

Navy Environmental Health Center





NAF Atsugi, Japan Human Health Risk Assessment

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Navy Environmental Health Center Environmental Programs Directorate

NAF Atsugi, Japan Final Human Health Risk Assessment Summary of Findings, Health Effects Evaluation and Conclusions June 2002

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Preface

The Shinkampo Incineration Complex (SIC), which began operation in 1985, was successfully shut down in April 2001. This document is the summary of activities conducted by the Navy Environmental Health Center (NEHC) and its contractors to address health concerns at Naval Air Facility (NAF) Atsugi, that were related to the operation of the adjacent SIC (also called Envirotech and Jinkampo Incineration Complex).

Multiple air quality studies were conducted from 1989 until the SIC was closed in April 2001. All of the air quality studies, except the one used for the final health risk assessment (HRA), were designed for "compliance purposes". That is, they were used to dispute SIC permit compliance in diplomatic negotiations with various Japanese governmental agencies and in legal action against the SIC owner. The U. S. began negotiations with the Kanagawa Prefectural Government in 1989, the U.S. - Japan Environmental Subcommittee in 1990, and the Status of Forces Agreement Japan Joint Committee in 1996.

A screening HRA was performed by NEHC in 1995, using air quality data collected for compliance purposes in 1994. This screening HRA indicated that the air quality at NAF Atsugi could raise the excess lifetime cancer risk to levels higher than the U.S. Environmental Protection Agency's (USEPA's) acceptable cancer risk range (i.e., 1 in 10,000 to 1 in 1,000,000 excess cases of cancer) for children (under the age of six) spending a normal three year tour of duty at NAF Atsugi.

In May 1997, the Navy Inspector General (NIG) noted increasing concerns among residents over the apparent lack of progress of diplomatic negotiations, which elevated efforts to resolve the situation. As a result, the Commander Naval Forces Japan (CNFJ) and NAF Atsugi requested that NEHC conduct a full HRA. Prior to initiating sampling for the full HRA, the Navy conducted another air quality study, during July through September 1997, to support increased diplomatic negotiations with the government of Japan (GOJ). The increased diplomatic negotiations resulted from the SIC owners request to renew their operating permit and expand their hours of operation. The data was used to develop a letter of objection to the GOJ in an effort to revoke the SIC's license vice renew it and allow for expanding operating hours. In August 1997, the Kanagawa Prefectural Government (KPG) renewed the SIC's license and

allowed for operating hours to increase from 8 hours each day to 24 hours each day. The Commander in Chief, U. S., Pacific Fleet (CINCPACFLT) requested NEHC to conduct another screening HRA with 1997 air quality data to assist with negotiations. The second screening HRA supported the first with regard to indicating a similar level of concern for calculated cancer risk and non-cancer health effects in the exposed population.

These screening assessments were pivotal in focusing the concerns at NAF Atsugi from those related to compliance with government regulations to potential health concerns for our NAF Atsugi community, as well as the local Japanese community. However, data collected for compliance purposes are collected differently than that collected to assess health risk related to potential exposures of different groups of individuals (e.g., children, adults, pregnant women, etc.). Some of the areas in which they differ can include the length of time and period that sampling is performed, the location at which samples are collected, and the detection level of the sampling method used. For compliance monitoring, one is merely trying to indicate that a certain standard has been exceeded for the time period for which the standard is expressed; however, for health risk assessment purposes, the data collected must be representative of an individual's exposure. That is why these initial health risk assessments were considered to be screening assessments, only.

In October 1997, the Bureau of Medicine and Surgery (BUMED) was given the lead to conduct a comprehensive HRA. Sampling for the assessment was conducted from March 1998 until July 2000. There were many challenges/limitations encountered in conducting the HRA as a result of the logistics presented on foreign soil. The most significant challenge/limitation resulted from the inability to conduct stack monitoring and the constraints to locate air monitoring equipment only on base property, for sampling site security/data integrity, as well as political ramifications that developed between Japan and the U.S. due to the operation of the SIC. Sampling was conducted to collect representative data that is spatially and temporally distributed over various seasons and various weather and incinerator operating conditions. As a result, this final and comprehensive HRA is the only document that contains representative data that can specifically address potential health impacts related to the operation of the SIC, on specific population groups who worked, lived or went to school at NAF Atsugi. Since the SIC has been closed, health effects related to its "air" pollutants resulting from its operation are not applicable to future populations at NAF Atsugi. However, the results of "soil" sampling are still

applicable to both current and future populations. Considering that thousands of military personnel and their families that were stationed and lived at NAF Atsugi from 1985 until April 2001, when the SIC discontinued operations, this final and comprehensive HRA is vital in responding to current and future concerns of military personnel and their families regarding health conditions associated with previous exposure to the soil and ambient air quality at NAF, during their tour(s) of duty. It is important to note that the risks addressed in this HRA for soil can be related to soil conditions during SIC operation for past, current and future populations.

Several different Standard Form 600s (SF 600s) were used at NAF Atsugi to respond to concerns from NAF Atsugi military personnel and their families about medical documentation and full disclosure of their potential exposure and possible health effects. All SF 600s are to be retained in personnel and family permanent health records. The Branch Medical Clinic (BRMEDCLINIC), with BUMED approval, developed a SF 600 that listed the maximum sampling concentrations measured in 1994 for 12 chemicals exceeding USEPA or New York State ambient air quality standards during the air quality study conducted by NFESC. Cancer risks were also provided on this SF600. Beginning 1 March 1996, this SF 600 was inserted in medical records of all individuals that requested the documentation. During health risk communication and consultation at NAF Atsugi, which began in June 1998, a revised SF 600 was completed for every individual at NAF Atsugi and those with orders to NAF Atsugi. This SF 600 documented full disclosure of potential exposures and possible health effects, related to environmental conditions, for each military member and/or family member based upon their medical history. The SF 600 was signed by each adult family member (18 years and older) to acknowledge receipt of risk communication. The sponsor or spouse signed the SF 600 for children under the age of 18. Additionally, all service members and family members over the age of 17 indicated that they received a risk communication briefing by signing an "Administrative Remarks NAVPERS 1070/613 (Rev. 10-81)," commonly referred to as a "Page 13" entry to be retained in their military record. Prior to PCS departure from NAF Atsugi, another SF 600 form was completed (SF-600 PCS Departure from NAF Atsugi) to document arrival and departure dates and locations of residence, schools attended and employment, while assigned to NAF Atsugi.

This final HRA is the only document that can be used to answer current and future questions concerning potential exposures of individuals living and/or working at NAF Atsugi

during the operation of the SIC. Additionally, it is the only document that can be used to respond to questions concerning soil exposures for the many individuals to be stationed at NAF Atsugi in years to come.

In addition to completing the two screening HRAs and this final HRA, BUMED, NEHC and BRMEDCLINIC Atsugi provided a tremendous level of support to assist with assessing and communicating health issues related to the operation of the SIC and responding to a multitude of requests for information. This support included responding to medical concerns of military personnel assigned to NAF Atsugi and those with orders to NAF Atsugi; responding to Executive and Congressional inquiries received by various offices within the Department of Defense (DOD) and other federal offices such as the House and Senate Armed Services Committees; conducting briefings for U. S. and Japanese officials, INCONUS and OCONUS, concerning health effects related to the SIC; conducting health studies to respond to community concerns; developing a health risk communication and health consultation plan; developing fact sheets and posters for public availability sessions; responding to frequently asked questions; communicating health risks related to SIC operation to every adult community member at NAF Atsugi and conducting health consultations for those families considered to be at a higher level of risk. A detailed description of Navy Medicine's involvement with the NAF Atsugi issue follows.

History of Medical Involvement

NEHC Conducts First Screening Health Risk Assessment

At the request of the Commanding Officer (CO), NAF Atsugi, in 1994, NEHC conducted a screening HRA with data collected by Naval Facilities Engineering Services Center (NFESC). A combined NFESC air quality study and NEHC HRA report was provided to CO, NAF Atsugi in October 1995. The National Academy of Sciences Committee on Toxicology (NAS COT) and the USEPA validated the screening health risk assessment report. After conducting the initial screening health risk assessment in 1995, NEHC actively participated, during 1996, in an informal working group with Chief of Naval Operations (CNO) and BUMED to discuss the results of the assessment and potential actions.

NEHC Briefs the 1995 Screening Health Risk Assessment

In May 1997, the NIG noted increasing concerns among residents over an apparent lack of progress to mediate the threat, of the SIC, which fueled heightened efforts to resolve the situation. Simultaneously, Commander Naval Forces Japan (CNFJ)/NAF Atsugi requested that NEHC conduct a full HRA and provide medical and epidemiological assistance. In May 1997, NEHC immediately responded to the request for assistance and traveled to Japan, within days, to brief the results of the 1995 combined air quality study and screening risk assessment to key U.S. and GOJ officials, to assist in plan development for a full HRA, to determine the status of medical surveillance conducted, and to determine epidemiological data needs. BUMED and NEHC provided many briefs to various individuals throughout this period. Some of the initial briefs to specifically address the findings of the 1995 screening HRA were as follows:

Commanding Officer, NAF Atsugi—CAPT Sweigert on 3 June 1997

Officer in Charge, BRMEDCLINIC Atsugi—CDR Halliwell on 3 June 1997

Commander, U. S. Forces Japan; USFJ—Major General Murray on 4 June 1997

Commander Naval Forces Japan—RADM Haskins on 5 June 1997

Government of Japan Officials (at the U. S. - Japan Joint Committee; Environmental Subcommittee) on 6 June 1997;

Commander in Chief, U. S. Pacific Fleet—RADM Church on 11 June 1997

Bureau of Medicine and Surgery—VADM Koenig, RADM Fisher and RADM Engel on 18 June 1997

Assistant Secretary of the Navy (ASN I&E)—Mr. Pirie on 19 June 1997

Assistant Secretary of the Navy (ASN E&S)—Ms. Elsie Munsell on 19 June 1997

Medical Inspector General—RADM Sanford on 29 September 1997

Navy Medicine Participates in Formal Working Group

Chief of Naval Operations (CNO) established a formal working group (by CNO ltr 5090 Ser N45E/7U595858 of 9 Jun 97) to determine short and long-term solutions to the SIC issue. The working group included staff members of Chief of Naval Operations (CNO—N1, N4, N8),

CINCPACFLT, BUMED, Bureau of Naval Personnel (BUPERS), Chief of Information (CHINFO), Commander Naval Facilities Engineering Command, Pacific Division (COMNAVFACENGCOM, PACDIV), NIG, Commander Naval Forces Japan (CNFJ), NEHC and NAF Atsugi.

BUMED Officially Given Lead on Comprehensive Health Risk Assessment

In October 1997, BUMED was given the lead to conduct a comprehensive health risk assessment. However, sampling could not be initiated until March 1998, as a result of CNFJ actively conducting compliance monitoring in an effort to develop data for a Letter of Objection, presented to the GOJ on 22 August 1997, to show compelling reasons why the SIC's operating permit should be revoked. However, on 31 August 1997, the Kanagawa Prefectural Government (KPG) not only renewed the SIC's license, but expanded the operation, allowing for 24 hours per day operation vice 8 hours per day. In addition, the KPG was entertaining the request to increase the allowable hazardous waste materials burned from 30 tons per day to 90 tons per day.

BUMED Participates in Flag Officer Working Group

In November 1997, the formal working group gave way to a Flag Officer Working Group from the same Navy Commands. The Bureau of Medicine and Surgery (BUMED), Deputy Surgeon General (DSG) became Navy Medicine's spokesperson.

NEHC Participates in Public Availability Session

The renewal of the SIC's license resulted in escalating concerns for the NAF Atsugi community, as it precluded hope for a reduction in emissions. At the request of CNFJ and NAF Atsugi, NEHC again traveled to NAF Atsugi to discuss the findings of the screening HRA with community members. Fact sheets and posters were developed by NEHC to address the health risks during a public availability session held on 20 November 1997. Approximately 300 community members were in attendance. NAF Atsugi environmental personnel responded to environmental questions, GOJ negotiation status and other NAF Atsugi issues. BUMED, NEHC and BRMEDCLINIC Atsugi representatives were available to respond to community health concerns that primarily dealt with potential health effects of pollutants on children, asthma and other respiratory effects, rashes, effects on nursing mothers, and effects on pregnant women and fetuses.

NEHC Conducts Second Screening Health Risk Assessment

CINCPACFLT requested NEHC conduct a second screening HRA with the compliance air quality data collected to develop the Letter of Objection. This second screening HRA was again provided to the USEPA and NAS COT for peer review and was once again validated by both organizations.

BUMED Directed to Develop Health Risk Communication and Health Consultation Plan

In April 1998, Dr. Bernard Rostker, Assistant Secretary of the Navy (ASN) for Manpower and Reserve Affairs, directed BUMED to take the lead in developing a comprehensive Health Risk Communication and Health Consultation Plan for NAF Atsugi. The plan was extensively coordinated with CINCPACFLT, CNFJ, NAF Atsugi, BRMEDCLINIC Atsugi and BUPERS. The purpose of the plan was to set forth implementing procedures to provide formal risk communication to everyone on board NAF Atsugi and personnel with orders to Atsugi. It also set forth implementing procedures to conduct mandatory health consultations for high-risk individuals assigned to NAF Atsugi. The plan was designed to allow Navy personnel and their families to make informed choices for themselves and/or their families.

The Health Consultation Plan required that Navy Detailers mention the air quality issue and refer military members to medical and base points of contact for further information. It required that overseas medical screeners discuss the health risks and provide a focused health consultation for individuals with orders to NAF Atsugi and a fact sheet that addressed potential risks of living and working at NAF Atsugi. A phased approach was established to inform individuals of potential risks to adults and children living or working at NAF Atsugi.

"Phase One" was conducted June through August 1998. In this phase, mandatory health risk communication was provided to all adults at NAF Atsugi over a three-month period. Briefings were provided to groups of not more than 50 people. Medical personnel from BUMED and NEHC discussed the results of the 1995 screening health risk assessment. One on one health consultations were mandatory for all adults extending for more than six years on station, all adults who had children under the age of six, those with chronic respiratory conditions and pregnant or nursing women. Other individuals received a health consultation upon request. This phase required a team of active duty and contract medical personnel arranged through BUMED.

The health consultations provided an opportunity for anyone to ask questions concerning their individual health and/or environmental exposure.

"Phase Two" was initiated in August 1998. This phase integrated the health risk communication into the "Orientation Week" for new arrivals and provided one on one health consultation for anyone not receiving it at the time of the medical overseas screen. It also established a SF 600 NAF Atsugi Departure Questionnaire with specific information regarding the locations of an individual's or family member's residence, employment and school during their tour(s) at NAF Atsugi, for entry into their medical record. NAF Atsugi and BRMEDCLINIC Atsugi supported this phase.

The Health and Environmental Risk Communication Plan also addressed the means for providing information to the community (e.g., base newspaper articles, public availability sessions, fact sheets, web sites, library repositories). Implementing this plan was the responsibility of NAF Atsugi.

NEHC Conducts Health Studies

To respond to NAF Atsugi community concerns, NEHC conducted two health studies, a Children's Respiratory Health Study (children at Yokosuka and those on and off-base at Atsugi) and a Pregnancy Loss Study for Women at NAF Atsugi. In January 1998, with the implementation of the Ambulatory Data System, BRMEDCLINIC Atsugi continued to monitor health status indicators for health effects that could be attributed to the SIC. There were 39 different International Classification of Disease (ICD) codes identified that could be related to air pollution to help assess whether there was any increase in morbidity due to the SIC.

NEHC Conducts Comprehensive Health Risk Assessment

Using the sampling data collected from March 1998 until July 2000, NEHC completed the comprehensive HRA. The USEPA and the NAS COT completed a thorough review of the draft final HRA. Their review comments are included as appendices to this NEHC summary report of findings and conclusions. Both organizations indicated confidence in the accuracy of the data collected and the quality of the sampling techniques. Both responses provided recommendations on certain aspects of the analyses and interpretation of the data. Recommendations were provided for addressing the degree of scientific uncertainties in the

numerical risk estimates, limitations of the risk assessment, and the need for additional risk characterization information to integrate the hazard identification, dose-response and exposure assessments by using a combination of qualitative, quantitative and uncertainty information to accompany the numerical risk estimates. NEHC responded to each of the comments and recommendations made by the peer reviewers. NEHC responses follow the particular comment or recommendations made by each peer reviewer and are included as appendices to this summary report.

As a result of the peer reviews, some changes were made in the risk assessment report. The USEPA and NAS COT comments and recommendations are reflected in this NEHC summary report of findings and conclusions. Changes to the draft report primarily included extensive risk characterization and the significant addition of text to respond to specific judgment statements made by the NAS subcommittee, published by the National Research Council in 2000. These judgment statements, such as those addressing the adequacy of statistical analysis and planning, resulted because of information not being readily available to the NAS COT, during their review. Given the sensitivity of the ongoing Department of Justice legal actions with the Government of Japan concerning the NAF Atsugi issue, the Navy declined meetings with the NAS COT as a result of their open nature (e.g., open to the media), so as not to compromise the legal actions.

The report that follows is a summary of the findings and conclusions of the work conducted by NEHC and all of its contractors. This comprehensive HRA is the only document that contains representative data that can specifically address future questions regarding potential health impacts related to the operation of the SIC on specific population groups who previously worked, lived or went to school at NAF Atsugi. Additionally, at this time, it is the only document that can be used to respond to questions concerning soil exposures, related to SIC emissions, for the many individuals to be stationed at NAF Atsugi in the future.

Public Health Summary

Background

The Navy Environmental Health Center (NEHC) completed a comprehensive human health risk assessment at Naval Air Facility (NAF) Atsugi, Japan. The purpose of the health risk assessment was to estimate the potential human health risks to Navy personnel and their families resulting from exposure to chemicals in the:

- ambient (outside) air,
- indoor air,
- indoor dust, and
- soil

that could be related to ambient air emissions at NAF Atsugi. The risk assessment also investigated how much of the overall health risk could be caused by the nearby Shinkampo Incineration Complex (SIC).

Prior to this comprehensive health risk assessment, two screening health risk assessments were conducted by NEHC in 1995 and 1998. Both screening assessments indicated that the air quality at NAF Atsugi could pose an unsafe risk to human health using U.S. Environmental Protection Agency (USEPA) regulatory guidance. The National Academy of Sciences and the USEPA reviewed each screening assessment. Both agencies supported NEHC's recommendation to conduct a comprehensive health risk assessment to provide the additional information needed to fully assess health risks at NAF Atsugi.

Risk assessors estimated the health risk using the methodology from the Superfund Risk Assessment Guidance for Superfund (EPA 1989). Air sampling data, collected over a 14-month period, and soil samples were used to calculate both cancer and non-cancer health risk estimates.

Human Health Risk Assessment Results

The results of the comprehensive health risk assessment were similar to the results of the two previous screening risk assessments. The cancer and non-cancer risks were higher for children of 0-6 years of age, than for adults because of children's typical hand-to-mouth behavior, and their play activities closer to the ground, which increases the amount of soil and

dust that they are likely to ingest. Since they are smaller, they get higher chemical doses per body weight.

Cancer Risk

Cancer risk is an estimate of how exposure to a chemical may increase the normal or expected (background) rate of getting cancer in a certain population of people. Generally, for risk management purposes an upper bound cancer risk (highest expected to occur) is calculated. USEPA considers an increase of "more than" 1 additional case of cancer (or greater) in 10,000 people (1×10^{-4}) to be unsafe. An increase to lifetime cancer risk of 1 case in 1,000,000 people or less (1×10^{-6}) is considered safe. The USEPA generally considers the range between 1 in $10,000 \ (1 \times 10^{-4})$ and 1 in 1,000,000 (1×10^{-6}) as a safe range and actions to reduce the risk may or may not be required based on the various factors specific to that site. USEPA typically considers additional actions, including regulations, to reduce risks that are close to or greater than 1 in $10,000 \ (1 \times 10^{-4})$ people.

Child

The calculated cancer risk for children (under the age of 6) living on base for a 3 or 6-year tour of duty suggested that a child's exposure to contaminants from air and soil during a 3-year tour of duty could potentially result in a cancer risk greater than 1 in 10,000 (10⁻⁴ level) above the current rate of cancer in the U. S. population during their lifetime.

Adult

The calculated cancer risk for adults living or working on base for a 3 or 6-year tour of duty suggested that an adult's exposure to contaminants from air and soil falls within the increased cancer risk range of 1 in 10,000 and 1 in 1,000,000 above the current rate of cancer in the U. S. population during their lifetime.

Non-Cancer Health Effects

Non-cancer health effects are expressed by a number known as the "hazard quotient" or "HQ." The HQ is a ratio that compares the amount of a chemical that people may have been exposed to over a specified time period with the amount that is considered to be safe. If people have been exposed to an amount greater than that considered safe for a particular chemical, then the ratio is greater than one. Because people can be exposed to more than one chemical at a

time, the HQs for different chemicals are added together to give an overall "Hazard Index," or "HI, unless data is available to indicate that they should not be added. It is important to keep in mind that a HI is NOT a probability that health effects will occur. USEPA policy considers chemical concentrations resulting in a HI above 1 to be of concern for developing potential non-cancer health effects. It is not a bright line that triggers action to reduce the exposure but the greater the number the greater the level of concern about health effects. Professional judgment must be used to evaluate the potential non-cancer health effects related to the concentration of these chemicals to determine if actions to reduce the risk are needed.

Child

All Hazard Indexes calculated for children were greater than 1, and are therefore at a level of concern for non-cancer health effects, according to USEPA policy.

Adult

All Hazard Indexes calculated for adults were greater than 1, and are therefore at a level of concern for non-cancer health effects, according to USEPA policy.

Acrolein, acetaldehyde, PM₁₀ and formaldehyde contribute approximately 91% of the potential non-cancer health effects at NAF Atsugi. Health effects related to the individual chemicals that cause respiratory effects, generally, are reversible when an individual leaves NAF Atsugi. However, there is some concern that repeated long-term exposure to chemicals, in combination with others, might result in long-term, non-cancer health effects. In our professional judgment, a closer evaluation of the concentrations for non-cancer health effects of the various chemicals measured at Atsugi indicates that one air pollutant, acrolein, may cause long-term and short-term adverse respiratory effects.

The cancer and non-cancer risks were significantly higher downwind from the SIC. In addition, air dispersion modeling results and the pattern and amount of dioxin found in soil are further indications that emissions from the SIC have a significant contribution to the air quality at NAF Atsugi. An incinerator in the United States with uncontrolled emissions and similar levels of dioxin, as those emitted by the SIC, would have been forced to either shut down or implement controls to reduce the potential for adverse health effects to occur.

This report is a summary of the results of the investigations that have been performed and it was written to assist the risk managers in making decisions regarding the protection of the

health of our military and civilian personnel and their families living at NAF Atsugi. Because risk assessments use many assumptions and estimates, the final risk numbers always contain some uncertainty. The risk could actually be over- or underestimated. Because of this, the numbers need to be interpreted with caution, as they could be as high as the estimated risk number or as low as zero. In the U.S., risk assessment results similar to those found at NAF Atsugi would result in additional USEPA regulatory action to lower the estimated risk..

Formal Risk Communication Plan

In April 1998, Dr. Bernard Rostker, Assistant Secretary of the Navy (ASN) for Manpower and Reserve Affairs, directed BUMED to take the lead in developing a comprehensive Health Risk Communication and Health Consultation Plan for NAF Atsugi. The plan was extensively coordinated with CINCPACFLT, CNFJ, NAF Atsugi, BRMEDCLINIC Atsugi and BUPERS.

The purpose of the plan was to implement procedures to ensure everyone on board NAF Atsugi and personnel with orders to NAF Atsugi, were aware of the poor air quality issue at NAF Atsugi. It established the details for providing information to the community (e.g., base newspaper articles, public availability sessions, fact sheets, web sites, library repositories). The plan also set forth procedures to conduct mandatory health consultations for high-risk individuals assigned to NAF Atsugi. The plan was designed to allow Navy personnel and their families to make informed choices for themselves and/or their families. The plan established responsibilities for specific Navy commands, as follows:

BUPERS

- Ensured each military member negotiating orders to Atsugi were aware of the air quality issue and referred them to medical and base points of contact for further information.
- Approved Voluntary Relocation of existing personnel.
- Informed personnel with PCS assignment to NAF Atsugi by providing facts sheets developed by NEHC and providing BUMED points of contact to answer health related questions.

• Developed a service record review worksheet to assist commands in identifying other priority recipients based on the length of time assigned to NAF Atsugi.

BUMED

- Provided health risk communication to all adults at NAF Atsugi over a three-month
 period and for incoming and outgoing personnel to ensure that everyone was given the
 opportunity to make informed decisions about their family health management.
- Conducted one-on-one health consultations for all adults extending for more than six years on station, all adults who had children under the age of six, those with chronic respiratory conditions and pregnant or nursing women.
- Developed a standard entry on a SF-600 form describing potential exposure conditions at NAF Atsugi for input into medical records.
- Conducted two epidemiological studies to ascertain the health status of children and pregnant women.

Branch Medical Clinic Atsugi

- Provided health care with a heightened awareness of the potential effects relating to the poor air quality.
- Participated in the one-on-one health consultations and presentations made during the risk communication briefs.
- Reviewed medical records/conducted health consultations for personnel and family members reporting to and departing NAF Atsugi.

NAF Atsugi

- Implementing this risk communication plan locally was the responsibility of NAF Atsugi.
- Established a Risk Communication Office to help implement the plan.
- Established an information section in the base library where interested parties could obtain information on studies conducted to evaluate health risks.

 Held monthly health risk communication briefs for current residents and incoming personnel.

Navy Actions To Reduce Exposure

From the time the results of the 1995 Screening Health Risk Assessment indicated a concern that the air quality at NAF Atsugi could pose an unsafe risk to human health, NAF Atsugi and the highest levels of the Navy and U.S. Government officials were committed to protecting the health and well being of their military personnel and their families stationed at NAF Atsugi. The Navy continued to take actions to protect their health by implementing the following actions.

Local Actions by NAF Atsugi and CNFJ

- Requested medical assistance from Branch Medical Clinic Atsugi and Navy Environmental Health Center.
- Initiated diplomatic efforts with Government of Japan to implement pollution control measures and/or shutdown the Shinkampo Incinerator.
- Provided residents with portable air cleaners to improve indoor air quality from potential infiltration of incinerator emissions into the home environment.
- Established an Air Quality Advisory/Warning System that warned residents to curtail outside activities indoors when emissions from the Shinkampo were blowing on base.
- Conducted training for childcare providers and schoolteachers to educate them
 about soil contamination and actions to reduce exposure by washing children's
 hands, toys and keeping them indoors when emissions from the Shinkampo were
 blowing on base.
- Established the Shinkampo Action team to support the legal efforts.
- Offered Voluntary Relocation of existing personnel.

Department of the Navy

- CNO established a formal flag-level working group to address short and long-term solutions to the Shinkampo issue. The working group included staff members of Chief of Naval Operations, Commander in Chief, U. S. Pacific Fleet, Bureau of Naval Personnel, Navy Office of Information, Commander Naval Facilities Engineering Command, Pacific Division, Naval Inspector General, Commander Naval Forces Japan, Bureau of Medicine and Surgery, Navy Environmental Health Center and NAF Atsugi.
- Conducted legal action in concert with the U. S. Department of Justice to pursue the shutdown of the Shinkampo Incinerator, which was successfully accomplished in May 2001.

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Section 1 — Introduction

1.1 Background

In 1995, an air quality and impact study was conducted at Naval Air Facility (NAF) Atsugi, Japan by the Naval Facilities Engineering Services Center (NFESC), at Port Hueneme, CA, to evaluate potential health effects associated with exposure to emissions from the off-base neighboring Japanese owned Shinkampo Incineration Complex (SIC). The reason for the study was that community concerns had been raised at NAF Atsugi regarding health impacts due to the emission of air pollutants from the incinerator and also during the storage, handling and disposal of liquid and solid waste material on-site from the SIC. Base personnel and family members complained of headaches, nausea, skin rashes, irritation of the nose, throat and eyes, allergy-like symptoms, and an increase in the frequency and duration of existing asthma problems and upper respiratory disease.

At the request of the Commanding Officer, NAF Atsugi, the Navy Environmental Health Center (NEHC), through the U. S. Naval Hospital Yokosuka, Japan, performed an initial screening human health risk assessment (HRA) using the data collected by NFESC. The air quality data collected by NFESC was not intended for risk assessment purposes, as it was collected over a limited period of time, of short duration and air was the only medium sampled. Therefore, the HRA was considered to be a screening assessment. The results of this screening HRA indicated that the calculated cancer risk, associated with poor air quality at NAF Atsugi, was higher than U.S. Environmental Protection Agency's (USEPA) upper range of acceptable cancer risk of 1 in 10,000 for children living on base for 3 years duration. The HRA also indicated that there was concern for children and adults, who lived on base, of developing non-cancer health effects as a result of the calculated hazard index being greater than the USEPA acceptable hazard index of 1.

NEHC was requested to conduct a second health risk assessment with data collected by Earth Tech Environmental Corporation (Earth Tech) in 1997. The data collected by Earth Tech was collected to address compliance issues, as a result of the SIC owner's request to modify the operating permit to allow for an increase in operating hours and throughput. Therefore, the second health risk assessment was also a screening assessment. The results of the second health screening HRA, conducted in 1998, were consistent with the first screening HRA.

Both screening health risk assessments were reviewed by the National Academy of Sciences (NAS), Committee on Toxicology (COT) and the USEPA. Recommendations were made in both peer review reports to conduct a comprehensive HRA. Consequently, the Navy Bureau of Medicine and Surgery (BUMED) was given the lead to conduct a comprehensive health risk assessment at NAF Atsugi. As the Navy's designated health risk assessors for BUMED, NEHC served as the project and technical managers for the risk assessment.

Following completion of the screening health risk assessments, NAF Atsugi initiated risk communication activities. These activities addressed diplomatic efforts to close or modify operations of the SIC, findings of the screening HRAs, interim risk reduction measures to mitigate the potential impact of the air quality on the health of people living in the residential areas and plans for performing the comprehensive HRA to characterize more completely the health risks. The *Comprehensive Risk Communication and Health Consultation Plan for Naval Facility Atsugi, September 1998* was written by the Navy Bureau of Medicine and Surgery (BUMED), at the direction of Dr. Bernard Rostker, Assistant Secretary of the Navy (ASN) Manpower and Reserve Affairs (M&R). Dr. Rostker directed BUMED to take the lead in developing the plan, in April 1998. BUMED extensively coordinated the plan with Commander in Chief, U.S. Pacific Fleet; Commander Naval Forces Japan; Naval Air Facility Atsugi; Branch Medical Clinic Atsugi and Bureau of Naval Personnel. Dr. Vincent Covello, Center for Risk Communication, New York, New York, validated the plan. The plan is attached to the report as Appendix A.

The purpose of the plan was to set forth implementing procedures to provide formal risk communication to everyone on-board NAF Atsugi as well as personnel with orders to NAF Atsugi. It also set forth implementing procedures to conduct mandatory health consultations for high-risk individuals assigned to NAF Atsugi. This plan was designed to provide the best possible and most comprehensive health risk communication and health consultation available so as to allow Navy personnel and their families to make personal and informed decisions.

The comprehensive health risk assessment is addressed in this report. The objectives of this health risk assessment were to:

1. Estimate the potential human health risks to U.S. Navy personnel and their families and other individuals living and working on NAF Atsugi, Japan, resulting from

exposure to constituents of concern (COCs) in soil, ambient air, indoor air, and indoor dust. This estimate focuses solely on COCs that are likely to be associated with ambient air emissions and/or subsequent deposition from point and non-point sources impacting the air quality at NAF Atsugi.

2. Estimate the contribution of the risk attributable to the emissions from the Shinkampo Incineration Complex (SIC).

1.2 Site Description

NAF Atsugi

NAF Atsugi is located in the Kanto Plain area on the island of Honshu, Japan (Figure 1-1). The Japanese Navy constructed the base in 1941 and it was commissioned in 1950 as U.S. Naval Air Station Atsugi. In 1971, the name of the base was changed to Naval Air Facility (NAF) Atsugi and the official joint use of the base with the Japanese Maritime Self Defense Force (JMSDF) began. NAF Atsugi's primary mission is to support the 120 aircraft and 8,000 U.S. military and civilian personnel, their family members, and Japanese employees of Carrier Air Wing FIVE (CVW 5), Helicopter Anti-Submarine Squadron Light FIVE ONE (HSL 51), and 40 tenant commands. NAF Atsugi is the only U.S. naval air base to support all seven aircraft types that compose a typical carrier air wing, attached to the U.S. Navy's only forward-deployed carrier battlegroup.

At the time of this risk assessment, the NAF Atsugi population was approximately 7,500, comprised of active duty, civil service, and foreign Nationals of which 81% was composed of active duty members and their dependents, 1% were Department of Defense employees such as teachers and their dependents, 5% were Civil Service employees and their dependents and 13% were Master Labor Contractors, including Japanese nationals. The U.S. Navy operates NAF Atsugi jointly with the JMSDF, whose personnel add another 2,200 active duty and 1,800 dependents living in family housing units on the base.

Approximately 75% of the population lived on base and 25% off base. It is estimated that approximately 6,000 of the 7,500 were adults. The family housing area, within a kilometer of the SIC, was home to approximately 3,500 U.S. active duty, U.S. Civil Service employees and their family members. There are approximately 446 dependents under 6 years of age and about 916 dependents between 6 and 18 years of age living on base versus 129 and 180, respectively, living off base. Active duty members were typically out to sea for 4.5 months per year.

Shirley Lanham Elementary School educated over 800 students, and a Child Development Center (CDC) cared for over 200 pre-school age children on the base. Middle school and high school students (12 – 18 years) were bussed daily during the school year (September – June) to Camp Zama, which was located several miles from the SIC. The normal tour of duty was 3 years, but it could be extended additional tours of duty several times. Some military and civilian members, particularly those with Asian spouses or Asian backgrounds, have chosen to remain for additional tours.

Figure 1-2 presents the layout of NAF Atsugi at the time of this risk assessment. The base occupied approximately 500 hectares and was level except for a small ravine formed by the Tade River which generally runs north-south and divided the facility into east and west sectors. Residential areas, including Residential Towers and townhouses, are located on the southern and western portions of the base, northwest of the SIC. Buildings 3101 and 3102, located within 300 meters from the incinerator, opened for occupancy in May 1996 and May 1997, respectively. Both towers, which were the closest towers to the incinerator complex, were sited and constructed before the 1995 screening HRA report documented the level of health risks. An Elementary School, youth center, and a day care center were located near the residential areas on the south side of the base, within 500 meters from the SIC. The parade grounds, two gymnasiums, softball fields, volleyball courts, a nature trail, and various parks were located in the western sector. Recreational areas on the eastern sector included the Golf Course and shooting range. The Golf Course extended to the east from the fence line of the base located at 200 meters from SIC up to the flightline, and to the north end of the base located 2300 meters from the SIC. Aviation-related facilities where petroleum products, oils and lubricants are stored or used (such as the aircraft maintenance shops) and the runway occupied the eastern sector of the base.

Shinkampo Incineration Complex

The SIC, shown in Figures 1-3a and 1-3b, was a private owned Japanese waste and combustion disposal facility. NAF Atsugi is located at a slightly higher elevation adjacent to the SIC, which is situated in the Tade River valley approximately 150 meters south of the NAF Atsugi fence line. Three incinerators were located inside this Complex, which was approximately 4 to 5 acres (2 hectares) in size. The complex was comprised of three incinerators and areas for storing and staging ordinary and specially controlled liquid and solid industrial

waste. It also contained facilities for refuse-derived fuel. The incinerator stack heights were only slightly higher than the plateau where NAF Atsugi was located. Base personnel have made numerous visual observations of the SIC visible emissions at ground level near the Ground Electronics Maintenance Building (GEMB), immediately north of the SIC, when the winds are brisk and from the south. Environmental concerns have been raised at NAF Atsugi regarding the emission of air pollutants resulting from this fumigation condition and through fugitive emissions during the storage, handling, and disposal of liquid and solid waste material at the SIC.

An air photo, presented in Figure 1-4, shows the spatial relationship of the SIC to the airfields and surrounding industrial and urban areas. South and west of the complex is the Ayase Industrial Park. Using visual observations and permit descriptions, incinerator and stack design experts have determined that the incinerators were configured similar to multiple chamber starved-air/controlled-air type incinerators. Waste appeared to arrive at the SIC in dump trucks, flatbed trucks, and municipal waste dumpsters (packers). The dump trucks tended to carry industrial construction or demolition waste. Flatbed trucks were observed carrying barrels of undetermined substances. The packer-type dump trucks were presumably carrying waste paper and waste plastics from industrial facilities. Wastes were stockpiled at the southern end of the complex until they could be processed. A shredder was located west of the incinerators for larger materials to be processed. Near the incinerator, materials including solids and liquids were laid out on a concrete pad for additional mixing, sorting, and material removal prior to incineration. It appeared that the incinerators were equipped with a quench system, and it was suspected that waste alkaline materials were used as scrubbing materials. Cyclones were also present to control dust; but the visible plumes from the SIC stacks indicated that they were adequate for controlling only the smaller particulate matter.

In April 1985, the Kanagawa Prefectural Government issued the SIC an intermediate ordinary industrial waste disposal license. It authorized the operation of three incinerators for the intermediate disposal of municipal and industrial wastes. The SIC subsequently replaced the incinerators, one each in November 1987, July 1991, and April 1993. An intermediate specially controlled industrial waste disposal license became effective in December 1993.

Intermediate disposal included incineration, rock and concrete crushing, gravel manufacturing, mechanized waste separation, volume reduction, and solidification. Wastes that

could be disposed of or otherwise managed at the SIC included the following, based upon the SIC permit or visual observation:

- Size reduction: plastic, rubber, metal, glass, ceramics, and construction debris;
- Mechanical separation: plastic, paper, wood, fabric, rubber, metal, glass, ceramics, and construction debris;
- Solidification: ash, sludge, slag, and dust, including stabilization/neutralization areas;
- Separation: oily water;
- Incineration: sludge, non-chlorinated oil, acid, alkaline, paper, wood, fabric, animal and plant remains, and rubber;
- Incineration: refuse-derived fuel consisting of plastic, paper, and wood; and
- Incineration: infectious waste, including congealed blood, scalpels, hypodermic needles, and x-ray film.

Visual observations of the number of vehicles containing waste entering the SIC facility indicated that much more than the permitted amount of waste was being incinerated. The SIC typically shut down every week on Sunday afternoon at about 1600 hours and restarted again on Monday afternoon at about 1600 hours. During certain Japanese holidays, this shutdown schedule might be shifted to Monday afternoon.

The SIC was permitted to operate 24 hours per day with up to 10 tons of waste in each of the three incinerators. The facility requested an increase to 30 tons per day per incinerator. In September 1998, the Kanagawa Prefecture approved the SIC's permit amendment request to increase its incinerator throughput from 30 to 90 metric tons/day. The facility was permitted to burn 90 metric tons of waste per day during a 24-hour period. However, during the sampling period, the facility was requested by the Government of Japan (GOJ) to reduce the daily throughput to 30 metric tons per day. The actual throughput of the incinerators, during the sampling period, was not known. The Navy did not have access to the SIC records.

Emissions from the complex were primarily the result of the combustion of municipal and industrial waste. It may have included wood products, plastics, construction debris, solvents,

alkalis, acids, rubber, infectious waste (i.e. blood, soiled garments, used needles, human tissue, etc) and oil with explosive properties. Fugitive emissions also occurred from the stockpiles of waste and ash that were open to the atmosphere. Several factors related to the SIC contributed to the degradation of the air quality at NAF Atsugi. The discharge heights of the incinerator stacks were only slightly higher than the ground surface of the plateau on which NAF Atsugi was located. Since the SIC was located in a valley adjacent to the base and due to its short incinerator stack height relative to the base located on the plateau, emissions from the incineration complex were carried parallel to the stack height downwind towards the base, at ground level. In addition, as documented by video surveillance conducted by base personnel, the SIC bypassed air pollution controls, allowing throughput that was too high for complete combustion, dumped liquid wastes directly onto open piles of solid waste, and engaged in poor practices of bottom ash handling and storage procedures.

Historically, the SIC emissions generally had the greatest impact on air quality at NAF Atsugi during the late spring, summer, and early fall when the wind blows predominantly from the south toward the base, as depicted by the wind rose presented in Figure 1-5. A wind rose shows the wind speed as a function of wind direction. The "spokes" of the rose show the wind speed ranges for each of the 16 wind direction vectors. The wind speed ranges are shown as an increasingly thicker spoke that is proportional to the percent of time the wind was from that given direction. The other numeric value displayed for each "spoke" is the percent of time the wind blew from that wind direction vector for the specific time period.

During the sampling period the incinerators were equipped with pollution control equipment consisting of primary quench chambers, cyclone separators, secondary quench sections and 23-meter smokestacks, as shown in Figure 1-3b. Later, the SIC removed the cyclone separators and secondary quench sections, extended the primary quench chambers, and installed bag filters on all three incinerators. On April 30, 2001, the government of Japan purchased the SIC, shut it down and subsequently disabled it.

1.3 Screening HRAs

1995 Screening Health Risk Assessment

The first screening health risk assessment used data collected by NFESC for an air quality assessment. It was not intended for risk assessment purposes, as it was collected on only

nine different days during the time period between 26 July 1994 to 10 September 1994, for a short duration and the only medium sampled was outdoor air. Therefore, the health risk assessment (HRA) conducted was considered to be a screening assessment. NFESC performed ambient air sampling at two different sites on NAF Atsugi, a site predicted by air dispersion modeling to be the area of highest impact by the SIC and an upwind background site. The exposure point concentrations for volatile organic compounds (methylene chloride, chloroform, benzene, trimethyl benzene), furans (2,3,4,7,8-PeCDF_and 1,2,3,4,5,7,8-HxCDF) and metals (chromium III) were higher than the EPA Region III Risk Based Concentration (RBC) screening values for ambient air (EPA 2000b). Health risks were calculated for adults and children living on base for 3, 6 and 30 years. The results of this screening HRA indicated that the calculated cancer risk, associated with poor air quality at NAF Atsugi, was higher than U.S. Environmental Protection Agency's (USEPA) upper range of acceptable cancer risk of 1 in 10,000 for children living on base for 3 years. The screening HRA also indicated that there was concern for children and adults, who lived on base, of developing non-cancer health effects as a result of the calculated hazard index being greater than the USEPA acceptable hazard index of 1.

This screening HRA was peer reviewed by the USEPA and the National Academy of Sciences (NAS). The peer reviewers concluded that the report contained sufficient and compelling evidence to warrant public-health concern and to justify further evaluation of the problem. The reviewers also agreed with NEHC recommendations: (1) for interim measures to reduce the potential exposures of Navy personnel and their families, especially children, to emissions from the incinerator complex and (2) that the Navy conduct additional sampling of air and other environmental media to form the basis of a more comprehensive risk assessment.

1998 Screening Health Risk Assessment

NEHC was requested to conduct a second health risk assessment with data collected by Earth Tech in 1997. The data collected by Earth Tech was to be used for addressing compliance issues, as a result of the SIC owner's request to modify the operating permit to allow for an increase in operating hours and throughput. Therefore, the second health risk assessment was also a screening assessment. The data used in the screening risk assessment was for ambient air only, collected at nine different locations at NAF Atsugi on 23 different days from 27 July 1997 to 01 September 1997. These locations were selected based on the potential areas of exposure

for base personnel and their families, other previously sampled locations, as well as a new background location. These sites were: (1) Skeet Range, (2) Golf Course South, (3) Golf Course North, (4) Fenceline, (5) Ground Electronics Maintenance Building, (6) Residential Tower 3102, (7) Residential Tower 3101, (8) Playground/Picnic Area, (9) Child Development Center. Sites 2 to 9 were sampled between 27 July 1997 and 07 August 1997. It is important to note that not all sites were sampled at the same time during all 23 sampling days. Site 1 was sampled on 27 days, site 5 on 18 days, site 7 on 10 days, site 3 on 14 days and sites 2, 4, 6, 8, and 9 on 8 days during the same period. The background site; however, was sampled everyday that sampling was performed.

Cancer risks and non-cancer hazard indices were calculated for a residential scenario involving adults and children (0 to 6 years of age) living on the base for 3, 6 and 30 years, at all 9 locations. An industrial scenario was calculated for adults working at the Ground Electronics Building (location 5) for 3, 6 and 30 years. The limited sampling could not be used to fully characterize health risks to the Atsugi community. The detection limits for some of the chemicals were above the levels of concern, resulting in additional uncertainty in the estimation of the risk contributed by these chemicals.

The results of this second screening assessment were similar to the first screening assessment. The results of this screening HRA once again indicated that the calculated cancer risk, associated with poor air quality at NAF Atsugi, was higher than U.S. Environmental Protection Agency's (USEPA) upper range of acceptable cancer risk of 1 in 10,000 for children living on base for 3 years. It again indicated that the non-cancer acceptable exposure levels corresponding to a hazard index of 1 were exceeded for both children and adults living on base for a normal tour of duty. The hazard index ranged from 5.2 to 41 for children and 1.9 to 15 for adults. There were 11 chemicals that contributed at least 1% to the total cancer risk at one or more of the sites; 3 metals which included arsenic, cadmium, and beryllium; 7 volatile organic hydrocarbons, including formaldehyde, 1,2-dichloroethane, benzene, carbon tetrachloride, chloroform, vinyl chloride, chloromethane; and 2,3,7,8-tetrachloro dibenzo dioxin. Particulate matter less that 10 microns (PM₁₀) was responsible for the majority of the hazard index (non-cancer health effects) at each location. 1,2-dichlopropane and acetaldehyde also contributed to the hazard indices at some locations.

The 1998 screening HRA was also reviewed by the National Academy of Sciences Committee on Toxicology (NAS COT). On the basis of the data presented in this second screening HRA, the NAS COT concurred with NEHC's 1998 estimate of carcinogenic risk, which were similar to those calculated in 1995, even though the pollutants contributing to those risks were different. The reviewers agreed, with the NEHC report, that there were serious limitations in the air sampling and analysis methods, which were for compliance purposes and not for completing a HRA. They concluded that the data was insufficient to determine that the incineration complex was the major source of pollution contributing to the observed concentrations of airborne contaminants during the air-sampling period. They stated that such a determination would require better characterization of background concentrations. They suggested that a better approach would be to compare results from an upwind location with downwind locations on days when the wind direction was out of the south-southwest and relatively constant.

1.4 Comprehensive Health Risk Assessment

The Navy Bureau of Medicine and Surgery (BUMED) was given the lead to conduct a comprehensive health risk assessment at NAF Atsugi. NEHC selected the USEPA Risk Assessment Guidance for Superfund methodology to conduct the risk assessment for a number of reasons. The primary reason that this methodology was selected is because it is the standard (and required) approach used in the United States for calculating risk; therefore providing a basis for comparison with other risks calculated at DoD activities in the U. S. Additionally, USEPA risk assessment principles and practices draw on many sources, including the environmental laws administered by USEPA, the National Research Council's 1983 report on risk assessment, the USEPA's Risk Assessment Guidelines, and various program specific guidance (e.g., the Risk Assessment Guidance for Superfund). The regulatory framework for performing human health risk assessments has been established through a series of guidance documents issued by USEPA and other regulatory agencies since the early 1980s. Through these documents, guidelines for performing both qualitative and quantitative human health risk assessments have been defined. The guidelines provide a clear explanation of USEPA processes for evaluating hazard, dose response, exposure, and other data that are relevant to the development of the health risk assessment. The USEPA has also released specific guidance for applying the general human health risk assessment methodologies to incineration facilities. USEPA (EPA 1994a) guidance

for hazardous waste combustion facilities regulated under the Resource Conservation and Recovery Act (RCRA) specifies a two-step approach for assessing human health risks. The first step is a screening-level risk assessment performed to determine if a more detailed, site-specific evaluation of risk is warranted, as was conducted at NAF Atsugi. Finally, the USEPA has twenty years of experience in developing, defending, and enforcing risk assessment based regulation.

As the Navy's designated health risk assessors for BUMED, NEHC served as the project and technical managers for the comprehensive health risk assessment. In this capacity, NEHC managed the project and provided oversight for the:

- Sampling and analysis plan,
- Monthly and quarterly monitoring reports,
- Equipment and procedures audit reports,
- Final monitoring summary, and
- Health risk assessment.

To support NEHC in the more complete assessment of potential impacts of emissions from the SIC on human health, the Naval Facilities Engineering Command, Atlantic Division (LANTDIV), procured and provided management of the Radian International LLC (Radian) contract to execute the environmental monitoring. They also managed the equipment and performance audit contracts for the air monitoring systems. Radian conducted the environmental monitoring program for soil, and ambient and indoor air, from March 1998 through June 1999 (14 months) and prepared the interim monthly, quarterly and final monitoring summary reports. Pioneer conducted the comprehensive human health risk assessment at NAF Atsugi, Japan and also provided interim monthly and quarterly reports based on the results of the comprehensive sampling program carried out by Radian.

Workplan Development

This health risk assessment workplan was designed to collect data to meet the objectives of the comprehensive health risk assessment which were:

- 1. To estimate the potential human health risks to U.S. Navy personnel and their families and other individuals living and working on NAF Atsugi, Japan, resulting from exposure to constituents of concern (COCs) in soil, ambient air, indoor air, and indoor dust. This estimate focused solely on COCs that were likely to be associated with ambient air emissions and/or subsequent deposition from point and non-point sources impacting the air quality at NAF Atsugi.
- 2. To determine the contribution of the risk attributable to the Shinkampo Incineration Complex (SIC).

It is important to note that many challenges and limitations were encountered in conducting a project of this magnitude overseas. These challenges and limitations were significant from the beginning. They started with the air quality monitoring operation and logistics presented on foreign soil and the inability to conduct stack monitoring and the constraints to only locate air quality monitoring equipment within the confines of base property, for sampling site/data integrity security, as well as political ramifications that developed between Japan and the U.S. due to the operation of the SIC.

The comprehensive health risk assessment was initiated in January of 1998, when a site visit was conducted to assist in locating the outdoor air, indoor air and soil sampling sites that would potentially be used for data collection (Radian 1998). The site visit began on 12 January 1998, with an opening meeting with NAF Atsugi representatives and a bus tour of the base. The next two days were field days to observe site conditions and gather data. A wrap-up meeting to discuss our observations and propose the monitoring plan for this project was held on 15 January 1998. These activities are summarized in the site visit plan (Radian 1997).

Based on this site visit, evaluation of the data from the two previous screening risk assessments (1995 and 1998), recommendations from peer reviewers and consultation with professionals from the National Center for Environmental Assessment (NCEA), Washington, D.C. and the National Exposure Research Lab (NERL), Research Triangle Park, N.C., a sampling and Quality Assurance/Quality Control (QA/QC) plan was developed by Radian (Radian 1998). The sampling and QA/QC plan supported the field sampling and analytical efforts for collecting the data needed to prepare a comprehensive HRA, while reducing uncertainties related to exposure (e.g., collecting data representative throughout the year). The

plan addressed the collection of ambient and indoor air samples, meteorological data and surface soil samples, since these were media of concern determined by the previous screening HRAs. Surface water, groundwater and food pathways would have been incorporated later if they became a concern, as determined by an exposure pathway analysis (Radian 1998b) conducted soon afterwards. However, the exposure pathways analysis determined that surface water, groundwater and food were not completed pathways.

Because this comprehensive HRA was designed to support risk management decisions regarding the health risks at NAF Atsugi, it was critical to reduce, as much as possible, the uncertainties regarding sampling and analytical procedures. To reduce these uncertainties in the sampling methodology, sampling methods were selected based on their ability to collect samples with sufficiently low detection limits to perform health-based risk analysis. Ambient air sampling frequency was established to replicate an every-sixth-day sampling program commonly used in the United States (EPA 1998a) to measure particulate and air toxic compounds for one year. Thus, any variability due to the day of the week, season, or other temporal effects could be assessed. Soil sampling and indoor air sampling frequency were determined by statistical analysis based on historical data. The sampling data was evaluated throughout the monitoring period to ensure accurate analytical data capture and ensure that data quality objectives were being met. This was accomplished by quarterly data evaluations, meetings and peer reviews, equipment and procedures audits, and analytical lab audits. NEHC also conducted quarterly review meetings with the project team to continuously evaluate the data to ensure data quality objectives were met, and make any necessary changes in sampling and analytical procedures. Analytical data were evaluated monthly to monitor for potential conditions that would require interim actions to mitigate health risks (Radian 1998f – 1998j, 1999b – 1999e, 1999g - 1999h; and Pioneer 1998b – 1998d, 1999b – 1999i). Throughout the monitoring program and development of the health risk assessment, NEHC consulted with USEPA experts in various scientific fields. They provided the latest scientific information to address health concerns, as well as input on sampling and analytical methodologies, to ensure that the health risk assessment would be developed with the minimum degree of uncertainty.

If this project were undertaken in the U.S., stack emissions, incinerator feedstock characteristics, and incinerator operating parameters would have been obtained and evaluated to determine the impact of the incinerator on air quality and human health. However, since the SIC

was a privately owned Japanese incinerator complex, this information was not available. Consequently, a network of ambient air monitoring stations was established around the SIC, mainly on NAF Atsugi property (for equipment security and political reasons), to determine the impact of emissions from the SIC on human health.

The key uncertainties associated with collecting ambient air data were emission from other point sources (e.g., smaller incinerators and industrial companies) as well as non-point sources (e.g., vehicles), which made the task of identifying chemicals emitted by the SIC, and the determination of background concentrations, much more difficult. To account for variability in incinerator feedstock, operating parameters, meteorological conditions (e.g., wind direction, precipitation, inversions, etc), and temporal and seasonal changes, a 12-month sampling plan was instituted. Samples were collected at irregular intervals to prevent the SIC from modifying its operations and reduce emissions during sampling events. Methodologies and sampling rationale are described in greater detail in the sampling plan document (Radian 1998a).

1.5 Scientific Peer Reviews of the Comprehensive HRA

USEPA Peer Review

USEPA scientists reviewing the final HRA generally concurred with the study design, methodologies and conclusions of the Navy's risk assessment, especially given the limitations placed on the Navy and its contractors because the incinerator was inaccessible. The USEPA peer reviewers identified no fundamental problems in the two documents (NEHC 2000 and Pioneer 2000), although a number of improvements were suggested and questions posed regarding issues that were not adequately covered in the two documents. USEPA suggested that the Navy provide additional explanations in the HRA Summary to assist the reader in understanding the scenario development, data analysis and decision options, especially if this document (NEHC 2000) was intended as a stand-alone summary. USEPA comments mainly focused on the need to more succinctly summarize what was known about risk at the site, to identify which risks were most likely attributable to the SIC, and to specify the strength of the association of the various chemicals, measured in air, to the SIC.

NEHC responded to the USEPA comments and questions by providing additional information, recalculating the risk according to their recommendations and focusing on the key messages to be addressed in the risk assessment conclusions. USEPA recommendations have

either been incorporated into the Pioneer and NEHC reports or addressed in the responses to their comments, which are presented in Appendix B.

NAS COT Subcommittee Peer Review

In reviewing the final HRA, the NAS COT subcommittee was provided the same supporting documents given to the USEPA, including the risk assessment report prepared by Pioneer (Pioneer 2000); sampling plans, sampling results, and summaries prepared by Radian (Radian 1998a,b,c,d, 1999a,b,c,d, 2000a,b,c,d); statistical analyses prepared by Research Triangle Institute (RTI 1999); and quality-assurance reports (IT 1999; UAI 1999). A brief summary of the subcommittee's review of the risk assessment is provided below.

Positive comments were made by the NAS COT regarding their confidence in the quality of the sampling techniques, data collected and meteorological monitoring. However, they also stated that NEHC's analyses of the resulting data did not consistently meet the objectives of estimating the potential health risks to people living at NAF Atsugi and determining the contribution of the incinerator to those risks. More specifically they indicated that:

- (1) Aspects of the analyses and interpretation of the data without taking into account the meaning and limitations of the risk assessment process, not the underlying data themselves, constituted the main limitation of the risk assessment;
- (2) Given that a Superfund type of risk assessment was conducted, there was no distinction when drawing conclusions and making recommendations between those based on science and those based on policy;
- (3) The most appropriate methods were not used to determine the contribution of the incinerator complex to health risks at NAF Atsugi; and
- (4) Contrary to the opinion of their own Committee on Toxicology, in previous peer reviews of the screening risk assessments, the Subcommittee did not support some of the interim risk-reduction strategies recommended by NEHC, such as washing of hands, forearms, face, tools, toys, and closing windows and doors when emissions from the SIC were blowing directly on base.

The NAS COT peer review comments focused on four different areas for improvement of the NEHC draft report on the findings, conclusions and recommendations of the final HRA:

- (1) Provide more in-depth discussion of purpose, objectives and methodology. Refine risk calculations while better stating the assumptions, limitations and uncertainties involved with the risk assessment to meet the original objectives of the health risk assessment. NEHC has added new and revised information to this report, which address these concerns.
- (2) Clearly distinguish science-based from policy-based conclusions and recommendations. NEHC has added discussions throughout this report that address risk assessment methodology and risk interpretation according to science or policy, clearly pointing out each.
- (3) Provide a complete toxicity evaluation for risk characterization that will include a qualitative assessment of risk to accompany the quantitative assessment to better assist the risk managers in their decision making process, thereby providing information that augments understanding of numerical risk estimates. NEHC responded by conducting further evaluation of health effects to reduce uncertainty in areas most likely to impact risk management decisions such as reproductive/developmental health effects, children's health effects, more in-depth toxicology profiling for risk drivers and a review of the literature for toxicological information for detected substances without peer reviewed toxicity values, and the derivation of missing toxicity values, where possible.
- (4) Reevaluate the contribution of the incinerator to potential health risks according to NAS COT recommendations. NEHC did not implement this recommendation which would have involved reevaluating the SIC contribution by conducting additional analysis through the use of air dispersion modeling/correlation analysis. The subcommittee indicated that air-dispersion modeling, used in conjunction with correlation analyses, would be the most appropriate method to determine the contribution of the incinerator complex to the health risks at NAF Atsugi (NRC 2000). NEHC sent a written request to the NAS COT chair for clarification on how best to apply the dispersion modeling approach to determine the contribution of the incinerator complex. In response, a

telephone conference was held 10 April 2001, with only two members of the subcommittee. During the telephone conference, the two reviewers withdrew their recommendation that the dispersion modeling and correlation analyses be used to determine the risk attributable to the incinerator facility. They were unable to provide a clearly stated methodology to conduct their recommended approach and could not guarantee that the approach would provide useful results with reduced uncertainty. In the absence of a better, time-tested approach, NEHC has retained the upwind-downwind approach initially presented to the NAS COT for review. The NAS COT peer reviewers, of the 1998 screening risk assessment (NRC 1998), previously recommended that an upwind-downwind approach be used to determine the risk attributable to the SIC. USEPA reviewers also encouraged the Navy to continue to pursue this strategy (EPA 2000a). NEHC believes that the upwind downwind analysis is a valid and reasonable approach, despite its uncertainties. Other NAS COT recommendations have been incorporated into the Pioneer and NEHC reports or addressed in the responses to their comments, which are presented in Appendix C.

1.6 Document Organization

This document is organized as follows:

- Section 1 Introduction: This section presents the purpose of this report, describes the NAF Atsugi and the SIC, and describes the planning efforts to evaluate air quality and determine the health risks.
- Section 2 Air Monitoring and Soil Sampling Methodology and Results: This section summarizes the methodology and results of the comprehensive air and soil-sampling program.
- Section 3 Human Health Risk Assessment Results: This section summarizes the results of the Human Health Risk Assessment.
- Section 4 Health Risk Evaluation: This section presents additional information that is used to help interpret the risks presented in Section 3, including the cancer and non-cancer health effects of chemicals of concern.

Section 5 – Risk Assessment Summary of Findings and Conclusions: This section presents conclusions of the risk assessment.

Figure 1-1 — Location of NAF Atsugi, Japan

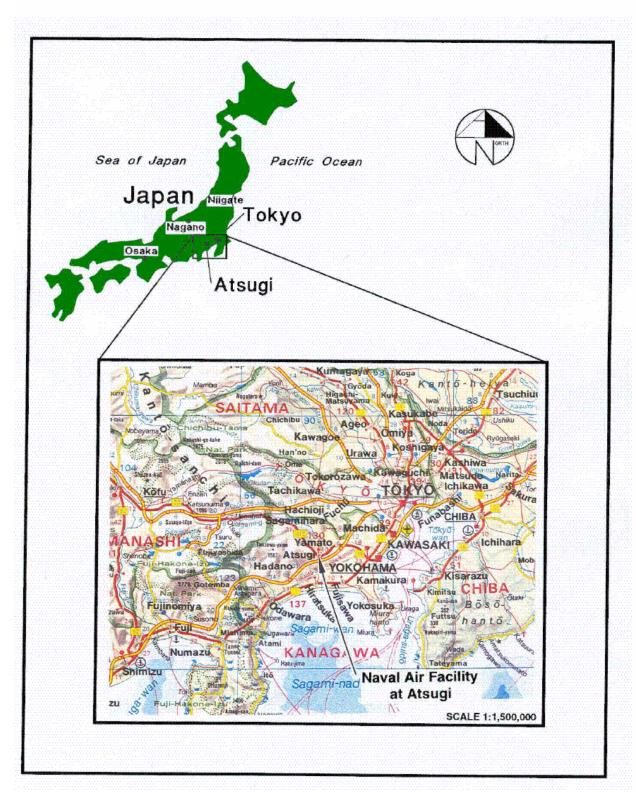


Figure 1-1 — Location of NAF Atsugi, Japan

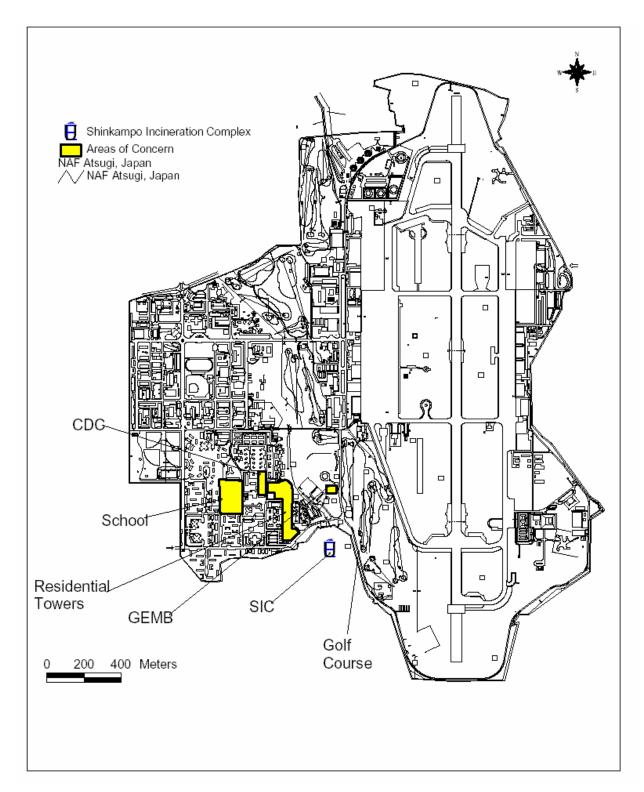


Figure 1-2 — NAF Atsugi Base Layout



Figure 1-3a — Shinkampo Incineration Complex

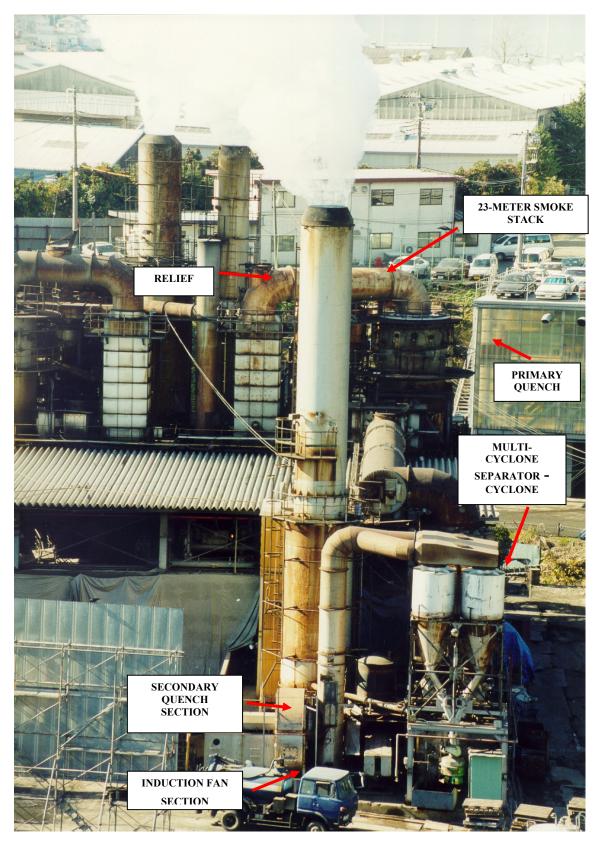


Figure 1-3b — Shinkampo Incineration Complex

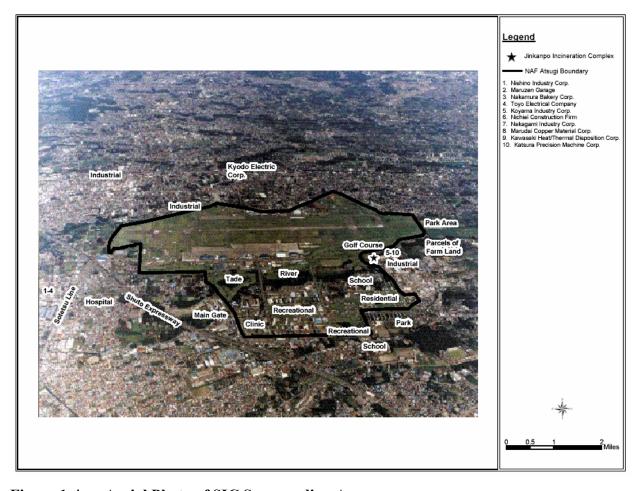


Figure 1-4 — Aerial Photo of SIC Surrounding Area

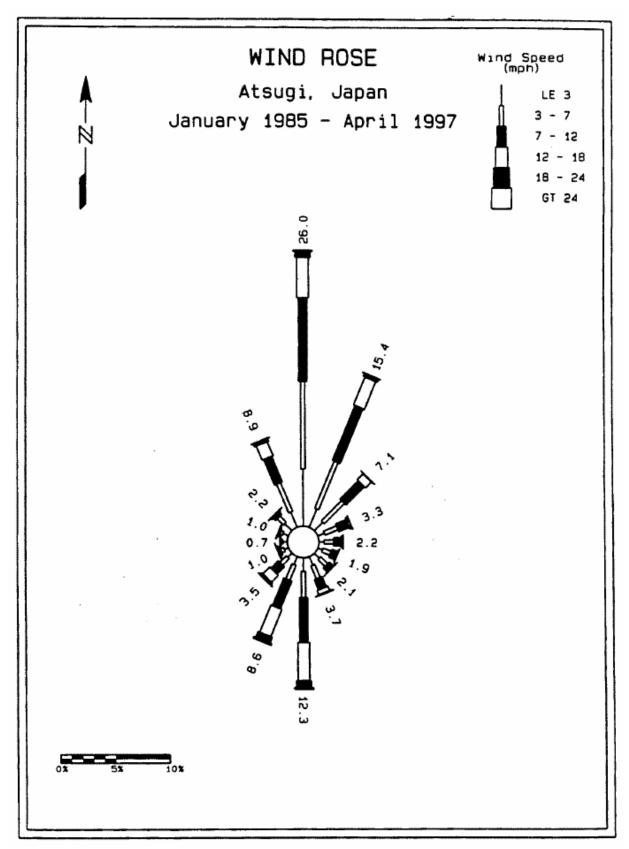


Figure 1-5 — Historical Wind Rose

Section 2 — Air Monitoring and Soil Sampling Methodology and Results

This section presents the results of the comprehensive environmental sampling program conducted by Radian from March 1998 to June 1999, to support the comprehensive HRA. The sampling program included sampling of the ambient (outdoor) air, indoor air and dust, and soil.

2.1 Sampling Objectives

The primary purpose of the ambient air monitoring and soil sampling were to collect data of sufficient quality to support the objectives of the comprehensive HRA:

- 1. Estimate the potential human health risks to U.S. Navy personnel and their families and other individuals living and working on NAF Atsugi, Japan, resulting from exposure to constituents of concern (COCs) in soil, ambient air, indoor air, and indoor dust. This estimate focused solely on COCs that were likely to be associated with ambient air emissions and/or subsequent deposition from point and non-point sources impacting the air quality at NAF Atsugi.
- 2. Evaluated the contribution of the potential health risk attributable to the Shinkampo Incineration Complex (SIC).

2.2 Ambient Air Sampling

The ambient air sampling was designed to provide data to answer the following questions for the HRA:

- What was the inhalation exposure risk for sensitive individuals in outdoor locations at NAF Atsugi, impacted by the SIC and other point and non-point air pollution sources?
- What was the risk contribution from the SIC?

Prior to this sampling effort, only short and periodic sampling was conducted in an attempt to capture "worst case" events when the SIC was burning hazardous materials and the wind was blowing over NAF Atsugi. The data from these sampling events indicated that there was a health threat, but because of the sporadic sampling periods, there was not enough data to

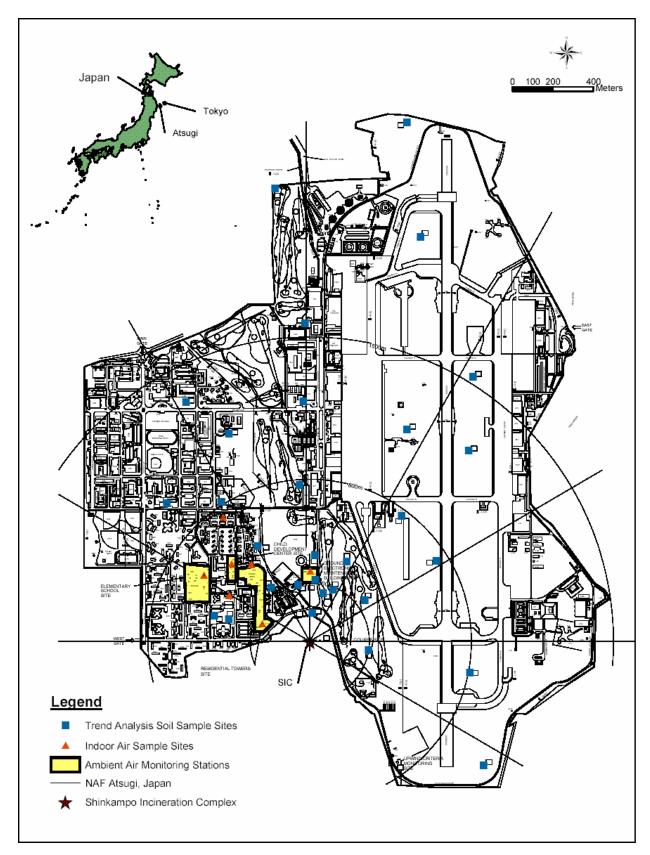


Figure 2-1 — Air and Soil Sampling Locations

	Oute	door	Ind	oor
Parameter	Number of Locations	Sampling Frequency	Number of Locations	Sampling Frequency ¹
VOCs	5	6 th day	7	2x Quarterly
Aldehydes and Ketones	5	12 th day	7	2x Quarterly
Heavy Metals	5	12 th day	7	2x Quarterly
Mercury	5	6 th day	7	2x Quarterly
Acid Gases	5	6 th day	7	2x Quarterly
PM ₁₀	5	12 th day	7	2x Quarterly
PM _{2.5} (manual)	1	12 th day	0	N/A
PCBs/Pesticides	5	12 th day	7	2x Quarterly
PCDDs/PCDFs	5	6 th day	7	2x Quarterly
SVOCs	5	12 th day	7	2x Quarterly
Criteria Pollutants	1	Continuous	0	N/A

Table 2-1 — Summary of Air Analytes, Sampling Sites, and Sample Frequency

¹The Ground Electronics Maintenance Building site was sampled every 12th day instead of quarterly, beginning with first quarterly indoor air monitoring event in June 1998.

VOCs - volatile organic compounds

PM – particulate matter

PCBs – polychlorinated biphenyls

PCDDs/PCDFs - dioxins/furans

SVOCs – semivolatiles

N/A – not applicable

determine the overall health risks. Therefore, for this sampling program, the ambient air monitoring was performed at frequent intervals to establish trends and determine the variety of pollutants coming from the SIC and reaching NAF Atsugi personnel. Table 2-1 presents a summary of the number of monitoring stations, parameters to be monitored, and frequency of monitoring for the outdoor and indoor air sampling programs.

Four monitoring stations were set up close to the SIC, at locations where people live, work, play, and go to school. These monitoring stations, shown in Figure 2-1, included one of the Residential Towers (Building 3101), the Ground Electronics Maintenance Building (GEMB), the Golf Course, and the Elementary School. One upwind/criteria monitoring station was located at the Skeet Range to monitor the upwind air quality when the wind was blowing toward the base, past the incinerator in the northerly direction. Although this was considered the upwind

site, this site was not a true upwind site because it might be downwind for part of a 24-hour sampling period and upwind or crosswind for the remainder of the period.

The chemicals chosen for monitoring included those considered to be a concern from a human health impact, and either likely to be stored at the SIC (such as the volatile organic compounds), or potentially a significant emission from the SIC (such as the chlorinated dioxins and furans and hydrochloric acid). The air was monitored for eight groups of chemicals, including those that are likely to be related to the SIC. Full 24 hr-monitoring runs were conducted at an average frequency of every 12 days and consisted of acid gases; aldehydes and ketones; heavy metals, particulate matter less than 10 and 2.5 micrometers in aerodynamic diameter (PM₁₀ and PM₂₅), polychlorinated biphenyls (PCBs), pesticides, semivolatile organic compounds (SVOCs), volatile organic compounds (VOCs), mercury, acid gases, and dioxins/furans. Partial 24 hr-monitoring runs were conducted every 6 days and consisted of VOCs, mercury, acid gases and the dioxins/furans. This program was established to replicate an every sixth-day sampling program commonly used in the United States to measure particulate and air toxic compounds for one year (EPA 1998a). This schedule rotates through the 7 days of the week and over one-year produces a nearly equal number of samples from each weekday. This schedule is used for USEPA's Urban Air Toxics Monitoring Program (UATMP) and their Photochemical Assessment Monitoring Stations (PAMS) studies.

Criteria Pollutants

At the Skeet Range (upwind/criteria site), the air was continuously monitored for "criteria pollutants." These are chemicals routinely measured by the USEPA to evaluate air for quality in the United States. Criteria pollutant sampling was conducted to answer the following questions:

 How did the local air quality compare with the USEPA National Ambient Air Quality Standards (NAAQS) for criteria pollutants? At measured concentrations, how did these criteria pollutants impact air quality?

The criteria pollutants that were continuously monitored, at the skeet range, during the project included sulfur dioxide (SO₂), nitrogen dioxide (NO₂), carbon monoxide (CO), ozone (O₃), and fine particulate matter (PM_{2.5}). These pollutants were monitored at a site located southeast and generally upwind of the SIC on top of the large dirt mound south of the skeet

range. An air monitoring enclosure was used to house the criteria pollutant continuous monitoring equipment.

An additional $PM_{2.5}$ monitor was operated at the elementary school and PM_{10} monitors were operated at each of the five monitoring stations. These additional 6 criteria pollutant monitors were not operated on a continuous basis but every 6 to 12 days. Lead analysis was conducted using the PM_{10} filters.

The results of the criteria pollutants monitoring indicated that except for carbon monoxide, sulfur dioxide and lead, the air quality failed to meet USEPA standards for all criteria pollutants during the 14-month period. A summary of the USEPA National Ambient Air Quality Standards (NAAQS) is presented in Table 2-2.

Due to limited sources of sulfur dioxide levels in the area, all SO₂ values were well below the USEPA NAAQS for SO₂ and do not represent a significant impact on ambient air quality.

Carbon monoxide values were consistent with data collected from other air monitoring programs in United States urban areas. The 8-hour and 1-hour NAAQSs were not exceeded.

The only applicable standard for NO₂ is the annual USEPA NAAQS arithmetic mean of 53 ppb. During the study, there were a number of individual 1-hour averages that exceeded the annual standard, with the highest hourly average being 108 ppb. However, the arithmetic mean at NAF Atsugi was 24 ppb, as compared to the 53 ppb standard.

The 1-hour USEPA NAAQS for ozone of 125 ppb was exceeded 7 times during the monitoring program and the 8-hour standard of 80 ppb was exceeded 19 times. Based on the data collected during this program, this area would certainly be considered non-attainment for ozone if it were located in the U.S.

 PM_{10} concentrations exceeded the USEPA 24-hour NAAQS of 150 μ g/m³ between one and three times per monitoring site. The annual arithmetic mean PM_{10} concentrations exceeded the annual NAAQS of 50 μ g/m³ at all five sites.

The USEPA NAAQS for lead is 1.5 ug/m³ and is based on a quarterly average value that is not to be exceeded. Lead concentrations measured for comparison with the USEPA lead standard should be collected for total suspended particulates (TSP). For this study, lead was measured using PM₁₀ samples; therefore, the average quarterly lead concentrations obtained at

NAF Atsugi are not directly comparable with the USEPA NAAQS. Lead was detected in all PM₁₀ samples analyzed during the program, with concentrations ranging from 0.009 ug/m³ to over 16 ug/m³. At the GEMB, the PM₁₀ lead concentrations exceeded the NAAQS for lead of 1.5 ug/m³ on six sampling days but no quarterly average exceeded this value. No other site had lead concentrations exceeding the USEPA NAAQS. Since lead concentrations were determined for particulate matter less than 10 microns in diameter (PM₁₀), instead of TSP, lead concentrations for comparison with the USEPA NAAQS could be severely underestimated.

The PM_{2.5} concentrations measured during the sampling period were consistently high, particularly during the fall and winter months. The 24-hour standard was exceeded on 146 days during the monitoring program, representing about one third of the total days sampled. In July 1997, USEPA promulgated new standards for particulate matter finer than 2.5 micrometers (PM2.5), in response to the Clean Air Act including its 1990 amendments. However, the PM2.5 standard is currently under litigation. Since the Supreme Court is currently reviewing this standard, the U.S. Government cannot enforce it at this time.

Table 2-2 — Summary of USEPA National Ambient Air Quality Standards (NAAQS) for the Criteria Pollutants

Pollutant	U.S. Standard
Carbon Monoxide (CO)	
8-Hour Average	9 ppm
1-Hour Average	35 ppm
Nitrogen Dioxide (NO ₂)	
Annual Arithmetic Mean	53 ppb
Oxides of Nitrogen (NO _x)	None
Ozone (O_3)	
1-Hour Average	125 ppb (non-attainment only)
8-Hour Average	80 ppb
Sulfur Dioxide (SO ₂)	
Annual Arithmetic Mean	30 ppb
24-Hour Average	140 ppb
3-Hour Average	500 ppb (secondary)
Particulate <10 μm (PM ₁₀)	
Annual Arithmetic Mean	$50 \mu \text{g/m}^3$
24-Hour Average	$150 \mathrm{\mu g/m^3}$
Particulate <2.5 μm (PM _{2.5})	
Annual Arithmetic Mean	$15 \mu g/m^3$
24-Hour Average	$65 \mu\text{g/m}^3$
¹ Lead (Pb)	
Quarterly Average	$1.5 \mu\mathrm{g/m}^3$

Lead standard applies to total suspended particulate (TSP) samples, not PM₁₀ samples.

Source: http://www.epa.gov/airs/criteria.html

Air Toxics

In addition to the USEPA criteria pollutants, eight groups of air pollutants were monitored outdoors. Table 2-3 presents the sampling and analytical methods used in the air toxics sampling. The eight groups of air pollutants monitored during this program were:

- Acid gases;
- Aldehydes and ketones;
- Polychlorinated dibenzo(p)dioxins and polychlorinated dibenzo(p)furans (PCDDs/PCDFs);
- PCBs and pesticides;
- Particulate matter (PM₁₀ and PM_{2.5}) and heavy metals;
- Mercury;
- Volatile organic compounds (VOCs); and
- Semivolatile organic compounds (SVOCs).

Acid Gases

The concentrations of hydrochloric acid (HCl) were generally well below the EPA Region 3 Risk Based Concentrations (RBC). However, the RBC was exceeded on some occasions at three of the five sites during the program. Overall, the GEMB site had significantly higher HCl concentrations, by a factor of two in mean concentration, than the other sites. The EPA Region 3 RBCs represent a concentration of chemicals below which little or no adverse health effects are observed even in the most sensitive populations such as children and the elderly. They do not necessarily constitute an enforcement policy but are used primarily to screen out chemicals that are to be carried through baseline health risk assessments.

Aldehydes and Ketones

A total of 13 aldehydes and ketones were measured during this monitoring program. Of these, seven have published RBC values. Four of these seven air pollutants had sample concentrations that exceeded the RBC. During the monitoring program, acetaldehyde and formaldehyde were detected in all samples from all sites, while acrolein was detected in 79% of all samples and crotonaldehyde was detected in 37% of all samples.

Table 2-3 — Summary of Sampling and Analytical Methods

Analyte	Sampling Method	Sampling Reference ^a	Analytical Method	Analytical Reference ^a	Sampling Duration (hours)
VOCs	SUMMA® Canister	TO-15	GC/MS	TO-15	24
Aldehydes and ketones	DNPH coated silica gel tubes	TO-11	HPLC	TO-11	24
Heavy metals	PM ₁₀ high volume sampling	40 CFR Part 50	Trace ICP	Method 6010B	24
Mercury	Gold Dosimeter	N/A	Jerome Analyzer	N/A	24
Mercury ^b	Hopcolite Resin	NIOSH 6009	Gold Vapor AA	Method 7470	24
Acid Gases	Denuder	IO-4.2	Ion chromatography	EPA Method 300	24
PCBs/Pesticides	PS-1 PUF sampler	TO-4	GC/ECD	Method 8081	24
PCB Congeners ^b	PS-1 PUF sampler	TO-9	High resolution GC/MS	Method 680	24
PM_{10}	Grasby PM ₁₀ sampler	40 CFR Part 50	Gravimetric	QA Handbook Vol. II	24
PM _{2.5}	Grasby PM _{2.5} sampler	40 CFR Part 50	Gravimetric	QA Handbook Vol. II	24
PCDDs/PCDFs	PS-1 PUF sampler	TO-9A	High resolution GC/MS	Method 8290	24
SVOCs	PS-1 PUF sampler	TO-13	GC/MS	Method 8270C	24

aln some cases these methods have been modified. See the Sampling QA/QC Plan to Assess Health Risks Related to Air Quality of NAF Atsugi for a complete discussion of the sampling and analytical protocols. ^bSpecial Study only.

Dioxins and Furans

Of the seventeen 2,3,7,8-substituted PCDDs/PCDFs congeners, 12 were detected in every sample analyzed and four more were detected in all but one sample. The congener with the highest toxicity, 2,3,7,8-TCDD (tetrachloro dibenzo-p-dioxin), was detected in 71% of the samples.

PCBs and Pesticides

Most of the pesticide compounds were detected sporadically. There were no PCB Aroclors (specific commercial preparations) detected in any of the samples. A significant number of coplanar PCB congeners of very light molecular weight, from monochloro- through tetrachloro congeners, was present, but they tended to be those with the lowest toxicity of the various congener classes.

Particulate Matter and Heavy Metals

Twelve heavy metals were quantitatively analyzed from the PM₁₀ filter samples. Continuous monitoring of PM_{2.5} was only conducted at the Elementary School site. Twelve of the 24 valid PM_{2.5} sampling runs were collected concurrently with the PM₁₀ sampling runs. The heavy metal results from these 12 PM_{2.5} samples, when compared to the PM₁₀ heavy metal results collected on the same day for lead, arsenic, cadmium, and beryllium concentrations, indicated that the majority of the metals were contained in the fine particulate fraction. Over 90% of the lead and virtually all the cadmium, arsenic, and beryllium were contained in this fine PM fraction.

Mercury

Mercury was detected in 57% of the ambient air samples collected during the monitoring program.

VOCs

There were 11 different VOCs with a frequency of detection exceeding 50% and a maximum value that exceeded the EPA Region 3 RBC value. The VOCs were 1,2,4-trimethylbenzene, benzene, chloromethane, methylene chloride, 1,2,4-trimethylbenzene, toluene, trichloroethylene, carbon tetrachloride, 1,3-butadiene, chloroform, and acetonitrile. In addition, nine other compounds with a frequency of detection ranging from 0.3% to 36% had maximum concentrations in excess of the RBC.

SVOCs

The SVOC compounds were fairly low in both concentration and frequency of detection. Only six of the SVOCs were detected in more than 90% of the samples. Fourteen of the more than 100 SVOCs on the target list were detected in over 50% of the samples.

Table 2-4 summarizes the 14-month air monitoring results, by providing summary statistics for 45 chemicals ordered by frequency of detection with maximum concentrations detected during the 14-month sampling period at any site exceeding the EPA Region 3 RBCs. During this sampling period the chemicals detected 100% of the time were one acid gas (hydrochloric acid), two aldehydes (acetaldehyde and formaldehyde), dioxin, PM₁₀, two PM₁₀ metals (cadmium and lead), five VOCs (benzene, chloromethane, methylene chloride, toluene, 1,2,4-trimethylbenzene) and one SVOC (1,4-dichlorobenzene). The highest air pollutant concentrations were observed at the Ground Electronics Maintenance Building. Sampling results for the individual sites and more details on the ambient air-monitoring program are found in Section 2 of the Radian Air Monitoring Summary report (Radian 2000).

RBC Exceedances

Prior to completion of the comprehensive HRA, the results of the air toxics monitoring were compared monthly and quarterly to the RBCs for air as a screening tool to help determine if the chemical concentrations required interim risk management actions necessary to protect the health of the NAF Atsugi community (Radian1998f – 1998j, 1999b – 1999e, 1999g - 1999h, and Pioneer 1998b – 1998d, 1999b – 1999i).

The EPA Region 3 RBCs (EPA2000a) were calculated for standard exposure scenarios to give chemical concentrations that correspond to fixed levels of risk (i.e., a hazard quotient of 1, or lifetime cancer risk of 10⁻⁶, whichever occurs at the lower concentration) that may occur in tap water, ambient air, fish, industrial soil and residential soil.

Table 2-4 — Summary Statistics for Representative Pollutants of Concern Sampled 21 April 1998 through 25 June 1999 (Ordered by Frequency of Detection)

Compound	Number Detected Results/ Total Samples	Range of Det	ected Values	Range of I Limits for No Sam	on-Detected	Median Value	Mean	Standard Deviation	Lower 95% Confidence Limit	Upper 95% Confidence Limit	RBC a.
		Minimum	Maximum	Minimum	Maximum				-	-	
ACID GASES (µg/m³)											
Hydrochloric Acid	361/361	0.491	42.2	NA	NA	2.48	3.83	4.67	3.35	4.32	21.0
	ALDEHYDES AND KETONES (µg/m³)										
Acetaldehyde	216/216	0.792	18.0	NA	NA	3.21	3.49	1.97	3.23	3.76	0.810
Formaldehyde	216/216	0.211	22.9	NA	NA	1.98	2.46	2.19	2.17	2.76	0.140
Acrolein	171/216	0.0792	3.53	0.0800	0.290	0.278	0.335	0.354	0.288	0.383	0.0210
Crotonaldehyde	79/216	0.0792	3.43	0.0791	0.241	0.0525	0.277	0.450	0.216	0.337	0.00330
				DIOXIN	$IS (pg/m^3)$						
Dioxin TEQ ^{b.}	344/344	0.0700	39.4	NA	NA	0.760	1.57	2.99	1.25	1.89	0.0420
1,2,3,7,8,9-HxCDD	343/344	0.0260	4.78	0.0205	0.0205	0.158	0.295	0.518	0.241	0.350	1.40
2,3,7,8-TCDD	243/344	0.00936	0.590	0.00470	0.0348	0.0200	0.0346	0.0575	0.0286	0.0407	0.0420
			PC	CBs AND PES	STICIDES (1	ng/m³)					
alpha-BHC	61/203	0.252	1.45	0.0523	0.193	0.0625	0.254	0.349	0.206	0.302	0.990
Dieldrin	44/203	0.136	0.714	0.0940	0.666	0.218	0.221	0.134	0.202	0.239	0.390
Aldrin	18/203	0.123	1.49	0.0787	1.34	0.0882	0.171	0.248	0.137	0.205	0.370
Heptachlor epoxide	14/203	0.293	0.748	0.0828	0.881	0.233	0.211	0.114	0.195	0.227	0.690
				PM ₁₀ MET	ALS (μg/m	3)					
Cadmium	232/232	0.000127	0.343	NA	NA	0.00171	0.00608	0.0257	0.00275	0.00941	0.000990
Lead	232/232	0.00936	16.3	NA	NA	0.0633	0.262	1.16	0.112	0.412	1.50
PM_{10}	231/231	7.45	245	NA	NA	59.2	69.6	41.9	64.1	75.0	150
Arsenic	215/232	0.0000246	0.0552	0.000941	0.00200	0.00176	0.00237	0.00381	0.00188	0.00287	0.000410
Beryllium	190/232	0.00000768	0.00120	0.0000443	0.00111	0.0000954	0.000235	0.000283	0.000198	0.000271	0.000750
				PM _{2.5} MET	ALS (μg/m						
Cadmium	21/24	0.00117	0.00871	0.00125	0.00196	0.00204	0.00266	0.00211	0.00177	0.00355	0.000990
$PM_{2.5}$	21/24	6.70	126	1.00	1.00	18.8	26.3	27.7	14.6	37.9	65.0

Table 2-4 — Summary Statistics for Representative Pollutants of Concern Sampled 21 April 1998 through 25 June 1999 (Ordered by Frequency of Detection)

	Number Detected Results/ Total			Range of Detection Limits for Non-Detected		Median		Standard	Lower 95% Confidence	Upper 95% Confidence	
Compound	Samples	Range of Det	ected Values	Samj		Value	Mean	Deviation	Limit	Limit	RBC a.
		Minimum	Maximum	Minimum	Maximum						
Beryllium	17/24	0.000417	0.00171	0.000417	0.000417		0.000663	0.000421	0.000486	0.000841	0.000750
Arsenic	13/24	0.000917	0.00750	0.00529	0.0127	0.00289	0.00355	0.00186	0.00277	0.00434	0.000410
				SVOC	$s (\mu g/m^3)$						
1,4-Dichlorobenzene	197/197	0.121	4.22	NA	NA	0.933	1.18	0.760	1.07	1.29	0.280
Acetophenone	75/197	0.0944	0.938	0.00100	0.0310	0.00456	0.121	0.197	0.0935	0.149	0.0210
Hexachlorobenzene	1/197	0.00500	0.00500	0.00100	0.0370	0.00390	0.00388	0.00162	0.00365	0.00410	0.00390
				VOCs (GC	/MS) (µg/m ³	3)					
1,2,4-Trimethylbenzene	349/349	0.366	11.1	NA	NA	1.67	2.02	1.46	1.86	2.17	6.20
Benzene	349/349	0.861	18.5	NA	NA	3.41	3.75	2.11	3.53	3.98	0.220
Chloromethane	349/349	0.954	3.48	NA	NA	1.63	1.74	0.461	1.69	1.79	1.80
Methylene Chloride	349/349	1.07	676	NA	NA	5.06	11.0	43.4	6.42	15.6	3.80
Toluene	349/349	4.17	523	NA	NA	20.4	24.5	30.1	21.3	27.7	420
Trichloroethylene	343/349	0.217	10.0	0.215	0.858	1.50	1.76	1.34	1.62	1.90	1.00
Carbon Tetrachloride	340/349	0.247	1.73	0.212	1.07	0.615	0.639	0.185	0.620	0.659	0.120
Tetrachloroethylene	329/349	0.0677	10.4	0.466	0.778	0.920	1.22	1.06	1.11	1.33	3.10
1,3-Butadiene	321/349	0.0802	5.74	0.0883	0.603	0.389	0.478	0.488	0.426	0.529	0.00350
Chloroform	270/349	0.0707	1.32	0.0975	1.07	0.195	0.232	0.135	0.218	0.246	0.0770
Acetonitrile	200/349	0.171	701	0.187	6.83	1.09	12.2	57.1	6.16	18.2	62.0
Hexachloro-1,3- Butadiene	125/349	0.128	11.1	0.469	15.8	0.649	1.46	1.80	1.27	1.65	0.0800
1,4-Dioxane	120/349	0.105	10.0	0.355	5.83	0.651	0.955	1.16	0.833	1.08	0.570
1,1,2,2- Tetrachloroethane	108/349	0.122	4.13	0.198	1.57	0.250	0.428	0.447	0.381	0.475	0.0310
1,1-Dichloroethylene	72/349	0.0459	0.676	0.0791	0.862	0.108	0.122	0.0822	0.114	0.131	0.0360
Acrylonitrile	70/349	0.0377	2.21	0.0867	2.02	0.179	0.257	0.263	0.229	0.285	0.0260
1,2-Dichloroethane	47/349	0.0808	1.07	0.105	0.901	0.169	0.181	0.108	0.170	0.193	0.0690
1,2-Dibromoethane	31/349	0.0767	1.85	0.0767	1.81	0.147	0.172	0.138	0.158	0.187	0.00820

Table 2-4 — Summary Statistics for Representative Pollutants of Concern Sampled 21 April 1998 through 25 June 1999 (Ordered by Frequency of Detection)

Compound	Number Detected Results/ Total Samples	Range of Detected Values		Range of Detection Limits for Non-Detected Samples		Median Value	Mean	Standard Deviation	Lower 95% Confidence Limit	Upper 95% Confidence Limit	
1		Minimum	Maximum	Minimum	Maximum						
1,2-Dichloropropane	28/349	0.0876	1.36	0.0655	0.600	0.0900	0.115	0.122	0.102	0.128	0.0920
Vinyl Chloride	24/349	0.0765	0.638	0.0663	0.561	0.0919	0.108	0.0773	0.100	0.116	0.0210
1,1,2-Trichloroethane	15/349	0.0545	1.21	0.0898	1.31	0.182	0.215	0.122	0.202	0.227	0.110
Bromodichloromethane	1/349	0.943	0.943	0.0929	6.69	0.164	0.275	0.562	0.216	0.334	0.100
Dibromochloromethane	1/349	1.11	1.11	0.170	8.50	0.216	0.330	0.715	0.255	0.405	0.0750

^aThe value listed for lead, PM_{2.5}, and PM₁₀ is from NAAQS. Otherwise, the value is from the April 1999 USEPA Region 3 Risk Based Concentration (RBC).

Median, mean, standard deviation, and confidence limits are estimated using proxies (dl/2) and detected results.

^bDioxin TEQs are calculated values, not measured.

NA = Not Applicable, because the analyte was detected in all samples.

While calculating the RBCs, EPA Region 3 used conservative assumptions for the exposure factors used in the risk assessment equations, to be protective of children and the elderly. In applying standard risk assessment methodologies to develop these screening concentrations, EPA Region 3 made the following assumptions:

Target Cancer Risk - 1 X 10⁻⁶ (one in a million or 1E-06)

Target Hazard Index - 1

Averaging Time - 70 years

Exposure Frequency - 350 days per year

Exposure Duration - 30-years

Body Weight - 70 kg (adult), 15 kg (child)

Other factors used in the risk equations were conversion factors such as the cancer slope factor (for carcinogens) and the reference concentration (for non-carcinogens). These factors were derived for each chemical based on animal studies and used to extrapolate toxicity results from animals to humans.

The maximum concentrations observed for acetaldehyde, formaldehyde, crotonaldehyde, and acrolein were above their corresponding RBCs. Maximum, median and mean concentrations of dioxin concentrations also exceeded its RBC. None of the pesticides had mean or median concentrations that exceeded the RBCs. Four of the metals (arsenic, cadmium, lead, and beryllium) had maximum concentrations exceeding the RBC. Two metals (arsenic and cadmium) had mean and median concentrations in excess of the RBCs as well. None of the detected values for mercury were in excess of the 310 ng/m³ RBC. There were 11 VOC air pollutants with a frequency of detection exceeding 50% and a maximum value that exceeded the EPA Region 3 RBC value. They were benzene, chloromethane, methylene chloride, 1,2,4-trimethylbenzene, toluene, trichloroethylene, carbon tetrachloride, tetrachloroethylene, 1,3-butadiene, chloroform, and acetonitrile. In addition, nine other VOCs had a frequency of detection ranging from 0.3% to 36% with maximum concentrations in excess of their RBC. Three of the SVOCs had maximum concentrations that slightly exceeded their RBC: 1,4-dichlorobenzene, acetophenone, and hexachlorobenzene.

Meteorological Monitoring

A 10-meter tower was used at Skeet Range (criteria site) to collect the meteorological parameters. The following meteorological parameters were monitored at this site;

- Wind speed;
- Wind direction;
- Wind standard deviation (σ-theta);
- Ambient temperature;
- Delta temperature (2 and 10 meters);
- Solar radiation;
- Precipitation; and
- Barometric Pressure.

The criteria site provided all the meteorological data used in evaluating the impact of the SIC. Because of the siting and selection of meteorological sensors located at this site, it provided data that was most representative of the meteorological conditions across NAF Atsugi. Furthermore, for SIC impact determinations, the upwind meteorology provided a better indication of the wind direction passing over the SIC stacks before impacting NAF Atsugi. The other monitoring sites were equipped with sonic anemometers and only measured wind speed and wind direction. These sites did not meet the same stringent USEPA criteria for meteorological station siting and sensor accuracy that the criteria site met. These data were used to assess whether there was any micrometeorology that may affect the correlation statistics used to estimate the SIC contribution to air quality.

The meteorological data from the various sites were compared to determine if significant differences existed between the various sites both on a monthly and quarterly basis. This comparison was performed by evaluating the differences between the percentage of time that the wind blew from each of the 16 wind direction vectors at the criteria site and each of the other four sites. Since very small differences in the wind direction angle can change the wind vector, the percentage of time the wind was from a combined set of vectors (e.g., south southeast to south southwest) was also evaluated. In general, the wind roses were quite similar between the

sites, with some slight variation at the Residential Towers site. The difference at the Residential Towers was most likely due to updrafts, building wake effects, and the effect of various structures on top of the tower. Based on the evaluation of these data, it appeared that there was little micrometeorology at NAF Atsugi and that the 10-meter tower data at the criteria site was representative of the meteorological conditions at the other four sites. The historical wind patterns were quite similar to what was observed during the NAF Atsugi monitoring program. Details on the meteorological monitoring are presented in the air monitoring summary report (Radian 2000a).

2.3 SIC Contribution to Air Quality

In the U.S., stack sampling is conducted to assess the risk from an incinerator. Since stack sampling could not be conducted for this Japanese owned and operated incinerator complex, several statistical methods were employed which used the ambient air sampling to assess the overall contribution to health risk. The method used to identify the chemicals in air that were emitted from the SIC was to correlate wind direction, specifically the percentage of time an individual monitoring site was downwind of the SIC, to the chemical concentrations observed in ambient air at the various sites. The basis of the correlation analysis performed was an assumption that if an analyte was emitted by the SIC, there would be a positive linear relationship between concentration at a particular site and the percentage of time that particular site was downwind (i.e., percent downwind) of the SIC. In other words, the hypothesis was that, for chemicals that were emitted from the SIC, the chemical concentration (and also risk) increased as the percent of time the wind blew emissions from the SIC onto the base increased. For example, if an analyte exhibits a positive correlation between concentration and percent downwind, then the concentration should increase as the percent downwind increases from 20% to 50% to 100%.

The fundamental assumption underlying the correlation analysis was that the analytical composition of the incineration feedstock, and consequently the stack emissions, were consistent from day to day so that a correlation could be established. However, the SIC was a municipal waste incinerator that had a highly variable feedstock and thus the analytical composition of the stack emissions were also highly variable. This significantly impacted the effectiveness of the correlation analysis because the hypothesis that the concentration of analytes that are associated

with emissions from the SIC increase as the percentage of time downwind increases, is only true if the analytical composition necessary to emit the analyte is present in the feedstock.

Radian and Research Triangle Institute (RTI) performed three statistical evaluations. This was to determine if a relationship existed between individual chemical concentrations measured at a particular site and the amount of time that site was downwind of the SIC (i.e., % downwind) at each site. The following methods were carried out to determine which specific chemicals would be associated with emissions from the SIC: (1) standard correlation and linear regression analysis [Radian], (2) mixed model analysis [RTI] and (3) non-parametric analysis [RTI]. These methods used meteorological data and concentrations measured for COCs at NAF Atsugi (e.g., the cancer and non-cancer risk drivers) to determine if the COCs were associated with SIC operations.

Radian's correlation and regression analysis approach analyzed one site and the SIC's condition at a time, basing examination of one site's data (Criteria Site, Residential Towers, Elementary School, Golf Course) on the positive results of statistical tests from another site (GEMB, the site with the highest percent downwind). In other words, the results for the other sites were examined in detail only if the GEMB showed a positive and significant correlation when the SIC was on. This approach identified six chemicals that were related to SIC operations. These chemicals were hydrochloric acid, dioxin, lead, cadmium, arsenic, and PM10. This was surprising since numerous analytes (50 - 100s) are typically present in stack samples of incinerators in the United States. The statistical analysis conducted by Radian is presented in detail in the air monitoring summary report (Radian 2000a)

Because factors such as variable feedstock, variable SIC operating conditions, and different meteorological conditions may have prevented the identification of all of the chemicals related to the SIC, RTI performed statistical analysis (RTI 1999) that involved a mixed model analysis and a non-parametric correlation analysis. In the case of variable wind speed and direction, from one week to the next, if the emissions were constant but the wind speed and direction were not the same, the correlation with percent downwind would be different. The confidence in the correlation of wind direction versus concentration was related to the number of observations that were used to calculate the correlation coefficient and the wind directions that were observed. The mixed model was based on a multiple regression analysis in which the

relationship between chemical concentration and % downwind was evaluated simultaneously at all of the ambient air monitoring stations. It used a more consolidated approach that attempted to adjust for day-to-day variations related to SIC feedstock, operating parameters or other sources. The non-parametric analysis, which utilized virtually all the data, was insensitive to outliers, did not depend on the measurement scales chosen for concentration data and incorporated an adjustment for day-to-day variations. For these reasons, the non-parametric methods were considered more robust.

The statistical analyses performed by RTI on the top thirty-two chemicals contributing to the risk in the risk assessment indicated that arsenic, benzene, cadmium, 2,3,7,8 TCDD TEQ, 1,2-dichloropropane, lead and PM₁₀ showed a strong association with the SIC. Other risk drivers such as 1,3-butadiene, acetaldehyde, acetophenone, chloromethane, and dieldrin showed an association with less confidence with the SIC.

The designations "strongly associated" and "with less confidence" were categories used by RTI to express the association between wind direction and air concentrations as a means of determining SIC contribution to air quality. RTI 's two approaches evaluated the degree of evidence of association, based on six statistical criteria that considered the last two methods and used a weight-of-evidence approach, regarding positive correlations. "No association" means that the chemicals met one or less of the six criteria or were assigned this category for other reasons. "Strongly associated" means that at least 5 of the six criteria were satisfied. "With less confidence" means a possible association exists, indicating that although significant positive correlations were found by the parametric analysis, they were not supported by the mixed model approach. The results of all statistical analyses were as follows:

- All three methods determined with confidence that 5 of the various chemicals (dioxin, PM₁₀, lead, cadmium, and arsenic), which contributed the majority of risk, were associated with the incinerator.
- Hydrochloric acid, which is normally associated with incineration as a result of combustion of plastics, was correlated well with the incinerator, using the standard correlation and linear regression method.

- The two more robust methods determined that benzene and 1,2- dichloropropane were strongly associated with the incinerator and 1,3-butadiene, acetaldehyde, acetophenone, chloromethane, and dieldrin were associated, but with less confidence.
- Acrolein showed a significant positive correlation with the SIC at the GEMB site based on the log of the concentration scale, one of the criteria of the mixed model analysis. This was similar to the chemicals that were strongly associated with the SIC. However, the correlation for SIC on days was virtually identical to SIC off days. In addition, unlike the chemicals strongly associated with the SIC, there was no correlation at the GEMB site based on the concentration scale (another criterion). At three other sites the correlation was greater for SIC off days. Even though the monitoring program was an extensive effort, SIC off days (13) did not match the number of SIC on days (60), which limited the power of the comparative analysis. It is expected that the SIC was a major contributor to acrolein concentrations; however, the data suggests that there may have been other sources of acrolein in the area. Automobiles are known for contributing acrolein to the environment.

Since the literature indicates that many more chemicals are generally associated with incineration, many more are expected to be associated with the SIC. There are a number of reasons why their association with the incinerator could not be demonstrated, which include:

- Other potential background sources of air pollution at Atsugi, as well as its proximity to Tokyo, a large industrial center
- Various feedstock and operating conditions of the incinerator
- Variable wind conditions and wind speeds
- No stack sampling

2.4 Estimation of Background Ambient Air Concentrations

Background is defined as the airborne concentrations that would be expected if the SIC had not existed. Many site-specific factors made the task of determining which chemicals originated from the SIC from those that were present in background ambient air challenging. For example, NAF Atsugi is located in a heavily industrialized area proximate to multiple point and non-point sources of airborne contaminants. Furthermore, Japan's primary mechanism for

disposing of waste is incineration, which results in higher background concentrations of many airborne contaminants such as particulates and dioxins. Also, meteorological conditions such as low percentage of downwind days, make the task of defining upwind concentrations or background from downwind concentrations difficult.

Five different alternatives were considered during this project for evaluating the potential impact of emissions from the SIC on the ambient air at NAF Atsugi. Each of these approaches involved comparing background concentrations with concentrations measured at sites downwind of the SIC.

- 1. Comparing the concentrations of COCs in ambient air when the SIC was ON to the concentrations when the SIC was OFF (Radian 2000).
- 2. Comparing the COC in ambient air at a downwind site when the SIC was ON to the COC when the SIC was OFF (Radian 2000).
- Comparing the COCs in ambient air at a site downwind of the SIC when the SIC was
 ON, to the concentrations when the site is not downwind of the SIC and the SIC was ON
 (Radian 2000).
- 4. Using the results of the correlation analysis to model and compare concentrations, when a site was downwind of the SIC and when the site was not downwind of the SIC (not performed, but considered).
- 5. Comparing the concentrations of COCs in ambient air at a site downwind of the SIC to the concentrations at another site which was upwind of the SIC on the same days (when the SIC was ON) (i.e., an "Upwind" versus "Downwind" evaluation).

Each method utilized a slightly different approach to characterize background and the contribution of the incinerator to the health risk at NAF Atsugi. Each method suffered from confounding factors contributing to the uncertainty in the quantification of the contribution of the incinerator to the health risks.

For the purposes of this risk assessment, the "Upwind" versus "Downwind" approach (#5 above) was selected to quantify the contribution of SIC emissions to the risk estimates.

Explanation of Why Alternatives 1, 2, 3, and 4 were not Selected to Quantify Background and the Contribution of SIC to the Risk Estimates

Alternatives 1, 2 and 3 compared the conditions when the SIC was ON and the wind was blowing toward NAF Atsugi with background air concentrations which were determined as follows:

- 1. Alternatives 1 and 2 Background was defined as the airborne concentrations measured when the SIC was OFF.
- 2. Alternative 3 Background was defined as the airborne concentrations measured at sites that were not downwind of the SIC when the SIC was ON.

Alternatives 1 and 2

Alternatives 1 and 2 were not selected because of the uncertainty associated with defining the background concentrations. The SIC was OFF only on Sundays, the same day that most other industries in the area were closed. Using only these data would potentially result in an underestimation or overestimation of the background concentrations. The underestimation of background concentrations could result because most industries were closed on Sundays — potentially resulting in a decrease in emissions to ambient air. The overestimation of background concentrations could result from industries, such as the SIC, that were emitting COCs to the air during the day as they were powered down.

Alternative 3

Alternative 3 was not selected because background concentrations defined under this condition were very ambiguous. The reason for this uncertainty is that there was not a clear cut-off between "wind blowing toward" and "wind blowing away." In fact, winds were blowing toward the base in varying degrees. Consequently, for some of the sites, the "most affected" days are low percent downwind days in absolute terms.

Alternative 4

From a risk assessment perspective, alternative 4 was not a good method because it only accounted for six chemicals that were identified in the correlation analysis as exhibiting a statistically significant correlation between concentration and percent downwind of the SIC. Experience from risk assessments performed on municipal waste incinerators in the United

States indicated that many more chemicals were emitted from incinerator facilities (EPA 1998b). Using the correlation analysis approach to quantify the contribution of the incinerator to the health risks would result in underestimating of the SIC's contribution. This is especially true for non-cancer health effects since a large number of expected chemicals were not found. Cancer risks would also be underestimated; however, the level of underestimation was not expected to be significant because dioxin-TEQ, which typically dominated the cancer risks, was one of the six chemicals that correlated with % downwind of the SIC. Also, as detailed in the Radian 2000 Report, there were many site-specific factors (e.g., the variable composition of municipal waste and emissions from multiple point and non-point sources) that should be considered when evaluating the results of the correlation regression analysis method.

Explanation of Why Alternative 5 was Selected to Quantify Background and the Contribution of SIC to the Risk Estimates

One of the primary goals of the risk assessment was to determine the contribution of health risks at NAF Atsugi attributable to emissions from the SIC. The "Upwind vs. Downwind" method was used to determine the risks attributable to the SIC because it was the only method that accounted for the risks associated with all analytes potentially being emitted from the SIC. This approach, which is complementary to the correlation regression analysis approach, was needed because of the limitations of the correlation analysis. If the correlation regression analysis method and/or air dispersion modeling methods had been used exclusively, only six of the 240 analytes that were detected in ambient air would have been evaluated (i.e., hydrochloric acid, 2,3,7,8-TCDD-TEQ, lead, cadmium, arsenic, and PM10). This would have resulted in an underestimate of the risks attributable to emissions from the SIC, especially for non-cancer health effects, because a large number of analytes being emitted from the SIC would not have been evaluated. Cancer risks would have also been underestimated. However, the level of underestimation for cancer risks was not expected to be significant, because 2,3,7,8-TCDD-TEQ, which typically dominates the cancer risks, was one of the six chemicals that would have been evaluated. The rationale for the decision to use the "Upwind vs. Downwind" method was based on experience from previous risk assessments performed on municipal waste incinerators that indicated that multiple chemicals (i.e., 50 - 100s) were likely to be emitted from incinerators (EPA 1998c).

In alternative 5, the approach used to identify COCs that were associated with emissions from the SIC involved comparing the analytical data from a site (i.e., the GEMB) that was downwind of the SIC with the analytical data from a site (i.e., the Golf Course or Upwind Criteria Site) that was upwind of the SIC during the same monitoring period (see Figure 2-2). Both the Golf Course and Criteria sites were considered "upwind" of the SIC when the wind was blowing from the south to the north as was the case with the data selected for this analysis. The

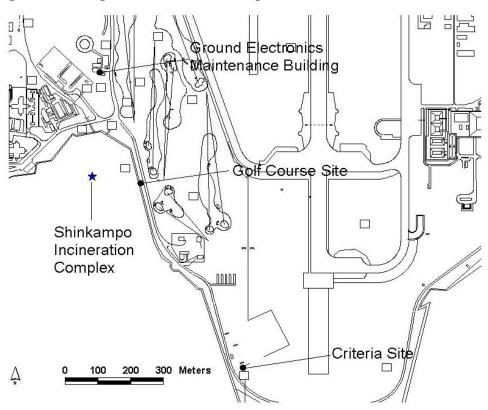


Figure 2-2 — Upwind/Downwind Sample Locations

Golf Course site was selected for the Upwind vs. Downwind comparison because it was closer to the SIC than the Criteria Site and would not be impacted by emissions located between the Criteria Site and the SIC generated from other sources. If the Criteria Site was used in the comparison, then the risks attributable to the SIC could potentially be overestimated as a result of emission sources located between it and the SIC.

This approach involved a comparison of the ambient air concentrations at the GEMB (i.e., the downwind site in this analysis) and at the Golf Course (i.e., the upwind site in this

analysis) using a sub-set of samples that were collected on the same day and time. It is important to note the terms downwind and upwind are relative to wind direction. For example, if the GEMB, which was located north of the SIC, was <u>downwind</u> of the SIC 100% of the time on a given day, then the Golf Course, which was located east of the SIC, was <u>upwind</u> of the SIC 100% of the time on that same day. Background does not mean "pristine" or "unimpacted; but background concentrations reflected anthropogenic (man-made) sources of airborne contaminants that were located proximate and remote to the SIC.

The goal of this "paired" comparison was to minimize, to the maximum extent possible, the potential contribution of other point and non-point sources on the concentrations being evaluated. Ideally, the upwind site would be downwind of the SIC 0% of the time during the day while the downwind site would be 100% downwind of the SIC. The components of the concentrations measured at the downwind and upwind sites under these ideal conditions are presented in Table 2-5

Table 2-5 — Components of Airborne Concentrations When the GEMB is 100% Downwind of the SIC

Site	Components of Airborne Concentrations
Golf Course Site Concentrations =	Background + Other Point and Non-Point Sources ¹ (emissions)
GEMB Site Concentrations =	Background + Other Point and Non-Point Sources (emissions) + SIC (emissions)

¹Only point and non-point sources located south of the SIC could impact the concentrations because the wind is blowing from the south to the north 100% of the time

Based on their locations relative to the SIC and the wind direction, the components of airborne concentrations measured at the Golf Course were (Background + Other Point and Non-Point Source emissions) and the components at the GEMB were (SIC + Background + Other Point and Non-Point Source emissions). As indicated in Table 2-5, the only difference between the airborne concentrations, and consequently risk, at the GEMB and the airborne concentrations at the Golf Course should be related to emissions associated with the SIC. There was some uncertainty associated with assessing the SIC's contribution to the risks using this method, including potential chemicals that were not associated with the SIC. However, based on the criteria used to select the sub-set of upwind and downwind samples, and the locations of the GEMB and Golf Course relative to the SIC, it was believed that this uncertainty was minimal.

Upwind -Downwind Analysis

The upwind-downwind analysis approach involved the following statistical approach for evaluating the null and alternative hypotheses.

The null and alternate hypotheses were as follows:

 H_o : The concentration of various chemicals observed at the GEMB (downwind site) were higher than those observed at the Golf Course (upwind site). In other words, the concentrations, and therefore risks, observed at the GEMB were "enriched" by emissions from the SIC when compared to the Golf Course.

 H_a : The concentrations of various chemicals observed at the GEMB (downwind site) were not higher than those concentrations observed at the Golf Course (upwind site). In other words, the concentrations, and therefore risks, observed at the GEMB were not "enriched" by emissions from the SIC when compared to the Golf Course.

For <u>all</u> analytes that were detected in at least one sample:

- The days when samples were collected at the GEMB when it was ≥ 80% downwind
 from the SIC were identified. Samples collected at the Golf Course for the
 corresponding days were also identified. These samples were downwind of the SIC <
 5% of the time.
- 2. The average and reasonable maximum exposure (RME) concentrations were calculated for both sets of data.

The exposure point concentration for the average exposed individual was calculated based on the arithmetic mean concentration and the maximum detected concentration in instances where the arithmetic mean concentration exceeded the maximum detected concentration.

The exposure point concentration for the RME individual was calculated based on the following criteria:

1. The 95% Upper Confidence Limit (95% UCL) on the mean concentration for normally distributed and non-lognormally distributed data sets.

- 2. The Log 95% UCL of the mean concentration for all lognormally distributed data sets.
- 3. The maximum detected concentration in instances where the 95% UCL or Log 95% UCL exceeded the maximum detected concentration.

Table 2-6 presents the paired comparison of ambient air concentrations during conditions established in step 1 on the same day for the GEMB and the Golf Course in these upwind and downwind locations. They were calculated based upon an RME Adult stationed at NAF Atsugi for 6 years (i.e., 2 tours of duty). Shading indicates the higher concentrations, of the two locations sampled.

Table 2-6 — Upwind-Downwind Paired Comparisons of Ambient Air Concentrations of COCs

COCs	Golf Course (Upwind)	GEMB(Downwind) Exposure Point
	Exposure Point Conc. (mg/m³)	Conc.(mg/m³)
Acetaldehyde	7.93E-02	8.22E-02
Acetonitrile	2.19E-01	7.51E-03
Acrolein	3.31E-04	1.31E-03
Acrylonitrile	5.61E-04	2.21E-03
Antimony	7.84E-06	3.63E-04
Arsenic	5.77E-06	3.62E-05
Benzene	3.00E-03	4.63E-03
1,3-Butadiene	4.45E-04	7.51E-04
Cadmium	1.22E-06	2.54E-04
1,2-Dibromoethane	2.30E-04	7.66E-05
1,4-Dichlorobenzene	1.58E-03	1.18E-03
1,4-Dioxane	1.85E-03	8.88E-03
Formaldehyde	8.84E-04	2.24E-03
Hexachlorobutadiene	3.51E-03	9.58E-04
Hydrochloric Acid	2.10E-03	2.43E-02
Nickel	1.82E-05	9.34E-05
PM_{10}	2.31E-01	2.34E-01
1,1,2,2-Tetrachloroethane	9.40E-04	7.87E-04
Total 2,3,7,8-TCDD TEQs	1.85E-09	2.92E-08

As shown in Table 2-6, except for acetonitrile, 1,2-dibromoethane, 1,4-dichlorobenzene, hexachlorobutadiene and 1,1,2,2-tetrachoroethane, all chemicals that contributed to the health risks at these sites had higher ambient air concentrations downwind from the SIC, including the

chemicals that were found to be associated with the SIC, which were acetaldehyde, arsenic, benzene, 1,3-butadiene, cadmium, hydrochloric acid, PM10 and 2,3,7,8-TCDD TEQs.

Uncertainties Associated with the Upwind Downwind Analysis Approach

The two main site-specific uncertainties associated with this approach were the sample size and the location of the Golf Course site. First, there were a limited number of samples (i.e., 3-8) that met the percent downwind criteria presented above. This decreases the overall confidence in the results by increasing the probability of committing Type I and Type II errors. Unfortunately, the only way to increase the sample size used in the comparison was to modify the percent downwind criteria (e.g., reduce the percent downwind criteria at the GEMB from 100% to 60%). The problem with this approach was that while the uncertainty was reduced by increasing sample size, significant uncertainty was introduced because 40% of the airborne concentrations at the GEMB could be associated with sources other than the SIC. Second, based on the component analysis presented in Table 2-5, the hypothesis was that the difference in the concentrations between the GEMB and the Golf Course were due solely to emissions from the SIC. The highest degree of uncertainty associated with this hypothesis was the assumption that the "other point source components" of the airborne concentrations were consistent between the GEMB and Golf Course. The reason for this uncertainty was that the Golf Course was located due east of the SIC. In order to fully minimize the uncertainties associated with this approach, an upwind monitoring station would have been placed due south of the SIC so that, when winds were blowing from the south to the north, all air sampling would be captured for the vector immediately upwind and downwind of the SIC. Unfortunately, it was not possible to place a monitoring station due south of the incinerator because this land is not under the control of the U.S. Navy. While this uncertainty was acknowledged, its impact on this upwind downwind analysis was considered minimal because no significant point sources were identified immediately south of the SIC (Radian 1998b). Consequently, based on its proximity to the SIC (i.e., approximately 150 meters) the airborne concentrations at the SIC and the Golf Course were expected to have similar component compositions.

2.5 Air Dispersion Modeling

There were two primary objectives for the dispersion modeling analysis. The first objective was to estimate emission rates from the SIC by comparing modeled impacts to actual

measured concentrations at each NAF Atsugi ambient air monitoring site. The second objective was to use the SIC estimated emission rates to predict average ground level concentrations across NAF Atsugi for the period 21 April 1998 through 25 June 1999. The USEPA Industrial Source Complex-Short Term (ISCST3 Version 98356) model was used for this analysis.

Air dispersion modeling during this period was conducted for six chemicals that showed a strong correlation with the SIC. These air pollutants showed a statistically significant correlation between their measured concentrations and percent of the time that a particular monitoring site was downwind of the SIC during the time period for which the concentrations were measured. The correlation regression analysis indicated that six air pollutants were likely related to SIC emissions; therefore, dispersion-modeling results are presented for the following air pollutants:

Dioxin TEQ;

Hydrochloric Acid;

Cadmium;

Lead;

Arsenic; and

PM₁₀.

To meet the first air dispersion-modeling objective, ISCST3 model runs were developed to estimate impacts at each of the ambient monitoring locations using a unit emission rate of one gram per second. Hourly concentrations were predicted for each hour with valid meteorological data. The hourly concentration data were then extracted from the ISCST3 output files. Ambient concentrations of most air pollutants were measured over a 24-hour integrated sampling interval. The hourly-modeled concentrations from the unit emission rate modeling were averaged over the coincident time period for each ambient air measurement. This resulted in a unit emission rate impact for each site location and sampling period.

During a typical 24-hour sampling period, any given measurement would include impacts resulting from emissions from all upwind sources. To best isolate impacts resulting from the SIC, the monitoring site that was downwind from the SIC the greatest percentage of the time was chosen. The site that was downwind of the SIC the smallest percentage of the time (always two

or fewer hours downwind during a 24-hour period) was used to estimate the background concentrations. The emission rate was then determined on each day of monitoring by subtracting the measured background concentration from the measured downwind concentration and dividing the difference by the sum of the modeled unit-emission-rate concentrations for the three incinerator stacks. The background was subtracted from the downwind concentrations to help isolate impacts resulting from SIC emissions only. Modeling showed that maximum impacts occurred just north of the SIC. High impacts extended just northeast from this location. The lowest modeled impacts, only 0.5 percent of the overall maximum concentration, occurred east of the SIC on the eastern NAF Atsugi property line. This location was only about 1,400 meters from the region of maximum impacts.

The results of the emission rate estimation analysis showed that the ratio of minimum to maximum emission rates during sampling events varied over four orders of magnitude for lead and over two orders of magnitude for the remaining air pollutants that had been associated with the SIC by the correlation regression analysis. Since modeled impacts were linearly proportional to the emission rate, impacts would also vary by these orders of magnitude if the range of emission rates were modeled. While this variability in emission rates did not seem unreasonable, it was impossible to determine if this variability was primarily due to day-to-day SIC operation and waste combustion versus inaccuracies in the emission rate back calculations procedure.

To meet the second air dispersion-modeling objective, an ISCST3 model run was generated to predict average ground-level concentrations for the 21 April 1998 through 25 June 1999 time period at receptor locations across NAF Atsugi. The NAF Atsugi site wide receptor grid was modeled and the ISCST3 period option was invoked. Since the SIC typically did not operate from 4 PM Sunday through 4 PM Monday, an emission rate of zero grams per second was modeled during these hours. An emission rate of one gram per second was modeled for all other hours during the period. Figure 2-3 presents the results of this analysis as a contour plot of the average ground-level concentrations from the unit emission rate modeling.

The highest impact among the five monitoring locations was $34.15~\mu g/m^3$, which occurred at the GEMB. The second highest impact of $18.30~\mu g/m^3$ was modeled at the Residential Towers. Impacts of $5.70~\mu g/m^3$, $5.60~\mu g/m^3$, and $3.75~\mu g/m^3$ were modeled at the Upwind/Criteria Site, Golf Course, and Elementary School, respectively. These concentrations

could only be used to compare the relative magnitude of impacts between the monitoring sites. The concentration multipliers shown in Figure 2-3 must be used to determine the actual compound-specific modeled concentrations at each NAF Atsugi location.

2.6 Indoor Air

The close proximity of the SIC to sensitive populations located on the base (i.e., Residential Towers, Elementary School, Child Development Center) required evaluation of potential impacts through indoor air sampling. It was difficult to distinguish the contribution of the SIC to indoor air, but the sampling provided some information to understand if there was a correlation between indoor and outdoor air concentrations. For comparison purposes, the indoor air sampling was collected using the same methods and parameters as for the outdoor ambient air-sampling program. Table 2-1 presented earlier in subsection 2.2 indicates the number of monitoring stations, parameters to be monitored, and frequency of monitoring for the indoor ambient air sampling program.

The objective of the indoor sampling was to determine the exposure risk for the occupants/residents of selected, representative NAF Atsugi buildings. The indoor air sampling was intended to help address the following two questions to meet the objectives of the HRA:

- 1. What were the risks to sensitive receptors from inhalation of indoor air and dermal contact or incidental ingestion of dust contaminated by COCs infiltrating indoors that are likely to be associated with ambient air emissions and/or subsequent deposition from point and non-point sources impacting the air quality at NAF Atsugi?
 - 2. What was the extent of deposition of particulates from the SIC in indoor surfaces?

There were two modes in which ambient air emissions could have impacted indoor environmental air quality. The first mode was direct and immediate, in which emissions from the SIC passed over NAF Atsugi and produced elevated concentrations of gases, vapor and dust in the ambient air. Ambient air could be drawn into buildings, especially those with active fresh air ventilation systems, producing an inhalation exposure. In this mode, indoor air concentrations would be elevated above indoor background levels, but would always be less than the simultaneous ambient air concentrations for chemicals associated with the SIC. The actual

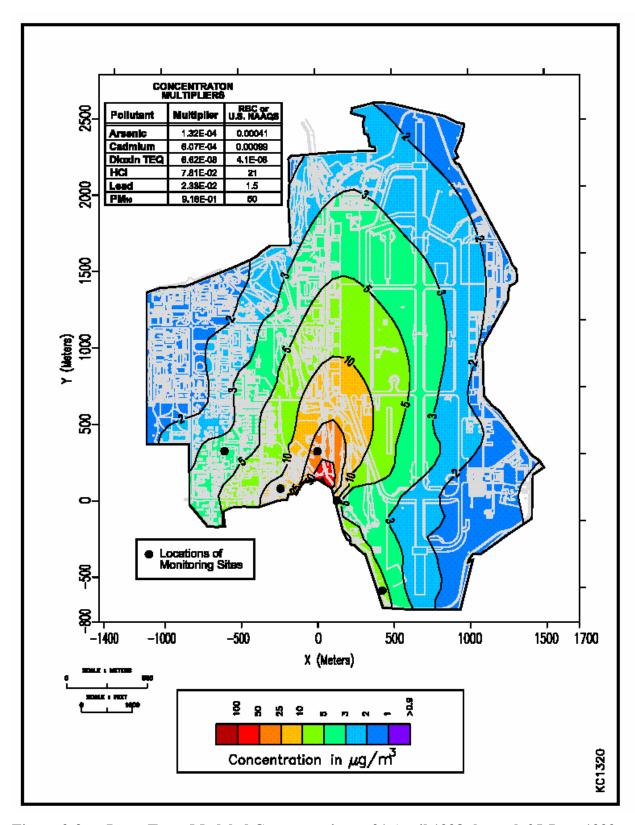


Figure 2-3 — Long-Term Modeled Concentrations - 21 April 1998 through 25 June 1999

indoor air composition would be the sum of components present in the background (e.g. from indoor emission sources) and components present in the incoming ambient air.

The other mode was indirect and applied to dust. The dust from the polluted ambient air could deposit on soil, vegetation, carpeting and indoor surfaces. This dust could produce exposure due to ingestion or dermal contact, but would have to become airborne again to produce an inhalation exposure. The sampling of indoor carpet/floor dust and surface wipe samples measured the accumulation of dust on surfaces at the time of the first air sampling at all indoor sites listed below.

Table 2-1 presents the number of monitoring stations, parameters to be monitored, and frequency of monitoring for the indoor air sampling program.

Indoor air sampling and analysis also included:

7 indoor carpet/floor dust samples;

7 indoor surface wipe samples;

A microscopical analysis of the seven carpet/floor dust samples (Micro-Sciences 1999).

A microscopical analysis of the dust catch on two PM₁₀ filters collected inside and outside the Ground Electronics Maintenance Building (Micro-Sciences 1999).

The decision to collect eight samples from each area of concern (AOC) was based on the number of samples required to obtain reasonable exposure point concentrations for risk assessment calculations. Exposure point concentrations were based on 95% upper confidence limits (UCLs) for the mean. The UCL was computed as $\bar{x} + t_{0.95,n-1} \frac{s}{\sqrt{n}}$, where \bar{x} was the sample mean; s was the sample standard deviation; s was the sample size, and s to distribution with n-1 degrees of freedom. The quantity s reflects the expected precision in the estimate of the mean (i.e., the distance that s could be from the true (population) mean). The smaller the variability (reflected by s), and the larger the sample size, the closer s was expected to be to the population mean. The UCL reflected this expectation. The larger s was and the smaller s was, the larger the quantity

 $t_{0.95,n-1} \frac{s}{\sqrt{n}}$ would be, and hence the higher the UCL (and the exposure point concentration) would be.

Because the exposure point concentration was based on the UCL, it was 95% certain to overestimate the true average exposure concentration. The degree to which the true average could be overestimated depended on the sample size (n) relative to the variability (s). For indoor air, preliminary discussions led to the conclusion that an assumed relative standard deviation (standard deviation divided by the mean) of 75% was a reasonable *a priori* estimate of variability within an AOC. Based on plots of precision versus sample size for a relative standard deviation of 75%, eight samples were 95% certain to yield a mean that was within 50% of the true mean. This was considered to be a reasonable sample size.

There were three sampling frequencies that were relevant for the indoor environment:

- Quarterly monitoring of indoor air at seven sites, including the Shirley Lanham
 Elementary School; housing units in Residential Towers 3101, 3102, 3043;
 Residential Townhouse 3025; and a work area, the Ground Electronics Maintenance
 Building, with two sequential samples collected at each site to yield 14 air samples
 plus 2 field blanks per quarter (whenever possible, ambient air samples were
 concurrently collected at these sites);
- Twice per month (approximately) indoor air monitoring at Site 7, the GEMB,
 between the quarterly air monitoring periods (collected concurrently with ambient air samples); and
- One time sampling for indoor dust and surface wipes at each of the seven sites, with one dust and one wipe sample collected at each site prior to the initiation of indoor air sampling in the first quarterly episode (16-20 July 1998). Therefore, indoor air monitoring was conducted for two weeks each quarter at all seven locations. The target chemicals and methods for sampling and analysis of indoor air were identical to those used for ambient air except for sampling duration, which was eight (daytime) hours per sample. The shorter duration of indoor air sampling (8 vs. 24 hours for ambient air) was necessitated by several factors: the smaller indoor interior volumes, the objective of sampling when educational facilities were occupied, and of sampling

A fairly consistent picture emerged over the four quarterly episodes of indoor air sampling in terms of the frequency of detection and the magnitude of indoor air concentrations relative to the RBC. There were 26 to 28 (depending on the quarter) chemicals detected in the indoor air, either with a frequency of detection of 50 to 100%, or whose concentrations exceeded their respective RBC value. For about 20 to 22 of these chemicals, depending on the quarter, the frequency of detection and the range of indoor air concentrations did not vary much between quarterly indoor air sampling episodes. The indoor air concentrations of these 20 to 22 chemicals were generally greater than the ambient (outdoor) air concentrations at the same time and place. However, there were six chemicals consistently found in the indoor air that did have two important characteristics:

- 1. Their concentrations in the ambient air were usually higher than in the indoor air; and
- 2. Their elevated concentrations in the ambient air were found to have a positive correlation with winds coming from the SIC to NAF Atsugi.

These six chemicals were: (1) dioxin TEQ, (2) hydrochloric acid, (3) cadmium, (4) lead, (5) arsenic, and (6) PM_{10} . Although it is possible that these six chemicals may have come from sources other than the SIC, on two occasions the data indicated that emissions from the SIC were related to an increase in their concentrations indoors.

As presented in Table 2-7, base wide average chemical concentrations found at all locations indicated that about 23 chemicals detected in greater than 50% of the indoor air samples were above the USEPA RBCs. Approximately 20 of these chemicals had maximum indoor air concentrations higher than maximum outdoor concentrations on the same day, suggesting that they are most likely originating from indoor emission sources such as building materials, furnishings and life style factors (e.g. smoking, cooking, cleaning, pets, etc.).

Table 2-7 shows the maximum ambient air concentrations for the listed chemicals, the ratio of this maximum to the RBC and the ratio of the maximum indoor air to maximum ambient air concentration (IA/AA). For the 28 chemicals listed in Table 2-7, the IA/AA ratio indicated that the maximum indoor air concentrations were usually greater than the maximum ambient air

concentrations measured for the same period. This result was not completely unexpected because indoor air concentrations for some pollutants (e.g., benzene, 1,4-dichlorobenzene), measured in the U.S., are generally higher than those measured in the ambient air. Outdoor air levels of dioxin TEQ, cadmium and lead generally were higher than indoor air levels. These chemicals were identified in the correlation regression analysis performed by Radian as being associated with emissions from the SIC.

The dioxin TEQ air concentration data were used as the best indicator of SIC impact on indoor air. Two notable instances were documented in which the elevated outdoor concentrations of the dioxin TEQ had a measurable effect on the indoor concentrations at sites with active ventilation (i.e., locations where air was actively pumped from outside to inside the building) versus passive ventilation. No elevation of dioxin concentrations was detected indoors at sites with passive ventilation, which would be expected since ambient air was not introduced into the buildings except through doors and windows being opened.

Since concentrations for the majority of the constituents exceeding RBCs were found to be higher indoors than outdoors indicating probable indoor air sources (e.g., insulation, carpets, and household chemicals); and ambient air was the source of constituents in indoor air that are associated with emissions from the SIC, indoor-air samples could not be used for the purposes of quantitative risk assessment because they would overestimate the impact of the SIC and other ambient air point and non-point sources. The results could be even higher if taken during occupancy, because of activities such as cooking, use of household cleaners and smoking. Since indoor air concentrations could not be used in the HRA to calculate indoor air exposures from outdoor air infiltration without overestimating the risk, ambient air concentrations detected on the same sampling day were used as surrogate indoor air concentrations.

Public buildings and areas such as the corridor in residential tower 3101 or 3102, the Ground Electronics Maintenance Building, the Child Development Center and the Elementary School had active, forced fresh air ventilation systems (i.e., high volume blowers, ducts, registers, etc.). Base residential units had no active forced fresh air ventilation systems (passive ventilation). They included residential units in the newer towers 3101 and 3102, a residential unit in an older tower 3043 and several residential townhouse units (e.g., 3025, 3023, 3043,

Table 2-7 — Representative Target Chemicals Detected in Atsugi Indoor Air that Often Exceeded Their RBC Value

Group					Indo	or Air		Ambi	ent Air	Ratio	
Group	Target Chemicals	RBC	Units	Freq %	Mean	Maximum	Max/RBC	Max	Max/RBC	IA/AA	Target Chemicals
1	VOCs										VOCs
	Benzene	0.22	$\mu g/m^3$	100	3.92	10.7	48.6	9.76	44.4	1.1	Benzene
•	Carbon tetrachloride	0.12	μg/m ³	98.5	0.62	1.22	10.2	1.03	8.6	1.2	Carbon tetrachloride
	Chloromethane	1	$\mu g/m^3$	100	1.66	3.01	3.0	2.90	2.9	1.0	Chloromethane
	Tetrachloroethylene	3.1	$\mu g/m^3$	92.3	1.32	3.39	1.1	4.08	1.3	0.83	Tetrachloroethylene
•	Trichloroethylene	1	μg/m ³	95.4	1.67	5.37	5.4	9.09	9.1	0.59	Trichloroethylene
	Chloroform	0.077	μg/m ³	83.1	0.54	2.44	31.7	0.95	12.3	2.6	Chloroform
•	Hexachloro-1,3-	0.8	μg/m ³	24.2	0.43	2.17	1.4	6.41	8.0	0.34	Hexachloro-1,3-
	butadiene										butadiene
	1,3-Butadiene	0.0035	μg/m ³	87.7	0.38	1.38	394	1.08	309	1.3	1,3-Butadiene
	Methylene chloride	3.8	$\mu g/m^3$	100	95.4	944	248	393	103	2.4	Methylene chloride
	Acetonitrile	51	$\mu g/m^3$	76.9	94.11	1445	28	716	14.0	2.0	Acetonitrile
2	Aldehydes/Ketones										Aldehydes/Ketones
	Acetaldehyde	0.81	$\mu g/m^3$	100	12.75	63.6	78.5	10.2	12.6	6.2	Acetaldehyde
	Acrolein	0.021	$\mu g/m^3$	64.5	0.75	3.39	161	0.41	19.5	8.3	Acrolein
	Formaldehyde	0.14	$\mu g/m^3$	100	26.15	86.5	618	10.2	73	8.5	Formaldehyde
	Crotonaldehyde	0.0033	$\mu g/m^3$	27.1	0.10	3.3	1000	1.15	348	2.9	Crotonaldehyde
3	PM_{10}	150	$\mu g/m^3$	100	76.23	388	2.6	239	1.6	1.6	PM_{10}
	Metals										
•	Cadmium	0.00099	μg/m ³	93.8	0.003	0.0248	25.1	0.167	169	0.15	Cadmium
•	Arsenic	0.00041	μg/m ³	62.5	0.003	0.0191	46.6	0.0071	17.3	2.7	Arsenic
•	Chromium	0.0021	μg/m ³	100	0.011	0.0584	27.8	0.0174	8.3	3.4	Chromium
-	Lead	1.5	$\mu g/m^3$	100	0.11	0.557	0.4	1.11	0.7	0.5	Lead
4	Mercury	0.310	μg/m ³	78	0.109	2.68	8.6	24.8	80.0	0.1	Mercury
5	Acid Gases		-								Acid Gases
	Hydrochloric acid	21	$\mu g/m^3$	100	2.28	31.4	1.50	26.8	1.27	1.2	Hydrochloric acid
6	Pesticides										Pesticides
	Chlordane	0.0049	μg/m ³	40	0.001	7.9	1.6	0.82	0.2	9.6	Chlordane

Table 2-7 — Representative Target Chemicals Detected in Atsugi Indoor Air that Often Exceeded Their RBC Value

Group					Indo	or Air		Ambi	ent Air	Ratio	
Group	Target Chemicals	RBC	Units	Freq %	Mean	Maximum	Max/RBC	Max	Max/RBC	IA/AA	Target Chemicals
	BHC (Lindane)	0.0048	μg/m ³	46.15	0.003	64.7	13.5	4.2	0.9	15.4	BHC (Lindane)
	Heptachlor	0.0014	μg/m ³	40	0.004	46.2	33.0	1.37	0.3	33.7	Heptachlor
7	PCDDs/PCDFs										PCDDs/PCDFs
	Dioxin	0.042	pg/m ³	100	0.6	2.07	49.3	13.2	315	0.16	2,3,7,8-TCDD TEQ
8	SVOCs										SVOCs
	1,4-Dichlorobenzene	0.28	$\mu g/m^3$	100	18.39	422	1507	2.33	8.3	181	1,4-Dichlorobenzene
	Naphthalene	3.3	μg/m ³	98.5	1.60	14.9	40.2	0.82	0.2	18.1	Naphthlene
	Acetophenone	0.021	$\mu g/m^3$	66.1	1.76	19.7	938	0.65	31.0	30.3	Acetophenone

3045, 3053 and 3068). A fairly consistent picture emerged over the four quarterly sampling periods in terms of frequency of detection and the magnitude of indoor air concentrations relative to risk based concentrations.

Details on the indoor air sampling can be found in Section 5 of the air monitoring summary report developed by Radian (Radian 2000a).

Indoor Dust

Dioxins and furans were selected as indicators as to whether contaminants from the SIC were infiltrating or being tracked into homes and therefore to evaluate the potential for an ingestion route of exposure due to deposited or tracked-in dust. The indoor dust and wipe samples were analyzed only for dioxins and furans to maximize the collection of information within the available resources, since they were the chemicals that were most likely to only be related to the SIC versus other chemicals that could be related to household cleaning, smoking, food odors, etc. Given that dioxin concentrations in indoor carpet/floor dust and in the dust on indoor surfaces were available, they were used to evaluate dermal/ingestion risk due to deposited or "tracked in dust". Since only dioxins and furans concentrations were available for indoor dust, soil surrogate concentrations were used in the comprehensive HRA to estimate dermal/ingestion risk for other chemicals potentially infiltrated or tracked into homes.

Two types of surface samples were collected at each site. The carpet or floor dust samples were collected according to the American Standards of Testing Materials (ASTM) Method D5438-93: Standard Practice for Collection of Dust from Carpeted Floors for Chemical Analysis using the CS₃ Inc. Model HVS3 High Volume (vacuum) Sampler. Carpeted or floor areas of 5-10 m² were vacuumed. The vacuumed material included fine (sieved) dust samples of 0.7-10 grams. The carpeted or floor area vacuumed to obtain the dust sample was selected to be in a high traffic/easily accessible area at the sampling site. As such, it was selected to be representative primarily of "tracked in" dust, although the sample could also have had some contribution from prior airborne deposition.

The surface wipe samples were collected using an industrial hygiene approach in which a (4 inch by 4 inch) cotton gauge pad (pre-extracted and wet with isopropyl alcohol) was wiped over a horizontal surface to pick up the surface dust. Multiple horizontal surfaces were wiped at each site to collect as much dust as the wipe would accommodate. Areas of undisturbed dust

were sought out, e.g. the top of the refrigerator, light fixtures, tall bookcases, etc. As such, the areas selected for wipe sampling were not readily accessible to the residents/occupants of the room and would be more representative of accumulated dust of variable age. The area accessible for wiping and the dust loading was highly variable from site to site, so that the quantity of dust per wipe was not quantifiable, but could have varied between 0.1 to 1.0 gram per wipe for wiped areas between 0.5 to 2 m².

The dioxin range of concentrations in the carpet/floor dust samples was 17-210 pg TEQ/g, which were higher than maximum dioxin (TEQ) concentrations in the soil on base, indicating that outdoor soil and/or air contamination were infiltrating in the homes. The dioxin levels in the wipe samples had a large range (86-4200 pg TEQ/m²). Details on these analytical results can be found in the Radian air summary report (Radian 2000a).

The results of the microscopical analysis of the floor dust samples indicated that the dust particles were primarily comprised of minerals, clay, cotton fibers, and/or plant or starchy tissue. Only low trace amounts of any components that could be attributed to a combustion product were found. Details on the results of this microscopical analysis can be found in the report developed by Micro Sciences (Micro-Sciences 1999a). The method used to collect floor dust samples was an ASTM method that collected particles smaller than 5 µm.

Since no combustion particles were found in the fraction containing particles less than 5 μ m and following the assumption that combustion products may be found in particles greater than 5 μ m, two PM₁₀ filter samples (one indoor and another outdoor) were also subjected to a microscopical analysis. The results of the analysis indicated major amounts of charred carbonized fragments that are indicative of a combustion source such as an incinerator. The PM₁₀ samples also contained a significant level of fine (sub micrometer-size) carbon particles that are characteristic of vehicle exhaust (e.g. diesel exhaust). Details on the results of this microscopical analysis can be found in the report developed by Micro Sciences (Micro-Sciences 1999b).

2.7 Soil Sampling

The emissions from the SIC were known to have a large amount of particulates which were expected to be deposited in the soil over the NAF Atsugi area. In addition, the strong

winds carried the stockpiled fly ash at the SIC over to NAF Atsugi property. Again, the close proximity of the recreational and residential areas located at NAF Atsugi warranted a concern for potential dermal and ingestion exposures from the pollutants being deposited in the soil.

The objectives of the soil-sampling program were to:

- 1. Provide data to help determine the risk from soil at each specified AOC; and
- 2. Determine whether there had been any soil contamination at NAF Atsugi that could be attributed to emissions from the SIC.

The purpose of the soil sampling was to help address the following two questions:

- What were the risks to sensitive receptors from dermal contact or incidental
 ingestion of soil contaminated by COCs that were likely to be associated with
 ambient air emissions and/or subsequent deposition from point and non-point
 sources impacting the air quality at NAF Atsugi?
- What was the extent of deposition in the soil of particulates from the SIC?

The sampling program monitored the soil from 0 to 12 inches below the ground surface to determine the extent of deposition of pollutants from the SIC. Samples were collected across NAF Atsugi property with the majority being collected at AOCs where receptors normally cluster, such as at associated playground, outdoor eating, or common areas at the Residential Towers, Elementary School, and Child Development Center. Other samples were collected farther away from the SIC to determine how far deposition may have occurred. Reference samples were collected from areas believed to be minimally impacted for subsequent comparison of results to those from impacted areas. The soil sampling in the AOCs was meant to help address the first of these questions, and the samples collected throughout the base were intended to address the second question. The aim of the sampling performed in the potential reference areas was to collect data from areas that were unaffected, or minimally affected, by the SIC or other potential contaminant sources. These data would be used to ascertain the degree of impact over "natural" background. Table 2-8 presents the number of soil samples collected by area for the first round (Phase I) of soil sampling described in this plan. The results of this Phase I soil sampling were used to establish where potential second event samples should be located, and what analyses would be performed in case data gaps were identified.

Sufficient soil samples were collected to be 95% certain that the sample mean would be within 50% of the true mean. The number of samples needed to provide this level of significance was determined from a curve of precision (maximum percent difference expected between the sample mean and the true mean) versus sample size. The curve was calculated under the assumption that the variability among concentrations was such that the standard deviation relative to the mean was 75%. Although the correct standard deviation relative to the mean varied by location and chemical, this assumed value was considered to be reasonable based on historical soil sampling data. The precision curve indicated that for each AOC, eight surface soil samples would yield an adequate sample size. Since the decision to treat the Residential Towers area as one exposure unit or as two would occur subsequent to the data evaluation, twelve surface soil samples were collected for the Residential Towers, six from each tower.

A site visit was performed prior to sampling to determine where samples would be collected for a soil trend analysis. Rather than using a statistical evaluation to determine the number and tentative locations of samples to be collected for the trend analysis, a sampling scheme was based on best professional judgment. Locations selected were undisturbed areas, likely to have been impacted by incinerator depositions, but not by other chemical contamination practices.

To determine the deposition trends across NAF Atsugi, the base was divided into areas defined by seven radii starting at the SIC and extending to the north, with transects at distances of less than 300 m, 300 m to 800 m, 800 m to 1500 m, and greater than 1500 m from the SIC, as shown in Figure 2-1. For trend analysis purposes, samples were collected from areas of potential sediment accumulation, stressed vegetation, and those lacking evidence of erosion or ground cover, where possible.

Consequently, Radian performed soil sampling in two phases. Phase I was conducted from 5 to 18 March 1998 at NAF Atsugi to support the HRA. A total of 102 field samples were collected from AOCs, potential reference (similar to "background") areas, and at individual locations across the base. Concentrated sampling was performed at the designated AOCs, which included the Child Development Center, the Shirley Lanham Elementary School, the area surrounding Residential Housing Towers 3101 and 3102, and at the two potential reference areas located on the far western side of NAF Atsugi. Less dense sampling was performed at the 33.

Table 2-8 — Phase I Soil Sampling at NAF Atsugi, Japan

	Samplir	ng Event #1	
Sample Area	Surface Soil Samples (0-3 in)	Subsurface Soil Samples (3-12 in)	Total Field Samples
Specified Areas of Concern			
Child Development Center	8	4	12
Elementary School	8	4	12
Residential Housing Towers 1&2	12	4	16
Dispersion Trend Locations			
Ambient Air Monitoring Stations	3	3	6
Basewide	30	8	38
Reference Locations (2)	12	6	18
Total Field Samples	73	29	102
QC Samples ^a			
Field duplicates			11
Equipment Rinsates ^b			11
Matrix Spikes (MS)			5
Matrix Spike Duplicates (MSD)			5
Field Blanks			1
Total QC Samples			33
Total Phase #1 Samples			135

^a QA/QC samples included 10% field duplicates, 5% MS, 5% MSDs (or 1 MS/MSD pair per twenty samples), one equipment rinsate per day (rinsates from every other day were analyzed; -- others were held and analyzed only if there was evidence of contamination), and one field blank per event.

^b Assumed Phase I soil sampling would last 10 days.

locations interspersed across the base to support the trend analysis. Of the 102 samples, 73 were from the surface interval (0-3 in.) and 29 were from the subsurface interval (3-12 in.).

The results were evaluated statistically and reported in the March 1998 Soil Sampling report (Radian 1998d). During statistical evaluations in report preparation, it was apparent that one of the reference areas (Reference Area 1) was less impacted by contaminant sources than the other (Reference Area 2); therefore, only those data from Reference Area 1 were used in site-to-reference comparisons. By using only half of the potential reference area sample results, the confidence and power of the statistical comparisons for risk assessment purposes were reduced. Following data and report release and risk calculations, questions about the true "background" concentrations of some metals at NAF Atsugi arose. Overall, both the reference area and overall base-wide concentrations of several metals were higher than expected, or found in nature. Thus, the assigned risks from metals were questioned. For example, if the reference area soil samples had been impacted by a contaminant source, leading to higher concentrations, then a comparison to other locations across the base (e.g., AOCs) would not indicate a source-related problem, when actually present.

After several discussions with the overall project team (risk assessors, and soil samplers), the decision was made to locate an area on the base which was possibly less impacted by SIC operations or other potential sources than the reference area used for statistical comparisons with Phase I sample data. The Phase I potential reference areas were selected based on a review of potential source locations and historical wind directions and land use, but were nevertheless in areas where the soil was exposed to the atmosphere. Therefore, Phase II sampling was conducted on 18 and 19 January 1999, to collect soils in an area(s) unlikely to have been impacted by SIC. Sixteen samples were collected for metal analysis beneath and outside Building 47, the Bachelors Enlisted Quarters, scheduled for demolition. Since this building was constructed prior to the introduction of the SIC, the area under this building was selected as a non-impacted site suitable for collecting background samples. Eight extra samples were collected from background Reference Area 1, previously sampled in Phase I. In addition, three randomly selected, co-located (with metals) samples were collected for organic compound analyses to further verify the "background", or uncontaminated condition of the soils, one from each of the above listed sample areas. To confirm or modify the reference area metal concentrations, which were used in the Phase I statistical comparisons and to determine the least

impacted area(s), three sequential comparisons were performed. For each comparison, one of two conclusions was reached: either one area was determined to have statistically significant lower concentrations than the other, or the two areas were found to have comparable concentrations. The comparisons were as follows:

- Chemical concentrations from beneath Building 47 were compared to concentrations from outside Building 47. Concentrations were found to be lower in samples beneath Building 47 than those outside the building.
- Chemical concentrations in Reference Area 1 samples collected during Phase I were compared to Phase II Reference Area 1. The concentrations were found to be comparable.
- Chemical concentrations from beneath Building 47 were compared to Reference Area 1 samples (Phase I and Phase II combined). The concentrations in samples from beneath Building 47 were found to be lower than those in combined samples from Reference Area 1.

The conclusion of these comparisons was that soil from beneath Building 47 appeared to be the least impacted by contaminant sources. Thus, the recommendation was to use the samples collected from beneath Building 47 as being representative of reference concentrations of inorganic compounds in soil. These background concentrations were used in the HRA to eliminate COCs associated with natural background.

All samples were analyzed for Contract Laboratory Program (CLP) semivolatile organic compounds (SVOCs), CLP organochlorine pesticides and polychlorinated biphenyls (PCBs), CLP metals (including cyanide), polychlorinated dibenzo-p-dioxins and furans (PCDDs and PCDFs), percent moisture, and pH. A subset of the samples was analyzed for anions (chloride, fluoride, sulfate, and nitrate), total organic carbon, and particle size distribution (PSD). GP Environmental Services, Inc. performed all analyses except those involving dioxins and PSD. Dioxins were analyzed by Triangle Laboratories and PSD analyses was performed by Radian. Following analyses, the data for CLP and dioxin analyses were validated by EcoChem, Inc. None of the soil data was rejected.

Areas of Concern

The results for the surface soil investigation (0-3 inches) at the Child Development Center, Elementary School, and Residential Towers are summarized in Table 2-9, which presents average and RME point concentrations. Eight surface soil and four subsurface soil samples were collected from each of these AOCs. These samples were analyzed for moisture, CLP SVOCs, CLP organochlorine pesticides and PCBs, CLP metals, and polychlorinated dibenzo-p-dioxins and furans (PCDDs and PCDFs). Details of the Phase I soil sampling, including the number of samples and analyses performed on samples from each site, are presented in the Radian soil report (EPA 1998b). Details of the Phase II soil sampling can be found in the Phase II soil sampling addendum report (Radian 1999f).

Surface Soil

As a risk-screening tool, soil concentrations were compared with EPA Region 3 RBCs. A comparison of surface soil results with the RBCs indicated that total dioxin and furans (2,3,7,8 TEQs) exceeded the RBC of 0.0000043 mg/kg for the average and the RME concentrations.

Among several metals that were detected only average and RME concentrations for arsenic exceeded the RBC of 0.43 mg/kg. SVOCs, organochlorine pesticides, PCBs, and cyanide were not found at concentrations exceeding the RBCs in any of the surface soil samples from these sites. Aluminum, arsenic, iron, manganese, thallium and vanadium were detected at all three sites at concentrations exceeding the RBCs. The concentrations of metals in the surface soils were not significantly different than those in background Reference Area 1. Lead was detected in all locations at levels below 100 mg/kg, which was well below the RBC (400 mg/kg).

Subsurface Soil

A comparison of subsurface soil results with the RBCs indicated that none of the toxic PCDD/PCDF congeners were found in the subsurface soil sampled at the Child Development Center, Residential Towers, except for the Elementary School, at concentrations exceeding the respective RBCs. Organochlorine pesticides, PCBs, and cyanide were not found at concentrations exceeding the RBCs in any of the surface soil samples from these sites. SVOCs (benzo(a) pyrene and dibenzo(a,h) anthracene), which exceeded the RBC of 0.0087 mg/kg, were detected only in one sample at the Elementary School. Benzo(a) pyrene was detected in only

Table 2-9- NAF Atsugi, Japan Surface Soil (0-3") Concentrations

		Range of Detected Values		Range of Detection Limits for Non- Detected Samples						
Compound	Number of Detected Results/Total Samples	Minimum	Maximum	Minimum	Maximum	Mean ^a	Median ^a	Standard Deviation ^a	Upper 95% Confidence Limit ^a	RBC ^{b.}
	·	•		Child Dev	elopment Ce	nter				
Anions and Nitrate (mg/kg)										
Chloride	1/1	1.15	1.15	NA	NA	1.15	1.15	NA	NA	No RBC
Fluoride	1/1	2.04	2.04	NA	NA	2.04	2.04	NA	NA	No RBC
Sulfate	1/1	7.28	7.28	NA	NA	7.28	7.28	NA	NA	No RBC
Nitrate	1/1	6.36	6.36	NA	NA	6.36	6.36	NA	NA	782.14
рН										
Н	9/9	5.25	9.14	NA	NA	6.87	6.83	1.36	7.71	No RBC
norganics (mg/kg)										
Aluminum	9/9	6530.00	44000.00	NA	NA	21428.89	16200.00	13472.02	29781.54	7821.43
Antimony	3/9	0.63	1.80	0.02	0.43	0.44	0.17	0.58	0.80	3.13
Arsenic	9/9	1.10	3.70	NA	NA	2.78	3.30	0.96	3.37	0.04
Barium	9/9	12.10	79.60	NA	NA	32.69	17.60	23.83	47.46	547.50
Beryllium	6/9	0.009	0.21	0.16	0.22	0.07	0.06	0.06	0.11	15.64
Cadmium	9/9	0.12	1.10	NA	NA	0.56	0.43	0.34	0.77	7.82
Calcium	9/9	3130.00	12200.00	NA	NA	8505.56	7900.00	3213.29	10497.79	No RBC
Chromium	9/9	5.00	26.10	NA	NA	12.11	7.80	7.60	16.83	No RBC
Cobalt	9/9	2.20	17.30	NA	NA	7.82	4.50	5.66	11.33	469.29
Copper	9/9	8.20	92.00	NA	NA	35.50	14.20	31.38	54.96	312.86
Cyanide	9/9	0.43	0.97	NA	NA	0.63	0.56	0.18	0.74	156.43
ron	9/9	6090.00	38700.00	NA	NA	19191.11	14500.00	11474.02	26305.01	2346.43
Lead	9/9	3.1	23.80	NA	NA	13.17	14.50	7.96	18.10	No RBC

Table 2-9- NAF Atsugi, Japan Surface Soil (0-3") Concentrations

		Range of Detected Values		Range of Detection Limits for Non- Detected Samples						
Compound	Number of Detected Results/Total Samples	Minimum	Maximum	Minimum	Maximum	Mean ^a	Median ^a	Standard Deviation ^a	Upper 95% Confidence Limit ^a	RBC ^{b.}
Magnesium	9/9	1370.00	7400.00	NA	NA	3992.22	3050.00	2247.70	5385.80	No RBC
Manganese	9/9	76.40	767.00	NA	NA	326.07	203.00	243.20	476.85	156.43
Mercury	7/9	0.01	0.15	0.02	0.02	0.04	0.03	0.04	0.07	No RBC
Nickel	9/9	4.20	24.00	NA	NA	11.20	6.30	7.32	15.74	156.43
Potassium	9/9	252.00	1000.00	NA	NA	585.33	637.00	231.64	728.95	No RBC
Selenium	1/9	0.40	0.40	0.04	0.30	0.12	0.09	0.11	0.19	39.11
Silver	8/9	0.008	0.29	0.15	0.15	0.12	0.09	0.09	0.17	39.11
Sodium	9/9	340.00	1200.00	NA	NA	770.89	864.00	326.64	973.40	No RBC
Γhallium	6/9	0.15	0.79	0.54	0.59	0.39	0.28	0.22	0.53	0.55
Vanadium	9/9	19.10	151.00	NA	NA	67.74	43.00	48.47	97.80	54.75
Zinc	9/9	26.50	125.00	NA	NA	75.36	63.20	38.76	99.39	2346.43
Organics (mg/kg)										
1,4'-DDE	7/9	0.0006	0.03	0.0002	0.0002	0.01	0.01	0.01	0.02	0.19
1,4'-DDT	7/9	0.002	0.05	0.0002	0.0002	0.02	0.008	0.02	0.03	0.19
Butylbenzylphthalate	1/9	0.13	0.13	0.04	0.04	0.03	0.02	0.04	0.05	1564.29
Dieldrin	1/9	0.002	0.002	0.0002	0.0003	0.0003	0.00010	0.0007	0.0008	0.004
alpha-Chlordane	3/9	0.0008	0.0010	0.0002	0.0002	0.0004	0.0001	0.0004	0.0007	0.18
ois(2-Ethylhexyl)phthalate	8/9	0.09	0.46	0.04	0.04	0.22	0.22	0.13	0.31	4.56
li-n-Butylphthalate	3/9	0.05	0.20	0.04	0.06	0.06	0.03	0.06	0.10	782.14
li-n-Octylphthalate	1/9	0.05	0.05	0.04	0.06	0.02	0.02	0.01	0.03	156.43
gamma-Chlordane	2/9	0.0009	0.0010	0.0002	0.0002	0.0004	0.0001	0.0004	0.0007	0.18
Γotal Dioxin/Furans (2,3,7,8-										
	9/9	0.00000091	0.00003	NA	NA	0.00001	0.00001	0.00001	0.00002	0.0000004

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Table 2-9- NAF Atsugi, Japan Surface Soil (0-3") Concentrations

		Range of Detected Values		Range of Detection Limits for Non- Detected Samples						
Compound	Number of Detected Results/Total Samples	Minimum	Maximum	Minimum	Maximum	Mean ^a	Median ^a	Standard Deviation ^a	Upper 95% Confidence Limit ^a	RBC ^{b.}
				Eleme	ntary School					
Anions and Nitrate (mg/kg)										
Chloride	2/2	0.67	8.17	NA	NA	4.42	4.42	5.31	28.11	No RBC
Fluoride	1/2	1.63	1.63	0.27	0.27	0.88	0.88	1.06	5.60	No RBC
Sulfate	2/2	0.27	13.40	NA	NA	6.83	6.83	9.29	48.30	No RBC
Nitrate	2/2	0.71	16.30	NA	NA	8.51	8.51	11.02	57.72	782.14
рН										
рН	9/9	6.74	8.98	NA	NA	8.00	8.43	0.89	8.55	No RBC
Inorganics (mg/kg)										
Aluminum	9/9	8290.00	72600.00	NA	NA	28632.22	19000.00	22029.43	42290.47	7821.43
Antimony	6/9	0.46	2.50	0.32	0.42	0.77	0.46	0.78	1.26	3.13
Arsenic	9/9	1.80	6.50	NA	NA	3.96	3.80	1.34	4.79	0.04
Barium	9/9	12.50	143.00	NA	NA	44.42	18.80	44.20	71.82	547.50
Beryllium	9/9	0.07	0.35	NA	NA	0.15	0.11	0.10	0.21	15.64
Cadmium	9/9	0.14	1.30	NA	NA	0.46	0.25	0.40	0.71	7.82
Calcium	9/9	9570.00	18700.00	NA	NA	12395.56	11400.00	2924.87	14208.97	No RBC
Chromium	9/9	4.90	51.40	NA	NA	15.96	10.20	14.99	25.25	No RBC
Cobalt	9/9	2.90	27.60	NA	NA	10.89	7.70	8.85	16.38	469.29
Copper	9/9	7.10	152.00	NA	NA	49.30	26.70	52.23	81.68	312.86
Cyanide	1/9	0.16	0.16	0.22	0.48	0.15	0.13	0.04	0.17	156.43
Iron	9/9	10000.00	64100.00	NA	NA	26088.89	19600.00	18456.93	37532.19	2346.43
Lead	9/9	2.70	61.50	NA	NA	12.81	3.70	18.97	24.57	No RBC

Table 2-9- NAF Atsugi, Japan Surface Soil (0-3") Concentrations

		Range of Detected Values		Range of Detection Limits for Non- Detected Samples						
Compound	Number of Detected Results/Total Samples	Minimum	Maximum	Minimum	Maximum	Mean ^a	Median ^a	Standard Deviation ^a	Upper 95% Confidence Limit ^a	RBC ^{b.}
Magnesium	9/9	2140.00	9970.00	NA	NA	5822.22	5480.00	2828.79	7576.07	No RBC
Manganese	9/9	128.00	1140.00	NA	NA	446.67	294.00	350.77	664.14	156.43
Mercury	3/9	0.01	0.13	0.02	0.02	0.03	0.01	0.04	0.06	No RBC
Nickel	9/9	4.20	37.50	NA	NA	15.21	13.60	11.33	22.24	156.43
Potassium	9/9	492.00	1060.00	NA	NA	723.22	640.00	212.40	854.91	No RBC
Selenium	1/9	0.51	0.91	0.005	0.31	0.23	0.14	0.29	0.41	39.11
Silver	9/9	0.01	0.50	NA	NA	0.15	0.09	0.15	0.25	39.11
Sodium	9/9	502.00	1210.00	NA	NA	795.44	781.00	260.46	956.93	No RBC
Γhallium	1/9	1.40	1.60	0.07	0.62	0.52	0.28	0.57	0.87	0.55
Vanadium	9/9	25.10	263.00	NA	NA	92.56	56.40	82.98	144.01	54.75
Zinc	9/9	22.10	274.00	NA	NA	66.71	37.40	79.58	116.05	2346.43
Organics (mg/kg)										
4,4'-DDD	1/9	0.12	0.12	0.0002	0.0003	0.01	0.00010	0.04	0.04	0.27
4,4'-DDE	3/9	0.003	0.04	0.0002	0.0002	0.008	0.00010	0.01	0.02	0.19
4,4'-DDT	3/9	0.003	0.05	0.0002	0.0002	0.009	0.00010	0.02	0.02	0.19
Aroclor-1254	1/9	0.04	0.04	0.0002	0.0003	0.005	0.00010	0.01	0.01	0.03
Butylbenzylphthalate	1/9	0.10	0.10	0.04	0.05	0.03	0.02	0.03	0.04	1564.29
Chrysene	1/9	0.07	0.07	0.04	0.05	0.03	0.02	0.02	0.04	8.75
Diethylphthalate	1/9	0.05	0.05	0.04	0.07	0.02	0.02	0.010	0.03	6257.14
Heptachlor	1/9	0.01	0.01	0.0002	0.0003	0.001	0.00010	0.004	0.004	0.01
Total Carcinogenic PAHS	1/9	0.08	0.08	0.08	0.12	0.05	0.04	0.01	0.06	0.009
alpha-Chlordane	1/9	0.003	0.22	0.0002	0.0003	0.02	0.00010	0.07	0.07	0.18

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Table 2-9- NAF Atsugi, Japan Surface Soil (0-3") Concentrations

		Range of Detected Values		Range of Detection Limits for Non- Detected Samples						
Compound	Number of Detected Results/Total Samples	Minimum	Maximum	Minimum	Maximum		Median ^a	Standard Deviation ^a	Upper 95% Confidence Limit ^a	RBC ^{b.}
bis(2-Ethylhexyl)phthalate	9/9	0.14	0.98	NA	NA	0.36	0.24	0.27	0.53	4.56
di-n-Butylphthalate	2/9	0.09	0.35	0.04	0.05	0.10	0.02	0.14	0.18	782.14
gamma-Chlordane	1/9	0.22	0.22	0.0002	0.0003	0.02	0.00010	0.07	0.07	0.18
Total Dioxin/Furans (2,3,7,8-	9/9	0.00000084	0.00009	NA	NA	0.00002	0.00003	0.00003	0.00003	0.0000004
ГСDD TEQs) ^b				Dag!da	 ential Towers					
Anions and Nitrate (mg/kg)	T			Reside	muai Towers				Ι	T
Chloride	3/3	2.29	11.40	NA	NA	7.54	8.93	4.71	15.48	No RBC
Fluoride	2/3	0.14		0.27	0.27	0.64	0.14	0.87	2.11	No RBC
Sulfate	3/3			NA	NA	43.77	44.90	9.95	60.54	No RBC
Nitrate	3/3			NA	NA	17.71	23.10	11.75	37.52	782.14
ρΗ										, , , , , , , , , , , , , , , , , , , ,
эН	13/13	6.02	8.88	NA	NA	7.26	7.54	1.02	7.76	No RBC
Inorganics (mg/kg)										
Aluminum	13/13	13500.00	78800.00	NA	NA	49469.23	55100.00	22987.90	60830.73	7821.43
Antimony	10/13	0.77	2.70	0.29	0.42	1.44	1.40	0.81	1.85	3.13
Arsenic	13/13	2.60	8.30	NA	NA	4.27	3.90	1.52	5.02	0.04
Barium	13/13	13.80	609.00	NA	NA	108.72	86.20	154.07	84.87	547.50
Beryllium	7/13	0.09	0.36	0.15	0.25	0.17	0.12	0.10	0.22	15.64
Cadmium	13/13	0.14	2.30	NA	NA	1.00	0.96	0.63	1.31	7.82
Calcium	13/13	3520.00	27700.00	NA	NA	14239.23	11600.00	6901.14	17650.03	No RBC
Chromium	13/13	6.30	47.90	NA	NA	30.14	36.50	14.85	37.48	No RBC

Table 2-9- NAF Atsugi, Japan Surface Soil (0-3") Concentrations

		Range of Detected Values		Limits	Detection for Non- I Samples					
Compound	Number of Detected Results/Total Samples	Minimum	Maximum	Minimum	Maximum	Mean ^a	Median ^a	Standard Deviation ^a	Upper 95% Confidence Limit ^a	RBC ^{b.}
Cobalt	13/13	3.50	29.00	NA	NA	19.55	21.90	9.47	24.23	469.29
Copper	13/13	7.50	150.00	NA	NA	97.61	107.00	52.51	123.56	312.86
Cyanide	10/13	0.09	1.90	0.17	0.23	0.61	0.37	0.61	0.92	156.43
Iron	13/13	11100.00	64400.00	NA	NA	43430.77	52700.00	19641.72	53138.45	2346.43
Lead	13/13	3.00	97.50	NA	NA	28.39	23.40	29.78	43.11	No RBC
Magnesium	13/13	2450.00	11700.00	NA	NA	8179.23	8990.00	3385.12	9852.29	No RBC
Manganese	13/13	173.00	1200.00	NA	NA	775.62	891.00	374.85	960.88	156.43
Mercury	13/13	0.01	0.14	NA	NA	0.06	0.05	0.04	0.08	No RBC
Nickel	13/13	5.70	38.80	NA	NA	26.19	31.40	12.04	32.14	156.43
Potassium	13/13	198.00	1110.00	NA	NA	696.08	676.00	278.37	833.66	No RBC
Selenium	2/13	0.40	0.74	0.02	0.45	0.20	0.15	0.19	0.29	39.11
Silver	13/13	0.05	0.43	NA	NA	0.27	0.30	0.13	0.33	39.11
Sodium	13/13	533.00	1970.00	NA	NA	1158.54	1240.00	439.45	1375.73	No RBC
Thallium	9/13	0.14	2.50	0.16	0.99	1.23	1.20	0.84	1.65	0.55
Vanadium	13/13	34.80	287.00	NA	NA	173.87	190.00	88.70	217.71	54.75
Zinc	13/13	25.50	246.00	NA	NA	107.35	94.30	69.65	141.78	2346.43
Organics (mg/kg)										
4,4'-DDD	5/13	0.004	0.02	0.0002	0.0003	0.003	0.0001	0.005	0.006	0.27
4,4'-DDE	10/13	0.002	0.17	0.0002	0.0002	0.04	0.02	0.05	0.07	0.19
4,4'-DDT	10/13	0.002	0.24	0.0002	0.0002	0.05	0.04	0.06	0.08	0.19
Benzo(a)anthracene	1/13	0.14	0.14	0.04	0.07	0.03	0.03	0.03	0.05	0.09
Benzo(a)pyrene	1/13	0.23	0.23	0.04	0.07	0.04	0.03	0.06	0.07	0.009

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Table 2-9- NAF Atsugi, Japan Surface Soil (0-3") Concentrations

			Range of Detected Values		Range of Detection Limits for Non- Detected Samples					
Compound	Number of Detected Results/Total Samples	Minimum	Maximum	Minimum	Maximum	Mean ^a	Median ^a	Standard Deviation ^a	Upper 95% Confidence Limit ^a	RBC ^{b.}
Benzo(b)fluoranthene	1/13	0.18	0.18	0.04	0.07	0.04	0.03	0.04	0.06	0.09
Benzo(g,h,i)perylene	1/13	0.17	0.17	0.04	0.07	0.04	0.03	0.04	0.06	No RBC
Benzo(k)fluoranthene	1/13	0.20	0.20	0.04	0.07	0.04	0.03	0.05	0.06	0.87
Butylbenzylphthalate	2/13	0.09	0.10	0.04	0.07	0.04	0.03	0.03	0.05	1564.29
Chrysene	2/13	0.05	0.17	0.04	0.07	0.04	0.03	0.04	0.06	8.75
Diethylphthalate	2/13	0.08	0.11	0.04	0.06	0.04	0.03	0.03	0.05	6257.14
Fluoranthene	5/13	0.06	0.11	0.04	0.07	0.04	0.03	0.03	0.06	312.86
Heptachlor	1/13	0.0009	0.0009	0.0002	0.0004	0.0002	0.0001	0.0002	0.0003	0.01
Heptachlor epoxide	2/13	0.001	0.002	0.0002	0.0004	0.0003	0.0001	0.0005	0.0006	0.007
Pyrene	7/13	0.06	0.16	0.04	0.06	0.05	0.06	0.04	0.07	234.64
Total Carcinogenic PAHS	2/13	0.06	0.29	0.08	0.16	0.08	0.06	0.07	0.11	0.009
alpha-Chlordane	5/13	0.002	0.008	0.0002	0.0004	0.002	0.0001	0.002	0.003	0.18
bis(2-Ethylhexyl)phthalate	13/13	0.06	0.76	NA	NA	0.29	0.20	0.22	0.40	4.56
di-n-Butylphthalate	7/13	0.06	0.20	0.04	0.06	0.08	0.06	0.07	0.11	782.14
gamma-Chlordane	5/13	0.002	0.008	0.0002	0.0004	0.002	0.0001	0.002	0.003	0.18
Total Dioxin/Furans (2,3,7,8- TCDD TEQs) ^b	13/13	0.0000007	0.00009	NA	NA	0.00002	0.000009	0.00003	0.00004	0.0000004

^aMedian, mean, standard deviation, and confidence limits are estimated using detected results and/or proxie concentrations equal to one-half of the detection level (dl/2) for those samples at or below the detection level.

^b The value listed is from the April 1999 USEPA Region 3 Risk Based Concentrations (RBCs).

^c Dioxin TEQ are calculated values, not measured.

NA = Not Applicable

one sample at the Residential Towers at a concentration which exceeded the RBC. Aluminum, arsenic, iron, manganese, thallium and vanadium were detected at all three sites at concentrations exceeding the RBCs. The concentrations of metals in the subsurface soils were not significantly different than those in background Reference Area 1.

Soil Trend Analysis

For the trend analysis, 33 surface and 11 subsurface samples were collected where maximum deposition was expected and/or minimal soil disturbance had occurred. Samples were collected from areas of potential sediment accumulation, stressed vegetation, and those lacking evidence of erosion or ground cover. The subsurface data was collected to determine if there were significant differences in comparison to the surface soils for evaluation of surface accumulation of airborne contaminants. Figure 2-1 shows sampling locations for risk assessment and for trend analysis.

Metals

The trend analysis showed that several metals exhibited very similar patterns across the base. Concentrations of antimony, barium, cadmium, copper, lead, mercury, silver and zinc all had maximum surface and subsurface concentrations at a site immediately north of the SIC (sample location 4, Figure 2-1). Concentrations at this one site had a tremendous impact on the interpolated soil distributions for these metals. Since these high concentrations were found in samples near the SIC, it is likely that the SIC was the source of the contamination.

Except for zinc, all of these metals were present in subsurface soils at concentrations exceeding their RBCs. Generally, higher concentrations of these elements were found in surface soils. Copper exhibited a similar distribution as the other seven but differed in that higher concentrations were found in subsurface soil rather than surface soil.

Aluminum, manganese, and vanadium exhibited no clear pattern in either the surface or subsurface soils. It appears that soil concentrations of manganese and vanadium, across the entire base, exceeded the RBC in some areas. Aluminum soil concentrations were above its respective RBC in specific locations across the base. The uniform distribution of concentrations across the base suggested that the presence of these constituents did not result from SIC operations.

Arsenic and chromium were found at elevated levels near the SIC, but were also found at elevated levels in other locations on the base. For both metals, it appeared that the SIC could have affected surface and subsurface soil. However, other sources of these metals appeared to be present in other locations on the base, especially for arsenic. All of the interpolated concentrations of arsenic exceeded the RBC and most of the values for chromium exceeded the RBC.

Thallium concentrations were elevated in several surface soil samples collected north of the SIC; however, concentrations at some of the background locations also were elevated. It was unclear how this pattern related to SIC operations. All concentrations were below the RBC, although a majority of the observed and interpolated concentrations exceeded the RBC. No clear pattern was evident for subsurface soil. Thallium concentrations were similar to those found in the surface.

Pesticides/PCBs

Organochlorine pesticides and PCBs were not detected at or above RBCs in either surface or subsurface soils.

Semivolatiles

Benzo(a)anthracene, benzo(a)pyrene, benzo(b)fluoranthene, dibenz(a,h)anthracene and indeno(1,2,3-cd)pyrene all exhibited similar distribution patterns. For each of these compounds, the maximum concentration was found in the surface soil sample south of the base at the Criteria Site, sample location #1 (Figure 2-1). However, all of the subsurface soil maximum results were found in sample location #27 (Figure 2-1). Only benzo(a)pyrene and dibenz(a,h)anthracene exceeded the RBCs in the subsurface soil. The concentrations in the surface soil were higher than in the subsurface; and the maximum result for all five SVOCs exceeded the RBCs. Based on the lack of spatial trends, and the generally isolated occurrence of the SVOCs, their presence in soils did not appear to be associated with the SIC.

Dioxins

TEQ values exceeded the RBCs for 2,3,7,8-TCDD in surface and subsurface soils. There was a definite trend of high concentrations near the SIC in both surface and subsurface soil, primarily focused around the sample location immediately north (trend analysis sample #4 in

Figure 2-1) in the vicinity of the incinerator. However, sample location #6 (Figure 2-1), which was to the east and on the Golf Course, also exhibited a relatively high TEQ concentration.

The soil trend analysis indicated that a spatial correlation between concentration and distance from the SIC was evident for Total 2,3,7,8-TCDD TEQs. This is displayed in Figure 2-4. Consequently, concentrations of Total 2,3,7,8-TCDD TEQs in soil samples on the base typically increased as the distance from the SIC decreased. The soil trend analysis also indicated that the concentrations of Total 2,3,7,8-TCDD TEQs exceeded RBCs throughout the base for surface (0 to 3- inch) soil samples, and approximately one-half of the base for subsurface (3 to 12-inch) soil samples.

As shown in Figure 2-4, a definite footprint of dioxin deposition associated with air emissions from the SIC was evident from high concentrations near the SIC, decreasing with increasing distance from the SIC. The analysis of this dioxin footprint conducted by Mr. Matthew Lorber, USEPA National Center for Environmental Assessment, Washington D.C., (EPA 1998d) is included in Appendix D.

Maximum concentrations for dioxins were always found in the samples taken near the fenceline north of the incinerator and at the Golf Course between the third and fourth holes, east of the incinerator. Elevated dioxin concentrations found at the Golf Course (600 ppt) between the third and fourth holes (Figure 2-1, sample location #6,), east of the incinerator, appeared to be an anomaly, perhaps associated with blowing ash. The congener profile associated with this 600 ppt 2,3,7,8 TCDD concentration indicated a congener profile, which was different from that of the next five highest soil sample concentrations, with octachlorodibenzo-*p*-dioxin (OCDD) dominating the profile.

Summary

In summary, the SIC appeared to have affected the distribution of analytes in surface and subsurface soil at NAF Atsugi. A clear trend, especially with dioxins, was evident. Concentrations of several metals were also clearly elevated near the SIC. Although SVOC concentrations were elevated at location #1, it is possible that the source of these compounds was not the SIC. Details on the soil trend analysis and sampling results for the areas of concern can be found in the Radian soil sampling report (Radian 1998c).

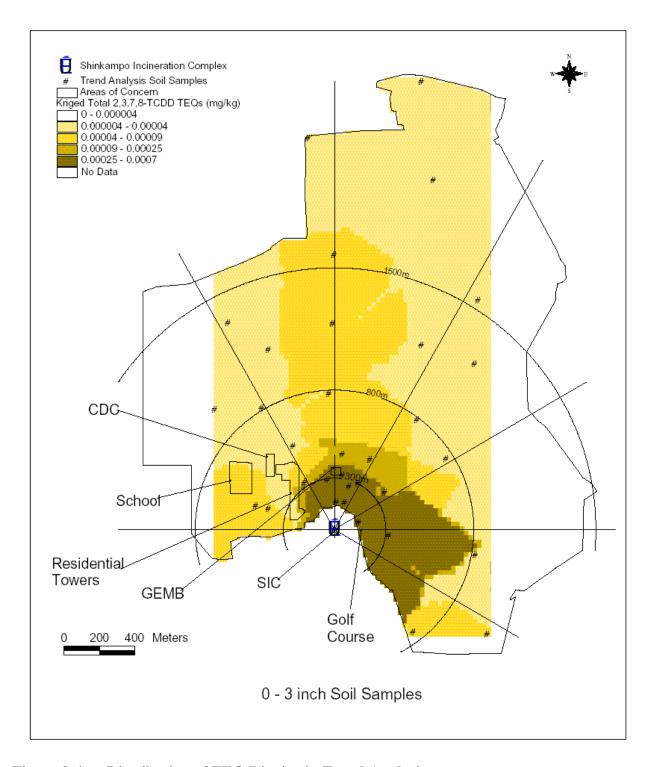


Figure 2-4 — Distribution of TEQ Dioxins in Trend Analysis

2.8 Fourier Transform Infrared (FTIR) Monitoring

A mobile, dual-cell extractive infrared Fourier Transform Infrared (FTIR) system was used to add flexibility to the sampling strategy.

The two primary objectives of the FTIR monitoring were:

- 1. To determine what concentrations of VOCs and acid gases NAF personnel were exposed to on a continuous basis and to determine if there was a correlation between outdoor and indoor air concentrations.
- 2. To determine the contribution of the SIC regarding VOCs and acid gases to the air quality at the base through confirmation of pollutants both upwind and downwind of the SIC, and if variations existed in VOC and acid gas concentrations across the base and at different times.

The FTIR was chosen for these objectives because it was capable of providing continuous measurements for any chemical in gas form that absorbs infrared energy, including VOCs and acid gases, while monitoring meteorological conditions.

The FTIR system was able to perform real-time analysis of up to 50 chemicals simultaneously in each of its two cells. The continuous measurement of acid gases, in particular, could assist in pinpointing the SIC as a source of emissions, because the combustion of chlorinated solvents and plastics that occurred at the SIC would produce emissions of hydrochloric acid. The mobility and the continuous monitoring were the two features that made the FTIR system attractive to meet these objectives.

One of the two cells of the dual-cell extractive FTIR was "scrubbed" to remove water vapor and carbon dioxide, producing lower detection limits for chemicals otherwise obscured by water vapor and carbon dioxide (e.g., benzene and toluene). The contents of the two cells were analyzed alternately using a 5-minute sampling period, with four or five samples from each cell being analyzed each hour. The data were converted to hourly averages for reporting purposes. The meteorological data (wind direction, wind speed, barometric pressure, and ambient air temperature) were integrated with the FTIR data in real time, providing a means for identifying the approximate direction of the source of the chemicals being measured.

The analytical list developed for the Atsugi FTIR system included chemicals that had been identified in the SIC plume emissions using the open path FTIR. This open path FTIR had been previously set up under another project conducted by the NAF Atsugi Public Works Department, Environmental Division to evaluate SIC plume emissions for a legal case being developed against the SIC. In addition, some chemicals that had been identified in a previous screening risk assessment for NAF Atsugi as chemicals of potential concern (with respect to human health) were also included. The entire list of chemicals monitored by the FTIR is included in Table 2-10.

The mobile FTIR system had inherent strengths and limitations. Its strong points included its mobility; its ability to conduct continuous, real-time analyses; and its ability to conduct simultaneous analyses for multiple chemicals. The system's primary limitation was that its minimum detection limit (the lowest concentration of a chemical the system was capable of measuring under ideal conditions) was higher for most chemicals than the minimum detection limit possible using other more conventional analytical methods. The FTIR system can typically measure the presence of a chemical at concentrations below 100 parts-per-billion, and for many chemicals at concentrations below 10 parts-per-billion. Other methods can detect the presence of chemicals in the parts-per-trillion range. A second limitation was that the FTIR system could only measure the presence of chemicals in the form of a gas. If a chemical, such as hydrochloric acid, was present as a gas and also present in the form of solid particles or aerosols, the FTIR would only measure the portion present in the form of a gas.

Since, in previous screening risk assessments (NEHC 1995), maximum concentrations of VOCs above the FTIR detection limit were observed for benzene (84 μ g/m³), ethylbenzene (100 μ g/m³), toluene (420 μ g/m³), o-xylene (42 μ g/m³) and p-xylene (130 μ g/m³) collected in 30-second Summa canisters samples, it was anticipated that the FTIR would have been an appropriate real time indicator of air quality. The intended primary location for the mobile FTIR system was in the parking lot adjacent to the Elementary School, approximately 500 meters northwest of the SIC. This location was chosen to provide real time monitoring of ambient air where sensitive receptors such as children spent a great deal of time. However, during this monitoring effort, SIC operating conditions may have changed so that gaseous concentrations of the target pollutants were not present above the FTIR system's minimum detection limits with a

Table 2-10 — Air Pollutants Included in Mobile FTIR Analytical Method

Pollutant	Measurement Source	Minimum Detection Limit (μg/m³)
Acetaldehyde	Both Cells	36
Acrolein	Both Cells	5
Acrylonitrile	Both Cells	65
Ammonia	Unscrubbed Cell	2
1,3-Butadiene	Both Cells	13
Carbon monoxide	Unscrubbed Cell	8
Carbon tetrachloride	Unscrubbed Cell	13
Crotonaldehyde	Both Cells	6
Dichloromethane	Unscrubbed Cell	73
Formaldehyde	Unscrubbed Cell	3.5
Freon-22	Unscrubbed Cell	14
Hydrogen chloride	Unscrubbed Cell	7
Hydrogen cyanide	Unscrubbed Cell	9
Hydrogen sulfide	Unscrubbed Cell	1,400
Methane	Unscrubbed Cell	4
Methanol	Unscrubbed Cell	4
Nitric acid	Unscrubbed Cell	26
Nitric oxide	Unscrubbed Cell	3.5
Phenol	Unscrubbed Cell	96
Trichloroethylene	Unscrubbed Cell	54
Benzene	Scrubbed Cell	80
Ethylbenzene	Scrubbed Cell	36
Meta-xylene	Scrubbed Cell	23
Ortho-xylene	Scrubbed Cell	36
Para-xylene	Scrubbed Cell	10
Toluene	Scrubbed Cell	190

frequency that would allow a statistical analysis addressing either question above. Therefore, the system was later moved to provide continuous monitoring at other locations across the base, such as the 19th Hole Golf Course Parking Lot, and the GEMB. Table 2-11 shows the prioritized locations for the mobile FTIR system, along with the rationale for selecting the locations. The site locations are presented in Figure 2-5.

By mid-December 1998, the FTIR system had been positioned at all of the planned locations. At that time, the decision was made to move the system to the location adjacent to the GEMB as the system's final, permanent location. This decision was based on several factors.

First, the FTIR system had not measured chemicals at concentrations above the FTIR system's detection limits at other locations at a frequency that allowed detailed analysis. It was

Location	Priority	Rationale
Elementary School Parking Lot (primary location)	1	Centrally located among many sensitive receptors; In an area often affected by the SIC; Within 300 meters of indoor measurement locations.
Ground Electronics Maintenance Building (GEMB) Parking Lot	2	Near the fence line between NAF Atsugi and the SIC; Frequently in the SIC plume.
19th Hole Golf Course Parking Lot	3	Collect continuous data for determining variations across the base.
Cryogenics (liquid oxygen) Building	4	Collect continuous data for determining variations across the base.
Recreational Area (softball field)	5	Collect continuous data where strenuous outdoor activity occurred.
Upwind Site adjacent to Criteria Site	6	Collect continuous upwind data;. Co-located with an ambient monitoring site.

Table 2-11 — Mobile FTIR/Meteorological Station Site Locations and Rationale

believed that by positioning the system permanently at the location nearest the SIC and downwind from the SIC during periods when the wind was blowing from the SIC toward the base, the opportunity for the FTIR to detect chemicals being emitted by the SIC might be improved.

Second, at this site, the FTIR system would be co-located with a conventional monitoring location and adjacent to a building at which indoor air quality measurements were taken. The

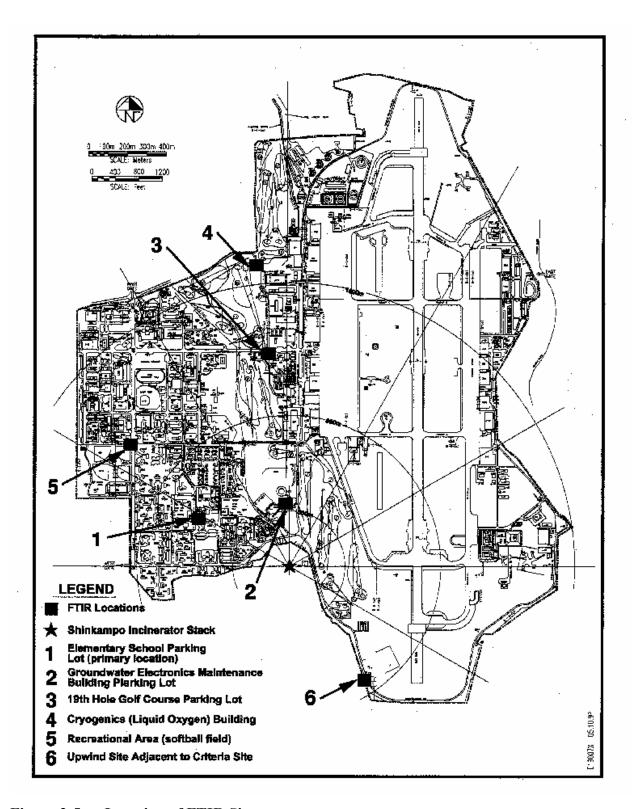


Figure 2-5 — Location of FTIR Sites

FTIR system was moved to the GEMB site on 22 December 1998, and remained there through 3 May 1999, when data collection by the FTIR system ended.

Despite major problems causing significant outages, of the FTIR system, such as generator and auto fill problems, liquid nitrogen refill problems and power supply failures, the overall data capture rate during the project was approximately 85%, including the initial start-up problems. The FTIR data capture rate for the last seven months of the project was above 90%.

In summary, gaseous concentrations of the target pollutants were not present above the FTIR system's minimum detection limits with a frequency that would allow a statistical analysis addressing the objectives. However, the following general statements were made: The FTIR was intended to be used as a real time indicator of air quality. Although the detection limits for the FTIR were much higher than those for ambient air monitoring, the continuous FTIR monitoring did show spike concentrations, in general, that were below the RBCs. Formaldehyde was the notable exception; since the FTIR's minimum detection limit for formaldehyde (3.5 $\mu g/m^3$) is well above the RBC for formaldehyde (0.14 $\mu g/m^3$). All concentration spikes for formaldehyde measured by the FTIR were above the RBC.

- Nine pollutants out of the list of 21 target pollutants included in the FTIR analytical
 method were detected during the project. These included carbon monoxide, methane,
 nitric oxide, methanol, formaldehyde, ammonia, freon-22, toluene, and
 dichloromethane.
- The FTIR system did not detect hydrochloric acid, an incinerator tracer compound, during the project. Hydrochloric acid was measured by other methods, when colocated with the FTIR system, in concentrations above the FTIR system's minimum detection limit (7µg/m³). We concluded from these results that substantial percentages of hydrochloric acid, as measured by the ambient air program, were present in the form of particles and aerosols. Therefore, the percentage present in the form of a gas was always below the FTIR system's minimum detection limit.

Details on the FTIR sampling can be found in the air monitoring summary report developed by Radian (Radian 2000a).

2.9 Audits

Throughout the 14-month environmental monitoring period, three different contractors performed three air quality performance and systems audits that included sampling and

meteorological equipment. The contractors included: Roy F. Weston, Inc., IT Corporation and UAI Environmental, Inc. The purpose was to conduct quality assurance on several components of the ambient air-monitoring program. The quality assurance audits included a qualitative and quantitative assessment of the accuracy of individual sampling devices as well as entire measurement systems. The audits were conducted following the guidance in "USEPA Quality Assurance Handbook – Volume II: Ambient Air Specific Methods" and "USEPA Quality Assurance Handbook – Volume IV: Meteorological Measurements" (EPA 1998). The audit conclusions indicated that the ambient air monitoring system was well designed, being operated in a manner consistent with the USEPA recommendations, and was well maintained. Complete information on the three air quality performance and systems audits can be found in the following Weston, IT and UAI reports (Weston 1999, IT 1999, UAI 1999).

USEPA National Exposure Research Laboratory, Research Triangle Park conducted audits of the three analytical laboratories performing chemical analyses. The laboratories audited were Radian International, Alta Analytical and Czartech Analytical (EPA 1999a, 1999b). The results of the audits indicated that the laboratories were well maintained, had excellent instrumentation, followed proper analytical methods and QA/QC procedures and produced good quality data.

ManTech Environmental Technology Incorporated conducted a quality assurance review of the FTIR spectra (ManTech 1999). The review flagged and recommended eighteen days of data be removed for cell 1 and one day for cell 2. With these exceptions, the concentration data for the various gases indicated that the instrument was functioning properly for the entire time and there was no technical reason to flag any other data.

2.10 Departures from Quality Assurance Project Plan (QAPP)

NEHC, Radian, BUMED, Pioneer and USEPA participated in quarterly meetings to determine if any changes were needed in the sampling protocol. Changes were made at various times, during the data evaluation, to better focus the investigation.

One of the greatest challenges in this project was to meet the second objective, i.e., to determine the SIC contribution to the health risk. The method used to identify the chemicals in air emitted from the SIC was to correlate wind direction, specifically the percentage of time an individual monitoring site was downwind of the SIC, to the chemical concentrations observed in

ambient air at the site for the various monitoring periods. The hypothesis was that, for chemicals that were emitted from the SIC, the chemical concentration (and also risk) increased as the percent of time the wind blew emissions from the SIC onto the base increased. In the case of variable wind speed and direction, from one week to the next, if the emissions were constant but the wind speed and direction were not the same, the correlation with percent downwind would be different. The confidence in the correlation of wind direction versus concentration was related to the number of observations that were used to calculate the correlation coefficient and the wind directions that were observed.

An analysis of the wind patterns observed during the 1998 sampling program indicated the winds had been atypical for the period. Specifically, when compared to historical meteorological data, there were fewer periods of southerly winds, which carried emissions from the SIC onto the base. May and June historically had significant periods of southerly winds, and extending sampling could result in ambient air concentrations that were more representative of historical conditions for evaluation in the risk assessment if the winds during the extension period were consistent with the historical wind patterns. In other words, during the 1998 ambient air sampling study, there were few periods of southerly winds, even fewer than that observed by historical wind roses. As a result, there were relatively few data points to correlate concentrations/percent downwind in an effort to assess SIC contribution. Therefore, sampling was extended for an additional 2 months and samples were collected on days predicted to be downwind days (i.e., toward NAF Atsugi from the SIC) to complete the correlation plots. The extended sampling included 6 additional sampling days when the wind was blowing toward the base past the SIC to complete the correlation plots for a better representation of historical conditions. Since this could result in an underestimation of long-term exposure conditions and consequently risk, the additional number of sampling days needed to provide additional information for the correlation analysis plots was determined by statistical analysis so that no bias would result by overweighing particular wind directions and overestimating the contribution from the SIC.

The health risks calculated based on the 1998 sampling program data could potentially have been higher than calculated if the winds during the sampling period had been consistent with historical data. A decision to not extend the sampling period could have resulted in too few

periods of southerly winds sampled in comparison to historical wind conditions and an unrepresentative data set for long-term exposure.

To maximize the use of financial resources, a decision was made not to include the Golf Course in the extended sampling. Resources were concentrated on obtaining additional information to refine the correlation plots on the AOCs where sensitive receptors (i.e. children) would spend most of their time (i.e, the Elementary School, the Residential Towers), and the most impacted area (GEMB). Sampling for pesticides was not done based on the justification that this class of chemicals did not show a reason for concern during the prior 12 months of sampling.

The QAPP was closely followed during the program for the vast majority of the activities conducted during the 14-month period. There were, however, several scope-of-work modifications during the program that had not been anticipated during the development of the original QAPP. These modifications included:

- The addition of an alternative PCB method;
- The addition of an alternate mercury monitoring method;
- Modifications of the USEPA Method TO-11A analysis, for aldehydes and ketones due to interference with the crotonaldehyde peak; and
- Modification of site locations of the FTIR unit.

As data were collected, there were questions concerning both the presence of smaller molecular weight PCBs and mercury species in addition to elemental mercury. Questions were raised regarding smaller molecular weight coplanar PCBs, because pesticide/PCB analysis showed a large number of co-eluting peaks. Therefore, a short-term monitoring program was initiated to determine the specific PCB congeners that were present in the ambient air. This monitoring used a very specific sampling and analytical method, known as high resolution GC/MS to determine over 50 specific PCB congeners. Since mercury was detected in the ambient air samples at concentrations lower than expected, an additional sampling and analytical method that could detect mercuric chloride in addition to elemental mercury was employed for a short-term study, to assess the potential for underestimating the ambient mercury concentrations.

Results of the additional monitoring efforts are discussed in the Radian air monitoring summary report (Radian 2000a) and are presented in Appendix N of the Radian report (Radian 2000c).

USEPA Method TO-11 guidance was used during the course of the monitoring program to collect and analyze samples for aldehydes and ketones. This was the standard method to collect and analyze aldehydes and ketones in ambient air. Among the target chemicals quantitated by this method, acrolein was known to exhibit low recovery on the dinitro-phenyl-hydrazine (DNPH)-coated collection media due to the breakdown of the primary DNPH derivative. This had been demonstrated by Radian in several previous monitoring programs. During the NAF Atsugi study, it appeared that crotonaldehyde was also experiencing a similar decomposition. This was not surprising because the two compounds are quite similar chemically, differing only by a carbon group. Crotonaldehyde decomposition had not been noted during previous Radian studies in the U.S., because crotonaldehyde was not detected in sufficient concentrations to warrant further evaluation.

FTIR sampling was conducted at all on-site locations described in the QAPP. The original QAPP called for the FTIR unit to be left at the Elementary School. However, the FTIR was moved to the GEMB on 22 December 1998 and remained there until it was decommissioned.

Section 3 — Human Health Risk Assessment Results

This section presents the results of the comprehensive HRA, including a description of the cancer and non-cancer risks.

3.1 Overview of Risk Assessment Methodology

Risk assessment is an established approach to evaluate the potential for adverse health effects from exposures to toxic constituents. Risk assessment is a management-decision tool and does not provide absolute statements about possible human health effects. Risk assessments typically focus on constituents and exposure pathways directly related to the site of concern. These assessments do not address risks from other sources of exposure (e.g., dietary exposures) or risks from other constituents that are not associated with the site under evaluation.

The general approach for preparing this HRA is provided by the USEPA Risk Assessment Guidance for Superfund (EPA 1989).

The human health risk assessment process is comprised of the following five steps:

- 1. Data Evaluation, Reduction, and Screening. This step identifies potential constituents of concern from analytical data obtained from the field-sampling program. Constituents detected in at least one sample during the field investigation are identified and screened against risk-based concentrations to obtain a final list of constituents to be evaluated in the risk assessment.
- **2. Exposure Assessment**. This step identifies potentially exposed populations (i.e., receptors), exposure scenarios, exposure pathways, and exposure factors. The algorithms used to calculate intake are also identified.
- **3. Toxicity Assessment**. This step identifies toxicity values for the COCs identified in step 1. Toxicity values include non-carcinogenic reference doses and carcinogenic slope factors.
- **4. Risk Characterization**. This step presents the human health risks associated with exposure to the COCs that were calculated using the information developed in steps 1 3.
- **5. Uncertainty Analysis**. This step identifies key uncertainties inherent in the evaluation that should be considered when assessing the risks.

3.2 Data Evaluation, Reduction, and Screening

Table 3-1 summarizes the media sampled at NAF Atsugi by areas of concern (AOC).

AOC	Soil Samples (0-3")	Ambient Air Samples	Indoor Air Samples ¹	Indoor Dust Samples ²
Child Development Center	•		•	•
Elementary School	•	•	•	•
Ground Electronics Maintenance Building		•	•	•
Apartment in Residential Towers (3101/3102)	•	•	•	•
Corridor in Residential Towers (3101/3102)		•	•	•
Residential Towers (3043)			•	•
Residential Townhouse (3025)			•	•
Golf Course		•		
Criteria Site ³		•		

Table 3-1 — Summary of Media Sampled at NAF Atsugi, Japan

The analytical data for soil, ambient air, indoor air, and indoor dust were analyzed using SiteSTATTM software, and a preliminary list of COCs was identified for each medium. Details on how this list was developed can be found in Section 2 of the Pioneer HRA report (Pioneer 2000). Site-specific background concentrations for ambient air, indoor air, and indoor dust were not available, because it was not possible to identify a location on the base that was not impacted by emissions from the SIC. Therefore, site-specific background screening of these media was not performed. However, for soil samples a suitable site-specific background site was identified, as described in the Phase II Soil Sampling Report (Radian, 1999f). The maximum detected concentration for these data was compared to the maximum detected in soil for each of the AOCs. If the maximum detected concentration in soil exceeded the background concentration, then the analyte was retained for further evaluation in the risk assessment. If the maximum detected concentration in soil was less than or equal to the background concentration, then the

^{●=} Media sampled at this location.

^{-- =} Media not sampled at this location.

¹The results of the indoor air samples were qualitatively evaluated in this assessment.

²Indoor dust samples were only analyzed for Dioxins/Furans.

³There were no exposed populations at this location; therefore, this site was not quantitatively evaluated in the risk assessment.

analyte was eliminated from further consideration in the risk assessment. The maximum detected concentrations of the COCs in each area were compared to $1/10^{th}$ the appropriate EPA Region 3 RBCs (i.e., soil and indoor dust concentrations were compared to soil RBCs and ambient air and indoor air concentrations were compared to ambient air RBCs). These values corresponded to a cancer risk of $1x10^{-7}$ (1 in 10 million) and a non-cancer hazard index of 0.1, calculated for a residential exposure scenario. If the maximum detected concentration for a constituent was greater than the RBC, then the constituent was retained for further consideration in the risk assessment. In addition, constituents lacking an RBC were retained for evaluation in the risk assessment. The exposure point concentrations calculated for this assessment are presented in Appendix B of the Pioneer HRA report (Pioneer 2000).

3.3 Exposure Assessment

An exposure assessment determines the specific chemicals and amounts of each to which a population might be exposed, the routes of exposure and the magnitude, duration and timing of the exposure. It involves a determination of the time patterns that are involved in certain activities. This was accomplished through a multi-pathway analysis.

Multi-Pathway Analysis

Exposure assessment of airborne emissions generally not only involves an analysis of the inhalation pathway, but also non-inhalation pathways of indirect exposure. These indirect exposures occur as a result of deposition of airborne material onto surface water, soils, and subsequent contact and consumption of food products (i.e., ingestion of meat, fish, eggs, fruits, vegetables, dairy products, etc.) affected by the emissions. A multi-pathway analysis was conducted to determine the pathways of concern. An exposure pathway is the process by which an individual is exposed to contaminants that originate from some source. An exposure pathway consists of five elements:

- 1. Source of contamination
- 2. Environmental media and transport mechanism
- 3. Point of exposure
- 4. Route of exposure
- 5. Receptor population

The multi-pathway analysis assessed the various pathways in light of the above elements to determine whether or not a pathway was complete at NAF Atsugi and subsequently addressed in the HRA. Emphasis was specifically placed on pathways that were associated with emissions from the SIC. For the purposes of this analysis, the following categories were used in making the determination:

- Complete Pathway Exists if all five elements link the contaminant source to a receptor;
- Potentially Complete Pathway Exists when one or more elements appear to be missing
 as insufficient information is available to make the determination of complete or
 incomplete;
- Incomplete Pathway Exists if one or more of the elements are known not to be present.

Figure 3-1 presents the results of the multi-exposure pathway analysis conducted by Radian (Radian 1998b). Individuals may be exposed to emissions from the SIC both directly and indirectly.

Air and Soil

The air and soil pathways were considered direct pathways due to the proximity of the areas of concern to the SIC, its visible emissions and consequent soil depositions, and since the constituents of concern (COCs) in soil and dust were deposited at NAF Atsugi during downwind events. Consequently, inhalation of particulates and vapors, and incidental soil ingestion and dermal contact were evaluated. The findings and conclusions of the pathway analyses related to groundwater, surface water and consumption of food were addressed as follows:

Grou<u>ndwater</u>

The multi-pathway analysis determined that the groundwater pathway was incomplete. Although groundwater was the source of potable water on base, it was not considered a complete pathway of concern, because recent drinking water sampling conducted to ensure that it meets U. S. drinking water standards indicated that the groundwater had not been impacted by incinerator operations (Dames & Moore 1999).

Surface Water

Surface water was also not considered a complete pathway since the Tade River, which runs through the base, was known to be polluted from the surrounding industries, and therefore,

was not used for swimming or other recreational uses by the NAF Atsugi community (Radian1998b). No one was observed near the river when the multi-pathway analysis was conducted. However, personal communication with Michelle Norman, from the NAF Atsugi Environmental Office, indicated that children might play near the river. To ensure that this pathway continues to be incomplete, the Navy should institute administrative controls (e.g., posting signs, educating parents).

Consumption of Food

Indirect exposure to COCs through consumption of fruits, vegetables, meat and milk were not considered pathways of concern due to the fact that most of the food purchased on base and consumed by base personnel and their families originated in the U.S. The vast majority of food was purchased at the commissary because it was much less expensive than at the local markets. A few fruits and vegetables labeled as "local" at the commissary were grown in larger farms located in northern and southern Japan away from the local area and therefore were not impacted by the SIC. Eggs were received from three farms from Yokohama, Sasebo, and Iwakuni. Therefore, indirect exposures to locally grown food products (e.g., meat, fish, fruits, vegetable, etc.), which are typically significant when evaluating bioaccumulative contaminants such as dioxins/furans, were not evaluated in this risk assessment.

Exposure Scenarios

Since the air and soil pathways were considered direct pathways due to the proximity of the areas of concern to the SIC, inhalation of particulates and vapors, and incidental soil ingestion and dermal contact were evaluated.

This risk assessment focused on exposure scenarios applicable to each of the AOCs likely to be frequented by sensitive receptors (i.e., children), as described in the Exposure Assessment section of the HRA, developed by Pioneer (Pioneer 2000). These are scenarios represented by exposed populations who work, spend time or live at the Child Development Center, the Elementary School, the Residential Towers, the Ground Electronics Maintenance Building or the Golf Course, respectively. These locations were AOCs because they were in close proximity to

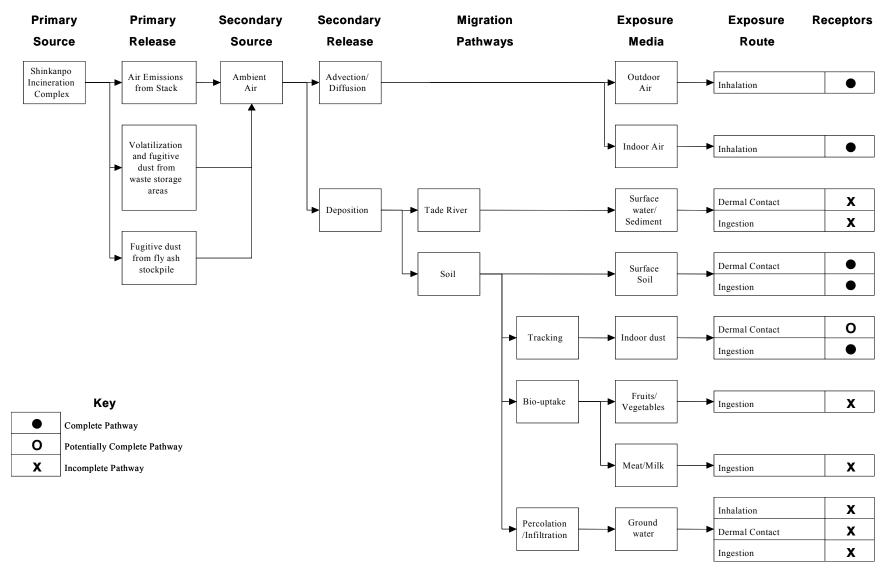


Figure 3-1 Conceptual Site Model, NAF Atsugi, Japan (Multi-Exposure Pathway Analysis)

the SIC, where children and adults spent most of their time. In particular, the newest buildings are the two towers closest to the incinerator, Buildings 3101 and 3102, which opened for occupancy in May 1996 and May 1997, respectively. Both towers were sited and constructed before the 1995 and the 1997 screening HRA reports documented the level of health risks. Figure 2-1 in Section 2 presents the locations of the AOCs and the exposure scenario for each location for which a quantitative risk assessment was conducted is described below.

Child Development Center, Building 2910 (Child and Adult Worker)

The Child Development Center was located approximately 450 meters northwest of the SIC. Approximately 400 infants to pre-school age children were at this facility for a maximum of 10 hours per day. Infants to pre-school aged children were at this facility under the direction of adult workers. Children were typically outside two times a day for 45 minutes each excursion. Outdoor activities were restricted during bad weather or when emissions from the incinerator were blowing towards the base. Central air conditioning was used for temperature control in the buildings, except for April through May when the weather conditions were cool enough to allow the windows to be opened. Students and adult workers were exposed to indoor air and dust while indoors. A new facility was constructed that was several meters closer to the SIC.

Students and workers were exposed to the emissions from the SIC, especially when outdoors or the windows were opened. Indoor air and dust (once) were monitored on a quarterly basis. Soil samples were also collected in the playground areas. Ambient air was monitored on a routine basis for a 14-month period at the nearby Elementary School. The data collected at this site was used to evaluate human health risks for inhalation of indoor air, dermal contact with soil, incidental ingestion of dust indoors and inhalation of ambient air using the data from the Elementary School as representative for this site. The children were not allowed to play outdoors when the wind was blowing from the direction of the SIC.

Shirley Lanham Elementary School, Building 993 (Adolescent and Adult Worker)

The Shirley Lanham Elementary School was located approximately 500 meters northwest of the SIC. Children attended Kindergarten through sixth grade and were ages 5 through 12. The Elementary School held class for 180 days per year for approximately 800 Kindergarten through 6th grade students. Teachers and adult workers were also present. Students and workers were exposed to the emissions from the SIC, especially when outdoors or the windows were

opened. The school year ran from the end of August to the beginning of June. Children were typically outside before and after school, during recess, and lunch. Outdoor activities were restricted during bad weather or when emissions from the incinerator were blowing towards the base, however children were still outside before and after school. Air conditioning was used to cool the rooms from April to the end of the school year. Steam heat was used to warm the rooms during the colder months; however, at times the rooms got too warm and the windows were opened to cool them. During the springtime, the windows are opened to cool the rooms. Each room was equipped with air filters. Students and adult workers were exposed to indoor air and dust while indoors.

Indoor air was monitored once each quarter. Indoor dust was monitored once at this site, prior to the air monitoring being conducted for the first quarter. Ambient air was monitored on a routine basis for a 14-month period. This data was used to evaluate human health risks for inhalation of indoor and ambient air, and incidental ingestion of dust indoors. Although safety tiles on the playground areas did not allow contact with the soil, dermal contact or incidental ingestion of contaminants may have occurred through touching playground equipment where deposition of contaminants may have occurred. For this reason, hands were washed as the students returned to their classrooms to limit dermal contact and incidental ingestion of contaminants.

Residential Towers, Buildings 3101 and 3102 (Child and Adult Residents)

Building 3102 Residential Tower was located approximately 300 meters northwest of the SIC. American military and government civilian employees lived in apartments in this building. Prevailing winds frequently blew from the SIC to this Residential Tower. Child and adult residents were exposed to these emissions and any deposition of constituents in the surrounding soils. Constituents from outside may have entered the towers as dust when doors were opened or may have been tracked indoors on shoes.

Ambient air was monitored on a routine basis for a 14-month period; indoor air was monitored on a quarterly basis; and soil samples were collected in the surrounding area. The data collected at this site was used to evaluate human health risks for inhalation of outdoor and indoor air, dermal contact with and incidental ingestion of soil, and incidental ingestion of dust tracked indoors.

Ground Electronics Maintenance, Building 1061 (Adult Worker)

The Ground Electronics Maintenance Building (GEMB) was located directly downwind (north) of the SIC, approximately 300 meters away. Air dispersion modeling indicated that this site was the highest impacted area from incinerator emissions. Prevailing wind direction was often from the SIC to the GEMB. Incinerator emissions had been observed to be at ground level at this site.

Indoor air was collected on a routine basis at this site during the sampling program. Ambient air was monitored on a routine basis for a 14-month period. Indoor dust was sampled once at this site. This data was used to evaluate human health risks to workers for inhalation of both indoor and ambient air, dermal contact with soil and incidental ingestion of indoor dust. These workers were typically indoors, but did have equipment located outside the building that had to be attended, on a routine basis.

Golf Course (Recreational User)

The first nine holes of the Golf Course were located east and northeast of the SIC. Hole 9 was the closest to the SIC and frequently received ground level emissions from the incinerator stacks. The manager and an assistant were the only American workers. All maintenance workers were Japanese civilians and were not being evaluated for risks. Both American and Japanese military and civilian government workers used the Golf Course. Ambient air was routinely monitored at this site for a 14-month period. Soil samples were also collected throughout the Golf Course area. The data collected at this site was used to evaluate human health risks for the recreational users. Inhalation was the most predominant exposure pathway, but incidental soil ingestion may have also occurred.

In addition to these AOCs, there were other AOCs for which sampling data was collected, but were not quantitatively evaluated in the HRA. These areas were:

Residential Townhouse, Building 3025

Building 3025 was located approximately 800 meters northwest of the SIC. American military and government civilian employees lived in these townhouses. Child and adult residents were exposed to indoor air and dust while at home. Indoor air and dust samples only were collected at this site and used for comparison with other indoor sites with a different heating and air conditioning system.

Old Residential Towers, Building 3043

Building 3043 was located approximately 500 meters northwest of the SIC. American military and government civilian employees lived in apartments in this building. Prevailing winds frequently blew from the SIC to this building. Child and adult residents were exposed to indoor air and indoor dust while at home. Indoor air and dust samples collected at this site were only used for comparison with other indoor sites to determine if there were any differences in concentrations of chemicals indoors, resulting from differences in heating and air conditioning systems (active and passive ventilation systems).

Criteria Site

The criteria site was located southeast of the SIC. Workers, residents, and recreational users were not located at this site. Therefore, it was not evaluated in the risk assessment; however, the sampling results are discussed in the Radian air monitoring summary report (Radian, 2000). Since this site was located in a predominantly upwind location based on historical trends, soil samples were collected at this site for the trend analysis only and ambient air samples were collected to provide data on reference or background concentrations.

Quantifying Exposures

Quantifying the magnitude, frequency, and duration of exposure for the selected populations and exposure pathways was the last step in the exposure assessment. The basic equation used to calculate human intake of a chemical was:

$$DI = C*HIF*M$$
, where,

Parameter	Definition
DI	Daily intake (mg of COC per kg of body weight per day) [mg/kg-day])
С	Concentration of the COC (mg/kg or mg/m ³)
HIF	Human intake factor [(day) ⁻¹]
MF	Exposure Pathway and Constituent Specific Modifying Factors (e.g.,
	percutaneous absorption rate) (variable units)

Table 3-2 — Exposure Parameters^(a) - Incidental Soil Ingestion

⁽a) Exposure factors without footnotes are U.S. Environmental Protection Agency Standard Defaults (USEPA, 1991b).

⁽b) For the 30-year residential exposure scenario (i.e., 6 years as a child and 24 years as an adult) the integrated intake equation was used to calculate intake. For all other exposure scenarios the standard intake equation was used to calculate intake. Parameters with an "a" subscript are for adults and parameters with a "c" subscript are for children.

⁽c) The Average and RME concentrations were calculated as described in the Exposure Point Concentrations section of this report.

⁽d)Outdoor and indoor exposure to soil and dust were partitioned based on the amount of time an individual was outdoors. For adult and child residents it was assumed that 30% of time was spent outdoors. This value is based on information presented in the Standard Default Exposure Factors, which indicates that residents spend 5 out of 16 waking hours outdoors. For all other exposure scenarios it was assumed that an individual spends a total of 2 hours outdoors per day.

⁽e) Based on professional judgment, the adolescent incidental soil ingestion rate was chosen as the midpoint between the residential child and adult values (i.e., 200 mg/day and 100 mg/day). It was assumed that an adolescent would potentially receive more soil contact than an adult, but that this contact was likely to be less than a child under age 6.

⁽f) Exposure duration was based on tours of duty of 3 or 6 years or 30 years for a civilian worker. For the 30-year residential exposure scenario the integrated ingestion daily intake equation (e.g., 6 years as a child and 24 years as an adult) was used to calculate intake. For all other exposure scenarios the standard ingestion daily intake equation was used to calculate intake.

⁽⁹⁾⁵ days per week at the day care for 37 weeks each year (52 weeks/year - (2 weeks for vacation + 2 weeks rain + 11 weeks cold weather)).

^(h)School days per year.

⁽¹⁾ Assumes that an individual played golf once a week for 37 weeks each year (52 weeks/year - (2 weeks for vacation + 2 weeks rain + 11 weeks cold weather))

⁽i) Adolescent body weight was determined by averaging the average weight for boys and girls combined from year 6 through 12 (USEPA, 1996).

Table 3-3 — Exposure Parameters^(a) - Incidental Indoor Dust Ingestion

⁽a) Exposure factors without footnotes are USEPA Standard Defaults (EPA, 1991b).

⁽b) For the 30-year residential exposure scenario (i.e., 6 years as a child and 24 years as an adult) the integrated intake equation was used to calculate intake. For all other exposure scenarios the standard intake equation was used to calculate intake. Parameters with an "a" subscript are for adults and parameters with a "c" subscript are for children.

⁽c) The Average and RME concentrations were calculated as described in the Exposure Point Concentrations section of this report.

⁽d)Outdoor and indoor exposure to soil and dust were partitioned based on the amount of time an individual was outdoors. For adult and child residents it was assumed that 30% of time was spent outdoors. This value is based on information presented in the Standard Default Exposure Factors, which indicates that residents spend 5 out of 16 waking hours outdoors. For all other exposure scenarios it was assumed that an individual spends a total of 2 hours outdoors per day.

⁽e) Based on professional judgment, the adolescent incidental soil and dust ingestion rate was chosen as the midpoint between the residential child and adult values (i.e., 200 mg/day and 100 mg/day). It was assumed that an adolescent would potentially receive more soil and dust contact than an adult, but that this contact was likely to be less than a child under age 6.

⁽f) Exposure duration was based on tours of duty of 3 or 6 years or 30 years for a civilian worker. For the 30-year residential exposure scenario the integrated ingestion daily intake equation (e.g., 6 years as a child and 24 years as an adult) was used to calculate intake. For all other exposure scenarios the standard ingestion daily intake equation was used to calculate intake.

⁽⁹⁾⁵ days per week at the day care for 50 weeks each year (52 weeks/year – 2 weeks for vacation).

⁽h)School days per year.

⁽i)Adolescent body weight was determined by averaging the average weight for boys and girls combined from year 6 through 12 (EPA, 1996).

Table 3-4 — Exposure Parameters^(a) - Dermal Contact With Soil

$$Daily Intake \binom{mg}{kg-dary} = \frac{C_s \times CR \times SA \times AB \times ED \times EF \times FI \times CF}{BW \times AT}$$

$$\frac{(^{\text{to}}) Integrated Adult and Child Daily Intake \binom{mg}{kg-dary}}{C} = \frac{C_s \times FI \times CF \times AB \times \frac{\left(\frac{CR_{cols} \times SA_{cols} \times SA_{cols} \times EI_{cold}}{BW_{cold}}\right) \times ED_{child}}{C} + \frac{\left(\frac{CR_{cols} \times SA_{cols} \times EI_{cold}}{BW_{cold}}\right) \times ED_{child}}{C} \times ED_{child}} \times ED_{child} + \frac{\left(\frac{CR_{cols} \times SA_{cols} \times EI_{cold}}{BW_{cold}}\right) \times ED_{child}}{C} \times ED_{child} \times ED_{child}} \times ED_{child} + \frac{\left(\frac{CR_{cols} \times SA_{cols} \times EI_{cold}}{BW_{cold}}\right) \times ED_{child}}{C} \times ED_{child} \times ED_{child}} \times ED_{child} \times ED_{child} \times ED_{child} \times ED_{child} \times ED_{child} \times ED_{child}} \times ED_{child} \times$$

⁽a) Exposure factors without footnotes are U.S. Environmental Protection Agency Standard Defaults (EPA, 1991b).

⁽b) For the 30-year residential exposure scenario (i.e., 6 years as a child and 24 years as an adult) the integrated intake equation was used to calculate intake. For all other exposure scenarios the standard intake equation was used to calculate intake. Parameters with an "a" subscript are for adults and parameters with a "c" subscript are for children.

⁽c) The Average and RME concentrations were calculated as described in the Exposure Point Concentrations section of this report.

⁽d) Chemical-Specific Absorption Factor (See Table 3-7).

⁽e) Values are EPA Region X Standard Defaults. The child exposure value is based on the assumption that the arms, legs, hands, and feet of a child are exposed. Adult surface area assumes 25% of the time at 5,000 cm² and 75% of the time at 1,900 cm² (USEPA, 1991b).

⁽f) Skin Surface area available for exposure was determined based on the data presented in the Exposure Factors Handbook: Volume I - General Factors (USEPA, 1996). It was assumed that a 6 to 12 year-old adolescent arms and hands were exposed during 75% of the year and that their arms, hands, legs, and feet were exposed for 25% (i.e., summer) of the year.

⁽g) Skin Surface area available for exposure was determined based on the data presented in the Exposure Factors Handbook: Volume I - General Factors (USEPA, 1996). Value is based on the head and hands of an adult.

⁽h) Exposure duration was based on tours of duty of 3 or 6 years or 30 years for a civilian worker. For the 30-year residential exposure scenario the integrated dermal daily intake equation (e.g., 6 years as a child and 24 years as an adult) was used to calculate intake. For all other exposure scenarios the standard inhalation daily intake equation was used to calculate intake.

⁽¹⁾5 days per week at the day care for 37 weeks each year (52 weeks/year - (2 weeks for vacation + 2 weeks rain + 11 weeks cold weather)).

⁽i)School days per year.

⁽k) Adolescent body weight was determined by averaging the average weight for boys and girls combined from year 6 through 12 (EPA, 1996).

Table 3-5 — Exposure Parameters (a) - Inhalation of Particulates and Vapors in Ambient Air

	Inhalation Daily Intake $\binom{mg}{kg-day} = \frac{C_a \times IR \times ET \times ED \times EF \times FI}{BW \times AT}$									
(b) Integr										
Exposure Parameter	Definition	Units		Area						
				Child Development Elementary School Center				Residential Towers (3101/3102)		Golf Course
			Child (0-6) Student	Adult Care Provider	Adolescent (6 - 12) Student	Adult Teacher	Child (0 - 6) Resident	Adult Resident	Adult Worker	Adult Recreator (Golfer)
Ca	Constituent concentration in ambient air ^(c)	mg/m ³	Average or RME	Average or RME	Average or RME	Average or RME	Average or RME	Average or RME	Average or RME	Average or RME
FI	Fraction from contaminated source	%	100%	100%	100%	100%	100%	100%	100%	100%
IR	Inhalation rate	m ³ /hour	1.0 ^(d)	0.833	1.0 ^(d)	0.833	0.5	0.833	0.833	0.833
ET	Exposure time ^(e)	hours/day	2	2	2	2	5	5	2	5 ^(f)
ED	Exposure duration	years	3 or 6	3 or 6	3 or 6	3 or 6	$3, 6, \text{ or } 30^{(g)}$	$3, 6, \text{ or } 30^{(g)}$	3, 6, or 30	3, 6, or 30
EF	Exposure frequency	days/year	185 ^(h)	185 ^(h)	180 ⁽ⁱ⁾	180 ⁽ⁱ⁾	350	350	250	37 ^(j)
BW	Body weight	kg	15	70	38 ^(k)	70	15	70	70	70
At _{nc}	Averaging time – non- carcinogenic (3, 6, 30- years) (Calculated as the Exposure Duration x 365 days/year)	days	1,095; 2,190	1,095; 2,190	1,095; 2,190	1,095; 2,190	1,095; 2,190; 10,950	1,095; 2,190; 10,950	1,095; 2,190; 10,950	1,095; 2,190; 10,950
At _c	Averaging time - carcinogenic (lifetime) (Calculated as the 70 year lifetime expectancy x 365 days/year)	days	25,550	25,550	25,550	25,550	25,550	25,550	25,550	25,550

⁽a) Exposure factors without footnotes are U.S. Environmental Protection Agency Standard Defaults (USEPA, 1991b).

⁽b) For the 30-year residential exposure scenario (i.e., 6 years as a child and 24 years as an adult) the integrated intake equation was used to calculate intake. For all other exposure scenarios the standard intake equation was used to calculate intake. Parameters with an "a" subscript are for adults and parameters with a "c" subscript are for children.

(c) The Average and RME concentrations were calculated as described in the Exposure Point Concentrations section of this report.

^(d)Inhalation rate for light activities for adults and children (USEPA, 1996).

⁽e) Outdoor and indoor inhalation exposures were partitioned based on the amount of time an individual was outdoors. For adult and child residents it was assumed that 30% of time was spent outdoors. This value is based on information presented in the Standard Default Exposure Factors, which indicates that residents spend 5 out of 16 waking hours outdoors. For all other exposure scenarios it was assumed that an individual spends a total of 2 hours outdoors per day.

⁽f) Assumes that it takes 5 hours to complete 18 holes of golf.

⁽⁹⁾ Exposure duration was based on tours of duty of 3 or 6 years or 30 years for a civilian worker. For the 30-year residential exposure scenario the integrated inhalation daily intake equation (e.g., 6 years as a child and 24 years as an adult) was used to calculate intake. For all other exposure scenarios the standard inhalation daily intake equation was used to calculate intake.

⁽h)5 days per week at the day care for 37 weeks each year (52 weeks/year - (2 weeks for vacation + 2 weeks rain + 11 weeks cold weather)).

⁽i)School days per year.

⁽i) Assumes that an individual played golf once a week for 37 weeks each year (52 weeks/year - (2 weeks for vacation + 2 weeks rain + 11 weeks cold weather))

⁽k) Adolescent body weight was determined by averaging the average weight for boys and girls combined from year 6 through 12 (USEPA, 1996).

Table 3-6— Exposure Parameters^(a) - Inhalation of Particulates and Vapors in Indoor Air

	Inhalation Daily Intake $\binom{mg}{kg-day} = \frac{C_a \times IR \times ET \times ED \times EF \times FI}{BW \times AT}$								
(b) Integr									
Exposure Parameter	Definition	Units							
			Child Development Elementary School Residential Towers GEMB Center (3101/3102)						GEMB
			Child (0-6) Student	Adult Care Provider	Adolescent (6 - 12) Student	Adult Teacher	Child (0 - 6) Resident	Adult Resident	Adult Worker
Ca	Constituent concentration in indoor air (c)	mg/m ³	Average or RME	Average or RME	Average or RME	Average or RME	Average or RME	Average or RME	Average or RME
FI	Fraction from contaminated source	%	100%	100%	100%	100%	100%	100%	100%
IR	Inhalation rate	m ³ /hour	1.0 ^(d)	0.833	1.0 ^(d)	0.833	0.5	0.833	0.833
ET	Exposure time ^(e)	hours/day	6	8	6	8	19	19	8
ED	Exposure duration	years	3 or 6	3 or 6	3 or 6	3 or 6	3, 6, or 30 ^(f)	3, 6, or 30 ^(f)	3, 6, or 30
EF	Exposure frequency	days/year	250 ^(g)	250 ^(g)	180 ^(h)	180 ^(h)	350	350	250
BW	Body weight	kg	15	70	38 ⁽ⁱ⁾	70	15	70	70
At _{nc}	Averaging time – non- carcinogenic (3, 6, 30- years)	days	1,095; 2,190	1,095; 2,190	1,095; 2,190	1,095; 2,190	1,095; 2,190; 10,950	1,095; 2,190; 10,950	1,095; 2,190; 10,950
At _c	Averaging time - carcinogenic (lifetime) (Calculated as the 70 year lifetime expectancy x 365 days/year)	days	25,550	25,550	25,550	25,550	25,550	25,550	25,550

⁽a) Exposure factors without footnotes are U.S. Environmental Protection Agency Standard Defaults (USEPA, 1991b).

⁽b) For the 30-year residential exposure scenario (i.e., 6 years as a child and 24 years as an adult) the integrated intake equation was used to calculate intake. For all other exposure scenarios the standard intake equation was used to calculate intake. Parameters with an "a" subscript are for adults and parameters with a "c" subscript are for children.

⁽c) The Average and RME concentrations were calculated as described in the Exposure Point Concentrations section of this report.

⁽d)Inhalation rate for light activities for adults and children (USEPA, 1996).

⁽e) Outdoor and indoor inhalation exposures were partitioned based on the amount of time an individual was outdoors. For adult and child residents it was assumed that 30% of time was spent outdoors. This value is based on information presented in the Standard Default Exposure Factors, which indicates that residents spend 5 out of 16 waking hours outdoors. For all other exposure scenarios it was assumed that an individual spends a total of 2 hours outdoors per day.

⁽f) Exposure duration was based on tours of duty of 3 or 6 years or 30 years for a civilian worker. For the 30-year residential exposure scenario the integrated inhalation daily intake equation (e.g., 6 years as a child and 24 years as an adult) was used to calculate intake. For all other exposure scenarios the standard inhalation daily intake equation was used to calculate intake.

⁽⁹⁾⁵ days per week at the day care for 50 weeks each year (52 weeks/year – 2 weeks for vacation).

⁽h)School days per year.

⁽i)Adolescent body weight was determined by averaging the average weight for boys and girls combined from year 6 through 12 (USEPA, 1996).

Each variable in this equation has a range of possible values associated with it. The intake variable values for a given pathway are selected so that the combination of all intake variables resulted in a realistic upper bound estimate (or RME) of the possible exposure by that pathway.

Quantitative characterization of carcinogenic and non-carcinogenic effects required estimating the potential human exposure levels for each COC. Exposure levels for carcinogens were averaged over the lifetime of the exposed individual (i.e., 70 years) while exposure levels for non-carcinogens were averaged over the duration of exposure.

The intake of a constituent was estimated using at least six basic factors: exposure frequency, exposure duration, contact rate, constituent concentration, body weight, and averaging time. In this assessment, intake was normalized for time and body weight, and was expressed in milligrams of constituent per kilogram of body weight per day (mg/kg-day). The exposure factors and algorithms used in this assessment to quantify exposure are presented in Tables 3-2 through 3-6. The average daily dose (ADD) and lifetime average daily dose (LADD) are presented in Appendix C of the Pioneer Report (Pioneer 2000).

3.4 Toxicity Assessment

The purpose of the toxicity assessment was to identify constituent and route-specific toxicity criteria for each COC to quantify the potential health impacts to exposed people. These toxicity values were used in conjunction with the information presented in the exposure assessment to calculate risks.

Both carcinogenic and non-carcinogenic health effects must be considered when evaluating potential human health impacts. Cancer toxicity values (carcinogenic slope factors [CSFs]) and non-carcinogenic toxicity values (reference doses [RfDs]) are derived through an evaluation of the relationship between the amount of an agent that changes certain aspects of the biological system. USEPA has evaluated numerous constituents and has published the corresponding toxicity values, which have undergone peer review. The following sources were consulted to identify toxicity values for this assessment:

• The Integrated Risk Information System (IRIS) (EPA 1999c).

- The Health Effects Assessment Summary Tables—Annual Update (HEAST) (EPA 1997).
- Provisional Toxicity Values Available from the National Center for Environmental Assessment (NCEA) Superfund Health Risk Technical Support Center (NCEA-CIN) (EPA 1999d).
- California EPA (Cal EPA) Toxicity Values including:
 - Technical Support Document for Describing Available Cancer Potency
 Factors and Hot Spots Unit Risk and Summary Table of Cancer Potency
 Values (Cal EPA 1999a); and
 - Technical Support Document for the Determination of Non-cancer Chronic Reference Exposure Levels (RELs) and Proposed Office of Environmental Health Hazard Assessment (OEHHA) Chronic Inhalation REL Summary (Cal EPA 1999b).

The values presented in IRIS have been "verified" by either the USEPA Reference Dose/Reference Concentration (RfD/RfC) Work Group or the Carcinogen Risk Assessment Verification Endeavor (CRAVE). These agency work groups conduct a verification process that leads to internal agency scientific consensus regarding risk assessment information for a agent. All of the toxicity values presented in the HEAST document are considered "provisional" by USEPA because they have not been verified by an agency work group (EPA, 1997). Provisional values are not listed in IRIS. Additional provisional and internal USEPA toxicity values were obtained from USEPA. The values provided by NCEA-CIN include chronic and subchronic toxicity values, unit risks, and slope factors. The values that have been peer reviewed are considered provisional, while the values that have not been peer reviewed are considered internal USEPA values. The cancer unit risk and potency factor values developed by Cal EPA were reviewed by a Cal EPA working group to ensure agency-wide consistency and harmonization. The Cal EPA inhalation exposure levels (i.e., non-carcinogenic toxicity values) are intended to protect the public from a lifetime of exposure to hazardous airborne substances. These healthbased chronic exposure levels are intended for risk characterization of routine industrial emissions.

Since multiple toxicity values were available for some constituents, the sources of toxicity information were prioritized as follows to select the toxicity values used in the assessment to calculate risk:

- 1. IRIS values
- 2. HEAST values
- 3. USEPA provisional values
- 4. USEPA internal values
- 5. USEPA provisional subchronic non-cancer toxicity values (which were converted to chronic toxicity values by dividing the subchronic value by 10)
- 6. USEPA internal subchronic non-cancer toxicity values (which were converted to chronic toxicity values by dividing the subchronic value by 10)
 - 7. Cal EPA toxicity values

The potential for producing carcinogenic effects was limited to certain COCs (i.e., carcinogens), while adverse non-carcinogenic health effects were potentially resulting from exposure to any constituent. Therefore, where available, cancer toxicity values were obtained for those constituents identified by Cal EPA as carcinogens and non-cancer toxicity values were obtained for each of the COCs.

The toxicity of any constituent depends on its route of entry into the body. In some cases a constituent may produce toxicity only at or near a specific route of entry and may not be toxic through other routes of exposure. Therefore, the route-specific toxicity value was used for each constituent.

Toxicity Values

Tables 4-2 and 4-3 of the Pioneer Health Risk Assessment (Pioneer 2000) present ingestion and inhalation constituent-specific toxicity values for COCs assessed in the human health evaluation. The number and type of toxicity values identified for this evaluation can be characterized as follows:

• Oral RfDs were available for 95 of the 246 COCs.

- Inhalation RfDs were available for 58 of the 246 COCs.
- Oral carcinogenic slope factors were available for 44 of the 246 COCs.
- Inhalation carcinogenic slope factors were available for 43 of the 246 COCs.
- Toxicity values for 86 of the 246 COCs were not available (see Table 4-4 of the Pioneer Health Risk Assessment (Pioneer 2000)). In response to review comments from the Subcommittee on Toxicology, the National Academy of Sciences, an assessment of these 86 chemicals is provided in Appendix E and summarized in Section 4. COCs lacking toxicity information were not evaluated further in the risk assessment.

Carcinogenic Health Criteria

The mechanism for carcinogenesis is referred to as a "non-threshold" process, since any level of exposure to such an agent poses a small, but finite, probability of generating a carcinogenic response. CSFs were used in this assessment to evaluate carcinogenic risks. A CSF is a numerical estimate of the potency of a constituent, which, when multiplied by the average lifetime dose, gives the probability of an individual developing cancer over a lifetime.

Non-carcinogenic Health Criteria

The term RfD was developed by USEPA to refer to an estimate (with uncertainty spanning perhaps an order of magnitude or greater) of a daily intake of a constituent to which an individual, including sensitive subpopulations, can be exposed without any expectation of adverse noncarcinogenic health effects (e.g., organ damage, biochemical alterations, birth defects) (EPA, 1989).

Non-carcinogenic Toxic Endpoints and Critical Effects

For non-cancer health effects, hazard quotients are added across COCs when they target the same organ, or produce the same critical effect, to calculate a segregated hazard index. Segregation of hazard indices requires the identification of the major effects of each constituent, including those seen at higher doses than the critical effect (e.g., the constituent may cause liver damage at a dose of 100 mg/kg-day and neurotoxicity at a dose of 250 mg/kg-day). Major effect categories include:

- Neurotoxicity
- Developmental toxicity
- Reproductive toxicity
- Immunotoxicity
- Adverse effects by target organ (i.e., hepatic, renal, respiratory, cardiovascular, gastrointestinal, hematological, musculoskeletal, and dermal/ocular effects).

Although higher exposure levels may be required to produce adverse health effects other than the critical effect, the RfD can be used as the toxicity value for each effect category as a conservative and simplifying step (EPA 1989). The toxic endpoints and critical effects for the constituents comprising the majority of the non-carcinogenic hazard indices are presented in Table 4-5, section 4 of the Pioneer Health Risk Assessment (Pioneer 2000).

3.5 Risk Characterization

Risk characterization requires integrating exposure and toxicity information into a quantitative estimate of non-carcinogenic hazard indices and carcinogenic risks. Risk estimates for the individual exposed to the average exposure and the RME were calculated for each COC, exposure pathway, and AOC. The exposure parameters used to calculate human health risks for the average exposure and the RME exposed individual were identical. The only difference between the calculations was the exposure point concentration used to calculated intake. The risks associated with each COC and exposure pathway were summed for each location to estimate the total human health risks.

Evaluation of Non-carcinogenic Effects

Adverse non-carcinogenic effects from exposure to a COC are quantitatively expressed as a hazard quotient. The hazard quotient is the ratio of the estimated dose of a particular constituent that a human receives to the RfD of the constituent:

HQ = ADD/RfD

where,

Parameter	Definition
HQ	Hazard Quotient; the ratio of the estimated dose of a constituent to the RfD
ADD	Average daily dose of constituent (mg/kg-day)
RfD	Reference dose for constituent (mg/kg-day)

Only chronic hazard quotients were evaluated in this assessment, since the subchronic effects within a given exposure scenario were typically less than or equal to the chronic effects for the same scenario. All hazard quotients for constituents were summed to yield a total hazard index. If the total hazard index was less than 1.0, it indicates that adverse non-carcinogenic health effects are unlikely. If the total hazard index was greater than 1.0, it indicates that adverse health effects were possible. However, the hazard index does not represent a probability of occurrence or a quantification of the magnitude of non-carcinogenic health effects.

Evaluation of Carcinogenic Effects

The risk of cancer from exposure to a constituent is described in terms of the probability that an exposed individual will develop cancer during a lifetime from that exposure. The risk estimate is calculated by multiplying the daily intake of a particular constituent over a lifetime by the carcinogenic slope factor.

$$RISK = LADD*SF$$

where,

Parameter	Definition
RISK	Lifetime probability of developing cancer due to exposure to a constituent in
	the environment.
LADD	Lifetime average daily dose to constituent (mg/kg-day).
SF	Carcinogenic slope factor for constituent (mg/kg-day) ⁻¹ .

All carcinogenic risks for constituents for each scenario and receptor were summed to yield the total carcinogenic risk. A 1 in 1,000,000 cancer risk (i.e., 1E-06) means that in a population of 1,000,000 people exposed under an identical exposure scenario (i.e., everyone had exactly the same daily intake of a carcinogen over the same period), there could be one

additional case of cancer in the population above the background number of cancer cases expected.

It is important to note that the human health risks, presented in Table 3-8, were calculated based upon the exposure for the particular scenario being evaluated and the time period that individuals were expected to remain at the various AOCs. For example, the health risk for a child going to school or an adult working at the Shirley Landham Elementary School was based upon being at the school for a period of 10 hours each day and 180 days each year. No additional exposures were calculated for the adults or children for the remaining 14 hours each day and 185 days each year. Likewise, no additional exposures were calculated for adults working at the Ground Electronics Maintenance Building, attending or working at the day care facility, or golfing. Individuals living or remaining on base for longer periods of time than the period used to calculate health risk, for a particular exposure scenario, may be at an increased health risk.

For each exposure scenario, the health risk that was calculated, considered a representative amount of time that individuals were likely to spend out of doors and indoors. For example, a child living at the Residential Tower was anticipated to be out of doors 5 hours each day and indoors for the remainder of the 24 hours each day. Likewise, a child at the Shirley Landham Elementary School was anticipated to spend 2 hours out of doors and the remainder of the 10 hours indoors.

In other words, exposures at each AOC were evaluated independently (i.e. exposures were not combined across AOCs). There are a myriad of exposure scenarios that could be evaluated for the NAF Atsugi population. Since it is not possible to evaluate every combination of exposure that may occur at NAF Atsugi, plausible upper bound risk estimates of these exposure combinations are presented later in Section 4, Table 4-9.

Based on the potentially exposed populations identified at NAF Atsugi in Table 3-7 and the exposure pathways that were evaluated in this assessment, health risks were calculated for adults and children living on the base; working at the GEMB, day care or Elementary School; attending school or attending day care. Risk calculations were conducted for 3 years and 6 years, representing one and two tours of duty, and 30 years representing the USEPA residential default

scenario for comparison. As indicated in Table 3-7 specifically, day care, Elementary School, industrial, residential, and recreational golfer exposure scenarios were evaluated. Site-specific exposure parameters were used, where appropriate, to model human exposure to average and Reasonable Maximum Exposure (RME) concentrations of COCs in the environment. The RME is the maximum exposure that is reasonably expected to occur at a site. The RME concentration is the 95% upper confidence limit on the arithmetic average concentration. This information was combined with toxicity information to quantify the potential risks. Table 3-8 presents the hazard indices and carcinogenic risks for each AOC evaluated at NAF Atsugi.

Carcinogenic Risk Summary

As indicated in Table 3-8, the carcinogenic risks for every exposure scenario were less than the upper limit of the USEPA acceptable risk range of 1 x 10⁻⁴ (1 in 10,000) except for the residential scenario at the Residential Towers and the industrial scenario at the GEMB. The carcinogenic risks at the Residential Towers exceeded 1 x 10⁻⁴ (1 in 10,000) for the child resident based on a 3-year and 6-year exposure. The carcinogenic risks at the GEMB only exceeded 1 x 10⁻⁴ (1 in 10,000) for the adult worker based on a 30-year exposure. The highest carcinogenic risk was 5.2 x 10⁻⁰⁴ (5.2 in 10,000) at the Residential Towers, assuming a 30-year residential exposure. The inhalation exposure pathways accounted for greater than 80% of the carcinogenic risk at each location with the exception of the Golf Course. Tables 3-9 and 3-10, which present the hazard quotients and carcinogenic risks summed by exposure pathway.

Non-carcinogenic Effects Summary

As indicated in Table 3-8, the non-carcinogenic hazard indices for every exposure scenario are greater than the USEPA acceptable Hazard Index of 1, except for the Recreational Golfer Scenario. The highest average hazard index calculated for the average exposed individual was 53 at the Residential Towers for the child 3-year and 6-year residential exposure scenarios. Likewise, the highest hazard index calculated for the reasonable maximum exposed individual was 67 at the Residential Towers for the child 3-year and 6-year residential exposure scenarios. The child residential scenarios had higher hazard indices than all other exposure scenarios. The inhalation exposure pathways accounted for greater than 95% of the hazard index at each location, as shown in Table 3-9.

Exposed Population	Incidental Soil Ingestion	Dermal Contact with Soil	Inhalation of Particulates and	Inhalation of Particulates and	Incidental Indoor Dust
			Vapors in Ambient	Vapors in Indoor	Ingestion ³
			Air	Air ²	
	Child Developme	nt Center, Building	291		
Child at Day Care (0-6)	•	•	•1	•	•
Adult Child Care Provider	•	•	•1	•	•
	Elementary S	chool, Building 993			
Adolescent at School (6-18)	•	•	•	•	•
Adult Worker	•	•	•	•	•
	Residential Towe	ers, Building 3101/31	02		
Child at Home (0-6)	•	•	•	•	•
Adult Resident	•	•	•	•	•
Groun	d Electronics Maint	enance Building, Bu	iilding 1061		
Adult Worker	•4	•	•	•	•
	Go	lf Course			
Adult Recreator (Golfer)	•4	0	•	NC	NC

Table 3-7 — Summary of Exposed Populations at NAF Atsugi

- 1. All exposed populations were evaluated based on Average and Upper-Bound (RME) exposure point concentrations.
- 2. Residents were evaluated in the risk assessment assuming 3-year, 6-year, and 30-year exposure durations. The 3-year and 6-year exposure durations correspond with 1 and 2 tours of duty, respectively. The 30-year exposure duration corresponds to the USEPA standard default residential exposure duration.
- 3. Workers were evaluated in the risk assessment assuming 3-year, 6-year, and 30-year exposure durations. The 3-year and 6-year exposure durations correspond with 1 and 2 tours of duty, respectively. The 30-year exposure duration corresponds to the USEPA standard default industrial worker exposure duration.
- 4. Recreators (Golfers) were evaluated in the risk assessment assuming 3-year, 6-year, and 30-year exposure durations. The 3-year and 6-year exposure durations correspond with 1 and 2 tours of duty, respectively. The 30-year exposure duration corresponds to the USEPA standard default residential exposure duration.

5.

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^{• =} Exposure pathway evaluated in this assessment.

^{○ =} Not considered a significant exposure pathway for this receptor.

NC = Not a complete exposure pathway.

¹Ambient air concentrations were not collected from this location. However, for the purposes of the risk assessment the ambient air concentrations from the Elementary School were used as the exposure point concentration at this site because the Elementary School is the closest ambient air monitoring site at approximately 200 meters.

²Ambient air concentrations were used as surrogate for indoor air concentrations. The actual measured indoor air concentrations collected during the 14-month monitoring study were qualitatively evaluated to determine if indoor air concentrations at Atsugi are comparable with indoor air concentrations in the United States.

³Indoor dust samples were only analyzed for Dioxins/Furans. Consequently, outdoor soil concentrations were used as surrogate indoor dust concentrations for all other COCs. The ingestion rates for Incidental Soil Ingestion (outdoor) and Incidental Dust Ingestion (indoor) were partitioned such that 30% of total exposure comes from outdoors and 70% of exposure comes from indoors. This is calculated assuming that a person spends a total of 5 out of 16 hours outdoors per day (5/16 = 0.31). The five hours outdoors is based on the Standard Default Residential Inhalation value, which assumes 5 hours outdoors per day.

⁴Soil samples collected for the trend analysis were used to evaluate the risks associated with exposure to soil at these locations. Notes:

Table 3-8 — Total Average and Reasonable Maximum Non-carcinogenic Effects and Carcinogenic Risks for the AOCs at NAF Atsugi, Japan

Scenario	Receptor	Exposure Duration	Location	Hazard	l Index	Cance	er Risk
				Average	RME	Average	RME
Residential	Child (0 - 6)	3-year	Residential Towers	53	67	6.2E-05	1.1E-04
Residential	Child (0 - 6)	6-year	Residential Towers	53	67	1.2E-04	2.3E-04
	Range			53 -	- 67	6.2E-05	- 2.3E-04
Residential	Adult	3-year	Residential Towers	19	24	2.0E-05	3.7E-05
Residential	Adult	6-year	Residential Towers	19	24	4.0E-05	7.4E-05
Residential	Child & Adult	30-year	Residential Towers	26	33	2.8E-04	5.2E-04
	Range			19 -		2.0E-05	- 5.2E-04
Day Care	Child (0 - 6)	3-year	Child Development Center	30	42	2.7E-05	3.4E-05
Day Care	Child (0 - 6)	6-year	Child Development Center	30	42	5.3E-05	6.7E-05
	Range			30 -	- 42	2.7E-05	- 6.7E-05
Day Care	Adult Care Provider	3-year	Child Development Center	7	10	5.7E-06	7.2E-06
Day Care	Adult Care Provider	6-year	Child Development Center	7	10	1.1E-05	1.5E-05
	Range		Center	7 –	10	5.7E-06	- 1.5E-05
Elementary School	Adolescent (6 - 12)	3-year	Elementary School	9	13	8.2E-06	1.1E-05
Elementary School	Adolescent (6 - 12)	6-year	Elementary School	9	13	1.6E-05	2.2E-05
	Range			9 –		8.2E-06	- 2.2E-05
Elementary School	Adult Teacher	3-year	Elementary School	5	7	4.4E-06	5.9E-06
Elementary School	Adult Teacher	6-year	Elementary School	5	7	8.7E-06	1.2E-05
	Range	T		5 -			- 1.2E-05
Recreational Golfer	Adult	3-year	Golf Course	0.6	0.8	7.3E-07	1.3E-06
Recreational Golfer	Adult	6-year	Golf Course	0.6	0.8	1.5E-06	2.6E-06
Recreational Golfer	Adult	30-year	Golf Course	0.6	0.8	7.3E-06	1.3E-05
	Range	 	i e	0.6 -	t		- 1.3E-05
Military/Industrial Worker	Adult	3-year	Ground Electronics Maintenance	8	13	9.2E-06	1.2E-05
Military/Industrial Worker	Adult	6-year	Ground Electronics Maintenance	8	13	1.8E-05	2.3E-05

Table 3-8 — Total Average and Reasonable Maximum Non-carcinogenic Effects and Carcinogenic Risks for the AOCs at NAF Atsugi, Japan

Scenario	Receptor	Exposure Duration	Location	Hazard Index		Canc	er Risk
		Duration		Average	RME	Average	RME
Military/Industrial Worker	Adult	30-year	Ground Electronics Maintenance	8	13	9.2E-05	1.2E-04
	Range			8 –	13	9.2E-06	- 1.2E-04

Note: The risks are not directly comparable between AOCs because different site-specific exposure scenarios were evaluated at each location. Shaded values indicate risks that exceeded the regulatory benchmarks of a hazard index of 1 and/or a carcinogenic risk of 1 x 10⁻⁴.

Table 3-9 — Hazard Indices by Exposure Pathway

				Child Hazard Indices (HI) ¹					Adult Hazard Indices (HI)						
	Exposure Point	Exposure		Inhalation of	Inhalation of	Ingestion of Indoor	Ingestion	Dermal Contact with	Total	Inhalation of	Inhalation of	Ingestion of Indoor	Ingestion	Dermal Contact	Total
Scenario	Concentration	Duration	Location	Ambient Air		Dust	of Soil	Soil		Ambient Air	Indoor Air	Dust	of Soil	with Soil	Hl
			Child Development						20.2		- 0				
Day Care	Average	3-year	Center	6.0	24.3	0	0	0	30.3	1.1	5.8	0	0	0	6.9
Day Care	Average	6-year	Child Development Center	6.0	24.3	0	0	0	30.3	1.1	5.8	0	0	0	6.9
Day Care	RME	3-year	Child Development Center	8.3	33.8	0	0	0	42.1	1.5	8.0	0	0	0	9.5
Day Care	RME	6-year	Child Development Center	8.3	33.8	0	0	0	42.1	1.5	8.0	0	0	0	9.5
Recreational Golfer	Average	3-year	Golf Course							0.6			0.02		0.6
Recreational Golfer	Average	6-year	Golf Course							0.6			0.02		0.6
Recreational Golfer	Average	30-year	Golf Course							0.6			0.02		0.6
Recreational Golfer	RME	3-year	Golf Course							0.7			0.02		0.8
Recreational Golfer	RME	6-year	Golf Course							0.7			0.02		0.8
Recreational Golfer	RME	30-year	Golf Course		-1			-	-	0.7	-	-	0.02	-	0.8
Commercial Worker	Average	3-year	GEMB							1.7	6.7	0.000009	0.000002	0	8.4
Commercial Worker	Average	6-year	GEMB					-	-	1.7	6.7	0.000009	0.000002	0	8.4
Commercial Worker	Average	30-year	GEMB		1			1	1	1.7	6.7	0.000009	0.000002	0	8.4

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Table 3-9 — Hazard Indices by Exposure Pathway

				Child Hazard Indices (HI) ¹				Adult	Hazard Ind	ices (HI)					
Scenario	Exposure Point Concentration	Exposure Duration	Location	Inhalation of Ambient Air		Ingestion of Indoor Dust	Ingestion of Soil	Dermal Contact with Soil		Inhalation of Ambient Air		Ingestion of Indoor Dust	Ingestion of Soil	Dermal Contact with Soil	Total Hl
Commercial				7 timorent 7 tii	mgoor / m	Dust	01 5011	5011	111	7 tillolelit 7 til	mgoor /m	Dust	01 5011	with 50h	111
Worker	RME	3-year	GEMB							2.6	10.3	0.000009	0.000002	0	12.8
Commercial Worker	RME	6-year	GEMB							2.6	10.3	0.000009	0.000002	0	12.8
Commercial Worker	RME	30-year	GEMB							2.6	10.3	0.000009	0.000002	0	12.8
Elementary School	Average	3-year	Elementary School	2.3	6.9	0.0004	0.0001	0.0003	9.2	1.0	4.2	0.00007	0.00002	0.0001	5.2
Elementary School	Average	6-year	Elementary School	2.3	6.9	0.0004	0.0001	0.0003	9.2	1.0	4.2	0.00007	0.00002	0.0001	5.2
Elementary School	RME	3-year	Elementary School	3.2	9.6	0.003	0.001	0.003	12.8	1.4	5.8	0.0006	0.0001	0.001	7.2
Elementary School	RME	6-year	Elementary School	3.2	9.6	0.003	0.001	0.003	12.8	1.4	5.8	0.0006	0.0001	0.001	7.2
Residential	Average	3-year	Residential Towers	11.0	41.7	0.01	0.006	0.001	52.7	3.9	14.9	0.002	0.0007	0.0002	18.8
Residential	Average	6-year	Residential Towers	11.0	41.7	0.01	0.006	0.001	52.7	3.9	14.9	0.002	0.0007	0.0002	18.8
Residential	Average	30-year	Residential Towers							5.3	20.3	0.004	0.002	0.0005	25.6
Residential	RME	3-year	Residential Towers	14.0	53.3	0.03	0.01	0.002	67.3	5.0	19.0	0.003	0.001	0.0004	24.0
Residential	RME	6-year	Residential Towers	14.0	53.3	0.03	0.01	0.002	67.3	5.0	19.0	0.003	0.001	0.0004	24.0
Residential	RME	30-year	Residential Towers							6.8	25.9	0.007	0.003	0.0008	32.7

Note: The risks are not directly comparable between AOCs because different site-specific exposure scenarios were evaluated at each location.

⁻⁻Receptor and/or exposure pathway was not evaluated at this location.

1A child (0-6) was evaluated at the Child Development Center and the Residential Towers. An adolescent (6-12) was evaluated at the Elementary School.

Table 3-10 — Carcinogenic Risks by Exposure Pathway

					Child Carcinogenic Risks (CR) ¹					Adult Carcinogenic Risks (CR)					
								Dermal						Dermal	
		Б				Ingestion		Contact				Ingestion		Contact	
Scenario	Case	Exposure Duration	Location	Inhalation of		of Indoor	Ingestion	with		Inhalation of		of Indoor	Ingestion	with	Total
Scenario	Case	Duration		Ambient Air	Indoor Air	Dust	of Soil	Soil	(CR)	Ambient Air	Indoor Air	Dust	of Soil	Soil	(CR)
			Child Development												
Day Care	Average	3-year	Center	4.8E-6	2.0E-5	1.7E-6	1.6E-7	3.6E-7	2 7E-5	8.6E-7	4.7E-6	9.5E-8	6.6E-9	5.8E-8	5.7E-6
Buy cure	riverage	3 y car	Child	1.02 0	2.02.0	1.72 0	1.02 /	3.0E 7	2.72 3	0.02 /	1.72 0	7.51	0.02)	5.6E 6	3.7E 0
			Development												
Day Care	Average	6-year	Center	9.6E-6	3.9E-5	3.3E-6	3.1E-7	7.2E-7	5.3E-5	1.7E-6	9.3E-6	1.9E-7	1.3E-8	1.2E-7	1.1E-5
			Child												
		_	Development												
Day Care	RME	3-year	Center	6.2E-6	2.5E-5	1.7E-6	2.3E-7	5.3E-7	3.4E-5	1.1E-6	5.9E-6	9.5E-8	9.7E-9	8.4E-8	7.2E-6
			Child												
Day Care	RME	6-year	Development Center	1.2E-5	5.0E-5	3.3E-6	4.5E-7	1.1E-6	6 7F-5	2.2E-6	1.2E-5	1.9E-7	1.9E-8	1.7E-7	1.5E-5
Recreational		0 year	Center	1.2L 3	3.0L 3	J.JL 0	T.3L /	1.1L 0	0.7L 3	2.21 0	1.2L 3	1.71	1.7L 0	1.7L /	1.31 3
Golfer	Average	3-year	Golf Course							5.0E-7			2.3E-7		7.3E-7
Recreational															
Golfer	Average	6-year	Golf Course							1.0E-6			4.6E-7		1.5E-6
Recreational		• •													
Golfer	Average	30-year	Golf Course							5.0E-6			2.3E-6		7.3E-6
Recreational Golfer	RME	2	Calf Causs							6.5E.7			6.2E-7		1.2E.6
Recreational		3-year	Golf Course							6.5E-7			0.2E-/		1.3E-6
Golfer	RME	6-year	Golf Course							1.3E-6			1.2E-6		2.6E-6
Recreational	1	o y cui	3011 204130							1.02 0			1.22 0		2.02 0
Golfer	RME	30-year	Golf Course							6.5E-6			6.2E-6		1.3E-5
Commercial		-													
Worker	Average	3-year	GEMB							1.7E-6	6.8E-6	4.2E-7	4.4E-8	2.6E-7	9.2E-6
Commercial	I I														
Worker	Average		GEMB							3.4E-6	1.4E-5	8.4E-7	8.8E-8	5.3E-7	1.8E-5
Commercial	Average	30-year	GEMB							1.7E-5	6.8E-5	4.2E-6	4.4E-7	2.6E-6	9.2E-5

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Table 3-10 — Carcinogenic Risks by Exposure Pathway

				Child Carcinogenic Risks (CR) ¹			Adult Carcinogenic Risks (CR)								
						Ingestion		Dermal Contact				Ingestion		Dermal Contact	
Scenario	Case	Exposure Duration	Location	Inhalation of		of Indoor	Ingestion	with			Inhalation of		Ingestion	with	Total
Worker	Case	Duration	Location	Ambient Air	Indoor Air	Dust	of Soil	Soil	(CK)	Ambient Air	Indoor Air	Dust	of Soil	Soil	(CR)
Commercial															
Worker	RME	3-year	GEMB							2.2E-6	8.7E-6	4.2E-7	4.4E-8	2.6E-7	1.2E-5
Commercial	TUIL	3 year	GENIE							2.22 0	0.72 0	1.22 /	1.12 0	2.02 /	1.22 3
Worker	RME	6-year	GEMB							4.4E-6	1.7E-5	8.4E-7	8.8E-8	5.3E-7	2.3E-5
Commercial															
Worker	RME	30-year	GEMB							2.2E-5	8.7E-5	4.2E-6	4.4E-7	2.6E-6	1.2E-4
Elementary			Elementary	4.05.6				4 45 =	0.00	0.45.5		4 4 5 =	0.500	ć 47. o	
	Average	3-year	School	1.9E-6	5.6E-6	5.7E-7	5.8E-8	1.4E-7	8.2E-6	8.4E-7	3.4E-6	1.1E-7	8.5E-9	6.4E-8	4.4E-6
Elementary School	Average	6-vear	Elementary School	3.7E-6	1.1E-5	1.1E-6	1.2E-7	2.7E-7	1.6E-5	1.7E-6	6.7E-6	2.2E-7	1.7E-8	1.3E-7	8.7E-6
Elementary	Average	0-year	Elementary	3.7E-0	1.1E-3	1.1L-0	1.2L-/	Z./L-/	1.015-3	1./L-0	0.7E-0	2.2E-7	1./L-0	1.3L-/	8.7E-0
School	RME	3-year	School	2.4E-6	7.1E-6	5.8E-7	3.1E-7	7.9E-7	1.1E-5	1.1E-6	4.3E-6	1.1E-7	4.5E-8	3.7E-7	5.9E-6
Elementary			Elementary												
School	RME	6-year	School	4.7E-6	1.4E-5	1.2E-6	6.1E-7	1.6E-6	2.2E-5	2.1E-6	8.6E-6	2.3E-7	8.9E-8	7.4E-7	1.2E-5
			Residential												
Residential	Average	3-year	Towers	1.1E-5	4.1E-5	8.0E-6	6.2E-7	1.0E-6	6.2E-5	3.9E-6	1.5E-5	8.6E-7	6.6E-8	1.7E-7	2.0E-5
D :1 ::1			Residential	2.25.5	0.25.5	1 (1) 7	1.00	2.15.6	1.05.4	7.05.6	2.05.5	1.70.6	1.05.7	2.25.7	4.00. 5
Residential	Average	6-year	Towers	2.2E-5	8.3E-5	1.6E-5	1.2E-6	2.1E-6	1.2E-4	7.8E-6	3.0E-5	1.7E-6	1.3E-7	3.3E-7	4.0E-5
Residential	Average	30-year	Residential Towers							5.3E-5	2.0E-4	2.3E-5	1.8E-6	3.4E-6	2.8E-4
Residential	Average	30-year	Residential							3.3L-3	2.0L-4	2.51-5	1.0L-0	J.4L-0	2.0L-4
Residential	RME	3-year	Towers	2.0E-5	7.8E-5	8.1E-6	2.4E-6	4.4E-6	1.1E-4	7.3E-6	2.8E-5	8.7E-7	2.5E-7	7.0E-7	3.7E-5
			Residential												
Residential	RME	6-year	Towers	4.1E-5	1.5E-4	1.6E-5	4.8E-6	8.8E-6	2.3E-4	1.5E-5	5.5E-5	1.7E-6	5.1E-7	1.4E-6	7.4E-5
			Residential												
Residential		30-year	Towers							9.9E-5	3.8E-4	2.3E-5	6.8E-6	1.4E-5	5.2E-4

Note: The risks are not directly comparable between AOCs because different site-specific exposure scenarios were evaluated at each location.

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⁻⁻Receptor and/or exposure pathway was not evaluated at this location.

1A child (0-6) was evaluated at the Child Development Center and the Residential Towers. An adolescent (6-12) was evaluated at the Elementary School.

Table 3-11 presents the results of segregating the hazard indices by target organ and/or critical effect. The segregated hazard indices were greater than the regulatory benchmark of 1, for respiratory effects, for every exposure scenario except the Recreational Golfer Scenario. Target organs/critical effects were identified for all constituents with a hazard quotient greater than 0.1. The methodology for grouping constituents into the respective target organ/critical effect categories is presented in Subsection 4.5 of Section 4.0 of the Pioneer Human Health Risk Assessment report (Pioneer 2000).

3.6 Risk Assessment Uncertainties

The risks presented in this assessment are conditional estimates based on a number of assumptions about exposure and toxicity, given a particular scenario. Uncertainties are created in a risk assessment because a range of values could be used for each assumption (i.e., parameter). Consistent with USEPA policy, more conservative (i.e., upper bound) values are generally chosen for each parameter, while other values (i.e., values closer to the central tendency) may be more representative of site-specific conditions (EPA 1989). Choosing upper bound values for each parameter typically results in overly conservative risks that do not reflect site-specific conditions.

Sources of uncertainty are normally associated with data collection and evaluation, exposure assessment, toxicity values and risk characterization. A qualitative discussion regarding the various uncertainties in the risk assessment follows. Table 3-12 addresses the sources of uncertainty and professional judgment regarding the direction and the magnitude of these uncertainties.

Uncertainties in Data Collection and Evaluation

Because this study was designed to support risk management decisions regarding the health risks at NAF Atsugi, it was critical to reduce, as much as possible, the uncertainties regarding data collection and evaluation. Most of these uncertainties regarding identification of COCs present in soil and ambient air at NAF Atsugi in the sampling methodology were reduced using site-specific information to develop the sampling work plan and focus sampling efforts. By collecting samples with sufficiently low detection limits to perform health-based risk analysis and ensuring that the number and frequency of samples collected was statistically

Table 3-11 — Summary of Hazard Indices by Target Organ/Critical Effect at Each Location

Target Organ/	Scenario	Dogonton	Exposure	Hazard Index	Hazard Index
Critical Effect	Scenario	Receptor	Duration	Average	RME
		Child Development Ce	nter		
Body Weight	Day Care	Child (0-6)	3 or 6 Years	1.4	1.9
Dermal/Ocular	Day Care	Child (0-6)	3 or 6 Years	1.2	1.5
Developmental	Day Care	Child (0-6)	3 or 6 Years	0.08	0.08
Hematological	Day Care	Child (0-6)	3 or 6 Years	0.08	0.08
Immunotoxicity	Day Care	Child (0-6)	3 or 6 Years	0.08	0.08
Kidney	Day Care	Child (0-6)	3 or 6 Years	0.06	0.07
Liver	Day Care	Child (0-6)	3 or 6 Years	0.06	0.07
Neurotoxicity	Day Care	Child (0-6)	3 or 6 Years	0.14	0.15
Not Classifiable – NOAEL	Day Care	Child (0-6)	3 or 6 Years	21.5	30.0
Not Provided	Day Care	Child (0-6)	3 or 6 Years	0.51	0.59
Reproductive	Day Care	Child (0-6)	3 or 6 Years	1.2	1.3
Respiratory	Day Care	Child (0-6)	3 or 6 Years	27.8	38.7
Body Weight	Day Care	Adult Care Provider	3 or 6 Years	0.31	0.43
Dermal/Ocular	Day Care	Adult Care Provider	3 or 6 Years	0.28	0.35
Developmental	Day Care	Adult Care Provider	3 or 6 Years	0.02	0.02
Hematological	Day Care	Adult Care Provider	3 or 6 Years	0.02	0.02
Immunotoxicity	Day Care	Adult Care Provider	3 or 6 Years	0.02	0.02
Kidney	Day Care	Adult Care Provider	3 or 6 Years	0.01	0.02
Liver	Day Care	Adult Care Provider	3 or 6 Years	0.01	0.02
Neurotoxicity	Day Care	Adult Care Provider	3 or 6 Years	0.03	0.04
Not Classifiable – NOAEL	Day Care	Adult Care Provider	3 or 6 Years	4.9	6.8
Not Provided	Day Care	Adult Care Provider	3 or 6 Years	0.12	0.13
Reproductive	Day Care	Adult Care Provider	3 or 6 Years	0.27	0.30
Respiratory	Day Care	Adult Care Provider	3 or 6 Years	6.3	8.8
		Elementary School	I		
Body Weight	Elementary School	Adolescent (6-12)	3 or 6 Years	0.42	0.58
Dermal/Ocular	Elementary School	Adolescent (6-12)	3 or 6 Years	0.37	0.47
Developmental	Elementary School	Adolescent (6-12)	3 or 6 Years	0.02	0.03
Hematological	Elementary School	Adolescent (6-12)	3 or 6 Years	0.02	0.03
Immunotoxicity	Elementary School	Adolescent (6-12)	3 or 6 Years	0.02	0.03
Kidney	Elementary School	Adolescent (6-12)	3 or 6 Years	0.02	0.02
Liver	Elementary School	Adolescent (6-12)	3 or 6 Years	0.02	0.02
Neurotoxicity	Elementary School	Adolescent (6-12)	3 or 6 Years	0.04	0.05
Not Classifiable – NOAEL	Elementary School	Adolescent (6-12)	3 or 6 Years	6.5	9.1
Not Provided	Elementary School	Adolescent (6-12)	3 or 6 Years	0.16	0.18
Reproductive	Elementary School	Adolescent (6-12)	3 or 6 Years	0.36	0.41
Respiratory	Elementary School	Adolescent (6-12)	3 or 6 Years	8.4	11.8
Body Weight	Elementary School	Adult Teacher	3 or 6 Years	0.24	0.33
Dermal/Ocular	Elementary School	Adult Teacher	3 or 6 Years	0.21	0.27
Developmental	Elementary School	Adult Teacher	3 or 6 Years	0.01	0.01

Table 3-11 — Summary of Hazard Indices by Target Organ/Critical Effect at Each Location

Target Organ/	Campuia	Document	Exposure	Hazard Index	Hazard Index
Critical Effect	Scenario	Receptor	Duration	Average	RME
Hematological	Elementary School	Adult Teacher	3 or 6 Years	0.01	0.01
Immunotoxicity	Elementary School	Adult Teacher	3 or 6 Years	0.01	0.01
Kidney	Elementary School	Adult Teacher	3 or 6 Years	0.01	0.01
Liver	Elementary School	Adult Teacher	3 or 6 Years	0.01	0.01
Neurotoxicity	Elementary School	Adult Teacher	3 or 6 Years	0.02	0.03
Not Classifiable – NOAEL	Elementary School	Adult Teacher	3 or 6 Years	3.7	5.2
Not Provided	Elementary School	Adult Teacher	3 or 6 Years	0.09	0.10
Reproductive	Elementary School	Adult Teacher	3 or 6 Years	0.20	0.23
Respiratory	Elementary School	Adult Teacher	3 or 6 Years	4.8	6.7
		Residential Tower	·s		
Body Weight	Residential	Child (0-6)	3 or 6 Years	2.4	3.4
Dermal/Ocular	Residential	Child (0-6)	3 or 6 Years	2.1	2.7
Developmental	Residential	Child (0-6)	3 or 6 Years	0.15	0.17
Hematological	Residential	Child (0-6)	3 or 6 Years	0.15	0.17
Immunotoxicity	Residential	Child (0-6)	3 or 6 Years	0.15	0.17
Kidney	Residential	Child (0-6)	3 or 6 Years	0.21	0.29
Liver	Residential	Child (0-6)	3 or 6 Years	0.21	0.29
Neurotoxicity	Residential	Child (0-6)	3 or 6 Years	0.36	0.45
Not Classifiable – NOAEL	Residential	Child (0-6)	3 or 6 Years	35.8	43.4
Not Provided	Residential	Child (0-6)	3 or 6 Years	1.2	1.4
Reproductive	Residential	Child (0-6)	3 or 6 Years	1.4	1.5
Respiratory	Residential	Child (0-6)	3 or 6 Years	48.6	62.4
Body Weight	Residential	Adult	3 or 6 Years	0.87	1.2
Dermal/Ocular	Residential	Adult	3 or 6 Years	0.75	0.98
Developmental	Residential	Adult	3 or 6 Years	0.05	0.06
Hematological	Residential	Adult	3 or 6 Years	0.05	0.06
Immunotoxicity	Residential	Adult	3 or 6 Years	0.05	0.06
Kidney	Residential	Adult	3 or 6 Years	0.08	0.10
Liver	Residential	Adult	3 or 6 Years	0.08	0.10
Neurotoxicity	Residential	Adult	3 or 6 Years	0.13	0.16
Not Classifiable – NOAEL	Residential	Adult	3 or 6 Years	12.8	15.5
Not Provided	Residential	Adult	3 or 6 Years	0.44	0.51
Reproductive	Residential	Adult	3 or 6 Years	0.50	0.52
Respiratory	Residential	Adult	3 or 6 Years	17.3	22.3
Body Weight	Residential	Child & Adult	30 years	1.2	1.6
Dermal/Ocular	Residential	Child & Adult	30 years	1.0	1.3
Developmental	Residential	Child & Adult	30 years	0.07	0.08
Hematological	Residential	Child & Adult	30 years	0.07	0.08
Immunotoxicity	Residential	Child & Adult	30 years	0.07	0.08
Kidney	Residential	Child & Adult	30 years	0.10	0.14

Table 3-11 — Summary of Hazard Indices by Target Organ/Critical Effect at Each Location

Target Organ/			Exposure	Hazard Index	Hazard Index
Critical Effect	Scenario	Receptor	Duration	Average	RME
Liver	Residential	Child & Adult	30 years	0.10	0.14
Neurotoxicity	Residential	Child & Adult	30 years	0.18	0.22
Not Classifiable – NOAEL	Residential	Child & Adult	30 years	17.4	21.1
Not Provided	Residential	Child & Adult	30 years	0.60	0.69
Reproductive	Residential	Child & Adult	30 years	0.68	0.71
Respiratory	Residential	Child & Adult	30 years	23.6	30.3
	'	Golf Course	1		
Body Weight	Recreational Golfer	Adult	3, 6, or 30 Years	0.02	0.02
Dermal/Ocular	Recreational Golfer	Adult	3, 6, or 30 Years	0.01	0.02
Developmental	Recreational Golfer	Adult	3, 6, or 30 Years	0.001	0.002
Hematological	Recreational Golfer	Adult	3, 6, or 30 Years	0.001	0.002
Immunotoxicity	Recreational Golfer	Adult	3, 6, or 30 Years	0.001	0.002
Kidney	Recreational Golfer	Adult	3, 6, or 30 Years	0.001	0.001
Liver	Recreational Golfer	Adult	3, 6, or 30 Years	0.001	0.001
Neurotoxicity	Recreational Golfer	Adult	3, 6, or 30 Years	0.003	0.003
Not Classifiable – NOAEL	Recreational Golfer	Adult	3, 6, or 30 Years	0.38	0.49
Not Provided	Recreational Golfer	Adult	3, 6, or 30 Years	0.010	0.01
Reproductive	Recreational Golfer	Adult	3, 6, or 30 Years	0.02	0.02
Respiratory	Recreational Golfer	Adult	3, 6, or 30 Years	0.50	0.65
	'	GEMB	1		
Body Weight	Military/Industrial Worker	Adult	3, 6, or 30 Years	0.30	0.42
Dermal/Ocular	Military/IndustrialWorker	Adult	3, 6, or 30 Years	0.26	0.33
Developmental	Military/Industrial Worker	Adult	3, 6, or 30 Years	0.02	0.02
Hematological	Military/Industrial Worker	Adult	3, 6, or 30 Years	0.02	0.02
Immunotoxicity	Military/Industrial Worker	Adult	3, 6, or 30 Years	0.02	0.02
Kidney	Military/Industrial Worker	Adult	3, 6, or 30 Years	0.02	0.02
Liver	Military/Industrial Worker	Adult	3, 6, or 30 Years	0.02	0.02
Neurotoxicity	Military/Industrial Worker	Adult	3, 6, or 30 Years	0.04	0.04
Not Classifiable – NOAEL	Military/Industrial Worker	Adult	3, 6, or 30 Years	5.5	8.3
Not Provided	Military/Industrial Worker	Adult	3, 6, or 30 Years	0.13	0.14
Reproductive	Military/Industrial Worker	Adult	3, 6, or 30 Years	0.32	0.39
Respiratory	Military/Industrial Worker	Adult	3, 6, or 30 Years	7.4	11.3

Table 3-12 — Summary of Uncertainties in the Human Health Evaluation and Site-Specific Characteristics

Source of Uncertainty	Direction	Magnitude	Comment
		Data Evaluation	
Identification of COCs present in soil at the Base	+/-	0	Used site-specific information to develop the sampling work plans and focus sampling efforts.
Identification of COCs present in ambient air at the Base	+/-	0	Used site-specific information to develop sampling work plans and focus sampling efforts. Samples were collected over a 14-month time frame to reflect temporal and seasonal changes in weather patterns at the base.
Identification of COCs present in indoor air at the Base	+/-	2	Used ambient air concentrations as surrogate indoor air concentrations for quantitative evaluation in the risk assessment. This may result in an underestimation or overestimation of the risks.
Identification of COCs present in indoor dust at the Base	+/-	2	The indoor dust samples were only analyzed for dioxins/furans. Used soil concentrations as surrogate indoor dust concentrations for all other COCs. This may result in an underestimation or overestimation of the risks.
Treatment of soil sample duplicates as discrete values	+/-	0	Assumed that since the risk contribution was >80% from air, double weighting caused by the use of soil sample field duplicates as discrete samples did not contribute a significant change in the total risk.
Quality of Analytical Data	+/-	0	Used quality-assured data in evaluation.
		Exposure Assessme	nt
No attenuation or enrichment of COC concentrations in soil or indoor dust occurred over time	+/-	2	Assumed that no attenuation or enrichment of soil concentrations occurred over time. This may have resulted in an underestimation or overestimation of the risks.
Exposure Assumptions	+/-	2	Used site-specific and U.S. EPA Standard Default Exposure Factors in the evaluation.
Experimental Dermal Absorption Rates	+/-	2	Used experimentally derived dermal absorption rates to evaluate dermal contact with soil
		Toxicity Assessmen	
Failure to include all COCs because of lack of U.S. EPA approved toxicity values	-	3	May have resulted in an underestimation of the risks. Oral RfDs were available for 95 of the 246 COCs and Inhalation RfDs were available for 48 of the 246 COCs. Oral slope factors were available for 44 of the 246 COCs and inhalation slope factors were available for 43 of the 246 COCs.
Extrapolation from animal studies to human toxicity	+	3	Used U.S. EPA's conservative approach incorporating safety factors and upper-bound estimates
Lack of COC-specific dermal toxicity values	-	1	Used oral toxicity values as surrogates for dermal toxicity values in order to evaluate risks associated with dermal exposure. This may have resulted in an underestimation of the risks.

Table 3-12 — Summary of Uncertainties in the Human Health Evaluation and Site-Specific Characteristics

Source of Uncertainty	Direction	Magnitude	Comment
Using dose-response	-	1	This may have underestimated the risks.
information from			
homogeneous animal			
populations or healthy			
human populations to predict			
effects that may have occurred			
in the general population,			
including sensitive			
subpopulations.			
		Calculation of Risk	T.S.
Assumed that health effects of	+/-	3	Assumed that health effects of COCs are
COCs were additive.			additive in risk calculations. Antagonistic and
			synergistic effects of COC mixtures were not
			evaluated.

Direction of Effect on Risk Calculations

- + = May result in risks that were overly conservative

Magnitude of Effect on Risk Calculations

- = May result in risks that were not conservative 0 = Negligible impact on risk calculations
- 1 = Small effect on risks calculations 2 = Medium effect on risk calculations

3 = Large effect on risk calculations Direction and Magnitude values based on professional judgment

determined, it was possible to minimize the possibility of over- or underestimating the health risks associated with the identification of COCs in ambient air and soil. The sampling frequency of every 6 to 12 days for more than one year guaranteed that each day of the week, as well as each week of the year, would be represented to account for any variability due to the day of the week, season, or other temporal effects. There was some uncertainty with the use of ambient air as a surrogate for indoor air concentrations.

Uncertainties in the Exposure Assessment

Standard uncertainties associated with exposure assessment are the use of site specific and USEPA standard default assumptions regarding age, body weight, period exposed, life expectancy, population characteristics, and intensity and amount of activity in lifestyle, which may not be representative of the exposed population. Therefore, the risk can be under- or overestimated. The amount of media (air, soil, water, etc.) intake is assumed to be constant and representative of the exposed population. This has a moderate potential to overestimate the exposure by one to two orders of magnitude.

Since it was assumed that no attenuation or enrichment of constituent concentrations in soil or indoor dust occurred overtime, this might have resulted in an underestimation or overestimation of the risk of 2 orders of magnitude. Likewise, the use of dermal absorption rates may have under- or overestimated the risk by two orders of magnitude.

Uncertainties in the Toxicity Assessment

There were several standard uncertainties inherent to a toxicity assessment that may under- or over estimate risk. These include the lack of toxicity values for some chemicals, which prevents their consideration in the risk assessment and may have resulted in an underestimation of risk. In this HRA, since initially oral RfDs were only available for 95 of the 246 COCs and inhalation RfDs were only available for 48 of the 246 chemicals of concern, the non-carcinogenic risks were underestimated. Likewise, since oral slope factors were initially available for only 44 of the 246 COCs and inhalation slope factors were available for only 43 of the 246 COC, this may have resulted in an underestimation of the cancer risk.

For the toxicity values that were available, there was another standard uncertainty associated with cancer slope factors and reference doses in the use of USEPA's conservative

approach. The EPA approach incorporates uncertainty factors of 10 to 1000 and upper-bound estimates on toxicity values to account for the extrapolation among animal species and from animal to human, as well as extrapolations from high to low doses. The toxicity value that is generally used to calculate non-cancer risks is a reference dose (RfD) or reference concentration (RfC). The RfD or RfC, for a specific chemical, is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily oral exposure to the human population (including sensitive subgroups) of that chemical that is likely to be without an appreciable risk. It can be derived from a no-observed-adverse-effect level (NOAEL), a lowest-observed-adverse effect level (LOAEL), or benchmark dose, with uncertainty factors generally applied to reflect the limitations of the data used. The RfC/RfD is derived by the consistent application of uncertainty factors (UFs) to account for recognized uncertainties in the extrapolation from the experimental data and exposure conditions to an estimate appropriate to the assumed human lifetime exposure scenario. The UFs generally consist of multiples of 10 (although values less than 10 are sometimes used) with each factor representing a specific area of uncertainty inherent in the extrapolation from the available data.

- A UF of 10 is used to account for variation in the general population to protect sensitive subpopulations (e.g. elderly, children).
- A UF of 10 is used when extrapolating from animals to humans to account for interspecies variability.
- A UF of 10 is used when a NOAEL derived from a subchronic instead of a chronic study is used as the basis for a chronic RfD.
- A UF of 10 is used when a LOAEL is used instead of a NOAEL to account for the uncertainty associated with extrapolating from the LOAELs to the NOAELs.
- Other chemical–specific uncertainty factors (modifying factors) may also be applied for individual chemicals depending on the existing health effects data set. Therefore, the UFs that are generally applied range from a factor of three to an order of magnitude. The composite UF depends on the number of extrapolations required. RfCs have been derived using composite UFs that range from 10 to 3000, with most RfCs using factors of 100 to 1000.

The greater the UF (i.e., the more individual UFs that were combined to get the total UF), the more conservatism is included. The precision of "an order of magnitude" should be applied on the average. There is less precision implied in the case of an RfC with overall UF of 1000 and more precision implied for RfCs with lower overall Ufs, such as less than 100. The relative precision and the magnitude of the composite UFs must be considered in decisions involving comparisons of HQ for different chemicals as in the assessment of the HI for a mixture of chemicals.

The lack of chemical specific dermal toxicity values leads to the use of oral toxicity values as surrogates for dermal toxicity values to evaluate risks associated with dermal exposure, and may result in an underestimate of the risk. The use of dose-response information from homogeneous animal populations or healthy human populations to predict effects that may occur in the general population, including sensitive populations, may also underestimate the risk.

Uncertainties in the Calculations of Risks

Standard uncertainty in risk characterization results from the assumption that health effects of chemicals are additive in risk calculations. Little is known about the effects of exposure to more than one chemical at a time. Exposure to multiple chemicals may be additive, synergistic (the effect of both is greater than if they were added) or antagonistic (the effect of both is less than expected if they were added together). To be protective, the risk from various chemicals are generally added in risk assessments; however, this assumption can result in under-or overestimating the risk.

3.7 Impacts of Emissions from the SIC on the Risk Estimates

The "Upwind" versus "Downwind" approach was selected to quantify the contribution of emissions from the SIC to the risk estimates. This effort involved comparing the risks at a site that was downwind of the SIC (i.e., the GEMB) with the risks for a site that was upwind of the SIC (i.e., the Golf Course) on the same days. For the purposes of determining impacts from the SIC, only the risks from inhalation of ambient air at the GEMB and the Golf Course were calculated using identical exposure parameters (i.e., a 3, 6, and 30-year child and adult residential exposure scenario). The only difference between the exposure factors used to calculate the risks at both sites was the exposure point concentrations. Table 3-13 presents a

comparison of the risks calculated at the GEMB and Golf Course under the "Downwind" and "Upwind" evaluation.

Table 3-13 — Comparison of Downwind Versus Upwind Risks at NAF Atsugi, Japan

	Hazard Index		Canc	er Risk
	Average	RME	Average	RME
Child	(0-6) - 3-Year Res	sidential Exposu	re	
GEMB – Downwind	133	265	1.8E-04	3.2E-04
Golf Course – Upwind	65	100	4.8E-05	8.5E-05
Potential Incremental Risk	50	137	1.3E-04	2.4E-04
Attributable to Emissions from the SIC				
Child	(0-6) – 6-Year Res	sidential Exposu	re	
GEMB – Downwind	133	265	3.7E-04	6.4E-04
Golf Course – Upwind	65	100	9.6E-05	1.7E-04
Potential Incremental Risk	50	137	2.7E-04	4.7E-04
Attributable to Emissions from the SIC				
Adi	ult – 3-Year Reside	ential Exposure		
GEMB – Downwind	47	95	6.5E-05	1.1E-04
Golf Course – Upwind	23	36	1.7E-05	3.1E-05
Potential Incremental Risk	18	49	4.8E-05	7.9E-05
Attributable to Emissions from the SIC				
Adi	ult – 6-Year Reside	ential Exposure		
GEMB – Downwind	47	95	1.3E-04	2.3E-04
Golf Course – Upwind	23	36	3.4E-05	6.1E-05
Potential Incremental Risk	18	49	9.6E-05	1.7E-04
Attributable to Emissions from the SIC				
Integrated Chi	ld and Adult – 30-	Year Residential	Exposure	
GEMB – Downwind	64	129	8.9E-04	1.5E-03
Golf Course – Upwind	31	49	2.3E-04	4.2E-04
Potential Incremental Risk	24	67	6.6E-04	1.1E-03
Attributable to Emissions from the SIC				

Notes: Ambient air samples at the GEMB were samples collected at wind conditions greater than or equal to 81% downwind of the SIC. Ambient air samples at the Golf Course were samples collected at wind conditions less than or equal to 4% downwind of the SIC.

The non-carcinogenic hazard indices were significantly higher, two to four times higher at the downwind location (i.e., GEMB) than the upwind location (i.e., Golf Course). The carcinogenic risks were also significantly higher, one and one-half orders of magnitude higher at the downwind location (i.e., GEMB) than the upwind location (i.e., Golf Course).

Section 4 — Health Risk Evaluation

The purpose of this section is to interpret and provide a context for presenting the results of the risk assessment, to discuss health concerns associated with the risk and to further characterize the estimated health risks based upon the sampling and risk analysis performed at NAF Atsugi. This section specifically discusses the nature of the health risks, estimation of their likelihood in various exposed populations and the strength of evidence and uncertainty associated with the health risks. It places the cancer and non-cancer risks from various air pollutants in perspective, identifies chemicals that may cause acute health effects, addresses other chemicals of concern, including dioxin, lead, and respirable particulates.

4.1 Cancer Risk and Non-Cancer Health Effects

Cancer Risk Evaluation

Excess lifetime cancer risk is calculated by multiplying an intake daily dose estimate times a cancer slope factor. The value that results is an expression of the probability that exposure to a chemical will lead to excess cancer risk. This value could be as low as zero. The value is expressed as a population risk, such as 1E-04, which means that no more than 1 in 10,000 exposed persons are expected to develop cancer in addition to what is already expected in the general population. To determine total cancer risk, individual cancer risks have been summed. This is consistent with the National Academy of Sciences (NAS) recommendations for risk assessment.

Table 3-8 in Section 3 indicates the total average and reasonable maximum cancer risk for each exposure scenario evaluated. As can be seen by the shaded areas, the reasonable maximum cancer risks for children residing at NAF Atsugi, for three and six years, were above the USEPA upper limit of acceptable cancer risk of 1 in 10,000 (1E-04) additional cases of cancer. Cancer risks for adults living at NAF Atsugi for the same time periods were an order of magnitude lower. Cancer risks were calculated for 30 years to compare with an USEPA residential default scenario. The other calculated cancer risks, which were above the USEPA acceptable risk range, were for the 30-year resident and the 30-year GEMB worker. As a result of the much shorter duration that most U.S. military and civilian personnel and their families spent at NAF Atsugi, the 30-year risks does not apply to most U.S. military and their families,

but applies to the American and Japanese civilian workforce and military that worked and lived at NAF Atsugi for extended and multiple tours of duty.

When the results of a human health cancer risk assessment fall in the range of 1E-06 to 1E-04 (which equals 1 in 1,000,000 to 1 in 10,000 additional cancer cases per lifetime, respectively), the USEPA typically considers using additional activities including regulations, to reduce the risk, particularly if the results are close to or greater than 1E-04. The results of this risk assessment suggested that a child's exposure to contaminants from air and soil during a 3-year tour of duty could potentially result in an upper bound lifetime estimate of risk at the 1E-04 level, but that adults would not likely reach this level, even with two tours of duty (6 years of exposure).

Adult Cancer Risk

Table 4-1 shows the chemicals which contributed to the vast majority of the reasonable maximum exposure cancer health risk for adults stationed at NAF Atsugi for 6 years (or 2 tours of duty) at each of the five sites monitored. This table indicates that 17 different chemicals each contributed at least 1% of the cancer risk.

The percentages in Table 4-1 indicate that the contribution that a specific chemical contributed to the overall risk was site-dependent. For example, at the GEMB and Golf Course, total 2,3,7,8-TCDD TEQ comprised the greatest contribution of risk at 26.8% and 52.5%, respectively. The second and third ranked chemicals contributing to risks at these two locations were 1,3-butadiene and acetaldehyde. At the Residential Towers, hexachlorobutadiene contributed the highest percent of cancer risk (27.2%), followed by 1,3-butadiene (14.1%) and acetaldehyde (9.5%). At the CDC and Elementary School, 1,3 butadiene contributed most to cancer risk (22.2% and 20.7%, respectively), but the second and third contributors at the Elementary School were total 2,3,7.8-TCDD TEQ and acetaldehyde, respectively. At the CDC, the second and third contributors were acetaldehyde and total 2,3,7,8-TCDD TEQ, respectively.

COC Elementary Residential Golf Course Child Ground Development School, Towers, Electronics Building 993 Building 3101 Center, Maintenance, Building 2910 Building 1061 Acetaldehyde 14.6 9.5 8.2 14.3 15.6 Acrylonitrile 2.9 2.7 2.4 1.6 2.6 1.5 1.1 2.0 Arsenic 1.6 1.0 Benzene 5.3 3.4 2.7 3.8 5.6 Benzyl Chloride 8.6 1.4 1.3-Butadiene 22.2 20.7 14.1 14.8 18.4 Cadmium 0.6 1.0 0.3 6.2 0.6 Carbon Tetrachloride 1.5 1.5 1.1 0.8 1.1 7.0 1.9 2.9 5.5 1,2-Dibromoethane 6.5 1,4-Dichlorobenzene 2.9 2.4 1.4 2.1 3.1 Methylene Chloride 0.8 0.8 0.7 1.3 0.4 0.9 1,4-Dioxane 1.6 1.5 1.1 0.5 Formaldehyde 8.1 7.6 4.5 2.5 4.7 5.9 27.2 2.9 Hexachlorobutadiene 6.3 2.7 4.5 2.2 1,1,2,2-Tetrachloroethane 4.8 3.0 3.6 Total 2,3,7,8-TCDD 11.0 16.7 11.9 52.5 26.8 **TEOs** Vinyl Chloride 1.8 1.6 1.2 0.7 1.2 2.5 5.5 6.7 4.3 4.1 All other constituents 100% 100% 100% 100% 100% Total Percentage

Table 4-1 — Percentage Contribution by COCs to the Adult Carcinogenic Risk at Each Location

Notes: Percentages were calculated based on an RME adult stationed at NAF Atsugi for 6-years (i.e., 2 tours of duty). Also, the risks were not directly comparable between AOCs because different site-specific exposure scenarios were evaluated at each location.

7.4E-05

2.5E-06

2.3E-05

1.2E-05

1.4E-05

Total Carcinogenic Risk

Child Cancer Risk

Table 4-2 provides the percentage that specific chemicals contributed to the cancer risk for children who resided at NAF Atsugi, attended Elementary School or attended the Child Development Center for three years.

The overall contribution for a child for certain chemicals was similar to the contribution for adults. For example, the three chemicals that contributed most to the adult cancer risk were the same three chemicals that contributed most to the child cancer risk. However, the order of contribution in which they are ranked was somewhat different for the child versus the adult. Since children's risks associated with soil were higher than for adults, the risk contribution due to chemicals found in soil were higher for children. Some of the chemicals, such as 2,3,7,8-TCDD TEQ were measured in soil and some such as 1,3-butadiene and acetaldehyde were not. Therefore, the percentages of risk for children, for those chemicals found in soil, were higher.

^{-- =} Not a COC for this location

COC	Child Development	Elementary School,	Residential Towers, Building.
	Center, Building 2910	Building 993	3101 – Residence
Acetaldehyde	14.9	13.7	8.9
Acrylonitrile	2.7	2.5	2.2
Arsenic	1.5	1.4	1.0
Benzene	5.4	4.9	3.2
Benzyl Chloride			8.1
1,3-Butadiene	21.1	19.4	13.2
Carbon Tetrachloride	1.5	1.4	1.0
1,2-Dibromoethane	6.7	6.1	1.8
1,4-Dichlorobenzene	3.0	2.7	2.3
1,1-Dichloroethene	1.1	1.0	0.7
1,4-Dioxane	1.5	1.4	1.0
Formaldehyde	7.7	7.1	4.2
Hexachlorobutadiene	6.0	5.5	25.4
1,1,2,2-Tetrachloroethane	4.6	4.2	3.3
Total 2,3,7,8-TCDD	15.2	21.6	17.4
TEQs			
Vinyl Chloride	1.7	1.5	1.1
All other constituents	5.5	6.5	5.9
Total Percentage	100%	100%	100%
Total Carcinogenic Risk	3.4E-05	1.1E-05	1.1E-04

Table 4-2 — Percentage Contribution by COCs to the Child Carcinogenic Risk at Each Location

Notes: Percentages were calculated based on an RME child stationed at NAF Atsugi for 3-years (i.e., 1 tour of duty). Also, the risks were not directly comparable between AOCs because different site-specific exposure scenarios were evaluated at each location.

Non-Cancer Health Effects Evaluation

Non-cancer risk is determined by comparing daily intakes of a chemical of concern with a toxicity value, called a reference dose (RfD) or reference concentration (RfC). These reference values are daily doses that even sensitive populations can receive without developing an appreciable risk of adverse health effects during a lifetime. The ratio of this comparison for each individual chemical is called a hazard quotient. Hazard quotients are generally summed in a risk assessment to provide a cumulative value of non-cancer health risks referred to as a Hazard Index (HI). Concentrations generating a HI above 1 indicate that there may be concern for non-cancer health effects. The HI assumes that there is a level of exposure below which it is unlikely that even sensitive persons will experience adverse health effects. This level of exposure is a Reference Dose (RfD) generally considered to have uncertainty spanning one to five orders of magnitude. A HI of 1 should not be considered a bright line, which automatically triggers remedial action, but rather indicates some degree of concern and the need for professional judgment following further evaluation. A HI greater than 1, indicates an increasing risk for

^{-- =} Not a CDC for their location

developing some type of adverse non-cancer health effect. Non-cancer risk characterization generally does not involve quantitative predictions of how much someone's risk of adverse effects is increased when exposure exceeds a certain reference dose. For example, an HI of 20 does not mean that there is 20 times more likelihood of a non-cancer effect to occur, but rather as the number increases there is some level of additional concern that it will occur. Exposures above an HI of 1 do not necessarily imply unacceptable risk or that adverse health effects are expected. Because of the uncertainties inherent to the derivation of the RfCs/RfDs used to calculate the HI, the significance of exceedances must be evaluated on a case-by-case basis, considering such factors as the confidence level of the assessment, the size of the uncertainity factors (Ufs) used, the slope of the dose-response curve, the magnitude of the exceedence, and the number or types of people exposed at various levels above the RfD or RfC.

The Presidential/Congressional Commission on Risk Management and Risk Assessment evaluated the EPA risk assessment approach for assessing hazardous air pollutant sources to implement section 112 of the Clean Air Act. Since the 1990 amendments do not set a threshold for considering health risks other than cancer, the Commission has set a HI threshold of 10 in a screening risk assessment for identifying high priority source categories when determining and managing risk. They chose a threshold index of 10, instead of 1 because there are few hazardous air pollutants with inhalation RfCs that are within a factor of 10 of their NOAELs. Typically, RfCs are one-thousandth of a NOAEL, so a hazard index of 10 in these cases would still leave a margin of exposure of 100.

Table 3-8 in Section 3 indicates the total average and reasonable maximum hazard index for each exposure scenario evaluated. As can be seen by the shaded areas, the reasonable maximum hazard index was greater than 10 for all scenarios evaluated except for the adult teacher and the golfer. The average hazard index exceeded 10 for the adult and child resident and for children of 0-6 years of age who attended the Child Development Center .

Adult Non-cancer Health Effects

Table 4-3 indicates the chemicals, which make up the vast majority of the RME non-cancer health risk for adults. These figures indicate the specific chemicals that comprised at least 1% of the total HI. Among HI contributors, acrolein, acetaldehyde, formaldehyde and PM₁₀ are respiratory irritants. They comprised approximately an average of 91% of the non-cancer risk at

the CDC, Elementary School, Residential Towers, and GEMB; with the lowest being 85% at the Golf Course. The specific chemicals indicated on Table 4-3, making up the remainder (e.g., 9% to 15%) of the non-cancer risk for the various locations have various endpoints. Hazard indices are often segregated by target organ endpoints; particularly in the situations where it is questionable as to whether or not an HI would exceed unity for one target organ. If the HI for several chemicals together were above unity, but the individual hazard index for the various target organs were less than unity, non-cancer health effects are likely to be of no concern. There were no other chemicals that had a hazard quotient of 1 and no other endpoints for which a hazard index of one or greater could be calculated. This indicated that respiratory concerns were the non-cancer health effects that could be anticipated.

Table 4-3 — Percentage Contribution by COCs to the Adult Hazard Indices at Each Location

COC	Child Development Center, Building 2910	Elementary School, Building 993	Residential Towers, Building. 3101 - Residence	Golf Course	Ground Electronics Maintenance, Building 1061
Acetaldehyde	11.8	14.0	17.1	16.2	15.2
Acetonitrile	1.3	1.5	0.3	5.9	0.5
Acrolein	69.8	69.8	64.2	59.4	64.3
Aluminum				1.5	
1,2-Dibromoethane	2.8	2.8	1.5	2.6	2.6
Formaldehyde	3.7	3.7	4.1	2.5	2.6
PM_{10}	3.5	3.5	5.2	5.8	4.2
1,2,4-Trimethylbenzene	1.1	1.1	1.6	1.2	0.9
All other constituents	6.0	3.6	6.0	4.9	5.8
Total Percentage	100%	100%	100%	100%	100%
Total Hazard Index	9.5	7.2	24.0	0.8	12.8

Notes: Percentages were calculated based on an RME adult stationed at NAF Atsugi for 6-years (i.e., 2 tours of duty). Also, the risks were not directly comparable between AOCs because different site-specific exposure scenarios were evaluated at each location.

Data on the effects of exposure to a mixture of chemicals are generally not available; therefore, when two or more chemicals act on the same organ system, their combined effect should be considered as additive, unless there is evidence to the contrary. The health effects resulting from exposure to respiratory irritants such as acrolein, acetaldehyde, and formaldehyde are likely to be reversible; however, combination with other chemicals such as respirable particulates is cause for concern. These chemicals can adhere to fine particulates and potentially

^{-- =} Not a COC for this location.

result in deep lung penetration and extended/prolonged retention. Continuous impact of these types of particulates has the potential to result in irreversible effects.

Child Non-cancer Health Effects

Table 4-4 provides the percentage that specific chemicals contributed to the non-cancer risk for children that resided at NAF Atsugi, attended Elementary School or attended the Child Development Center for three years. As with the cancer risk, the overall contribution for the specific chemical was similar to the contribution for adults but the actual contribution for the specific chemicals that were measured in soil may be slightly higher than the contribution for that particular chemical for adults. The percentage of chemical contribution was the same as for adults, but the total Hazard Index at the same sites for children are much higher than for adults.

~ ~ ~		T =	T =
COC	Child Development Center	Elementary School	Residential Towers,
			Building 3101
Acetaldehyde	14.1	14.1	17.4
Acetonitrile	1.5	1.5	0.3
Acrolein	70.4	70.4	65.1
Aluminum			
1,2-Dibromoethane	2.8	2.8	1.6
Formaldehyde	3.7	3.7	4.1
PM_{10}	3.5	3.5	5.3
1,2,4-Trimethylbenzene	1.1	1.1	1.7
All other constituents	2.9	2.9	5.0
Total Percentage	100%	100%	100%
Total Hazard Index	42	13	66

Table 4-4 — Percentage Contribution by COCs to the Child Hazard Indices at Each Location

Notes: Percentages were calculated based on an RME child stationed at NAF Atsugi for 3-years (i.e., 1 tour of duty). Also, the risks were not directly comparable between AOCs because different site-specific exposure scenarios were evaluated at each location.

4.2 Health Effects of Chemicals Lacking Peer-reviewed Toxicity Values

Since toxicity values for 86 of the 246 chemicals of concern were not available from the secondary sources consulted (U.S. EPA Integrated Risk Information System, IRIS; U.S. EPA Health Effects Assessment Summary Tables, HEAST; USEPA National Center for Environmental Assessment, NCEA; or California EPA), an exhaustive search of all available scientific peer-reviewed databases was conducted to determine whether sufficient toxicological information exists to calculate an interim Inhalation Reference Dose (RfDi) or cancer slope factor (CSF). For those chemicals where it was determined that sufficient toxicological information for non-carcinogenic effects was available, an RfDi was estimated using the

^{-- =} Not a COC for this location.

identical toxicological methodology recommended by the National Academy of Sciences and later adopted by USEPA. This methodology is currently used to develop toxicity values that are presented in IRIS. A detailed description of this study of the 86 chemicals is presented in Appendix E and is entitled "Toxicological Evaluation of Chemicals of Concern at NAF Atsugi Lacking Known Toxicity Values".

Ten different U.S. regulatory or governmental agency databases and twenty toxicological databases were extensively queried, including the following: IRIS, EPA Region 9 Toxicity Information, California Office of Environmental Health Hazard (OEHHA)/Air Resources Board (ARB) Database, Cal EPA Department of Toxic Substances Control (DTSC), Occupational Safety and Health Administration (OSHA)/National Institute for Occupational Safety and Health (NIOSH), Carcinogenic Potency Database (CPDB), and the Registry of Toxic Effects of Chemical Substances (RTECS). Eight RfDi values were identified. Through literature searches of the National Library of Medicine (NLM), over 11,000 abstracts were first reviewed and over 300 of these were collected and further reviewed to identify useful quantitative toxicological information. Based on careful review of these published studies, it was possible to develop an interim RfDi for nine of the chemicals, a small subset of the 86 chemicals. This subset included the following chemicals: propionaldehyde, 1-hexene, butyl acrylate, cyclohexane, cyclopentene, ethanol, 1-butene, m-diethylbenzene, and n-nonane. There was no indication in any of the numerous databases analyzed or scientific papers reviewed that any of the 86 chemicals could be classified as a human carcinogen. That is, all 86 chemicals had a lack of toxicological information for assigning a toxicological classification other than Category D, Not Classifiable As To Human Carcinogenicity. A structure activity analysis was performed on the 86 chemicals and there was little justification for suspecting potential carcinogenic effects.

In the final analysis, there was insufficient toxicity information for most of the 86 chemicals in this study. The reason so few toxicological studies have been conducted on these particular chemicals is that their toxicological significance is of no consequence. That is, they likely possess low inherent toxicity. For that reason, the chemicals lacking toxicity values are best viewed as chemicals posing very low health hazards, especially at the low levels detected at NAF Atsugi. For those eight chemicals where toxicity values have been identified in EPA Region 9 Preliminary Remediation Goal (PRG)Tables (or other regulatory databases) or for the nine chemicals where toxicity values have been developed based on careful review of the

literature, the estimated health hazards are extremely low and can be considered *de minimus* (least, smallest, slightest). These interim RfDi values were used to estimate a Hazard Index for three areas at NAF Atsugi (Residential Towers, GEMB site, and the school site). With the exception of ethyl alcohol, all hazard quotients and indices were below 1.0. The sole reason the hazard quotient for ethyl alcohol exceeded one was high level of uncertainty in the toxicity database. It is highly unlikely that ethyl alcohol poses a real health hazard at NAF Atsugi at the concentrations detected.

Therefore, based on this evaluation, it was determined that the 86 chemicals do not pose unacceptable threats to human health at the concentrations detected in each of the three areas. Additional health risk cannot be attributed to these 86 chemicals.

4.3 Considerations for Children

Infants and children are qualitatively and quantitatively different from adults in their exposure to airborne contaminants and they are different in their susceptibility to harm from these exposures. As a result, the calculated carcinogenic and non-carcinogenic risks are greater for children, as documented in this report for the risks at NAF Atsugi. There are numerous factors that increase the exposure of infants and children to environmental toxicants and their susceptibility to adverse effects. Children have different diets than adults and they have activity patterns that change their exposure profile compared to adults. Crawling on the floor or ground, putting their hands and foreign objects in their mouths, and raising dust and dirt during active play all increase exposure potential in children (Manton et al, 2000). Being closer to the ground increases their exposure to some toxicants. In addition to exposure differences between children and adults, children are growing and developing, which makes them more susceptible to certain types of toxicants. Effects of early childhood exposures, including neurobehavioral effects and cancer, may not be apparent until later in life. Therefore, special considerations for children and infants have been included in the risk assessment process (Klaassen, 2001).

The risk calculations use different assumptions for children due to physiological and biochemical differences from adults. On a body weight basis, children breath more air than adults and have higher incidental rates of ingestion. Due to their behavior, they ingest more soil and dust than adults. Air intake of a resting infant is twice that of an adult. Since infants and children are smaller than adults, they get higher doses per body weight. Infants' metabolic

pathways may be immature and their ability to metabolize and excrete certain toxicants may be very different from an adult.

Outdoor air pollution in the U.S. and many places abroad, including Japan, have contributed to adverse health effects among the most susceptible members of society, infants and children in particular (Holland et al., 1979). Health problems from air pollution in children depend on predisposing health factors, such as asthma, the frequency and duration of the exposure, and the child's access to medical care. Adverse acute health effects from exposure to outdoor air pollution are primarily respiratory effects. Children with asthma have been found to be a sensitive sub-population for acute responses to outdoor air pollution (Clark et al, 1999). Exposure to elevated levels of air pollution was associated with decreased peak expiratory flow rates, increased respiratory symptoms, increased prevalence of school absence, and fever in asthmatic children (Peters et al, 1997). For children under 2 years of age, a 0.05 mg/m³ increase in PM₁₀ was associated with a 4-12 % increase in lower respiratory symptoms (Ostro et al, 1999). Chronic exposure has been associated with increased bronchitis and decreased pulmonary function in children. One study found an association between ambient pollutants and poorer gain of pulmonary volumes in preadolescent children living in more polluted areas (Jedrychowski et al, 1999). Other effects include the increase in hospitalizations that coincide with acute episodes of air pollution as measured by increases in ozone, PM₁₀ and PM_{2.5}, and sulfur dioxide.

Environmental toxins of particular concern for infants and children include heavy metals and dioxins, primarily due to their potential adverse neurobehavioral and developmental effects that have been well documented in the scientific and medical literature. Heavy metals, including lead, cadmium, and arsenic, are among the risk drivers at NAF Atsugi. The existence of high air pollution incidents caused *in utero* exposures to heavy metals that resulted in developmental disorders and behavioral impairments in children (Otto et al, 1997). Prenatal exposure to heavy metals, including cadmium and lead, resulted in adverse effects on childhood cognitive skills and illnesses (Lewis et al, 1992). Another study documents the differences in exposure and metabolic response of infants and adults to lead and cadmium (Mahaffey, 1983). The group of compounds known as dioxins are also among the primary risk drivers at NAF Atsugi. Due to the modulation of many different hormones and their receptors, dioxins are known to cause a spectrum of morphological and functional deficits that result in adverse developmental effects

that are irreversible. Children exposed prenatally to complex mixtures of dioxins and other endocrine disrupting chemicals may be smaller and have delayed puberty, hearing deficits, increased respiratory disease, and intelligence and behavioral deficits (Birnbaum, 1995). There might be effects on thyroid function in infants exposed to dioxin from breast milk (Yonemoto, 2000).

Due to concerns in regards to exposure of infants and children to heavy metals and dioxins, reproductive and developmental effects from all the chemicals of concern are discussed further in Section 4.8, Health Effects of Various Chemicals.

4.4 Risk Comparisons

Indoor Air

There is not an extensive database of comparable indoor air concentrations measured in the U.S. with which to compare the results of this risk assessment. In fact, this study at NAF Atsugi represents one of the most comprehensive studies of indoor air ever conducted.

In the early 1980s, the USEPA conducted a comprehensive study of personal exposure of 600 residents in seven U.S. cities to 20 target chemicals (EPA 1987). This study was called the Total Exposure Assessment Method (TEAM) study. This study was unique in its scope and methods, with high levels of quality control. It measured the target chemicals in ambient air, indoor air, exhaled breath and drinking water. The study found 11 of the 20 target chemicals to be prevalent in these personal exposures: (1) 1,1,1-trichloroethane, (2) m-and p-xylene, (3) benzene, (4) ethylbenzene, (5) perchloroethylene, (6) o-xylene, (7) m- and p- dichlorobenzene, (8) chloroform, (9) trichloroethylene, (10) carbon tetrachloride and (11) styrene, in decreasing order of geometric mean concentrations. The study found that mean personal air exposures to essentially every one of the 11 prevalent target chemicals were greater than mean outdoor concentrations (EPA 1987b).

In 1992, the USEPA compiled a national VOCs database for concentrations of organic compounds measured indoors (EPA 1992a). Based on a review of the literature from 1979 through 1990, including early data on VOC concentrations in residences, the database contains information on over 220 compounds. The California Air Resources Board (ARB) (Cal EPA 1998) has also developed a report including individual summaries of general exposure and health

effects information for the 243 substances included on the ARB Toxic Air Contaminant Identification List as part of the State of California's air toxics program. These summaries provide readily available information on the physical properties, sources and emissions, ambient concentrations, indoor sources and concentrations, atmospheric persistence, risk assessment information, and potential health effects of each substance, using information specific to California where possible. Both these databases cite mean values; therefore, the mean value reported at NAF Atsugi can be compared to these results. Measured concentrations from the Atsugi indoor air sites were compared to the results of various indoor air studies performed in the U.S. in Table 4-5. This comparison was used primarily as a point of reference for indoor air quality. Shading on table 4-5 shows whether indoor chemical air concentrations were higher in the U.S. studies or NAF Atsugi homes. The comparison indicates that, in general, indoor air concentration averages for most of the chemicals, for which data was available in the U.S. studies, were lower or within the same range at NAF Atsugi. Only butanol, 1,2 dibromoethane, 1.4 dioxane, styrene, di-n-butylphthalate and bis(2-ethyl hexyl)phthalate means were higher at NAF Atsugi than the California study. The higher concentrations of metals found in indoor air at NAF Atsugi, especially cadmium, arsenic and lead which are strongly associated with the SIC, could have been due to SIC particulate emissions deposited in soil and tracked indoors via shoes, as well as accumulation of indoor dust, originating from the SIC emissions transported indoors via open windows and doors.

Ambient Air

The results of the criteria pollutants monitoring as compared to cities in the U.S. are shown in Table 4-6. The comparison indicates that levels of carbon monoxide at NAF Atsugi were lower in comparison to U.S. cities and only slightly higher than Honolulu. The levels of nitrogen dioxide are similar to the levels found in Houston, Baltimore and Atlanta. Ozone levels at NAF Atsugi were considered elevated, but no more elevated than levels found in Atlanta and Baltimore. PM₁₀ levels at NAF Atsugi were 66 mg/m³ and were slightly elevated in comparison with 45 mg/m³ in Los Angeles, the highest level in the U.S. cities used as a comparison.

Table 4-5— Comparison of Indoor Air Concentrations between NAF Atsugi and U.S. Homes

Analyte	NAF Atsugi Mean	U.S. Studies Mean	Reference				
,	$(\mu g/m^3)$	$(\mu g/m^3)$					
	ALDEHYDES AND						
Acetaldehyde	12.749	5.4 - 27.0	Cal EPA 1998				
Acetone	32.506	39	EPA 1992				
Acrolein	0.745	7.1	Cal EPA 1998				
Benzaldehyde	3.062	17.8 - 20.7	EPA 1992				
Formaldehyde	26.154	12.3 - 615	Cal EPA 1998				
VOCs							
Butanol	15.5	3	EPA 1992				
1,1,1-Trichloroethane	0.835	14 - 24	Cal EPA 1998				
		2.8 - 48	EPA 1992				
1,1,2,2-Tetrachloroethane	0.356	2.27 - 6.72	Cal EPA 1998				
1,1-Dichloroethylene	0.149	< 0.2	Cal EPA 1998				
,		10.7 - 13	EPA 1992				
1,2,4-Trimethylbenzene	3.713	11	EPA 1992				
1,2-Dibromoethane	0.169	< 0.15	Cal EPA 1998				
1,2-Dichloroethane	0.457	< 0.8	Cal EPA 1998				
,		12.3	EPA 1992				
1,2,3- Trimethylbenzene	0.86	3.5	EPA 1992				
1,3,5-Trimethylbenzene	1.088	1.4 - 13	EPA 1992				
1,3-Butadiene	0.382	2.7 - 10	Cal EPA 1998				
1,4-Dioxane	0.938	0.15 - 0.53	Cal EPA 1998				
Acrylonitrile	0.376	9.1	Cal EPA 1998				
Benzene	3.923	0.28 - 16.4	Cal EPA 1998				
		1.6 - 52	EPA 1992				
Carbon Tetrachloride	0.616	0.63 - 1.37	Cal EPA 1998				
		0.2 - 45	EPA 1992				
Chlorobenzene	0.164	0.31	Cal EPA 1998				
Chloroform	0.538	1.9 – 43.9	EPA 1992				
Cumene	0.417	1.3 - 10.6	EPA 1992				
Cyclohexane	1.730	7.4	EPA 1992				
Ethylbenzene	10.723	5.02 - 6.64	Cal EPA 1998				
-		0.7 - 27	EPA 1992				
Methylene Chloride	95.392	83	Cal EPA 1998				
·		14.3 - 670	EPA 1992				
Styrene	2.724	0.72 - 2.4	Cal EPA 1998				
·		0.3 - 2.5	EPA 1992				
Tetrachloroethylene	1.319	2.27- 6.72	Cal EPA 1998				
		1.7 - 18	EPA 1992				
Toluene	28.788	27.2 - 96	EPA 1992				
Trichloroethylene	1.669	0.63 -3.97	Cal EPA 1998				
•		0.8 - 18	EPA 1992				
Vinyl Chloride	0.300	<40	Cal EPA 1998				
a-Pinene	1.423	1.2 - 102	EPA 1992				
n-Decane	5.543	8.9 - 14	EPA 1992				
n-Nonane	2.328	6.4 - 27	EPA 1992				
n-Octane	0.877	1.0 - 14	EPA 1992				
n-Undecane	3.270	1.1 - 12	EPA 1992				
o-Xylene	7.674	3.71 - 12.9	Cal EPA 1998				

Table 4-5— Comparison of Indoor Air Concentrations between NAF Atsugi and U.S. Homes

Analyte	NAF Atsugi Mean	U.S. Studies Mean	Reference					
	$(\mu g/m^3)$	$(\mu g/m^3)$						
p-Xylene + m-Xylene	15.838	9.3 - 33.6	Cal EPA 1998					
	PESTICIDE	S						
Heptachlor	0.004	0.072 - 0.16	Cal EPA 1998					
		0.01 - 1.8	EPA 1992					
alpha-BHC	0.001	0.22 - 0.32	Cal EPA 1998					
gamma-BHC	0.003	0.001 -0.02	Cal EPA 1998					
		0.02	EPA 1992					
gamma-Chlordane	0.001	0.22 - 0.32	Cal EPA 1998					
		0.14 - 5.8	EPA 1992					
	METALS							
Arsenic (PM ₁₀)	0.003	>90% samples ND	Cal EPA 1998					
Cadmium (PM ₁₀)	0.003	>90% samples ND	Cal EPA 1998					
Copper (PM ₁₀)	6.340	0.007 - 0.1	Cal EPA 1998					
Lead (PM ₁₀)	0.112	0.027	Cal EPA 1998					
Silver (PM ₁₀)	0.005	>90% samples ND	Cal EPA 1998					
Zinc (PM ₁₀)	0.280	0.038 - 0.36	Cal EPA 1998					
Mercury	0.109	>90% samples ND	Cal EPA 1998					
	SVOCs							
1,4-Dichlorobenzene	18.386	55	EPA 1992					
Di-n-butylphthalate	6.950	0.62	Cal EPA 1998					
bis(2-Ethylhexyl)phthalate	0.530	0.12	Cal EPA 1998					

Table 4-6 — Criteria Pollutant Data for Various Cities in the United States in Comparison to NAF Atsugi

	Los Angeles,	Atlanta,	Houston,	Norfolk,	Baltimore,	Honolulu,	NAF	NAQQS
Criteria Pollutant	CA	GA	TX	VA	MD	HI	Atsugi 1	
Carbon Monoxide								
2 nd Maximum 8-hour (ppm)	15	4	7	6	4	3	3.4	9
Nitrogen Dioxide								
Arithmetic Mean (ppm)	0.045	0.027	0.023	0.018	0.027	0.003	0.024	0.053
Ozone								
2 nd Maximum 1-hour (ppm)	0.20	0.13	0.18	0.1	0.13	0.05	0.137	0.125
PM10								
Arithmetic Mean (µg/m3)	45	29	40	21	29	19	66	50
Sulfur Dioxide								
Arithmetic Mean (ppm)	0.004	0.008	0.006	0.007	0.008	0.002	0.002	0.030
24-hour Maximum (ppm)	0.011	0.028	0.046	0.025	0.028	0.009	0.008	0.140

Source: National Air Quality and Trends Report, 1996 USEPA Office of Air and Radiation Data collected 21 April 1998 through 25 June 1999.

POLLUTANT JAPANESE STANDARD Carbon Monoxide (CO) 24-Hour Average 10 ppm 8-Hour Average 20 ppm Nitrogen Dioxide (NO₂) 24-Hour Average 40- 60 ppb Sulfur Dioxide (SO₂) 24-Hour Average 40 ppb 1-Hour Average 100 ppb Suspended Particulate Matter (SPM) (diameters less than or equal to 10 um) 0.10 mg/m^3 24-Hour Average 0.20 mg/m^3 1-Hour Average Photochemical oxidants 1-Hour Average 60 ppb

Table 4-7 — Summary of Japanese Air Quality Standards for the Criteria Pollutants

Source: www.env.go.jp/en/lar/regulation/aq.html on 24 April 2002 (Modified table format)

Japanese air quality standards for criteria pollutants are presented in table 4-7. There is no specific standard for ozone; however, there is a standard for photochemical oxidants, which is lower than the NAQQS standard for ozone. Although there is a standard for suspended particulate matter, there are no standards for PM₁₀ and PM_{2.5}. The only Japanese standards that are directly comparable with the U.S. standards are the 8-hour average standard for carbon monoxide, which is higher than the U.S. standard, and the 24- average standard for sulfur dioxide, which is lower than the U.S. standard. NAF Atsugi ambient air concentrations for carbon monoxide and sulfur dioxide did not exceed the Japanese air quality standards.

Table 4-8 presents concentrations of air pollutants measured in U.S. cities for comparison purposes. The table provides average or maximum concentrations for a number of organic chemicals (aldehydes, ketones, SVOCs and VOCs) and metals that have been measured at various locations in the U.S. (specific cities are noted), as well as the maximum and average concentrations of the same chemicals measured in ambient air at NAF Atsugi. Additionally, the table provides 25% and 75% daily quartile concentrations for some of the chemicals for which information regarding averages and maximums were not available. Although these numbers may not be directly comparable with the average and maximum concentration for the chemical that was measured at NAF Atsugi, they provide a frame of reference, particularly if the Atsugi average concentration was greater than the 75% quartile. Shading on Table 4-8 indicates that the chemical found at NAF Atsugi was at a higher average concentration than in the U.S.

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Table 4-8 — Air Pollutant Data for Various Cities in the United States Compared to NAF Atsugi

Pollutant	¹ Midlothian, Texas Data 1993-1995 Maximum 24-hour values	² U.S. 1994	³ U.S. Data 1994-1995 Average 24-hour Values	⁴ U.S. Data 1980-1988 25% and 75% Daily Quartiles	NAF Atsugi Mean Value	NAF Atsugi Maximum Value
Organics, μg/m³ unless o	therwise noted ¹					-
Acetaldehyde			2.7 (Baltimore, MD)	0 - 9.80	3.5	18.0
Acetone				0 - 6.72	6.3	28.3
Benzene	65.7		13.7 (Baltimore, MD) 28.4 (Houston, TX)	2.01 – 10.7	3.8	18.9
1,3-Butadiene	<1.4		20.4 (max) ⁵ 0.96 (mean) ⁵	0.34 – 1.49	0.52	5.9
Carbon tetrachloride	21.4			0.44 - 0.82	0.64	1.8
Chloroform	<3.1			0.05 - 0.88	0.25	1.3
Chloromethane				1.25 – 1.49	1.8	3.6
Dibromomethane				0 - 0.09	0.31	1.9
1,2-Dichloroethane	<2.1			0 - 0.22	0.33	1.1
1,2-Dichloropropane	2.0			0.05 - 0.30	0.33	1.4
1,4-Dioxane				0 - 0.14	1.5	10.2
Dioxin, TEQ		0.095 pg/m^3			1.6	39.4
Ethylbenzene	6.0		9.6 (Baltimore, MD) 13.5 (Houston, TX)			54.4
Formaldehyde			17.2 (max) ⁵ 5.0 (mean) ⁵	2.3 – 12.0	2.7	22.9
n-Hexane	9.6		33.8 (Houston, TX)		2.9	22.5
Hexachloro – butadiene				0.01 - 0.06	1.2	11.3
Methane				1050 – 1413		
Methylene chloride	1.4			1.09 - 6.28	11.2	690
Naphthalene				0.02 - 5.70	0.38	0.83
Styrene	<3.8		2.1 (Baltimore, MD) 0.69 6.0 (Houston, TX)		0.69	4.3
Trichloroethane	<1.0			0.70 - 3.33	0.67	4.3
Trichloroethylene	5.0				1.81	10.2

Pollutant	¹ Midlothian, Texas Data 1993-1995 Maximum 24-hour values	² U.S. 1994	³ U.S. Data 1994-1995 Average 24-hour Values	⁴ U.S. Data 1980-1988 25% and 75% Daily Quartiles	NAF Atsugi Mean Value	NAF Atsugi Maximum Value
1,2,4-Trimethylbenzene			62.8 (max) ⁵ 2.02 (mean) ⁵			
Vinyl chloride	<1.2			0 - 0.77	0.29	0.65
m-, p-Xylene	10.4		38.6 (Baltimore, MD) 40.4 (Houston, TX)		6.0	62.8
o-Xylene	7.8		16.5 (Baltimore, MD) 16.1 (Houston, TX)		2.4	24.5
Metals, μg/m ³						
Arsenic	< 0.004 - 0.003				0.0025	0.055
Beryllium	< 0.0008				0.0003	0.0012
Cadmium	< 0.02 - 0.014				0.0061	0.343
Chromium	< 0.02 - 0.058				0.0065	0.116
Lead	< 0.007 - 0.068				0.262	16.3
Nickel	<0.002 - 0.024				0.0065	0.140

NOTE: Data on this table has been altered from the references (e.g., all concentrations that were reported in units of ppbv have been converted to units of μg/m³.) Shaded rows represent those constituents where NAF Atsugi's mean value is greater than those values reported in the United States. Sources:

¹Texas Natural Resources Conservation Commission 1996. <u>Critical Evaluation of the Potential Impact of Emissions From Midlothian Industries: A Summary Report.</u>: Office of Air Quality/Toxicology and Risk Assessment Section. AS-71 (Revised). This report was referenced because the data represents an area with 3 cement kilns that burn waste derived fuel

²EPA 1994. Estimating Exposure to Dioxin Like Compounds. Volumes 1, 2, and 3. EPA/600/6-88/005Ca-c. June 1994. External Review Draft.

³EPA 1997. National Air Quality and Emissions Trends Report, 1996. Office of Air and Radiation.

⁴G₂ Environmental, Inc. National Ambient Volatile Organic Compounds (VOCs) Data Base Update. EPA 600/3-88/010(A). January 1988

⁵Data from U.S. EPA AIRS Database. Max Value represents maximum site mean value while mean value represents mean of all individual sites.

Table 4-8 indicates that some of the VOCs and aldehydes/ketones were higher in the U.S. and some were higher at NAF Atsugi. For some of the chemicals, the average was higher in the U.S. but the maximum concentration was higher at NAF Atsugi. The concentrations of aldehydes and ketones at NAF Atsugi appear to be somewhat greater than those in the U.S. With the exception of lead, the metals measured at NAF Atsugi were lower than concentrations found in Midlothian, Texas. Concentrations of dioxin and lead are clearly orders of magnitude greater at NAF Atsugi than in the U.S.

Incinerators in the U.S.

In the U.S., the USEPA has established stringent standards to regulate the operation of incineration facilities. The standards are based on the performance of maximum achievable control technology and implement section 112 of the Clean Air Act which regulates hazardous air pollutants, such as dioxins and furans, toxic organic compounds and mercury.

The operation of a hazardous waste incinerator in the U.S. involves issues such as air monitoring, stack monitoring, risk assessment, trial burns, fugitive emissions and waste characterization. A number of cases within the past six years have resulted in the review and/or modification of incineration facilities. Several incinerators were shut down or kept from being built as with the Vertac Chemical Incinerator in Jacksonville, Arkansas, and the Brio Refining Superfund Site near Houston, Texas. In other cases, incinerators were required to be upgraded, requiring engineering assessments, equipment replacement, additional studies, and/or the control of fugitive emissions as with the Times Beach Incinerator in Missouri, the Thermal Destruction Facility Drake Chemical in Lockhaven, Clinton County, Pennsylvania, the Shattuck Chemical Co. in Denver, Colorado, and the Stauffer Chemical Plant in Tarpon Springs, Florida (EPA 2000).

The Columbus Waste to Energy Facility, Ohio incinerator was shut down in the mid 1990s. It was considered to be the number one dioxin emitting trash incinerator. The average concentration of five dioxin measurements taken from the Columbus stack was more than 13,000 nanograms per cubic meter. This was 430 times higher than the amount the EPA was proposing for the burning of hazardous waste. Dioxin ambient air monitoring conducted over two 48 hour periods in March and April of 1994 showed a maximum ambient air monitored dioxin concentration of 0.352 picograms TEQ/m³ measured at a distance of approximately 2 kilometers

to the east of the Facility. In September 1994, finding that the facility "may present an imminent and substantial endangerment" to public health, the EPA ordered Columbus to stop running the facility until it completed a major overhaul of its pollution control system. On November 1, 1994, faced with having to spend at least \$65 million for the upgrades, the governing board for the plant voted unanimously to stop the burning of garbage in Columbus.

At NAF Atsugi dioxin ambient air monitoring was conducted every 6 days over 14 months from April 1998 to June 1999. An average ambient air concentration of 1.57 picograms TEQ/m³ was measured within a 1.5 Km radius from the SIC and an average ambient air concentration of 3.49 picograms TEQ/ m³ within 300 meters. By comparison with the Columbus Waste to Energy Facility, the SIC, an incinerator with uncontrolled emissions and high dioxin emission levels, would have been forced to either shut down or implement controls to come into compliance and mitigate the risk of health hazards caused by its uncontrolled

4.5 Base-Wide Risk Estimates

Calculated cancer and non-cancer risks for children and adults in all of the various scenarios presented in Table 3-2, were calculated as if the population of concern only spends time on base for the specific activity conducted (e.g., a child spends 8 hours at the Elementary School) and the exposure for the remainder of the hours of the day is zero. Given the fact that Japan is highly industrialized, it is readily apparent that the exposure for the remaining hours of the day will not be zero. However, the health risk assessment only addressed exposures on base because of the inherent sampling constraints presented by being in a foreign country. Many individuals do not remain on base after working, attending school or daycare. Therefore, calculated health risks provided on Table 4-9, only address their risks at each location as if they spent 24 hours at each location.

There are a myriad of other exposure scenarios that could occur for individuals or groups of individuals that routinely spend 24 hours a day at NAF Atsugi (e.g., resides on base and attends or works at the Elementary School, Child Development Center or works at the GEMB). To assist in determining the plausible upper bound inhalation risks for these individuals, Pioneer compared the air pathway health risk only, at each location, under identical exposure assumptions. This was performed for the air pathway only since the inhalation pathway is responsible for the majority of risk at each location (e.g., $\geq 85\%$). The results, presented in Table

4-9 indicate that health risks were similar throughout the areas monitored. Table 4-9 indicates that the cancer risk was similar at all locations for children. This was also true for adults. The hazard indices (HI) for children at the Child Development Center, the Elementary School and the Golf Course were the same; for the residential area it was slightly less and it was slightly more elevated at the GEMB. The same pattern was observed for adults. This table indicates that the plausible upper bound cancer risk estimate for the air pathway was similar at all locations on base. It further indicates that the risks for children and adults routinely spending 24 hours each day on base will be similar to the cancer (1.1E-05 for child and 3.7E-05 for adults) and noncancer risks (67 for child and 24 for adults) for an RME exposure of 3 years presented for the Residential Towers (see Table 3-8). The cancer and non-cancer risks calculated for inhalation pathway only for adults under identical exposure assumptions at the five sites included in the risk assessment were calculated using actual ambient air concentrations. These results are consistent with the dispersion modeling results conducted for the six chemicals (arsenic, cadmium, dioxins, HCl, lead and PM₁₀) associated with the SIC. Dispersion modeling used to assist in predicting the risk at other locations (e.g., other residential areas, the Bachelor Officers Quarters (BOQ), etc.) as a result of the SIC emissions only, predicted SIC impacts were similar at all

Table 4-9 — Comparison of Total Inhalation Risks Between AOCs Calculated Using Identical Exposure Assumptions

Location		velopment nter	Elementa	ry School	Residential Towers, Building 3101		Golf (Course	Mainte	lectronics enance ding
Receptor	Child	Adult	Child	Adult	Child	Adult	Child	Adult	Child	Adult
	(0-6)		(0-6)		(0-6)		(0-6)		(0-6)	
Hazard	94	34	94	34	66	24	93	33	113	40
Index										
Cancer	7.0E-05	2.5E-05	7.0E-05	2.5E-05	9.8E-05	3.5E-05	8.3E-05	3.0E-05	1.0E-04	3.6E-05
Risk										

Notes: Risks were calculated based on an RME child and adult stationed at NAF Atsugi for 3-years (1 tour of duty). The risks were calculated using identical exposure parameters and assuming a 24-hour exposure to ambient air at each location. The only difference between the calculations was the exposure parameters and assuming a 24-hour exposure to ambient air at each location.

residential areas. Therefore, it is reasonable to generalize that the risk attributed to SIC emissions was similar at all residential areas.

4.6 Contribution of Risk from the Shinkampo Incineration Complex

The Upwind-Dowwind approach assessed SIC contribution to risk by comparing the difference in risk between upwind and downwind monitoring sites. This comparison was made

when the prevailing wind toward the GEMB, the site having the greatest apparent impact from the SIC, was nearly 100% and the wind at an upwind site, the Golf Course, was approximately 0%, on the same day. There were few days in which these conditions were actually met. Therefore, the analysis itself has low power; however, it provides a good prediction of the contribution on those days. The results of that method indicated non-cancer risks downwind of the GEMB are approximately 2 to 4 times higher than risks upwind at the Golf Course on the same day. The cancer risk is one and one-half orders of magnitude higher at the GEMB than the risk at the Golf Course. The primary risk drivers contributing to the upwind versus downwind cancer and non-cancer risk are presented in Tables 4-10 and 4-11.

The noncarcinogenic hazard indices are significantly higher at the downwind location (i.e., GEMB) than the upwind location (i.e., Golf Course). Table 4-10 identifies the constituents that are responsible for the majority of the hazard index for each location. Acetaldehyde, acetonitrile, acrolein, and PM₁₀ contributed the majority of the HI for each location. The most significant increase in risk at the downwind location was observed for acrolein. The hazard index increased from 15.9 at the upwind location to 63.0 at the downwind location.

The hazard quotient for acrolein at the GEMB was approximately 4 times greater that that at the Golf Course. Acrolein was not determined to be directly related to the incinerator by the three statistical methods used to evaluate chemicals that could be associated with SIC operations. It is not clear as to why this occurred but it may have resulted due to a combination of factors, which included other sources of acrolein (e.g., automobiles or other small combustions sources) variable feedstocks, various operating parameters and variable weather/wind conditions.

The carcinogenic risks are significantly higher at the downwind location (i.e., GEMB) than the upwind location (i.e., Golf Course). Table 4-11 identifies the constituents that are responsible for the majority of the carcinogenic risk for each location. Acetaldehyde, 1,3-butadiene, hexachlorobutadiene, and total 2,3,7,8-TCDD TEQs are responsible for the majority of the risks at the Golf Course. Cadmium and total 2,3,7,8-TCDD TEQs are responsible for the majority of the risks at the GEMB. The most significant increase in risk at the downwind

Table 4-10 — Percentage that COCs Contributed to the Hazard Indices at the Upwind and Downwind Locations

	Golf Course			GEMB		
	(Upwind)		Golf Course	(Downwind)		GEMB
	Exposure	Golf Course	(Upwind)	Exposure	GEMB	(Downwind)
	Point Conc.	(Upwind)	% of Total	Point Conc.	(Downwind)	% of Total
COC	(mg/m^3)	Hazard Index	Hazard Index	(mg/m^3)	Hazard Index	Hazard Index
Acetaldehyde	7.93E-02	8.5	23.6	8.22E-02	8.7	9.3
Acetonitrile	2.19E-01	3.5	9.8	7.51E-03	0.1	0.1
Acrolein	3.31E-04	15.9	44.4	1.31E-03	63.0	66.6
Acrylonitrile	5.61E-04	0.3	0.8	2.21E-03	1.1	1.1
Antimony	7.84E-06	0.2	0.5	3.63E-04	8.7	9.2
1,2-	2.30E-04	1.1	3.1	7.66E-05	0.4	0.4
Dibromoethane						
Formaldehyde	8.84E-04	0.3	0.8	2.24E-03	0.7	0.8
Hydrochloric	2.10E-03	0.1	0.3	2.43E-02	1.2	1.2
Acid						
Nickel	1.82E-05	0.4	1.0	9.34E-05	1.8	1.9
PM_{10}	2.31E-01	4.4	12.4	2.34E-01	4.5	4.8
All other		1.1	3.3		4.5	4.6
constituents						
Total		35.8	100%		94.8	100%

Notes: Percentages were calculated based on an RME adult stationed at NAF Atsugi for 6 years (i.e., 2 tours of duty).

Table 4-11 — Percentage that COCs Contributed to the Carcinogenic Risk at the Upwind and Downwind Locations

	Golf Course		Golf Course	GEMB		GEMB
	(Upwind)	Golf Course	(Upwind)	(Downwind)	GEMB	(Downwind)
	Exposure	(Upwind)	% of Total	Exposure	(Downwind)	% of Total
	Point Conc.	Carcinogenic	Carcinogenic	Point Conc.	Carcinogenic	Carcinogenic
COC	(mg/m^3)	Risk	Risk	(mg/m^3)	Risk	Risk
Acetaldehyde	7.93E-02	1.4E-05	23.5	8.22E-02	1.5E-05	6.6
Acrylonitrile	5.61E-04	3.1E-06	5.1	2.21E-03	1.2E-05	5.5
Arsenic	5.77E-06	2.0E-06	3.4	3.62E-05	1.3E-05	5.7
Benzene	3.00E-03	2.0E-06	3.4	4.63E-03	3.2E-06	1.4
1,3-Butadiene	4.45E-04	1.0E-05	16.8	7.51E-04	1.7E-05	7.6
Cadmium	1.22E-06	1.8E-07	0.3	2.54E-04	3.8E-05	16.6
1,2-Dibromoethane	2.30E-04	4.2E-06	6.8	7.66E-05	1.4E-06	0.6
1,4-Dichlorobenzene	1.58E-03	1.5E-06	2.4	1.18E-03	1.1E-06	0.5
1,4-Dioxane	1.85E-03	1.2E-06	1.9	8.88E-03	5.6E-06	2.5
Formaldehyde	8.84E-04	9.4E-07	1.6	2.24E-03	2.4E-06	1.1
Hexachlorobutadiene	3.51E-03	6.3E-06	10.4	9.58E-04	1.7E-06	0.8
Nickel	1.82E-05	3.9E-07	0.6	9.34E-05	2.0E-06	0.9
1,1,2,2- Tetrachloroethane	9.40E-04	4.5E-06	7.4	7.87E-04	3.8E-06	1.7
Total 2,3,7,8-TCDD TEQs	1.85E-09	6.5E-06	10.7	2.92E-08	1.0E-04	45.3
All other constituents		4.2E-06	5.7		1.0E-05	3.2
Total		6.1E-05	100%		2.3E-04	100%

Notes: Percentages were calculated based on an RME adult stationed at NAF Atsugi for 6 years (i.e., 2 tours of duty).

location was observed for 2,3,7,8-TCDD TEQs. The carcinogenic risk increased from 6.5E-06 (6.5 in 1,000,000) at the upwind location to 1.0E-04 (1.0 in 10,000) at the downwind location, an increase greater than one order of magnitude.

4.7 Acute Health Effects

Health risk assessments typically evaluate the potential impacts associated with chronic or long-term health risks. However, at the request of the Navy Environmental Health Center, the Toxicology Detachment of the Naval Health Research Center at Wright-Patterson Air Force Base conducted literature research on the health effects of various chemicals detected in ambient air at NAF Atsugi to evaluate the potential acute, or short-term health risks. The purpose of the research was to determine potential acute non-carcinogenic health effects for the specific 24-hour concentrations measured at NAF, Atsugi. Old Dominion University performed the search for the Toxicology Detachment. They did a very thorough search to identify toxicity criteria for humans as well as animals, from a variety of sources. Toxicity information on all chemicals detected at NAF Atsugi was collected from the following major sources:

- USEPA's on-line Envirofacts
- National Toxicology Program's on-line NIEHS Chemical Repository
- Texas Natural Resource Conservation Commission's Short-term Effects Screening Levels
- Reprotext
- Hazardtext
- Registry of Toxic Effects of Chemical Substances (RTECS)
- Hazardous Substances Data Bank (HSDB)
- Tomes/MEDITEXT
- New Jersey Health Hazard Information
- Toxic Substances Control Act Test Submission Database
- Other USEPA documents
- Chemical Fact Sheet prepared by Office of Pollution Prevention & Toxics (USEPA)

- Department of Health and Human Services publications
- Agency for Toxic Substances and Disease Registry (ATSDR) Toxicological Profiles
- American Conference of Governmental Industrial Hygienists (ACGIH)
 Documentation of the Threshold Limit Values & Biological Indices
- Patty's Industrial Hygiene and Toxicology
- Toxicology of the Eye, 3rd Edition
- California EPA's Inhalation Reference Exposure Levels
- Numerous other sources of information cited in Appendix E

Information collected during this literature search is presented in Appendix F, "Comparison of Maximum 24-Hour Concentrations of Ambient Chemicals Detected at Atsugi With Acute Health Effect Levels." Appendix F presents not only ATSDR Minimal Risk Levels (MRLs), but also all data that was found in the literature regarding air concentration levels for all chemicals detected at NAF Atsugi. This includes various acute health effect levels for varying periods of time with descriptions of the corresponding effects. In most cases these are occupational levels at much greater concentrations than those measured at Atsugi; however, they are included here for comparison purposes with the much lower MRLs. It was more appropriate to compare the maximum 24-hour concentrations to the acute MRLs, since they too are based upon 24-hour exposure time. A review of Appendix F indicates that the only chemical found to exceed any acute health concentration level was acrolein. Appendix F shows the maximum 24-hour concentrations of all chemicals, at any location, measured at NAF Atsugi. All chemicals with maximum 24-hour concentrations less than the corresponding MRL were subsequently screened out from further consideration for acute health effects.

An MRL is an estimate of the daily human exposure to a hazardous substance that is likely to be without appreciable risk of adverse non-cancer health effects over a specified duration of exposure. MRLs are derived when ATSDR determines that reliable and sufficient data exists to identify the target organ(s) of effect or the most sensitive health effect(s) for a specific duration for a given route of exposure to the substance. MRLs are derived for acute (1-14 days), intermediate (15-364 days), and chronic (365 days and longer) exposure durations, and for the oral and inhalation routes of exposure. MRLs are generally based on the most

sensitive substance-induced end point considered to be of relevance to humans. ATSDR states that MRLs are used as screening tools to assist public health officials with identifying geographical areas that may be expected to cause adverse health effects. Most MRLs contain a degree of uncertainty because of the lack of precise toxicological information on the people who might be most sensitive (e.g., infants, elderly, nutritionally or immunologically compromised) to the effects of chemicals. ATSDR uses a conservative approach to address this uncertainty, consistent with the basic public health principle of prevention. Although human data is preferred, MRLs often must rely on animal studies in the absence of human studies and assumes that humans are more sensitive to the effects of hazardous substances than animals, particularly for sensitive individuals. Thus, the resulting MRL may be as much as a hundred fold below levels that have been shown to be nontoxic in laboratory animals. According to ATSDR, these levels are not intended to serve as action levels or clean-up levels, but should be used as a tool to indicate the need for further evaluation. Exposure to a level above the MRL does not mean that adverse health effects will occur.

At a glance, it appears that acrolein, benzene, formaldehyde and vinyl acetate may have exceeded their respective MRLs. Although measured NAF Atsugi concentrations for benzene, formaldehyde, and vinyl acetate exceeded the intermediate MRL concentrations for one or two days, they did not exceed them for the exposure frequency for which the MRLs are established. (15-364 days). The maximum concentration measured for these chemicals and their respective MRLs are provided in Table 4-12, below.

For acrolein, 77 of 216 air samples, collected at various sampling locations, exceeded the acute inhalation MRL of 0.00005 parts per million (ppm). This MRL is based on eye irritation. All samples exceeded the intermediate MRL of 0.000009 ppm. The intermediate MRL is based on respiratory effects of acrolein exposure as seen in laboratory animals. In the 1990 Toxicological Profile for acrolein, ATSDR states that the only known effects of acrolein exposure in humans are general respiratory congestion and eye, nose, and throat irritation. Persons with pre-existing eye, skin, respiratory, allergic, asthmatic or heart diseases might be at increased risk due to acrolein exposure. Individuals with cystic fibrosis or asthma should be excluded from acrolein exposure (Micromedex 1999).

Analyte	Atsugi Maximum 24 Hour Concentration (ppm)	Inhalation MRL Concentration (ppm)	Health Effects
Acrolein	0.0008	Acute = 0.00005	Eye Irritation
Actorem	0.0008	Intermediate = 0.000009	Respiratory Irritation
Benzene	0.0063	Acute = 0.05	Immunological
Delizene	0.0003	Intermediate $= 0.004$	Neurological
		Acute = 0.04	Respiratory
Formaldehyde	0.0162	Intermediate $= 0.03$	Respiratory
Formatdenyde	0.0102	Chronic = 0.008	Respiratory
Vinyl Acetate	0.0114	Intermediate = 0.01	Respiratory

Table 4-12—Constituents Equivalent to or Exceeding ATSDR's Acute and Intermediate Inhalation MRLs

Source: Agency for Toxic Substances and Disease Registry Minimum Risk Levels

Note: Acute MRLs refer to exposures of 1-14 days. Intermediate MRLs refer to exposures of 15 – 364 days. Chronic MRLs refer to exposures of 365 days and longer.

The intermediate MRL for formaldehyde is 0.03 ppm and the chronic MRL is 0.008 ppm and they are based on a study of nasopharyngeal irritation in laboratory animals. The maximum air concentration of 0.0162 ppm was less than the intermediate and acute MRLs on one day, but exceeded the chronic MRL. The most common symptoms of exposure to formaldehyde are irritation of the eyes, nose, and throat. Increased tearing with irritation of mucous membranes are associated with airborne levels of 0.4 - 3 ppm. Persons with eye, skin, respiratory, or allergic conditions may be more sensitive to the effects of formaldehyde. Asthmatics sensitized to formaldehyde may be more sensitive to formaldehyde at lower concentrations than non-sensitized individuals (Micromedex 1993).

The intermediate MRL for benzene is 0.004 ppm, which is based on no adverse effect on neurological functions. On one of the 70 days sampled, the benzene concentration exceeded 0.004 ppm at 3 sampling locations. The maximum concentration measured was 0.0063 ppm. Since the intermediate MRL applies to 15-364 days of exposure, one out of 70 days of sampling does not present a concern.

The intermediate inhalation MRL for vinyl acetate is 0.01 ppm, based on respiratory effects of vinyl acetate exposure to laboratory animals. The intermediate inhalation MRL was exceeded on 2 of the 70 days that were sampled. The maximum vinyl acetate measured was 0.0114 ppm. Since the intermediate MRL applies to 15-364 days of exposure, two out of 70 days of sampling do not present a concern.

Although acrolein was the only chemical out of 167 chemicals found to exceed the acute inhalation MRL, exposures were not limited to a single chemical, but a mixture of chemicals. It is important to note that the toxicological and epidemiological studies that form the basis of the MRLs are based on exposure to a single chemical. The results of research on the effects of exposure to a mixture of chemicals is not complete; therefore, when two or more chemicals act on the same organ system, their combined effect should be considered as additive, unless there is evidence to the contrary. The health effects resulting from exposure to acrolein are likely to be reversible; however, combination with other chemicals such as respirable particulates is cause for concern. These chemicals can adhere to fine particulates and potentially result in deep lung penetration and retention.

The following chemicals that were present in the ambient air with acrolein cause transient and reversible irritation of mucosal membranes, including the eyes, nose, throat, and respiratory tract: acetone, antimony, benzene, dichlorobenzene, dichloroethane, dichloroethylene, butadiene, dioxane, butanol, octene, propanol, methylhexane, acetaldehyde, acetone, acetonitrile, pinene, benzyl chloride, chloroform, hexene, crotonaldehyde, cyclohexane, ethanol, formaldehyde, hydrochloric acid, hydrofluoric acid, diethylbenzene, methylisobutylketone, methyphenol, xylene, naphthalene, hexane, perchloroethylene, sulfuric acid, tetrachloroethane, toluene, vinyl acetate, and vinyl chloride. Acting in an additive fashion, mucosal membrane irritants were of concern in the ambient air of NAF Atsugi.

4.8 Health Effects of Various Chemicals

The following section addresses the health effects related to selected chemicals of concern that include the primary non-carcinogenic and carcinogenic risk drivers in the health risk assessment and NAAQS criteria pollutants. Acrolein, acetaldehyde, formaldehyde, and particulate matter account for the majority of non-carcinogenic risks (97%). Acetaldehyde, 1,3-butadiene, formaldehyde, hexachlorobutadiene, and dioxin account for the majority of carcinogenic risks (80%). Lead and particulate matter are specific criteria pollutants that exceeded the NAAQS. In the following discussion, emphasis has been placed on known and suspected adverse human health effects from monitored exposure conditions at NAF Atsugi. Effects through inhalation, the primary route of entry, are of most interest. Also, special

attention has been given to effects on asthma, any potential chronic effects, as well as any known reproductive or developmental effects from the chemicals of concern.

Acrolein

Acrolein contributed approximately 59 - 70 % of the non-carcinogenic risk at NAF Atsugi through inhalation. It is an unsaturated aldehyde compound found in polluted air that is formed as a reaction product of the photo-oxidation of hydrocarbons from combustion of fossil fuels, tobacco smoke, and pyrolyzed animal and vegetable fats. It contributes to the odor as well as eye and sensory irritations of photochemical smog. Acrolein can be extremely toxic to humans if in high enough concentrations. The acute and chronic effects of acrolein in humans consist mainly of effects on the lung, such as upper respiratory tract irritation and congestion and eye irritation. Exposure to low concentrations of acrolein may produce mild irritation of the eyes and throat. Symptoms of exposure at high concentrations can include eye, skin, and mucous membrane irritation, decreased lung function, labored breathing and a burning sensation in the throat. These effects can be intensified when acrolein is present in mixed atmospheres containing particles, such as in NAF Atsugi.

The USEPA reference concentration (RfC) for acrolein is 0.00002 mg/m³. The USEPA estimates that inhalation of this concentration or less, over a lifetime, would not likely result in the occurrence of chronic, non-carcinogenic effects, such as general respiratory congestion and eye, nose, and throat irritation. The RfC for acrolein is based on squamous metaplasia and neutrophilic infiltration of nasal epithelium in rats. The USEPA has high confidence in the studies on which the RfC was based because adequate numbers of animals were used, careful attention was paid to experimental protocol, and together they demonstrated a consistent profile of histopathological changes in the respiratory system. The principal study for deriving the RfC, Kutzman (1981), used a lowest observed adverse effect level (LOAEL) (adjusted for humans) of 0.02 mg/m³ before dividing by uncertainty and modifying factors totaling 1000. A factor of 10 was incorporated to account for interspecies extrapolation; a factor of 10 was used to protect sensitive human subpopulations; and a factor of 10 was used to account for extrapolation from a sub-chronic to a chronic exposure. The maximum 24-hour concentration of acrolein at any one sampling site at NAF Atsugi was 0.004 mg/m³, which is five times less than the above LOAEL. However, this level is two hundred times greater than the RfC. Therefore, if an individual were

exposed to this concentration for a lifetime, it is possible that chronic effects could occur. Also, as discussed in the subsection on acute health effects (page 159), a concentration of 0.004 mg/m³, which exceeds the inhalation MRL, would be expected to cause respiratory track congestion and eye, nose, and throat irritation. The minimum and average 24 hour concentrations of acrolein at the same site at NAF Atsugi were 0.00008 mg/m³ and 0.00033 mg/m³, respectively. The USEPA compiled information from 1961 to 1980 for two urban locations that reported a mean concentration of 0.014 mg/m³ with a range of concentrations from 0.008 to 0.024 mg/m³. Therefore, acrolein levels at NAF Atsugi are many times less than those that have been measured in urban areas of the U.S. several decades ago.

There is no information available on the carcinogenic effects of acrolein in humans. There is limited animal cancer data—one inhalation study in rats reported no evidence of tumor in the respiratory tract or in other tissues and organs (ATSDR 1998), while another study reported an increased incidence of adrenocortical tumor in female rats exposed to acrolein in drinking water (HSDB 1993). USEPA has classified acrolein as a Group C, possible human carcinogen, based on limited evidence of carcinogenicity in animals, the structural similarity of acrolein to substances possibly carcinogenic to humans, the carcinogenic potential of one of its metabolites, and the lack of human data (EPA 1993). USEPA has not yet developed a carcinogenic slope factor or unit risk factor for acrolein.

There is no information on the reproductive or developmental effects of acrolein in humans. In one reproductive animal study, rats were exposed to acrolein by inhalation, with no effects observed on the number of pregnancies or the number and weights of the fetuses.

Acrolein has been reported to cause birth defects in rats when injected directly into the embryonic tissue (ATSDR 1989a).

Acetaldehyde

Acetaldehyde contributed approximately 12 to 17 % of the non-carcinogenic risk and approximately 8 to 16 % of the carcinogenic risk at NAF Atsugi through inhalation. It is an aldehyde compound found in polluted air that is formed as a reaction product of the photooxidation of hydrocarbons. It is not as irritating as acrolein because of lesser solubility in airway fluids; however, it still contributes to the odor as well as eye and sensory irritations of photochemical smog. It is also a product of incomplete combustion in fireplaces and

woodstoves, coffee roasting, burning of tobacco, vehicle exhaust, coal refining, and waste processing. It is formed in the body from the breakdown of ethanol. Acetaldehyde can be extremely toxic to humans if in high enough concentrations. The primary acute effect of inhalation of low concentrations is irritation of the eyes, skin, and respiratory tract. Exposure to higher concentrations can result in erythema, coughing, pulmonary edema, and depression of the central nervous system. Exposure to extremely high concentrations can result in respiratory paralysis and death. Repeated exposure to high concentrations can affect the central nervous.

The maximum 24-hour concentration of acetaldehyde at any one sampling site at Atsugi was 0.28 mg/m³ and the average concentration at the same site was 0.028 mg/m³. These levels of acetaldehyde at Atsugi seem to be similar to the concentrations of acetaldehyde in various urban areas in the United States. Concentrations of acetaldehyde measured in air samples taken from different locations vary, depending on several conditions, including weather. Acetaldehyde has been detected in ice fog, rain, cloud mist, and fog. Urban concentrations measured 0.009 mg/m³ in Baltimore, MD; 0.061 mg/m³ in Claremont, CA (severe smog); and 0.297 mg/m³ in another unspecified California City (foggy conditions) (CHEMFATE 1994). Acetaldehyde concentrations reached 1.03 mg/m³ in clouds over California (site not specified), and levels reached 0.005-0.023 mg/m³ in Grand Canyon, AZ (CHEMFATE 1994). Ambient air sampling of two rural regions, Point Barrow, Alaska and Whiteface Mountain, New York, indicate "background" acetaldehyde concentrations are from 0 to 0.001 mg/m³ (ARB 1993). The USEPA also reported concentrations of acetaldehyde from 14 study areas during 1989. The overall acetaldehyde average concentration in these areas was 0.002 mg/m³ (EPA 1993a).

The RfC for acetaldehyde is 0.009 mg/m³. The USEPA estimates that inhalation of this concentration or less, over a lifetime, would not likely result in the occurrence of chronic, non-carcinogenic effects, such as respiratory tract, eye, and skin irritation. The RfC for acetaldehyde is based on degeneration of olfactory epithelium during short-term rat inhalation studies. The USEPA has medium confidence in the principal studies since appropriate histopathology was performed on an adequate number of animals, but the duration was short and only one species was tested. The principle studies for deriving the RfC, (Appleman et al 1986, 1982), used a NOAEL (adjusted for humans) of 8.7 mg/m³ before dividing by uncertainty and modifying factors totaling 1000. A factor of 10 was applied to account for interspecies extrapolation; a factor of 10 for taking into account the most sensitive human sub-population; and a factor of 10

to account for extrapolating sub-chronic to chronic exposures. The maximum 24-hour concentration of acetaldehyde was 0.28 mg/m³, which is thirty one times less than the above NOAEL. However, this level is thirty one times greater than the RfC. Therefore, if an individual were exposed to this concentration for a lifetime, it is possible that chronic effects could occur.

Human data regarding the carcinogenic effects of acetaldehyde are inadequate. An increased incidence of nasal tumors in rats and laryngeal tumors in hamsters has been observed following inhalation exposure to acetaldehyde. USEPA has classified acetaldehyde as a Group B2, probable human carcinogen of low carcinogenic hazard, with an inhalation unit risk estimate of 2.2E-6 per ug/m³. So, if an individual were to breathe air-containing acetaldehyde at 50 ug/m³ over his or her entire lifetime, that person would theoretically have no more than a one-in-ten thousand increased chance of developing cancer (EPA1993b).

Information is not available on the reproductive or developmental effects of acetaldehyde in humans. Acetaldehyde has been shown, in animals, to cross the placenta to the fetus. Data from animal studies suggest that acetaldehyde may be a potential developmental toxin (IARC 1985).

Formaldehyde

Formaldehyde contributed approximately 3 to 4 % of the non-carcinogenic risk and approximately 3 to 8 % of the carcinogenic risk at NAF Atsugi through inhalation. It is an aldehyde compound found in polluted air that is formed as a reaction product of the photooxidation of hydrocarbons. Formaldehyde is a primary sensory irritant. Since it is very soluble in water, it is easily absorbed in mucous membranes in the nose, upper respiratory tract, and eyes. Symptoms of exposure at high concentrations can include eye, nose, throat, and respiratory tract irritation, lacrimation, cough, and bronchitis spasm. Prolonged exposure may produce skin and respiratory sensitizations. The dose-response curve for formaldehyde is steep: 0.5 to 1 ppm creates a detectable odor; 2 to 3 ppm produces mild irritation; and 4 to 5 ppm is intolerable to most people. Formaldehyde is thought to act via sensory nerve fibers to reflexively induce bronchoconstriction, which induces an increase in airflow resistance. Respiratory frequency and minute volume also become decreased. The no observed effect level (NOEL) using these lung function criteria is about 0.05 ppm, which is more than two times greater than

0.02 ppm (0.0162 mg/m³), the maximum 24-hour concentration of formaldehye at any one sampling site at NAF Atsugi. USEPA has not established a reference concentration (RfC) for formaldehyde. However, 0.02 ppm does exceed the chronic Maximum Risk Level (MRL) set at 0.008 ppm by ATSDR. Formaldehyde can also interact with water-soluble particles during inhalation and produce irritancy beyond that expected for the vapor alone. In addition to polluted outside air, formaldehye can be present in indoor atmospheres as an off-gassed product of construction materials such as plywood or improperly installed urea-formaldehyde foam insulation. Children were found to have lower peak expiratory flow rates in homes with 60 ppb than did unexposed children. Asthmatic children were affected below 50 ppb. Therefore, this irritant vapor can cause respiratory effects at common exposure levels (Krzyzanowski et al 1990).

Limited human studies have reported an association between formaldehyde exposure and lung and nasopharyngeal cancer. Animal inhalation studies have reported an increased incidence of nasal squamous cell cancer. USEPA has classified formaldehyde as a Group B1, probable human carcinogen of medium carcinogenic hazard, with an inhalation unit risk estimate of 1.3E-5 per ug/m³. So, if an individual were to breathe air containing 16 ug/m³ over his or her entire lifetime, that person would theoretically have no more than a two-in-ten thousand increased chance of developing cancer (EPA 1993c).

Reproductive effects, such as menstrual disorders and pregnancy problems, have been reported in women workers exposed to formaldehyde. No other reproductive or developmental effects have been reported in humans or animals (WHO 1989).

Hexachlorobutadiene

Hexachlorobutadiene contributed approximately 3 to 27 % of the carcinogenic risk at NAF Atsugi through inhalation. It is a chlorinated compound with a turpentine-like odor. There is no information available on the acute or chronic effects of hexachlorobutadiene in humans. Animal studies have reported irritant effects on the respiratory system from acute inhalation exposure. The USEPA has not established a reference concentration (RfC) for this compound. Also, there is no information available on the carcinogenic effects of this compound in humans or animals from inhalation exposure. However, one study reported kidney tumors in rats exposed to hexachlorobutadiene orally. As a result, the EPA has classified this compound as a

Group C, possible human carcinogen of low carcinogenic hazard, with an inhalation unit risk estimate of 2.2E-5 per ug/m³. The maximum 24-hour concentration of hexachlorobutadiene was 10 ug/m³; and if an individual were to breathe air containing this concentration over his or her entire lifetime, that person would theoretically have no more than a two-in-a-ten thousand increased chance of developing cancer. Information is not available on the developmental or reproductive effects of hexachlorobutadiene in humans. However, reduction in fetal body weights from inhalation exposure has been reported in animal studies. 0.2 to 460 ug/m³ of this compound has been reported near industries where hexachlorobutadiene is formed or used (EPA 1994c).

Particulate Matter (PM₁₀ and PM_{2.5})

Particulate matter (PM_{10}) contributed approximately 4 to 6 % of the non-carcinogenic risk at NAF Atsugi through inhalation. Particulate matter (PM) is a term used for a mixture of solid particles and liquid droplets found in air. PM consists of organic, inorganic, and biological materials whose composition depends primarily upon local point sources. Coarse particles or PM_{10} range in size from 2.5-10 micrometers in diameter and come from a variety of sources including windblown dust and grinding operations. Fine particles or $PM_{2.5}$ consists of particles less than 2.5 micrometers in diameter that often come from such things as motor vehicles, power plants, fireplaces and wood stoves. Fine particles can also be formed in the air from gases such as sulfur dioxide and nitrogen oxides. PM_{10} showed a strong association with the SIC at NAF Atsugi.

Both PM₁₀ and PM_{2.5} are capable of penetrating deep into the lungs, including the alveoli. Organic chemicals commonly found in air pollution, such as polycyclic and semivolatile compounds, could adhere to particulate matter, resulting in a prolonged residence time at deposition sites deep within the respiratory tract. Therefore, PM can elicit both short- and long-term health effects, such as local irritation at the sites of deposition, and the effects of associated chemicals. Epidemiological studies have shown consistent positive associations of exposure to ambient PM with health effects and resulting related impacts, including: premature mortality; respiratory related hospital admissions and emergency room visits; aggravated asthma; acute respiratory symptoms, including aggravated coughing; breathing difficulties; chronic bronchitis; decreased lung function; and work and school absences. Individuals living in industrialized areas

are more likely to experience respiratory allergic symptoms from asthma than those living in rural areas (D'Amato 1999). Allergic respiratory diseases, such as asthma, have become more common worldwide in the last two decades, and outdoor air pollution may be a major responsible factor (D'Amato et al 2001; Jones 2000). In asthmatics, epidemiological studies generally show a positive correlation between the particulate fraction of air pollution and increased morbidity (Goldsmith and Kobzik 1999). Epidemiology has associated particulate pollution with asthma exacerbations and other cardiorespiratory illnesses (Linn and Gong 1999). Epidemiological studies indicate increased health risks associated with exposure to PM, alone or in combination with other air pollutants. Analyses support the use of PM₁₀ as an indicator of ambient particle exposures associated with human health effects and the use of a PM₁₀ standard for protecting public health (EPA 1996a).

Individuals with cardiovascular or pulmonary disease, such as congestive heart disease, asthma, or chronic obstructive pulmonary disease, especially if elderly, are more likely to suffer severe health effects, such as mortality or hospitalization, related to PM exposure than are healthy young adults. A recent study suggests that elevated concentrations of PM_{2.5} may transiently elevate the risk of myocardial infarctions within a few hours and days after exposure occurs (Peters et al 2001). Children and asthmatics are also susceptible to certain PM effects, e.g., increased respiratory symptoms and decreased lung function. Smokers also constitute a population group at increased risk for ambient PM exposure effects. Exposure to PM can increase susceptibility to respiratory infections and can aggravate existing respiratory diseases, such as asthma and chronic bronchitis, causing increased medication use and more doctors visits (EPA 1996a).

Health-based NAAQS for particulate matter in the U.S. (EPA 2001a) are as follows:

- PM₁₀ Annual Standard 0.050 mg/m³
- PM_{10} 24-hour Standard 0.150 mg/m³
- PM_{2.5} Annual Standard 0.015 mg/m³
- PM_{2.5} 24-hour standard 0.065 mg/m³

The average concentrations of PM_{10} and $PM_{2.5}$ at NAF Atsugi during the 14-month data collection period for the comprehensive health risk assessment were 0.070 mg/m³ and 0.026

mg/m³ respectively, well above the U.S. Annual Standards. The levels of particulate matter in the air at NAF Atsugi were roughly equivalent to the levels measured in Los Angeles County, California, in 1997. The Los Angeles County data was collected as part of a health risk assessment to assess the potential health benefits of attaining the revised NAAQS for particulate matter. This study concluded that excess morbidity and mortality related to particulate matter in Los Angeles could be reduced by as much as 62.8% by attaining the revised NAAQS for particulates (EPA 2001a).

There are significant demographic differences between the populations of NAF Atsugi and Los Angeles County. In general, the NAF Atsugi population is younger and healthier. In addition, the Navy's active overseas screening program ensures that those individuals at greatest risk of air quality related morbidity and mortality are not stationed at NAF Atsugi. Regardless, these differences make it impossible to quantify the health impact of a significant reduction in airborne particulate at NAF Atsugi, although some reduction in overall respiratory illness would be expected.

The maximum 24-hour concentration at any sampling site at NAF Atsugi was 0.24 mg/m³ for PM₁₀ and 0.13 mg/m³ for PM_{2.5}. These concentrations also far exceed the NAAQS and are considered unhealthful. PM₁₀ concentrations greater than 0.150 mg/m³ can cause mild aggravation of symptoms in susceptible persons, with irritation symptoms in healthy individuals. At these concentrations persons with heart or respiratory illnesses are advised to stay indoors and reduce physical activity (EPA 1996b).

1,3-Butadiene

1,3-Butadiene contributed approximately 13 to 22 % of the carcinogenic risk at NAF Atsugi through inhalation. It is a colorless gas with a mild gasoline-like odor. Acute exposure by inhalation of 2,000 – 18,000 ppm in humans results in irritation of the eyes, nasal passages, throat, and lungs. More severe symptoms of respiratory tract irritation, such as coughing, and neurological effects, such as blurred vision, headache, drowsiness, vertigo, and fatigue, can occur at levels greater than 18,000 ppm. In spite of this, the USEPA has not yet established a reference concentration (RfC) for this compound. Also, there is no information available on the reproductive or developmental effects of 1,3-butadiene in humans. Animal studies have reported developmental effects, such as skeletal abnormalities and decreased fetal weights, and

reproductive effects, including an increased incidence of ovarian atrophy and testicular atrophy, from inhalation exposure (EPA 1994d).

Several epidemiological studies of workers in styrene-butadiene factories have shown an increased incidence of respiratory, bladder, and stomach cancers. However, these studies are considered inadequate to determine a causal association between 1,3-butadiene exposure and cancer due to confounding factors. Animal studies have reported tumors in a variety of organs and tissues following inhalation exposure with the formation of hemangiosarcomas of the heart, lymphomas and alveolar and bronchiolar adenomas and carcinomas. As a result, USEPA has classified 1,3-butadiene as a Group B2, probable human carcinogen. They have calculated an inhalation unit risk estimate of 2.8E-4 per ug/m³. The maximum 24-hour concentration of 1,3-butadiene was 6 ug/m³ and the average 24-hour concentration at the same location was 0.6 ug/m³. If an individual were to breathe air containing these concentrations over his or her entire lifetime, that person would theoretically have no more than a two-in-one thousand and two-in-ten thousand increased chance of developing cancer. 1,3-butadiene has been detected in ambient air in cities and suburban areas at an average of 0.6 ug/m³. Higher concentrations have been measured in highly industrialized cities or near oil refineries, chemical manufacturing plants, and plastic and rubber factories (EPA 1994d).

Dioxin

Dioxin contributed approximately 11 to 53 % of the calculated carcinogenic risk at NAF Atsugi through inhalation and was not a risk driver for the calculated non-carcinogenic risk. The term "dioxin" refers to a group of chemical compounds that share certain similar chemical structures, mode-of-action and biological characteristics. A total of 30 of these dioxin-like compounds exist and are members of three closely related chemical families: the chlorinated dibenzo-p-dioxins (CDDs), chlorinated dibenzofurans (CDFs) and certain polychlorinated biphenyls (PCBs). The term dioxin is also used for the most well-studied and one of the most toxic dioxins, 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD). CDDs and CDFs are not created intentionally, but can be produced inadvertently in nature and by a number of human activities. Dioxins are emitted from incinerators that burn medical waste, municipal solid waste, hazardous waste sewage sludge, and tires. Combustion, chlorine bleaching of pulp and paper, certain types of chemical manufacturing and processing, and other industrial processes all can create small

quantities of dioxins. They are products of incomplete combustion when chlorine and complex mixtures containing carbon are present (EPA 2001b). Dioxins are absorbed on airborne particulate and on industrial effluent and then deposited on soil and eventually bind to other organic substances and bottom sediment in lakes and rivers. Atmospheric dioxins are deposited on vegetation, which farm animals consume. Humans can then ingest crops, fish, meat, and dairy products, resulting in a body burden of dioxin (Scorecard, 2001).

Dioxins are believed to cause toxic effects in similar ways; that is, they share a "common mechanism of toxicity". As a result, USEPA and others use an approach that adds together the toxicity of individual dioxins in order to evaluate complex environmental mixtures to which people are exposed. Because dioxins differ in their toxic potential, the toxicity of each component in the mixture must be accounted for in estimating the overall toxicity. To do so, international teams of scientists have developed Toxicity Equivalency Factors that compare the toxicity of different dioxins. Given these factors, the toxicity of a mixture can be expressed in terms of its Toxicity Equivalents (TEQ), which is the amount of TCDD it would take to equal the combined toxic effect of all the dioxins found in that mixture (EPA 2001b).

The use of the TEQ approach represents a key assumption upon which many of the conclusions of the USEPA in the recent reassessment of dioxin exposure and human health effects since 1991 are based. It is important to note that the USEPA will not use the conclusions of the most recent draft dioxin reassessment for regulatory purposes until the science peer review process is completed. External peer review of the reassessment was completed in July 2001; the revised draft was sent to the USEPA Science Advisory Board (SAB) Dioxin Reassessment Review Subcommittee and then to the SAB Executive Committee, who has endorsed the reassessment and forwarded it to the USEPA Administrator. Final revisions are being completed now in response to SAB recommendations and public comments. Upon completion, the USEPA will release the comprehensive reassessment of dioxin exposure and human health effects in a document entitled "Exposure and Human Health Reassessment of 2,3,7,8-Tetrachlorodibenzo-p-Dioxin (TCDD) and Related Compounds". The recent USEPA reassessment that is progressing toward completion finds that dioxins are potent animal toxicants with potential to produce a broad spectrum of carcinogenic and non-carcinogenic adverse effects in humans. Dioxins can alter the fundamental growth and development of cells in ways that have the potential to lead to many kinds of impacts (EPA 2001b).

Acute exposure of humans to high concentrations of dioxins has caused chloracne (a severe acne-like condition that sometimes persists for many years), liver toxicity, skin rashes, nausea, vomiting, and muscular aches and pains. Animal studies have shown weight loss, hyperkeratosis, facial alopecia, inflammation of the eyelids, and loss of fingernails and eyelashes. The immune system is very sensitive to high concentrations of dioxin. Animal studies have shown thymic atrophy and changes in the spleen, lymph nodes, and bone marrow. Chronic exposure of animals to dioxins has resulted in splenic and testicular atrophy and abnormal neurological findings. The USEPA has not established a reference concentration (RfC) or an oral reference dose (RfD) for TCDD. As a result, dioxin's contribution to non-carcinogenic risk has not been quantitatively estimated in human health risk assessment.

TCDD at lower concentrations is a known carcinogen in rodents and is considered a potential carcinogen in humans. Human studies which have reported cancer increases are inconclusive due to inadequate data. Animal studies have shown increases in tumors of the liver, lung, nasal turbinates, and the hard palate. The International Agency for Research on Cancer has classified TCDD as Group 1: Human Carcinogen, based on sufficient evidence in humans (Scorecard, 2001). USEPA now characterizes TCDD as a "human carcinogen" based on the weight of evidence of animal and human studies and characterizes other dioxins as "likely human carcinogens" (EPA 2001b).

Human studies on the adverse reproductive and developmental effects of dioxins are inconclusive. However, animal studies have shown TCDD to be teratogenic and fetotoxic. Reproductive and teratogenic effects observed in animals include: cleft palate, kidney abnormalities, decreased fetal weight and survival, hydrocephalus, and infertility (Scorecard 2001).

The USEPA reassessment proposes that most dioxins enter ecological food webs by being deposited from the atmosphere, either directly following air emissions or indirectly by processes that return dioxins already in the environment to the atmosphere. Once they reach the environment, dioxins are highly persistent and can accumulate in the tissues of animals. USEPA estimates that most dioxin exposure occurs through the diet, with over 95% of dioxin intake for a typical person coming through dietary intake of animal fats. Small amounts of exposure occur from breathing air containing trace amounts of dioxin on particles and in vapor form, from

inadvertent ingestion of soil containing dioxin, and from absorption through the skin contacting air, soil, or water containing minute levels. These processes result in widespread, low-level exposure of the general population to dioxins (EPA 2001b).

USEPA estimates that the amount of dioxin found in the tissues of the general human population (which is known as the "body burden") closely approaches (within a factor of 10) the levels at which adverse effects might be expected to occur, based on studies of animals and highly exposed human populations. Despite the potential risks, currently there is no clear indication of increased disease in the general population attributable to dioxin-like compounds. This may be due to limitations of current data and scientific tools rather than indicating that dioxin exposure is not causing adverse effects. For cancer, USEPA estimates that the risks for the general population based on dioxin exposure may exceed 1 in 1,000 (10⁻³) increased chance of experiencing cancer related to dioxin exposure. Actual risks are unlikely to exceed this value and may be substantially less. This cancer risk indicates an about 10-fold higher chance than estimated in USEPA's earlier (1994) draft of this reassessment, which was 1 in 10,000 (10⁻⁴). This reflects a proposal for increasing the cancer slope factor for dioxin to a value at least six times more conservative than the one that is currently in use and which was used in this particular risk assessment for NAF Atsugi (EPA 2000). The background excess lifetime cancer risk in the U.S., attributable to dioxin exposure via all pathways, as shown in Table 4-7, is 1.0 X 10⁻⁴ or about 1 per 10,000, which is consistent with the earlier draft of this reassessment.

Table 4-13 — PCDD/F TEQ Cancer Risk Estimates for Adult and Child* Overall U.S. Background Exposures and For Selected Pathways

Description	Adult Lifetime Cancer Risk**	Child Lifetime Cancer Risk **		
Overall	1.0 E-04	2.7 E-05		
Inhalation	3.6 E-06	6.7 E-07		
Soil Ingestion	1.3E-06	8.8 E-07		
Soil Dermal Contact	3.1 E-07	1.8 E-08		

Source: Personal communication note from Matthew Lorber, USEPA, NCEA, Washington, DC

Most U.S. citizens come to NAF Atsugi with this same background lifetime cancer risk (1.0 X 10⁻⁴, using the earlier reassessment). Since most of the food consumed by U.S. service personnel while at NAF Atsugi comes from U.S. sources, the contribution of diet to lifetime cancer risk, from dioxin, is likely to be the same. Information regarding background

^{*}Child (1-5 years of age)

^{** 95%} Upper bound risk

concentrations of chemicals in food is not readily available for most chemicals sampled at Atsugi. The USEPA has conducted national surveys to determine the concentrations of dioxins in various types of foods in the U.S. Human exposure to dioxins in the U.S. is attributed primarily to consumption of animal products such as meat, dairy products and fish. The exposure portion of the USEPA dioxin reassessment has concluded that over 90% of human exposure to dioxin (and related compounds) occurs via food ingestion, primarily meats, dairy products and fish. Using the background concentration of dioxin in food in the U.S., as determined by USEPA, background exposure levels of dioxin result in an upper bound cancer risk estimate of about one in one thousand to one in ten thousand (i.e. 1.0 X 10⁻³, using the current reassessment to 1.0 X 10⁻⁴, using the earlier reassessment). Consumption of food accounts for a substantial portion of total dioxin intake, regardless of the food source. Consumption of substantial amounts of foods from sources that contain higher concentrations of dioxin than that in U.S. food would elevate risk above U.S. background for foodstuffs.

As illustrated in Table 4-14, the average concentration of dioxin in soil (15 ppt TEQ) at NAF Atsugi to which individuals were likely to be exposed is essentially the same as the average concentration found in an urban setting in the U.S. (2-21 ppt TEQ), which is only marginally higher than the average background soil concentration found in rural settings. In addition, the maximum detected soil concentration of dioxin at NAF Atsugi, about 600 ppt TEQ, is well below the USEPA remediation standard of 1000 ppt. However, this concentration is much greater than the average rural concentration of 0.1-6 ppt TEQ and the highest average urban concentration of approximately 20 ppt TEQ in the U.S.. At the same time it is recognized that soil concentrations nearer the SIC, on the GEMB site and Golf Course where much less exposure is expected to occur, are significantly higher at greater than 100 ppt for some samples.

The concentration of dioxin in the air at NAF Atsugi (1.57 pg TEQ/m³) was much higher than the background concentration found in the urban air (0. 03–0.2 pg TEQ/m³) in the U.S. These elevated dioxin concentrations in air and in soil were attributed to emissions from the SIC, as indicated by the gradation of soil concentrations, highest near the SIC and decreasing concentrations associated with distance from the SIC and statistical analysis of air sampling data.

Environmental Media	Location	Dioxin (ppt TEQ)* Dioxin (pg TEQ/m³) +
Soil	NAF Atsugi	15*
Soil	Urban U.S.	2-21*
Soil	Rural U.S.	0.1 – 6*
Air	NAF Atsugi	1.57+
Air	Urban U.S.	$0.03 - 0.2^{+}$
Air	Rural U.S.	$0.01 0.02^{+}$

Table 4-14 — Soil and Airborne Dioxin Levels for NAF Atsugi and United States

Source for U.S Urban, Rural Levels: USEPA, 2000. Exposure and Human Health Reassessment of 2,3,7,8-Tetrachlorodibenzo-p-Dioxin (TCDD) and Related Compounds. Part I, Chapter III: Integrated Summary and Risk Characterization for 2,3,7,8-Tetrachlorodibenzo-p-Dioxin (TCDD) and Related Compounds. National Center for Environmental Assessment, Office of Research and Development. September 2000 SAB Review Draft. EPA/600/P-00/001Bg

Neither incidental soil ingestion nor skin contact with the concentrations of dioxin found in soil at NAF Atsugi significantly added to the lifetime cancer risk each U.S. citizen brought with them from dioxin exposures in the U.S. However, the higher airborne concentrations of dioxin at NAF Atsugi may have as much as doubled the dioxin related lifetime cancer risk. Therefore, the risk of exposure to dioxin found in the soil at NAF Atsugi is of little concern as compared to the risk of exposure to dioxin through inhalation of ambient air. Neither soil nor airborne concentrations of dioxin at NAF Atsugi have contributed to non-carcinogenic risks.

It is important to note that children and other populations that have experienced elevated exposures to dioxin are of special concern. Fetuses, infants, and children may be more sensitive to dioxin exposure because of their rapid growth and development. Data on risk to children are limited; however, and it is not known if the children in the general U.S. population are experiencing adverse effects from dioxin. Although breast milk appears to be a significant source of dioxin exposure for nursing infants, the overwhelming body of evidence supports the health benefits of breastfeeding despite the potential presence of dioxin. Other populations have experienced elevated exposures to dioxin as a result of food contamination incidents around the world, through the workplace or from industrial accidents, or from consumption of unusually high amounts of fish, meat, or dairy products containing elevated levels of dioxins. In some cases, such as USAF personnel exposed to the herbicide Agent Orange contaminated with dioxin during the Vietnam War, dioxin exposure has been associated with adverse health effects (EPA 2001b). Follow-up of the population exposed to substantial quantities of TCDD vented directly

^{*}TEQ - Toxic Equivalent Concentration

⁺Average concentrations

into the atmosphere after the 1976 industrial accident in Seveso, Italy, generated results that support dioxin as carcinogenic to humans and corroborate the hypotheses of its association with other health outcomes, including cardiovascular and endocrine-related effects (Bertazzi et al 2001).

Lead

Although an estimation of the contribution of lead to non-carcinogenic and carcinogenic risks at NAF Atsugi was not determined, lead is a chemical of concern because it exceeded the NAAQS and its toxicity is of special concern, especially in regards to children. Human exposure to lead usually occurs through a combination of inhalation and oral exposure, with inhalation contributing a greater proportion of the dose for occupationally exposed groups, and the oral route generally contributing a greater proportion of the dose for the general population, especially children. However, the health effects of lead are the same regardless of the route of exposure and are correlated with internal exposure, as blood lead levels. Inhalation of airborne lead has been of most concern in NAF Atsugi. Until the introduction of lead-free gasoline, the largest source of lead in the atmosphere has been from leaded gasoline combustion. Other airborne sources of lead include combustion of solid waste, coal, and oils. Lead in the atmosphere exists as dust or particulates of lead dioxide, or in the form of vapors. About 90 % of lead particles in ambient air are of sufficiently small size to be easily deposited and retained in the lungs. Absorption of retained lead trough the alveoli is efficient and complete (Klaassen 2001).

Even at very low dose levels, lead is a very toxic element. Brain damage, kidney damage, and gastrointestinal distress are seen following acute exposure to high levels of lead in humans. Chronic exposure to lower levels of lead in humans results in effects on the blood, central nervous system, blood pressure, kidneys, and vitamin D metabolism. Children are particularly sensitive to the chronic effects of lead, with slowed cognitive development and reduced growth. Reproductive effects, such as decreased sperm count in men and spontaneous abortions in women, have been associated with lead exposure. The developing fetus is at particular risk from maternal lead exposure, with low birth weight and slowed postnatal neurobehavioral development. Human studies have been inconclusive in regards to lead exposure and the development of cancer, while animal studies have shown an increase in kidney

cancer from lead exposure by the oral route. As a result of these findings, USEPA has classified lead as a Group B2, probable human carcinogen (EPA 1994e).

Since the introduction of lead-free gasoline, concentrations of lead in air have been usually less than 1.0 ug/m³ in most industrialized cities. In NAF Atsugi, the maximum 24-hour concentration at any sampling location was 20 ug/m³ with a corresponding average 24-hour concentration of 0.26 ug/m³. The USEPA NAAQS for lead is 1.5 ug/m³ and is based on a quarterly average that is not to be exceeded (EPA 2001a). Although the maximum and average 24-hour concentrations cannot be directly compared with the NAAQS, it is very likely that lead concentrations exceeded this value. Also, as previously mentioned in the exposure assessment section of this report, since lead concentrations were determined from particulate matter less than 10 microns in diameter, instead of total suspended particulates, the measured lead concentrations could be significantly underestimated.

Due to toxicological concern and potential exposure to lead in NAF Atsugi, lead was included in the risk assessment qualitatively. The traditional risk assessment approach for evaluating non-carcinogenic effects from exposure to chemicals of concern (COCs) involves comparison of chemical intakes to a reference dose (RfD). This approach could not be taken with lead because a NOAEL for lead has not been identified, and therefore there is no RfD for lead. In its place, non-carcinogenic risks for lead exposures were evaluated using USEPA's Integrated Exposure Uptake Biokinetic Model for Lead (IEUBK) (EPA 1994f). Blood lead concentrations are accepted as the preferred measure of chronic low level lead exposures. Blood lead concentrations provide an index for evaluating the likelihood of adverse effects from lead exposure. A blood lead level of $10~\mu g/dL$ has been identified by the Centers for Disease Control as the target action level for evaluating exposure to lead. The USEPA defines a greater-than-5 percent probability of exceeding the $10~\mu g/dL$ criterion value as posing an unacceptable threat to human health.

The site-wide RME concentrations of lead in soil and air for NAF Atsugi (i.e., 26.5 mg/kg and 3.9 ug/m³, respectively) were evaluated using the IEUBK model to determine the potential for health effects associated with exposure to lead. The results of the modeling effort indicated that there is a 0.02% probability of a blood lead level of 10 μ g/dL at NAF Atsugi for children. This value is well below the Centers for Disease Control target action level of greater-

than-5 percent probability. Furthermore, the Atsugi Branch Medical Clinic was contacted regarding pediatric blood lead levels measured in children as part of the Pediatric Lead Poisoning Prevention Program (PLPP). The current PLPP policy is to administer a "lead risk" questionnaire to parents rather than to routinely draw pediatric blood for testing. Family practice physicians review the questionnaire and, based on the results, order blood lead testing for children who are potentially at risk. Of the 372 children tested under the Pediatric Lead Poisoning Prevention Program (PLPP) at Atsugi between 1995 – 1999, one child (over 6 years old) who lived on base, was found to have a blood lead between $10 - 19 \mu g/dL$ in 1997. The Department of Defense policy on the assessment of health risk from lead is that blood lead levels of 10-19 ug/dL require confirmatory blood lead determination within one month of the first result. Confirmed 10-19 ug/dL blood lead results require a reassessment of the risk factors for exposure education concerning diet and personal hygiene. If levels persist, the policy requires the initiation of individual case management, environmental investigation, and lead hazard abatement. Rescreening is also required every 3 months. Upon confirmatory blood lead determination, the child was found to have a blood lead level of less than 10 ug/dL, ruling out pediatric lead poisoning.

4.9 Chemicals of Concern Measured in the Air With Potential Reproductive and Developmental Effects

In addition to the chemicals discussed already, the following chemicals of concern have potential reproductive and developmental effects: acetonitrile, acrylonitrile, arsenic, benzene, benzyl chloride, cadmium, carbon tetrachloride, 1,2-dibromoethane, 1,4-dichlorobenzene, methylene chloride, 1,4-dioxane, 1,1,2,2-tetrachloroethane, 1,2,4-trimethylbenzene, and vinyl chloride.

Acetonitrile

Acetonitrile contributed up to 6 % of the non-carcinogenic risk at NAF Atsugi by inhalation. The average 24-hour concentration of acetonitrile at the site of the maximum 24-hour sample was 0.01 mg/m³, which is less than the inhalation RfC of 0.06 mg/m³ (EPA 1999e). The USEPA estimates that inhalation of this concentration or less, over a lifetime, would not likely result in the occurrence of chronic, non-cancer effects. There is no information available on the reproductive or developmental effects of acetonitrile in humans. However, animal studies

suggest that acetonitrile may cause developmental and reproductive effects such as a decrease in average fetal body weight and a significant increase in the number of malformed offspring (EPA 1987d).

Acrylonitrile

Acrylonitrile contributed 2 to 3 % of the carcinogenic risk at NAF Atsugi by inhalation. The average 24-hour concentration of acrylonitrile at the site of the maximum 24-hour sample was 0.0003 mg/m3, which is less than the inhalation RfC of 0.002 mg/m3 (EPA 1998e). The USEPA estimates that inhalation of this concentration or less, over a lifetime, would not likely result in the occurrence of chronic, non-cancer effects. There is no information available on the reproductive or developmental effects of acrylonitrile in humans. However, fetal malformations have been reported in rats exposed to acrylonitrile by inhalation. In mice orally exposed to acrylonitrile, degenerative changes in testicular tubules and decreased sperm count were observed (ATSDR 1990).

Arsenic

Arsenic contributed 1 to 2 % of the carcinogenic risk at NAF Atsugi by inhalation. The average 24-hour concentration of arsenic at the site of the maximum 24-hour sample was 2.4E-06 mg/m³. An RfC is not available for comparison at this time. Human data suggest a relationship between inhalation exposure to inorganic arsenic in humans and an increased risk of reproductive effects, such as spontaneous abortions. However, these data are not sufficient to suggest a cause and effect relationship in humans. Oral animal studies have reported inorganic arsenic to be fetotoxic and to cause birth defects (ATSDR 1989b).

Benzene

Benzene contributed 3 to 6 % of the carcinogenic risk at NAF Atsugi by inhalation. The average 24-hour concentration of benzene at the site of the maximum 24-hour sample was 0.0038 mg/m³. An RfC is not available for comparison at this time. Menstrual disorders and a decreased size of ovaries have been observed in women occupationally exposed to high levels of benzene. Several occupational studies suggest that benzene may impair fertility in women exposed to high levels. However, these studies are limited due to lack of exposure history, simultaneous exposure to other substances, and lack of follow-up. Available human data on the

developmental effects of benzene are inconclusive due to concomitant exposure to other chemicals, inadequate sample size, and lack of quantitative exposure data. Adverse effects on the fetus, including low birth weight, delayed bone formation, and bone marrow damage, have been observed where pregnant animals were exposed to benzene by inhalation (ATSDR 1991a).

Benzyl Chloride

Benzyl chloride contributed 1 to 9 % of the carcinogenic risk at NAF Atsugi by inhalation. The average 24-hour concentration of benzyl chloride at the site of the maximum 24-hour sample was 0.002 mg/m³. An RfC is not available for comparison at this time. There are no studies regarding developmental or reproductive effects in humans. In one rat study in which rats were given benzyl chloride orally, increases in embryonic mortality were observed along with retarded development of the offspring (EPA 1986).

Cadmium

Cadmium contributed up to 6 % of the carcinogenic risk at NAF Atsugi by inhalation. The average 24-hour concentration of cadmium at the site of the maximum 24-hour sample was 6E-06 mg/m³. An RfC is not available for comparison at this time. Cadmium has been shown to be a developmental toxicant in animals, resulting in fetal malformations and other effects, but no conclusive evidence exists in humans. These effects could be seen from acute, as well as chronic exposures (ATSDR 1992a).

Carbon Tetrachloride

Carbon tetrachloride contributed 1 to 2 % of the carcinogenic risk at NAF Atsugi by inhalation. The average 24-hour concentration of carbon tetrachloride at the site of the maximum 24-hour sample was 6.4E-04 mg/m³. An RfC is not available for comparison at this time. There is no information available on the reproductive or developmental effects of carbon tetrachloride in humans. Reproductive effects, such as decreased fertility in rats, decreased sperm production in male rats, degenerative changes in the testes, and a decreased survival rate of newborns, have been observed in animals exposed to carbon tetrachloride orally and by inhalation. Birth defects have not been observed in animals (ATSDR 1992b).

1,2-Dibromoethane

1,2-Dibromoethane contributed 2 to 7 % of the carcinogenic risk and 2 to 3 % of the non-carcinogenic risk at NAF Atsugi by inhalation. The average 24-hour concentration of 1,2-dibromoethane at the site of the maximum 24-hour sample was 1.7E-04 mg/m³, which is less than the provisional RfC of 2E-04 mg/m³. USEPA estimates that inhalation of this concentration or less, over a lifetime, would not likely result in the occurrence of chronic, non-cancer effects. Developmental effects have not been documented in humans exposed to 1,2-dibromoethane. Limited data on men occupationally exposed to it at concentrations much higher than found at NAF Atsugi indicate that long-term exposure can impair reproduction by damaging sperm cells in the testicles. Animal studies have demonstrated reproductive and developmental effects from 1,2-dibromoethane. Animals that breathed or ate food containing it for short or long periods were less fertile than control animals or had abnormal sperm. Pregnant animals that were sick from exposure to 1,2-dibromoethane have had pups with birth defects (ATSDR 1992c).

1,4-Dichlorobenzene

1,4-Dichlorobenzene contributed 1 to 3 % of the carcinogenic risk at NAF Atsugi by inhalation. The average 24-hour concentration of 1,4-dichlorobenzene at the site of the maximum 24-hour sample was 1.4E-03 mg/m³, which is less than the provisional RfC of 0.8 mg/m³. USEPA estimates that inhalation of this concentration or less, over a lifetime, will not likely result in the occurrence of chronic, non-cancer effects. Information on the reproductive or developmental effects of 1,4-dichlorobenzene in humans is not available. In one animal study, exposure of pregnant rats to 1,4-dichlorobenzene via inhalation did not result in developmental effects in the offspring. In another study, an increase in the incidence of an extra rib was reported in the fetuses of pregnant rats administered 1,4-dichlorobenzene by gavage, i.e., placing the chemical experimentally in the stomach (ATSDR 1993a).

Methylene chloride

Methylene chloride contributed up to 1 % of the carcinogenic risk at NAF Atsugi by inhalation. The average 24-hour concentration at the site of the maximum 24-hour sample was 0.01 mg/m³. An RfC is not available for comparison at this time. There are no studies regarding developmental or reproductive effects in humans from inhalation or oral exposure. Animal

studies have demonstrated that methylene chloride crosses the placental barrier, and lowered fetal body weights have been noted (ATSDR 1991b).

1,4-Dioxane

1,4-Dioxane contributed 1 to 2 % of the carcinogenic risk at NAF Atsugi by inhalation. The average 24-hour concentration at the site of the maximum 24 hour sample was 9.5E-04 mg/m³. A RfC is not available for comparison at this time. Information is not available on the reproductive and developmental effects in humans. There is no evidence of gross, skeletal, or visceral malformations in the offspring of rats exposed via gavage. Embryo toxicity was observed only at the highest dose (EPA 1987e).

1,1,2,2-Tetrachloroethane

1,1,2,2-Tetrachloroethane contributed 2 to 5 % of the carcinogenic risk at NAF Atsugi by inhalation. The average 24-hour concentration at the site of the maximum 24-hour sample was 4.2E-04 mg/m³. An RfC is not available for comparison at this time. Information is not available on the reproductive and development effects in humans from inhalation or oral exposure. Animal studies have not reported reproductive effects from inhalation exposure, while an oral study in rats reported histopathological changes in the testes (ATSDR 1987).

1,2,4-Trimethylbenzene

1,2,4-Trimethylbenzene contributed 1 to 2 % of the non-carcinogenic risk at NAT Atsugi by inhalation. The average 24-hour concentration at the site of the maximum 24-hour sample was 2E-03 mg/m³. An RfC is not available for comparison at this time. Information is not available on the reproductive and developmental effects in humans. Results of testing in animals indicate that the C9 fraction of petroleum, which contains 1,2,4-trimethylbenzene, can adversely affect the reproductive system and the developing offspring (EPA 1994g).

Vinyl chloride

Vinyl chloride contributed 1 to 2 % of the carcinogenic risk at NAF Atsugi by inhalation. The average 24-hour concentration at the site of the maximum 24-hour sample was 1E-04 mg/m³, which is less than the RfC of 1E-01 mg/m³. USEPA estimates that inhalation of this concentration or less, over a lifetime, will not likely result in the occurrence of chronic, non-cancer effects. Human studies have suggested an association between men occupationally

exposed to vinyl chloride and miscarriages in their wives' pregnancies although other studies have not supported these findings. Several human studies have reported an association between vinyl chloride exposure in pregnant women and an increased incidence of birth defects, while other studies have not supported these findings. Several case reports suggest that male sexual performance may be affected by vinyl chloride. However, these studies are limited by lack of quantitative exposure information and possible concomitant exposure to other chemicals. Animal studies have reported decreased fetal weight and birth defects at levels that are also toxic to maternal animals in the offspring of rats exposed to vinyl chloride through inhalation. Testicular damage to rats exposed for up to 12 months has been reported at levels as low as 10 ppm. It would be prudent to consider vinyl chloride as posing both reproductive and developmental hazards (ATSDR 1993b).

4.10 Health Outcome Studies at NAF Atsugi

Evaluation of Pregnancy Loss at NAF Atsugi Japan (June 1995- May 1998)

As documented earlier (NEHC 1999a) and presented as Appendix G in this report, a study designed to describe the rate of miscarriage at NAF Atsugi and other naval facilities in Japan was conducted in the summer of 1998. Hospital and clinic records for Navy personnel and their dependents, which were pregnant and living in Japan at some time between June 1995 and May 1998, were examined. Information used to calculate miscarriage rates came from delivery logs at U.S. Naval Hospital, Yokosuka (NHY), pathology records at NHY, and the prenatal log at Atsugi Branch Medical Clinic. Data were collected on the number of live births and the number of miscarriages (unintentional pregnancy loss at up to the 28th week of pregnancy). The miscarriage or pregnancy loss rate was defined as the number of miscarriages divided by the total number of pregnancies examined (the number of babies born plus the number of miscarriages).

A total of 1862 pregnancies with known outcomes from NHY (including Atsugi, Yokosuka, Sasebo, and Iwakuni) were examined. There were 1701 live births and 130 miscarriages between June 1995 and May 1998. The corresponding miscarriage rate for this period was 7.1 %. The rate at NAF Atsugi, determined from review of the prenatal log during the same period, was 8.8 %. This rate was based on the examination of 353 total pregnancies, with 322 live births and 31 miscarriages. The average range of miscarriages in the U.S.

population is 10 % to 15 % for recognized pregnancies. The results of this study suggest that the occurrence of miscarriage at NAF Atsugi and other naval facilities within Japan is at the low end of the expected range described for the population of the United States.

Evaluation of Children's Respiratory Health at NAF Atsugi

PM₁₀ was monitored during a health study (Appendix H) to determine if air pollutants from the SIC in NAF, Atsugi, were affecting the respiratory health of children attending school at Atsugi, compared to those at Yokosuka, Japan. The 24-hour maximum PM₁₀ concentration measured during the four-week study period in 1998 was 0.105 mg/m³. Data review indicated no unusual differences between the respiratory health of the Atsugi children from the Yokosuka children, who lived approximately 50 km away from the SIC. Pulmonary function testing indicated normal lung function. The only difference in respiratory symptoms between the groups was a higher incidence of rhinorrhea reported by Atsugi children, which might be attributed to the irritant effects of the emissions from the SIC (NEHC 1999b).

4.11 Discussion of Acceptable Risk – Policy versus Science

During the late 1970's and early 1980's regulatory agencies in the U.S. and abroad frequently adopted a cancer risk criteria of one-in-one million (1E-06) as a negligible risk when fairly large populations might be exposed to a suspect carcinogen. The Food and Drug Administration (FDA) developed this concept. Other federal agencies have also used a one-in-one-million increased risk over a lifetime as a reasonable criterion for separating high risk problems warranting agency attention from negligible risk problems that do not.

The public and the media often misunderstand this risk level, which is not an actual risk. For example, the FDA does not expect one out of every million people to get cancer if they drink decaffeinated coffee, which may contain potentially carcinogenic chemical compounds. Rather, it is a mathematical risk based on scientific assumptions used in risk assessment. In developing this concept, the FDA used a conservative estimate (health protective) to ensure that the risk is not underestimated. This conservative estimate was necessary because animal test results must be interpreted conservatively, and uncertainty factors applied when we extrapolate risks to humans. When the FDA uses the risk level of 1E-06 (1 in 1,000,000), it is confident that the risk to humans is virtually non-existent. A one-in-one million-cancer risk estimate, which is often

assumed by some policy-makers to represent a trigger level for regulatory action, actually represents a level of risk that is so small as to be of negligible concern.

The other misperception within the risk assessment arena is that all environmental regulations have as their goal a theoretical maximum cancer risk of 1E-06. Travis et al (Travis 1987) conducted a retroactive examination of the level of risk that triggered regulatory action in 132 federal regulatory decisions. The results of his examination revealed that every chemical with an individual lifetime risk above 4E-03 (4 in 1,000) received regulation, while those with values below 1E-06 remained unregulated. For small populations, regulatory action never resulted for individual risks below 10⁻⁴. However, for potential effects resulting from exposures to the entire U.S. population, a risk level below 10⁻⁶ never triggered action. Above 3E-04, it always triggered action.

U.S. Federal and most State regulatory agencies have adopted the 1E-06 (1 in 1,000,000) cancer risk as being of negligible concern in situations where large populations (for example, 200 million people) are involuntarily exposed to suspect carcinogens (for example, food additives). When smaller populations are exposed to suspect carcinogens, theoretical cancer risk of up to 1E-04 have (1 in 10,000) been considered acceptable.

The USEPA National Contingency Plan (EPA 1992b) set the acceptable risk range between 1E-04 (1 in 10,000) and 1E-06 (1 in 1,000,000) at hazardous waste sites regulated under the Comprehensive Environmental Response, Compensation and Liability Act (CERCLA). For non-cancer effects, the National Contingency Plan has adopted that a Hazard Index less than 1 is of no concern. A Hazard Index of 1 means that for systemic contaminants, acceptable exposure levels represent concentration levels to which the human population, including sensitive subgroups, may be exposed without adverse effect during a lifetime or part of a lifetime, incorporating an adequate margin of safety. both costs and benefits of anticipated courses of action should be thoroughly evaluated. However, actual data and risk estimates of common human activities, regulatory precedent, and the relationship between the magnitude and variance of background and incremental risk estimates all provide convincing support for the adoption of the *de minimis* risk level of 1E-05 (1 in 100,000) for regulatory purposes.

However, to arrive at its decision-making, the USEPA evaluates environmental risk through an assessment process that uses a considerable amount of scientific data and analysis

with much judgment and uncertainty. The evaluation of risks from exposure to environmental agents through this process concludes in a characterization of risks. According to the Presidential Commission on Risk Assessment and Risk Management (CRARM 1997a, CRARM 1997b) "Many risk characterizations have relied primarily on mathematical estimates of risk to communicate risk assessment findings often conveying an unwarranted sense of precision while failing to cover the range of scientific opinions. They are particularly difficult for audiences unfamiliar with risk assessment to comprehend. Effective risk management is impeded without effectively communicating information about who is at risk, how they might be affected, what the severity and reversibility of adverse effects might be, how confident the risk assessors are in their prediction and other qualitative information that is critical to decision making."

The National Academy of Sciences (NAS 1996) refers to the risk characterization as "the process of organizing, evaluating and communicating information about the nature, strength of evidence and the likelihood of adverse health or ecological effects from particular exposures". Their emphasis on fully characterizing the scope, uncertainties, limitations, and strengths of the risk assessment and on the social aspect of interacting with decision makers and stakeholders in a iterative, analytic and deliberative process is to ensure that the assessment will be useful for the purposes for which the risk assessment was intended and that it will be understood. This is where policy based decision-making ends and the science-based decision-making begins.

At NAF Atsugi we could not solely rely on determination of risk according to regulatory policy, especially considering the fact that environmental pollution causing health concerns was generated in a foreign country where USEPA regulatory policy did not apply. Therefore, in addition to the mathematical estimate of risk, the science based determination of risk which considers uncertainties, limitations, and strengths of the risk assessment, must be achieved to ensure that the risk managers have adequate health risk information input for their risk management decisions.

Section 5 — Risk Assessment Summary of Findings and Conclusions

5.1 Risk Assessment Input Information

NEHC directed a comprehensive health risk assessment at NAF Atsugi, Japan, to estimate the potential human health risks of individuals living and working at NAF Atsugi, resulting from exposure to constituents of concern (COCs) in soil, ambient air, indoor air, and indoor dust associated with ambient air emissions and/or subsequent deposition from point and non-point sources impacting the air quality. Another objective of the final health risk assessment was to estimate the contribution of the risk attributable to the SIC.

This risk assessment focused on exposure scenarios applicable to each of the areas of concern (AOCs) likely to be frequented by NAF Atsugi community members, to include sensitive receptors (i.e., children), as described in the Exposure Assessment Section of the *NAF Atsugi, Japan, Human Health Risk Assessment* developed by Pioneer (Pioneer 2000). These are scenarios representative of exposed populations who worked, spent time or lived at the Child Development Center, the Elementary School, the Residential Towers, the Ground Electronics Maintenance Building or the Golf Course. These locations were considered to be AOCs because they were in close proximity to the SIC where children and adults spent most of their time.

The health risk was estimated using the methodology from the Superfund Risk Assessment Guidance for Superfund (EPA 1989). The primary reason that this methodology was selected is because it is the standard approach used in the United States for calculating risk; therefore providing a basis for comparison with risks calculated at DoD activities in the U. S. Additionally, USEPA risk assessment principles and practices draw on many sources, including the environmental laws administered by USEPA, the National Research Council's 1983 report on risk assessment, the USEPA's Risk Assessment Guidelines, and various program specific guidance (e.g., the Risk Assessment Guidance for Superfund). The regulatory framework for performing human health risk assessments has been established through a series of guidance documents issued by USEPA and other regulatory agencies since the early 1980s. Through these documents, guidelines for performing both qualitative and quantitative human health risk assessments have been defined. The exposed individuals that were considered included an adult and a child in a *residential* scenario; adult workers in an *industrial* scenario; a golfer in a *recreational* scenario; and an adult working at or a child attending the day care or elementary

school. Site specific information regarding average and reasonable maximum exposure (RME) air concentrations, exposure frequency and duration, and USEPA default values for body weight and averaging time were used to calculate the cancer risk and non-cancer hazard index.

The health risk assessment was conducted using air monitoring data and soil samples collected over a 14 month time period. Sampling plans took into account information from the two previous screening risk assessments.

5.2 Exposure Assessment and Risk Characterization

Health risks were calculated for adults and children living on the base for 3 years and 6 years, representing one and two tours of duty, and 30 years to represent the USEPA residential default scenario, for comparison purposes. Industrial, residential, day care, elementary school, and recreational exposure scenarios were evaluated. Site-specific exposure parameters were used in the health risk assessment.

Human activity patterns were determined in conjunction with a multi-pathway analysis, to calculate population specific health risks. The sampling scheme and use of realistic exposure parameters resulted in the collection of data best representative of actual site conditions, to as accurately as possible, assess potential health risks to the NAF Atsugi community.

Cancer Risk

The calculated carcinogenic risk for a child living at the Residential Tower for 3 or 6 years or an individual living at the Residential Towers for 30 years (residential scenarios) was above the USEPA acceptable cancer risk range of 1 x 10⁻⁴ (1 in 10,000) to 1x 10⁻⁶ (1 in 1,000,000). The carcinogenic risk for an adult working at the GEMB (industrial scenario) for 30 years was also above the USEPA acceptable cancer risk range. The highest cancer risk calculated was 5.2 x 10⁻⁴ (5.2 in 10,000) at the Residential Towers, assuming a 30-year residential exposure. The inhalation exposure pathway accounted for greater than 80% of the carcinogenic risk, at each location, with the exception of the Golf Course.

Acetaldehyde, 1,3-butadiene, and 2,3,7,8-TCDD TEQs (dioxins) were responsible for the majority of the carcinogenic risks at all five AOCs.

Non-Cancer Risk

The non-carcinogenic hazard indices were greater than the USEPA acceptable Hazard Index of 1, for every exposure scenario, except for the Recreational Golfer Scenario. For every exposure scenario, except the Recreational Golfer Scenario, the segregated hazard indices, for respiratory effects alone, were greater than the acceptable Hazard Index of 1. The highest average hazard index of 53, calculated for the "average" exposed individual, was observed at the Residential Towers for the 3 and 6-year child resident. The highest hazard index calculated for the reasonable maximum exposed individual was 67, calculated for a child resident living at the Residential Towers for 3 or 6 years. The inhalation exposure pathway accounted for greater than 95% of the hazard index at each location.

Acrolein, acetaldehyde, PM₁₀ and formaldehyde comprised approximately 91% of the non-cancer health effects. Health effects that could be anticipated from exposure to the specific concentrations of these chemicals, measured at NAF Atsugi, were respiratory system related.

Base-Wide Risk Estimates

Since 24-hour risk estimates were only conducted for the residential scenario, calculations were performed to compare the risks for each AOC on a 24-hour exposure basis. Since the inhalation pathway was responsible for the majority of the risk at each location (e.g., $\geq 85\%$), the plausible upper bound inhalation risks for individuals remaining on base 24 hours each day was determined only considering the air pathway. When a comparison of the air pathway health risk was performed at the Elementary School, Residential Towers, Golf Course, Child Development Center and Ground Electronics Maintenance Building, under identical exposure assumptions, the results indicated that the plausible upper bound risk estimate was similar at all locations on base. This indicated that the risks for children and adults, routinely spending 24 hours per day on base (e.g., working on base or going to school on base and living on base, as well), is expected to be similar to the cancer and non-cancer risks calculated for the Residential Towers.

Ambient air dispersion modeling indicated that the maximum impacts occurred just north of the SIC, in the vicinity of the GEMB. The lowest modeled impacts occurred east of the SIC on the eastern NAF Atsugi property line. Using emission rates, which were back calculated from the ambient air monitoring samples, the dispersion modeling predicted air concentrations of total

2,3,7,8-TCDD TEQs, at levels higher than RBCs across the entire base. Based on the modeling, arsenic concentrations exceeded the RBCs across approximately two thirds of the base. Lead and PM₁₀ concentrations were greater than the quarterly and annual USEPA NAAQS, respectively, in a small area north of the SIC. Hydrochloric acid (HCl) concentrations were below the RBC at all locations (Radian, 2000).

5.3 SIC Risk Contribution

The ability to statistically associate all of the specific chemicals, known to be related to incineration, with SIC operations were limited. Factors such as variable feedstock, variable SIC operating conditions, and different meteorological conditions may have prevented the statistical methods from identifying a greater number of pollutants associated with the SIC. To determine which specific chemicals would be associated with emissions from the SIC, three statistical evaluations were performed by Radian and Research Triangle Institute (RTI). These evaluations determined if a relationship existed between individual chemical concentrations and the amount of time a site was downwind of the SIC (i.e., % downwind).

The statistical analysis conducted by Radian identified only six chemicals likely to be associated with SIC operations. These chemicals were hydrochloric acid, 2,3,7,8 TCDD, lead, cadmium, arsenic, and PM₁₀. Statistical analyses performed by RTI on the top thirty-two chemicals determined to be contributing the greatest risk (risk drivers) in the risk assessment indicated that arsenic, benzene, cadmium 2, 3, 7, 8 TCDD TEQ, 1,2-dichloropropane, lead and PM₁₀ showed an association with the SIC. Other risk drivers, including 1,3-butadiene, acetaldehyde, acetophenone, chloromethane, and dieldrin showed a possible association with the SIC.

Comparison between the risks from a site that was downwind of the SIC (i.e. the GEMB) with the risks for a site that was upwind of the SIC (i.e., the Golf Course) indicated that the cancer and non-cancer risks were significantly higher at the downwind location (the GEMB) than the upwind location (Golf Course). Acetaldehyde, acetonitrile, acrolein, and PM₁₀ contributed the majority of the hazard index at each location. The most significant increase in risk at the downwind location was observed for acrolein. The hazard index increased from 15.6 at the upwind location to 63.1 at the downwind location. Acetaldehyde, 1,3-butadiene, hexachlorobutadiene, and total 2,3,7,8-TCDD TEQs were responsible for the majority of the

cancer risks at the Golf Course. Cadmium and total 2,3,7,8-TCDD TEQs were responsible for the majority of the risks at the GEMB. The most significant contributor of cancer risk shown in this comparison was total dioxins/furans. The cancer risk at the GEMB was calculated to be one and one-half orders of magnitude higher than that at the Golf Course, further indicating the SIC as a major source of dioxins/furans.

A soil trend analysis for emission deposition also indicated that the SIC appeared to have affected the distribution of chemicals in surface and subsurface soil at NAF Atsugi. A definite footprint of dioxin deposition associated with air emissions from the SIC was evident in the way dioxin concentrations decreased with increasing distance from the incinerator.

A list of chemicals statistically associated with the SIC, the increase in cancer risk by more than one order of magnitude and non-cancer hazard index downwind from the SIC, air dispersion modeling results and the soil trend analysis, together, indicated that emissions from the SIC significantly contributed to the poor air quality at NAF Atsugi.

5.4 Context with Superfund

The results of the final health risk assessment were similar to the results of the two previous screening risk assessments (NEHC 1995 and NEHC 1998). The results indicated that a child's exposure to contaminants from air and soil during a 3-year tour of duty could potentially result in a cancer risk as high as of 1 in 10,000 (1 x 10⁻⁴ level) above the current lifetime background rate of cancer (1 in 3 females and 1 in 2 males) in the U. S. population. They also indicated that adults would not likely reach this level even with two tours of duty (6 years of exposure). The National Oil and Hazardous Substance Pollution Contingency Plan (NCP) (USEPA 1992b) discusses the risk range of 1 x 10⁻⁴ (1 in 10,000) to 1 x 10⁻⁶ (1 in 1,000,000) as generally being acceptable. USEPA generally uses the upper boundary of the risk range, 1 x 10⁻⁴ (1 in 10,000), as the point at which risk management decisions are made. Action to reduce risk is generally warranted when the cumulative site risk to an individual using reasonable maximum exposure assumptions exceeds the 1 x 10⁻⁴ (1 in 10,000) lifetime excess cancer risk. A risk manager may also decide that a lower level of risk to human health is unacceptable, due to site-specific reasons, and that action to reduce risk is warranted.

The non-cancer Hazard Index (HI) was higher than 1 for both children and adults, indicating concern for potential non-cancer health effects. According to USEPA guidance

(USEPA 1989), concentrations generating a HI above 1 indicate that there could be concern for potential non-cancer health effects to occur within a population. An HI of 1 should not be considered a bright line, which triggers remedial action, but rather indicates some degree of concern and the need for professional judgment to further characterize the potential for health effects to occur.

5.5 Context with Health Effects

Acute Health Effects

The maximum 24-hour concentrations of all ambient chemicals detected at NAF Atsugi were compared to the Agency for Toxic Substances and Disease Registry (ATSDR) Maximum Risk Levels (MRLs) and all other acute health based concentration levels found in the literature. The only chemical found to exceed any acute health based concentration level was acrolein. The acute MRL for acrolein is based on eye irritation. The only known effects of acrolein exposure in humans are general respiratory congestion and eye, nose, and throat irritation. Persons with pre-existing eye, skin, respiratory, allergic, asthmatic or heart diseases might be at increased risk due to acrolein exposure above the acute MRL. Individuals with cystic fibrosis and asthma, especially children, should be excluded from acrolein exposure greater than the MRL. Although acrolein was the only chemical found to exceed the acute inhalation MRL, exposures were not limited to a single chemical, but a mixture of chemicals. Research on the effects of exposure to a mixture of chemicals is not complete; therefore, when two or more chemicals act on the same organ system, their combined effect should be considered as additive, unless there is evidence to the contrary. The health effects resulting from exposure to acrolein are likely to be reversible; however, combination with other chemicals such as particulate matter (PM₁₀ and PM_{2.5}), acetaldehyde, and formaldehyde is cause for concern. Repeated long-term exposure to these chemicals in combination may result in irreversible effects due to mucosal membrane irritation.

Children As Sensitive Populations

Since children differ from adults in their exposure to contaminants in the air and soil and in their susceptibility to harm from these exposures, the calculated carcinogenic and non-carcinogenic risks were greater for children than for adults. The risk calculations use different assumptions for children due to physiological, biochemical, and behavioral differences from adults. The results of the risk assessment suggested that the exposure of a child resident to

contaminants from air and soil at NAF Atsugi, during a 3-year tour of duty, could potentially result in a cancer risk as high as 1 x 10⁻⁴ (1 in 10,000) above the current lifetime background rate of cancer in the population. The HI's for children were higher than the HI's for adults; but the HI's for both populations were much greater than one. Infants and children are known to be among the most susceptible members of society to the adverse health effects from outdoor air pollution. Children are susceptible to certain particulate matter effects, including: increased respiratory symptoms; decreased lung function; an increased susceptibility to respiratory infections; and the aggravation of existing respiratory diseases, such as asthma and chronic bronchitis. Through a health study, included as Appendix H, a higher incidence of rhinorrhea (runny nose/post nasal drip) was reported by Atsugi school children. Environmental toxins of particular concern for infants and children that were measured at NAF Atsugi and were major risk drivers, included heavy metals, such as lead, cadmium, and arsenic, and the group of compounds known as dioxins.

5.6 Context With Risk Comparisons

Indoor Air

A comparison between average indoor air concentrations found at NAF Atsugi and in U.S. homes indicated that, in general, indoor air concentration averages for most of the chemicals, for which data was available in U.S. homes, were lower or within the same range at NAF Atsugi. The higher concentrations of metals found in the indoor air at NAF Atsugi could be due to deposition from the incinerator onto the ground that was tracked indoors, via shoes, and then re-suspended in the air. Another potential source of metals in indoor air, especially cadmium, arsenic and lead, which were strongly associated with the SIC, was transportation of particulates in ambient air indoors via open windows or doors.

Ambient Air

The comparison between ambient air concentrations of criteria pollutants, in major cities in the U.S. and NAF Atsugi, indicated that levels of carbon monoxide at NAF Atsugi were low in comparison to U.S. cities and only slightly higher than Honolulu. The levels of nitrogen dioxide were similar to the levels found in Houston, Baltimore and Atlanta. Ozone levels at NAF Atsugi were considered elevated, but no more elevated than levels found in Atlanta and

Baltimore. PM_{10} levels at NAF Atsugi were 66 mg/m³ and were slightly elevated in comparison with 45 mg/m³ in Los Angeles, the highest level in the U.S. cities used as a comparison.

Some VOCs and aldehydes/ketones concentrations were higher in the U.S. and some were higher at NAF Atsugi. For some of the chemicals, the average was higher in the U.S. but the maximum concentration was higher at NAF Atsugi. The concentrations of aldehydes and ketones at NAF Atsugi appeared to be somewhat greater than those in the U.S. With the exception of lead, the metals measured at NAF Atsugi were lower than concentrations found in Midlothian Texas, a very highly industrialized area in the U.S. Concentrations of dioxin and lead were clearly orders of magnitude greater at NAF Atsugi than in the U.S.

Comparison With Incinerators In The U.S.

In the U. S., because of community opposition to incineration and strict USEPA emission standards, most of the incinerator proposals developed in the last five years have been abandoned, and many incinerators have been shut down due to violations of the Clean Air Act.

The Columbus Waste to Energy Facility, Ohio, incinerator was shut down in the mid 1990s. It was considered to be the number one dioxin emitting trash incinerator. Dioxin ambient air monitoring conducted over two 48-hour periods in March and April of 1994 showed a maximum ambient air monitored dioxin concentration of 0.352 picograms TEQ/m³ measured at a distance of approximately 2 kilometers to the east of the Facility. In September 1994, the USEPA finding that the facility "may present an imminent and substantial endangerment" to public health, ordered Columbus to stop running the facility until it completed a major overhaul of its pollution control system. On November 1, 1994, faced with having to spend at least \$65 million for the upgrades, the governing board for the plant voted unanimously to stop the burning of garbage in Columbus.

At NAF Atsugi, dioxin ambient air monitoring was conducted every 6 days over 14 months from April 1998 to June 1999. An average ambient air concentration of 1.57 picograms TEQ/m³ was measured within a 1.5 kilometers radius from the SIC and an average ambient air concentration of 3.49 picograms TEQ/ m³ within 300 meters. By comparison with the Columbus Waste to Energy Facility, the SIC, an incinerator with uncontrolled emissions and high dioxin emission levels, would have been forced to either shut down or implement controls to come into compliance and mitigate the risk of health hazards caused by its uncontrolled emissions.

5.7 Limitations and Uncertainties

The risk estimates presented in this risk characterization are limited by the uncertainties inherent in the models used to estimate risk and in the data used in the models. NEHC attempted to minimize uncertainties by:

- a) Collecting site-specific air monitoring data every 6 to 12 days, for 14 months to account for any variability due to the day of the week, season, or other temporal effects.
- b) Selecting sampling methods based on their ability to collect samples with sufficiently low detection limits to perform health-based risk analysis.
- c) Evaluating sampling data throughout the monitoring period to ensure accurate analytical data capture and to ensure that the data quality objectives were being met. This was accomplished by quarterly data evaluations, meetings and peer reviews, equipment and procedure audits, and analytical lab audits.

However, the uncertainty associated with the identification of COCs in indoor air and indoor dust could not be minimized since ambient air concentrations were used as surrogate indoor air concentrations for quantitative evaluation in the risk assessment. This may have resulted in an underestimation of the risk

Standard uncertainties associated with the exposure assessment are the use of site specific and USEPA Standard Default assumptions regarding body weight, period exposed, life expectancy, population characteristics, and lifestyle, which may not be representative of the exposed population. Therefore the risks may have been under- or over-estimated.

In this HRA, since oral RfDs were only available for 95 of the 246 chemicals of concern (39%) and inhalation RfDs were only available for 58 of the 246 chemicals of concern (24%), the non-carcinogenic risks were likely underestimated. Similarly, since oral slope factors were available for only 44 of the 246 (18%) chemicals of concern and inhalation slope factors were available for only 43 of the 246 (17%) chemicals of concern, the cancer risk may have resulted in an underestimation of the cancer risk. Standard uncertainties in risk characterization result from an assumption that health effects of chemicals are additive, and the non-evaluation of antagonistic and synergistic effects of chemical mixtures.

This risk assessment focused on exposure scenarios applicable to each of the AOCs likely to be frequented by NAF Atsugi community members, including sensitive receptors (i.e., children), as described in the Exposure Assessment section of the NAF Atsugi, Japan, Human Health Risk Assessment, developed by Pioneer (Pioneer 2000). Although there may have been other receptors, behaviors and activities in the NAF Atsugi population that could have been evaluated, this comprehensive risk assessment was limited to scenarios represented by exposed populations who worked, spent time or lived at the Child Development Center, the Elementary School, the Residential Towers, the Ground Electronics Maintenance Building or the Golf Course.

The main site-specific uncertainty associated the determination of the risk contribution from the SIC, using the upwind-downwind approach, was the limited number of samples (i.e., 3-8) that met the percent downwind criteria used in this analysis. The only way to increase the sample size used in the comparison would have been to modify the percent downwind criteria (e.g., reduce the percent downwind criteria at the GEMB from 100% to 60%). However, reducing the uncertainty related to a small sample size would have significantly increased the uncertainty that the emissions were only related to the SIC vice the SIC and other sources. The hypothesis used in this approach was that the difference in the concentrations between the GEMB and the Golf Course were due solely to emissions from the SIC. The highest degree of uncertainty associated with this hypothesis was the assumption that the other point source components of the airborne concentrations were consistent between the GEMB and Golf Course. The reason for this uncertainty was that the Golf Course was located due east of the SIC. However, while this uncertainty is acknowledged, its impact on this upwind/downwind analysis was considered minimal because no significant point sources have been identified immediately south of the SIC (Radian 1998b). Therefore, based on its proximity to the SIC (i.e., approximately 150 meters) the airborne concentrations at the SIC and the Golf Course were expected to have similar component compositions.

5.8 Conclusions

The estimated potential human health risks for individuals that lived and worked at NAF Atsugi, Japan, resulting from exposure to constituents of concern (COCs) in soil, ambient air, indoor air, and indoor dust associated with ambient air emissions and/or subsequent deposition

from all sources impacting the air quality at NAF Atsugi were calculated for children and adults. Risks were calculated for exposures of 3, 6 and 30 years. The calculated carcinogenic risk for a child living at the Residential Tower for 3 or 6 years, or an individual living at the residential tower for 30 years (residential scenarios), was above the USEPA acceptable cancer risk range of 1 x 10⁻⁴ (1 in 10,000) to 1x 10⁻⁶ (1 in 1,000,000). The carcinogenic risk for an adult working at the GEMB (industrial scenario) for 30 years was also above the USEPA acceptable cancer risk range. The highest cancer risk calculated was 5.2 x 10⁻⁰⁴ (5.2 in 10,000) at the Residential Towers, assuming a 30-year residential exposure. The inhalation exposure pathway accounted for greater than 80% of the carcinogenic risk, at each location, with the exception of the Golf Course. The non-cancer Hazard Index (HI) was higher than 1 for both children and adults, for all scenarios evaluated except for the Recreational Golfer Scenario, indicating concern for potential non-cancer health effects.

As a result of inherent uncertainties in the risk assessment process, the USEPA acceptable cancer risk range and HI should not be interpreted as a bright line, as it does not represent a level at which health effects will be actually seen in a population. They are values that would typically alert the USEPA to consider additional activities including regulations, to lower the estimated risk. Since USEPA regulatory policy does not apply in a foreign country, NEHC conducted extensive risk characterization to help understand the degree of confidence to be placed on the cancer risk and hazard index estimates. The risk characterization indicated that the potential health concerns related to the SIC, primarily through the inhalation exposure pathway, included excess lifetime cancer risks and non-cancer adverse health effects. For children, cancer risks above the USEPA acceptable cancer risk range were calculated for children spending 3 years at NAF Atsugi. For adults cancer risks above the USEPA acceptable cancer risk range were calculated for adults spending 30 years at NAF Atsugi. Non-cancer adverse health effects anticipated were primarily associated with the respiratory system. The only chemical found to exceed any acute health based concentration level was acrolein. Acrolein exposure causes general respiratory congestion and eye, nose, and throat irritation. Persons with pre-existing eye, skin, respiratory, allergic, asthmatic or heart diseases might be at increased risk due to acrolein exposure. Individuals with cystic fibrosis and asthma, especially infants and children, should be excluded from acrolein exposure at levels measured at NAF Atsugi. The acute health effects resulting from exposure to acrolein were likely reversible; however,

combination with other known chemicals present, such as particulate matter, acetaldehyde, and formaldehyde, were cause for additional concern. Repeated long-term exposure to these chemicals in combination may have resulted in irreversible non-cancer effects due to mucosal membrane irritation.

The results of the evaluation of the contribution of the risk attributable to the SIC showed that the cancer and non-cancer risks were significantly higher downwind from the SIC than upwind from the SIC. Emissions from the SIC had a detrimental contribution to the air quality at NAF Atsugi, as indicated by:

- An increase in cancer and non-cancer risks downwind from the SIC,
- Statistical analysis showing a number of chemicals associated with the SIC,
- Results of air dispersion modeling, and
- Deposition trends of dioxin in soil across the base.

By comparison with incinerators in the U.S., an incinerator with uncontrolled emissions and high emission levels of dioxin, such as the SIC in the U.S., would have been forced to either shut down or implement controls to come into compliance and mitigate the risk of health hazards.

References

- ARB (Air Resources Board) 1993. Acetaldehyde as a Toxic Air Contaminant. Technical Support Document. Exposure Assessment. Stationary Source Division. Sacramento, CA.
- ATSDR (Agency for Toxic Substances and Disease Registry) 1987. Toxicological Profile for 1,12,2-Tetrachlorethane. U.S. Public Health Service, U.S. Department of Health and Human Services, Atlanta, GA.
- ATSDR (Agency for Toxic Substances and Disease Registry) 1989a. Toxicological Profile for Acrolein, U.S. Public Health Service, U.S. Department of Health and Human Services, Atlanta, GA.
- ATSDR (Agency for Toxic Substances and Disease Registry) 1989b. Toxicological Profile for Arsenic. U.S. Public Health Service, U.S. Department of Health and Human Services, Atlanta, GA.
- ATSDR (Agency for Toxic Substances and Disease Registry) 1990. Toxicological Profile for Acrylonitrile. U.S. Public Health Service, U.S. Department of Health and Human Services, Atlanta, GA.
- ATSDR (Agency for Toxic Substances and Disease Registry) 1991a. Toxicological Profile for Benzene. U.S. Public Health Service, U.S. Department of Health and Human Services, Atlanta, GA.
- ATSDR (Agency for Toxic Substances and Disease Registry) 1991b. Toxicological Profile for Methylene Chloride. U.S. Public Health Service, U.S. Department of Health and Human Services, Atlanta, GA.
- ATSDR (Agency for Toxic Substances and Disease Registry) 1992a. Toxicological Profile for Cadmium. U.S. Public Health Service, U.S. Department of Health and Human Services, Atlanta, GA.
- ATSDR (Agency for Toxic Substances and Disease Registry) 1992b. Toxicological Profile for Carbon TetrachlorideU.S. Public Health Service, U.S. Department of Health and Human Services, Atlanta, GA.

- ATSDR (Agency for Toxic Substances and Disease Registry) 1992c. Toxicological Profile for 1,2-Dibromoethane. U.S. Public Health Service, U.S. Department of Health and Human Services, Atlanta, GA.
- ATSDR (Agency for Toxic Substances and Disease Registry) 1993a. Toxicological Profile for 1,4-Dichlorobenzene. U.S. Public Health Service, U.S. Department of Health and Human Services, Atlanta, GA.
- ATSDR (Agency for Toxic Substances and Disease Registry) 1993b. Toxicological Profile for Vinyl Chloride. U.S. Public Health Service, U.S. Department of Health and Human Services, Atlanta, GA.
- Appleman, L. M., Woutersen, R. A., and Feron, V. J. 1982. Inhalation toxicity of acetaldehyde in rats. I. Acute and subacute studies. <u>Toxicology</u> 23:293-297.
- Appleman, L. M., Woutersen, R. A., Feron, V. J., Hooftman, R. N., and Notten, W. R. F. 1986. Effect of variable versus fixed exposure levels on the toxicity of acetaldehyde in rats. <u>J. Appl. Toxicol.</u> 6(5):331-336.
- Bertazzi, P. A., Consonni, D., Bachetti, S., Rubagotti, M., Baccarelli, A., Zocchetti, C., and Pesatori, A. C. 2001. Health effects of dioxin exposure: A 20-yer mortality study. <u>Am. J. Epidemiol.</u> 153(11):1031-1044.
- Birnbaum, L. S. 1995. Developmental effects of dioxins and other endocrine disrupting chemicals. Neurotoxicology 16(4):748
- Cal EPA (California Environmental Protection Agency) 1997. Toxic Air Contaminant Identification List Summaries, Toxic Contaminant Fact Sheets; September 1997. California Air Resources Board, [Online]. Available:

 (http://www.arb.ca.gov/toxics/tac/tac.htm)
- Cal EPA (California Environmental Protection Agency) 1999a. Air Toxics Hot Spots Program
 Risk Assessment Guidelines Part II Technical Support Document for Describing
 Available Cancer Potency Factors and Hot Spots Unit Risk and Cancer Potency Values.
 [Online]. Available: http://www.oehha.org/scientific/hsca2.htm.
- Cal EPA (California Environmental Protection Agency) 1999b. Air Toxics Hot Spots Program Risk Assessment Guidelines Part III Technical Support Document for the

- Determination of Noncancer Chronic Reference Exposure Levels, SRP Draft and Proposed OEHHA Chronic Inhalation REL Summary. [Online]. Available: http://www.oehha.org/hotspots/RAGSII.html.
- CHEMFATE 1994. Syracuse Research Corporation's Environmental Fate Data Bases (for acetaldehyde). Syracuse Research Corporation, Syracuse, NY.
- Clark, N. M., Brown, R. W., Parker, E., Robins, T. g., Remick, Jr., D. G., Philbert, M. A., Keeler, G. J., and Israel, B. A. 1999. <u>Environmental Health Perspectives</u> 107 (Suppl. 3):421-429.
- CRARM (Commission on Risk Assessment and Risk Management) 1997. Framework for Environmental Health Risk Management, Final Report, Volume 1, Washington, DC.
- CRARM (Commission on Risk Assessment and Risk Management) 1997. Risk Assessment and Risk Management in Regulatory Decision-Making, Final Report Volume 2, Washington, DC.
- D'Amato, G.,1999. Outdoor air pollution in urban areas and allergic respiratory diseases.

 Monaldi Arch. Chest Dis.54(6):470-474.
- D'Amato, G., Liccardi, G., D'Amato, M., and Cazzola, M., 2001. The role of outdoor air pollution and climatic changes on the rising trends in respiratory allergy. <u>Respir. Med.</u> 95(7):606-611.
- Dames and Moore (Chiyoda Dames & Moore Co., Ltd) 1999. Tade River Water Quality Survey

 Third Quarter U.S. Naval Air Facility Atsugi, Japan. Prepared by Chyioda Dames &

 Moore Co., Ltd, Tokyo, Japan for NAF Atsugi Public Works Department, Environmental

 Division, NAF Atsugi, Japan.
- EPA (U.S. Environmental Protection Agency) 1986. Health and Environmental Effects Profile for Benzyl Chloride. 600/x-86-148. Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Office of Research and Development, Cincinnati, OH.
- EPA (U.S. Environmental Protection Agency) 1987a. Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities. Office of Solid Waste and Emergency Response, EPA530-D-98-001A), Washington, D.C.

- EPA (U.S. Environmental Protection Agency) 1987b. The Total Assessment Method (TEAM) Study, Wallace, L.A., September 1987, EPA Report 600/56-87/002, Washington, D.C.
- EPA (U.S. Environmental Protection Agency) 1987c. Health Assessment Document for Acetaldehyde. EPA/600/8-86-015A. Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Office of Research and Development, Research Triangle Park, NC.
- EPA (U.S. Environmental Protection Agency) 1987d. Health Effects Assessment for Acetonitrile, Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Office of Research and Development, Cincinnati, OH.
- EPA (U.S. Environmental Protection Agency) 1987e. p-Dioxane Health Advisory. Office of Drinking Water, Washington, DC.
- EPA (U.S. Environmental Protection Agency) 1989. U.S. Environmental Protection Agency. Risk Assessment Guidance for Superfund: Human Health Evaluation Manual Part A. Interim Final. Office of Emergency and Remedial Response. Washington, D.C. 9285.701A. EPA/540/1-89/002.
- EPA (U.S. Environmental Protection Agency) 1992a. Indoor Air Quality Data Base for Organic Compounds", Samfield, M. M., February 1992, EPA-600-R-92-025, U.S. Environmental Protection Agency, Indoor Air Branch, Research Triangle Park, N.C.
- EPA (U.S. Environmental Protection Agency) 1992b. National Oil and Hazardous Substances Pollution Contingency Plan (The NCP). EPA 9200.2-14/PB92-963261. Office of Emergency and Remedial Response, Washington DC.
- EPA (U.S. Environmental Protection Agency) 1993. Integrated Risk Information System (IRIS) on Acrolein. Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Office of Research and Development, Cincinnati, OH.
- EPA (U.S. Environmental Protection Agency) 1993a. Ambient Concentration Summaries for Clean Air Act Title III Hazardous Air Pollutants. Kelly, T.J., Ramamurthi, M., Pollack, A. J., Spicer, C.W. and Cupitt, L.T. U.S. EPA Contract No. 68-D80082.

- EPA (U.S. Environmental Protection Agency) 1993b. Integrated Risk Information System (IRIS) on Acetaldehyde. Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Office of Research and Development, Cincinnati, OH.
- EPA (U.S. Environmental Protection Agency) 1993c. Integrated Risk Information System (IRIS) on Formaldehyde. Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Office of Research and Development, Cincinnati, OH.
- EPA (U.S. Environmental Protection Agency) 1994a. Implementation Guidance for Conducting Indirect Exposure Analysis at RCRA Combustion Units (Draft). U.S. Environmental Protection Agency, Office of Solid Waste and Emergency Response, Washington, DC.
- EPA (U.S. Environmental Protection Agency) 1994b. Unilateral Administrative Order for Protective Measures to the Solid Waste Authority of Central Ohio and the City of Columbus, Ohio, U.S. Environmental Protection Agency, Region 5, Chicago, IL
- EPA (U.S. Environmental Protection Agency) 1994c. Health Effects Notebook for Hazardous Air Pollutants-Draft, Section on Hexachlorobutadiene. EPA/452/D-95-00,PB95-503579.
- EPA (U.S. Environmental Protection Agency) 1994d. Health Effects Notebook for Hazardous Air Pollutants-Draft, Section on 1,3-Butadiene. EPA/452/D-95-00, PB95-503579.

 December
- EPA (U.S. Environmental Protection Agency) 1994e. Health Effects Notebook for Hazardous Air Pollutants-Draft, Section on Lead. EPA/452/D-95-00, PB95-503579.
- EPA (U.S. Environmental Protection Agency) 1994f. Guidance Manual for the Integrated Exposure Uptake Biokinetic Model for Lead in Children. NTIS/PB93-963510.

 Technical Review Workgroup for Lead (TRW).
- EPA (U.S. Environmental Protection Agency) 1994g. OPPT Chemical Fact Sheet. Chemicals in the Environment: 1,2,4-Trimethylbenzene. Office of Pollution Prevention and Toxics, Cincinnati, OH.
- EPA (U.S. Environmental Protection Agency) 1996a. Air Quality Criteria for Particulate Matter. EPA/600/P-95/001aF. National Center for Environmental Assessment, Office of Research and Development, Research Triangle Park, NC.

- EPA (U.S. Environmental Protection Agency) 1996b. Measuring Air Quality: The Pollution Standard Index. EPA/451/K-94-001, Office of Research and Development, Cincinnati, OH.
- EPA (U.S. Environmental Protection Agency) 1997. The Health Assessment Summary Tables FY- 1997 Update. EPA/540-R-97-036. PB97-921199. U.S. Environmental Protection Agency, Office of Solid Waste and Emergency Response, Washington, DC.
- EPA (U.S. Environmental Protection Agency) 1998a. Quality Assurance Handbook for Air Pollution Measurement Systems, EPA/454/R-98-004. U.S. Environmental Protection Agency, Office of Air Quality Planning and Standards, Research Triangle Park, N.C.
- EPA (U.S. Environmental Protection Agency) 1998b. Methodology for Assessing Health Risks Associated With Multiple Pathways of Exposure to Combustor Emissions. EPA 600/R-98/137. [Online]. Available: http://www.epa.gov/nceawww1/combust.htm. [June 23, 2000]. U.S. Environmental Protection Agency, Office of Research and Development, Research Triangle Park, NC.
- EPA (U.S. Environmental Protection Agency) 1998c. Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities. Vol.1. Peer Review Draft. EPA530-D-98-001A. [Online]. Available: http://www.epa.gov/epaoswer/hazwaste/combust/risk.htm. [May 16, 2000]. U.S. Environmental Protection Agency, Office of Solid Waste and Emergency Response, Washington, DC.
- EPA (U.S. Environmental Protection Agency) 1998d. Results of March 1988 Soil Sampling NAF Atsugi, Japan, August 1998, Memorandum from Mr. Matthew Lorber, Environmental Protection Agency, National Center for Environmental Assessment, Washington, D.C.
- EPA (U.S. Environmental Protection Agency) 1998e. Integrated Risk Information System (IRIS) on Acrylonitrile. Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Office of Research and Development, Cincinnati, OH.
- EPA (U.S. Environmental Protection Agency) 1999a. Laboratory Audit of Alta Analytical Laboratories, Inc and Laboratory Audit of Radian International. Letter from Mr. Curtis

- M. Morris, Environmental Protection Agency, National Exposure Research Laboratory, Research Triangle Park, N.C.
- EPA (U.S. Environmental Protection Agency) 1999b. Laboratory Audit of Czartech Analytical Letter from Mr. Curtis M. Morris, Environmental Protection Agency, National Exposure Research Laboratory, Research Triangle Park, N.C.
- EPA (U.S. Environmental Protection Agency) 1999c. The Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, Ohio. 4th Quarter 1999 Update.
- EPA (U.S. Environmental Protection Agency) 1999d. Provisional Toxicity Values obtained from the National Center for Environmental Assessment Superfund Health Risk Technical Support Center. U.S. EPA Memo From Femi Adeshina and Patricia A. Daunt to Mark Greenberg, Dec. 2, 1999.
- EPA (U.S. Environmental Protection Agency) 1999e. Integrated Risk Information System (IRIS) on Acetonitile. Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Office of Research and Development, Cincinnati, OH.
- EPA (U.S. Environmental Protection Agency) 2000a. Consolidated Peer Review Comments by Staff of the U.S. Environmental Protection Agency (EPA) on the *Comprehensive Health Risk Assessment for NAF*, *Atsugi*, *Japan*. Memorandum from Bruce D. Rodan, Medical Officer (Research)/Senior Health Scientist, U.S. EPA, National Center for Environmental Assessment, to Commanding Officer, Navy Environmental Health Center, May 31, 2000.
- EPA (U.S. Environmental Protection Agency) 2000b. EPA Region III Risk-Based Concentration

 Table October 2000 Update [Online]. Available:

 (http://www.epa.gov/reg3hwmd/risk/riskmenu.htm)
- EPA (U.S. Environmental Protection Agency) 2000c. Preliminary Report of the National Ombudsman on the Waste Technologies Industries (WTI), Liverpool Ohio Office of Solid Waste and Emergency Response, U.S. Environmental Protection Agency, Washington, D.C.

- EPA (U.S. Environmental Protection Agency) 2001a. National Ambient Air Quality Standards (NAAQS). Office of Air Quality Planning and Standards, Cincinnati, OH, [Online]. Available: (http://www.epa.gov/airs/criteria.html).
- EPA (U.S. Environmental Protection Agency) 2001b. USEPA Information Sheet 1—Dioxin: Summary of the Dioxin Reassessment Science, Office of Research and Development, Washington, DC 20460.
- Goldsmith, C. W. and Kobzik, L. 1999. Particulate air pollution and asthma: A review of Epidemiological and Biological Studies. Reviews on Environmental Health 14(3): 121-134.
- Holland, W. W., Bennett, A. E., Cameron, I. R., Florey, D. V., Leeder, S. R., Schilling, R. S. F.,
 Swan, A. V., and Waller, R. E. 1979. Health effects of particulate pollution:
 reappraising the evidence, Chapter 5. Exposure to particulate pollution: studies in children. <a href="https://doi.org/no.1007/jmailto.com/mailto.co
- HSDB (Hazardous Substances Data Bank) 1993. U.S. Department of Health and Human Services, Hazardous Substances Data Bank (HSDB, online database). National Toxicology Information Program, National Library of Medicine, Bethesda, MD.
- IT (IT Corporation). 1999. Quality Assurance Audit Report for the Ambient Air Quality and Meteorological Monitoring Program. IT Job No. 778604. Prepared by IT Corporation, Air Quality Services, Knoxville, TN, for Commander, Atlantic Division Naval Facilities Engineering Command, Norfolk, VA.
- IARC (International Agency for Research on Cancer) 1985. IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans: Allyl Compounds, Aldehydes, Epoxides and Peroxides. Volume 36. World Health Organization, Lyon, France.
- Jedrychowski, W., Flak, E., and Mroz, E. 1999. The adverse effect of low levels of ambient air pollutants on lung function growth in preadolescent children. <u>Environmental Health</u>

 <u>Perspectives</u> 107(8):669-674.
- Jones, A. P. 2000. Review article: Asthma and the home environment. <u>Journal of Asthma</u> 37(2):103-124.

- Klaassen, C. D., Editor. 2001. <u>Casarett and Doull's Toxicology—The Basic Science of Poisons</u>, Sixth Edition, McGraw-Hill, New York, NY, pages 828, 992.
- Krzyzanowski, M., Quackenboss, J. J., and Lebowitz, M. D. 1990. Chronic respiratory effects of indoor formaldehyde exposure. <u>Environ. Res.</u> 52:117-125.
- Kutzman, R. S. 1981. A subchronic inhalation study of Fischer 344 rats exposed to 0, 0.4, 1.4, or 4.0 ppm acrolein. Brookhaven National Laboratory, Upton, NY. National Toxicology Program: Interagency Agreement No. 222-Y01-ES-9-0043.
- Lewis, M., Worobey, J., Ramsay, D. S., and McCormack, M. K. 1992. Prenatal exposure to heavy metals: Effect on childhood cognitive skills and health status. <u>Pediatrics</u> 89 (6 Part 1):1010-1015.
- Linn, W. S., and Gong, Jr., H. 1999. The 21st century environment and air quality influences on asthma. <u>Current Opinion in Pulmonary Medicine</u> 5:21-26.
- Mahaffey, K. R. 1983. Differences in exposure and metabolic response of infants to lead,cadmium and zinc. In <u>Reproductive and Developmental Toxicity of Metals</u>, edited by T.W. Clarkson, G. F. Nordberg, and P. R. Sager. Plenum Press, New York, pages 777-806.
- ManTech (Mantech Environmental Technology, Inc.) 1999. Quality Assurance Review of the FTIR Spectra Acquired at the Naval Air Facility In Atsugi, Japan. Prepared by George M. Russwurm, ManTech Environmental Technology Inc., Research Triangle Park, North Carolina for Navy Environmental Health Center, Norfolk, VA.
- Manton, W. I., Angle, C. R., Stanek, K. L., Reese, Y. R., and Kuehnemann, T. J. 2000.

 Acquisition and retention of lead by young children. Environmental Research (Section A) 82(1):60-80.
- Micromedex (Micromedex, Inc.) 1999. Reprotext® System. Dabney, B. J. (Editor). Denver, CO
- Micro-Sciences (Micro-Sciences, Ltd.). 1999a. Microscopical Identification of Indoor Dust Samples from Atsugi, Japan. Final Report. Prepared by Micro-Sciences, Ltd., Arlington Heights, IL, for Radian International, Austin, TX.

- Micro-Sciences (Micro-Sciences, Ltd.). 1999b. Microscopical Identification of Aerosols on PM10 Filters from Atsugi, Japan. Final Report. Prepared by Micro-Sciences, Ltd., Arlington Heights, IL, for Radian International, Austin, TX.
- NEHC (Navy Environmental Health Center). 1995. Human Health Preliminary Risk Evaluation of Jinkanpo Incineration Complex Activities on Naval Air Facility Atsugi, Japan. U.S. Navy Environmental Health Center, Norfolk, VA.
- NEHC (U.S. Navy Environmental Health Center). 1998. Screening Level Air Human Health Risk Assessment. NAF Atsugi, Japan. Technical memorandum. U.S. Navy Environmental Health Center, Norfolk, VA.
- NEHC (U.S. Navy Environmental Health Center). 1999a. Pregnancy Loss at NAF Atsugi Japan (June 1995-May 1998). Prepared by Laurel A. May and David Sack. U.S. Navy Environmental Health Center, Norfolk, VA.
- NEHC (U.S. Navy Environmental Health Center). 1999b. Is Air Pollution from the Shinkampo Incinerator Associated with Adverse Respiratory Effects Among Children at NAF Atsugi? Prepared by David Sack and Jeffrey Hyman. U.S. Navy Environmental Health Center, Norfolk, VA.
- NEHC (U.S. Navy Environmental Health Center). 2000. NAF Atsugi, Japan. Human Health Risk Assessment: Summary of Findings, Conclusions and Recommendations. Draft Final. U.S. Navy Environmental Health Center, Norfolk, VA.
- NRC (National Research Council). 1996. Understanding Risk: Informing Decisions in a Democratic Society, eds. Paul C. Stern and Harvey V. Fineberg, National Academy Press, Washington, DC.
- NRC (National Research Council) 1998. Review of a Screening Level Risk Assessment for the Navy Air Facility at Atsugi, Japan; Committee on Toxicology, Board on Environmental Studies on Toxicology, Commission on Life Sciences, National Academy Press, Washington DC.
- NRC (National Research Council) 2000. Review of the US Navy's Human Health Risk Assessment of the Naval Air Facility at NAF Atsugi, Japan; Committee on Toxicology,

- Board on Environmental Studies on Toxicology, Commission on Life Sciences, National Academy Press, Washington DC.
- Ostro, B. D., Eskeland, G. s., Sanchez, J. M., and Feyzioglu, T. 1999. Air pollution and health effects: A study of medical visits among children in Santiago, Chile. <u>Environmental</u> Health Perspectives 107(1): 69-73.
- Otto, D., Skalik, I., Bahboh, R., Hudnell, K., and Sram, R. 1997. Neurobehavioral performance of Czech school children born in years of maximal air pollution (1982-1983).

 Neurotoxicology 18(3):903
- Peters, A., Dockery, D. W., Muller, J. E., and Mittleman, M. A. 2001. Increased particulate air pollution and the triggering of myocardial infarction. <u>Circulation</u> 103(23):2810-2815.
- Peters, A., Dockery, D. W., Heinrich, J., and Wichmann, H. E. 1997. Short-term effects of particulate air pollution on respiratory morbidity in asthmatic children. <u>European Respiratory Journal</u> 10(4):872-879.
- Pioneer (Pioneer Technologies Corporation). 1998a. Draft Soil Human Health Risk Assessment NAF Atsugi, Japan. Prepared by Pioneer Technologies Corporation, Olympia, WA, for U.S. Navy Environmental Health Center, Norfolk, VA.
- Pioneer (Pioneer Technologies Corporation). 1998b. Monthly Comparison of Ambient Air Concentrations to EPA Region 3 RBSCs for April, May, June and July 1998, NAF Atsugi, Japan. Draft Rev.: 2. Prepared by Pioneer Technologies Corporation, Olympia, WA, for U.S. Navy Environmental Health Center, Norfolk, VA.
- Pioneer (Pioneer Technologies Corporation). 1998c. Comparison of Ambient Air Concentrations to EPA Region 3 RBSCs for First Quarter (April July, 1998), NAF Atsugi, Japan. Draft Rev.: 2. Prepared by Pioneer Technologies Corporation, Olympia, WA, for U.S. Navy Environmental Health Center, Norfolk, VA.
- Pioneer (Pioneer Technologies Corporation). 1998d. Monthly Comparison of Ambient Air Concentrations to EPA Region 3 RBSCs for August and September 1998, NAF Atsugi, Japan. Draft Rev.: 0 Prepared by Pioneer Technologies Corporation, Olympia, WA, for U.S. Navy Environmental Health Center, Norfolk, VA.

- Pioneer (Pioneer Technologies Corporation). 1999a. Japan Indoor Air and Indoor Dust Human Health Risk Assessment, NAF Atsugi, Japan. Draft Rev:0 NAF Atsugi Prepared by Pioneer Technologies Corporation, Olympia, WA, for U.S. Navy Environmental Health Center, Norfolk, VA.
- Pioneer (Pioneer Technologies Corporation). 1999b. Monthly Comparison of Ambient Air Concentrations to EPA Region 3 RBSCs for October 1998, NAF Atsugi, Japan. Draft Rev.: 0 Prepared by Pioneer Technologies Corporation, Olympia, WA, for U.S. Navy Environmental Health Center, Norfolk, VA.
- Pioneer (Pioneer Technologies Corporation). 1999c. Comparison of Ambient Air Concentrations to EPA Region 3 RBSCs for Second Quarter (August October, 1998), NAF Atsugi, Japan. Draft Rev.: 2. Prepared by Pioneer Technologies Corporation, Olympia, WA, for U.S. Navy Environmental Health Center, Norfolk, VA.
- Pioneer (Pioneer Technologies Corporation). 1999d. Monthly Comparison of Ambient Air Concentrations to EPA Region 3 RBSCs for November December 1998, NAF Atsugi, Japan. Draft Rev.: 0 Prepared by Pioneer Technologies Corporation, Olympia, WA, for U.S. Navy Environmental Health Center, Norfolk, VA.
- Pioneer (Pioneer Technologies Corporation). 1999e. Monthly Comparison of Ambient Air Concentrations to EPA Region 3 RBSCs for January 1999, NAF Atsugi, Japan. Draft Rev.: 0 Prepared by Pioneer Technologies Corporation, Olympia, WA, for U.S. Navy Environmental Health Center, Norfolk, VA.
- Pioneer (Pioneer Technologies Corporation). 1999f. Comparison of Ambient Air Concentrations to EPA Region 3 RBSCs for Third Quarter (November 1998- January 1999), NAF Atsugi, Japan. Draft Rev.: 2. Prepared by Pioneer Technologies Corporation, Olympia, WA, for U.S. Navy Environmental Health Center, Norfolk, VA.
- Pioneer (Pioneer Technologies Corporation). 1999g. Monthly Comparison of Ambient Air Concentrations to EPA Region 3 RBSCs for February and March 1999, NAF Atsugi, Japan. Draft Rev.: 0 Prepared by Pioneer Technologies Corporation, Olympia, WA, for U.S. Navy Environmental Health Center, Norfolk, VA.

- Pioneer (Pioneer Technologies Corporation). 1999h. Monthly Comparison of Ambient Air Concentrations to EPA Region 3 RBSCs for April 1999, NAF Atsugi, Japan. Draft Rev.: 0 Prepared by Pioneer Technologies Corporation, Olympia, WA, for U.S. Navy Environmental Health Center, Norfolk, VA.
- Pioneer (Pioneer Technologies Corporation). 1999i. Comparison of Ambient Air Concentrations to EPA Region 3 RBSCs for Third Quarter (February April 1999), NAF Atsugi, Japan. Draft Rev.: 2. Prepared by Pioneer Technologies Corporation, Olympia, WA, for U.S. Navy Environmental Health Center, Norfolk, VA.
- Pioneer (Pioneer Technologies Corporation). 2000. NAF Atsugi, Japan. Human Health Risk Assessment, Draft Rev.: 0. Prepared by Pioneer Technologies Corporation, Olympia, WA, for U.S. Navy Environmental Health Center, Norfolk, VA.
- Presidential/Congressional Commission on Risk Assessment and Risk Management. 1997a. Framework for Environmental Health Risk Management. Final Report. Vol.1. GPO, Washington, DC.
- Presidential/Congressional Commission on Risk Assessment and Risk Management. 1997b. Risk Assessment and Risk Management in Regulatory Decision-Making. Final Report. Vol.2. GPO, Washington, DC.
- Radian (Radian International). 1997. Site Visit Plan for the Jinkanpo Incineration Complex NAF Atsugi, Japan. Prepared by Radian International, Austin, TX, for Atlantic Division Naval Facilities Engineering Command, and Navy Environmental Health Center, Norfolk, VA.
- Radian (Radian International). 1998. Site Visit Report to Support Monitoring Impacts of the Jinkanpo Incineration Complex, NAF Atsugi, Japan. RCN 801230.04. Prepared by Radian International, Austin, TX, for Atlantic Division Naval Facilities Engineering Command, and Navy Environmental Health Center, Norfolk, VA.
- Radian (Radian International). 1998a. Draft Final Sampling and QA/QC Plan to Access Health Risks Related to Air Quality at NAF Atsugi, Japan. RCN 80067903.03. Prepared by Radian International, Austin, TX, for Atlantic Division Naval Facilities Engineering Command, and Navy Environmental Health Center, Norfolk, VA.

- Radian (Radian International). 1998b. Exposure Pathways Analysis, Shinkampo Incineration Complex NAF Atsugi, Japan RCN 8012301.21. Prepared by Radian International, Austin, TX, for Atlantic Division Naval Facilities Engineering Command, and Navy Environmental Health Center, Norfolk, VA.
- Radian (Radian International). 1998c. Soil Sampling Plan to Demonstrate Health Impacts from the Jinkanpo Incineration Complex NAF Atsugi, Japan. RCN 8012301.08. Prepared by Radian International, Austin, TX, for Atlantic Division Naval Facilities Engineering Command, and Navy Environmental Health Center, Norfolk, VA.
- Radian (Radian International). 1998d. Results of March 1998 Soil Sampling, NAF Atsugi, Japan. RCN 8012301.10. Prepared by Radian International, Austin, TX, for Atlantic Division Naval Facilities Engineering Command, and Navy Environmental Health Center, Norfolk, VA.
- Radian (Radian International). 1998e. NAF Atsugi, Japan. First Quarterly Monitoring Summary 21 April 31 July 1998. RCN 801230.2302. DCN. 98.801230.05. Prepared by Radian International, Austin, TX, for Atlantic Division Naval Facilities Engineering Command, and Navy Environmental Health Center, Norfolk, VA.
- Radian (Radian International). 1998f. NAF Atsugi, Japan. Ambient Air Monitoring Summary
 April May 1998. RCN 801230.2301. DCN. 98.801230.02. Prepared by Radian
 International, Austin, TX, for Atlantic Division Naval Facilities Engineering Command, and Navy Environmental Health Center, Norfolk, VA.
- Radian (Radian International). 1998g. NAF Atsugi, Japan. Ambient Air Monitoring Summary June 1998. RCN 801230.2301. DCN. 98.801230.03. Prepared by Radian International, Austin, TX, for Atlantic Division Naval Facilities Engineering Command, and Navy Environmental Health Center, Norfolk, VA.
- Radian (Radian International). 1998h. NAF Atsugi, Japan. Ambient Air Monitoring Summary July 1998. RCN 801230.2301. DCN. 98.801230.04. Prepared by Radian International, Austin, TX, for Atlantic Division Naval Facilities Engineering Command, and Navy Environmental Health Center, Norfolk, VA.

- Radian (Radian International). 1998i. NAF Atsugi, Japan. Ambient Air Monitoring Summary August 1998. RCN 801230.2301. DCN. 98.801230.05. Prepared by Radian International, Austin, TX, for Atlantic Division Naval Facilities Engineering Command, and Navy Environmental Health Center, Norfolk, VA.
- Radian (Radian International). 1998j. NAF Atsugi, Japan. Ambient Air Monitoring Summary September 1998. RCN 801230.2301. DCN. 98.801230.06. Prepared by Radian International, Austin, TX, for Atlantic Division Naval Facilities Engineering Command, and Navy Environmental Health Center, Norfolk, VA.
- Radian (Radian International). 1999a. NAF Atsugi, Japan. Second Quarterly Monitoring

 Summary 1 August 31 October 1998. RCN 801230.2302. DCN. 98.801230.09.

 Prepared by Radian International, Austin, TX, for Atlantic Division Naval Facilities

 Engineering Command, and Navy Environmental Health Center, Norfolk, VA.
- Radian (Radian International). 1999b. NAF Atsugi, Japan. Ambient Air Monitoring Summary January 1999. RCN 801230.2301. DCN. 99.801230.08. Prepared by Radian International, Austin, TX, for Atlantic Division Naval Facilities Engineering Command, and Navy Environmental Health Center, Norfolk, VA.
- Radian (Radian International). 1999c. NAF Atsugi, Japan. Ambient Air Monitoring Summary February 1999. RCN 801230.2301. DCN. 99.801230.09. Prepared by Radian International, Austin, TX, for Atlantic Division Naval Facilities Engineering Command, and Navy Environmental Health Center, Norfolk, VA.
- Radian (Radian International). 1999d. NAF Atsugi, Japan. Ambient Air Monitoring Summary
 March April 1999. RCN 801230.2301. DCN. 99.801230.10. Prepared by Radian
 International, Austin, TX, for Atlantic Division Naval Facilities Engineering Command,
 and Navy Environmental Health Center, Norfolk, VA.
- Radian (Radian International). 1999e. NAF Atsugi, Japan. Ambient Air Monitoring Summary May - June 1999. RCN 801230.2301. DCN. 99.801230.11. Prepared by Radian International, Austin, TX, for Atlantic Division Naval Facilities Engineering Command, and Navy Environmental Health Center, Norfolk, VA.

- Radian (Radian International). 1999f. Phase II Soil Sampling Report Addendum to the March 1998 Report. NAF Atsugi, Japan. RCN 801230.2701. Prepared by Radian International, Austin, TX, for Atlantic Division Naval Facilities Engineering Command, and Navy Environmental Health Center, Norfolk, VA.
- Radian (Radian International). 1999g. NAF Atsugi, Japan. Ambient Air Monitoring Summary
 March April 1999. RCN 801230.2301. DCN. 99.801230.10. Prepared by Radian
 International, Austin, TX, for Atlantic Division Naval Facilities Engineering Command,
 and Navy Environmental Health Center, Norfolk, VA.
- Radian (Radian International) 1999h. NAF Atsugi, Japan. Ambient Air Monitoring Summary
 May June 1999. RCN 801230.2301. DCN 99.801230.11. Prepared by Radian
 International, Austin, TX, for Atlantic Division Naval Facilities Engineering Command, and Navy Environmental Health Center, Norfolk, VA.
- Radian (Radian International) 2000a. NAF Atsugi, Japan. Final Monitoring Summary, April 1998 -June 1999. Vol. I: Text. RCN 801230.2302. DCN 99.801230.11. Prepared by Radian International, Austin, TX, for Atlantic Division Naval Facilities Engineering Command, and Navy Environmental Health Center, Norfolk, VA.
- Radian (Radian International) 2000b. NAF Atsugi, Japan. Final Monitoring Summary, April 1998 -June 1999. Vol. III: Appendices E-G. RCN 801230.2302. DCN 99.801230.11. Prepared by Radian International, Austin, TX, for Atlantic Division Naval Facilities Engineering Command, and Navy Environmental Health Center, Norfolk, VA.
- Radian (Radian International). 2000c. NAF Atsugi, Japan. Final Monitoring Summary, April 1998 -June 1999. Vol. IV: Appendices H-N. RCN 801230.2302. DCN 99.801230.11. Prepared by Radian International, Austin, TX, for Atlantic Division Naval Facilities Engineering Command, and Navy Environmental Health Center, Norfolk, VA.
- Radian (Radian International). 2000d. NAF Atsugi, Japan. Final Monitoring Summary, April 1998 -June 1999. Volume II: Appendices A-D. RCN 801230.2302. DCN 99.801230.11. Prepared by Radian International, Austin, TX, for Atlantic Division Naval Facilities Engineering Command, and Navy Environmental Health Center, Norfolk, VA.

- RTI (Research Triangle Institute). 1999. Statistical Analysis of Ambient Air Data for the NAF Atsugi Health Risk Assessment. RTI No. 7732-000. Prepared by Research Triangle Institute, Research Triangle Park, NC, for Navy Environmental Health Center, Norfolk, VA.
- Scorecard (Scorecard). 2001. Chemical Profile for 2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD). Environmental Defense. [Online]. Available: http://www.scorecard.org/chemical-profiles.
- Travis, C.C., S.A. Richter, E.A.C. Crouch, R. Wilson, and E. Klema. 1987. Cancer Risk Management a Review of 132 Federal Regulatory Decisions. Environ. Sci. Technol. 21(5): 415-420.
- UAI (UAI Environmental, Inc). 1999. Quality Assurance Audit Report for the Ambient Air Quality and Meteorological Monitoring Program. Naval Air Facility Atsugi, Japan. Prepared by UAI Environmental, Inc., Reading, PA, for Commander, Atlantic Division Naval Facilities Engineering Command, Norfolk, VA.
- WHO (World Health Organization) 1989. Environmental Health Criteria for Formaldehyde. Volume 89. World Health Organization, Geneva, Switzerland.
- Weston (Roy F. Weston, Inc.) 1999. Quality Assurance Audit Report for the Air Quality/Meteorological Monitoring Program at the Naval Air Facility in Atsugi Japan. W.O. No. 10220-050-001. Prepared by Roy F. Weston Inc., West Chester, PA, for Commander, Atlantic Division Naval Facilities Engineering Command, Norfolk, VA.
- Yonemoto, J. 2000. The effects of dioxin on reproduction and development. <u>Industrial Health</u> 38:259-268







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Comprehensive Risk Communication and Health Consultation Plan for Naval Air Facility Atsugi

Forward

1. Introduction.

a. Air Quality at Naval Air Facility (NAF) Atsugi.

- (1) Air quality at Naval Air Facility (NAF) Atsugi is generally poor. NAF Atsugi is located on Japan's Kanto Plain. The Kanto Plain is one of the most densely populated and heavily industrialized regions of Japan. There are many sources of air pollution, including vehicle exhaust as well as air emissions from industry. One major source of air pollution, which directly impacts NAF Atsugi, is the Jinkanpo (Shinkampo) Incinerator Complex. Jinkanpo (Shinkampo) is located adjacent to NAF Atsugi and has been in operation since 1985. There are three incinerators at Jinkanpo (Shinkampo). Under its existing license, Jinkanpo (Shinkampo) operates 24 hours per day and burns up to 30 tons of waste per day. Approval to expand operations to 90 tons per day per incinerator is expected in the near future.
- (2) Jinkanpo (Shinkampo) operates under a general waste disposal permit which means it can burn materials such as municipal and industrial wastes, wood products, plastics, industrial materials, construction debris, alkalis, waste oils, waste acids, and numerous other wastes. Incinerator operators frequently soak solid waste materials with liquid wastes prior to burning. In addition, on windy days piles of ash at the complex contribute to particulate emissions from the incinerators.
- (3) The incinerators are equipped with control equipment consisting of a dry quench, an acid gas reaction chamber, an electrostatic precipitator, cyclone separators, and a wet quench scrubber. However, observations by Navy environmental professionals indicate that incinerator operators may frequently bypass the air pollution control equipment.

- (4) The location of Jinkanpo (Shinkampo) in relation to NAF Atsugi influences the impact of incinerator operations on the air facility. Jinkanpo (Shinkampo) is situated in a small river valley south of NAF Atsugi. NAF Atsugi is positioned on a plateau at the head of the valley, about 20 meters higher than the tops of the incinerator stacks. The river valley channels the wind in the direction of NAF Atsugi, especially during the months of April through October when prevailing winds are from the south. During this portion of the year, fumigation incidents are common in the most densely populated areas of the air facility, including family housing which has one high-rise/high density unit only 250 meters from the incinerator complex.
- b. Navy Efforts to Address Poor Air Quality and Impact of Jinkanpo at NAF Atsugi. The Navy is concerned with the poor air quality at NAF Atsugi and for the potential health effects on Navy personnel and their families. The proximity of Jinkanpo (Shinkampo) to the air facility particularly focuses concern on exposures to incinerator stack emissions and fugitive emissions from storage, handling and disposal of waste materials. Committed to protecting the health of our people, the Navy has engaged in a number of activities to address the situation.
 - (1) The U.S. first expressed concerns about Jinkanpo to the Government of Japan (GOJ) in September 1990. GOJ responded with a report that Jinkanpo (Shinkampo) was in compliance with Japanese law. Since that time the U.S. has engaged in continuous negotiations with GOJ to close Jinkanpo (Shinkampo) or mitigate emissions from Jinkanpo (Shinkampo).
- (2) The Navy has conducted three air quality studies; 1991, 1994, and 1997; to characterize air quality at NAF Atsugi. To augment the second and third air quality studies, the Navy Environmental Health Center (NEHC) performed screening health risk assessments (HRAs) with the air quality data. The findings of both screening HRAs indicated the potential for an increase in non-cancer and cancer health effects with ongoing exposure to the poor air quality at NAF Atsugi. A full HRA is now underway to characterize more completely the health risks at NAF Atsugi.
 - (3) NAF Atsugi initiated formal risk communication activities, following completion of the first screening HRA, dated October 1995. Communication activities to date have addressed: ongoing diplomatic efforts to close

Jinkanpo (Shinkampo) or modify Jinkanpo's (Shinkampo) operating procedures; findings of the screening HRAs; interim risk reduction measures to moderate the potential impact of the air quality on the health of area residents; and plans for a full HRA to characterize more completely the health risk.

- 2. Objectives of the Comprehensive Risk Communication and Health Consultation Plan. The Navy Bureau of Medicine and Surgery (BUMED) has prepared this Comprehensive Risk Communication and Health Consultation Plan for NAF Atsugi to guide the risk communication and health consultation process and establish a coordinated and consistent message.
- Risk Communication. Health risk communication is designed to provide factual information concerning potential health risks without causing unwarranted fear or concern. The plan lays out the mechanisms and accountabilities for informing all potentially affected parties about (1) the nature and magnitude of health risks associated with the air quality, (2) specific actions being taken and that can be taken by residents to reduce those health risks, and (3) the status of ongoing environmental studies, such as the full HRA designed to characterize more completely the health risk. Multiple communication channels must be used to ensure that appropriate information reaches the right audiences. Specific target audiences for these risk communication efforts include individuals and families prior to transfer to and from NAF Atsugi as well as individuals and families at NAF Atsugi during the summer of 1998. August 1998, Risk Communication Briefs will be a required element of the NAF Atsugi check-in process for newly arriving community members and will be presented during the Area Orientation program. Risk Communication will also be required for departing NAF Atsugi community members and will be incorporated into individual and family Health Consultations provided by Branch Medical Clinic (BMC) Atsugi providers. Completion of the required Health Consultation and Risk Communication prior to permanent change of station departure from NAF Atsugi will be included in the NAF Atsugi check-out process. From another perspective, key target audiences include active duty personnel, Navy and Department of Defense civilian personnel, family members of active duty and civilian personnel, and Japanese Nationals who work at NAF Atsugi. Different communication channels will be required to reach each target audience. Specific communication channels and procedures to be employed for each of these target audiences are covered in Sections 2.0 through 4.0 respectively. These sections also address the

specific roles and responsibilities of various commands in the delivery of risk communication.

- Health Consultation. In addition to establishing a program for risk communication, this plan provides for oneon-one health consultation for active duty personnel and their families at the time of the overseas medical screen, prior to transfer to NAF Atsugi, for individuals and family members over the age of six who will exceed a total stay time of six years at NAF Atsugi or a total stay time of three years at NAF Atsugi for individuals with family members under the age of six, and for all community members at the time of permanent change of station departure from Atsugi. The plan also provides for health consultations for current NAF Atsugi residents and employees who are at greatest potential health risk. Individuals potentially most sensitive to exposure to poor air quality include those with chronic respiratory diseases, children under the age of six years, and possibly pregnant or nursing women. addition, the plan provides opportunities for other concerned individuals to receive health consultations. Principal health concerns potentially related to the air quality at NAF Atsugi, as expressed by area residents and employees, have included acute health effects such as mucous membrane and respiratory track irritation and headaches, as well as general concerns for the health of unborn and young children. Moderate to severe chronic respiratory conditions are generally disqualifying for assignment to NAF Atsugi. Development of significant acute health effects, such as exacerbation of asthma, sinusitis, or migraine headaches, while at Atsugi may require relocation of affected individuals. Pregnant women and families with children under six years of age will receive focused education on measures to take to minimize exposures to incinerator emissions and air pollution fallout. BUMED has developed guidance to conduct the health consultations, as further detailed in Sections 2.0 through 3.0.
- c. Community Outreach Program. This comprehensive health risk communication and health consultation plan is designed to encourage two-way dialogue and ensure that timely, accurate and consistent information is provided to future and current members of the NAF Atsugi community. The plan specifically addresses the requirements of a community outreach program, as detailed in Section 4.0, for current residents. NAF Atsugi is responsible for developing an execution plan to carry out the requirements of the community outreach program, which includes formal means to evaluate the effectiveness of community outreach and risk communication activities.

- d. Training in Health Risk Communication. It is essential that individuals throughout the Navy organization who will be involved with the oversight and execution of this comprehensive plan be familiar with the principles of health risk communication. Those charged with the responsibility of delivering health risk communication must be trained in risk communication in order to be most effective. In January 1997, May 1998, and June 1998, several individuals involved in the NAF Atsugi risk communication efforts attended NEHC-sponsored Health and Environmental Risk Communication Workshops in Tokyo, Japan, Washington D.C., Millington, Tennessee, and Atsugi, Japan. Section 5.0 details the NEHC plan for providing ongoing risk communication training to key personnel.
- e. Resource Requirements. The level of effort required to fully execute this plan will require resources above current operating funds. Section 6.0 details the additional requirements BUMED has identified to provide risk communication support and training and to conduct one-on-one health consultations. NAF Atsugi is responsible for identifying additional resource requirements to execute the community outreach program and related risk communication.

3. Previous Air Quality Studies and Screening Health Risk Assessments (HRAs).

- a. The air quality studies conducted by the Navy in 1994 and 1997 indicate that the air quality at NAF Atsugi meets Japanese air standards, but does not meet US Environmental Protection (EPA) Standards for breathable dusts and a number of chemicals, including a number of organic compounds and heavy metals.
- b. To evaluate the potential for human health effects from exposure to the poor air quality at NAF Atsugi, NEHC used the data of the 1994 and 1997 air quality studies to conduct two separate screening HRAs. NEHC released the first screening HRA in 1995. NEHC completed the draft HRA based on the 1997 air quality study in January 1998, which has since undergone peer review by the National Academy of Science's Committee on Toxicology and the ATSDR. The findings of both screening HRAs are comparable and indicate that there is an increase in lifetime risk for non-cancer and cancer health effects with ongoing exposure to the poor air quality at NAF Atsugi.
- c. Risk for Non-cancer Health Effects. Many of the air pollutants at NAF Atsugi are mucous membrane and lung

irritants. Consequently, the predominant non-cancer health effects impact the respiratory system and the eyes. severity of symptoms depends on a number of factors such as type of pollutants present, the concentrations of pollutants, duration of exposure, related weather conditions, and susceptibility of the exposed individual. As these parameters can vary widely during the course of a day or from day to day, the effect on an individual's health can vary considerably as well. For most individuals symptoms tend to be short-lived and directly related to exposure. Those with chronic respiratory conditions, such as asthma, chronic bronchitis, or sinusitis, and young children may be particularly sensitive to the irritating effects of air pollution and prone to more chronic symptoms as duration of exposure increases. Determination of suitability for assignment at Atsugi must be handled on a case-by-case basis. As stated earlier, moderate to severe chronic respiratory conditions will generally be considered disqualifying for assignment to NAF Atsugi. Development of significant acute health effects, such as exacerbation of asthma, sinusitis, or migraine headaches, while at Atsugi may require relocation of affected individuals.

- d. Risk for Cancer Health Effects. The HRA process involves the use of many assumptions about exposures and the potential of health effects at low levels of exposure. The process is designed to apply assumptions conservatively to ensure protection of public health. As such the actual lifetime cancer risk is likely to be less than that calculated from an HRA.
- (1) Adults and Children Six and Older. Based on the screening HRAs, for adults and children ages six and older, the air quality at NAF Atsugi could result in as much as one additional case of cancer over the course of a lifetime in a population of 10,000 individuals who resided at NAF Atsugi for six years. This represents an increase over and above the background lifetime expected incidence of cancer as estimated by the American Cancer Society, which is 5,000 cases in 10,000 American men and 3,333 cases in 10,000 American women. In other words, after six years of exposure at NAF Atsugi, it is expected that the number of men at risk for cancer is 5,001 in a population of 10,000 and the number of women at risk for cancer is 3,334 in a population of 10,000.
- (2) Children Under Six Years of Age. For children under the age of six, the same level of risk, one additional case of cancer, over the course of a lifetime, in a population of 10,000 individuals, is reached after three

years of exposure to the environmental conditions at NAF Atsugi.

- (3) Comparison of Excess Cancer Risks. To help individuals put the excess cancer risk at Atsugi into perspective, the increase in cancer risk from exposure to cosmic radiation acquired by living in Denver, Colorado, a mile above sea level, as opposed to living at sea level, is provided for comparison. For Denver residents, the excess lifetime cancer risk is one additional case of cancer in 10,000 individuals after two years of residence in Denver, when compared to a population living at sea level.
- (4) Based on the inherent limitations and assumptions of the HRA process and the limited air quality data from which the screening HRAs were conducted, normal tour lengths equating to six years for adults and children over six and to three years for children under six present acceptable levels of lifetime cancer risk, as per EPA standards, pending completion of the full HRA and complete definition of the environmental health risk.
- 4. Previous Community Outreach Activities. NAF Atsugi has had the lead in communicating the health risk to residents. Public health professionals with experience in health risk communication at the Navy Environmental Health Center (NEHC) have assisted the staff of NAF Atsugi and Branch Medical Clinic (BMC) Atsugi regarding effective risk communication tools to convey health risk in proper perspective and community health education to provide information on how individuals can reduce their health risk.
 - a. NAF Atsugi has hosted the following formal events:
- Oct 95: Public Availability Session: The public exhibit reviewed the background and results of the 1994 air quality study and 1995 screening health risk assessment in understandable language, addressed health and medical issues, discussed base initiatives, and discussed related political issues.
- Voluntary health screening was offered to concerned personnel.
- A plan to educate base personnel on sensible precautions to protect their health was addressed.
- Feb 97: Shirley Lanham Elementary School teachers brief.
- APR 97: Public exhibit.
- Apr 97: Shirley Lanham Elementary School 4th Grade Class Brief.

- Nov 97: Public Availability Session:
- NAF Atsugi hosted a Public Availability Session to update residents on ongoing efforts by the Navy to resolve the incinerator issue. Public exhibits and information booths with subject matter experts were set up to provide information on the nature of air pollution at Atsugi, related medical risk, and precautionary measures and engineering controls to reduce exposure to poor air quality and incinerator emissions.
- The CO NAF Atsugi and representatives from Commander Naval Forces Japan (CNFJ) and NEHC met with a group of concerned citizens to discuss specific issues.
- Jan 98: NAPRA (tenant command) safety standdown brief.
- **Feb 98**: Branch clinic sponsored training for Child Development Center, Youth Center and Family Home Care Providers.
- Mar 98: Branch clinic sponsored training for family members in measures to take to minimize exposure to air pollution and its fallout.
- b. NAF Atsugi has not used any formal means to evaluate the effectiveness of these past community outreach activities. However, sufficient information has been gathered via these previous activities to assemble a listing of frequently asked questions. These questions appear in the Frequently Asked Questions (FAQ) Fact Sheet (Appendix T).
- c. NAF Atsugi and the BMC Atsugi continue to be engaged in ongoing community outreach efforts designed to communicate health risk, evaluate potential health effects of poor air quality, and educate residents on effective measures to protect their health.
- 5. Risk Reduction Activities at NAF Atsugi. Risk reduction efforts at NAF Atsugi pre-date the 1995 screening HRA. Early efforts consisted of posting placards to warn joggers about air emissions from the Jinkanpo Incinerator and to inform the community on the status of negotiations with the Government of Japan (GOJ) and the Kanagawa Prefectorate Government (KPG) about the incinerator's emissions. With the release of the 1995 screening HRA, NAF Atsugi intensified risk reduction efforts. Actions have been taken on three fronts: (1) diplomatic initiatives, (2) engineering controls, and (3) community health education.
- a. Diplomatic efforts have been ongoing with GOJ and KPG since shortly after the incinerator opened in 1985. Only recently has noticeable progress been made in that KPG

has temporarily withheld approval for expanded operations at the incinerator and GOJ has begun to discuss the merits of raising the stack height to better disperse incinerator emissions. Negotiations continue regarding options to close the incinerator or to otherwise significantly modify incinerator emissions and their impact on the environment.

- b. Engineering controls include purchasing and distributing portable air cleaners to residents of Navy family housing and the Bachelor Officers' Quarters and Bachelor Enlisted Quarters. In addition, the design for retrofitting the existing heating, ventilation, and air conditioning system of the high-rise family housing units with a state of the art air cleaning system is nearly complete.
- c. Community health education efforts include providing education about minimizing exposure to incinerator emissions, providing education about minimizing exposure to soot from incinerator emissions and other sources of poor air quality, and developing a base wide pollution advisory and alert system which is broadcast over the local public access television station.
- d. BUMED and NEHC have developed four epidemiology studies in response to concerns about the environmental exposures at Atsugi and the potential for adverse health effects. The findings of these studies will assist the Navy in better characterizing the health risks at Atsugi and identifying further actions to reduce health risk.
- 6. Summary of Key Elements and Perspective for Delivering Risk Communication and Health Consultation on the Environmental Health Risk at NAF Atsugi. The key elements and perspective for provision of risk communication and health consultation on the potential environmental health risk at NAF Atsugi, as addressed in this forward, include the following:
- a. The Navy is committed to protecting the health and well being of our people.
- b. The Navy is aggressively engaged in diplomatic negotiations with GOJ to close Jinkanpo (Shinkampo) or significantly reduce incinerator emissions and will keep the Atsugi community informed regarding the status of negotiations.
- c. The Navy will communicate a current factual description of the environment at NAF Atsugi, the poor air

quality due to pollution, and the impact of Jinkanpo (Shinkampo) on the facility. Related to this, the Navy is conducting a comprehensive Health Risk Assessment to characterize more completely the environmental health risk at Atsugi and several epidemiology studies to evaluate the impact of the environment on the health of the people at Atsugi. The Navy will provide status reports on these studies and share findings on these studies as they are completed.

- d. The Navy will communicate the known and potential non-cancer and cancer health effects related to exposure to the air pollution and identify those at greater risk for these health effects.
- e. The Navy will provide one-on-one health consultations for those identified to be at greater risk and will offer health consultations to all who request them.
- f. The Navy will provide education to the Atsugi community regarding measures individuals and families can take to reduce exposures to environmental pollution and incinerator emissions.
- g. The Navy is pursuing the use of various engineering controls at NAF Atsugi to reduce exposure to air pollution and incinerator emissions. The Navy will keep the community apprised on the status and effectiveness of these efforts.

1.0. Definitions.

- 1.1. Risk Communication. The term "risk communication," as used in this plan, applies to the process of informing and educating people regarding the environmental health risk at Atsugi and continuing actions being taken to mitigate the risk.
- 1.2. Health Consultation. The term "health consultation," as used in this plan, applies to the conduct of a clinical health assessment of an individual with the specific intent to review the environmental health risk at Atsugi and determine the potential impact of the health risk on health of the individual.

1.3. Abbreviations.

- 1.3.1. BMC Atsugi is the Branch Medical Clinic Atsugi, Japan.
- 1.3.2. **BUMED** is the Bureau of Medicine and Surgery for the Department of the Navy.
- 1.3.3. BUPERS is the Bureau of Naval Personnel.
- 1.3.4. CPF is the command of Commander in Chief Pacific Fleet.
- 1.3.5. CNFJ is the command of Commander Naval Forces Japan.
- 1.3.6. NAF Atsugi is Naval Air Facility Atsugi, Japan.
- 1.3.7. NEHC is the Navy Environmental Health Center.
- 1.3.8. Jinkanpo is the name of the privately owned Japanese

incinerator located adjacent to the fence line at NAF Atsugi. It has also been called **Jinkampo** and **Shinkampo** in translated documents.

Risk Communication and Health Consultation for Individuals Prior to Transfer to NAF Atsugi. The Navy will ensure health risk communication regarding the environment at NAF Atsugi is provided to all active duty personnel, family members of active duty, and civilian personnel prior to transfer to Atsugi. The Navy will coordinate with the Department of Defense (e.g. DOD Dependent Schools (DoDDS)) and other Federal agencies to provide them with health risk communication materials they may use to inform their employees of the environmental health risk prior to relocation to the area. Provision of health risk information will enable personnel and family members to make informed decisions regarding their move to Atsugi. The Navy will also provide one-on-one health consultations to active duty personnel and their families to discuss the implication of the health risk on an individual basis and to ensure medical suitability of transfer to the Atsugi environment.

2.1. Risk Communication.

- 2.1.1. Risk Communication for Active Duty Personnel. BUPERS and BUMED are jointly responsible for providing environmental health risk communication to active duty personnel who are receiving orders to commands stationed at NAF Atsugi and the aviation commands assigned to the aircraft carrier homeported in Japan.
- 2.1.1.1. <u>BUPERS Role</u>. At the time orders to Atsugi are negotiated, the detailers at BUPERS will provide information on the environmental pollution at Atsugi and the potential for impact on health to active duty personnel. Detailers will refer personnel who desire additional information to knowledgeable points of contact at NAF Atsugi and NEHC. BUPERS commenced risk communication on 29 May 98.
- 2.1.1.1.1. BUPERS will identify all requisitions for Atsugi-based commands with an "auto remark" which will prompt detailers to provide the risk communication. The auto remark will read:

"Use Detailer Script to provide environmental information to the member."

2.1.1.1.2. Detailers will use an abbreviated Detailers' script to present the risk communication (see Appendix A). As detailers are not expert health risk communicators, detailers will provide the information on the abbreviated detailers' script and direct personnel who desire more information to points of contact at NAF Atsugi and NEHC. As part of the risk communication, detailers will also provide

- information on access to NAF Atsugi and NEHC home pages for those interested in learning more about the issue.
- 2.1.1.1.3. BUPERS will maintain the text of the Detailers' Script on the BUPERS home page and in the Job Advertisement and Selection System (JASS) Open Mind as a general note for all to see.
- 2.1.1.1.4. Detailers will insert a standard statement in each service member's orders to an Atsugi-based command to document provision of this risk communication (See Appendix B).
- 2.1.1.1.5. Detailers were offered training in risk communication in May 1998 by NEHC. The training was done in Arlington, Virginia on 12-13 May 98 and in Millington, Tennessee on 25-26 May 98. This training provided detailers with a general understanding of risk communication, background information on the situation at NAF Atsugi, and an understanding of their role in the risk communication process as the first point of contact for active duty members considering orders to Atsugi. (See section 5.0, Training in Health Risk Communication, for further details.)
- 2.1.1.2. <u>BUMED Role</u>. BUMED through their medical staff at Navy medical treatment facilities (MTFs) will provide more in-depth environmental health risk communication to active duty personnel at the time of the overseas medical screen. BUMED first directed MTFs to incorporate this risk communication at the time of the overseas medical screen by message dated 26 Feb 98 (see Appendix C). BUMED prepared additional guidance to execute the plan as detailed below and released updated guidance in a message dated 7 July 1998 (see Appendix D).
- 2.1.1.2.1. Medical staff are directed to review a background summary document, developed by BUMED, entitled Response to Environmental Conditions at Naval Air Facility Atsugi: A Review, prior to conducting the medical overseas screen and risk communication (see Appendix E). To standardize communication of the environmental health risk at Atsugi to the active duty member, BUMED has also developed a Health and Environmental Fact Sheet (see Appendix F) that the medical provider will review with each individual or family undergoing medical overseas screening for suitability for assignment to NAF Atsugi.
- 2.1.1.2.2. Medical staff will provide a copy of the current Health and Environmental Fact Sheet on general health risk information related to the environment at Atsugi prepared by

BUMED to each service member and adult family member at the time of risk communication.

- 2.1.1.2.3. Medical staff will document provision of risk communication to the active duty member on a special overprinted Standard Form (SF) 600 (see Appendix G) developed by BUMED to summarize the environmental health risk at Atsugi.
- 2.1.1.2.4. The active duty member will countersign his/her SF 600 to acknowledge receipt of the risk communication.
- 2.1.1.2.5. In addition to providing medical staff with detailed guidance on the conduct of the risk communication, BUMED will also coordinate with NEHC to offer these staff more formal training in risk communication through the use of a 36 minute video specifically prepared for this purpose and scheduled for distribution in September 1998. (See section 5.0. Training in Health Risk Communication for further details.)
- 2.1.2. Risk Communication for Family Members of Active Duty Personnel. As for active duty members, BUMED through their medical staff at Navy medical treatment facilities (MTFs) will provide environmental health risk communication to all adult family members (18 years and older) of active duty at the time of the overseas medical screen. Family members will receive the same services as detailed for active duty members in section 2.1.1.2. Whenever possible the overseas medical screen should be completed as a family unit with sponsor, spouse, and children present.
- 2.1.2.1. Medical staff will document provision of risk communication to family members on the special overprinted SF 600 (see Appendix G) developed by BUMED to summarize the environmental health risk at Atsugi. One SF 600 will be completed for each family member.
- 2.1.2.2. Each adult family member over the age of 17 will countersign his/her SF 600 to acknowledge receipt of the risk communication. The sponsor or spouse will sign the SF600 for children under 18 years.
- 2.1.3. Risk Communication for Civilian Employees. BUMED is currently working with ODASN (CP/EEO) and CPF to establish a plan for providing risk communication to civilian employees working for the U. S. Navy and employees of DoD Dependents Schools (DODDS).

- 2.2. Health Consultation for Active Duty Personnel and Family Members, a BUMED Role. In addition to providing environmental health risk communication, BUMED through their medical staff at Navy medical treatment facilities (MTFs) will also provide one-on-one health consultation to active duty personnel and each of their family members as part of the overseas medical screen for transfer to Atsugi. In follow up to the message dated 26 Feb 98 which tasked MTFs to conduct the risk communication, BUMED released additional guidance regarding the one-on-one health consultation to be incorporated into the medical overseas screen on 7 July 1998 (see Appendix D).
- 2.2.1. The Overseas Medical Screen. NAVMEDCOMINST 1300.1C, Suitability Processing for Overseas Assignment of Navy and Marine Corps Members and their Accompanying Dependents, and BUMEDNOTE 1300, Suitability Screening for Overseas and Remote Locations, provide direction for performing an overseas medical screen. The standard overseas medical screen includes a medical record review, a current medical history (SF 93) on each beneficiary, and a physical examination, when medically necessary.
- 2.2.1.1. A special overprinted SF 600 (see Appendix G) is used to summarize findings of the medical record review and the current medical history and to document any potentially disqualifying diseases or conditions per the directions of NAVMEDCOMINST 1300.1C. Potentially disqualifying conditions for assignment to NAF Atsugi are specifically addressed in section 2.2.2.1.
- 2.2.1.2. NAVMED 1300/1, the Medical and Dental Overseas Screening Review for Active Duty or Dependents, is used to summarize chronic medical conditions, requiring routine or continuing access to care or access to specialized medical care, and to recommend suitability for assignment to the overseas location.
- 2.2.1.3. NAVPERS 1300/16, Report of Suitability for Overseas Assignment, is used to compile findings of the medical overseas screenings for the service member and his/her family members into one document and to document whether or not the service member and each family member is medically recommended for the assignment to the overseas location.
- 2.2.2. Suitability for Assignment based on Environmental Health Risk at NAF Atsugi. As part of the overseas medical screen, and in addition to communicating the environmental health risk at NAF Atsugi, medical staff will discuss

potential impact of the risk on any medical conditions identified at the time of the overseas medical screen and determine the suitability of assignment to Atsugi. Potential non-cancer and cancer health effects of exposure to the poor air quality at Atsugi will be discussed and are highlighted in the Forward to this document.

- 2.2.2.1. At present, chronic respiratory conditions, such as asthma or chronic obstructive pulmonary disease, are the only potentially disqualifying conditions for assignment to Atsugi. Level Three and Level Four asthma, as defined in New Asthma Therapy Recommendations: May 1997 Guidelines for the Diagnosis and Management of Asthma Expert Panel Report 2 of the National Institutes of Health National Heart Lung and Blood Institute, are specifically disqualifying for assignment to NAF Atsugi. For other chronic conditions, medical staff must use their professional judgement to determine suitability of assignment to Atsugi, however any moderate to severe chronic respiratory conditions may be considered disqualifying on a case-by-case basis. Medical staff conducting the overseas medical screen and suitability assessment are encouraged to contact the Branch Medical Clinic at NAF Atsugi to discuss suitability determinations.
- 2.2.3.2. Medical staff will annotate on the risk communication SF 600(see Appendix G) whether or not a potentially disqualifying condition exists and the medical determination of suitability or qualification for assignment to Atsugi.
- 2.2.3.3. Medical staff will also annotate the details of the disqualifying condition on the overseas screening SF 600, NAVMED1300/1 and NAVPERS 1300/16.

- 3.0. Risk Communication and Health Consultation for Individuals Employed by, Assigned to, or Residing at NAF Atsugi and Tenant Commands.
- 3.1. New Arrivals. Newly arrived Service members and their families as well as civilian employees and their family members are required to participate in an Area Orientation Brief (AOB) and a separately scheduled Risk Communication Session for New Arrivals provided by NAF Atsugi. Although all newly arrived personnel should have received risk communication and health consultation during their medical overseas screen prior to departure from CONUS, the Health Risk Communication Office at NAF Atsugi will schedule all new arrivals over the age of 17 for an additional risk communication session. This session will provide an opportunity for NAF Atsugi Command representatives and BMC Atsugi staff to inform newly arrived individuals of the updated status of environmental concerns and ongoing actions that may reduce exposure to environmental pollution. If any newly arrived personnel or family member did not receive the Health Consultation during the medical overseas screening evaluation in CONUS, that individual and his family will be referred to BMC Atsugi for a Health Consultation.
- 3.1.1. Risk Communication for Active Duty and Family Members. NAF Atsugi is responsible for providing Environment and Health Risk Communication Briefings to all new arrivals, including military members, as well as all family members over the age of seventeen years.

3.1.1.1. Presentation of Risk Communication.

3.1.1.1. The Risk Communication Session will be given by a team consisting of an NAF Atsugi Command representative and a BMC Atsugi representative. The initial presentation will last approximately thirty minutes, followed by a poster availability session where community members will have an opportunity to address specific questions or concerns with the Risk Communication Team members. The briefing session will review the air quality issues at NAF Atsugi, actions being taken to address these issues, potential health effects related to poor air quality, and recommended personal protective measures that individuals may adopt to minimize environmental exposure for themselves or family members.

- 3.1.1.1.2. The Risk Communication Session should follow the same format as BUMED/NEHC/BMC Atsugi/NAF Atsugi developed for use with the current residents of NAF Atsugi. This is described in section 3.2.
- 3.1.1.1.3. A poster availability session should immediately follow the Risk Communication Session in order to provide an opportunity for individuals to meet with local informed experts.
- 3.1.1.4. The NAF Atsugi Health and Environmental Fact Sheet will be distributed at the Risk Communication Session.
- 3.1.1.1.5. Consultants from the Environmental Programs Directorate of NEHC will assist NAF Atsugi with developing updated briefing packets when new information from ongoing environmental studies at NAF Atsugi becomes available.

3.1.1.2. Documentation of Risk Communication.

- 3.1.1.2.1. NAF Atsugi must retain documentation of provision of Risk Communication to all new arrivals.
- 3.1.1.2.2. A page 13 entry, developed by NAF Atsugi will document provision of Risk Communication (see Appendix H).
- 3.1.1.2.3. For those individuals who have had previous assignments to NAF Atsugi, the page 13 entry must also document when the individual, and all family members over the age of 6 years, will reach a total stay time of 72 months at NAF Atsugi.
- 3.1.1.2.4. For those individuals with children under the age of 6 years, the page 13 entry must document when the family will complete a total stay time of 36 months at NAF Atsugi.
- 3.1.1.2.5. Each individual over the age of 17 years will be required to provide signature to the page 13 to document receipt of the Risk Communication Brief.

3.1.1.3. Scheduling of Health Consultations.

3.1.1.3.1. At the time of the Risk Communication session, the Risk Communication Team will inform all individuals that the Navy is committed to providing each individual and family member an opportunity to meet with a health care provider to discuss the possible health effects of the environmental conditions at NAF Atsugi. Every individual

- will be advised of the opportunity to schedule an elective individual Health Consultation at BMC Atsugi. The Health Consultation schedules forty minutes for a family of four and twenty minutes for an individual appointment.
- 3.1.1.3.2. During the Risk Communication session, new arrivals will be advised that if a Health Consultation was not performed during the medical overseas screening process prior to arrival at NAF Atsugi, the individual and all family members will be referred to BMC Atsugi for the necessary Health Consultation.
- 3.1.1.3.3. NAF Atsugi will notify the BMC Atsugi of any individuals or families requesting or requiring a Health Consultation.
- 3.1.2. Risk Communication for Civilian Employees and Family Members of Civilian Employees. Until further guidance is issued, NAF Atsugi will ensure that all newly arriving Civilian Employees and Family Members of Civilian Employees are offered and included in the same process of Risk Communication provided to Military members and their family members.
- 3.1.2.1. Until further guidance is issued, documentation of Risk Communication for Civilian Employees and Family Members of Civilian Employees at NAF Atsugi will utilize the same process and forms as previously described in section 3.1.1.2.
- 3.1.2.2. NAF Atsugi will retain documentation of Risk Communication provided for Civilian Employees and Family Members of Civilian Employees.
- 3.1.3. Health Consultations for Active Duty and Family Members. The BMC Atsugi is responsible for conducting Health Consultations for every Service member and his family according to guidelines based upon length of stay at NAF Atsugi and the age of family members, but in general, all Service and family members will complete a Health Consultation at time of permanent change of station (PCS) departure from NAF Atsugi. The purpose of the Health Consultation is to review the environmental health risk status at NAF Atsugi and conduct a clinical medical assessment to determine the potential impact of the environment upon individual health.

3.1.3.1. Required Health Consultations

3.1.3.1.1. Health Consultations will be provided for all

individuals and family members who arrive at NAF Atsugi without having received a Health Consultation prior to departure from their previous Command and at the time of their medical overseas screening evaluation for suitability for assignment to NAF Atsugi. BMC Atsugi is responsible for reviewing the medical records as part of Command check in to determine whether the required Health Consultation was performed.

- 3.1.3.1.1.1. A special SF 600 overprint, signed by the health care provider and the Service member or family member over the age of 17, will document provision of Risk Communication and Health Consultation for NAF Atsugi as required.
- 3.1.3.1.1.2. If the special SF 600 overprint is not in the medical record at the time of arrival in NAF Atsugi, BMC Atsugi will notify the originating Command and schedule the Service member and family members for the required Health Consultation.
- 3.1.3.1.2. Health Consultations will be conducted for all individuals and family members prior to PCS departure from NAF Atsugi.
- 3.1.3.1.3. If a family has children under the age of 6 and is requesting retour at NAF Atsugi or extension of their current tour such that the total tour will exceed 36 months, the Service member and his family must complete a Health Consultation prior to exercising extension or retour at NAF Atsugi.
- 3.1.3.1.4. Any Service member or individual whose family members were over the age of 6 years during the time of assignment to NAF Atsugi must complete a Health Consultation if requesting a retour or extension of current tour at NAF Atsugi such that total stay time at NAF Atsugi will exceed 72 months.

3.1.3.2. Elective Health Consultations.

- 3.1.3.2.1. Any individual assigned to NAF Atsugi who has health concerns related to environmental conditions at NAF Atsugi may schedule a Health Consultation at BMC Atsugi at any time.
- 3.1.3.3. <u>Focused Health Consultations</u>. BMC Atsugi will take a proactive position to identify individuals who may be more likely to experience health concerns or health effects

related to the environmental conditions at NAF Atsugi. These individuals will be offered Health Consultations.

- 3.1.3.3.1. Families with children under six years.
- 3.1.3.3.2. Pregnant or nursing women.
- 3.1.3.3.3. Individuals with chronic respiratory conditions.
- 3.1.3.4. Elements of the One-on-One Health Consultation.
- 3.1.3.4.1. Medical health care providers will read the background summary document entitled, Response to Environmental Conditions at Naval Air Facility Atsugi: A Review (See Appendix E). This document will prepare providers to review the environmental health risk at NAF Atsugi with the active duty member and his/her family.
- 3.1.3.4.2. Medical staff will provide a copy of the current NAF Atsugi Health and Environmental Fact Sheet (see Appendix F) to the active duty member and all family members over the age of 17 years.
- 3.1.3.4.3. Medical staff will document review of risk communication with the active duty member and family on a special overprinted SF 600 that summarizes the environmental health risk at NAF Atsugi (see Appendix G).
- 3.1.3.4.4. If the Health Consultation is performed at time of departure or tour extension at NAF Atsugi, the SF 600 will also document history on where service members and family members lived, worked, or attended school or day care while at NAF Atsugi.
- 3.1.3.4.5. One SF 600 overprint will be completed for each active duty member and each member of his/her family.
- 3.1.3.4.6. Medical staff will review current and past medical history, perform a physical examination if indicated, to document whether or not a condition exists which may be caused or exacerbated by the environmental conditions at NAF Atsugi. Any medical conditions identified that are potentially disqualifying for continued overseas assignment to NAF Atsugi will be recorded.
- 3.1.3.4.6.1. At present, chronic respiratory conditions, such as step 3 or step 4 asthma as described in section 2.2.2.1., are disqualifying for continued assignment to NAF

- Atsugi. Other moderate to severe respiratory conditions should be evaluated on a case by case basis.
- 3.1.3.4.6.2. If disqualifying conditions are identified, the individual or family should be managed by referral to NAF Atsugi with recommendation that the Commanding Officer activate the Early Return of Dependents process.
- 3.1.3.4.6.2. Findings and recommendations will be discussed with the individual and family member with signature of provider and all persons over the age of 17 years required to document discussion of findings and recommendations on the SF 600 overprint.
- 3.1.3.4.7. All individuals age seventeen or older, including family members, will complete a Health Risk Appraisal "Fit to Win" evaluation.
- 3.1.3.4.7.1. The Health Risk Appraisal addresses the status of risk factors such as tobacco use, quality of diet, level of exercise, alcohol use, blood pressure and cholesterol levels.
- 3.1.3.4.7.2. Identification of risk behaviors will support medical counseling on healthy lifestyles and recommendations for risk factor reduction.
- 3.1.3.4.7.3. Identification of risk behaviors will provide valuable information on baseline health status and risk factors that an individual possesses at the time of the Health Consultation.
- 3.1.3.4.8. BMC Atsugi will maintain a proactive stance with all patients to ensure patients are aware of the health risk imposed by the environment at NAF Atsugi and understand the personal protective precautions that will minimize exposure.
- 3.1.4. <u>Health Consultations for Civilian Employees and</u> Families of Civilian Employees.
- 3.1.4.1. Pending further guidance, NAF Atsugi and BMC Atsugi will offer to include these individuals in the Health Consultation process previously described for active duty members and family members.
- 3.1.4.2. There will be no payment required for the Health

Consultations conducted at the BMC Atsugi or offered as part of the medical overseas screening process for suitability of assignment through employment to NAF Atsugi.

- 3.1.4.3. The process and forms used to provide Health Consultations to civilian employees and family members will be as described in section 3.1.3.
- 3.1.4.4. BMC Atsugi will retain documentation of the Health Consultations provided to civilian employees and family members, pending further guidance.
- 3.2. Risk Communication and Health Consultation at NAF Atsugi June/July/August 1998. In order to ensure that the population of NAF Atsugi and each Tenant command is afforded an opportunity to receive current information about the environmental issues at NAF Atsugi and the many activities underway to address those issues (See Forward), NAF Atsugi, assisted by BMC Atsugi, NEHC, and BUMED, will conduct Risk Communication sessions for the entire population. Consultations will be required for families with children under the age of six years, individuals with chronic respiratory conditions, women who are pregnant or breastfeeding, and any individual requesting tour extensions or retours in NAF Atsugi that would extend the total time of assignment/employment at NAF Atsugi to 72 months for individuals or family members age six or older or 36 months if there is a family member under the age of six.
- 3.2.1. A joint CINCPACFLT, BUPERS, BUMED message 042358ZMay98 directed that Risk Communication and Health Consultation would occur for everyone currently onboard NAF Atsugi or any of its Tenant Commands (See Appendix I).
- 3.2.1.1. Risk Communication.
- 3.2.1.1.1. These briefings were to be conducted as small group sessions with no more than 50 participants.
- 3.2.1.1.2. A Risk Communication Team, consisting of a representative from NAF Atsugi, BMC Atsugi, and BUMED/NEHC, would conduct these briefings between the months of June and August.
- 3.2.1.1.3. The briefings were to relate the nature and magnitude of health risks related to air quality, inform individuals of specific actions to reduce health risks, and describe the ongoing studies, including the full Health Risk Assessment and the medical epidemiology studies, that are attempting to further determine risk.

- 3.2.1.1.4. The briefings were to identify individuals potentially at greater risk for health effects related to environmental conditions at NAF Atsugi. These individuals were to be referred for a Health Consultation. Any person could request a Health Consultation.
- 3.2.1.1.5. At the time of the Risk Communication briefing each individual would document by signature on a page 13 entry that they attended the Risk Communication, did or did not request a Health Consultation, or elected to defer the decision on the Health Consultation.
- 3.2.2. NAF Atsugi released a GENADMIN 210900ZMay98 (see Appendix J) directing all Naval Activities onboard NAF Atsugi to participate in the mandatory Risk Communication Sessions.
- 3.2.2.1. The small group sessions of Risk Communication began on 8 June 1998, and are inclusive of all military and civilian personnel, age 18 and over.
- 3.2.2.2. The risk communication brief is followed by a poster availability session (see Appendix K) during which the Risk Communication Team is available to answer individual questions or concerns.

3.2.1.2. Health Consultations.

- 3.2.1.2.1. Those individuals, potentially at higher risk for health effects and as described in section 3.1., were to be scheduled, at the time of the Risk Communication Briefing, for a subsequent Health Consultation at BMC Atsugi. Medical and personnel records were to be screened to identify persons in need of one-on-one Health Consultations.
- 3.2.1.2.2. Health Consultations began on 9 June 98 in the BMC Atsugi, conducted by the Occupational Health Team contracted by BUMED. Civilian employees and their family members are also offered the recommended or elective Health Consultation with no payment required.
- 3.2.1.2.3. The elements of the Health Consultation are as described in section 3.1.3.4.
- 3.3. Risk Communication and Health Consultation Prior to Permanent Change of Station Departure from NAF Atsugi and Tenant Commands. Prior to PCS departure from NAF Atsugí,

all Service members, civilian employees, and any of their family members must complete a Health Consultation at the BMC Atsugi. The PSD of NAF Atsugi and tenant commands must ensure that this required consultation is completed and documented prior to PCS departure of any family or individual completing an assigned tour at NAF Atsugi or any tenant commands. NAF Atsugi will make a page 13 entry to document completion of the required Health Consultation prior to PCS departure from NAF Atsugi.

3.3.1. Risk Communication.

- 3.3.1.1. Risk Communication will be provided as an element of the Health Consultation conducted by the staff of BMC Atsugi.
- 3.3.1.2. Risk Communication will be documented on the SF 600 overprinted completed by medical at the time of the Health Consultation.
- 3.3.1.3. Each Service member or civilian employee and all family members over the age of 17 years will receive a copy of the most current NAF Atsugi Health and Environmental Fact Sheet.

3.3.2. Health Consultation.

- 3.3.2.1. A one-on-one Health Consultation must be completed for each individual and family member prior to PCS departure from NAF Atsugi.
- 3.3.2.2. The Health Consultation will be conducted by the BMC Atsugi.
- 3.3.2.3. The elements of the Health Consultation are described in section 3.1.3.4.
- 3.3.2.4. The SF600 overprint completed at the time of the PCS departure Health Consultation (see Appendix U) will also document history on where service members and family members lived, worked, or attended school or day care while at NAF Atsugi.

4.0 Community Outreach Program at NAF Atsugi. The Community Outreach Program at NAF Atsugi is a multifaceted program conducted by NAF Atsugi in consultation with NEHC, BUMED, CNFJ, and CPF. The CINCPACFLT message 042358ZMay98 (Appendix I) directs that the Community Outreach Program at NAF Atsugi be broadened and intensified in accordance with the Comprehensive Plan for Risk Communication and Health Consultation at NAF Atsugi. The message also directs NAF Atsugi, with the assistance of BUMED, to develop a method of evaluating the effectiveness of the Community Outreach Program and Risk Communication.

4.1. Community Outreach Program Overview.

4.1.1. Goal. While the Navy has already conducted many community outreach actions to provide information and recommendations to the NAF Atsugi community members, an overarching plan will ensure that information continues to be regularly disseminated. The goal of the Community Outreach Program is to address health concerns and keep the community informed of the continuing actions the Navy is taking to assess the risk of the poor air quality at NAF Atsugi to health. The community must continue to be informed of actions to minimize exposure of the population to the poor air quality at NAF Atsugi. The community will be informed of progress in the ongoing diplomatic negotiations related to the operation of the adjacent Jinkanpo Incineration Complex. The Community Outreach Program will provide information to current NAF Atsugi residents, individuals and families considering transfer to NAF Atsugi, and members of the media.

4.1.2. Objectives.

- 4.1.2.1. Provide information to individuals and families in order to assist them in making informed decisions regarding their stay at NAF Atsugi.
- 4.1.2.2. Relay information to those individuals considering NAF Atsugi as a duty station.
- 4.1.2.3. Actively solicit and be responsive to the concerns of our military families, civilian personnel, DODDS employees, and other involved individuals.
- 4.1.2.4. Ensure updated information is regularly provided to our military families, civilian personnel, DODDS employees, and other involved individuals.
- 4.1.2.5. Ensure information is clear, accurate, and

consistent.

- 4.1.2.6. Ensure timely and regular release of all information.
- 4.1.2.7. Establish communication responsibilities.
- 4.1.2.8. Provide communication training appropriate to the level of responsibility of the communicator.
- 4.1.3. <u>Program Elements</u>. The Community Outreach Program will be conducted by NAF Atsugi and contains several elements.
- 4.1.3.1. History of Risk Communication Activities at NAF Atsugi. There have been many previous risk communication activities to address the dual issues of poor air quality and the risk to the health of individuals at NAF Atsugi. Risk communication is ongoing and newly arrived Service members, civilian employees, and all family members over the age of 17 are scheduled to attend a Risk Communication Session for New Arrivals shortly after PCS check in to NAF Atsugi or tenant commands. There are also ongoing diplomatic actions being conducted by United States Government representatives with the Government of Japan (GOJ) and Kanagawa Prefectural Government (KPG).
- 4.1.3.1.1. Past Risk Communication Activities.
- 4.1.3.1.1.1 NAF Atsugi has had the lead to communicate environmental risk information to residents.
- 4.1.3.1.1.2. Experts in health risk communication at the Navy Environmental Health Center (NEHC) have assisted the staff of NAF Atsugi and Branch Medical Clinic Atsugi with training in methods of effective risk communication and education. NEHC has also assisted NAF Atsugi staff with preparation of materials and presentation of risk communication activities. Risk communication has attempted to convey health risk in proper perspective as well as provide information on actions individuals can take to reduce their health risk.
- 4.1.3.1.1.3. NAF Atsugi has hosted the following formal events.
 - Oct 95: Public exhibit.
 - The public exhibit (1) reviewed background and results of the 1995 Engineering Study and HRA in understandable language, (2) addressed health and

- medical issues, (3) discussed base initiatives, and (4) discussed related political issues.
- Voluntary health screening was offered to concerned personnel.
- A plan to educate base personnel on sensible precautions to protect their health was addressed.
- Feb 97: Shirley Lanham Elementary School Teachers Brief.
- APR 97: Public exhibit.
- Apr 97: Shirley Lanham Elementary School 4th Grade Class Brief.
- Nov 97: Public meeting.
 - NAF Atsugi hosted a Public Meeting to update residents on ongoing efforts by the Navy to resolve the incinerator issue. Public exhibits and information booths with subject matter experts were set up to provide information on the nature of air pollution at Atsugi, related medical risk, and individual protective measures and engineering controls to reduce exposure to poor air quality and incinerator emissions.
 - The CO NAF Atsugi and representatives from CNFJ and NAVENVIRHLTHCEN met with a group of concerned citizens to discuss specific issues.
- Jan 98: NAPRA (tenant command) safety stand down brief.
- <u>Feb 98</u>: Branch clinic sponsored training for Child Development Center, Youth Center and Family Home Care Providers.
- Mar 98: Branch clinic sponsored training for family members.
- 4.1.3.1.1.4. NAF Atsugi has hosted training sessions in Risk Communication. With the assistance of NAVENVHLTCEN, the following training sessions have been provided in support of personnel participating in the Risk Communication activities at NAF Atsugi. Training was conducted by Dr. Vincent Covello, the DOD expert consultant in methods of health and environmental risk communication.
 - Jan 97: A three day off-site Health and Environmental Risk Communication Workshop was conducted for personnel at NAF Atsugi and USNH Yokosuka.
 - Jun 98: A day and a half training session in Risk Communication was held at NAF Atsugi for personnel designated as participants in the scheduled risk

- communication and health consultation activities planned for the summer of 1998.
- Jun 98: A two hour executive brief in techniques of effective risk communication was held for the invited senior leadership personnel at NAF Atsugi, CPF, USNH Yokosuka, and Branch Medical Clinic Atsugi.
- Jun 98: A one half day session of training in Risk Communication techniques was held for the remainder of the staff at Branch Medical Clinic Atsugi who were not designated as direct participants in the planned summer 1998 risk communication and health consultation activities at NAF Atsugi.
- 4.1.3.2. Communication Techniques. Several communication techniques will be used by NAF Atsugi to maintain two-way public dialogue. Appropriate use of these methods will ensure that the NAF Atsugi community is properly informed on health issues, educated in personal actions to take to reduce individual exposure to poor air quality, aware of the most recent findings of ongoing studies, and given the opportunity to ask questions and receive replies from Navy experts. Utilization of these techniques will also assist Navy leadership in remaining aware of changing community concerns. Communication techniques to provide a comprehensive public outreach program must inform the community and educate the community.
- 4.1.3.2.1. <u>Informing the Community</u>. NAF Atsugi will use several means of keeping the community informed of health issues and ongoing environmental studies related to the air quality at NAF Atsugi in order that individuals can make decisions as to whether they should remain at NAF Atsugi. Furthermore, the techniques used will ensure that the community members have adequate opportunities to ask and get responses to their questions concerning the various aspects of this issue. This section will address:

4.1.3.2.1.1. Process of Informing the Community.

4.1.3.2.1.1.1. Community members must continue to receive information in a timely manner. Building trust and credibility within the community will be clearly linked with the timeliness, clarity, and directness of the processes used to provide information to the community. In general, the less information people have, the more questions and concerns they will have.

- 4.1.3.2.1.1.2. Key community members and NAF Atsugi civilian employees will be identified and included in the process of channeling information to the NAF Atsugi community. Communications with these individuals will build and sustain awareness of the air quality assessment activities and ongoing activities to improve the air quality at NAF Atsugi.
- 4.1.3.2.1.1.3. Multiple channels and several techniques will be used to provide information to the military and civilian workforce, their family members, the media, and other interested individuals. NAF Atsugi Public Affairs Office will prepare and distribute information concerning ongoing environmental and medical epidemiology studies, recommendations for decreasing personal exposure to poor air quality, and, as appropriate, the status of governmental negotiations regarding incinerator operations. NAF Atsugi, through the Public Affairs Office, will coordinate distribution of information received from the Public Works Office, NEHC, CPF, CNFJ, and BUMED. All written questions and comments will receive a written response. The following communication techniques are or will be in use at NAF Atsugi.

4.1.3.2.1.1.3.1. <u>Information Repositories</u>.

- 4.1.3.2.1.1.3.1.1 Purpose. Information repositories will ensure that the public has regular access to information concerning NAF ATSUGI air quality issues.
- 4.1.3.2.1.1.3.1.2. Location. An information repository has been established at the NAF Atsugi Library.
- 4.1.3.2.1.1.3.1.3. Content. The information repository will contain the community outreach plan, public comments and responses to the comments, public notices, fact sheets, newspaper articles, reports of health and environmental studies, and videos of previous reports or interviews.

4.1.3.2.1.1.3.2. Public Notices.

- 4.1.3.2.1.1.3.2.1. Purpose. The primary purpose of public notices is to provide an official announcement of activities and plans concerning the air quality at NAF Atsugi.
- 4.1.3.2.1.1.3.2.2. Location. Public notices will be placed in the Skywriter (the NAF Atsugi newspaper), on the NAF Atsugi Website and on the Armed Forces Radio and Television Network (AFRIN).

- 4.1.3.2.1.1.3.2.3. Content. Public Notices will notify the community of the time and location for Public Availability Sessions and other important events related to the issue of air quality at NAF Atsugi.
- 4.1.3.2.1.1.3.3. Fact Sheets. BUMED and the NEHC have developed fact sheets that provide information regarding health and the environment at NAF Atsugi (see Appendix F and L). Fact sheets will be updated as additional information is available.
- 4.1.3.2.1.1.3.3.1. Purpose. Fact Sheets summarize technical information and help to inform the public through a concise summary that can be read by all community members.
- 4.1.3.2.1.1.3.3.2. Location. Fact sheets will be provided and reviewed at the time of the Medical Overseas Screening of Suitability for Overseas Assignment, prior to arrival at NAF Atsugi. Fact sheets will be distributed at the NAF Atsugi Base Orientation and available at all community outreach events such as public availability sessions, exhibits, etc. The fact sheet will also be in the NAF Atsugi Base Library information repository and posted on the NAF Atsugi and NEHC web site.
- 4.1.3.2.1.1.3.3.3. Content. Fact sheets are brief summaries that use non-technical terminology to describe the risk assessment process for environmental pollutants, the pathways through which an individual may be exposed to pollutants, how health risks are determined, the status of the ongoing health risk assessment at NAF Atsugi, and recommendations for actions that may decrease an individual's exposure to air pollution. Fact sheets will also provide point of contact information if additional information is desired.
- 4.1.3.2.1.1.3.4. <u>Newsletters</u>. Newsletters ensure that community members have a regular means of receiving updated information and point of contact information.
- 4.1.3.2.1.1.3.4.1. Purpose. Providing frequent updates on the progress of multiple actions addressing the air quality situation at NAF Atsugi is essential to keeping the community informed.
- 4.1.3.2.1.1.3.4.2. Location. Following review and concurrence by NAF Atsugi, the newsletter will be distributed to the community through appropriate Base channels such as the Omsbudsman program. The newsletter

- will also be placed in the information repository at the NAF Atsugi Base Library. The first newsletter will describe the interim environmental sampling results of the full health risk assessment study. The projected distribution date is September 1998.
- 4.1.3.2.1.1.3.4.3. Content. NEHC will produce a newsletter every three months that will describe progress to date on the full health risk assessment and the medical epidemiology studies. NAF Atsugi will add local information concerning actions to decrease exposure of the base population to poor air quality and progress on diplomatic negotiations to effect modification of the Jinkanpo (Shinkampo) Incinerator operations, if appropriate. The newsletter format supports the use of maps or other graphics that will allow the community to receive some additional aids to assist their understanding of technical information.
- 4.1.3.2.1.1.3.5. <u>Press Releases</u>. In coordination with CNFJ, NAF Atsugi will continue to utilize press releases to provide timely information to the NAF Atsugi base population and surrounding community members.
- 4.1.3.2.1.1.3.5.1. Purpose. Press releases describe the actions being taken to address the concerns of the Navy and the community. Press releases for the media will commonly be used to announce community outreach activities, to report the results of public meetings/public availability sessions, and to describe significant interim findings in environmental surveys or in the ongoing full health risk assessment.
- 4.1.3.2.1.1.3.5.2. Location. Following established review and coordination procedures with CNFJ, NAF Atsugi Public Affairs Office will distribute press releases to the base newspaper, "The Skywriter", and the local broadcast media. Press releases can also be placed on the NAF Atsugi Base Information Channel.
- 4.1.3.2.1.1.3.5.3. Content. Press releases will be used to announce community outreach events, such as public availability sessions. Press releases will also describe key technical milestones and findings in the ongoing environmental surveys and health risk assessment processes.
- 4.1.3.2.1.1.3.6. <u>Telephone Infoline (Commanders' Action</u> Line or Health Risk Communication Office).
- 4.1.3.2.1.1.3.6.1. Purpose. It is important for the

- public to have a relatively quick means of expressing their concerns directly to NAF Atsugi and getting questions answered.
- 4.1.3.2.1.1.3.6.2. Location. Interested individuals can call the Health Risk Communication Office or the NAF Atsugi Commanders' Action Line to leave a recorded phone message describing the questions or concerns. Individuals will receive a reply, by phone, within 24-48 hours. A phone log will be used to record all calls, questions asked, or response given.
- 4.1.3.2.1.1.3.6.3. Content. The Commanders' Action Line is an established process by which any resident, employee, or beneficiary can address any question or concern directly to the Command. While not a specific hot line for air quality concerns, the Commanders' Action Line is an established and familiar means through which any individual can directly address the NAF Atsugi Command and receive a prompt reply. The Health Risk Communication Line is a dedicated line through which any individual can express environmental concerns and questions.
- 4.1.3.2.1.1.3.6.4. Records. It is important to record the number of calls and the types of questions related to environmental exposure/health concerns that come to the Commanders' Action Line. This information will be a necessary component of the overall Community Outreach Program evaluation.

4.1.3.2.1.1.3.7. Website.

- 4.1.3.2.1.1.3.7.1. Purpose. An NAF Atsugi website will provide information to current, future, and past NAF Atsugi residents concerning the Jinkanpo (Shinkampo) Incinerator issue.
- 4.1.3.2.1.1.3.7.2. Location. The NAF Atsugi website will be maintained by the NAF Atsugi Public Affairs Office. It can be found via the internet at www.atsugi.navy.mil. There is a link to Jinkanpo (Shinkampo) and to the NEHC.
- 4.1.3.2.1.1.3.7.3. Content. The NAF Atsugi website will provide information such as the status of negotiations with the Japanese, upcoming community outreach events, the number to the NAF Atsugi Commanders' Action Line, answers to frequently asked questions and any other pertinent information. The NAF Atsugi website will link to the NEHC website which will provide fact sheets and information

concerning the status of the health risk assessment and epidemiological studies.

4.1.3.2.1.1.3.8. Questions and Answers.

- 4.1.3.2.1.1.3.8.1. Purpose. A Frequently Asked Question list will be used to address specific concerns at the community level of interest.
- 4.1.3.2.1.1.3.8.2. Location. NAF Atsugi has developed a comprehensive list of frequently asked questions. These questions and answers are posted on the NAF Atsugi web site and in the Information Repository at the NAF Atsugi Base Library. The Frequently Asked Questions (see Appendix T) were most recently updated in May 1998, and are also distributed at public exhibits and public availability sessions.
- 4.1.3.2.1.1.3.8.3. Content. While the development, editing, and distribution of the Frequently Asked Questions is the responsibility of NAF Atsugi, in future revisions NEHC will assist NAF Atsugi in providing responses to the questions, at the community level of interest. Close coordination between NAF Atsugi and NEHC will be essential to assuring that the responses are updated as additional information is acquired and that a clear and coordinated message is provided. Additional questions from the community can be gathered from ongoing monitoring of public availability sessions, Commanders' Action Line calls, and website inquiries.
- 4.1.3.2.2. Educating the Community. A key objective in any communication effort is to ensure that people have continuous and ready access to information and can understand the technical aspects of the issue of concern. NAF Atsugi will use public meetings, public exhibits, public availability sessions, and community advisory panels as means of educating the community in order to assist the community members' understanding of the technical information being collected. Through education, the community members will be better prepared to make educated decisions regarding their health and the health of their family members.

4.1.3.2.2.1. Process of Educating the Community.

4.1.3.2.2.1.1. Education activities must be timely and linked with completion of key activities such as the full health risk assessment or the medical epidemiology studies.

- 4.1.3.2.2.1.2. Education activities should include key community representatives as well as health or environmental experts.
- 4.1.3.2.2.1.3. Several forums will be used to educate the community and are described in the following segments.

4.1.3.2.2.1.3.1. Command Briefings/Community Meetings

- 4.1.3.2.2.1.3.1.1. Purpose. Command briefings or community meetings are meetings at which NAF Atsugi staff and other experts are available to present information and respond to questions/concerns. The residents are also offered the opportunity to ask questions, raise issues and receive feedback. This type of meeting should only be used as prelude to a public availability session immediately following the briefing.
- 4.1.3.2.2.1.3.1.2. Location. Public command briefings or community meetings will be scheduled by NAF Atsugi as new information becomes available through the ongoing medical epidemiology studies or the environmental studies and the full health risk assessment. The command meetings will be conducted in a NAF designated facility that can support movement of the community members from the larger community gathering to a nearby location housing the public availability session.
- 4.1.3.2.2.1.3.1.3. Content. NAF Atsugi designated representatives present summary information to the gathered community. General questions can be addressed, but the community is advised that additional experts can provide further detail and response to individual questions at the public availability session. NEHC and BUMED will provide representatives to address the findings of the full health risk assessment or the medical epidemiology studies at the request of NAF Atsugi.

4.1.3.2.2.1.3.2. Public Availability Sessions.

4.1.3.2.2.1.3.2.1. Purpose. Public availability sessions are designed as a follow on to public community meetings or command briefings or may be scheduled in place of large community gatherings in order to facilitate two way dialog between community members and topic experts. Public availability sessions provide an opportunity for individuals to ask questions and express concerns to those experts who are most knowledgeable in the area of interest. Public

exhibits such as poster presentations are often included in public availability sessions.

- 4.1.3.2.2.1.3.2.2. Location. Public availability sessions will be scheduled as an optional activity for any community member attending any command public meeting addressing any aspect of air quality actions at NAF Atsugi. The public availability session should be conducted in an area adjacent to but separate from the location of the command briefing or community meeting. All community members attending the public command briefing or community meetings will be invited to attend the public availability session.
- 4.1.3.2.2.1.3.2.3. Content. The public availability session will have a series of poster displays with visual aids that summarize ongoing activities and/or findings of ongoing investigational studies. Technical experts and NAF Atsugi command representatives will be available to discuss individual questions or concerns.

4.1.3.2.2.1.3.3. Public Exhibits.

- 4.1.3.2.2.1.3.3.1. Purpose. Public exhibits are a convenient one-way communication tool that offer a means of maintaining public awareness. NAF Atsugi, with the aid of NEHC, will develop and display public exhibits periodically.
- 4.1.3.2.2.1.3.3.2. Location. Public exhibits can be placed at any common area such as the Family Service Center or the Base Library. There can be continuous public exhibits or periodic public exhibits when new information is available.
- 4.1.3.2.2.1.3.3.3. Content. Public exhibits utilize poster presentations with graphics and other visual aids to present summaries of current information concerning overall status of NAF Atsugi air quality issues. Point of contact information is also provided. Public comment can be solicited through written questions deposited in a central collection box. If public comment is invited, all comment or questions must receive a response from designated representatives. Other printed information such as fact sheets or newsletters may also be distributed at Public Exhibits.

4.1.3.2.2.1.3.4. Small Community Group or Unit Briefings/Presentations.

4.1.3.2.2.1.3.4.1. Purpose. Individual groups, such as

- Childcare Center Caregivers or Teachers, may request briefings or presentations in a smaller setting than the Command Brief or Community Meeting. NAF Atsugi will coordinate scheduling and staffing for these sessions. Responding to these requests for informational updates fosters coordination of community actions and provides an opportunity for open discussion of concerns and recommendations.
- 4.1.3.2.2.1.3.4.2. Location. These briefings are hosted by the group requesting the presentation and are conducted at the worksite. These briefings will be scheduled at the request of the individual group.
- 4.1.3.2.2.1.3.4.3. Content. In general, the content of the program will include an update of the ongoing activities occurring in response to air quality issues at NAF Atsugi.
- 4.1.3.2.2.1.3.5. NAF Atsugi Community Advisory Panel (Jinkanpo/Shinkampo Action Team).
- 4.1.3.2.2.1.3.5.1. Purpose. The purpose of the community advisory panel (CAP) is to create an opportunity for key NAF Atsugi community members to receive information and provide input regarding the many activities addressing the issues of poor air quality at NAF Atsugi. Any interested community member may attend.
- 4.1.3.2.2.1.3.5.2. Location. The NAF Atsugi Community Advisory Panel, called the Jinkanpo (Shinkampo) Action Team, meets regularly in an advertised location on base. The meeting is announced in the Base newspaper.
- 4.1.3.2.2.1.3.5.3. Content. The Jinkanpo (Shinkampo) Action Team is the CAP. The CAP is the responsibility of NAF Atsugi and is chaired by the Executive Officer of NAF Atsugi. Membership includes the NAF Atsugi Omsbudsman, representatives from the Public Works Office, representatives from the school, representatives from Industrial units, representatives from Public Affairs Office, and any interested community members or tenant command representatives. The CAP meetings offer a forum for discussion of progress on the health risk assessment, medical epidemiological studies, or the ongoing negotiations with the Government of Japan and Kanagawa Prefecture regarding the operation of the Jinkanpo (Shinkampo) Incinerator. The Jinkanpo (Shinkampo) Action Team also reviews technical documents and provides advice about community concerns. Members also carry information to the

- community. A fact sheet is being developed that will describe the functions of the Jinkanpo (Shinkampo) Action Team and emphasize that membership is available to any interested community member.
- 4.1.3.2.3. Evaluating Community Outreach Activities. The specific communication techniques being used to conduct the community outreach program will be evaluated for effectiveness in providing information to the community and addressing the community's concerns. NAF Atsugi and the tenant commands of NAF Atsugi are responsible for ensuring ongoing evaluation of the overall community outreach program with the assistance of the Jinkanpo (Shinkampo) Action Team and the Health Risk Assessment Coordinator. NEHC can assist NAF Atsugi with development of evaluation instruments and analysis of the evaluation findings. Evaluation results will be reported quarterly to CPF and BUMED. If specific products or communication techniques are not effectively addressing community concerns revision of the community outreach program must occur.
- 4.1.3.2.3.1. Meeting and Activity Evaluation Forms. A comment and evaluation form will be developed by NAF Atsugi to distribute to participants at appropriate public outreach events, such as small group meetings, presentations, public meetings, etc. to solicit feedback on what took place. These forms will be utilized by NAF Atsugi staff to monitor effectiveness of specific activities and to make any changes or revisions. A similar evaluation card will be used to evaluate fact sheets, exhibits, etc. which the respondents can hand back or mail to the NAF Atsugi PAO Office.
- 4.1.3.2.3.2. Monitoring Community Response. NAF Atsugi will develop mechanisms for evaluating the community response to outreach activities. Techniques will include (1) monitoring news coverage for number, length, and tone of reports; (2) monitoring specific public events (i.e., public meetings, presentations) for the number of people attending and the types of interests that the attending community express; (3) monitoring distribution of fact sheets and mailing cards for the number and location; and (4) monitoring phone calls for the number and tone. It is important to record the kind of questions the community is asking as well as how those change over time. Interviews may also be used to monitoring the effectiveness of public outreach activities.
- 4.1.3.2.4. Community Outreach Program Revisions. NAF Atsugi will revise the community outreach program as indicated by the results of the program evaluations. Revisions will occur as the multiple activities addressing air quality

issues at NAF Atsugi progress. Community concerns will change. Information obtained from community input at Jinkanpo (Shinkampo) Action Team meetings, the Commander's Action Line, and other forums for two-way communication will provide a means of assessing the type of community concerns. This information will be used to determine the need for specific community outreach activities. Additional public outreach activities may be developed as needed to address issues emerging from the health risk assessment or other activities being conducted. Public outreach activities that are not effective or no longer needed will be discontinued. Also, any information that may have changed (points of contact, addresses, phone numbers) will be updated.

- Atsugi Residents. NAF Atsugi will take the lead to issue information to current NAF Atsugi residents, as soon as possible, after technical findings from environmental and epidemiology studies are confirmed. Information will be provided in a clear and concise manner. The Navy Environmental Health Center will assist NAF Atsugi in their risk communication efforts. Branch Medical Clinic Atsugi is also engaged in day-to-day operations designed to communicate the status of the actions being taken to assess the quality of the air at NAF Atsugi, evaluate the potential health effects of poor air quality, and educate residents on effective measures to protect their health.
- 4.2.1. NAF Atsugi Facility Personnel. NAF Atsugi is the responsible agent for performing several actions.
- 4.2.1.1. NAF Atsugi will include attendance at a risk communication presentation as one of the required actions to be completed during the permanent change of station check in process. All newly arrived personnel and family members over the age of 17 will be required to attend a Risk Communication Session for Newly Arrived Personnel.
- 4.2.1.2. NAF Atsugi will designate a command representative to deliver the Risk Communication Brief given at the time of the required Risk Communication Session for Newly Arrived Personnel.
- 4.2.1.3. NAF Atsugi will provide education on methods to minimize exposure to incinerator emissions on days when base air quality is poor or questionable.
- 4.2.1.4. NAF Atsugi will provide education to minimize exposure to soot from incinerator emissions and other sources of poor air quality.

- 4.2.1.5. NAF Atsugi will maintain a base wide pollution advisory and alert system.
- 4.2.1.6. NAF Atsugi will hold small group briefings for local commands upon request.
- 4.2.1.7. NAF Atsugi will give one-on-one briefs to concerned base residents.
- 4.2.1.8. NAF Atsugi will provide input for frequent articles to be published in the Skywriter or the Stars and Stripes.
- 4.2.1.9. In consultation with NEHC, NAF Atsugi will monitor and evaluate the community response to community outreach programs.
- 4.2.2. Branch Medical Clinic Atsugi. BMC Atsugi will continue to provide community health care with a heightened awareness of the potential exacerbating effects of chronic exposure to the poor air quality at NAF Atsugi. Branch Medical Clinic Atsugi will conduct Health Consultations, both elective and required in accordance with the overall Comprehensive Risk Communication and Health Consultation Plan. Branch Medical Clinic staff will participate or have participated in the following activities.
- 4.2.2.1. Designated BMC Atsugi staff will be co-presenters, with NAF Atsugi representatives, of the general Risk Communication Brief given at the time of the Area Orientation Brief.
- 4.2.2.2. One-on-one Health Consultations will be scheduled for any NAF Atsugi personnel or family member who requests evaluation of the potential environmental impact on their own individual health.
- 4.2.2.3. As personnel, active duty or civilian employees, and family members report aboard NAF Atsugi, the Branch Medical Clinic staff will review individual medical records to ensure that Risk Communication and Health Consultation was received at the time of the medical overseas screening. If not, individuals or families will be scheduled for a mandatory Health Consultation appointment.
- 4.2.2.4. Personnel departing NAF Atsugi at completion of a 36 month tour or persons requesting tour extension will be scheduled for a mandatory Health Consultation.

- 4.2.2.5. Medical staff have conducted voluntary health screening physical examinations for concerned individuals and documented provision of risk communication on overprint SF 600's.
- 4.3. Approach for Ongoing Communications With Media Relations and Public Information. The program details the proactive approach for providing information to the local newspapers and broadcasting network.
- 4.3.1. Presentation Development. Following receipt of updated information from NAF Atsugi concerning the status of ongoing diplomatic negotiations for modification of Jinkanpo (Shinkampo) incinerator operation, environmental sampling and survey status, Command actions to mitigate exposure, and timeline for completion of the Full Health Risk Assessment, NAVENVIRHLTHCEN will develop a full presentation for use by members of the NAF Atsugi Health Risk Communication Team members. To ensure that information is presented correctly and consistently, as new information is obtained and validated NEHC will assist with interim presentation updates. NEHC can coordinate the training of NAF Atsugi designated presenters to who will deliver the brief or function as NAF representatives for media conferences.
- 4.3.2. NAF Atsugi Role in Media Relations And Public Information.
- 4.3.2.1. NAF Atsugi Public Affairs Office is responsible for developing a plan for media relations and public information.
- 4.3.2.1.1. NAF Atsugi Public Affairs Office will carry out the public affairs plan for interacting with the media and providing public information as directed by NAF Atsugi.
- 4.3.2.1.2. NAF Atsugi Public Affairs Office will coordinate review through existing chain of command.
- 4.3.2.2. Goal. In coordination with CNFJ and CPF, release timely, regular and accurate information to the media and the general public and ensure prompt response to all inquiries.
- 4.3.2.3. Objectives.
- 4.3.2.3.1. <u>Provide Information</u>. Provide information to reporters and editors that will assist their effort to convey the story to their audiences.

- 4.3.2.3.2. Open Communication. Maintain open communication with key reporters and editors.
- 4.3.2.3.3. Response. Respond to inquiries from the media and members of the public.
- 4.3.2.3.4. Release Information on Latest Developments to the Media. Issue press releases on significant findings, milestone events, etc. Present technical results in context to make it easy for reporters to understand the story behind the data.
- 4.3.2.3.5. <u>Meet with Editorial Staff</u>. Request meetings with editorial staff of local and regional publications, and television and radio stations.
- 4.3.2.3.6. Conduct Tours for Media Contacts. Invite reporters and editors to individually tour the site.
- 4.3.2.3.7. Address inaccuracies. Prepare letters and other responses to the media correcting inaccurate information in a polite, conciliatory, but non-defensive manner.
- 4.3.2.3.8. Publicize Community Outreach. Continue to publicize community outreach activities and scientific studies that are ongoing.
- 4.3.2.3.9. <u>Provide Information</u>. Make on-going technical data/environmental safety and health information available to reporters even when data is not considered surprising or "significant."
- 4.3.3. BUMED Role in Providing Public Information.
- 4.3.3.1. Responsible organization.
- 4.3.3.1.1. Navy Environmental Health Center will assist With development of products to be used in Risk Communication activities and Health Consultation activities directed by NAF Atsugi.
- 4.3.3.1.1.1. Products Already Developed.
- 4.3.3.1.1.1. Health and Environmental Fact Sheet (Dec 97) (Appendix L).
- 4.3.3.1.1.1.1. Addresses the environment at Atsugi and the cancer and non-cancer health risks determined from the screening HRAs.

- 4.3.3.1.1.1.2. Designed by professional health risk communicators and written at the community level of concern.
- 4.3.3.1.1.1.3. Was revised on 8 June 1998, (Appendix C) for use in the medical overseas screening process and ongoing Risk Communication and Health Consultation activities offered to the entire population of NAF Atsugiduring the Summer of 1998.
- 4.3.3.1.1.1.4. Will be posted on the NAVENVIRHLTHCEN Website and made available to the community by NAF Atsugi.
- 4.3.3.1.1.1.2. Medical Provider Script (Dec 97) (Appendix M). Designed for the medical provider to communicate the health risk to the active duty member and his/her family members during the overseas medical screen or the one-on-one health consultation.
- 4.3.3.1.1.2.1. Replaced with a Medical Provider Background Document (April 1998) (Appendix E) that is offered as a preread for those providers who will be conducting the medical overseas screening evaluations for personnel and family members undergoing permanent change of station transfer to NAF Atsugi.
- 4.3.3.1.1.1.3. Overprint SF 600 (FEB 98) (Appendix N).
- 4.3.3.1.1.3.1. Designed to document delivery of the health consultation risk communication by a medical provider.
- 4.3.3.1.1.3.2. Revised in April 1998 (Appendix G) to support documentation of provision of Risk Communication and components of Health consultation.
- 4.3.3.1.1.1.4. Poster on Dioxin (Appendix O). Used in Nov 97 public exhibit. Designed to provide health information concerning dioxin, one of the major chemicals of concern with regard to soil and air pathways of exposure.
- 4.3.3.1.1.5. Poster on Risk Assessment (Appendix P). Designed to provide information concerning the process of risk assessment.
- 4.3.3.1.1.6. Risk Communication Briefing slides June 1998 (Appendix R). Complete briefing packet used in the basewide Summer 1998 Risk Communication sessions conducted by Risk Communication Team consisting of NAF Atsugi

representative, Branch Medical Clinic Representative, and NAVENVRNHLTHCEN/BUMED Representative.

- 4.3.3.1.1.1.7. <u>Public Availability posters</u> used in the Public Availability Session accompanying the Risk Communication Briefs during the Summer of 1998. (Appendix K).
- 4.3.3.1.1.1.8. NAF Atsugi Health Risk Assessment Progress Reports April 1998 (Appendix S) As started in April 1998, BUMED/NEHC will provide monthly technical reports to the Line on the status of the full HRA and the medical epidemiologic studies. A community progress report will be placed in the Information Repository at the NAF Atsugi Base Library approximately quarterly, or as soon as validated information is available.
- 4.3.3.1.1.1.8.1. As appropriate, information from the Monthly HRA Progress Reports and medical epidemiology studies will be posted on the NEHC home page.
- 4.3.3.1.1.2. The NEHC is available to provide assistance in health risk communication, including development/review of fact sheets, Qs&As, correspondence, posters, and other materials to be used in public outreach efforts; development/review of community educational materials; risk communication training for personnel involved with public outreach programs and assistance with providing expert staffing for public availability sessions.

- 5.0. Training in Health Risk Communication. Risk communication programs require different techniques and skills than those required in traditional communications. This is because risk communication addresses the emotional as well as informational needs of the community, and it assumes the difficult context of high concern and low trust. Risk communication principles are central to our community outreach plan. Risk communication training includes public involvement activities, public meeting participation or facilitation, community outreach initiatives, and media training.
- 5.1. <u>Individuals to Receive Training</u>. See Appendix Q for the schedule for risk communication training.

5.1.1. Workshops in Japan (Week of 1-5 June 1998).

- NAF Atsugi personnel conducting or participating in Risk Communication Brief or the Health Consultation process as well as others who participated in public meetings and/or provide responses to the NAF Atsugi community (e.g. environmental staff, PAO, JAG, etc. as determined by NAF Atsugi) attended a 1.5 day workshop on Risk Communication Techniques.
- NAF Atsugi Branch Medical Clinic (BRMEDCLINIC) personnel who were not designated briefers but have frequent contact with NAF Atsugi community participated in a 1/2 day Risk Communication Brief on 4 June 1998.
- DON Military and Civilian Executive Level Personnel participated in a 2 hour executive luncheon presentation of Risk Communication Principles and Techniques on 4 June 1998.

5.1.2. Workshops in Washington, DC.

- DON Military and Civilian Executive Level Personnel attended Power briefings (12-13 May 98).
- Detailers All detailers who were located in Washington DC were offered Risk Communication training 11-12 May 98.

5.1.3 <u>Training at Other Locations</u>.

• Medical personnel conducting overseas pre-screening physicals will be offered Risk Communication Training

with the use of a 36 minute educational videotape made by Dr Vincent Covello in coordination with the Naval School of Health Sciences. The videotape was filmed on 11 May 98. By Sept 98, this videotape will be distributed for use by the medical staff in the following locations:

- 31 Medical Treatment Facilities.
- 100 BRMEDCLINICs.
- Detailers in Millington, Tennessee, were offered Risk Communication Training on 25 and 26 May 98, conducted by NEHC.
- 5.1.4. Levels of Risk Communication Training. Risk communication training will proceed on three levels.

5.1.4.1. <u>Level One Training</u>.

- 5.1.4.1.1. Level One training attempts to establish a fundamental understanding of why and how risk communication is more successful and how it differs from conventional communication approaches. Level One training will involve substantial emphasis on skill building exercises in order to afford the participants an opportunity to improve their competence when handling difficult public communication situations. Completion of Level One training will provide the basic skills required to effectively communicate health risks to the public. These sessions will be offered by Dr. Vincent Covello or the NEHC Risk Communication Coordinator.
- 5.1.4.1.2. Level One training was presented in a 1.5 day Risk Communication workshop presented at NAF Atsugi and a two hour session for Command leadership and CNFJ. It is intended for NAF Atsugi personnel who will be participating in public availability sessions or preparing fact sheets and/or newsletters, NAF Atsugi Branch Medical Clinic (BRMEDCLINIC) personnel dealing with day to day issues regarding health risks and conducting one-on-one health consultations, and NAF Atsugi, CNFJ and other personnel who are interacting with the media.

5.1.4.1.3. Level One training consists of:

- Orientation to Risk Communication Theory
- Answering Tough Questions
- Seven Part Communication Model
- Message Development
- Community Outreach Techniques
- 5.1.4.1.4. BUMED and NEHC are responsible for providing Level One training annually for personnel at NAF Atsugi.

5.1.4.2. Level Two Training.

- 5.1.4.2.1. Level Two training will also establish an understanding of the fundamentals of Risk Communication theory and principles, but is provided in mostly lecture format with limited interactive skill building exercises. It is important that senior leadership understand the differences in Risk Communication techniques as compared to traditional communication approaches.
- 5.1.4.2.2. This training was conducted by Professor Covello in Washington, DC and at NAF Atsugi and/or CNFJ in a 2 hour "Power Luncheon" format for senior managers and executive level personnel.
- 5.1.4.2.3. Level Two training consists of:
 - Introduction to Risk Communication Theory
 - Implications of Communicating in High Concern/ Low Trust Situations
 - Alternatives to Town Hall Public Meetings
- 5.1.4.2.4. BUMED and NEHC are responsible for providing Level Two training annually for designated personnel at NAF Atsugi.
- 5.1.4.3. Level Three Training.
- 5.1.4.3.1. Level Three training is designed to provide a basic understanding of risk communication principles and theory.
- 5.1.4.3.2. Level Three training will be presented by NEHC staff personnel at various locations throughout the United States and consists of 1 to 4 hour training sessions for medical department personnel conducting NAF Atsugi prescreening physicals and detailers.

- 5.1.4.3.3. Level Three training will be scheduled at the request of the MTF or Branch medical clinic. Level Three training will focus on preparing medical providers to conduct Risk Communication in the course of conducting a medical overseas screening evaluation for personnel assigned to NAF Atsugi.
- 5.1.4.3.4. Level Three training will consist of:
 - Introduction to Risk Communication Theory
 - Communication Pitfalls

- 6.0. Resource Requirements. Execution of this comprehensive risk communication and health consultation plan for NAF Atsugi will require resources above current operating funds. CINCPACFLT and BUMED have the lead to identify additional resource requirements to execute their respective responsibilities. Selected costs for specific programs are estimated (see Appendix V).
- 6.1. <u>BUMED</u>. BUMED has the lead to provide risk communication support and training and to conduct one-on-one health consultations.
- 6.1.1. Risk Communication Support and Training. Provision of risk communication support and training to non-BUMED activities and personnel will be handled on a reimbursable basis.
- 6.1.1.1. <u>Risk Communication Support</u>. NEHC has the lead to provide risk communication support to CPF and NAF Atsugi. NEHC negotiates with CINCPACFLT on an ongoing basis for reimbursement of services.
- 6.1.1.2. Risk Communication Training. NEHC has the lead to oversee provision of risk communication training to those involved in communicating the health risk of the environmental conditions at NAF Atsugi. Appendix provides cost estimates for provision of specific training sessions this fiscal year.
- 6.1.2. One-on-One Health Consultation. BUMED has determined that the branch clinic at NAF Atsugi will require additional personnel to conduct one-on-one health consultations and related risk communication for new arrivals, current residents, and those who are completing a permanent change of station departure from NAF Atsugi.
- 6.1.2.1. <u>Permanent Branch Clinic Staff</u>. BUMED will need to augment the permanent staff at the clinic with one active duty ambulatory care register nurse and one hospital corpsman.
- 6.1.2.2. Contractor Support. BUMED entered into a personal services contract for Occupational Health Team Services at Naval Air Facility Atsugi, Japan. This team of health professionals conducted the mandatory one-on-one health consultations from June to August 1998 at NAF Atsugi. Appendix V gives an estimate of the duration and cost of the contractor which is dependent on the final workload.

6.2. <u>CINCPACFLT</u>. CINCPACFLT has the lead to oversee execution of the risk communication plan at NAF Atsugi. Details on additional resource requirements will be forthcoming.

7.0. Appendices.

- Appendix A. Detailer Script
- Appendix B. <u>Detailer's A Text for Orders to NAF Atsugi and</u> Tenant Commands.
- Appendix C. Overseas Screening for NAF Atsugi, Japan 26 Feb 98 BUMED message.
- Appendix D. Updated Guidance on Overseas Screening for NAF Atsugi and Tenant Commands 7 July 98 BUMED message.
- Appendix E. Medical Providers Background Document
- Appendix F. NAF Atsugi Health and Environmental Fact Sheet
- Appendix G. Overprint SF 600 for One-on-One Health Consultation and Risk Communication.
- Appendix H. NAF Atsugi Page 13 used for Summer 1998 Risk Communication Briefings.
- Appendix I. Joint CINCPACFLT/BUPERS/BUMED message May 98.
- Appendix J. NAF Atsugi Message May 98.
- Appendix K. Poster Availability Session Summer 1998.
- Appendix L. <u>December 1997 Fact Sheet</u>.
- Appendix M. February 1998 Medical Provider Script.
- Appendix N. February 1998 SF 600 Overprint.
- Appendix O. Poster entitled "What is Dioxin?"
- Appendix P. Poster entitled "What is Risk Assessment"
- Appendix Q. Schedule for Risk Communication Training
- Appendix R. Risk Communication Brief Summer 1998.
- Appendix S. Sample Technical HRA Progress Report.
- Appendix T. Frequently Asked Questions (FAQ) Fact Sheet/ NAF Atsugi Spring 1998.
- Appendix U. Draft SF 600 Overprint for PCS Departure Health Consultation.

Appendix V. Estimated Cost for Contract Health Consultation Team at NAF Atsugi Summer 1998.

Page	EPA Comment	NEHC Response
Page 2	Dioxin Levels at NAF Atsugi: As a general comment on the NAF Atsugi facility, the proximity to the Shinkampo Incineration Complex (SIC) has led to soil and air levels of dioxins that are generally considered to be of concern. Specifically, the SIC is only a few hundred meters from where people reside, and in a river valley such that its stacks are essentially at ground level with nearby base residential and school settings. The extensive ambient air monitoring program on the base, which was the basis for the estimation of inhalation impacts in this risk assessment, showed an average dioxin air concentration of 1.57 pg TEQ/m³ over all sampling dates and sampling locations (TEQ = Toxic EQuivalent concentrations, which is the sum of the dioxin congeners in a mixture weighted by their individual toxicities relative to the most toxic congener, 2,3,7,8-TCDD). Concentrations directly downwind at the nearest air monitor were as high as 100 pg TEQ/m³. This compares to EPA's national compilation of air monitoring data, done as part of the Dioxin Reassessment, showing air concentrations below 0.10 pg TEQ/m³ in urban background settings, and below 0.05 pg TEQ/m³ in rural background settings (where "background settings" are defined as those where there is no immediately identified source impacting the monitoring results). Measurements above 1.00 pg TEQ/m³ are rare in the United States, even in proximity to an incinerator. Soil concentrations were higher than US background as well, with concentrations were higher than US background as well, with concentrations were higher than US background as well, with concentrations were higher than US background as the edge of the base, to concentrations between 10 and 100 ppt where most of the exposure will occur at NAF Atsugi - at the school and residential settings. In the US, rural concentrations average less than 10 ppt TEQ, and urban concentration are in the range of 20 ppt TEQ. These levels of dioxin in the ambient environment at NAF Atsugi	NEHC's current medical recommendation is not to provide testing of dioxin in blood/breast milk for the following reasons: 1. Dioxin levels in blood/breast milk are not standardized medical tests; They are costly and are still primarily a research tool. Consequently, Dioxin values from testing would vary by method used in testing and by quality control efforts in the laboratory doing the test. 2. There are no medical guidelines for interpreting results in individuals. Consequently, knowing a person has X level of dioxin in their blood is all we would know. In addition, the NAF Atsugi population is of transient nature, which may make it difficult to differentiate prior from current exposures. Appropriate control groups may also be difficult to find. As pointed out by the reviewer, any increases would likely be small and may be difficult to distinguish from the control group(s). Instead the Navy recommendation is not to conduct blood or breast milk testing but rather continue to provide medical counseling and risk communication to base residents regarding the limitations of performing these tests.

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	raise a number of issues for the risk assessment which will be highlighted now, recognizing that these topics recur during the specific comments section: Measured dioxin levels in humans: Noting 1) the substantial outlay of resources on this assessment, 2) the ongoing medical studies reported at the end of the HHRA Summary, and 3) the central impact of dioxins on the risk estimates at NAF Atsugi, it is surprising that serum dioxin levels of NAF Atsugi personnel have not been taken. With persistent toxins such as dioxins, the ultimate measure of exposure is the actual concentration in the body. Measurement of serum dioxin levels in this population, especially if coupled with measurements of matched Navy personnel at other bases in Japan, could answer a number of questions simultaneously, e.g., are dioxin doses in naval personnel stationed in Japan higher than in the US and, most importantly, are dioxin doses experienced by NAF Atsugi staff and families higher than other sites in Japan, or in the United States? It would be important to locate a suitable comparison population in Japan for such a blood sampling, since this would be only way to determine what level of exposure to dioxin at NAF Atsugi can be attributed to the SIC. In performing such measurements, it should also be understood that any increases would likely be small and may be difficult to distinguish from the control group(s).	
Page 2	EPA Dioxin Reassessment: The Draft EPA Dioxin Reassessment is scheduled for release very shortly, and will likely propose increasing the cancer slope factor for dioxin considerably, as well as pointing to a number of non-cancer health risks at levels at or within an order of magnitude of the US background. As the dioxin reassessment will be in draft form for some time, it is recommended that the NAF Atsugi assessments should continue to rely on the previously calculated	The subsection on Dioxin in the Health Evaluation section has been expanded to address the Draft EPA Dioxin Reassessment in the revised NEHC Report.

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	cancer slope factor. However, it is important for the Navy to consider the technical and political implications of the dioxin reassessment in its evaluation of NAF Atsugi.	
Page 3	Dioxin TEQs: There is no indication in the HHRA Summary as to which toxicity equivalence factors were used in the assessment. Page 62 of the Pioneer Assessment references the old EPA possibly I-TEF values from 1994. The source of these TEFs should be more clearly listed in both reports. Also, the 1994 TEF values are out of date, as most international and revised EPA assessments are adopting the WHO TEF values from 1998. If possible, the assessment should be changed to reflect these newer values, or the use of the older values clearly noted and a brief discussion included describing the [presumed] minimal impact of these changes on the assessment.	The source of the TEF values used for the risk calculations is identified on page 62 of the PIONEER report. These values were obtained from EPA's web-site and are presented in a document titled "The US EPA TEF Values" (http://www.epa.gov/nceawww1/dchem.htm). The 1998 WHO TEF values are proposed in the Draft Dioxin Reassessment (released in June 2000) but have not been adopted by EPA. As recommended by the reviewer, the Dioxin TEQs were recalculated in the revised NEHC report using the latest WHO TEF values from 1998.
Page 3	Dioxin Cancer Slope Factor: The CSF value of 150,000 kg-day/mg for dioxin was cited for both ingestion and inhalation exposures (see Pioneer Assessment Table 4-3). No explanation was given as to how this oral CSF was converted into an inhalation slope factor. Such a conversion would necessitate both route to route extrapolations and absorption assumptions. For instance, is all the inhaled dioxin assumed to be taken into the body (no expiration, full absorption, etc.)? EPA staff were unable to find an explanation of how these values were converted, although assessments typically assume 100% absorption for both pathways and that would be appropriate here as well.	The oral and inhalation slope factors for Dioxin were obtained from EPA's Health Effects Assessment Summary Tables (HEAST Table 3 Page 3-33, 1997).
Page 3	HHRA Summary could be tightened, especially its conclusions on the impact of the SIC: Although the HHRA Summary covers a lot of information, it needs to be tightened to enhance readability. The HHRA Summary provides valuable information on outdoor levels,	The NEHC report has been revised to include more specific conclusions regarding the SIC contribution to the air quality and its impact on health risk. In the revised report these conclusions address a list of chemicals statistically associated with the SIC, the 50% increase in cancer and non-

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	indoor measures, models, winds, risks to the GEMB, etc., and yet loses the reader regarding its ultimate message. In order to make sense of the sometimes conflicting results, I was forced to prepare my own summary of results on a separate sheet of paper to try and piece together what was or was not attributable to the SIC. Thus, I would suggest that the HHRA Summary redo the conclusions section to succinctly summarize what is known about risks at the site, which risks are most likely attributable to the SIC, and these levels in comparison to risks to the US population. It is probable that the difficulty in summarizing this information relates to the inherent difficulties in apportioning the risks based on the available data. While both NAF Atsugi documents under review demonstrate admirable efforts by the Navy in trying to apportion these risks accurately, EPA reviewers suggest that the Navy needs to go a little further in tightening the link to the SIC and focusing their conclusions. One suggestion is to look to data on similar incinerators to determine which chemicals are emitted, and to use this information to refine the determination of what constituents are potentially related to the SIC. Clearly, dioxins, metals, and PM ₁₀ were linked, but some of the other chemicals such as hydrochloric acid would also presumably be linked. On the other hand, a number of EPA reviewers pointed out that some of the measured chemicals would be unlikely to relate to an incinerator, and that the Navy should look to other sources in the valley.	cancer risk downwind from the SIC, air dispersion modeling results and the soil trend analysis, which together are indications that emissions from the SIC have a significant contribution to the air quality at NAF Atsugi. A subsection has been added to compare the SIC with incinerators in the U.S.
Page 4	Further mathematical approaches to apportioning risks to the SIC were also suggested in Matt Lorber's comments, which, rather than re-write, are included verbatim: "Two separate efforts are described in this document to address the	The SIC Contribution to Air Quality subsection has been expanded to include more information on the statistical exercises to correlate air concentrations and percent downwind, and to include a discussion on the estimation of background concentrations as well as uncertainties associated with the Upwind-Downwind analysis approach. Ambient air concentrations

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	contribution of the SIC to exposure and risk. The first includes two statistical exercises, which attempt to correlate air concentrations found in monitors and meteorological conditions, such as % downwind, during the monitoring events. These analyses are summarized on pages 16 and 17 of the Summary document and further discussions are found in the Risk Assessment document. One analysis suggests that the SIC is the primary source for hydrochloric acid, dioxin, lead, cadmium, arsenic, and PM ₁₀ . The second shows an association between the SIC and arsenic, benzene, cadmium, 2,3,7,8-TCDD TEQ, 1,2,-dichloropropane, lead, and PM ₁₀ , with possible associations indicated for 1,3-butadiene, acetaldehyde, acetophenone, chloromethane, and dieldren. The second effort is a comparison of risks based on a "downwind" site and an "upwind" site. While individuals don't live at these sites, the air concentrations at the sites are significantly different, and it is justifiably concluded (given % downwind data) that these differences are due to the presence of the SIC. My comments on these approaches are: a) On page 38 of the Summary document, it is noted that, "The only difference between the assumptions used to calculate the risks at both sites was the exposure point concentrations." What was the concentration used - the overall average during the 14-month sampling period, concentration corresponding to an 80% downwind condition or otherwise at that monitor, another concentration? This	used to calculate the health risks upwind (Golf Course) and downwind (GEMB) from the SIC were average and RME air concentrations on specific days when concurrently the GEMB was greater than 80% of the time downwind and the Golf Course was less than 4% of the time upwind of the SIC (about 8 days).
	section should specify what air concentration was used. I do see that there is a "Note" on the bottom of Table 3-3, page 40, that provides this information. There are also discussions in the Risk Assessment [Pioneer Assessment] document, which describe the strategy in more detail. This information should be included and expanded upon in the text of the Summary document.	

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Page 4	b) On page 38 of the Summary document, it is noted that the HI increased most for acrolein, from 15.6 at the upwind location to 63.1 at the downwind location. However, acrolein was not identified in either statistical test as having come from the SIC. Why is this? Is there another source for acrolein at the GEMB? This should be clarified. It is discussed on p. 64-65 of the Summary document. This text should be brought forward.	Three statistical analysis were conducted to determine the chemical contribution of the SIC to overall air quality: (1) standard correlation and linear regression analysis, (2) mixed model analysis and (3) non-parametric analysis. These methods used meteorological data and concentrations measured for COCs at NAF Atsugi (e.g., the cancer and non-cancer risk drivers) to determine if the COCs were associated with SIC operations. Acrolein showed a significant positive correlation at the GEMB site based on the log of the concentration scale, one of the criteria of the mixed model analysis. This was similar to the chemicals that were strongly associated with the SIC. However, the correlation for SIC-on days was virtually identical to SIC-off days. In addition, unlike the chemicals strongly associated with the SIC there was no correlation at the GEMB site based on the concentration scale (another criterion). At three other sites the correlation was greater for SIC off days. Even though the monitoring program was an extensive effort, we do not have as many SIC-off days (13) as we do SIC-on (60), which limits the power of the comparative analysis. It is expected that the SIC is a major contributor to acrolein concentrations; however, the data suggests that there are other sources of acrolein in the area. Automobiles are known for contributing acrolein to the environment. This information has been clarified and brought forward in Section 2.3 SIC Contribution to the Air Quality in the revised NEHC report.
Page 4	c) For this downwind/upwind assessment, the cancer risk at the GEMB (downwind) is driven by dioxin, which explained 45% of the risk. Also, the disparity in risk between the GEMB and golf course (upwind) is driven by dioxin. In the overall risk assessment, as noted above, dioxin makes up only a small part of the overall risk - about 15% of the cancer risk. This obviously makes this exercise less relevant to the question it tries to answer - what is the proportion of the total risk for living at NAF Atsugi that can be attributed to the SIC.	This upwind and downwind risk analysis approach was used for days when the prevailing wind toward the GEMB, the site having the greatest apparent impact from the SIC, was nearly 100% and the wind at an upwind site, the golf course, was approximately 0%, on the same day. In section 4.3 of the Summary report we did state: "The results of that method indicate non-cancer risks downwind of the GEMB are approximately 2 to 4 times higher than risks upwind at the golf course on the same day. The cancer risk is 50% higher at the GEMB than the risk at the golf course." As one of the reviewers suggested, we agree that if we had applied this analysis also to

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	When looking at Table 3-3, page 40 of the Summary document, it becomes clear that the overall carcinogenic exposure is about four times higher at the GEMB as compared to the golf course. If one divides the cancer risk in the shaded area, described as "Potential Incremental Risk Attributable to Emissions from the SIC", with risks at the GEMB "downwind" site, the math suggests that 72% of the total carcinogenic risk calculated for the GEMB is due to the SIC. Unfortunately, because dioxin is driving this result, it cannot be used as a general conclusion for the proportion of overall risk for living at NAF Atsugi that can be attributed to the SIC. I think this risk assessment very importantly needs to be able to derive a statement like this: "It is estimated that about ????% of the overall cancer risk for living at NAF Atsugi can be attributed to the SIC" and all efforts should be made to figure out if such a statement can be developed. Obviously, the Navy spent a lot of time thinking about this issue and tried different things. The various approaches are summarized on p.82-83 of the Risk Assessment document. Although its not stated, it is possible that the Navy tried to do something like the downwind/upwind analysis for the Areas of Concern (AOCs) instead of locations not associated with living and schooling (i.e., the GEMB and the golf course). Given the utmost importance of trying to cull out the effects of the SIC from the background effects at the AOCs, I think the Navy should go further than they have. Perhaps they should display results from the several efforts they tried. If an overall qualitative result can be culled from the various efforts, such as, "between 40-70% of the overall, upper bound lifetime cancer risk increment from being at NAF Atsugi appears to be attributed to the SIC", then this might be very helpful for decision makers.	other sites besides the Golf Course and the GEMB we may have been able to arrive to an overall qualitative result regarding the SIC contribution. The analysis indeed provides a good prediction of the contribution on those days, but there were only few days in which these conditions were actually met at these sites. We searched the database for the same type of conditions for the other sites involving areas of concern and found even less instances where these conditions were met simultaneously within the 14 months monitoring period. Without extending the sampling period to collect this type of information for additional sites, the analysis itself would have low power. Since dioxin is strongly associated with the SIC, the percent of cancer risk contribution from dioxin is noticeably the highest downwind (45%). However, on this particular upwind/downwind analysis only 6 out of 15 chemicals showed an increase in downwind cancer risk and 4 out of 11 showed an increase in downwind non-cancer effects. From day to day depending on the feedstock there may be different chemicals being emitted, or the same chemicals at different concentrations which makes it difficult to actually assign a percent contribution from the incinerator on an annual basis. All we can say is that there is definitely a contribution to the risk from the SIC, but we cannot say how much. On an annual average basis, it is difficult to separate background from the SIC. To more clearly focus conclusions on the contribution of the SIC, we added a subsection in the Conclusions section which summarizes our discussion on the SIC Risk Contribution.

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	I actually did some calculations on my own from an early set of air concentration data for dioxins. I looked at air concentrations when the wind was blowing from the North to the South (i.e., away from the base in relation to the SIC rather than toward the base, akin to the 4% condition above) to see whether that might provide a reasonable estimate of the "background" air concentration. I found dioxin air concentrations to be about 0.45 pg TEQ/m³ for this North-to-South "background" condition. The overall average (calculated for all sampling dates, all wind conditions) for all air monitors for dioxin was 1.57 pg TEQ/m³. Therefore, perhaps, an overall NAF Atsugi average air concentration for dioxin attributed to the SIC is 1.12 pg TEQ/m³ (1.57 - 0.45). There are two problems I see with this simple procedure: 1) by not considering a background condition when the wind is blowing from the South to the North may neglect southern sources other than the SIC such that the "true" background dioxin concentration is higher than 0.45 pg TEQ/m³, or there may be no southern sources other than the SIC such that a more appropriate background concentration could be much lower than 0.45, and 2) an "upwind" condition could simply represent a recycling dioxins that were originally from the SIC anyway. It may be impossible to get a true "background". Another approach might be to monitor at a location several miles away, not near obvious sources. Other air concentration data on dioxins from Japan has similarly shown a background at around 0.40 pg TEQ/m³. The bottom line is, the capability to make a statement such as the one italicized above is really needed for this risk assessment, if there is some way to do it. I'm sure the Navy realizes this and struggled with this question much longer than I have."	
	Similar comments from other reviewers were evident on the value of	

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	the upwind/downwind comparison, such as that provided in Section 5.4 (p. 82) of the Pioneer Assessment, and on the need to more clearly focus assessment conclusions on the contribution of the SIC. The Pioneer Assessment conclusions in 7.1 (p. 91) were generally considered reasonable and succinct on this point.	
Page 6	Additional background information on the site and health complaints at NAF Atsugi: The Pioneer Assessment Introduction (and in its abridged form in the HHRA Summary) needs to include a brief history of the SIC and NAF relationship. The actual history of the SIC and the evolution of concerns regarding plant emissions would set the stage and provide a rationale for the assessment. Additional information on records of complaints or health impacts at the base would provide the reader with a real world perspective on the problem and an indication of any nuisance, discomfort or frank ill effects that had been reported. This latter point highlights the apparent absence in the current NAF Atsugi reports of any "real" health data on the personnel and families since the plant opened in 1985. Although the population is transient and small by epidemiological standards, there must be quite good medical history data and clinic/hospitalization information (emergency visits - like asthma, admissions, etc.) that could be informative as to the nature of the noncancer impacts.	The Introduction section of the revised NEHC report now includes more information on the history and operation of the SIC and summarizes information on health complaints at the base. While the Navy conducted the comprehensive environmental sampling study at NAF Atsugi, two health studies were conducted to identify certain acute health conditions that either could be associated with exposure to poor air quality or were health conditions that concerned the NAF Atsugi community. One of the studies, the Children's Respiratory Health Effects Study, compares peak respiratory flow between children at Atsugi and Yokosuka. The second, the Pregnancy Outcome Study, compares spontaneous abortion rates between Atsugi residents and residents of other bases in Japan. Additional surveillance on air pollution related morbidity, compares rates of skin conditions and respiratory symptoms seen at the NAF Atsugi Branch Medical Clinic and Naval Hospital Yokosuka. The first two studies presented in separate reports are included, as Appendices D and E, in the revised NEHC report. Naval Base Yokosuka served as the control location for the studies for two specific reasons. First, Yokosuka, which is approximately 25 kilometers from Atsugi, is also located on Japan's Kanto Plain. Its population, climate, and vegetation are similar to that at Atsugi. In addition, other than the highly visible point source of pollution at Atsugi i.e., the Shinkampo Incinerator Complex, sources of air quality degradation are similar. Secondly, Yokosuka is the site of the Navy's primary medical treatment

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Page	EPA Comment	facility in Japan, Naval Hospital Yokosuka, which provides access to several healthcare databases. The study on Respiratory Effects in Children had two primary goals: 1. Identify differences in respiratory symptoms and lung function between children who live or go to school at NAF Atsugi and similar children at Yokosuka. 2. Determine if there were more respiratory symptoms in children who live or go to school at NAF Atsugi on days when they are exposed to higher levels of pollutants from the Shinkampo Incinerator during the four week study period (7 May-5 June 1998). The study focused on children since their health is a major concern of the NAF Atsugi residents. Children's lungs also tend to be more sensitive to the effects of air pollution. Fifth and sixth grade students at Atsugi and Yokosuka DOD Schools participated. One hundred twenty-seven (127)
		Yokosuka DOD Schools participated. One hundred twenty-seven (127) students volunteered for the study. Eighty (80) of the students lived on base at NAF Atsugi, 17 lived off base at NAF Atsugi and 30 lived at Yokosuka. The children's lung function was tested each school day during lunchtime. Children recorded the number of hours spent outdoors as well as respiratory and/or air quality related symptoms such as, trouble breathing, coughing during the day or night, feeling bad, runny nose, cold, headache, and irritated eyes. A daily symptom score was given to each child based on the information recorded. Data from ambient air monitoring at Shirley Lanham School was also collected for PM10, nitrogen dioxide and sulfur dioxide, known to cause respiratory effects. Wind direction and wind speed were also recorded, in an attempt to associate health effects with environmental pollution conditions. The primary findings of this study were:

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		 There were no differences in the respiratory health of children living on or off base at NAF Atsugi and those at Yokosuka. Children living on base at Atsugi reported more runny noses than the Yokosuka children did. All other reports of symptoms were similar. There was no difference in the reported number of colds between the Atsugi on base and the Yokosuka groups. Children living off base at Atsugi did report more colds. Most of the children in the study group had lung function better than that of the general population in the United States. The wind was blowing toward the school for only a short period of time during the four-week study period. As a result, no clear relationship between wind direction and the levels of gases and dust particles could be identified.
		The study on Pregnancy Outcomes was conducted because many residents expressed concerns during the November 1997 NAF Atsugi Public Meeting about the health effects the Shinkampo Incinerator may be having on their families regarding miscarriages. Therefore the study was designed to describe the rate of miscarriage, at NAF Atsugi and other naval facilities in Japan. Information for the study was gathered by looking at hospital and clinic records for past pregnancies. This was a retrospective study where only documented miscarriages versus live births were considered. The study population consisted of Navy personnel or their dependents who were pregnant at some point between June 1995 and May 1998 and lived on or near NAF Atsugi or other naval facilities in Japan serviced by Naval Hospital Yokosuka (NHY). Information used to calculate the miscarriage rates came from three different sources, Delivery Logs and Pathology records at NHY and the Prenatal Log at the Atsugi Branch Medical Clinic

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		Data collection took place during the summer of 1998. It included the number of live births and the number of miscarriages. The total number of pregnancies with known outcomes during the study period was 1862. For the purposes of this study, a miscarriage was defined as an unintentional pregnancy loss at up to the 28 th week of pregnancy. Multiple births were excluded from the analysis. The miscarriage rate was defined as the number of miscarriages divided by the total number of pregnancies examined (the number of babies born plus the number of miscarriages).
		The findings of the study were: 1. The overall miscarriage rate for patients with known pregnancies from Atsugi, Yokosuka, Iwakuni and Sasebo between June 1995 and May 1998 was 7.1%. This rate was determined by review of the delivery log and pathology records at NHY. When the Atsugi patients are subtracted, the miscarriage rate for the other areas is 7.8%. 2. Review of the NAF Atsugi Branch Clinic prenatal log, during the same period, indicates a miscarriage rate at NAF Atsugi, of 8.8%. However, the data used in this study came from different sources and contain some different information. Therefore, the miscarriage rate at NAF Atsugi cannot be directly compared to that of the other naval facilities that were part of this study population. 3. The NHY and NAF Atsugi miscarriage rates during the study period were both lower than the documented rate of miscarriage for women in the United States, who know they are pregnant, which is between 10% - 15%. 4. This study was conducted with the limited information that was available in various records. The results suggested that the risk of miscarriage at NAF Atsugi and other naval facilities within Japan are at the low end of the expected risk range described for the population of the United States.
		The ADS is a medical data management information system. The ADS records and classifies all outpatient visits, including follow-up visits, to

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		Navy medical treatment facilities by the International Classification of Disease (ICD-9) coding system. ICD-9 codes exist for all possible diagnoses made in clinical medicine. NEHC examined data on 39 diagnoses potentially related to air pollution (i.e., asthma, upper respiratory illness (URI), conjunctivitis, etc.) during the study. The population categories used in the study include "Adult Active Duty" and "Adult Civilian" (above eighteen (18) years old) and "Children" (below eighteen (18) years). The continued surveillance of air pollution related morbidity indicated the following: 1. There were no significant differences in air quality related morbidity between the adult populations at Atsugi and Yokosuka during the study period., There were no significant differences in air quality related morbidity between the Child (below 18 years of age) populations at Atsugi and Yokosuka during the study period. 2. There was a peak period of respiratory disease complaints at Atsugi from June —August 1998. This is an artifact of the comprehensive risk communication and health consultation program that was at its height during that period. 3. There was a peak period of respiratory disease complaints at Yokosuka beginning in November 1998 and persisting through January 1999. This represents an outbreak of Japan Type A Influenza during that period. This study, Prospective Analysis of Specific Respiratory Diagnosis Between Atsugi and Yokosuka, is still in progress.
Page 6	Comparative assessment between NAF Atsugi and US environmental levels: One of the most interesting and informative aspects of the NAF Atsugi assessments was the comparison between the ambient and indoor levels measured at the base and those measured in US	We searched the scientific literature for information on ambient and indoor levels measured at other cities in Japan. We found a few references on suspended particulate matter, NO2, SO2, metals and some VOCs. However, since the data collection and analysis methodology cited in these

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	cities. This provides a very valuable reality check on what are otherwise somewhat amorphous exposure numbers. EPA suggests that the Navy further develop these comparisons by including data, where available, from Tokyo and Yokohama. These comparisons could be used to further refine the conclusions regarding health risks at NAF Atsugi, as discussed above. For instance, the comparison of criteria air pollutants indicated that, although NAF Atsugi has elevated levels of ozone, NO ₂ , PM ₁₀ and PM _{2.5} (see discussion later on PM _{2.5}) compared to the US NAAQS, only the PM ₁₀ level is higher (by quite some margin) than that experienced in comparable US cities.	references were different from those used at NAF Atsugi, or were not well described, we were not able to develop useful and meaningful comparisons to include in the revised NEHC report.
Page 6	As mentioned above, the dioxin levels in air and soil were also substantially greater than US levels. Both PM ₁₀ and dioxin are also statistically associated with the SIC (as were Pb, As, and Cd). These findings are reasonably clear evidence that the SIC is contaminating the NAF Atsugi base, as they are both statistically based and logical given our knowledge of incinerator emissions (and photographic evidence!). With this foundation, the association of other pollutants to the SIC can be developed based on general <i>a priori</i> knowledge of incinerator emissions, other data collected at NAF Atsugi, and ground-truthed to background levels in the Tokyo/Yokohama region.	Although three statistical analyses were conducted to determine chemicals associated with the SIC, the ability to statistically associate all of the specific chemicals, known to be related to incineration, with SIC operations is limited. Factors such as variable feedstock, variable SIC operating conditions, and different meteorological conditions may have prevented the statistical methods from identifying a greater number of pollutants associated with the SIC. However, the statistical analysis conducted by Radian identified six chemicals likely related to SIC operations. These chemicals were hydrochloric acid, 2,3,7,8 TCDD, lead, cadmium, arsenic, and PM ₁₀ . The statistical analyses performed by RTI on the top thirty-two chemicals contributing to the risk in the risk assessment indicated that arsenic, benzene, cadmium 2, 3, 7, 8 TCDD TEQ, 1,2-dichloropropane, lead and PM ₁₀ showed an association with the SIC. Other risk drivers such as 1,3-butadiene, acetaldehyde, acetophenone, chloromethane, and dieldrin showed a possible association with the SIC.
Page 6	Acrolein and Acetaldehyde and the SIC: One problematic issue raised by such a comparison to background levels is with acrolein, the principal chemical contributing to the non-cancer risks. Acrolein was	Three statistical analysis were conducted to determine the chemical contribution of the SIC to overall air quality: (1) standard correlation and linear regression analysis, (2) mixed model analysis and (3) non-parametric

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	not associated statistically with the SIC, was associated with southerly winds coming from the valley, contributed the majority of the pulmonary non-cancer hazard quotient, and yet is reported in the HHRA Summary (p. 77) as being far below comparable levels in the United States. What does this mean? Either the US population is generally exceeding a pulmonary hazard quotient due to acrolein, there is another source of acrolein in the valley, or there is an error somewhere. Any assessment of acrolein will need to clarify these issues, preferably using other data on incinerators to determine if acrolein is usually associated with incinerator emissions (as some EPA staff note that it is, along with acetaldehyde and 1,3,-butadiene¹), and comparison to background data on Tokyo air levels to determine if this pollutant bears any relationship to the SIC, to the industrial complex nearby, or to Tokyo/Yokohama levels in general.	analysis. These methods used meteorological data and concentrations measured for COCs at NAF Atsugi (e.g., the cancer and non-cancer risk drivers) to determine if the COCs were associated with SIC operations. The correlation and regression approach, analyzed one site and the SIC's condition at a time, basing examination of one site's data on the results of statistical tests from another site. The mixed model analysis, which used a more consolidated approach, dealt with all sites simultaneously and attempted to adjust for day-to-day effects resulting from daily variations in SIC feedstock and other sources' variations. The non-parametric analysis, which utilized virtually all the data, was insensitive to outliers, did not depend on the measurement scales chosen for concentration data and incorporated an adjustment for day-to-day differences. The designations "strongly associated" and "with less confidence" are categories formed by simultaneously considering the results of the analyses and evaluating the degree of evidence of association, based on six statistical criteria that considers the last two methods and uses a weight-of-evidence approach, regarding positive correlations. "No association", means that the chemicals either failed all six criteria, or met only one or were assigned this category for other reasons. "Strongly associated" means that at least 5 of the six criteria were satisfied. "With less confidence" means a possible association indicating that although significant positive correlations were found by the parametric analysis, they were not supported by the mixed model approach. Acrolein was not listed as associated with SIC although this

¹Please note that, as with dioxin, some IRIS assessments are under review, such as for 1,3-butadiene where the inhalation unit risk factor is expected to change by at least one order of magnitude, possibly to the range of 1.4E-5 to 2.1E-6. This change could substantially reduce risk estimates.

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		compound displayed the most significant downwind increase. Acrolein showed a significant positive correlation at the GEMB site based on the log of the concentration scale, one of the criteria of the mixed model analysis. This was similar to the chemicals that were strongly associated with the SIC. However, the correlation for SIC-on days was virtually identical to SIC-off days. In addition, unlike the chemicals strongly associated wit the SIC there was no correlation at the GEMB site based on the concentration scale (another criterion). At three other sites the correlation was greater for SIC-off days. Even though the monitoring program was an extensive effort, we do not have as many SIC-off days (13) as we do SIC-on (60), which limits the power of the comparative analysis. It is expected that the SIC is a major contributor to acrolein concentrations; however, the data suggests that there may be other sources of acrolein in the area. Automobiles are known for contributing acrolein to the environment.
Page 7	It is also recommended for principal contributing chemicals that the actual primary data used to derive the RfC be examined, and that the highest measured values from the plume be compared with the toxicological data to determine if acute effects could be occurring. For acrolein, for instance, the RfC was developed from a LOAEL for the critical effect of squamous metaplasia and neutrophil infiltration of the nasal epithelium in rats, leading to a human equivalent concentration of 0.02 mg/m ³ and an RfC of 2 x 10 ⁻⁵ mg/m ³ . The IRIS file also indicates that, in another study, eye irritation in humans was	The actual primary data used to derive the RfC has been examined, and the highest measured 24-hr concentrations detected at NAF Atsugi compared with the toxicological data to determine if acute effects could be occurring. This comparison has been made in the revised NEHC Summary report.

²Kutzman, R.S. (1981) A subchronic inhalation study of Fischer 344 rats exposed to 0, 0.4, 1.4, or 4.0 ppm acrolein. Brookhaven National Laboratory, Upton, NY. National Toxicology Program: Interagency Agreement No. 222-Y01- ES-9-0043.

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	observed during a 40 minute exposure to 0.17 ppm, and a NOAEL for this effect was not established. ³ For comparative purposes, the exposure point concentration for acrolein at the day care center was 0.00036 mg/m ³ , with a maximum concentration of 0.001 mg/m ³	
Page 7	Note also needs to be taken that another major contributor to the NAF Atsugi non-cancer risks was acetaldehyde, which was measured at levels comparable to similar US cities. If acetaldehyde is a substantial contributor to non-cancer hazard indices at NAF Atsugi, then it also would contribute to similar risks in US cities and Tokyo, and would be difficult to localize to the SIC. In this event, the comparable or lower ambient pollutant levels for these two chemicals (acrolein, acetaldehyde) compared to US cities makes it more difficult to support the case that living at NAF Atsugi is any worse than a similar assignment in the United States. Such a conclusion further focuses concern on dioxins and PM ₁₀ , where the elevations and links to the SIC are clearer, tempered by the recognition that a reductionist, chemical-by-chemical approach to breathing in an incinerator plume may not adequately convey the true risk of these mixed exposures.	The statistical analyses performed by the Research Triangle Institute on the top thirty-two chemicals contributing to the risk in the risk assessment did indicate that acetaldehyde showed a possible association with the SIC. Although acetaldehyde levels at NAF Atsugi are comparable to ambient levels in U.S. cities and Appendix A shows that the maximum acetaldehyde concentration observed at NAF Atsugi was 0.28 mg/m³, (well below the levels that could produce acute health effects) acetaldehyde is a major cancer and non-cancer risk driver at NAF Atsugi. Despite the fact that acrolein was the only chemical found to exceed established acute health effect level at NAF Atsugi, we acknowledge that exposures are not limited to a single chemical, but a mixture of chemicals. Since toxicological and epidemiological studies that form the basis of the toxicity values are based on exposure to a single chemical, research on the effects of exposure to a mixture of chemicals is not available; therefore, when two or more chemicals act on the same organ system, their combined effect should be considered as additive, unless there is evidence to the contrary. Our intent in providing a discussion on acrolein and acetaldehyde and other chemicals individually was to identify potential health effects, especially respiratory effects noted in anecdotal complaints.
Page 8	Elevated indoor levels versus outdoor do not exonerate the SIC: The NAF summary notes that, for the chemicals linked statistically to the	By stating that, for the chemicals linked statistically to the SIC, only for dioxin, lead and cadmium are the outdoor levels greater than indoor, we did

³Webber-Tschopp A, Fischer T, Gierer R et al. (1977) Experimental irritating effects of acrolein on man. Int Arch Occup Environ Health (German) cited in ATSDR (1990) Toxicological Profile for Acrolein.

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	SIC, only for dioxin, lead and cadmium are the outdoor levels greater than indoor. The HHRA Summary interprets this to indicate that other internal sources of arsenic, particulate matter and other pollutants are responsible for the elevations found indoor. Although alternative indoor sources may be part of the problem, it is also possible that the accumulation of indoor dust, originating from the outside air, is a contributing factor. This should be made clear in the text, and the indoor elevations should not be over-interpreted as exonerating the SIC, especially if the constituents are clearly those persistent chemicals and metals that are often linked to incineration.	not mean to imply that therefore the sources of arsenic, particulate matter and other pollutants were internal. The text will be clarified by editing the last paragraph in the subsection on Indoor Air as follows: "The higher concentrations of metals found in indoor air at NAF Atsugi (especially cadmium, arsenic and lead which are strongly associated with the SIC) could be due to SIC particulate emissions deposited in soil and tracked indoors via shoes, as well as accumulation of indoor dust, originating from the SIC emissions transported indoors via open windows and doors."
Page 8	HHRA Summary Lacking Exposure Scenario Information: One of the fundamental bases of reporting a risk assessment is to clearly step through the "person-related" scenarios that are being evaluated, whether average exposures or reasonable maximal estimates. These scenarios are based on humans and their relationships to locations, not on the locations, <i>per se</i> . The HHRA Summary, however, appears to convey risk information based on the golf course or the GEMB, for instance, which is initially confusing as it implies that these physical sites were at risk from SIC exposures. Recognizing that what was probably meant was exposure to individuals during their presence at these sites, it was then very difficult to determine the scenarios under consideration and the time periods of exposure for determining the risk estimates. The reader's confusion is compounded by HHRA Summary Table 4-3 (p. 62), which posits 24-hour exposure to infants on the golf course or at the ground electronics maintenance building. Fortunately, the Pioneer Assessment did provide tabular and brief written information on the exposure scenarios, and the reader is able to piece together what the HHRA Summary was attempting. EPA's recommendation is that, from the outset, the HHRA Summary describe the relevant exposure scenarios under consideration, possibly	These scenarios are summarized in Table 3-7 Summary of Exposed Populations at NAF Atsugi in Section 3 of the HHRA. Reference to Table 3-7 and the rationale for choosing the receptors, pathways and routes of exposure listed on the table have been added to the text.

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	by incorporating and expanding on text from the Pioneer Assessment.	
	Specific Comments: HHRA Summary	
Page 8	Public Health Summary, page 1 (you may wish to label the pages) Clarity on the objectives and purpose of the risk assessment: The first sentence is slightly misleading and establishes an expectation that is not fully met by the risk assessment. The sentence states that, "The Navy Environmental Health Center (NEHC) directed a comprehensive health risk assessment at Naval Air Facility (NAF) Atsugi, Japan to assess potential health effects associated with exposure to the neighboring Shinkampo Incineration Complex (SIC)." Rather, the primary goal of the health assessment appears to be to evaluate the potential health effects from residing at NAF Atsugi. This is better stated on HHRA Summary p. 41, "Given that the primary objective of the Health Risk Assessment (HRA) is to estimate the potential human health risks of individuals living and working at NAF Atsugi". A second and equally important goal was to evaluate how the SIC could contribute to that total risk. Thus, the first sentence here sets an expectation that probably wasn't met, noting the difficulties discussed above in evaluating impacts specific to the SIC. It would be better to rephrase the first sentence to reflect the dual purpose noted further down on the first page.	The objectives have been clarified throughout the revised NEHC Summary report.
Page 9	Public Health Summary, page 1, line 7: Suggest deleting text "whom supported" and substituting ", both of which supported".	The Public Health Summary has been extensively modified in the revised NEHC Summary report.
Page 9	Public Health Summary, page 1, full para 2: As no risk assessment is "accurate," EPA suggests caveating this word, such as " as accurately as possible."	The Public Health Summary has been extensively modified in the revised NEHC Summary report.
Page 9	Public Health Summary, page 2, overinterpretation of risk assessment	The text has been modified as suggested throughout the revised NEHC

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	results: On many occasions throughout the HHRA Summary (and to	Summary report.
	a lesser extent in the Pioneer Assessment), the risk assessment over-	
	interprets the 10 ⁻⁴ value as a benchmark or bright line. This is not the	
	case, and all of these numerical upper bound estimates must be	
	interpreted in light of risk management considerations, etc. The	
	importance of not making bright lines cannot be emphasized enough.	
	For instance, on page 2 of the Public Health Summary, the middle	
	paragraph begins, "The USEPA cancer risk benchmark" There is	
	no such thing, and all such references need to be re-phrased. The	
	Pioneer Assessment (section 5.0, page 66) has a reasonable summary	
	of the emphasis that should be accorded these values in risk	
	assessment and management decisions (see later comment).	
	Essentially, depending on the relevant legislative background and	
	interpretation of risks, upper bound lifetime cancer risk estimates	
	above 10 ⁻⁴ to 10 ⁻⁶ have generally been considered to warrant increased	
	regulatory consideration and possible intervention. Similarly, a	
	hazard index of 1 is also not an Agency benchmark. There is enough	
	uncertainty and variability in the procedures such that the strength of	
	conclusions in this paragraph is not warranted, i.e., that the $1.1 * 10^{-4}$	
	risk is <i>slightly</i> higher than this benchmark, and that the benchmark is	
	reached in a finite number of months in children and resident adults.	
	EPA suggests rewriting this paragraph in the following manner:	
	"When the results of a human health cancer risk assessment fall in the	
	range of 10 ⁻⁶ to 10 ⁻⁴ (which equals 1 in 1,000,000 and 1 in 10,000	
	additional cancer cases per lifetime, respectively), the US EPA	
	typically considers additional activities, including regulations, to	
	mitigate the risk, particularly if the results are close to or greater than	
	10 ⁻⁴ . The results of this risk assessment suggest that a child's	
	exposure to contaminants from air and soil during a 3-year tour of	
	duty could potentially result in an upper bound lifetime estimate of	
	risk at the 10 ⁻⁴ level, but that adults would not likely reach this level,	

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	even with two tours of duty (6 years of exposure)." EPA suggests that the Navy adopt this type of language in this paragraph and throughout the risk assessment document. EPA also strongly recommends deleting all mention of a specific number of months until a benchmark is reached. This issue occurs again on page 3 of the public health statement. The first difficulty with calculating 32 months, for instance, is that it creates an expectation of accuracy that is totally unfounded. Second, the calculation implies that this part of a child's or adult's life can be viewed in isolation from the rest of their life, which is not the case. The incremental risks from residing at NAF Atsugi must be added to the rest of one's life experience, and one cannot divorce a risk estimate of ~10 ⁻⁴ from other risk experiences occurring at other times and at other places. EPA's suggestion is that 32 months is so close to 36 months that it would be illogical to separate the difference.	
Page 10	Public Health Summary, page 2, second bullet: Add " as much as" before the 3.7 additional cancer risks. As the bullet before implies correctly, the risk may be as great as this, but is likely to be less, may be zero, and is certainly not a single point value.	The text has been modified as suggested throughout the revised NEHC Summary report.
Page 10	Public Health Summary, page 2, third bullet: As noted above under the discussion of acrolein, the reliance on the hazard index "benchmark" both overstates the importance of the "benchmark" bright line and obscures some of the concerns about which chemicals contribute to this level and how this differs from levels experienced in the United States.	The text has been modified to specify that an HI of 1 assume that there is a level of exposure below which it is unlikely that even sensitive persons will experience adverse health effects. An HI of 1 should not be considered a bright line, which triggers remedial action, since this level of exposure, called Reference Dose (RfD), includes an uncertainty factor that could be as high as 3,000. An HI greater than 1 indicates some degree of concern and the need for professional judgment to evaluate the concentrations and the potential non-cancer health effects related to the concentration of these

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		chemicals.
Page 10	Public Health Summary, page 2, fourth bullet: The "one air pollutant" is not stated and needs to be. It is probably acrolein. There is an extra comma after "respiratory effects," that should not be there.	The text has been edited as suggested.
Page 10	Public Health Summary, page 3, second line under Recommendations: The commas in this sentence seem problematic.	The Public Health Summary has been extensively modified in the revised NEHC Summary report. This sentence has been removed.
Page 10	Public Health Summary, page 3, "2. Source control." More information is needed on what kind of emission controls the SIC has in place already. Later in the document, a series on emission reduction devices are listed as in place at the SIC, including electrostatic precipitators, etc. The recommendations section of this report needs to explain why these current emission control devices are not adequate, and what additional devices are required to reach an adequate level of control, if that is indeed possible. Further, the assessment needs to highlight the eye-witness reports that the SIC bypasses the emission control devices, and to document how such bypasses are recognized by Navy personnel and how often they occur.	The Public Health Summary has been extensively modified in the revised NEHC Summary report. Recommendations have been deleted in the NEHC and Pioneer reports.
Page 10	Page iii, first line: NAAQS usually don't have an extra "s" added.	The text has been edited as suggested.

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Page 10	Page 1, Introduction: This introduction has a lot of grammatical rough spots, and should be tightened up considerably as it is the lead in to the report. e.g., commas and periods on line 2; ", as" needed to be inserted in para 2, line 4; media changed to medium on para 2, line 5; extra spaces on para 2, line 7.	The entire report has been edited.
Page 10	P.4, Shinkampo Incineration Complex: A number of reviewers inquired as to how long the incinerator has been there. This information (e.g., the SIC has been there since 1985) and the timing of Navy concerns and construction activities are outlined in the responses to Navy personnel questions (FAQs, 6/6/98; end HHRA Summary), and could be conveyed in more detail here. As mentioned earlier, this information would also be useful introductory sections of both NAF Atsugi documents.	More specific information on the SIC and its waste management operations have been included in the Introduction on the revised NEHC Summary report.
Page 10	Page 5, line 1: The report talks a lot about the constraints in performing a risk assessment on foreign territory. This is understandable regarding the inability to enforce Japanese laws and require inspections of the SIC facility. However, the constraints are harder to understand when looking at air monitoring sites. The assessment needs to more clearly explain why it was necessary for all monitoring sites to be on base, and why the Navy could not rent some space in an upwind location from the SIC. This may be a simple foreign military installation policy issue, but this needs to be stated.	It was necessary to locate air monitoring sites on base, because we could not guarantee the security of air monitoring stations off base. In addition, off base monitoring would greatly increase personnel resources requirements and political implications. These reasons have been added to the text in the revised NEHC report,
Page 11	Page 5, line 5: Delete "all the way", as this emotive form of statement is not consistent with a technical risk assessment report.	The entire sentence has been deleted.

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Page 11	Page 14 PM _{2.5} standard: The PM _{2.5} standard is under litigation, with Supreme Court review under consideration at the time of writing these comments. Thus, the U.S. Government's ability to enforce this standard at the present time is problematic and this needs to be noted in the report.	The text has been edited as suggested.
Page 11	Page 15, last paragraph on Risk Based Concentrations: This paragraph references and commences a strong reliance on the USEPA Region III Risk Based Concentrations (RBCs). The formulae, assumptions and caveats that go into the derivation of the RBCs needs to be more clearly outlined. Also, the Region III recommendations do not necessarily constitute US EPA policy. This limitation needs to be more clearly highlighted in the report, with reference to the Region III values being used only as a form of guidance.	Additional information on the assumptions and caveats that go into the derivation of the RBCs has been included in the report. The text has been edited as suggested.
Page 11	P. 16, Meteorological Monitoring: This paragraph states that other monitoring sites measured wind speed and direction but may not have been as robust as the criteria site. Nevertheless, given the importance of wind direction to the analysis, there should be some discussion of the wind data provided by these other sites. Micro-meteorological effects, including building impacts and terrain elevation gradients, can impact local flow and the wind roses from the other sites should be included to either verify the 10-meter tower data or provide a better picture of the local wind flow.	A discussion of the wind data provided by these other sites as compared to the 10-meter tower data has been included in the report.
Page 11	Page 18, table 2-2: The column under U.S. Data reports the number 44.2 ppm for carbon monoxide. What is U.S. Data in this context? Average levels? Standards? More importantly, why is the "U.S.	The column under U.S. Data has been deleted.

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	Data" higher for carbon monoxide than for any individual city, and higher than the NAAQS? Is there are typo here?	
Page 11	Page 27, first paragraph: This is not a new paragraph, but a continuation of the preceding.	Correction has been made in the text.
Page 11	Page 27, second paragraph: What is the foundation for the assumption that combustion products are found in particles greater than 5 μ m? I thought combustion products were in the fine fraction of PM ₁₀ , and that the more coarse fraction related to physical processes. Also, this sentence is missing a comma.	Since no combustion particles were found in the fine fraction (5 μm), it would be logical to try to identify them in a coarser fraction of PM10.
Page 11	Page 27, Further discussions and the use of air dispersion modeling: The discussion on this topic on p. 27 of the HHRA Summary is too brief. It should at least elaborate a bit on the modeling strategy - that the model back-calculated emission rates based on a calibration exercise where predictions of air concentrations were forced to match observations of air concentrations, as closely as possible. It is also unclear what role the dispersion modeling played in the risk assessment. The text indicates that the modeling was used to evaluate risk at areas without monitors, but it is unclear how this was done. The modeling could have been used to evaluate a worst-case risk scenario by using the maximum value predicted by the model, although admittedly this is complicated by the lack of real emissions data. Also, the modeling concentrations at the monitoring sites could have been compared with the ambient monitored values. If the comparison was favorable, it would have added credibility to using the model more extensively as a tool to evaluate risk and potential mitigation strategies. The fact that this modeling exercise generated emission rates also	Further discussions on the air dispersion modeling strategy and its results regarding predicted concentrations has been included in the revised report. The results were not used in the risk assessment, but rather to predict the average ground-level concentrations from the unit emission rate modeling of the six chemicals associated with the SIC as a contour plot to determine modeled impacts at specific NAF Atsugi locations. Comparison of concentrations of these chemicals with the EPA Region III RBCs was used to evaluate the relative long-term health risk impacts at locations across the NAF Atsugi Base. EPA's suggestion for bolstering Navy's argument that the SIC is contributing far more dioxin to the immediate environment than it is warranted based on its operating practice (i.e. amount burned), at least compared to US incinerators is very logical. However, it doesn't seriously consider the uncertainty with the resulting calculations and also how the information will be used (i.e., would we recommend that someone make a risk management decision based on the modeling data over the empirical data). It would be very difficult to accurately estimate the actual SIC emission concentrations and waste burning rate because of lack of stack

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	presents an opportunity for the Navy to make some important statements in support of a policy advocating emission reductions at SIC. In a previous comment sent by Matt Lorber to the Navy, he commented that: "Preliminary findings are as follows: the annual emissions of dioxins, as backcalculated from the air dispersion modeling exercise, are reasonably low and not out of the ordinary for US incinerators; however, the 30 tons per day waste burning rate is very low so that the emission factor - that is, the grams dioxin emitted per kg waste burned, is very high - much higher than any US category of incinerator." The Navy should pursue determining a dioxin emission factor for the SIC. This way they could bolster their argument that the SIC is contributing far more dioxin to the immediate environment than it is warranted based on its operating practice (i.e. amount burned), at least compared to US incinerators. EPA can supply the Navy with emission factors that have been determined for US incinerators for comparison purposes. If the Navy has a sense of the volume of air emitted, they could also estimate emission concentrations, which can be compared with EPA regulatory limits and to the limits adopted as Japanese standards.	emissions data; modeling uncertainty that include unknown SIC operation and waste composition, variable emission rates, establishment of background air concentrations, emissions from other SIC sources; and the accuracy of ISCST3 in simulating dispersion of SIC plumes. Associated magnitudes of error are likely quite high but are difficult to estimate without more detailed information concerning SIC operation. Therefore it would be also difficult to evaluate the worst case scenarios based on maximum concentration due to the lack of real emissions data. The uncertainty associated with the modeling inputs outweighs the benefits of performing more analysis. We could certainly calculate the amount of dioxin emitted in g/ton of throughput. This information could be compared with incinerators in the United States. However, there is a great deal of uncertainty in this calculation because we don't know: 1) Although they are permitted for 90 tons/day we don't know how much waste is actually burned per day because visual observations of waste truck loads indicate that higher throughputs. The average value could be anywhere along that spectrum. 2. The emission rate. Ours is based on a back calculation from the ambient air samples. This is highly uncertain.
Page 12	Page 29, soil trend analysis: The statement on p. 29 of the HHRA Summary document, "A definite footprint of dioxin deposition associated with air emissions from the SIC is evident in the way the congeners are distributed and their decreasing concentrations with increasing distance from the incinerator" is not quite true (or misleading). Actually, the congener distribution, or as we call it, the congener profile, is similar to the profile of dioxins in US background	Correction has been made in the text which will read: "A definite footprint of dioxin deposition associated with air emissions from the SIC is evident from high concentrations near the SIC with decreasing concentrations associated with increasing distance from the incinerator.

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	settings (with one exception, the soil sample most elevated to the east of the SIC and suspected of being caused by blowing ash or another source), and similar to the profile we saw on a study of the impacts from a US incinerator emitting large amounts of dioxin. The "footprint" really just applies to finding high concentrations near the incinerator, with decreasing concentrations associated with increasing distance from the SIC. There is no unique congener distribution associated with the soil concentrations at NAF Atsugi.	
Page 12	Page 29, end second paragraph: What is an SVOC? Semi volatile organic compound?	Correct.
Page 12	Page 35, end line 10: insert "of" between "most the".	This section of the NEHC report has been extensively revised and the sentence where correction is suggested has been deleted.
Page 13	Page 35, para 2, end first line: insert "and" between "years 6".	This section of the NEHC report has been extensively revised and the sentence where correction is suggested has been deleted.
Page 13	Page 35 - 38, Exposure Scenarios: As noted in the general comments above, there is absolutely no explanation of the exposure scenarios considered in the HHRA Summary text or in the tables. This is the fundamental basis of the risk assessment and is a major oversight. Included in this explanation needs to be information on how the average exposure is distinguished from the reasonable maximum exposure.	These scenarios are summarized in Table 3-1 Summary of Exposed Populations at NAF Atsugi in Section 3 of the HHRA. This section of the NEHC report has been extensively revised to include additional information on exposure scenarios. Reference to the table and the rationale for choosing the receptors, pathways and routes of exposure listed on the table has been added to the text.
Page 13	Review of the Pioneer Assessment indicates that the "residential exposures" are, in fact, all-encompassing exposures including 24 hours/day, 350 days/year. The air and soil concentrations are specific to those locations, but the concentrations used are really not that different from other nearby locations where exposures were	The independence of the various scenarios has been emphasized in the text of the revised NEHC report.

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	considered (school, daycare). The school exposures are 180 day/yr, 8 hr/day exposures, and the daycare are similarly 185 day/yr, 8 hr/day. One might initially think that to get a "total" cancer risk, one should add the risks for residential + school, for example. They might presume that a residential scenario only includes 16 hr/day. Adding residential plus school cancer risks for children is obviously not valid for this assessment. An appropriate way to explain the strategy behind the construction of the exposure scenarios is that they assumed: 1) for the residential scenarios, the contamination exists only at the home and not elsewhere, and the strategy is to be conservative with that environment (350 days/yr, 24 hrs/day), 2) for the school scenarios, the contamination exists only at the school and not elsewhere, and again the strategy is to be conservative for that environment (185 days/yr, 8 hr/day), and 3) for the worker or golfer scenarios, the exposure occurs only at the workplace (250 days/yr, 10 hrs/day) or on the golf course (37 days/yr, 5 hr/day) and not elsewhere. The discussions need to emphasize the independence of the various scenarios so that others are not tempted to add cancer risks, as several EPA reviewers were.	
Page 13	Page 37, Table 3-2: The #1 footnote is difficult to understand, and there is no footnote for #2.	Table 3-2 (Table 3-8 in the NEHC revised report) has been corrected. Footnote #1 has been assigned to the Hazard Index and Cancer Risk column headings only. Footnotes on items on the Scenario column have been deleted, including footnote #2.
Page 13	Page 38, non-carcinogenic hazard index: The HHRA Summary presents Hazard Indices, which are calculated as the sum of all Hazard Quotients calculated for all chemicals/pathways/target organs or effects. This is not fully appropriate, as hazard quotients can be summed for different chemicals and different pathways only if the	The text has been edited to reflect suggested corrections. Table 5-4, page 75, of the Pioneer Assessment which lists all the HIs organized by receptor and target organ/target effect has been added to the HHRA Summary report. The heading on the subsection Non-Carcinogenic Risks has been corrected to Non-Carcinogenic Effects.

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	target organ/target effect is the same. Most of the non-cancer hazard index, about 70%, is attributed to respiratory effects. Table 5-4, page 75, of the Pioneer Assessment lists all the HIs organized by receptor and target organ/target effect. To be correct, the HHRA Summary may wish to report only the HIs associated with respiratory effects, with text stating that several other HIs were developed for other effects such as neurotoxicity, liver, kidney, and other organs/effects, but that respiratory effects would comprise 70% of the total if all were added up. Note, too, that the hazard index is not a measure of risk, but a measure of a level below which no significant adverse effects are likely to occur. Risk, to the contrary, has a stochastic probability to it.	
Page 13	Page 40, table 3-3, 6 year old exposures: This table is quite confusing as it posits scenarios that just cannot be realistic. For instance, how can a child (0 - 6) undergoing a residential exposure scenario get exposed to SIC emissions at the Ground Electronics Maintenance Building, presumably a secure area for Navy personnel only. Another scenario appears to posit that these infants and children are now playing golf.	Table 3-3 presents a Comparison of Downwind versus Upwind Risks at NAF Atsugi. The purpose for this comparison was to estimate the potential impact of emissions from the SIC on the risk. We did not intend to present the risk for a child (0-6) at the golf course or at the GEMB, but to show the impact of the SIC on the risks, on the different scenarios.
Page 14	P. 46, over-interpretation issues: There are a number of smaller text issues on this page that require clarification. With regard to page 46, line 2, EPA does not use the term safety factors, but uses the term "uncertainty" factor. These uncertainty factors generally range from 10 - 1000, but may be 0 or may be up to 3000. In the second last paragraph, there are some caveats that should be inserted, such as "and the number could be as low as zero" at the end of the third sentence. The last line again overemphasizes the bright line nature of the "benchmark of 1 in 10,000."	The text on Section 4, Health Risk Evaluation of the NEHC report has been reorganized. Uncertainty in the risk assessment, including uncertainty in the toxicity assessment, is now discussed in more detail in Section 3, Human Health Risk Assessment Results where the text has been revised based on reviewers' comments.

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Page 14	Page 47, second paragraph: Change "children that reside" to "children who reside". Change vice to versus.	The text has been edited as suggested.
Page 14	Page 53, table 4.1, notes: The mention of different site specific scenarios for the AOCs just re-emphasizes the absolute need for more explanation of these scenarios.	These scenarios are summarized in Table 3-1 Summary of Exposed Populations at NAF Atsugi in Section 3 of the HHRA. This section of the NEHC report has been revised to include additional information on exposure scenarios. Reference to the table and the rationale for choosing the receptors, pathways and routes of exposure listed on the table has been added to the text.
Page 14	Page 53, text: The hazard index greater than 1 indicates an "increasing" risk for developing a non-cancer effect. "Increasing" needs to be inserted .	The text has been edited as suggested
Page 14	Page 54, para 1: The attempt to explain the Congressional Commission findings leaves the reader totally confused. Can this be re-written? Also, there really is no typical uncertainty factor in going from a NOAEL to an RfC or RfD, with the usual range going from about 30 to 1000 fold (mode of 100), but they are certainly not usually one thousand fold and are coming down quite markedly as the science improves. We would also voice a concern regarding the reported conclusion of the Commission report that it is OK to use a hazard index of 10 as a benchmark. The basis and context for this statement should be checked, noting that uncertainty factors are currently being peeled off as the science improves, and that the HI value of 1 is appearing, as it should, as based increasingly more on science than policy, albeit with a ways to go.	This paragraph was written to provide another perspective on the non-cancer risk evaluation. For clarification purposes the following has been replaced with the current paragraph: "The Presidential/Congressional Commission on Risk Management and Risk Assessment evaluated the EPA risk assessment approach for assessing hazardous air pollutant sources to implement section 112 of the Clean Air Act. Since the 1990 amendments do not set a threshold for considering health risks other than cancer, the Commission has set a HI threshold of 10 in a screening risk assessment for identifying high priority source categories when determining and managing risk. They chose a threshold index of 10, instead of 1 because there are few hazardous air pollutants with inhalation RfCs that are within a factor of 10 of their NOAELs. Typically, RFCs are one-thousandth of a NOAEL, so a hazard index of 10 in these cases would still leave a margin of exposure of 100."
Page 14	Page 55, last sentence. Change vice to versus.	The text has been edited as suggested.

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Page 14	Page 61, table 4-2: The title of this table needs to be changed to "Percentage Contribution by COCs to the Hazard Indices at Each Location."	The text has been edited as suggested.
Page 14	Page 62, table 4-3: The exposure scenario issue is further confused by this section – the first time that scenarios are mentioned in the report – which then posits 24-hours at each site.	These scenarios are summarized in Table 3-1 Summary of Exposed Populations at NAF Atsugi in Section 3 of the HHRA. Reference to the table and the rationale for choosing the receptors, pathways and routes of exposure listed on the table has been added to the text. Because there is a myriad of exposure scenarios not covered in this investigation, the purpose of Table 4-3 is to illustrate the plausible upper bound inhalation risks for individuals that routinely spend 24 hours a day at NAF Atsugi.
Page 14	P. 63 para 1 line 1: Change vice to versus	The text has been edited as suggested.
Page 14	P. 64 - first paragraph: This paragraph discusses an approach used to assess the SIC contribution to risk by comparing upwind and downwind monitoring sites. As noted under the general discussion above, EPA reviewers encourage the Navy to continue to pursue this strategy. The technique compared the downwind GEMB site on days when the wind was nearly 100% impacting the monitor and days when the upwind golf course site was impacted nearly 0% of the time. As the HHRA Summary states, this type of sustained wind flow over an entire day would not occur very often. Therefore, the number of days available for use in the analysis is limited. An alternative that may allow more days to be used, yet still provide meaningful information on the SIC contribution, would be to consider days where the GEMB site is impacted at least a significant part of the day (e.g., 50%). The prevailing wind flow is essentially bi-directional (i.e., 75% of the time either northerly or southerly, likely due to sea/land breeze interaction). The golf course site is directly east of the SIC	To be confident in the incremental risks that could potentially be attributed to emissions from the SIC the upwind-downwind analysis requires that these directly opposite conditions (>80 downwind and <4% upwind) be observed simultaneously. This simultaneous condition cannot occur when percentage downwind is much lower than 80% at the downwind site and much greater than 4% at the upwind site. The prevalent winds at NAF Atsugi are northerly or southerly, alternating during summer and winter seasons. The GEMB is the only site located directly north of the SIC with no obstructions, being the most impacted site. This facilitates the upwind-downwind analysis because they are located in the path of the most prevalent wind patterns. The analysis we conducted did include ambient air data using > 75% downwind condition at the downwind site which demonstrated the impact due to the SIC. Since the elementary school and the residential towers are not located in the path of the prevalent winds, the wind would still have to blow from the SIC toward the sites for which we have ambient air data (for example easterly or southerly).

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	stack and will be an "upwind site" most of the time. Therefore, even if the GEMB downwind site is only "downwind" for 50% of the time, the difference between the GEMB and the golf course daily concentrations could still be attributed to the SIC as long as the golf course site remained unimpacted. During wind flows from the north or east, both the GEMB and golf course site would be monitoring essentially the same background. Another consideration is to consider other sites as background depending on wind direction. For example, when the wind is from the south and switches to westerly during a 24-hour period, the school site may be a more appropriate background site. The modeling output in Figure 2-2 also suggests that, based on long-term impacts, the school site is the least impacted by SIC.	
Page 15	Page 65, line 5: The term "one and one-half orders of magnitude" is difficult to grasp. What is one and one-half orders of magnitude? 10E1.5 equals 31.6 times, or is this term meant to imply 50 or so?	An increase of one and a one half orders of magnitude means a 15-fold increase in cancer risk between the cancer risk values for the golf course and the GEMB, i.e., from 1.16E-05 to 1.62E-04.
Page 15	Page 66, acrolein exceeding acute health criterion: It is difficult to fully grasp the totality of breathing in an incinerator plume using a chemical-by-chemical approach that relies on reference concentrations and uncertainty factors. Regarding acute effects, the HHRA Summary indicates that only acrolein exceeds acute standards. A more complete approach would then analyze these data by researching the basis for the acrolein acute level and compare this level to that found at NAF Atsugi (see general points). An examination of reported adverse health effects (e.g., asthma, cough) by wind direction (+/- time lagging) might also be attemped as a way to more pragmatically look for acute impacts.	As indicated in a response to an earlier comment, in order to compare the highest levels of ambient air chemicals at NAF Atsugi with levels known to cause acute effects in humans, toxicological information was collected during a literature search and presented in Appendix A: "Comparison of Maximum Concentrations of Chemicals Detected at Atsugi with Acute Health Effect Levels." Appendix A presented not only ATSDR Maximum Risk Levels (MRLs) but all data, including IRIS database, that was found in the literature regarding air concentration levels for all chemicals detected and corresponding health effects. The actual primary data used to derive the RfC has been examined, and the highest measured 24-hr concentrations detected at NAF Atsugi compared with the toxicological data to determine if acute effects could be occurring. This comparison has been made in the revised NEHC Summary report. A review of Appendix A indicates that

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		acrolein was found to exceed any acute health concentration level. For acrolein, 77 of 216 air samples collected at various sampling locations exceeded the acute inhalation MRL of 0.00005 parts per million (ppm). This MRL is based on eye irritation. All samples exceed the intermediate MRL of 0.000009 ppm. The intermediate MRL is based on respiratory effects of acrolein exposure as seen in laboratory animals. In the 1990 Toxicological Profile for acrolein, ATSDR states that the only known effects of acrolein exposure in humans are general respiratory congestion and eye, nose, and throat irritation. While the Navy conducted the comprehensive environmental sampling study at NAF Atsugi, health studies were also conducted to identify certain acute health conditions that either could be associated with exposure to poor air quality or were health conditions that concerned the NAF Atsugi community. One of the studies, the Children's Respiratory Health Effects Study, compares peak respiratory flow between children at Atsugi and Yokosuka. Please refer to response to comment Additional background information on the site and health complaints at NAF Atsugi under General Comments.
Page 15	P. 67, second last line: The intermediate inhalation "what" for vinyl chloride?	The text has been edited to read "the intermediate inhalation MRL for vinyl chloride"
Page 15	P. 68, first full paragraph: EPA suggests that the term MRL requires clear definition, and that this be done on page 66, close to where the first use of the term takes place.	The text has been edited as suggested.
Page 15	P. 69, probability of exceeding the 10 μg/dL Pb level: Again, the tone of this section implies a bright line criterion for risk of lead poisoning, which is not the case. "Benchmark" needs to be deleted, especially when associated with the CDC figures. EPA also notes the benefits	The word "Benchmark" has been replaced with "the action level". In the Navy Pediatric Blood Lead Poisoning Prevention Program screening blood lead, screening of low risk infants may be suspended provided that some conditions are met. The first condition is that large numbers of percentages

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	and reassurance provided by actually measuring Pb levels in humans, a situation which could be repeated regarding the dioxin levels.	of children have been screened in the community and found not to have elevated blood lead levels. Routine testing of pediatric blood lead to date has not indicated elevated blood levels in the NAF Atsugi community.
Page 15	P. 70, first paragraph: The comment that Branch Medical Clinic Atsugi personnel would have evaluated this slight elevation is inadequate. Did they, and was there any potential relationship to the SIC?	The Department of Defense policy on the assessment of health risk from lead is that blood lead levels of 10-19 ug/dl require confirmatory blood lead determination within one month of the first result. Confirmed 10-19 ug/dl blood lead results require a reassessment of the risk factors for exposure, education concerning diet and personal hygiene. If levels persist the policy requires the initiation of individual case management, environmental investigation, and lead hazard abatement. Re-screening is also required every 3 months. Upon confirmatory blood lead determination the child with the previous slight blood level elevation was found to have a blood lead level of less than 10 ug/dl. This paragraph has been added to text to clarify the issue.
Page 16	P. 70, fifth bullet: Breathing is not a significant health problem it is a good thing. We think you mean breathing difficulties, or somesuch.	The sentence preceding the bullets has been edited as follows: "These particulates have been associated with significant health problems and resulting related impacts including:" The bullet "Breathing" will now state "Breathing difficulties."
Page 16	P. 71, last sentence: This sentence needs to be reworded because you cannot reduce mortality, because it is always 100% eventually. The mortality rate can be reduced, or other time-related metrics used.	The word "rate" has been added to the terms mortality and morbidity.
Page 16	P. 72, last sentence, p. 73 table 4.6: The quoted dioxin risk, as well as not being caveated adequately as an upper bound estimate, also needs to incorporate mention of the pending revisions to the dioxin reassessment.	The entire subsection on Dioxin has been revised and includes a reference on the dioxin reassessment. A caveat indicating that the estimate on the background lifetime cancer risk is an upper bound value will be added to the sentence along with a footnote on Table 4-6.

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Page 16	P. 73, second last paragraph, dioxin soil levels: The statement that soil levels at Atsugi are essentially the same as in the urban US is somewhat contrary to the data provided by the EPA dioxin exposure assessment group, which indicate that levels at NAF Atsugi are elevated. The discrepancy could be clarified by using actual numbers in the text.	The comparison was made between U.S. urban and rural soils, and the NAF Atsugi soil concentration. The NAF Atsugi soil concentration of 15 ppt TEQ to which individuals are likely to be exposed was calculated as the average concentration through the entire base. At the same time it is recognized that soil concentrations nearer the SIC, on the GEMB site and golf course where much less exposure is expected to occur, are significantly higher at greater than 100 ppt for some samples. This paragraph has been clarified by mentioning in the text the actual values used in the comparison and the higher soil concentrations found in individual samples near the SIC.
Page 16	P. 75 and 76: Acetaldehyde is stated to be a class B2 carcinogen and acrolein a class C. While this is correct, it is important to at some stage mention which set of EPA guidelines are being used to make this classification. All chemicals going online to IRIS currently use both the 1986 EPA cancer guidelines (the source for the above mentioned classifications) and the EPA Draft 1996 Carcinogen Assessment Guidelines, which report cancer characterizations in a different manner.	The classification according to the 1986 EPA cancer guidelines has been cited when referring to carcinogenic classification for both chemicals.
Page 16	P. 78, end second paragraph: How does outdoor air (second last sentence) differ from ambient air (last sentence)?	Section 4, Health Risk Evaluation has been revised and no longer contains this sentence.
	Jinkampo Incinerator Complex (FAQ), p. 2, second bullet: This bullet should make note that the skin problems are only found at high levels.	Comment has been noted.
Page 16	Jinkampo Incinerator Complex (FAQ), p. 3, last answer: This needs to include the phrase "up to" before the 110 additional cases.	Comment has been noted
Page 16	Jinkampo Incinerator Complex (FAQ), p. 6: Noting the timing of siting and construction decisions at NAF Atsugi in the 1990s, full and	The FAQ mentioned by the reviewer is "Why did the Navy open two new housing towers so close the smokestacks?" The answer was "The

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	fair disclosure would suggest that these details be included in the background information on the incinerator controversy. Also, it would be hoped that the known air quality issues at the time of construction would have prompted the incorporation of appropriate air conditioning and cleaning apparatus at the new residential and childcare facilities, a situation that might help mitigate ongoing exposures.	Government of Japan programs all major building projects on U.S. military bases five to seven years in advance. The two towers closest the incinerator, Buildings 3101 and 3102, opened for occupancy in May 1996 and May 1997, respectively. Both towers were sited and constructed before the 1995 screening HRA report documented the level of health risks. The Navy is investigating the feasibility of installing special filtration systems in all residential towers to clean the incoming air as much as possible. The Housing Division has also issued portable air cleaners to all on-base residents." It is important to note that the ventilation systems in all residential buildings were all similar and called passive because they required action on the part of the occupant to cause fresh air infiltration. The educational buildings such as the elementary school and the child development as well as the Ground Electronics Maintenance building had active (forced fresh air input) ventilation systems. The reviewer's suggestion for inclusion of details regarding timing and siting of the residential towers has been addressed in the background part of Section 1 of the HRA summary report with respect to the close proximity of
	Su caifin Commontae Pioneau Assessment	these buildings to the SIC. It has also been addressed in the rationale for the selection of the risk assessment scenarios considered in the risk assessment on Section 3.
D 15	Specific Comments: Pioneer Assessment	
Page 17	P. 12, Section 2.1.1.2., Soil Trend Analysis: This section describes the methods that were used to test if a spatial correlation exists between soil concentration of COCs and the distance from the SIC source. The two methodologies (Thiessen Polygons and Kriging Analysis) are briefly explained. It would be valuable if the general results of the soil trend analysis could be presented to the reader in this Section,	Additional text describing the soil trend analysis and the results/conclusions has been added to the main body of the report. The results of the soil trend analysis were not used directly in the risk assessment. The purpose of these results was to assist in making risk management decisions about the human health risks at NAF Atsugi, Japan.

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	especially if the results were actually used to determine some of the risk calculations. The reader should not merely be referred to Section 7 and Appendix D. Also, it would be useful to know if there was a positive correlation between the two methods (i.e., do both methods predict the same trend?).	The PIONEER Report has been revised to state: "An overview of the results of the trend analysis is presented below. Appendix E presents a detailed discussion of the soil trend analysis. Three COCs were selected for the trend analysis based on their relative toxicity and because they represent different chemical classes (i.e., inorganics, semi-volatile organics, and dioxins/furans): Arsenic The Total Benzo(a)pyrene equivalent concentration (Total BaP TEQ) The Total 2,3,7,8-Tetrachlorodibenzo-p-dioxin (2,3,7,8-TCDD) equivalent concentration (Total 2,3,7,8-TCDD TEQ) The results of the Thiessan Polygon and Kriging analysis were consistent. Elevated concentrations of Arsenic were observed throughout the base but no clear pattern of contamination was revealed via either method. Total BaP TEQs did not exhibit any spatial trends in concentration and distance from the SIC. Elevated concentrations were observed sporadically in the soil across the base. The concentrations of 2,3,7,8-TCDD TEQ clearly decrease as the distance from the SIC increases. Elevated concentrations of Total 2,3,7,8-TCDD TEQs in subsurface soil were typically collocated with elevated concentrations in surface soil."
Page 17	P. 12, Ambient Air Monitoring: This Section mentions how many samples were collected over a 14 month period but does not explain the timing or frequency of the sampling events. Other information is included, for instance, in Section 2.2 of the HHRA Summary, suggesting that a statistical sampling approach was developed that indicated six day sampling to be appropriate. It would be useful to include a little more discussion of this statistical approach and its implementation. For example, were the actual sampling events spread	Additional text has been added stating that sampling occurred on a six-day cycle for the first 12 months of sampling and that focused sampling (i.e., sampling during expected downwind events from the SIC) was performed for the last two months of sampling. In the text the reader has been referred to the Radian 2000 Sampling Plan and Site Characterization document for specific information on the sampling plan. The PIONEER Report was revised to state:

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	over equal time intervals during the 14 month period, or was sampling performed more often during time periods when the SIC was expected to have a higher impact on NAF Atsugi? Were more air samples collected at certain locations (e.g., living quarters)?	"In April of 1998 a 14-month ambient air monitoring program was instituted at NAF Atsugi in order to characterize the health effects associated with exposure to ambient air. The sampling occurred on a six-day cycle for the first twelve months of sampling. For the final two months focused sampling was implemented: samples were taken during expected downwind (from the SIC) events. For more information on the details of the sampling plan see the Radian 2000 Sampling Plan and Site Characterization. Over two thousand ambient air samples were collected and the results are described in the NAF Atsugi, Japan Ambient Air Monitoring Summary 21 April 1998 – 01 June 1999 (Radian, 2000). The samples were analyzed for multiple constituents including metals, semi-volatile organic compounds, pesticides, polychlorinated biphenyls, volatile organic compounds, and dioxins/furans."
Page 17	P. 13, Ambient Air Dispersion Modeling: This section explains that the ambient air monitoring results were used in combination with air dispersion modeling to calculate ground level air concentrations across NAF Atsugi based on the SIC as the point of emission. This section does not state which air model was used for the analysis. Was an Industrial Source Complex (ISC) point emission model used for this analysis or was a more general screening method used? It is difficult to visualize how a point emission model could be used if most of the SIC stack characteristics (e.g., gas flow velocity, average temperature, building downwash) are unknown. It would be helpful to include more explanation about how the model inputs were back-calculated from the air monitoring data.	The U.S. EPA Industrial Source Complex-Short Term (ISCST3 Version 98356) model was used for this analysis. Since ambient air dispersion modeling data was not used in the risk assessment, explanation about how the model inputs were back calculated from the air monitoring data has not been included in the Pioneer report, but more appropriately in the revised NEHC Summary report.
Page 17	P. 13 Section 2.1.3 Indoor Air and Indoor Dust: Under #1, the word "missions" should change to "emissions." Under #2, the acronym "RBSCs" is introduced without a definition. This term is discussed	Corrections have been made in the revised report.

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	later in Section 2.4. The same caveats apply, as noted above, concerning reliance on EPA Region III numerical screening values.	
Page 17	P. 14, Section 2.2, second bullet: Concerns were raised by EPA regional assessors that the treating of field duplicates as discrete observations reflected a misunderstanding of the Exposure Unit concept, and that field duplicates should either be averaged and subsequently included as one measurement, or the lower of the two values discarded.	The statement in the Pioneer report has been revised. Duplicate air samples were only collected at the GEMB for all chemical groups except for Hg, which was collected at every site. They were used to determine precision of sampling and they were not included in the determination of exposure concentrations. Duplicate soil samples were collected at every area of concern and used as discreet samples. However, since the risk contribution was 95% from air, double weighing caused by the use of duplicates as discreet samples does not contribute to a significant change in the total risk.
Page 18	P. 14, Section 2.2, page 14, eighth bullet: The steps involved in the CROP decision rule should be briefly explained, especially if the use of CROP figured prominently in the determination of exposure point concentrations.	The steps in the CROP decision rule have more clearly described in the revised report. In instances where analytical overlap occurred (i.e., results for a constituent were reported by different analytical methods for the same sample), a set of decision rules, called Compound Rules of Precedence (CROP), was applied to the data to select the concentration that should be used for risk assessment purposes (i.e., development of exposure point concentrations). CROP prioritize the selection based on the sensitivity of the analytical methods involved in the overlap. However, other factors, such as the whether or not the analyte was positively detected by both methods, are also considered. The CROP rules used to reduce the analytical data and develop the exposure point concentrations presented in Section 2.5 are described below.
		Analytical overlap was identified only in ambient air data for constituents in the following methods: 1. Gas Chromatography/Mass Spectroscopy (GC/MS; EPA Method TO-
		15) [CROP Level of Precedence: 1] and Semi-Volatile Organic

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		Compound (SVOC; SW8270) [CROP Level of Precedence: 2]
		2. GC/MS (EPA Method TO-15) [CROP Level of Precedence: 1] and Aldehydes/Ketones (EPA Method TO-11) [CROP Level of Precedence: 2]
		3. Mercury (Gold foil amalgamation) [CROP Level of Precedence: 1] and Hopcolite-Resin Mercury [CROP Level of Precedence: 2]
		A conditional level or precedence was used so that results with a higher level of precedence (indicated by the lower number) were used to develop EPCs in all cases except in instances where the result of a constituent with a higher level of precedence was not detected and the result for the lower level of precedence was detected. In these instances the lower level of precedence result was used to develop the EPC.
Page 18	P. 15., Section 2.3 Background Screening of COCs: On the same theme as the above comments, the Pioneer Assessment is overly brief in discussing the selection of the background soil site, and too quickly refers the reader back to a document not in the possession of the reader. It will be valuable to briefly explain the background soil site that was selected for NAF Atsugi and to explain why this background site would not be expected to be impacted by emissions from the SIC. Is this likely to be an "unimpacted site" or the least impacted site? This is an important point because the site was apparently used as background for both inorganic (possibly naturally occurring) and <i>organic</i> (not likely to be naturally occurring) constituents.	A brief explanation of the background site selection has been added to revised report. A suitable site-specific background soil site was identified as described in the <i>Phase II Soil Sampling Report Addendum to the March 1998 Report - NAF Atsugi, Japan</i> (Radian, 1999a). In summary, a list of optimum location criteria was developed. This list included several screening factors, with the main two being: 1) soils should be located under an impervious, protected cover; and 2) the cover should have been in place since before initiation of SIC operations (i.e., pre-1985). Multiple sites were identified and ultimately Building 47 – the Former Bachelor's Enlisted Quarters, located on the northwest portion of NAF Atsugi, was selected as the background sampling site. A total of twelve soil samples were collected from beneath the building.
Page 18	P. 15., Section 2.3.1 Soil: The second sentence states that the maximum detected background soil concentration of each COC was compared to the maximum detected soil concentration of each COC at	The text and tables have been clarified in the revised report. The maximum background soil concentration of each COC, not the average, was compared to the maximum detected soil concentration of each COC at the AOCs.

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	the AOCs. However, Appendix B, footnote 1 and table structure, imply that the average background soil concentration of each COC was compared to the maximum detected soil concentration of each COC at the AOCs. Any discrepancy in the actual approach should be resolved. EPA considers the latter approach (i.e., Appendix B) to be more conservative and appropriate to use for risk assessment.	Other summary statistics are presented to provide the reader with information on the range of concentrations observed.
Page 18	P.18., Section 3.1 Potentially Exposed Populations: The selection of potentially exposed populations and exposure pathways appears to be appropriate. Since the EPA was asked to address the methodologies and uncertainties associated with different risks for the various subpopulations, we note that the most recent EPA guidance for the assessment of risks from indirect pathways of exposure recommends that risks from breast feeding of infants should be evaluated (<i>Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities</i> ; Peer Review Draft; EPA530-D-98-001A; U.S. EPA; Washington D.C.; Office of Solid Waste; July 1998). The chemicals of concern for this situation are the highly lipophilic chemicals, which have the potential to bioaccumulate, and are transferred to milk fat (e.g., dioxins and other chlorinated hydrocarbons). As the risk from dioxin to the adult population at the residential towers is estimated at up to 10 ⁻⁵ (Table 5-6), it is possible that some additional exposure to infants could be occurring through breast milk.	NEHC's current medical recommendation is not to provide testing of dioxin in blood/breast milk for the following reasons: 3. Dioxin levels in blood/breast milk are not standardized medical tests; They are costly and are still primarily a research tool. Consequently, Dioxin values from testing would vary by method used in testing and by quality control efforts in the laboratory doing the test. 4. There are no medical guidelines for interpreting results in individuals. Consequently, knowing a person has X level of dioxin in their blood is all we would know. In addition, the NAF Atsugi population is of transient nature, which may make it difficult to differentiate prior from current exposures. Appropriate control groups may also be difficult to find. As pointed out by the reviewer, any increases would likely be small and may be difficult to distinguish from the control group(s). Instead the Navy recommendation is not to conduct blood or breast milk testing but rather continue to provide medical counseling and risk communication to base residents regarding the limitations of performing these tests.
Page 18	P. 20, Section 3, table 3-1, subscript 3: Five hours outdoors per day seems a very long time. Has this figure been checked, or does it apply to time outside the house for children, which may include time spent indoors elsewhere?	Text has been added to the revised report clarifying that the 5 hours is based on the EPA Standard Default Exposure Factors. Outdoor and indoor exposure to soil and dust were partitioned based on the amount of time an individual is outdoors. For adult and child residents it was assumed that 30% of time is spent outdoors. This value is based on information

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		presented in the USEPA Standard Default Exposure Factors, which indicates that residents spend 5 out of 16 waking hours outdoors. For all other exposure scenarios it was assumed that an individual spends a total of 2 hours outdoors per day.
Page 18	P. 22, Section 3, second paragraph: The assessment states that "exposure levels for carcinogens are(i.e. 70 years)." EPA assumes this means that for a 3 or 6 year residential exposure scenario the assessment takes the mean exposure level for each pollutant over that period of time and then averages over 70 years to come up with the cancer risk. The assessment may be improved by elaborating in this paragraph on just how the calculations were performed so that the reader will clearly understand. Also, Table 3-2 needs another footnote to define the acronyms in the equation at the top.	More text explaining how the risks for cancer and non-cancer risks were calculated has been added to Section 5 of the revised report. "The PIONEER Report has been revised to state: "Exposure levels for carcinogens are averaged over the lifetime of the exposed individual (i.e., 70 years). This assumes that exposure to a carcinogen could cause cancer to develop subsequent to exposure, at any time in your lifetime. Exposure levels for noncarcinogens are averaged over the duration of exposure, which assumes that the effects of exposure to a noncarcinogen are seen at the time of exposure, and are directly related to the period of exposure. This concept is incorporated into intake calculations as the Averaging Time (AT) parameter. Calculation of the AT is shown in Tables 3-2 through 3-6."
Page 19	Page 23: Additional justification should be given regarding the choice of the adolescent soil ingestion rate as being the midpoint between the adult and child levels.	This value was selected based on professional judgment – in an effort to be protective and a reasonable estimate of exposure. The USEPA Standard Default soil ingestion rate for adult residents is 100 mg/day and the default soil ingestion rate for child residents is 200 mg/day. Therefore, for adolescents 150 mg/day (i.e., the midpoint between the adult and child ingestion rates) was used.
Page 19	Page 24: Ingestion of 200 mg/day of indoor dust was considered by an EPA regional assessor to be high for a child. The studies from which the child's soil ingestion rate was derived did not distinguish between soil and dust (fecal tracer studies). Consequently, an additional qualification should be added that consideration of 200 mg soil (table 3.2) and 200 mg of dust (table 3.3) per day represents a protective set of assumptions. The issue of the independence of exposure scenarios is briefly discussed on page 67, but requires further clarification in the	The 200 mg/day was obtained from the USEPA Standard Default Exposure Factors Handbook.

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	table and more detailed scenario development up front.	
Page 19	P. 25, Table 3-4, Exposure Parameter "AB": The acronym "CCS" needs to be defined. Does this refer to the Percutaneous Absorption Factors shown in Table 3-7?	CSS has been defined in the revised report. The reader is referred to Table 3-7. CSS = Chemical Specific Absorption Factor and it does refer to the Absorption Factors shown in Table 3-7.
Page 19	P 25, Table 3-4: Concern was raised by a regional assessor that the contact rate or soil-skin adherence factor is too high. The Exposure Factors Handbook shows a value of about 0.07 mg/cm² based on experimental data.	The contact rate (Adherence Factor) has been changed to 0.07 mg/cm² in the revised report. The contact rate used in the draft report is a conservative value. For example, the Adherence Factors (AF) presented in USEPA's Risk Assessment Guidance for Superfund are 1.45 mg/cm² for commercial potting soil and 2.77 for kaolin clay. Current information suggests that contact rates are body part and activity pattern dependent and have a wide range (i.e., < 1mg/cm²-event to > 1 mg/cm²-event). These contact rates are considered protective and reasonable.
Page 19	P. 30, Section 4.1, third full paragraph: EPA recommends modifying the first two sentences in the paragraph beginning with "the values presentedconstituent." This is because the IRIS system was changed in 1996 and the RfC/RfD work group and CRAVE were disbanded in favor of a consensus approach across EPA Offices. A more appropriate text might be:	The text provided by the EPA has been incorporated to the revised report as appropriate.
	"Many of toxicological summaries on IRIS were developed prior to 1996 and the information and values presented were verified by either the USEPA Reference Dose/Reference Concentration (RfD/RfC) Work Group or the USEPA Carcinogen Risk Assessment Verification Endeavor (CRAVE) group. IRIS entries in 1997 to the present represent USEPA consensus information. Chemical-specific health assessment information on IRIS is a result of a comprehensive review of chronic toxicity data by U.S. EPA health scientists from several Program Offices, Regional Offices, and the Office of Research and Development."	

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	You may wish to add all or portions of the following, as well: "The Integrated Risk Information System (IRIS) is an EPA database containing Agency consensus scientific positions on potential adverse human health effects that may result from chronic (or lifetime) exposure to environmental contaminants. IRIS contains chemical-specific summaries of qualitative and quantitative health information in support of two steps of the risk assessment process, i.e., hazard identification and dose-response evaluation. IRIS information includes the reference dose for non-cancer health effects resulting from oral exposure (the RfD) and the reference concentration for non-cancer health effects resulting from inhalation exposure (the RfC) and the carcinogen assessment information."	
Page 20	P.31, numbered points: The seven listings at the top of this page are confusing. Numbers 3 through 6 are difficult to distinguish. EPA suggests taking the appropriate text information from the preceding paragraph and placing it in the listing so that each item number is described.	The text presents the prioritization scheme that was used to identify and select toxicity values for constituents. The sources presented in the list are described on the previous page (Page 30.)
Page 20	P.31, items #5 and #6: It is not standard practice to use subchronic noncancer toxicity values as a direct surrogate for chronic toxicity values. If the subchronic value is used, then an additional Uncertainty Factor (UF) would be applied for extrapolating the animal data from a subchronic NOAEL to the chronic NOAEL. (Refer to the EPA methodology at http://www.epa.gov/iris/rfd.htm). A UF of 10 would usually be applied, which would reduce the subchronic RfD or RfC by a factor of 10.	The risk assessment has been revised to incorporate the recommendation. Surrogate chronic toxicity values were derived from subchronic toxicity values by dividing the subchronic toxicity value by a factor of 10.
Page 20	P. 32, Table 4.1: Although the EPA weight of the evidence categories	In the revised report a footnote has been added to table 4.1 that indicates

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	listed are still operative, it should be clearly noted that the Agency is moving toward a more narrative carcinogen characterization as described in the 1996 Draft Carcinogen Assessment Guidelines.	that the EPA is moving towards a narrative carcinogen characterization scheme.
Page 20	P.32, Unit Risks text under Table 4.1: EPA suggests that you give an example calculation in addition to the brief text description of inhalation unit risks. As it stands, one may not know what is meant by 'inhalation unit risk' and what the units are. An example calculation will show how the units cancel out. Also, in the case of children, what body weight is used in this calculation?	An example calculation of how Unit Risks (URs) are converted to cancer slope factors (CSFs) as described on page 32, has been added to the revised report. URs are converted to CSFs by multiplying the UR by 70 (kg body weight) and 1,000 (ug/mg conversion factor) and dividing by 20 (m³/day inhalation rate.
Page 20	P. 32, section 4.3.2: Suggest modifying the title to read: Derivation of Oral Reference Doses and Inhalation Reference Concentrations.	The title has been changed Derivation of Oral Reference Doses and Inhalation Reference Concentrations in the revised report.
Page 20	P. 33, 2nd full paragraph: Suggest adding the following information on the RfC derivation: "The Inhalation Reference Concentration (RfC) is analogous to the oral RfD and is likewise based on the assumption that thresholds exist for certain toxic effects such as cellular necrosis. The inhalation RfC considers toxic effects for both the respiratory system (portal-of-entry) and for effects peripheral to the respiratory system (extrarespiratory effects). Inhalation RfCs were derived according to the Interim Methods for Development of Inhalation Reference Doses (EPA/600/8 88/066F August 1989) and subsequently, according to Methods for Derivation of Inhalation Reference Concentrations and Application of Inhalation Dosimetry (EPA/600/8 90/066F October 1994)." In the RfD calculation, EPA recommends using the term	The text has been changed in the revised report as suggested. The RfC calculation was not presented because RfCs were converted to RfDs for the purposes of evaluating multiple exposed populations in the risk assessment.
	"uncertainty" factor rather than "safety" factor. EPA does not use the	

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	word safety factor unless specifying a factor to be added for additional protection, which is more a policy than a scientific decision. As the same calculation holds for the RfC, why not use the same general calculation for both?	
Page 21	P.48, Table 4-3, Inhalation Toxicity Values: The units for this table are unusual, given that Reference Concentrations are usual given as mg/m³, and the inhalation slope factors as risk per μg/m³. Why are the column headings in kg-day/m³? Furthermore, EPA notes that for dioxin TEQs the oral cancer slope factor has been used. This is a reasonable action, but it does require a number of assumptions and conversions from the oral intake to the inhalation intake, such as percent absorbed, etc. EPA was unable to find clarifications to either of these questions in the table, and recommends that these be added. A clear separation is also needed between those RfCs/inhalation slope factors that come from IRIS, for instance, and those where some conversions were made.	As indicated in the text RfCs and Unit Risks were converted to RfDs and Cancer Slope Factors, respectively in order to evaluate multiple exposed populations. In the revised report a footnote has been added to Table 4-3 indicating this. The slope factor for dioxin is directly from EPA's HEAST FY-1997 Table 1 and was not derived or modified prior to use in the risk assessment. The source of the toxicity values presented in Table 4-2 and 4-3 are clearly presented in the SOURCE column of the table.
Page 20	P. 60, Table 4-4: Suggest defining in the table title precisely where the assessment searched for, and could not find, the toxicity information. For instance, there is copious toxicity information on ethanol, but, due to the as yet undefined data retrieval decision matrix for this table, this data apparently could not be used.	The sources of toxicity values that were searched are presented on Page 30. A footnote has been added to table 4-4 in the revised report indicating the sources that were searched to make it clear to the reader.
Page 20	P. 66, Purpose, bullet points: EPA recognizes that the discussion of what constitutes a safe level, benchmark or action level, etc., for cancer and non-cancer endpoints is difficult to summarize because there is always the need for the risk manager to take into consideration the site in question. As noted above, however, EPA is quite concerned that both the Pioneer Assessment and HHRA Summary have focused too strongly on the RfDs/RfCs/CSFs/10 ⁻⁴ etc.	Changes have been made in the NEHC report

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	values being benchmarks, and EPA emphasizes that these values must be interpreted in light of the exposure situation under consideration. That said, the interpretations of "acceptable" risk measures presented in these bullet points are reasonable. This language refers to the general risk interpretation for the Superfund program and risk management decisions for Superfund sites.	
	EPA has also presented some more specific risk management criteria for evaluating waste combustion facilities. The criteria are contained in a guidance titled: "Implementation Guidance for Conducting Indirect Exposure Analysis at RCRA Combustion Units" (EPA Office of Solid Waste; April 22, 1994). The criteria were presented as a set of "Acceptable Target Levels" intended to protect human health from risks posed by emissions from hazardous waste combustion units. The target levels are used to evaluate the results of risk assessments for stack emissions. They also provide a basis for recommending additional permit conditions and limits, if necessary, to ensure the protection of human health.	
	The acceptable target levels may be summarized as follows: A) The total cancer risk due to high-end individual exposure to carcinogenic constituents should not exceed 1E-5 (i.e., an upper bound lifetime risk of one predicted case of cancer in a population of 100,000);	
	B) For toxic chemicals, the high-end individual hazard index for the mixture of toxic constituents should not exceed 0.25; As stated in the OSW Guidance, these target levels were adopted in part to account for, and provide protection from, likely background exposure to contaminants that could occur in the vicinity of a given	

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	combustion unit. The Guidance states: "The selection of these [target] levels (as opposed to, for example, an incremental cancer risk of 10 ⁻⁴ and a hazard quotient of 1.0) was done in part to account for exposure to background levels of contamination (including indirect exposures from other combustion units) which should be considered as part of the risk estimation and decision-making process to set emission levels at a combustion unit. The unit will not likely be the only source contributing to exposures in the study area and to neglect other environmental sources may overestimate an allowable emission level, leading to unacceptable total risk to the public. In this case, background is defined as those exposures in drinking water, food, and air attributable to sources other than the combustion unit(s) being assessed. If detailed information on background sources is available for a particular area, the permit writer may choose to use this information to develop an alternative approach for incorporating background levels." The discussion above is presented by EPA for informational and comparative purposes, not as a formal recommendation for how the Navy should proceed on risk management issues at NAF Atsugi.	
Page 22	P. 66, Sections 5.0 and 5.1: The focus in these sections is on the RfD, which is unusual given that the principal risks are coming from the inhalation route, and thus the RfC and inhalation slope factors would be more relevant. As noted before, it should be made clear that the RfC is converted into an RfD before subsequent calculations are made, assuming this is the case. EPA's concerns on this section relate back to previously mentioned issues where it is difficult to follow the flow of the document because it is compartmentalized with no clear, up-front, explanation of what is being done. One suggestion would be to have a flow chart which shows all the calculations for both	In the revised report a note has been added to the section indicating that RfCs and Unit Risks were converted to RfDs and Cancer Slope Factors so that multiple exposure populations could be evaluated in the risk assessment. Table 5-3 presents the total risk by exposure pathway so that the reader can determine which exposure pathways are responsible for the majority of the risk. Table 5-4 presents the noncancer hazards segregated by the target organ/critical effect of the toxicity study that formed the basis of the RfC. Figure 3-1 (page 21) presents a flow-chart of the conceptual site model for NAF Atsugi, Japan Risk Assessment.

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	noncancer and cancer effects, accompanied by text and explanations as to the exposure scenarios under consideration. This would be extremely useful in trying to figure out what is being portrayed in very complex tables like tables 5-3 and 5-4, from whence the myriad calculation endpoints originate.	
Page 22	P. 80, Section 5.3.4 Lead Evaluation: EPA reviewers raised a concern that the finding of a 0.02% probability of a blood lead level of 10 ug/dL is abnormally low. This probability would result in a geometric mean blood lead below 2.5 ug/dL. Based on the listed inputs for NAF Atsugi (i.e., lead soil concentration of 26.6 mg/kg; lead air concentration of 3.9 ug/m³) in the IEUBK LEAD99d model, an EPA reviewer was unable to reproduce the listed result (The value of 26.6 mg/kg was used as the outdoor and indoor soil lead concentration together with a constant ambient air concentration of 3.9 ug/m³. The values found for the age range from 0 - 84 months were a 0.18% probability of blood lead at 10 ug/dL with a geometric mean of 2.6 ug/dL). In any case, there does not appear to be a significant probability that the CDC health recommendations for blood lead in children would be exceeded at NAF Atsugi. However, it should be noted that the site-wide RME lead air concentration of 3.9 ug/m³ is well above the quarterly lead NAAQS of 1.5 ug/m³.	The IEUBK model runs have been re-evaluated to ensure that they are correct. There was a typographical error in this section. The site-wide RME lead air concentration should read 0.39 ug/m3 not 3.9 ug/m3. Since 0.39 ug/m3 is below the NAAQS value for lead the note will not be added.
Page 23	P. 80, third paragraph: The lead level in the child is reported as 10 - 19 μg/dL. Why is such a broad range reported, and why are we not given the actual measure? For instance, the CDC lead guidelines make a clear distinction between 10 - 14 and 15 - 19, where an elevation to 11 is clearly different from an elevation to 19. It would be reassuring to have a little more information on this exceedance value, subject of course to patient confidentiality necessities.	The PIONEER Report has been revised to state: "Note: The Department of Defense policy on the assessment of health risk from lead is that blood lead levels of 10-19 ug/dl require confirmatory blood lead determination within one month of the first result. Confirmed 10-19 ug/dl blood lead results require a reassessment of the risk factors for exposure, education concerning diet and personal hygiene. If levels persist the policy requires the initiation of individual case management,

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		environmental investigation, and lead hazard abatement. Rescreening is also required every 3 months. Upon confirmatory blood lead determination the child was found to have a blood lead level of less than 10 ug/dl. This paragraph has been added to text to clarify the issue.
Page 23	P. 93, Recommendations: Recognizing that the EPA is not intimately familiar with the NAF Atsugi site and that the Navy has the lead in this, we offer the following thoughts on the recommendations for consideration. Regarding Recommendation #1, the Agency notes that limiting childhood exposures to 32 months and adults to 98 months cannot be considered a solution, in itself, but may be a valuable adjunct to other management actions. As stated above, these time limits are overly "accurate" and are actually associated with significant hazard indices and cancer risks. A particularly disturbing factor militating toward recommendation #10 is the information in Pioneer Assessment Section 1.2.1, where it is stated that the incinerator is already equipped with a battery of pollution control devices that may be underutilized or not properly operated. Even if more proper operation of the incinerator was achieved, the design characteristics and appropriate waste feed limits of the incinerator need to be examined. If this combustion device was designed to be a municipal waste incinerator, it may never effectively operate to provide the proper destruction efficiency and pollution control needed for proper treatment of the wide description of listed wastes, including trash and (apparently) hazardous waste. The first bullet under recommendation #2 could provide some risk reduction from acute exposures, but would not seem to be an effective chronic risk reduction method since outdoor contaminants migrate indoors and may actually achieve higher concentrations in the indoor	Risk management recommendations have been deleted from the Pioneer and NEHC reports.

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	environment. This recommendation could well be linked to recommendation 8, air conditioning of residential and school quarters, which presumably would not be difficult as the quarters are only a few years old and presumably have well performing equipment. The other recommendations seem logical, but only the implementation of recommendation #10 would result in a significant method to minimize SIC emissions and chemical exposures. The problem remains that the location of the quarters and facilities so close to the SIC emission stacks fundamentally exacerbates any problems. In this regard, EPA notes that the Japanese government is part of a global United Nations Environment Program negotiation on Persistent Organic Pollutants (POPs), scheduled for completion late 2000/early 2001. It is our understanding that the Japanese government is adopting, or has adopted, dioxin emission standards for new sources. Under the draft UNEP agreement, Parties are also encouraged to retrofit BAT to existing incinerators.	
Page 23	Appendix C, Table C-1 and following tables: These Tables appear to contain some misprints in the columns under "Carcinogenic Risk (CR)." For example, the child cancer risk for outdoor inhalation of acetaldehyde is listed as 6.05E-7 and 2.28%; but the outdoor inhalation cancer risk from Bis(2-ethylhexyl)phthalate is listed as 6.94E-1 and 0.00%. The "6.94E-1" value is probably actually "6.94E-10."	The tables in Appendix C have been corrected so that the cancer risks are correctly printed (i.e., the Carcinogenic Risks field needs to be made wider so that the risk numbers print correctly).

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	SUMMARY	
Page 2	A lack of proper planning was evident in the risk assessment and appeared to cause inconsistencies in the objectives of the data collection and risk assessment. The purpose and objectives of the risk assessment were not consistent and not clearly stated.	Although the subcommittee was provided with a sampling plan document and quarterly summary reports the subcommittee speculates that poor planning is the cause for differences in wording of the specific objectives of the risk assessment in different documents. While we recognize that the wording on the objectives was different in various documents that were written by different people, the objectives implied by each were the same. All individuals agreed on the objectives at the initial kick off meeting. However, when the NRC subcommittee addressed the issue of different objectives, it was clear how they derived a conclusion that the objectives were different. The sampling plan addresses all of the elements needed in a sampling and QA/QC plan to collect meaningful data. The documents provided to the subcommittee did reflect the extensive and continuous planning that was conducted during all phases of the data collection, risk assessment and data interpretation. Nevertheless, NEHC added a subsection in Section 1 on planning, that include the details on the planning that were not mentioned in the NEHC report before. In the revised report, NEHC has also referred to a site visit report and additional monthly project review reports that were not submitted
		to the subcommittee to demonstrate and emphasize that planning and periodic (monthly and quarterly) reviews were indeed carefully conducted.
Page 2	The purpose and objectives of the risk assessment were not consistent and not clearly stated.	NEHC has clarified the objectives to ensure consistency.
Page 2	For future risk assessments of this nature, NEHC should follow a general risk-assessment framework, such as those discussed in <i>Science and Judgment in Risk Assessment</i> (NRC 1994), and <i>Framework for Environmental Health Risk Management</i> and <i>Risk</i>	We are familiar with each of the documents cited, and are not clear what specific methodology is being recommended, since the NRC framework for conducting a risk assessment is identical to the EPA Superfund methodology. Upon request for clarification, the NRC subcommittee has

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	Assessment and Risk Management in Regulatory Decision-Making (Presidential/Congressional Commission on Risk Assessment and Risk Management 1997a,b) and should consider the use of independent peer reviewers to oversee the entire assessment process, including the planning stages.	neither provided specifics regarding the recommended methodology to follow nor citations that we can obtain where others have followed that recommended methodology. Considering that an EPA risk assessment was being conducted, NEHC did involve various independent peer reviewers from EPA through all stages of the risk assessment, beginning with the planning stages. EPA NCEA RTP and their FTIR contractor accompanied NEHC and its contractors on the initial site-scoping visit to Atsugi and continued their involvement throughout the process.
Page 3	The most appropriate methods were not used to determine the contribution of the incinerator complex to health risks at NAF Atsugi. The excellent and innovative air-dispersion modeling, used in conjunction with correlation analyses, would be the most appropriate method to determine the contribution of the incinerator complex to the health risks at NAF Atsugi.	The determination regarding the method to be used to assess the contribution of risk from the incinerator was thoroughly evaluated. A tremendous amount of deliberation went into selecting the method that we used. We addressed the issue with EPA personnel and also with a group of expert statisticians from the Research Triangle Institute, Research Triangle Park, North Carolina.
		The NRC selected very positive words to discuss a dispersion modeling approach to the risk assessment while many negative ones were used in discussing EPA RAGS methodology that uses actual versus modeled data. The underlying reason for the strong support of modeling is not clear for the following reasons: (1) The dispersion modeling approach determined that only 6 of the 336 chemicals monitored showed a strong correlation with the SIC. In addition, since dispersion modeling was performed using ambient air concentrations to estimate stack emissions, the use of dispersion modeling to predict the contribution of the SIC would only yield a circular logic (2) Associated magnitudes of error are likely to be high because of unknown SIC operation parameters (stack velocities, stack temperature, emission rate) and waste composition data (3) The emission rate estimation

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		analysis varied by 2-4 orders of magnitude. The NRC did not address the additional inherently large uncertainty with the use of dispersion modeling although other uncertainties are very clearly addressed by the NRC. This approach has been encouraged by EPA.
		NEHC sent a written request to the subcommittee for instructions on how to use the dispersion modeling approach used by Radian to determine the contribution of the incinerator complex to the health risks at NAF Atsugi. The subcommittee chose not to reply to our request for instructions in writing, but agreed to a phone conference that included only two members of the subcommittee. During the phone conference, the two reviewers withdrew their recommendation to use a dispersion modeling approach and did not articulate instructions on the approach. They recommended a new approach but couldn't articulate instructions on how to conduct the new approach. Furthermore they indicated that they could not ensure that the approach would provide valid results. As this recommendation to use the dispersion modeling has now been rescinded and these subcommittee members admitted that the new approach could include just as much uncertainty as the NEHC approach, NEHC chose to retain the approach that was initially presented to the subcommittee for review. The approach used by NEHC was actually the approach recommended by the previous NRC Committee on Toxicology that reviewed the 1998 screening risk assessment and stated the following:
		"Another approach that might be useful for getting a rough estimate of the contribution of incinerator emissions to ambient air, relative to the background, would be to compare results from Location 1 (upwind site) with those from downwind locations on days when the wind direction is out of the south-southwest and relatively constant." The subcommittee specifically mentioned that the "upwind site" (named so because of the

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		frequency at which it is upwind from the SIC) should be used for the upwind downwind comparison. However, it is intuitively obvious that upwind and downwind depend on specific wind direction on a particular day.
Page 3	NEHC used outdoor-air samples as surrogates for indoor air, but such substitution is not appropriate	The objective of the HRA was to determine the risk that could be attributed to ambient air emissions from point and non-point sources impacting the air quality at NAF Atsugi. Since concentrations for the majority of the constituents exceeding RBCs were found to be higher indoors than outdoors indicating probable indoor air sources (e.g., insulation, carpets, and household chemicals) and ambient air is the source of constituents in indoor air that are associated with emissions from the SIC, indoor-air samples could not be used for the purposes of quantitative risk assessment because, as pointed out by the subcommittee, they would overestimate the impact of the SIC and other ambient air point and non-point sources. The concentrations could be even higher if sampling occurred during occupancy, because of activities such as cooking, use of household cleaners and smoking. Since indoor air concentrations could not be used in the HRA to calculate indoor air exposures from outdoor air infiltration without overestimating the risk due to the contribution of indoor air sources, the most conservative alternative was to use ambient air concentrations as surrogates for indoor air concentration.
Page 3	At almost all sites, air sampling was conducted for 14 months, with the last 2 months of sampling apparently being collected at times when the contribution of the incinerator was expected to be high. The potential biases in that collection protocol are not discussed in the report or accounted for in the data analysis.	Since no stack sampling was permitted to be conducted on this foreign owned incinerator one of the greatest challenges in this project was to meet the second objective, i.e., to determine the SIC contribution to the health risk. The method we initially employed to determine the SIC contribution was to identify the chemicals in air that are emitted from the SIC by correlating wind direction (specifically the percentage of time an individual monitoring site was downwind of the SIC), to the chemical concentrations observed in ambient air at the site. The hypothesis is that, for chemicals

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#		that are emitted from the SIC, the chemical concentration (and also risk) increases as the percent of time that the wind blows emissions from the SIC onto the base increases. In the case of variable wind speed and direction, from one week to the next, if the emissions are constant but the wind speed and direction are not the same, the correlation with percent downwind will be different. The confidence in the correlation of wind direction versus concentration is related to the number of observations that are used to calculate the correlation coefficient and the wind directions that are observed. During the 1998 ambient air sampling study, there were few periods of southerly winds, even fewer than that indicated by historical wind roses. As a result, there were relatively few data points to correlate concentrations/percent downwind in an effort to assess SIC contribution. Therefore, sampling was extended for an additional 2 months and samples were collected on days predicted to be downwind toward NAF Atsugi from the SIC to complete the correlation plots. To complete the correlation plots, the extended sampling included 6 additional sampling days when the wind was blowing toward the base. The additional sampling also provided better representation of historical exposure conditions. Conducting the risk assessment without this additional data could result in an underestimation of long-term exposure conditions and consequently risk. Therefore the additional 6 days of data were used to calculate the risk. The additional number of sampling days needed to provide additional information for the correlation analysis plots was determined by statistical analysis so that no bias would result by overweighting particular wind directions and overestimating the contribution from the SIC.
Page 3	The interpretation of the risk-assessment results by NEHC is not appropriate, given that a Superfund type of risk assessment was conducted.	With this comment the subcommittee seems to place the Superfund type of risk assessment in a somewhat negative context, particularly since NEHC is directed subsequently to a number of NRC publications for other methodology. NEHC selected the superfund methodology because (1) It is

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"		widely used throughout the United States because of regulatory decision making and therefore has legal precedence; (2) It uses real data to reduce the uncertainty of modeling; (3) It has received much peer review from the scientific community including the NRC and is updated periodically by issuing supplemental guidance; (4) It provides a basis for measuring risk related to a particular source; (5) The specific methodology is the most used tool in the US for risk management; and (6) It is a process identical to NRC's and previously based on NRC documents.
Page 3	The NEHC report should characterize the uncertainties in the risk assessment, including all the principal uncertainties identified in the supporting documents.	NEHC has expanded the subsection on Uncertainties in Section 3 "Human Health Risk Assessment Results" to include additional uncertainties that were addressed in the supporting documents.
Page 3	The conclusions of the risk assessment should be presented in such a way that uncertainties in the data and process that led to the conclusions are evident.	The conclusions have been revised in the final NEHC report pointing out the uncertainties associated with the risk assessment.
Page 3	The report should discuss the health-surveillance studies that have been conducted at NAF Atsugi.	Health surveillace studies have been reported in two separate reports. The third study is still in progress. While the Navy conducted the comprehensive environmental sampling study at NAF Atsugi, health studies were also conducted to identify certain acute health conditions that either could be associated with exposure to poor air quality or were health conditions that concerned the NAF Atsugi community. One of the studies, the "Children's Respiratory Health Effects Study", compares peak respiratory flow between children at Atsugi and Yokosuka. The second, the "Pregnancy Outcome Study," compares spontaneous abortion rates between Atsugi residents and residents of other bases in Japan. An additional air pollution related morbidity medical surveillance is still in progress. It compares rates of skin conditions and respiratory symptoms seen at the NAF Atsugi Branch Medical Clinic and Naval Hospital Yokosuka. The

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#		first two studies are mentioned in the revised report and they are included as appendices D and E.
		Naval Base Yokosuka served as the control location for the studies. This was done for two specific reasons. First, Yokosuka, which is approximately 25 kilometers from Atsugi, is also located on Japan's Kanto Plain. Its population, climate, and vegetation are similar to that at Atsugi. In addition, other than the highly visible point source of pollution at Atsugi (i.e., the Shinkampo Incinerator Complex), sources of air quality degradation are similar. Secondly, Yokosuka is the site of the Navy's primary medical treatment facility in Japan, Naval Hospital Yokosuka, which provides access to several healthcare databases.
		The study on "Respiratory Effects in Children "had two primary goals: 1. Identify differences in respiratory symptoms and lung function between children who live or go to school at NAF Atsugi and similar children at Yokosuka. 2. Determine if there were more respiratory symptoms in children who live or go to school at NAF Atsugi on days when they are exposed to higher levels of pollutants from the Shinkampo Incinerator during the four week study period (7 May-5 June 1998).
		The study focused on children since their health is a major concern of the NAF Atsugi residents. Children's lungs also tend to be more sensitive to the effects of air pollution. Fifth and sixth grade students at Atsugi and Yokosuka DOD Schools participated. One hundred twenty-seven (127) students volunteered for the study. Eighty (80) of the students lived on base at NAF Atsugi, 17 lived off base at NAF Atsugi and 30 lived at Yokosuka. The children's lung function was tested each school day during lunchtime. Children recorded the number of hours spent outdoors as well as respiratory and/or air quality related symptoms such as, trouble breathing, coughing

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#		during the day or night, feeling bad, runny nose, cold, headache, and irritated eyes. A daily symptom score was given to each child based on the information recorded.
		Data from ambient air monitoring at Shirley Lanham School was also collected for PM10, nitrogen dioxide and sulfur dioxide, known to cause respiratory effects. Wind direction and wind speed were also recorded, in an attempt to associate health effects with environmental pollution conditions. The primary findings of this study were:
		 There were no differences in the respiratory health of children living on or off base at NAF Atsugi and those at Yokosuka. Children living on base at Atsugi reported more runny noses than the Yokosuka children did. All other reports of symptoms were similar. There was no difference in the reported number of colds between the Atsugi on base and the Yokosuka groups. Children living off base at Atsugi did report more colds. Most of the children in the study group had lung function better than that of the general population in the United States. The wind was blowing toward the school for only a short period of time during the four-week study period. As a result, no clear relationship between wind direction and the levels of gases and dust particles could be identified.
		Complete information on this study can be found in the report Air Pollution From the Shinkampo Incinerator Associated with Adverse Respiratory Effects Among Children at NAF Atsugi Study (NEHC Jul 99).
		The study on "Pregnancy Outcomes" was conducted because many residents expressed concern during the November 1997 NAF Atsugi Public

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#		Meeting about the health effects the Shinkampo Incinerator may be having on their families regarding miscarriages. Therefore the study was designed to describe the rate of miscarriage, at NAF Atsugi and other naval facilities in Japan. Information for the study was gathered by looking at hospital and clinic records for past pregnancies. This was a retrospective study where only documented miscarriages versus live births were considered.
		The study population consisted of Navy personnel or their dependents who were pregnant at some point between June 1995 and May 1998 and lived on or near NAF Atsugi or other naval facilities in Japan serviced by Naval Hospital Yokosuka (NHY). Information used to calculate the miscarriage rates came from three different sources, Delivery Logs and Pathology records at NHY and the Prenatal Log at the Atsugi Branch Medical Clinic.
		Data collection took place during the summer of 1998. It included the number of live births and the number of miscarriages. The total number of pregnancies with known outcomes during the study period was 1862. For the purposes of this study, a miscarriage was defined as an unintentional pregnancy loss at up to the 28 th week of pregnancy. Multiple births were excluded from the analysis. The miscarriage rate was defined as the number of miscarriages divided by the total number of pregnancies examined (the number of babies born plus the number of miscarriages).
		The findings of the study were: 1. The overall miscarriage rate for patients with known pregnancies from Atsugi, Yokosuka, Iwakuni and Sasebo between June 1995 and May 1998 was 7.1%. This rate was determined by review of the delivery log and pathology records at NHY. When the Atsugi patients are subtracted, the miscarriage rate for the other areas is 7.8%. 2. Review of the NAF Atsugi Branch Clinic prenatal log, during the same

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		period, indicates a miscarriage rate at NAF Atsugi, of 8.8%. However, the data used in this study came from different sources and contain some different information. Therefore, the miscarriage rate at NAF Atsugi cannot be directly compared to that of the other naval facilities that were part of this study population. 3. The NHY and NAF Atsugi miscarriage rates during the study period were both lower than the documented rate of miscarriage for women in the United States, who know they are pregnant, which is between 10% - 15%. 4. This study was conducted with the limited information that was available in various records. The results suggested that the risk of miscarriage at NAF Atsugi and other naval facilities within Japan are at the low end of the expected risk range described for the population of the United States.
		Complete information on this study can be found in the report <i>Pregnancy Loss at NAF Atsugi Japan (June 1995-May 1998) (NEHC Sep 99)</i> .
		The continuing air pollution related medical surveillance indicates that:
		1. There were no significant differences in air quality related morbidity between the adult populations at Atsugi and Yokosuka during the study period. There were no significant differences in air quality related morbidity between the Child (below 18 years of age) populations at Atsugi and Yokosuka during the study period.
		2. There was a peak period of respiratory disease complaints at Atsugi from June –August 1998. This is an artifact of the comprehensive risk communication and health consultation program that was at its height during that period.
		3. There was a peak period of respiratory disease complaints at Yokosuka beginning in November 1998 and persisting through January 1999. This represents an outbreak of Japan Type A Influenza during that period.

Page #	NAS Comment	NEHC Response
Page 3	Information on Japanese standards should be included in the report.	NEHC has edited the report to include information on Japanese air quality standards that are similar to the US National Ambient Air Quality Standards.
Page 3	The report should begin with a clear statement of the purpose and objectives of the risk assessment, and it should include more details of the methods, assumptions, uncertainties, and limitations of the risk assessment.	NEHC has edited the report to clarify the purpose and objectives of the risk assessment, and it has expanded the sections describing more details of the methods, assumptions, uncertainties, and limitations of the risk assessment in the report.
Page 3	The report should be thoroughly referenced, including references to specific sections of the many supporting documents.	NEHC has edited the text to include more specific references to the supporting documents. A reference section has been added to the NEHC report.
Page 3	When drawing conclusions and making recommendations, NEHC should clearly distinguish between those based on science and those based on policy	In the revised NEHC report, a specific subsection has been added to address policy-based versus science-based approaches for evaluating health risk. A summary discussion on policy-based and science—based conclusions has also been included in the conclusions section.
Page 3	NEHC should also describe the ways in which stakeholders have been involved in the policy decisions as recommended by many advisory groups for appropriate risk management practice.	This information was included in the risk communication plan of the Draft NEHC Summary document, which is now Appendix F in the revised NEHC Summary document.
	PLANNING OF THE RISK ASSSESSMENT	
Page 4	In future risk assessments, NEHC should consider the use of independent peer reviewers—beyond the management group mentioned in Appendix B—throughout the project (including the planning stages) to evaluate objectives and proposed methods, to ensure that the project remains focused on the objectives, and to critique the final document	Considering that an EPA risk assessment was being conducted, NEHC involved various independent peer reviewers from EPA throughout the development of the risk assessment, beginning with the planning stages. EPA NCEA and EPA RTP and their FTIR contractor accompanied NEHC and its contractors on the initial site-scoping visit to Atsugi and continued their involvement throughout the process.

Page #	NAS Comment	NEHC Response
Page 4	When planning a risk assessment, NEHC should follow the basic framework for making risk-management decisions that has been laid out for risk assessments NEHC is directed to those reports for general guidance and frameworks for design of risk assessments like the one at NAF Atsugi.	NEHC is familiar with the recommended reports and has followed the basic framework needed for risk management decisions that have been laid out for risk assessment. This framework is identical to the framework in the EPA Risk Assessment guidance for Superfund.
	Objectives of the Risk Assessment	
Page 4	Even the overall objective of the risk assessment is not clear; NEHC should be clear that it was trying to determine whether there is a problem at NAF Atsugi, not that it was trying to show that there is a problem.	NEHC has edited the report to clarify the purpose and objectives of the risk assessment. This project was designed to collect data to meet the objectives of the comprehensive health risk assessment which were: 1. Estimate the potential human health risks to U.S. Navy personnel and their families and other individuals living and working on NAF Atsugi, Japan resulting from exposure to constituents of concern (CoCs) in soil, ambient air, indoor air, and indoor dust. This estimate focuses solely on CoCs that are likely to be associated with ambient air emissions and/or subsequent deposition from point and non-point sources impacting the air quality at NAF Atsugi. 2. The contribution of the risk attributable to the Shinkampo Incineration Complex (SIC). NEHC did not have a preconceived hypothesis that there was/was not a problem at NAF Atsugi, but developed a sampling plan to make this determination. The mere presence of uncontrolled emissions from an incinerator adjacent to the base indicates the potential for exposure and therefore a potential risk.
Page 5	The main text of the NEHC report should identify the population at risk, define sensitive subpopulations that are of special concern, and describe who or what is meant by "sensitive receptors" (see Section 3.2.1; Radian 1998a).	NEHC has expanded Section 3 Human Health Risk Assessment Results to include details on all steps of the EPA risk assessment methodology used in this health risk assessment (Data Evaluation, Reduction, and Screening; Exposure Assessment; Toxicity Assessment; Risk Characterization;

Page #	NAS Comment	NEHC Response
		Uncertainty Analysis). The expanded subsection on Exposure Assessment identifies the populations at risk, defines populations that are of special concern and defines children as the sensitive receptors.
Page 5	The report should provide information on the size of the military population at NAF Atsugi, on the composition of that population (including age and percentage of women, children, and infants), and on the number of retired military and nonmilitary personnel employed on the facility.	NEHC has edited Section 1 of the report to include more details on the size and composition of the military population at the time of the survey conducted for the Exposure Pathways Analysis (June 1998), such as the number of active duty personnel and dependents, civilians and dependents, the number of children under 6 or in the age group of 6-18 years of age. The following text has been added: "The NAF Atsugi population is approximately 7,500 when sailors, residents, and workers are present, of which 81.1% is composed of active duty members and their dependents, 1.22% are Department of Defense employees such as teachers and their dependents, 5.02% are Civil Service employees and their dependents and 12.65% are Master Labor contractors including Japanese nationals. Seventy-five percent of the population live on-base and 25% off-base. It is estimated that approximately 6,000 are adults. There are approximately 446 dependents under 6 years of age and about 916 dependents between 6 and 18 years old living on base versus 129 and 180, respectively, living off-base."
Page 5	Information on the average duration of a tour at NAF Atsugi and the frequency with which the standard tour is extended would also be helpful.	NEHC has edited Section 1 of the report to include information on the average duration of a tour at NAF Atsugi and the frequency with which the standard tour is extended. The following text has been added: "Military personnel are typically stationed at NAF Atsugi for 3 years (1 Tour of Duty), however the tour can be extended to 6 years (2 Tours of Duty) or more."
	Sampling	

Page #	NAS Comment	NEHC Response
Page 5	If FTIR monitoring was expected to detect pollutants more often, the question arises of whether a change in the incinerator complex resulted in pollutant concentrations lower than in the past If that is the case (the supporting, documentation seems to indicate that contaminant concentrations were lower than expected), the report should discuss the decreases.	Since in previous screening risk assessments (NEHC 1995), maximum concentrations of VOCs above the FTIR detection limit were observed for benzene (84 µg/m³), ethylbenzene (100 µg/m³), toluene (420 µg/m³), oxylene (42 µg/m³) and p-xylene (130 µg/m³) collected in 30-second Summa canisters samples, it was anticipated that the FTIR would have been an appropriate real time indicator of air quality. However, during this monitoring effort, SIC operating conditions could have changed so that gaseous concentrations of the target pollutants were not present above the FTIR system's minimum detection limits with a frequency that would allow a statistical analysis addressing its objectives. This discussion has been added to the subsection on the FTIR in the report.
	The NEHC report (p. 11) states that a statistical method indicated that outdoor air sampling should be conducted every 6 days, but the subcommittee was unable to locate a discussion of any such statistical method or its application. The relevant section of the planning document (Radian 1998a; pp. 3-11 to 3-13) states only this: "Air samples will be collected approximately every six days. For each sampling event, analytical results will be obtained for VOCs [volatile organic compounds], mercury, acid gases, and PCDDs/PCDFs (dioxins/furans). Aldehydes and ketones, heavy metals, PCBs, pesticides, PM ₁₀ , PM _{2.5} , and SVOCs [semivolatile organic compounds] will be analyzed every other sampling event, or every 12 days. [The air-sampling plan] will yield approximately 60 outdoor air samples at the elementary school for those chemicals measured during each sampling event and approximately 30 for the remaining chemicals. Such sample sizes are likely to lead to a great deal of	No statistical analysis was performed to derive the every sixth day sampling schedule. U.S. EPA air toxics sampling programs call for the collection of air samples every 6 th or 12 th day. This schedule, which rotated through the 7 days of the week and over a one-year period, produced nearly equal numbers of samples from each weekday. This schedule is used for EPA's Urban Air Toxics Monitoring Program (UATMP) and their Photochemical Assessment Monitoring Stations (PAMS) studies. A draft paper ("Air Toxics Monitoring, Concept Paper", Office of Air Quality Planning and Standards, Revised Draft, February, 29, 2000) by EPA states: "The selection of sampling frequencies will be guided by the data quality objective (DQO) process. DQOs are currently under development and will provide recommendations on the need to sample according to the typical UATMP sample frequency of once every 12 days, the frequency of VOCs collected at PAMS sites of one in six days, or the frequency to be used at PM _{2.5} speciation trend sites and IMPROVE (Interagency Monitoring of Protected Visual Environments) sites of once in three days."

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#	precision in the estimated RMEs [reasonable maximum exposure].	
	For this comparison, either 30 or 60 upwind and either 90 or 180 downwind samples will be available. This is considered to be an adequate number of samples to make a valid comparison."	
	Statistical analyses, which should have been conducted using air measurements from previous years, were needed during the planning stage to determine the number and frequency of samples required if conclusions regarding the health risks and contributions of the incinerator were to be drawn. Such analyses were conducted for the soil monitoring, but not for the air monitoring.	
Page 6	It is not clear how the decision to collect only eight samples was made. It is also not clear how collecting eight indoor-air samples could answer the question "What is the inhalation exposure risk for sensitive receptors in buildings likely to be impacted by the Jinkampo Incineration Complex?" (Radian 1998a; Section 3.2.1) or could determine the impact of the incinerator complex.	The decision to collect eight samples from each AOC was based on the number of samples required to obtain reasonable exposure point concentrations for risk assessment calculations. Exposure point concentrations are based on 95% upper confidence limits (UCLs) for the mean. The UCL is computed as $\overline{x} + t_{0.95,n-1} \frac{s}{\sqrt{n}}$, where \overline{x} is the sample mean, s is the sample standard deviation, n is the sample size, and $t_{0.95,n-1}$ is the 95 th percentile from a student's t-distribution with n-1 degrees of freedom. The quantity $t_{0.95,n-1} \frac{s}{\sqrt{n}}$ reflects the expected precision in the estimate of the mean (i.e., the distance that \overline{x} may be from the true (population) mean). The smaller the variability (reflected by s), and the larger the sample size, the closer \overline{x} is expected to be to the population mean. The UCL reflects this expectation. The larger s is and the smaller n

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		is, the larger the quantity $t_{0.95,n-1}\frac{s}{\sqrt{n}}$ will be, and hence the higher the UCL (and the exposure point concentration) will be. Because the exposure point concentration is based on the UCL, it is 95% certain to overestimate the true average exposure concentration. The degree to which the true average may be overestimated depends on the sample size (n) relative to the variability (s). For indoor air, preliminary discussions led to the conclusion that an assumed relative standard deviation (standard deviation divided by the mean) of 75% was a reasonable a priori estimate of variability within an AOC. Based on plots of precision versus sample size for a relative standard deviation of 75%, eight samples is 95% certain to yield a mean that is within 50% of the true mean. This was considered to be a reasonable sample size.
Page 6	Furthermore, indoor-air samples were collected only for an 8-h period (NEHC 2000; p.17), which is not long enough to fulfill the study objectives. The only rationale provided for that sampling duration was a limitation in homes because the sampling pumps were noisy, but that limitation is not discussed in the planning documents, nor is the possibility of using quieter pumps. It was pointed out that further sampling would have been pointless for the high-volume samplers in the low-infiltration-rate locations because the samplers already sample all the available air several times over, but a rationale for the limitation in high-infiltration-rate locations is needed. In addition, it is not clear why a particular apartment was chosen for sampling and whether any consideration was given to other factors, such as smoking in the apartment.	There were several reasons for the 8-hour sampling periods, a number of which were logistical. An important objective of the study design was to "measure indoor air concentrations with a sensitivity and selectivity comparable to the ambient air measurements (Radian 1998, QAPP, page 4-16)." It was believed that this approach would be able to prevent the occurrence of exposure estimates for the risk assessment based on "not detected" default values. Therefore, it was proposed to use the same commercial high volume instrumentation and methodology as that used for the ambient monitoring since that would allow direct comparisons between the ambient and indoor measurements. Additionally, we know of no commercially available alternative sampling methods that would have been unobtrusive, quiet, and able to collect sufficient sample volumes, to directly compare with the ambient air methodology.

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#		We believe that the 8-hour sampling periods were sufficient to fulfill the study objectives. During each monitoring period, active outside ventilation was curtailed so that the various chemicals could be collected without continuous dilution. In practice, many of the units (day care center and school) were recycling internal air and had outside make-up air held to a minimum. For all the residential units, the volume of air sampled exceeded the actual volume of air inside the unit, therefore, additional sampling would not have collected additional constituents. Calculations were then performed (Appendix L) to account for this re-sampling. For the larger units (e.g., locations with larger internal volumes than the actual air sampled) this volume correction was not required and had the sampling continued for a longer period, we would have needed to perform volume calculations on these samples also. In order to find residential units for monitoring, Base Housing solicited
		volunteers from the various apartment units. Finding individuals who would volunteer their apartments was a difficult challenge each monitoring quarter. Lifestyle issues were not considered in apartment or townhouse selection. Specifically, however, smoking was not an issue as smoking was banned in all buildings on the base, including individual apartments and living quarters.
		In addition to finding residents to volunteer for the study, there were many logistical considerations and limitations to be considered. For instance, the school cafeteria could only be sampled when students were not present (after 14:00), the Ground Electronics Maintenance Building could only be monitored when Navy staff were present (7:00 to 16:00), the day care center could only be monitored when the center was vacant, and the residential units (apartments and townhouses) could only be sampled when the

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#		residents were not home between the hours of 7:00 and 17:00, at a mimimum. NEHC has included a discussion of the effects of lifestyle factors in the report. The effects were higher concentrations of chemicals indoors than outdoors, due to interfering factors, such as building materials, furnishings and life style factors, e.g. smoking, cooking, cleaning, pets, etc. except for the chemicals that were associated by correlation analysis with the incinerator.
Page 6	NEHC should also discuss other behavioral patterns that could affect the risk assessment; such as the proportion of time residents at NAF Atsugi spend indoors versus outdoors.	There are literally, a myriad of different risks that can be calculated for different activity patterns, simulating real-world exposures. For example, one could be at the elementary school for part of the day and at the high rise for the rest of the day; be at ground electronics building for part of the day and the high rise for the rest of the day; be at the golf course on weekends and work at the elementary school the rest of the week and live in the high rise, etc. This was pointed out in the draft NEHC report on page 61. The air pathway drives at least 85% of the risk at each of the five locations. Therefore to assist in determining if there would be a significant difference for individuals being on base 24 hours per day, regardless of when monitoring was conducted, we calculated the risk related to the air pathway for a 24-hour exposure at each of the 5 locations monitored. The results indicated that there was no significant difference or in some cases no difference at all in the risk, no matter where you are on base; therefore, the risk calculated at the high rise should be considered the plausible upper bound risk for individuals spending 24 hours on base, no matter the location in which those hours were spent.

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	Risk Communication	
Page 6	There does not seem to have been a coordinated strategy for risk communication. Such a strategy should have been developed with objectives that are among the overall objectives of the project.	Appendix B of the draft NEHC report (now Appendix F in the revised NEHC report) is a Risk Communication Plan, which was developed to address stakeholders' concerns regarding potential health impacts caused by the poor air quality at NAF Atsugi. The Comprehensive Risk Communication and Health Consultation Plan for Naval Air Facility Atsugi, September 1998 was written at the direction of Dr. Bernard Rostker, Assistant Secretary of the Navy (ASN) for Manpower and Reserve Affairs (M&R). Dr. Rostker directed BUMED to take the lead in developing the plan, in April 1998. BUMED extensively coordinated the plan with Commander in Chief, U.S. Pacific Fleet; Commander Naval Forces Japan; Naval Air Facility Atsugi; Branch Medical Clinic Atsugi and Bureau of Naval Personnel. Dr. Vincent Covello, Center for Risk Communication, New York, New York, validated the plan. The purpose of the plan is to set forth implementing procedures to provide formal risk communication to everyone on-board NAF Atsugi and personnel with orders to NAF Atsugi. It also sets forth implementing procedures to conduct mandatory health consultations for high-risk individuals assigned to NAF Atsugi. This plan is designed to provide the best possible and most comprehensive health risk communication and health consultation available so as to allow our Navy personnel and their families to make personal and informed choices. This plan will remain in effect until the NAF Atsugi health issues are resolved.
Page 7	The risk-assessment project also seems to lack a fundamental understanding of stakeholders' needs and concerns and a clear process that could be used to update and improve risk communication.	Prior to developing the risk communication plan, a public availability session was held which addressed stakeholder concerns. Additionally the base established a Shinkampo Action Team that was comprised of NAF Atsugi personnel and community members to establish an open dialogue with community members. The Branch Medical Clinic in Atsugi was also

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		very involved in addressing concerns of the community and they participated in the Shinkampo Action team meetings. Furthermore the health studies conducted (Appendices E and F of the revised NEHC report) were in direct response to the community.
	DATA ANALYSIS	
	Attributable Risk	
Page 7	The attributable risk has not been adequately evaluated. Pioneer Technologies Corporation listed five ways "to identify the potential impact of emissions from the SIC [Shinkampo Incinerator Complex]" (Pioneer 2000; p. 82): 1. Comparing the risks due to ambient air when the SIC is ON to when the SIC is OFF (Radian, 2000).	As acknowledged by the subcommittee in their summary of their peer review comments on page 2, the NEHC "report is a summary of more detailed reports prepared by Pioneer Technologies Corporation, Radian International, and other contractors, which performed the sampling and risk assessment."
	2. Comparing the risks due to ambient air when the SIC is ON, and a site is downwind of the SIC, to when the SIC is OFF (Radian, 2000). 3. Comparing the risks due to ambient air when the SIC is ON, and a site is downwind of the SIC, to when the site is not downwind of the SIC (Radian, 2000). 4. Using the results of the correlation analysis to model	Lengthy and complete discussions on the first three approaches are provided in the Radian Air Monitoring Summary Report (Radian 2000). Due to their lengths they were not included in the Pioneer or the NEHC report, but they were referenced as to where the reader could find these discussions. A discussion on all alternatives on how they do/do not meet the objective of the risk assessment has been added to the NEHC report.
	concentrations, and subsequently calculate and compare risks, at sites when the site is downwind of the SIC and when the site is not downwind of the SIC. 5. Comparing the risks due to ambient air when the SIC is ON and a site is downwind of the SIC to another site which is upwind of the SIC on the same days (i.e., an "Upwind" versus "Downwind" evaluation). Pioneer (2000) states that those approaches are discussed in various places in the report, but the subcommittee could not find, in any of the documents provided, an adequate evaluation of whether any of the	Alternatives 1 and 2 were not selected because there generally was no relationship between concentration and percent downwind for the SIC OFF background scenario for the six key chemicals that were found to be related to the SIC. This same lack of correlation is true for most of the other chemicals as well. In the few cases where there was a significant correlation when the incinerator was OFF, the relationship was not consistent across sites, and the concentrations when the incinerator was OFF were well within the range of typical concentrations (i.e., there are no cases where the most extreme concentrations occur when the incinerator is OFF).

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#	approaches met the objectives of the risk assessment.	Alternative 3 was not selected because the average concentrations under this background scenario (SIC ON and the site is not downwind from the SIC) were more variable during this scenario than when it was OFF. One potential explanation is that the SIC was only OFF on Sundays, the same day that most other industries in the area also were closed. Although it is possible that the increase in variability could be due to SIC effects, it is more likely that the increase is due to other industries in the area. From a risk assessment perspective, alternative 4 is not a good method because it only accounts for six chemicals that were identified in the correlation analysis as exhibiting a statistically significant correlation between concentration and percent downwind of the SIC. Experience from risk assessments performed on municipal waste incinerators performed in the United States indicates that multiple chemicals are being emitted from the SIC (EPA 1998a). Using the correlation analysis approach to quantify the contribution of the incinerator to the health risks would result in an underestimate of the SIC's contribution especially for non-cancer health effects since a potentially large number of chemicals are unaccounted for. Cancer risks would also be underestimated; however, the level of underestimation is not expected to be significant because Dioxin-TEQ, which typically dominate the cancer risks, is one of the six chemicals that correlate with % downwind of the SIC. Also, as detailed in the Radian 2000 Report [Section 2.5; p. 32 - 33], there are many site-specific factors (e.g., the variable composition of municipal waste and emissions from multiple point and non-point sources) that should be considered when evaluating the results of the correlation analysis method.
		quantify the contribution of emissions from the SIC to the risk estimates

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		because it accounts for chemicals present in air that may be attributable to the SIC but were not identified, as such, in the correlation analysis. This approach that is complementary to the correlation analysis approach was needed because of the limitations of the correlation analysis.
Page 7	Two methods—of the Research Triangle Institute (RTI 1999), and Radian (2000d)—that are similar to each other were used to determine the contribution of the incinerator facility by correlating the concentrations of various pollutants measured in the air samples with the fraction of the sampling period during which the measurement site was downwind of the incinerator (that is, the "percent downwind"). The Research Triangle Institute method (RTI 1999) also used a nonparametric correlation analysis to estimate the impact of the incinerator facility. Neither analysis was interpreted quantitatively, nor were the two methods' results compared.	Correlation analysis definitely is a quantitative analysis method (as opposed to just constructing plots and drawing conclusions based on visual inspection, for instance). RTI tested the significance of the results using statistical significance tests, which also are quantitative, and evaluated the assumptions behind the various calculations. It is incorrect to call this a "qualitative" analysis. We assume what the reviewer is calling qualitative is the fact that we did not use the correlation/regression results to construct prediction models or to estimate concentrations that could be used as input into a risk assessment. In all of the discussion/interpretation, we say the results either indicate a significant relationship between chemical concentrations and an opportunity to be impacted by the SIC or do not indicate a significant relationship. We typically did not quantify that relationship (e.g., we didn't say that a 10% increase in the time a site is downwind of the SIC corresponds to a concentration of X). That really isn't necessary because the interpretation of the results was entirely consistent with the goals of the data evaluation to assess whether there was an empirical relationship between the observed concentration and the opportunity to have been impacted by the SIC. It is unclear what additional quantification of the results in the interpretation would have added.
Page 7	The possibility of "sector-sampling" (switching pumps on and off as the wind direction changes) is discussed but dismissed as impractical and unnecessary for VOCs in the Field Sampling Plan (Radian 1998a,	Sector sampling devices collect samples only when the winds are from a pre-defined wind direction sector. To accomplish this task, each piece of monitoring equipment would require a direct link to a meteorological

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#	p. 4-13). The subcommittee believes, however, that "sector-sampling", or some similar method, might well be necessary to evaluate the incinerator contribution to exposure.	station, a data logger, and switching equipment. A program would also be required to turn each piece of equipment on and off and determine the period of time the wind would need to be in or out of the wind sector before the equipment was turned on or off.
		The only way comparable data could have been gathered during this program using a sector sampling approach would have been to use sector sampling for all sample types. To conduct sector sampling for the nine different sample media that were collected during the Atsugi project would have been a massive undertaking requiring an enormous amount of methods development and a substantial additional outlay of resources. As resources were not unlimited during this project, using more conventional and accepted methodology was warranted as the budget could not have supported the methods development required to implement a sector sampling approach for 9 different sample media.
		There are additional logistical constraints with using a sector sampling approach. The level of detection for each sampling method is based on the analytical detection limit and the sample volume (e.g., the volume of air collected during a sampling run). If a sector sampling approach were to have been employed, it is possible that the sample duration required for some of the methods to collect sufficient sample volume to achieve adequate detection limits could have taken days or even weeks. While this may have resulted in a more "focused" sample, the logistics of leaving sorbent media exposed for days or weeks would have produced passive deposition on the samplers and would have resulted in drastically increased
		blank concentrations for many of the techniques. If a defined sample period had been used (e.g., samples were run for a fixed time period regardless of sample volume), it is likely that some samples would not have collected sufficient sample volumes to achieve desired detection limits while other

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The state of the s		samples may have collected too much sample volume and experienced break-through. Trying to use sector sampling (e.g., motors being frequently turned on and off) with high volume air sampling techniques would also have placed significant stress on the induction motors used in the samplers. This would have resulted in a high rate of sampler failure and consequently reduced data capture rates. We believe that the approach of categorizing samples, based on the percentage of time the sampler was downwind of the incinerator, is a valid and useful approach. It is our opinion that a sector sampling approach would have resulted in reduced data capture and a much smaller data set of qualified data.
Page 8	The models used in the correlation analyses are not justified in either the RTI report (1999) or the Radian report (2000a). The correlation analyses consisted principally of fitting a straight line to the relation between percent downwind and the measured concentration or its logarithm, or between rescaled versions of those variables. Such relationships have no physical basis, so it is difficult to interpret the results.	We concur that a simple linear model with a single explanatory variable ("percent downwind" or a transformed version of this variable) does not provide a complete physical model for concentration. Other factors such as wind speed, temperature, and downwash certainly also affect the concentration at a given site on a given day. However, percent downwind is a reasonable surrogate for the degree to which a site may have been affected by the incinerator on a given day. The objective of the correlation analysis was to understand whether observed concentrations are related to the degree to which a site is potentially affected by the incinerator and <i>not</i> to create an atmospheric dispersion model. The models included in the Radian report successfully meet the objective. We do not concur that the results are difficult to interpret. A significant positive relationship indicates that concentrations tend to be higher when the location is downwind of the facility than when it is not, and that the concentrations increase as the

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#		percent of time a site is downwind of the incinerator increases.
Page 8	As is often the case with environmental measurements, the air-concentration data appear to be approximately lognormally distributed; the distribution should be taken into account in the statistical analysis. Either a least-squares approach to a nonlinear physical model (if the data are log-transformed) or a statistical approach that can account for nonnormal errors should be used.	Three statistical approaches applying correlation and regression analysis were used to determine which chemicals are associated with the SIC, one method used by Radian and two other methods used by RTI. Regarding the method used by Radian, we agree that the distribution of the data should be taken into account in the statistical analysis, and the distribution was taken into account. Appendix C of the Radian report provides an explanation of the assumptions behind the calculations presented in the report, one of which is that the regression residuals are normally distributed. As stated in the report, the distribution of the residuals was evaluated, and the specific form of the correlation was tailored to the outcome of that evaluation. For each analyte, three correlation analyses were run: one using untransformed concentrations, one using log-transformed concentrations, and one based on the ranks of the concentrations. The results presented in the report correspond to the model whose residuals were most consistent with normality. If neither the untransformed-concentration residuals nor the log-transformed-concentration residuals were normally distributed, then a nonparametric correlation based on the ranks of the data was used. In regard to the RTI statistical approaches, we disagree in two main respects with the statement that the air-concentration data appear to be lognormal and that this distribution should be taken into account in the analyses. (1) In fitting a statistical regression model that allows the mean concentration level to vary as a function of independent variables (e.g., site, percent downwind, day), the concentration data are not required to have any particular distribution; rather, it is the residuals from the model that may need to follow some specified distribution (e.g., normal or lognormal) in order to justify optimality of the estimation method and/or the validity of
		tests of significance for the model parameters. We believe that measurement

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π		errors in concentration measurements do tend to increase with concentration level, so that log-scaled concentrations are more likely to exhibit the homogeneous variances assumed in the regression estimation. Hence, we did utilize log-scaled concentrations in our mixed-model analyses. (2) An analysis that relies on the fact that data (or residuals) follow a particular distribution is generally more powerful than a nonparametric approach when the data do follow that distribution; when there are deviations from the assumption, however, such an analysis can produce inaccurate estimates and an analysis that does not rely on such an assumption is preferred. Hence, in addition to the regression methods, we utilized a nonparametric correlation approach that is not tied to any particular assumption about the underlying distributional form. We regard the fact that, in this analysis, we do not have to make any assumption about the underlying distribution as a strength, not a weakness, of the approach. Thus we did utilize "a statistical approach that can account for nonnormal errors."
Page 8	Pioneer (2000) selected what it called the upwind-versus-downwind approach, but no documentation provides the exact method used, and neither the method nor whether the designations of "upwind" and "downwind" are representative of overall exposures could be determined from the available documentation. The approach uses unverified and possibly unjustified assumptions, and the logic behind the selection of sites for comparison is not clear. The ground-electronics maintenance building (GEMB) is fairly obvious as the site most affected by the incinerator complex, but no rationale is provided for selecting the golf course as the upwind comparison site. The selection of the golf course is particularly surprising in that the "criteria site" - a site southeast of the incinerator facility and considered upwind of the incinerator – was original selected as the upwind site and designated as the background site in the sampling	The PIONEER and the NEHC Reports have been revised to clarify the upwind versus downwind comparison. Both the Golf Course and Criteria site are considered "upwind" of the SIC when the wind is blowing from the south to the north (as was the case with the data selected for this analysis). The Golf Course site was selected for the Upwind vs. Downwind comparison because it is closer to the SIC than the Criteria Site and would potentially be impacted by emission sources that are located between the Criteria Site and the SIC. If the Criteria Site was used in the comparison, then the risks attributable to the SIC could potentially be overestimated because the Criteria Site is not impacted by emission sources located between it and the SIC.

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	plan. The Pioneer document (Pioneer 2000; p. 8) provides the following rationale for ignoring the criteria site: The criteria site is located southeast of the SIC. No workers, residents, or recreational users are located at this site. Therefore, it was not evaluated in the risk assessment. That rationale, however, only addresses the use of the criteria site for the evaluation of the total health risk at NAF Atsugi. It does not address the use of the criteria site for evaluating the contribution of the incinerator.	
Page 8	The calculated risk estimates raise the related question of whether the incinerator contributions are important compared with variations among sites that could be caused by sources other than the incinerator. The large difference between "average" and "RME" estimates in Tables 5-2 and 5-10 of the Pioneer report (Pioneer 2000) suggests that the available data might not be sufficient to show important differences among sites and that the differences could be due to random variation. The subcommittee recommends that NEHC investigate whether there are statistically significant differences in risk estimates among the various sites.	By definition the RME estimate is the maximum exposure that is reasonably expected to occur at a site. Because of the uncertainty associated with any estimate of exposure concentration the upper confidence limit (i.e., the 95 percent upper confidence limit) on the arithmetic average is used for this variable. Large differences between the average and the RME are irrelevant to the subcommittee's statement that the large difference between "average" and "RME" estimates in Tables 5-2 and 5-10 of the Pioneer report (Pioneer 2000) suggests that the available data might not be sufficient to show important differences among sites and that the differences could be due to random variation. An investigation on whether there are statistically significant differences in risk estimates among the various sites may be interesting; however, it would require an extensive effort and the results of this investigation would not change the overall results of the risk assessment.
Page 8	The subcommittee believes that the dispersion analysis, in conjunction with the correlation analyses, provides the best approach for determining the contribution of the incinerator facility to pollution	This recommendation is not clear for the following reasons: (1) The dispersion modeling approach determined that only 6 of the 336 chemicals monitored showed a strong correlation with the SIC. In addition, since

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#	at NAF Atsugi. Although the model has some limitations, the subcommittee recommends that the dispersion modeling and correlation analyses, not an upwind-downwind comparison, be used to determine the risk attributable to the incinerator facility.	dispersion modeling was performed using ambient air concentrations to estimate stack emissions, the use of dispersion modeling to predict the contribution of the SIC would only yield a circular logic (2) Associated magnitudes of error are likely to be high because of unknown SIC operation parameters and waste composition data (3) The emission rate estimation analysis varied 2-4 orders of magnitude. The NRC does not address the additional inherently large uncertainty with the use of dispersion modeling although other uncertainties are very clearly addressed. The approach used by NEHC was actually the approach recommended by the previous Committee on Toxicology that reviewed the 1998 screening risk assessment and stated the following: "Another approach that might be useful for getting a rough estimate of the contribution of incinerator emissions to ambient air, relative to the background, would be to compare results from Location 1 (upwind site) with those from downwind locations on days when the wind direction is out of the south-southwest and relatively constant." The use of the upwind-downwind approach has also been encouraged by the EPA reviewers NEHC sent a written request to the subcommittee for instructions on how to use the dispersion modeling approach used by Radian to determine the
		contribution of the incinerator complex to the health risks at NAF Atsugi. The subcommittee chose not to reply to our request for instructions in writing, but in a phone conference held 10 April 2001, that included only two members of the. During the phone conference, the two reviewers withdrew the recommendation to use dispersion modeling and correlation
		analyses to determine the risk attributable to the incinerator facility. They

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#		suggested yet another new approach but did not articulate instructions on the new approach. Additionally they indicated that the new approach would be an extensive statistical effort, which may not provide any useful information. As this recommendation has now been rescinded and the subcommittee admits that new approach includes just as much uncertainty as the NEHC approach, NEHC chose to retain the upwind-downwind approach that was initially presented to the subcommittee for review
Page 9	The Pioneer risk-assessment report (Pioneer 2000) provides four reasons why ambient air concentrations were used as surrogates for indoor air concentrations (see p. 13), but they do not provide an adequate rationale for the substitution in light of the stated objectives for the overall risk assessment (Pioneer 2000; p. 1):	NEHC has edited the report to clarify the objectives of the overall risk assessment. As acknowledged by the subcommittee in this peer review, initially when the sampling plan was developed, the purpose of the indoor air samples was to provide exposure estimates for NAF Atsugi residents and dependents that would be used in the risk assessment. Another purpose of the indoor air sampling was to evaluate indoor air quality at Atsugi by comparing it with indoor air quality in the U.S. However, since the objective of the risk assessment for indoor air was to calculate risk due to ambient air sources, including the SIC, the ambient air concentrations had to be used as surrogates for indoor air concentrations in the risk assessment because: 1) Concentrations for the majority of the constituents exceeding RBCs were found to be higher indoors than outdoors indicating probable indoor air sources (e.g., insulation, carpets, and household chemicals). If we are trying to determine the risk due to ambient air infiltrating indoors, using indoor air samples that also measure contaminants generated by indoor sources would overestimate the impact of the SIC and other ambient air point and non-point sources 2) Passive ventilation systems are used at most locations which make attempts to quantify the contribution of risk attributable to emissions from

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#		the ambient air sources highly uncertain. If we are trying to determine the risk due to ambient air infiltrating indoors, using indoor air samples that also measure contaminants generated by indoor sources would overestimate the impact of the SIC and other ambient air point and non-point sources. 3) Ambient air is the source of constituents in indoor air that are associated with emissions from the SIC and other ambient air sources. If we are trying to determine the risk due to ambient air infiltrating indoors, using indoor air samples that also measure contaminants generated by indoor sources would overestimate the impact of the SIC and other ambient air point and non-point sources.
Page 10	The first reason provided for the substitution of outdoor air measurements for indoor air concentrations is: 1. The objective of collecting the indoor air samples stated in the Sampling and QA/QC Plan to Assess Health Risks Related to Air Quality at NAF Atsugi, Japan (Radian, 1998) was to make a comparison between concentrations found indoors in the United States with concentrations found at NAF Atsugi. The indoor air samples were not intended to determine the contribution of [emissions] from the SIC to concentrations of constituents in indoor air. The subcommittee, however, is unable to find any indication in the final sampling plan (Radian 1998a) that the objective of the indoor-air sampling was to compare indoor-air concentrations at Atsugi with	NEHC disagrees with the subcommittee that comparing risks at Atsugi and in Los Angeles was interpreted as the entire objective. To avoid misunderstanding of the indoor air sampling purpose this reason has been deleted from the list of reasons why surrogates were used in calculating indoor air risk. NEHC's lack of clarity in stating the objectives of the HRA has led the subcommittee to disagree with NEHC's approach regarding the use of outdoor surrogate concentrations to evaluate indoor air risk due to chemicals likely associated with ambient air emissions infiltrating indoors. In the revised NEHC report, NEHC has clarified the objectives to ensure consistency between the NEHC report and all supporting documents. The overall objectives have been clarified as follows: This project was designed to collect data to meet the objectives of the
	indoor-air concentrations in the United States. The indoor-air data- quality objectives clearly are based on the requirement to estimate risks associated with indoor inhalation, as is seen in Section 3.2.1 (Radian 1998a):	comprehensive health risk assessment which were: 1. Estimate the potential human health risks to U.S. Navy personnel and their families and other individuals living and working on NAF Atsugi, Japan resulting from exposure to constituents of concern (CoCs) in soil, ambient air, indoor air, and indoor dust. This

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	The question to be addressed by the indoor air sampling effort is: 1. What is the inhalation exposure risk for sensitive receptors in buildings likely to be impacted by the Jinkanpo Incineration Complex? That that was an objective is reinforced by a response to a comment on the sampling plan (Radian International, unpublished data, January 27, 2000): "The purpose of the indoor air monitoring is to provide exposure estimates for Atsugi residents and dependents that will be used in the risk assessment. These exposure estimates should be representative of a normal or typical total exposure. Although the incinerator emissions are expected to have an adverse effect on indoor air quality, it is not the intent of this project to measure or estimate the differential risk due to the operation of the incinerator. Rather, the goal is to estimate the total risk due to indoor chemical exposures at Atsugi, e.g., is there more risk to live at Atsugi vs. Los Angeles. "The example given in that comment-comparing risk at Atsugi and in Los Angeles- seems to have been interpreted as the entire objective. A sampling plan designed to compare indoor air at Atsugi and the United States, however, would not be designed to collect data suitable for the overall objectives of the project. In addition, what NEHC means by "normal or typical total exposure" should be explained.	estimate focuses solely on CoCs that are likely to be associated with ambient air emissions and/or subsequent deposition from point and non-point sources impacting the air quality at NAF Atsugi. 2. The contribution of the risk attributable to the Shinkampo Incineration Complex (SIC). Therefore to meet the objectives of the HRA, indoor air sampling was conducted to answer the following HRA question: What are the risks to receptors from inhalation of indoor air and dermal contact or incidental ingestion of dust contaminated by CoCs infiltrating indoors that are likely to be associated with ambient air emissions and/or subsequent deposition from point and non-point sources impacting the air quality at NAF Atsugi? As indicated by the subcommittee, the words "normal or typical total exposure" came from an unpublished response to a NEHC comment on the draft sampling plan (Radian International, unpublished data, January 27, 2000). These words are not in any of the reports. The Radian staff responding to the comment used these words to mean "representative exposure".
Page 10	The second stated reason for the substitution of outdoor air measurements for indoor air concentrations is: Concentrations for the majority of the constituents exceeding RBSCs [risk-based screening concentrations] were higher indoors than outdoors indicating probable indoor air sources (e.g., insulation, carpets, and household chemicals). Using indoor-air samples that also measure contaminants generated by indoor sources would not affect the use of those samples to	NEHC agrees with the subcommittee's observation that using indoor-air samples that also measure contaminants generated by indoor sources would not affect the use of those samples to estimate the potential health risks to people living at NAF Atsugi. However, as clarified in the revised report, our objective is to determine the indoor air risk due to chemicals likely associated with ambient air emissions infiltrating indoors. Having clarified this objective, it is clear that these samples might overestimate the impact of the incinerator unless the contributions of sources unrelated to the

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	estimate the potential health risks to people living at NAF Atsugi, although it might overestimate the impact of the incinerator unless the contributions of sources unrelated to the incinerator can be removed from the exposure estimate.	incinerator can be removed from the exposure estimate, as recognized by the subcommittee on page 10 of their peer review document.
Page 10	The third stated reason [for the substitution of outdoor air measurements for indoor air concentrations] is: Passive ventilation systems are used at most locations which make attempts to quantify the contribution of risk attributable to emissions from the SIC highly uncertain. The use of passive-ventilation systems does not preclude the use of those samples for estimating the overall health risks at NAF Atsugi.	As clarified in the revised report, our objective is to determine the indoor air risk due to chemicals likely associated with ambient air emissions infiltrating indoors. The use of passive ventilation (no outside air is forced indoors), will preclude the use of those samples for estimating indoor air risk due to chemicals likely associated with ambient air emissions infiltrating indoors.
Page 10	The fourth stated reason [for the substitution of outdoor air measurements for indoor air concentrations] is: Ambient air is the source of constituents in indoor air that are associated with emissions from the SIC. Although the emissions might be the source of the indoor contaminants, that does not preclude the use of indoor-air measurements to estimate the human health risks at NAF Atsugi.	As clarified in the revised report, our objective is to determine the indoor air risk due to chemicals likely associated with ambient air emissions infiltrating indoors. The use of indoor-air measurements to estimate the human health risks at NAF Atsugi due to ambient air chemicals infiltrating indoors might overestimate the impact of the incinerator unless the contributions of sources unrelated to the incinerator can be removed from the exposure estimate, as recognized by the subcommittee on page 10 of their peer review document.
Page 10	When comparing the concentrations of contaminants in indoor air at NAF Atsugi with the concentrations in US homes (NEHC 2000; p. 22, and Table 2.5, pp. 25-26), it is not stated whether the status of doors and windows was recorded during indoor sampling.	Unfortunately this information was not available from the studies.
Page 10	More complete information is essential, [on HVAC] especially if NEHC is trying to justify the use of outdoor-air concentrations as a	More information has been added to the NEHC report discussing the types of HVAC in each building sampled for indoor air.

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surrogate for indoor concentrations in its risk assessment.	
Use of Data from Final 2 Months of Sampling	
The subcommittee is concerned about potential biases in the monitoring because of the change from the original sampling plan to directed sampling (that is, when NAF Atsugi was predicted to be downwind of the incinerator complex) during the last 2 months. That change was not adequately justified and was not accounted for in the analysis.	Since no stack sampling was permitted to be conducted on this foreign owned incinerator, one of the greatest challenges in this project was to meet the second objective, i.e., to determine the SIC contribution to the health risk. One method we used to identify the chemicals in air that are emitted from the SIC was to correlate wind direction, specifically the percentage of time an individual monitoring site was downwind of the SIC, to the chemical concentrations observed in ambient air at that site. The hypothesis is that, for chemicals that are emitted from the SIC, the chemical concentration (and also risk) increases as the percent of time the wind blows emissions from the SIC onto the base increases. In the case of variable wind speed and direction, from one week to the next, if the emissions are constant but the wind speed and direction are not the same, the correlation with percent downwind will be different. The confidence in the correlation of wind direction versus concentration is related to the number of observations that are used to calculate the correlation coefficient and the wind directions that are observed. The rationale for a two-month extension of the ambient air sampling program at NAF Atsugi is summarized below: 1. An analysis of the wind patterns observed during the 1998 sampling program indicates the winds were atypical for the period. Specifically, when compared to historical meteorological data, there were fewer periods of southerly winds, which carry emissions from the Shinkampo Incineration Complex (SIC) onto the base. May and June historically had significant periods of southerly winds, and extending sampling
	surrogate for indoor concentrations in its risk assessment. Use of Data from Final 2 Months of Sampling The subcommittee is concerned about potential biases in the monitoring because of the change from the original sampling plan to directed sampling (that is, when NAF Atsugi was predicted to be downwind of the incinerator complex) during the last 2 months. That change was not adequately justified and was not accounted for in the

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		of historical conditions for evaluation in the risk assessment (if the winds during the extension period are consistent with the historical wind patterns). In other words, during the 1998 ambient air sampling study, there were few periods of southerly winds, even fewer than that observed by historical wind roses. As a result, there were relatively few data points to correlate concentrations/percent downwind in an effort to assess SIC contribution. Therefore, sampling was extended for an additional 2 months and samples were collected on days predicted to be downwind days (i.e., toward NAF Atsugi from the SIC) to complete the correlation plots. The extended sampling included 6 additional sampling days when the wind was blowing toward the base from the SIC to complete the correlation plots for a better representation of historical conditions. Since this could result in an underestimation of long-term exposure conditions and consequently risk, the additional number of sampling days needed to provide additional information for the correlation analysis plots was determined by statistical analysis so that no bias would result by overweighing particular wind directions and overestimating the contribution from the SIC.
		2. The health risks calculated based on the 1998 sampling program data are potentially much lower than would be calculated if the winds during the sampling period had been consistent with historical data. This adversely impacts the human health risk assessment because the fundamental assumption of the risk calculations, and any conclusions and recommendations made based on the risk assessment, is that the ambient air concentrations accurately reflect the long-term exposure conditions at NAF Atsugi. In this case, the current ambient air concentrations may underestimate the long-term exposure conditions, and consequently risk, because there were very few periods of southerly winds during the sampling period and therefore few opportunities for

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		emissions from the SIC to have impacted NAF Atsugi. NEHC has revised the report to include this information on a subsection on the departure from the Sampling and QA/QC plan.
Page 11	Another change made to the monitoring program during this period was a short-term mercury study using different sampling and analytical methods.	Since mercury was detected in the ambient samples at concentrations lower than expected, an additional sampling and analytical method that could detect mercuric chloride in addition to elemental mercury was employed for a short-term study to assess the potential for underestimating the ambient mercury concentrations. Results for the additional monitoring efforts are discussed in the Radian air monitoring summary report. NEHC has revised the summary report to include this information.
Page 11	For estimating mean exposures at NAF Atsugi, the inclusion of the final 2 months of samples could bias the results by overweighing particular wind directions and probably, if the targeting was successful, overestimating the contribution of the incinerator complex.	The inclusion of the final 2 months of sampling does not bias the results by overweighing particular wind directions. In fact it helps to account for the seasonal change in wind direction, and to provide the best estimate of long-term exposure conditions, because of the atypical wind conditions found during the 12 months sampling period.
		An analysis of over 12 years of meteorological data indicated that the predominant wind direction at NAF Atsugi during the summer months (i.e., May – August) is significantly different from the rest of the year. Specifically, the predominant wind direction during the summer months is from the south. The predominant wind direction during the other months of the year is from the north.
		The seasonal changes in wind direction have a significant impact on human health because the SIC is located south of the base. Therefore, the maximum risk to human health associated with exposure to emissions from the SIC is when the wind is from the south (i.e., when emissions from the

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		human health the wind is fi blown away wind direction conditions, a comparison of during the 19 there was a 2 compared to southerly win	n associated with rom the north (i. from the base). on, and to provide 12-month samp of the historical 198 sampling ev 21% decrease in the 12 years of 1 ands greatly increase wo primary objective.	n exposure to en e., when emission In order to accord the the best estimaling period was wind patterns we ents. During the southerly winds historical data.	s (for the SSE-SI The marked dec ainty of any resu mpling program.	e SIC is when C are being onal change in a exposure e 1 presents a terns observed y – August 1998 E vectors) when orease in alts presented to
		Based on a	a Comparison o	of 1998 Samplin and Patterns for	ng Events and t the Same Mon	
			May	June	July	August
		Vector	%	%	%	%
		GGE GE	Difference	Difference	Difference	Difference
		SSE-SE	-26.6%	-31.5%	29.0%	-53.3%
		S	-25.8%	-29.9%	-17.4%	-10.9%
		SSE-SSW	-11.8%	-17.5%	-14.8%	-21.7%
		SSW-SW	-5.4%	26.9%	-33.5%	-40.5%
		SE-SW SSE = South	-18.0%	-13.1%	-14.5% = South; SSW	-27.6%
			SW = SouthWe	,	5 – Bouin, BBW	– Soum-
		Risks calcula	ated based on 12	months data co	llected in 1998 i	may not

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		accurately characterize the long-term health risks associated with ambient
		air at NAF Atsugi, particularly at the Child Development Center, Elementary School, and the Residential Towers, which are locations
		receiving SSE-SE winds. In other words, the risks may be lower than if the
		winds had been consistent with the historical data (i.e., individuals would be
		exposed to emissions from the SIC less often because the wind did not blow
		emissions from the SIC onto the base). This adversely impacts the human
		health risk assessment because any conclusions or recommendations made
		based on the risk assessment may not be representative of historical conditions and could underestimate the actual risks.
		conditions and could underestimate the actual risks.
		The decrease in southerly winds also makes it difficult to quantify the
		contribution of health risk attributed to emissions from the SIC. In order to
		quantify the contribution of risk attributed to emissions from the SIC, the
		chemicals in air that are emitted from the SIC must be identified. The
		method for trying to identify the chemicals in air that are emitted from the
		SIC is to correlate wind direction, specifically the percentage of time an
		individual monitoring site was downwind of the SIC, to the chemical concentrations observed in ambient air at the site. The hypothesis is that,
		for chemicals that are emitted from the SIC, the chemical concentration
		(and also risk) increases as the percent of time the wind blows emissions
		from the SIC onto the base increases. The confidence in the correlation of
		wind direction versus concentration is related to the number of observations
		that are used to calculate the correlation coefficient and the wind directions
		that are observed. For the 1998 ambient air data there were few periods of
		southerly winds to use in the correlation analysis and, therefore, the
		confidence in the results of the analysis is less than if the winds had been consistent with the historical average. This is particularly true at the Child
		Development Center, Elementary School, and the Residential Towers.
		Results for the GEMB suggest that there is a relationship between the

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		concentrations of several chemicals and the wind direction. However, the Child Development Center, and the Elementary School, and the Residential Towers have not been downwind of the SIC often enough to confirm that a similar relationship exists there. If the 12 months sampling had not been extended the result would be that:
		Fewer chemicals would be identified as having a positive correlation with percent downwind of the SIC and concentration.
		2. The confidence in the quantifying the risk attributed to chemicals emitted from the SIC is reduced
	Soil Trend Analyses	
Page 11	Pioneer concluded that no spatial trends for arsenic or BaP were found in the Thiessen Polygon (also called Voronoi diagram) analysis. That conclusion for arsenic appears to be based on the apparent randomness of the values in Figure 4. Closer examination of the figure, however, indicates two possible arsenic-contamination areas: one in the southern area close to the incinerator and one in the northeast area. As shown in Figure D-2, zones along the southern border of NAF Atsugi 100 m from the incinerator at their highest points were estimated to have arsenic concentrations in the highest range (6.7–14.7 mg/kg) for the soil layers 0-3 in deep (0-7.6 cm deep) and 3-12 in deep (7.6-30.5 cm deep). Those zones are most frequently downwind of the incinerator. Also, the second-highest concentration range (4-6.7 mg/kg) fans out from a west-northwest direction to the northeast direction. The samples from the surface layer, which appear to have been collected in the Tade River valley north of the incinerator, had arsenic concentrations of 0.43-4 mg/kg. Those concentrations are lower than those of the samples on both sides of the valley. That pattern could be the result of erosion in the	NEHC is unclear on reviewer reference to "Figure 4". In order to address the subcommittee's comment we must assume that the reviewer means "northwest" instead of "northeast" for the direction of one of the areas with relatively elevated arsenic concentrations. The report documenting the soil results in question, Results of March 1998 Soil Sampling, NAF Atsugi (Radian International, 1998), and which was used by Pioneer to support the Human Health Risk Assessment, states that "arsenic was found at elevated levels near the SIC, but was also found at similar levels in other portions of the base. It appears that the SIC could have affected surface and subsurface soil with respect to arsenic. However, other sources of arsenic appear to be present in other portions of the base" (pg. 4-46). This "other portions of the base" is especially true in the northwest direction from the SIC, near the base boundary (i.e., quite a distance, and over less-affected intervening area). Additionally, the arsenic isoconcentration contouring performed in support of this earlier report showed a small, relatively elevated area of arsenic in surface and subsurface soils a short distance north/northeast from the SIC, and a larger area in the

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# # # # # # # # # # # # # # # # # # #	river-valley slopes. In addition, the 3- to 12-in. (7.6 to 30.5-cm) soil-layer map shows the highest arsenic concentrations just north of the incinerator. Those results are consistent with the suggestion that arsenic from the incinerator plume has deposited at NAF Atsugi. However, no objective way to confirm or deny those trends is presented. The possibility of deposition of arsenic should be investigated further and the presence or absence of such a trend should be discussed in the conclusions.	northwestern portion of the base. Arsenic appears to be naturally elevated across the entire base had arsenic concentrations well above the respective RBCs. The Pioneer report used different interpretive approaches, including the Thiessan Polygon and semi-variogram plots. The Thiessan Polygon approach yielded a similar arsenic distribution pattern to the previous Radian isoconcentration contouring, again showing the highest concentration areas to the immediate north/northeast (close) and northwest (distant) from the SIC. Concerning the reviewer hypothesis about areas immediately north of the incinerator possibly having lower values because of erosion associated with the Tade River valley, the four sample locations responsible for the lower-concentration pattern in this area are some distance from the Tade River. In fact, the more-westerly samples are nearer the river and exhibit relatively higher arsenic concentrations. Also, as stated in Appendix D, pg. 4, "a mathematical model describing the correlation of concentration and distance from the SIC could not be fitted to the semi-variogram of the arsenic data (normal or log transformed). This means that the arsenic data do not exhibit a spatial correlations between concentration and distance." Therefore, based on the various interpretive approaches, although there do appear to be relatively elevated arsenic concentrations in at least one location immediately north of the SIC, there does not appear to be a wide-spread deposition from the SIC similar to other analytes (e.g., total 2,3,78-TCDD TEQs). The merits of additional investigation into arsenic deposition should be weighed heavily against the findings that: 1) the
		known area of elevated arsenic concentrations immediately north/northeast of the SIC is not near identified areas of concern, 2) this small area of elevated arsenic concentrations is bounded by sample points with lower

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		concentrations (i.e., is already reasonably defined).
Page 12	It is unclear why no data from the Radian phase II soil sampling report (Radian 1999 d) were used in the risk assessment. The data from the Radian phase II soil-sampling report should be used in the risk assessment before such conclusions are made.	As indicated on page 15, section 2.3.1 of the Pioneer report, the Radian Phase II soil sampling was conducted to identify a suitable site specific background soil site which was used to compare with soil concentrations from areas of concern to determine the analytes that should be retained for further evaluation in the risk assessment.
	Missing Toxicity Values	
Page 12	It is inaccurate to characterize all those chemicals as having "no toxicity information". Primary literature and many useful secondary sources should be consulted for toxicity information that could be used in some cases to determine whether exposures to those chemicals at Atsugi might be of concern.	Although the subcommittee later questioned the need for this recommendation during our telephone conference of 10 April 2001 when we requested clarification to their recommendations, NEHC searched the scientific literature for toxicity information to derive screening toxicity values and determine whether exposure to those chemicals at NAF Atsugi might be of concern. The 86 chemicals with no toxicity values were comprehensively researched and analyzed for potential toxicity. An exhaustive search of all available scientific peer-reviewed databases was conducted for applicable toxicological information, including the following book: Gold L. S. and E. Zeiger, Editors. 1997. Handbook of Carcinogenic Potency and Genotoxicity Databases. Boca Raton, FL: CRC Press (also http://potency.berkeley.edu/cpdb.html). The sources of toxicological information consulted, the details of the methodology used for deriving toxicity values for a subset of the 86 chemicals, and the results and conclusions from this analysis are presented in Appendix B and is entitled "NAF Atsugi Toxicological Evaluation".
Page 12	Alternatively, a default cancer slope factor could be used in a Sensitivity Analysis to assess the impact of including in the risk assessment any of the 86 chemicals rated as potential carcinogens on the basis of weight of evidence. For instance, Caldwell et al (1998)	Pioneer identified all chemicals for which toxicity values were available and unavailable. Using a default cancer slope factor to develop a Sensitivity Analysis to assess the impact of including any of the 86 chemicals rated as potential carcinogens on the basis of the weight of evidence may be an

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	used the cancer slope factor of methylene chloride, 7.5 x 10 –3 (kg-day)/mg as a screening value	interesting academic project. Unfortunately funds are not available to develop an exercise in which we question its efficacy because of inherent uncertainties, and that in the end, may not be approved by the scientific community such as the NRC and EPA upon review.
	INTERPRETATION OF RISK ASSESSMENT RESULTS	
	Limitations of the Superfund Methodology	
Page 13	Careful consideration should be given to the characteristics of the exposures that are being estimated and to the derivation of the toxicity values with which the exposure estimates are compared for both the risk assessment itself and the communication of those risks. Estimates of exposure should be presented with ranges or confidence intervals. Appendix A of the NEHC report contains a comparison of the risk-assessment results with the levels at which acute health effects were seen, according to the results of a literature search "to determine potential acute health effects for the specific 24-hour concentrations measured at NAF, Atsugi" (p. 65). The comparison might seem to satisfy the need for an evaluation of acute, possibly reversible effects, but the concentrations measured at NAF Atsugi are compared with toxicity values that were compiled for various purposes and with various protocols. The minimal risk levels (MRLs) and reference exposure levels (RELs), for example, are similar to lowest-observed-effect levels. A discussion of how the assumptions and adjustment factors used in the derivation of toxicity values affect the risk assessment should be included in this type of comparison.	In the revised NEHC report we revised Appendix A (now Appendix B) to compare maximum 24-hr concentrations measured at NAF Atsugi with 24-hr studies and/or ATSDR acute (1-14 days) MRLs.
Page 13	Some of the California RELs presented in Appendix A are different from the May 2000 values posted at	The May 2000 California RELs had not yet been posted at the time the Pioneer health risk assessment and NEHC summary drafts reports were
	http://www.oehha.org/air/acute_rels/allAcRELs.html.	completed in January 2000. The updated values have been added to the

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		final NEHC report.
Page 13	Those RELs are based on 1 or 6 hr of exposure and are not appropriate for direct comparison with the "specific 24-hour concentrations measured at NAF Atsugi."	It was not feasible to obtain hourly sampling results required for 1 or 6 hr of exposure by conventional air monitoring because they would be insufficient to achieve detection limits below the risk-based concentrations required for the health risk assessment. Air concentrations were too low to be detected by real time monitoring instruments for a short-term reading. Appendix A (revised as Appendix C) presents not only ATSDR Maximum Risk Levels (MRLs), but also all data that was found in the literature regarding air concentration levels for all chemicals detected at NAF Atsugi. This includes various acute health effect levels for varying periods of time with descriptions of the corresponding effects. In most cases these are occupational levels at much greater concentrations than those measured at Atsugi; however, they are included in Appendix C for comparison purposes with the much lower MRLs. It was more appropriate to compare the maximum 24-hour concentrations to the acute MRLs, since they too are based upon 24-hour exposure time. However, since it was not feasible to obtain 1-hour readings we also compared maximum 24-hour air concentrations of all chemicals with available RELs (based on 1 or 6 hr exposures) and MRLs (based on 1-day to 14 days exposures) to evaluate acute health effects. Although NEHC acknowledges that data qualification needs to be made regarding this comparison, NEHC believes that it is still a useful comparison in the absence of 1-hour data.
Page 13	Similarly, the comparison with intermediate MRLs is not valid, because a 1-day exposure at or near the intermediate MRL is not necessarily a cause of concern.	As stated in the footnote on Table 4-5 of the NEHC draft report, NEHC acknowledges that intermediate MRLs are for 15-364 days of exposure. NEHC also acknowledged in the draft report that since the intermediate MRL applies to 15-364 days of exposure that one out of 70 sampling days does not present a concern. NEHC disagrees with the subcommittee that the comparison with intermediate MRL is not valid. Intermediate MRLs were

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		used for comparison in the event that concentrations above MRLs were identified in more than 15 days of exposure such as in the case of acrolein where the intermediate MRL was exceeded in all 216 sampling days.
Page 14	Because of those concerns with the comparisons in Appendix A, the subcommittee recommends that Appendix A be substantially revised or removed from the NEHC document.	Appendix A has been revised to reflect a more appropriate comparison between estimated exposures at Atsugi and specified reference values. Compounds that do not pose an acute health risk have been screened out. Comparisons that did not allow this have a more detailed discussion of the conclusions that can be made considering the limitations of using toxicity values that were derived for a variety of purposes. Appendix A has been redesignated Appendix C in the revised Human Health Risk Assessment report.
Page 14	The exposure conditions at NAF Atsugi should be kept in mind when one is considering the toxicity of the chemicals present. For example, NEHC discusses the potential health effects of cadmium, but cadmium is a slowly accumulating toxicant that causes adverse effects only after chronic exposure	The subsection on health effects of various chemicals has been revised in the final NEHC report to account for the exposure conditions at NAF Atsugi.
Page 14	A more appropriate comparison would be between the concentrations of pollutants at NAF Atsugi and the point of departure (the level at which effects are seen, before the addition of uncertainty and modifying factors, in a critical study on which a toxicity value is based). Such a comparison would be similar to the margin-of-exposure analyses recommended by the Presidential/Congressional Commission (1997b). Concentrations of pollutants measured at NAF Atsugi higher than the point of departure would be a cause of concern. The health effects are not clear for concentrations at Atsugi above the RfD but below that point of departure; what action to take in response to such an exposure is the subject of a policy decision, and this should	The NEHC report has been revised to show comparison between the concentration of pollutants at NAF Atsugi and the levels at which acute effects are seen. For those compounds presenting a possible acute health risk, the concentration at NAF Atsugi has been compared to the concentration used in the critical study establishing the toxicity value for that compound, before the addition of uncertainty and modifying factors.

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π	be clearly stated.	
	Interpretation of Risk Values	
Page 14	NEHC's interpretation and discussion of risk estimates as though they are bright lines are therefore not appropriate.	The conclusions have been revised in the final NEHC report pointing out the uncertainties that prevent the risk estimates from being considered as brightlines. In addition, in the revised NEHC report, a specific subsection has been added to address policy based versus science-based approaches for evaluating health risk. A summary discussion on policy-based and science-based conclusions and has also been included in the conclusions section
Page 14	NEHC should clarify the nature and history of the regulatory use of 10^{-6} and 10^{-4} cancer risk estimates.	Information on the nature and history of the regulatory use of 10 ⁻⁶ and 10 ⁻⁴ cancer risk estimates have been included in the revised NEHC report.
Page 14	Similarly, 10 ⁻⁴ is not a "cancer risk benchmark", but a value rooted in regulatory decision-making as opposed to medical practice.	In the revised NEHC report, the conclusions regarding the risk have been clarified by distinguishing policy-based from science-based conclusions.
Page 14	Although it is beyond the scope of this subcommittee's task to recommend policy decisions, it does recommend that NEHC clearly differentiate in its report which decisions are based on science and which on policy.	Although risk management recommendations have been removed from the NEHC report, the conclusions regarding risk have been distinguished as those based on policy and those based on science.
	Uncertainty	
Page 15	The report should indicate the amount of uncertainty in the results, at least qualitatively, and should clarify what is meant by the "minimum degree of uncertainty." An adequate discussion of uncertainty should include the sources of uncertainty mentioned in Appendix C of this report.	The subsection on Uncertainty has been expanded in the NEHC revised report.
Page 15	The previous COT report (NRC 1995) made a similar comment on the	The statements have been revised in the final NEHC report.

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	need to convey the uncertainty in risk assessment:	
	"The Navy should be urged to be far more careful in its presentation	
	of risk results. Statements such as that on page 13 of the NEHC	
	(1995) report ("The total cancer risk for carcinogens of 6.62 x 10 ⁻³	
	means that there is an increased risk of 7 cancer cases per 1,000	
	people over the normal lifetime cancer risk rate") can be misleading.	
	Both cancer and noncancer risks estimated using the methods that	
	NEHC employed are not as certain as such statements imply. To be	
	credible, all risk estimates should be accompanied by descriptions of	
	the assumptions and uncertainties that are associated with them."	
	f	
	The present subcommittee reiterates that thought and finds statements	
	like the following (p. 2 of Public Health Summary, NEHC 2000)	
	regarding the risk-assessment results potentially misleading:	
	1-8 m and 1 m more and 1	
	• The calculated cancer risk for the child resident, less than 6 years of	
	age, indicates that the air quality at NAF Atsugi could result in as	
	much as 1.1 additional cancer cases in a population of 10,000 after an	
	exposure period of approximately 3 years.	
	• The cancer risk for the adult resident is calculated at 3.7 additional	
	cancer cases in a population of 100,000 after an exposure period of 3	
	years.	
	Those statements imply that after 3 years of exposure cancer could	
	occur (the correct statement requires pointing out that this is the	
	lifetime risk of cancer) and does not mention the substantial	
	uncertainty in the estimates (the correct statement requires pointing	
	out that this is an upper-bound risk estimate, with a lower bound of	
	zero). They also fail to discuss appropriately that the increase is small	

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"	relative to lifetime cancer risk.	
Page 15	NEHC should consider conducting sensitivity analyses to characterize the uncertainty in the risk assessment as recommended by the National Research Council (1994, 1996) and the <i>Presidential/-Congressional Commission</i> (1997b).	No funds are available to conduct a Sensitivity Analysis to characterize uncertainty, an analysis that ultimately may not change the results.
	NEHC's Risk-Reduction Recommendation	
Page 16	One major problem with NEHC's risk-reduction strategies is the lack of planning for or conducting of evaluations of those strategies. Furthermore, Appendix B of the NEHC report shows that some risk-reduction strategies have been implemented before peer review or any assessment of their potential effectiveness. That is not appropriate for a risk-management process.	Risk reduction strategies were previously regarded as acceptable by both of the NRC Committees on Toxicology that reviewed the 1995 and the 1998 screening Health Risk Assessments, without recommending that an effectiveness assessment be done. The risk reduction recommendations are common sense and good Public Health practices. Washing hands after playing in soil and toys that have been outside are preventive measures that can be taken to decrease the risk of exposure to children.
Page 16	Some of the risk-reduction strategies recommended by NEHC are not supported by the findings in the risk-assessment report. For example, the recommendation to continue to monitor health-status indicators is vague and should specify which indicators are to be monitored and why, when they are to be monitored, in whom they are to be monitored, and who will monitor them.	Risk reduction strategies were previously regarded as acceptable by both of the NRC Committees on Toxicology that reviewed the 1995 and the 1998 screening Health Risk Assessments, without recommending that an effectiveness assessment be done. The risk reduction recommendations are common sense and good Public Health practices.
Page 16	The text pertaining to risk reduction is oversimplified. It should discuss how plausible the actions are, whether they can be enforced, and families' compliance with recommendations.	The risk reduction strategies are options that families can take to reduce risk. Compliances with the strategies are strictly up to the individuals. These same recommendations are made everyday in occupational environments and there are no measures for enforcement. The Navy conducted an education campaign to inform parents and individuals on actions they can take to reduce exposure.

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Page 16	Washing of hands, forearms, face, tools, toys, and so on, after outdoor activities that result in direct contact with soil or dust is good advice and practice, but risk reduction by such measures has not been determined (Pioneer 2000; p. 93).	Risk reduction strategies were previously regarded as acceptable by both of the NRC Committees on Toxicology that reviewed the 1995 and the 1998 screening Health Risk Assessments, without recommending that an effectiveness assessment be done. As acknowledged by the subcommittee, the risk reduction recommendations are common sense and good Public Health practices. Washing hands after playing in soil and toys that have been outside are preventive measures that can be taken to decrease the risk of exposure to children.
	INFORMATION GAPS	
	Health Surveillance Data	
Page 16	The subcommittee is aware of studies conducted by Laurel A. May and David Sack at NAF Atsugi and focusing on adverse pregnancy outcomes (including spontaneous abortions) and children's health. Appendix B of the NEHC report mentions the existence of surveillance studies but their design and results should be mentioned in the main section of the NEHC report. The subcommittee recognizes that such surveillance is not a part of traditional risk assessments but believes that it could provide useful, complementary information. Health surveillance of personnel and their families residing at NAF Atsugi is useful for assessing all but chronic effects with latent periods in excess of the period of residence. Because of the rather small number of people at Atsugi, epidemiological methods could attribute only large increases in incidence above background to	While the Navy conducted the comprehensive environmental sampling study at NAF Atsugi, short-term health studies were also conducted to identify certain acute health conditions that either could be associated with exposure to poor air quality or were health conditions that concerned the NAF Atsugi community. One of the studies, the Children's Respiratory Health Effects Study, compares peak respiratory flow between children at Atsugi and Yokosuka. The results of this study showed that there were no significant differences in the respiratory health of children living on or off base at NAF Atsugi and those at Yokosuka. For the measured parameters, children at Atsugi and Yokosuka had values, which are associated with "better lung function" than that of the general population in the United States.
	living at NAF Atsugi. Risk assessment is capable of assessing lifetime risk at low levels. But surveillance of outcomes amenable to this approach can eliminate many of the uncertainties inherent in risk assessment. Therefore, the subcommittee recommends that NEHC use both approaches, especially where data are already available.	The second study, the Pregnancy Outcome Study, compares spontaneous abortion rates between Atsugi residents and residents of other bases in Japan. The results indicated that the Naval Hospital Yokosuka and NAF Atsugi miscarriage rates during the study period were both lower than the reported miscarriage rate for U. S. women with known pregnancies. This

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		rate is between 10% - 15%. Since the military population is generally younger and healthier than that of the U. S. civilian population, we would expect their percentage of pregnancy loss to be lower than the population norms.
		A medical surveillance on, air pollution related morbidity, compared rates of skin conditions and respiratory symptoms seen at the NAF Atsugi Branch Medical Clinic and Naval Hospital Yokosuka. This medical surveillance has not yet been completed. However, preliminary results show that no significant differences in air quality related morbidity between the adult populations at Atsugi and Yokosuka measured during the surveillance period exist, and that no significant differences in air quality related morbidity between child (below 18 years of age) populations at Atsugi and Yokosuka during this period exist.
Page 17	An evaluation of the health risks that looks at acute, possibly reversible effects, as well as potential chronic effects, could also be useful.	The draft NEHC report did present an evaluation of acute health effects. A discussion on potential chronic health effects has been added to the revised report in the subsection on the health effects of various chemicals that contribute the majority of carcinogenic and non-carcinogenic risks.
Page 17	Stakeholders' concerns should be taken into account when designing surveillance programs.	During a public availability session held in 1997, the NAF Atsugi community voiced concerns, which resulted in developing the epidemiological studies. Medical Surveillance programs have been developed for incoming, current and outgoing military and civilian personnel and their families, such as health pre-screening prior to arriving at NAF Atsugi, development of database specific to document health complaints related to the incinerator such as respiratory conditions and health consultations prior to leaving NAF Atsugi.

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Page 17	NEHC should consider studying the potential health effects of peak contaminant concentrations (for example, correlating contaminant concentrations with anecdotal subjective reports) to assess irritation and other short-term reversible but unpleasant effects.	Children's health studies attempted to correlate potential health effects of peak contaminant concentrations with children's respiratory symptoms complaints, but no clear relationship between wind direction and the levels of gases and dust particles could be identified. The wind was blowing toward the school for only a short period of time during the four-week study period.
Page 17	Surveillance data could be used in conjunction with risk assessment, perhaps focusing the risk assessment on end points of particular interest.	While the Navy conducted the comprehensive environmental sampling study at NAF Atsugi, health studies were also conducted to identify certain acute health conditions that either could be associated with exposure to poor air quality or were health conditions that concerned the NAF Atsugi community. One of the studies, the Children's Respiratory Health Effects Study, compared peak respiratory flow between children at Atsugi and Yokosuka. The second, the Pregnancy Outcome Study, compared spontaneous abortion rates between Atsugi residents and residents of other bases in Japan. An ongoing medical surveillance on air pollution related morbidity is comparing rates of skin conditions and respiratory symptoms seen at the NAF Atsugi Branch Medical Clinic and Naval Hospital Yokosuka. Naval Base Yokosuka served as the control location for the studies for two specific reasons. First, Yokosuka, which is approximately 25 kilometers from Atsugi, is also located on Japan's Kanto Plain. Its population, climate, and vegetation are similar to that at Atsugi. In addition, other than the highly visible point source of pollution at Atsugi i.e., the Shinkampo Incinerator Complex, sources of air quality degradation are similar. Secondly, Yokosuka is the site of the Navy's primary medical treatment facility in Japan, Naval Hospital Yokosuka, which provides access to several healthcare databases.

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#		The study on Respiratory Effects in Children had two primary goals: 1. Identify differences in respiratory symptoms and lung function between children who live or go to school at NAF Atsugi and similar children at Yokosuka. 2. Determine if there were more respiratory symptoms in children who live or go to school at NAF Atsugi on days when they are exposed to higher levels of pollutants from the Shinkampo Incinerator during the four week study period (7 May-5 June 1998).
		The study focused on children since their health is a major concern of the NAF Atsugi residents. Children's lungs also tend to be more sensitive to the effects of air pollution. Fifth and sixth grade students at Atsugi and Yokosuka DOD Schools were eligible to participate. One hundred twenty-seven (127) students volunteered for the study. Eighty (80) of the students lived on base at NAF Atsugi, 17 lived off base at NAF Atsugi and 30 lived at Yokosuka.
		The children's lung function was tested each school day during lunchtime. Children recorded the number of hours spent outdoors as well as respiratory and/or air quality related symptoms such as, trouble breathing, coughing during the day or night, feeling bad, runny nose, cold, headache, and irritated eyes. A daily symptom score was given to each child based on the information recorded.
		Data from ambient air monitoring at Shirley Lanham School was also collected for PM10, nitrogen dioxide and sulfur dioxide, known to cause respiratory effects. Wind direction and wind speed were also recorded, in an attempt to associate health effects with environmental pollution conditions. The primary findings of this study were: 1. There were no differences in the respiratory health of children living on

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		or off base at NAF Atsugi and those at Yokosuka. 2. Children living on base at Atsugi reported more runny noses than the Yokosuka children did. 3. All other reports of symptoms were similar. 4. There was no difference in the reported number of colds between the Atsugi on base and the Yokosuka groups. Children living off base at Atsugi did report more colds. 5. Most of the children in the study group had lung function better than that of the general population in the United States. 6. The wind was blowing toward the school for only a short period of time during the four-week study period. As a result, no clear relationship between wind direction and the levels of gases and dust particles could be identified.
		Complete information on this study can be found in the report Air Pollution From the Shinkampo Incinerator Associated with Adverse Respiratory Effects Among Children at NAF Atsugi Study (NEHC Jul 99).
		The study on Pregnancy Outcomes was conducted because many residents expressed concerns during the November 1997 NAF Atsugi Public Meeting about the health effects the Shinkampo Incinerator may be having on their families regarding miscarriages. Therefore the study was designed to describe the rate of miscarriage, at NAF Atsugi and other naval facilities in Japan. Information for the study was gathered by looking at hospital and clinic records for past pregnancies. This was a retrospective study where only documented miscarriages versus live births were considered.
		The study population consisted of Navy personnel or their dependents who were pregnant at some point between June 1995 and May 1998 and lived on or near NAF Atsugi or other naval facilities in Japan serviced by Naval Hospital Yokosuka (NHY). Information used to calculate the miscarriage

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		rates came from three different sources, Delivery Logs and Pathology
		records at NHY and the Prenatal Log at the Atsugi Branch Medical Clinic
		Data collection took place during the summer of 1998. It included the number of live births and the number of miscarriages. The total number of pregnancies with known outcomes during the study period was 1862. For the purposes of this study, a miscarriage was defined as an unintentional pregnancy loss at up to the 28 th week of pregnancy. Multiple births were excluded from the analysis. The miscarriage rate was defined as the number of miscarriages divided by the total number of pregnancies examined (the number of babies born plus the number of miscarriages).
		The findings of the study were: 1. The overall miscarriage rate for patients with known pregnancies from Atsugi, Yokosuka, Iwakuni and Sasebo between June 1995 and May 1998 was 7.1%. This rate was determined by review of the delivery log and pathology records at NHY. When the Atsugi patients are subtracted, the miscarriage rate for the other areas is 7.8%. 2. Review of the NAF Atsugi Branch Clinic prenatal log, during the same period, indicates a miscarriage rate at NAF Atsugi, of 8.8%. However, the data used in this study came from different sources and contain some different information. Therefore, the miscarriage rate at NAF Atsugi cannot be directly compared to that of the other payal facilities that were part of
		be directly compared to that of the other naval facilities that were part of this study population. 3. The NHY and NAF Atsugi miscarriage rates during the study period
		were both lower than the documented rate of miscarriage for women in the United States, who know they are pregnant, which is between 10% - 15%.
		4. This study was conducted with the limited information that was available
		in various records. The results suggested that the risk of miscarriage at
		NAF Atsugi and other naval facilities within Japan are at the low end of the

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#		expected risk range described for the population of the United States. Complete information on this study can be found in the report Pregnancy Loss at NAF Atsugi Japan (June 1995-May 1998) (NEHC Sep 99). A preliminary review of the data collected during medical surveillance on air pollution morbidity period indicated the following: 1. There were no significant differences in air quality related morbidity between the adult populations at Atsugi and Yokosuka during the study period. There were no significant differences in air quality related morbidity between the Child (below 18 years of age) populations at Atsugi and Yokosuka during the study period. 2. There was a peak period of respiratory disease complaints at Atsugi from June –August 1998. This is an artifact of the comprehensive risk communication and health consultation program that was at its height during that period. 3. There was a peak period of respiratory disease complaints at Yokosuka beginning in November 1998 and persisting through January 1999. This represents an outbreak of Japan Type A Influenza during that period. This study, Prospective Analysis of Specific Respiratory Diagnosis Between Atsugi and Yokosuka, is still in progress.
Page 17	A surveillance program could also be helpful in risk-management decisions and in risk communication.	A surveillance program would be warranted if the studies mentioned above had indicated significant differences in health of the populations being compared in the studies.
	Indoor Dust	
Page 17	The Radian report indicates that the purpose of dust monitoring was to "evaluate the potential for an ingestion route of exposure due to	Dioxins and furans were selected as indicators as to whether contaminants from the SIC were infiltrating or being tracked into homes and therefore to

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#	deposited or "tracked-in" dust, surface sampling will be performed once at each of the seven indoor sites whose air will be tested." The NEHC report, however, does not connect the observed results to that stated purpose.	evaluate the potential for an ingestion route of exposure due to deposited or tracked-in dust. The indoor dust and wipe samples were analyzed only for dioxins and furans to maximize the collection of information within the available resources, since they were the chemicals that were most likely to only be related to a burning operation versus other chemicals that could be related to household cleaning, smoking, food odors etc. Given that dioxin concentrations in indoor carpet/floor dust and in the dust on indoor surfaces were available they were used to evaluate dermal/ingestion risk due to deposited or "tracked in dust" in conjunction with soil surrogate concentrations to estimate dermal/ingestion risk for other chemicals infiltrating or being tracked into homes. This information has been added to the NEHC report in the Indoor Air Subsection of Section 2.
Page 17	However, the dust samples were collected by a vacuum method (Micro-Sciences 1999; p. 12) that samples larger particles (greater than 5 µm), so the composition of smaller particles, not larger ones, might be at issue. If the quoted passage was meant to indicate that combustion products can be found in particles smaller than 5 µm (that is was written incorrectly)—and an appreciable fraction of the PM ₁₀ samples would be of such smaller particles—then a method that collected the particles of the desired sizes should have been used.	To clarify any confusion the text has been revised as follows: The vacuum method actually collects particles smaller than 5 μ m. Since no combustion particles were found in this fraction a microscopical analysis was performed on PM ₁₀ filters that contained particles larger than 5 μ m. The results of the analysis of the PM ₁₀ filters indicated major amounts of charred carbonized fragments that are indicative of a combustion source such as an incinerator. The PM ₁₀ samples also contained a significant level of fine (sub micrometer-size) carbon particles that are characteristic of vehicle exhaust (e.g. diesel exhaust).
Page 18	The dust samples were analyzed for dioxins and furans, but background apartment concentrations of dioxins and furans are not provided, so the measurements are difficult to interpret.	Background apartment concentrations should be zero or negligible considering that any indoor air concentrations of dioxins and furans would have originated from the ambient air, not from indoor sources.
Page 18	The dust samples should have been analyzed for heavy metals because dust and wipe samples typically are good indicators of air and soil pathways for heavy metals (such as, lead, cadmium, and arsenic).	The subcommittee did not offer an explanation as to why soil is a poor surrogate for indoor dust, but states that "The dust samples should have been analyzed for heavy metals because dust and wipe samples typically are

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"	Metals are common constituents of incinerator emissions, and metals can be cheaply and reliably analyzed. Furthermore, the average concentrations of all metals except selenium measured in the soil samples seemed to be higher at the residential towers than at the elementary school or the Child Development Center (see Table 2-6, NEHC 2000); that indicates that the soil might be contaminated by metals. Soil is a poor surrogate for indoor dust, however, and it is important to determine whether the dust is contaminated by metals.	good indicators of air and soil pathways for heavy metals (such as lead, cadmium, and arsenic)." If dust samples are good indicators of air (airborne dust) and soil pathways (tracked in dust) for heavy metals, it is reasonable to use soil as a surrogate for indoor dust as a conservative assumption that will be protective of human health in a health risk assessment. Naturally there are uncertainties associated with this assumption, which were already addressed in the uncertainty section of the Pioneer report and included in the revised version of the NEHC report.
Page 18	In particular, dust samples should have been monitored for lead. Although relatively low lead concentrations were found in the soil, indoor sampling for lead is needed because of the presence of a potential emission source nearby, because lead- contaminated dust is difficult to remove, and because dust is a main source of indoor lead exposure in children up to 4 years old (Manton et al. 2000). Experience at other sites has demonstrated that indoor-dust lead will slowly accumulate if there is a continuous emission source. Table 2-6 (NEHC 2000), which reports data on soil up to 3 in. deep (7.6 cm deep), suggests that lead and other metals might be slowly accumulating around the residential apartment buildings or towers. The average and the RME lead concentrations in soil of that depth are apparently higher at the residential buildings than at the elementary school. Therefore, lead concentrations in dust in the residential buildings could be high.	The indoor dust and wipe samples were analyzed only for dioxins and furans to maximize the collection of information within the available resources, since they were the chemicals that were most likely to only be related to a burning operation versus other chemicals that could be related to household cleaning, smoking, food odors etc. Given that dioxin concentrations in indoor carpet/floor dust and in the dust on indoor surfaces were available they were used to evaluate dermal/ingestion risk due to deposited or "tracked in dust" in conjunction with soil surrogate concentrations to estimate dermal/ingestion risk for other chemicals infiltrating or being tracked into homes. Therefore, although dust samples were not analyzed for lead, the lead risk was still evaluated in indoor air. In addition, as it was stated in page 69 of the NEHC report: "The site-wide RME concentrations of lead in soil and air for NAF Atsugi (i.e., 26.5 mg/kg and 0.39 ug/m³, respectively) were evaluated using the IEUBK model to determine the potential for health effects associated with exposure to lead. The results of the modeling effort indicated that there is a 0.02% probability of a blood lead level of 10 μg/dL at NAF Atsugi for children. This value is well below the Centers for Disease Control target action level of greater-than-5 % probability. Of the 372 children tested under the Pediatric Lead

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		Poisoning Prevention Program (PLPP) at Atsugi between 1995 – 1999, one child (over 6 years old) who lived on base, was found to have a blood lead between $10-19~\mu g/dL$ in 1997. But, upon confirmatory blood lead determination the child was found to have a blood lead level of less than 10 ug/dl.
		Although Table 2-6 may indicate that the lead average and RME concentration in surface soil at the Residential Towers are higher than at the Child Development Center, and Elementary School (28.39 mg/kg versus 13.17 mg/kg and 12.81 mg/kg respectively for average concentrations; 83.55 mg/kg versus 18 mg/kg and 44.14 mg/kg for RME) this does not necessarily indicate that lead and other metal might be slowly accumulating around the Residential Towers. Dioxins also originates from a continuous combustion emission source such as the incinerator and yet as indicated in Table 2-6, the average and RME concentrations for dioxins TEQ for the Residential Towers are the same as in the Elementary School.
	Other Data Gaps	
Page 18	NEHC states (pp. 4-5) that it could not conduct monitoring off NAF Atsugi, and the Pioneer report (Pioneer 2000; p. 15) states that site-specific background concentrations could not be evaluated, even in ambient air, because all sites on NAF Atsugi were affected by the incinerator complex. No other reason is stated for the failure to evaluate background concentrations. The NEHC report should clearly and specifically describe why off-site monitoring was not possible, even if the reasons are legal or political.	Lengthy discussions on the estimation of background concentrations for ambient air are presented in the Radian Air Monitoring Summary Report, which was submitted to the subcommittee for review. This analysis is fully presented in section 2.6 pages 39-46. These are the reasons why they were not presented in the NEHC and Pioneer reports. Many site-specific factors make the task of separating analytes originating from the SIC from those in background ambient air challenging. For example, NAF Atsugi is located in a heavily industrialized area proximate to multiple point and non-point sources of airborne contaminants. Furthermore, Japan's primary mechanism for disposing of waste is incineration, which results in higher background concentrations of many airborne contaminants such as particulates and dioxins. Meteorological conditions such as low percentage

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		of downwind days makes the task of defining upwind conditions and downwind conditions difficult, whether one is trying to separate upwind days from downwind days for a given location, or trying to define upwind locations and downwind locations on a given day. Offsite monitoring was not possible because not only equipment security regarding vandalism or sabotage could not be guaranteed, but sampling on foreign soil could jeopardize political relations between the U.S. and Japan. As stated in the Pioneer report, background soil concentrations from Phase II soil sampling were used in the soil risk assessment.
Page 18	In addition to US guidelines, NEHC should state what, if any, Japanese standards apply and should provide adequate comparisons with them.	There are few Japanese standards that apply. The only Japanese standards available are those similar to the U.S. NAAQS. There is no specific standard for ozone, however there is a standard for photochemical oxidants, which is lower than the NAQQS standard for ozone. Although there is a standard for solid particulate matter there are no standards for PM ₁₀ and PM _{2.5} . The only Japanese standards that are directly comparable with the U.S. standards are the 8-hour average standard for carbon monoxide, which is higher than the U.S. standard, and the 24-hr average standard for sulfur dioxide, which is lower than the U.S. standard. This information has been included in the revised NEHC report. Since the U.S. Government has raised concerns with the SIC, recently in the past couple of years Japan has promulgated a Dioxin TEQ guideline that ambient air concentrations in Japan may not exceed 0.6 pg/m³. Japan has also recently adopted a standard for dioxin TEQ in soil of 1000 ppt, which is the same concentration that drives cleanup in the U.S.
Page 18	The 2,3,7,8-tetrachlorodibenzo- <i>p</i> -dioxin (TCDD) toxicity equivalence factors for polychlorinated dibenzodioxins and furans are values agreed on by the scientific community through consensus reports. The latest such consensus report (Van den Berg et al. 1998) updated	The health risk for dioxins and therefore the total risk has been recalculated using the new World Health Organization toxicity equivalence factors recommended by the subcommittee for 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) toxicity equivalence factors for polychlorinated dibenzodioxins

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	those values, and NEHC should use the latest values in its risk assessment	and furans.
	SUGGESTED IMPROVEMENTS IN THE PRESENTATION AND ORGANIZATION OF THE NEHC REPORT	
Page 18	The NEHC report lacks a framework that would allow readers to examine what was done, why it was done, and what the results were. The report should begin with a clear statement of the purpose and objectives of the risk assessment.	The NEHC report was developed for the risk managers, with a Public Health Summary section in the beginning of the report for the community who are composed of the military and civilian personnel and their families at NAF Atsugi. This Public Health Summary addressed in simple terms what was done, why it was done and what the bottom line results were. Regarding the purpose and objectives of the risk assessment, more text has been added to the objectives previously stated in all reports (Radian, Pioneer and NEHC) to support better understanding of the purpose and the objectives of the risk assessment.
Page 19	The methods, assumptions, and limitations of the project and its results should be described more thoroughly.	The NEHC report has been extensively revised to include more information on the methods, assumptions, and limitations of the project.
Page 19	The rationales for using or not using particular monitoring techniques and methods and particular analyses are also essential.	NEHC has not included this information in the NEHC report because there aren't many more particular monitoring techniques, methods and particular analysis that are appropriate which could have been used for meeting the objectives of the health risk assessment. The NEHC report does not describe rationale for using the particular monitoring techniques that were used because NEHC believes that the risk managers for whom the report was written, are mostly interested in the results of the risk assessment, rather than rationales for using a particular monitoring technique or speculations on what other techniques could have been used. Most study reports focus on what was done rather than the myriad of things that could have been done.

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Page 19	The NEHC report seems to be a summary of the project, but the audience for the report is not clear. The report is too technical for lay readers but does not provide adequate details for a risk assessor to understand and evaluate the project—planning, sampling, measurement results, or the risk assessment itself. The expected audience of the report should be indicated, and the report should be geared toward that audience.	NEHC has revised the report to emphasize that the NEHC report indeed is a summary of the project, and the audience is the risk managers, who are mainly interested in the results of the health risk assessment and recommendations for risk reduction. The NEHC report was not written for risk assessors. Adequate details for a risk assessor to understand and evaluate the project—planning, sampling, measurement results, or the risk assessment itself are contained in the supporting documentation, i.e. the Pioneer and the Radian reports, which were submitted to the subcommittee for peer review.
Page 19	The NEHC report does not include enough details of the incinerator facility and NAF Atsugi. A paragraph explaining the Enviro-Tech incinerator complex should be added, including information on the number of bypass stacks present and the potential for fugitive emissions from waste and ash handling.	NEHC has revised Section 1 of the NEHC report to include more details of the incinerator facility and NAF Atsugi.
Page 19	The report lacks adequate citations, and it is difficult to evaluate some statements without them. References to specific sections or pages in the extensive supporting documents should be included.	The NEHC report has been extensively revised to include, citations and references to specific sections or pages in the Pioneer and Radian reports.
Page 19	Furthermore, the NEHC risk assessment is based on EPA methods, but the NEHC report and the report by Pioneer Technologies Corporation (Pioneer 2000) do not reference current EPA methods for assessing risks associated with indirect exposure to emissions from combustion (EPA 1998a). Similarly, the EPA Region VI incinerator risk-assessment protocol (EPA 1998b) is not cited or referenced.	Evaluation of risks associated with indirect exposure to emissions from combustion is out of the scope of this health risk assessment project.
Page 19	The subcommittee recommends that the NEHC report be professionally edited. Abbreviations should be spelled out the first	A list of abbreviations and acronyms was included in the draft NEHC report as well as in the Pioneer and Radian reports. Abbreviations were spelled

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	time they are used, and there should be a list of them.	out the first time they were used, and included in a list just before section 1 of the NEHC report.
Page 19	The subcommittee noted some instances of inadequate paraphrasing in the NEHC report of technical discussions and conclusions in the supporting documents. Direct quotation of the wording of contractor reports would be preferable, particularly for technically precise statements. Appendix D presents specific sections of the soil-trend analysis discussion that the subcommittee recommends be quoted directly.	In the revised NEHC report the entire text from supporting documents has been quoted on technical discussions and conclusions instead of paraphrasing.
Page 19	Outdoor air samples appear to have been collected mainly for a period of 24 h, although it is difficult to be sure, because Table 4-5 of the <i>Final Monitoring Summary</i> (Radian 2000a) contains an incomplete summary of the sample periods. It is not clear why some samples were collected for more or less than 24 h or why there was a deviation in some cases in measuring from midnight to midnight.	Five regular monitoring runs (Radian Final Monitoring Summary, page 2-3) were conducted during each of the first 12 months of the monitoring program. These "regular" sampling runs were conducted from midnight to midnight. These five runs were always conducted during periods when the incinerator was in operation. The only exception to the midnight-to-midnight sampling protocol occurred during the first six weeks of the monitoring program when some of the dioxin samples were collected for more than 24-hours to ensure that sufficient sample volumes were collected to achieve the desired detection limits. This approach was described on page 4-12 of the Quality Assurance Project Plan (QAPP). Once the laboratory data became available and it was determined that a 24-hour sample was sufficient to achieve desired detection limits, additional dioxin sampling runs were not conducted. In addition to the five "regular" sampling runs conducted each month, an additional sampling run was conducted each month during periods when the incinerator was not operating. These runs were commenced when the incinerator ceased operation, normally at 1600 hours on Sunday, and ran until the incinerator resumed operations on Monday, normally at 1600

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		hours. These times were approximate and were adjusted based on actual incinerator operation. Occasionally this schedule was shifted to a Monday shutdown and Tuesday resumption. Since all "incinerator off" sampling runs were manually initiated, if the incinerator did not shut down, then sample initiation was postponed until the incinerator did shut down. A description of the sampling approach is given in the Radian Final Report (Radian 2000a) on page 2-3. The techniques used would not produce a biased data set.
Page 19	Some essential information (such as sample times and comparison of contaminant concentrations when the incinerator was operating with and without bypass stacks) was not included in the available reports. NEHC should ensure that such information is included in the risk-assessment documents.	NEHC chose not to include information on sample times and comparison of contaminant concentrations when the incinerator was operating with and without bypass stacks in the report. Obtaining this information for the 24 hours of sampling would have been impossible. We were determining the risk based on operating conditions. Considering that the SIC was operated by a foreign entity, operational records that contained this information were not available and the effort involved in reviewing one year of tapes of the plume would not add anything to the risk assessment; particularly since we had no control over the SIC operations.
Page 19	If readers of the report are expected to be varied, it would be helpful to explain the "wind rose" better, that is, that it shows the direction from which the wind is coming, not the direction toward which the wind is going.	An explanation on how to read the wind rose has been included in Section 1 of the revised NEHC report.
Page 19	The PM _{2.5} concentration exceeds the National Ambient Air Quality Standard (NAAQS) and should be discussed.	The possible health effects of PM2.5 exceeding the U.S. health-based standard is discussed in the subsection of the revised report regarding health effects of various chemicals of the revised NEHC report.
	RESPONSIVENESS TO PREVIOUS NATIONAL RESEARCH COUNCIL COMMENTS	

Page	NAS Comment	NEHC Response
Page 20	NEHC partially responded to the following problems identified in the previous risk assessments but has not fully resolved them. In the previous report (NEHC 1998), NEHC assumed, without appropriate supporting data, that the concentrations measured at the background site were substantially affected by emissions from the incinerator complex. Better use of meteorologic data to define upwind sites (Radian 2000a, b) has addressed that issue in this risk assessment; however, as discussed in the section on attributable risk, the criteria used to determine the background site are still not clear.	NEHC has addressed the problems identified in the previous screening risk assessments, regarding upwind/background site. In this risk assessment the upwind site addressed in this risk assessment, i.e. the criteria site, was selected based on historical windroses, which indicate the prevalent wind direction from the incinerator toward the base. However, an upwind site selected according to the prevalent wind does not necessarily represent background. As acknowledged by the subcommittee, "because 24-hr sampling was used, a site might be downwind for part of a sampling period and upwind for the remainder of the period." Background is defined as a concentration level that would represent the conditions that could be expected if the SIC did not exist. Background does not mean "pristine" or "unimpacted, but background concentrations reflect anthropogenic sources of airborne contaminants that are located proximate and remote from the SIC. Therefore, to evaluate the SIC attributable risk, meteorological data was used to select the background site (upwind) as one with low percentage of downwind hours, which was compared to a site with a high percentage of downwind hours (downwind) on the same day at the same time. The difference in risk between the upwind and the downwind site indicated the SIC attributable risk.
Page 20	COT previously recommended that continuous or semicontinuous monitoring methods be used to correlate meteorologic data and emission-dispersion estimates with ambient concentrations of pollutants (NRC 1998). Some FTIR monitoring was done (Radian 2000a). The limitations of that monitoring are discussed in the section on attributable risk.	NEHC has addressed the problems identified in the previous screening risk assessments regarding correlation between air sampling and meteorological data. As presented in the draft NEHC report continuous monitoring was conducted to evaluate Criteria Pollutants and twenty-four hour air sampling was used to correlate ambient air concentrations with associated meteorologic data. As acknowledged by the subcommittee, the FTIR was used also as a continuous monitoring device.
Page 20	COT previously indicated that the 6 weeks of sampling is not representative of long-term exposure, and a 12-month sampling	NEHC has addressed sampling that is representative of long-term exposure by sampling over 12 months. The reasons for extending sampling beyond

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	period was recommended (NRC 1998). For the current risk	12 months are explained as follows. Since no stack sampling was permitted
	assessment, NEHC performed a 14-month study. The implications of	to be conducted on this foreign owned incinerator one of the greatest
	a 14-month, rather than a 12-month, sampling period are discussed	challenges in this project was to determine the SIC contribution to the
	earlier in this report.	health risk. One method we used to identify the chemicals in air that are
		emitted from the SIC was to correlate wind direction, specifically the
		percentage of time an individual monitoring site was downwind of the SIC,
		to the chemical concentrations observed in ambient air at the site. The
		hypothesis is that, for chemicals that are emitted from the SIC, the chemical
		concentration (and also risk) increases as the percent of time the wind blows
		emissions from the SIC onto the base increases. In the case of variable
		wind speed and direction, from one week to the next, if the emissions are
		constant but the wind speed and direction are not the same, the correlation
		with percent downwind will be different. The confidence in the correlation
		of wind direction versus concentration is related to the number of
		observations that are used to calculate the correlation coefficient and the
		wind directions that are observed. During the 1998 ambient air sampling
		study, there were few periods of southerly winds, even fewer than that observed by historical wind roses. As a result, there were relatively few
		data points to correlate concentrations/percent downwind in an effort to
		assess SIC contribution. Therefore, sampling was extended for an
		additional 2 months and samples were collected on days which were
		predicted to blow from the SIC to NAF Atsugi for a better representation of
		historical conditions. Six additional days were sampled. The additional
		number of sampling days needed to provide additional information for the
		correlation analysis plots was determined by statistical analysis so that no
		bias would result by overweighing particular wind directions and
		overestimating the contribution from the SIC. This additional data was
		collected to reduce the uncertainty on underestimating the risk as a result of
		sampling on too few days when the winds were from the North. NEHC has
		added this additional explanation to the revised NEHC report.

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Page 20	As recommended previously by COT (NRC 1998), multiple exposure pathways were explored in the current NEHC risk assessment. Appropriate methods have been used except for the use of outdoor-air concentrations as surrogates for indoor concentrations.	As acknowledged by the subcommittee in this peer review, initially when the sampling plan was developed, the purpose of the indoor air samples was to provide exposure estimates for NAF Atsugi residents and dependents that would be used in the risk assessment. Another purpose of the indoor air sampling was to evaluate indoor air quality at Atsugi by comparing it with indoor air quality in the U.S. However, since the true objective of the risk assessment for indoor air was to calculate risk due to ambient air sources, including the SIC, the ambient air concentrations had to be used as surrogates for indoor air concentrations in the risk assessment because: 1) Concentrations for the majority of the constituents exceeding RBCs were found to be higher indoors than outdoors indicating probable indoor air sources (e.g., insulation, carpets, and household chemicals). If we are trying to determine the risk due to ambient air infiltrating indoors, using indoor air samples that also measure contaminants generated by indoor sources would overestimate the impact of the SIC and other ambient air point and non-point sources
		 2) Passive ventilation systems are used at most locations which make attempts to quantify the contribution of risk attributable to emissions from the ambient air sources highly uncertain. If we are trying to determine the risk due to ambient air infiltrating indoors, using indoor air samples that also measure contaminants generated by indoor sources would overestimate the impact of the SIC and other ambient air point and non-point sources. 3) Ambient air is the source of constituents in indoor air that are associated with emissions from the SIC and other ambient air sources. Using indoor air samples that also measure contaminants generated by indoor sources would overestimate the impact of the SIC and other ambient air point and

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		non-point sources and would not indicate a risk due to ambient air infiltrating indoors.
Page 20	Other than a brief mention in Appendix B, potential sources of air pollutants near NAF Atsugi, other than the incinerator complex, are not characterized, and their potential contributions to total risk are not evaluated. Such an evaluation would be helpful in differentiating risks attributable to the incinerator from ambient background risks or risks posed by other activities.	NAF Atsugi is located in a heavily industrialized area proximate to multiple point and non-point sources of airborne contaminants. We agree that an understanding of the emissions released from each of the other sources may help in discussing ambient background. However, Japan's primary mechanism for disposing of waste is incineration, which results in higher background concentrations of many airborne contaminants such as particulates and dioxins. Also meteorological conditions such as low percentage of downwind days makes the task of defining upwind conditions and downwind conditions difficult, whether one is trying to separate upwind days from downwind days for a given location, or trying to define upwind locations and downwind locations on a given day.
Page 21	RfCs are still converted to RfDs in the current risk assessment. It was recommended (NRC 1998) that RfCs themselves be used to calculate hazard indexes, and the present subcommittee reiterates that recommendation. Exposures to inhaled pollutants are converted to estimated doses in milligrams per kilogram per day. That conversion is not appropriate for inhaled pollutants with portal-of-entry effects. Therefore, the present subcommittee reiterates the recommendation that exposure concentrations, rather than doses, be used for inhaled pollutants.	The conversion of RfCs to inhalation RfDs is based on the recommendations of the USEPA Superfund Program. While there may be valid technical reasons for not converting RfCs to inhalation RfDs, the reason for the conversion is that RfCs incorporate exposure assumptions [i.e., RfCs are developed based on a lifetime exposure] and therefore can only be used to evaluate one exposure scenario [i.e., continuous exposure over a lifetime]. Inhalation RfDs are calculated from RfCs by dividing by 70 kg (an assumed human body weight), multiplying by 20 m3/day (an assumed human inhalation rate), and adjusting by an appropriate absorption factor (USEPA HEAST 1997 Annual Update). In Superfund risk assessments, multiple exposure scenarios and exposed populations are typically evaluated (e.g., residential adults and children or recreational scenarios) where the exposure assumptions incorporated into the RfC are not appropriate. Because of the need to evaluate risks for many types of NAF Atsugi-specific exposure scenarios (e.g., children and adults with

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		exposure durations less than a lifetime), RfCs and Unit Risks were converted to the RfDs and inhalation cancer slope factors (CSFs).
		The policy decision of converting RfCs to inhalation RfDs is further explained in the USEPA's Health Effects Assessment Summary Table 1997 Annual Update:
		"Superfund recognizes the importance of these issues [i.e., the uncertainties associated with converting RfCs to inhalation RfDs) and is actively working with EPA's Office of Research and Development to evaluate the impacts of these changes on its program regulations and guidance. In the short-term, however, modification of program regulations and guidance is not a viable option. Therefore, the chairs of the RfD/RfC and CRAVE Work Groups were consulted regarding Superfund's need to make the conversion from a concentration in air to a dose. There was agreement that, in many cases, converting the air concentration data to a dose (in mg/kg-day) may not add significant uncertainty to the Superfund risk assessment process, and therefore may be a reasonable use of the data given appropriate circumstances and Superfund program objectives."
Page 21	The contribution of pollutants from the solid-waste piles and liquid-waste sources at the incinerator facility are not explicitly addressed. Although pollutants from such wastes are presumably measured in the air samples, those wastes do not appear to have been considered as sources in the air-dispersion modeling. They also do not appear to have been considered as potential sources in the computation of the periods when particular sites were downwind.	The comment raises several very valid technical points. Any fugitive emissions from the liquid waste stored at the site, garbage delivered to the site, and incinerator ash stored on the site would certainly have been measured in the air samples collected during the monitoring program. These fugitive emissions, however, were not considered in the modeling for a number of reasons: 1. The garbage piles and waste drum material was constantly changing in consistency, volume, and location within the incinerator complex. 2. There was no way of determining what wastes or constituents were in

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		the piles and/or drums (the facility was permitted to burn municipal, hazardous, and medical wastes) and if they even had measurable emissions (e.g., were the drums tightly sealed or leaking). 3. While estimates were made as to the overall volume (e.g., the number of trucks that entered the facility each day and the estimated volume/mass of each truck) of waste that was delivered each day to the incinerator facility, no effort was made to estimate the number of drums or the volume of the waste pile(s) on any given monitoring day. 4. Overall there was insufficient information on the waste piles and drums to make any assumptions as to the input parameters for the modeling. The modeling assumed that the majority of the emissions from the facility impacting the base would come from the stacks that were nearly at ground level with the base. Therefore, for modeling purposes, we focused on the stacks where we could make educated assumptions regarding input parameters.
Page 21	The 2000 NEHC report still does not provide enough information on the observational design and methods. Some of that information is present in supporting documents (Pioneer 2000; Radian 2000a,b,c,d) and should be included in the NEHC report, but even the supporting documents do not provide all the necessary information.	As indicated in the subcommittee's comment, information on the observational design and methods is found in the Radian and Pioneer supporting documents including the Radian Sampling Plan. The NEHC report did not include as much information as the subcommittee would like NEHC to include because the NEHC report is a summary of the project, and the audience is the risk managers, who are mainly interested in the results of the health risk assessment and recommendations for risk reduction. Additional information has been included in the NEHC revised report to address the specific subcommittee's comments made regarding the need for additional information.
Page 21	Values below the limit of measurement have been replaced by half the limit of measurement in calculating averages. Such replacement might not be appropriate—a sensitivity analysis for the effect of this	Values below the limit of measurement have been replaced by half the limit of measurement in calculating averages because not only is this practice driven by EPA guidelines for handling non-detects, but also because

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	assumption should be performed.	according to a reference cited by the subcommittee (Hornung and Reed 1990), "if there is a compelling reason to report a mean concentration level, Method 2 (L/2) should probably be used". Mean concentrations were necessary to estimate the average health risk.
	APPENDIX A	
	Objectives of the NEHC Risk Assessment	
	As discussed previously, the subcommittee noted many different, and sometimes conflicting, objectives within the NEHC report and the supporting documentation from the contractors. In the Pioneer document (Pioneer 2000; p. 1), the purpose of the risk assessment is stated to be: 1. Estimate the potential human health risks to U.S. Navy personnel and their families and other individuals living and working on NAF Atsugi, Japan resulting from exposure to constituents of concern (COCs) in soil, ambient air, indoor air, and indoor dust. 2. Estimate the contribution of the risk attributable to emissions from the SIC. Suitable sampling plans could be designed to help answer both of those questions; however, the data-collection and analysis requirements for each question are different, therefore, both objectives must be considered in sampling design. The purposes of the risk assessment, however, are not consistently incorporated into design objectives throughout the documents. [For example, [Section 1.3 (Radian 1998a), Section 3.1 (Radian 1998c), Section 3.2 (Radian 1998a), Section 3.3 (Radian 1998a), Section 3.4 (Radian 1998a), Section 4.1 (Radian 1998c)]. Those different statements of objectives could imply substantially different approaches. NEHC should ensure	Both objectives were considered in the sampling design. These objectives were incorporated in the sampling plan protocols designed for risk assessment. Although these objectives were worded somewhat different in different documents they were consistent in developing the sampling design and in the review of the process. In the revised NEHC report NEHC has clarified the objectives to ensure consistency between the NEHC report and all supporting documents. The objectives have been revised as follows: This project was designed to collect data to meet the objectives of the comprehensive health risk assessment which were: 1. Estimate the potential human health risks to U.S. Navy personnel and their families and other individuals living and working on NAF Atsugi, Japan resulting from exposure to constituents of concern (CoCs) in soil, ambient air, indoor air, and indoor dust. This estimate focuses solely on CoCs that are likely to be associated with ambient air emissions and/or subsequent deposition from point and non-point sources impacting the air quality at NAF Atsugi. 2. The contribution of the risk attributable to the Shinkampo Incineration Complex (SIC).
	that the objectives of each aspect of the risk-assessment project are consistent with the overall project objectives so that sampling is	

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	conducted to meet the overall objectives.	
	APPENDIX B	
	Air Dispersion Modeling	
Page 23	Although this is potentially an important aspect of the exposure assessment, the NEHC report provides few details of the modeling. The results are summarized in a single paragraph (p. 27) and a map (Fig. 2-2) (NEHC 2000). No information regarding the assumptions, data sources, methods, or intermediate results is presented.	In the revised NEHC report, the discussion on dispersion modeling has been expanded to provide more details on the modeling.
	General Comments	
Page 24	The dispersion-modeling approach used by Radian International (2000 a,d) might be thought of as a relatively sophisticated means of interpolating and extrapolating, spatially and temporally, measured contaminant concentrations—one that adjusts concentration estimates to account for the impact of meteorologic variables on pollutant transport. For the six contaminants modeled, the estimated concentrations might be better estimates of the exposure potential than the average measured concentrations because modeling was able to take into account meteorologic variation over almost the entire study period.	NEHC did not use air dispersion modeling results to estimate exposure because since only 6 contaminants were modeled the risks would be underestimated.
	Specific Comments	
Page 25	On pp. 27-28 of the NEHC report, the dispersion-modeling results are presented only briefly, and no discussion of the dispersion-modeling method is presented elsewhere in the NEHC report. Therefore, readers of the NEHC report cannot understand the modeling results, their interpretation, and their significance. More details should be included.	In the revised NEHC report, the discussion on dispersion modeling has been expanded to provide more details on the modeling.

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Page 25	The discussion of dispersion modeling on p. 13 of the Pioneer (2000) report should include at least a summary of the assumptions, data sources, and methods.	This discussion on the summary of the assumptions, data sources, and methods has been included in the revised NEHC report. Including air modeling details in the Pioneer risk assessment report would not be appropriate.
Page 25	Page 4-7 of the Radian report (2000a) states that results of a small number of studies indicate that the maximal discrepancies between the values predicted by the Industrial Source Complex-Short Term (ISCST3) model and measured concentrations are generally less than 30% for well-characterized sources. References should be provided for that statement. One validation study using that model gave a correlation coefficient of 0.97 between observed and predicted concentrations of particles at an industry fence line, but that was for long-term estimates (Heron et al. 1984). Correlations for short-term estimates can be much worse than for long-term estimates.	The source shown below generally supports the questioned statement and presents actual data comparisons between ISCST3 and ambient data. The reference for that statement is "U.S Environmental Protection Agency and American Meteorological Society, "Model Evaluation Results for AERMOD," draft document, December 17, 1998."
Page 25	On p. 4-8, the Radian (2000a) report notes that atmospheric stability class was not directly measured and lists it as a source of uncertainty. However, Appendix I states that atmospheric stability class was determined on the basis of solar-radiation and temperature-gradient measurements made on site (Radian 2000d; p.1-3). That method is one of the best for determining stability class-better than the most commonly used approach based on wind speed and cloud cover. Therefore, very little uncertainty would result from that determination.	Atmospheric stability can be estimated by several methods. One common historical method was to use the wind standard deviation, which is a measure of the variability in wind direction. Recently, the U.S. EPA recommended replacing this methodology with a method that uses the incoming solar radiation during the day and the difference in ambient temperature at two elevations (typically 2 and 10 meters) to derive an estimate of atmospheric stability. Otherwise, to directly determine the atmospheric stability, one needs to have access to upper air data to determine mixing height. Upper air data can be obtained through upper air profilers or acoustic sounders or though the use of twice per day balloon launches. These observations were not part of the program scope of work and therefore, the solar radiation/delta

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#		temperature (SRDT) method was used. This amounts to using an estimation of atmospheric stability instead of measuring it directly, which may introduce a small degree of uncertainty. Since estimating atmospheric stability is a standard approach, any differences in the modeled outputs between using the SRDT method and using a method based on direct measurement using upper air data should be quite minimal.
Page 25	On p. 4-8 (Radian 2000a), another listed source of uncertainty is the assumption that each of the three incinerator stacks always had equal emission rates. The approach for estimating emission rates on p. 4-6, however, allows calculation of the emission rates separately, as well as lumped together. It might not have been possible to distinguish the impact of one stack from another with the method described, but no rationale for using the lumped approach is given.	Because we did not have any direct information as to actual operating conditions of the various stacks (and consequently the actual emission rates of the various stacks) there is no way of knowing if, or to what degree there are differences in emission rates. Since this information was not available, we had to make some assumptions in the modeling approach, and one of the assumptions was that each stack had equal emission rates. While in reality, this is probably not true, there is no way of knowing otherwise. Therefore, since we had to assume that all stacks had equal emissions, calculating the results separately, or lumped together would have produced the same result.
	APPENDIX C Uncertainty	
Page 25	There is inadequate discussion of uncertainty in the NEHC report (2000). The report mentions uncertainty only in the context of saying that it is minimized. It fails to disclose the types or magnitudes of any source of uncertainty and to discuss the impact of uncertainty in the context of the risk-assessment results. The purpose and meaning of "minimum degree of uncertainty" is not clear. Combining this statement with the precise and unqualified estimates of risk in the report, readers might infer that the risk-assessment results are certain. That is not consistent with the limitations of risk assessment in general.	In the NEHC revised report, NEHC has expanded the subsection on Uncertainties to include discussions on the types of uncertainties addressed in the Pioneer report and the magnitude of their impact in the risk assessment results including uncertainties in data collection and evaluation, exposure assessment, toxicity assessment and risk calculations. The use of the term "minimum degree of uncertainty" is related to the numbers, types and length of sampling. Aside from the uncertainties inherent to the risk assessment process, NEHC tried to minimize those elements of uncertainty that we were able to control while working within the limitations placed on this project due to its location. As a result of being a summary report, all the uncertainties addressed in other documents were not incorporated in the

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	Nevertheless, the subcommittee recommends that NEHC characterize the magnitude of uncertainties before and after their minimization and determine their impacts on the results of the risk assessment.	NEHC draft report.
Page 26	A discussion of uncertainty appears on pp. 45-46, but the discussion is not adequate. The text fails to convey the uncertainty surrounding the risks and could result in confusion about risk-assessment methods. For example, it discusses issues related to extrapolation of animal-toxicity data to humans, discusses generic uncertainty issues, and states that "in calculating toxicity values for each chemical, safety factors of 10 to 1,000 are applied to the toxicity values to account for these extrapolations." The sentence apparently is directed at interpreting the application of uncertainty and modifying factors in estimating reference doses and reference concentrations for noncancer health effects, but the complete bases for the safety factors of 10-1,000 and when they are used should be clarified.	As a result of being a summary report, all the uncertainties addressed in other documents were not incorporated in the NEHC draft report. However for the purpose of including additional information, the discussion of uncertainty surrounding the risks has been expanded in the NEHC report. This discussion includes a more detailed explanation on the complete basis for the safety factors.
Page 26	The final paragraph in the section on uncertainties, on p. 46, implies that it is more controversial to evaluate uncertainties than it is not to, and that doing so requires more judgment than is required for a screening risk assessment. The subcommittee disagrees and recommends that the uncertainty in the point estimates of risks presented (such as a statement that actual risks are likely to be somewhere between zero and the upper-bound estimates provided) be more fully characterized and that NEHC reconsider the discussion of the benefits of characterizing uncertainty.	NEHC 's intent in this paragraph was not to imply that it is controversial to evaluate uncertainty, but to note that controversy is inherent to judgment when drawing conclusions regarding the risk. Science points out the uncertainty and judgment analyzes how the uncertainty determines one or more points of risk estimates. This paragraph has been edited and added to an earlier paragraph, which discusses how uncertainties in the characterization of the non-cancer risk should be interpreted.
Page 27	Of those 13 sources of uncertainty, the NEHC report (2000, pp. 45-46) mentions only the last four; the subcommittee believes that other	The subsection on Uncertainty has been expanded in the NEHC report to include all thirteen sources of uncertainty.

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	sources of uncertainty also warrant mention. The criteria used to assign qualitative magnitudes should be provided.	
Page 27	In addition, the magnitude rating of 2 for uncertainty associated with soil, indoor dust, and dermal absorption seems to be high, considering that little of the risk was attributable to those media. Therefore, absorption might not have a "medium" effect on risk estimates, as stated in the supporting document (Pioneer 2000; p. 88).	As explained in the Pioneer report uncertainty associated with indoor dust, can vary within two orders of magnitude because the indoor dust samples were only analyzed for dioxins and furans. Soil concentrations were used as surrogate indoor dust concentrations for all constituents. The uncertainty associated with identification of CoCs in soil was not 2, but 0.
		The uncertainty associated with dermal absorption can vary within two orders of magnitude because experimentally derived dermal absorption rates were used to evaluate dermal contact with soil.
Page 27	The subcommittee recommends that NEHC state the meanings of "negligible", "small", "medium", and "large" effects on risk calculations.	The meaning of "negligible", "small", "medium", and "large" effects on risk calculations is associated with the number of orders of magnitude the uncertainty impacts the results of the risk assessment. For example, if the degree of the uncertainty varies from 0 to 3 order of magnitude, 0 is negligible, 1 is small, 2 is medium and 3 is large. This has been explained in the Pioneer report.
Page 27	On p. 88 of the Pioneer (2000) report, the column labeled "action or result" in the table is confusing, and it is not clear whether the magnitude classification applies before or after the listed actions have been taken. It is not clear whether those magnitude classifications are related to the uncertainties in the characteristics themselves or to their impact on the overall results.	In the revised Pioneer report clarification has been provided.

e e a	Discussion of the exposure assumptions and scenarios should be expanded to convey the variability and uncertainty in exposure estimates. Some uncertainties mentioned elsewhere in the text do not	The exposure assumptions and scenarios are fully discussed in the revised NEHC report as well as the uncertainty associated with EPA default
	appear in the section on uncertainty. For example, the partitioning of the substances into particulate and gas phases is stated to be uncertain in some places but is not mentioned in the section on uncertainty.	assumptions and the site specific factors regarding age, duration and frequency of activity, which have generated the risk estimates for child and adult at the different areas of concern on base and the duration of the exposure for different tour lengths (i.e., 3, 6 years) and individuals living on base for 30 years.
		The subcommittee refers to partitioning of HCl into particulate and gas phases. This partitioning was mentioned to explain that FTIR sampling detected HCl in vapor phase as opposed to conventional sampling which collected both phases. Particulate and gas phase partitioning does not represent an uncertainty in the risk assessment because both particulates and gas phases were collected with conventional sampling which provided the concentrations used in the risk assessment, not the FTIR.
p	Measurements of particular compounds should be discussed as possible sources of uncertainty. For example, uncertainty might be associated with dioxin measurements (on the basis of the following statement p. 29, NEHC 2000): Maximum detections for dioxins were always found in the samples taken near the fenceline north of the incinerator and at the golf course between the third and fourth holes, east of the incinerator. Elevated dioxin concentrations found at the golf course between the third and fourth holes, east of the incinerator appear to be an anomaly perhaps associated with blowing ash.	Uncertainty with data collection and evaluation is presented in the NEHC revised report in the Uncertainty subsection. In regard to this specific example cited by the subcommittee, an anomaly on the dioxin concentration on the golf course does not represent an uncertainty associated with dioxin measurements. The anomaly was attributed to blowing ash deposition on the golf course near the incinerator gates, from trucks carrying ash leaving the incinerator complex, not uncertainty with dioxin measurements. The elevated dioxin concentration found at the golf course had a congener profile similar to that found in ash. This profile was different than the congener profile for maximum concentrations in soil from air emission deposition found near the fenceline north of the incinerator. Because this study was designed to support risk management decisions regarding the health risks at NAF Atsugi, it was critical to reduce, as much

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#		as possible, the uncertainties regarding data collection and measurements. Most of these uncertainties regarding identification of CoCs present in soil and ambient air at the Base in the sampling methodology were reduced using site-specific information to develop sampling work plan and focus sampling efforts. By collecting samples with sufficiently low detection limits to perform health-based risk analysis; and ensuring that the number and frequency of samples collected was statistically determined it was possible to control the possibility of over- or underestimating the health risks associated with the identification of CoCs in ambient air and soil. The sampling frequency for more than one year guaranteed that each day of the week, as well each week of the year, would be represented to account for any variability due to the day of the week, season, or other temporal effects could be assessed. Some uncertainty associated the identification of CoCs in indoor air and indoor dust could not be minimized since ambient air concentrations were used as surrogate indoor air concentrations for quantitative evaluation in the risk assessment.
Page 27	Because of the interpretation of the hydrogen chloride data, reported concentrations might have been higher than the actual concentrations at NAF Atsugi. FTIR did not detect hydrogen chloride (Radian 2000a; p. 3-3), although, according to conventional measurements, it was expected that FTIR would detect hydrogen chloride. It was assumed that FTIR failed to detect hydrogen chloride because it was in particulate or aerosol form, which would not be detected by FTIR. That assumption is illustrated in the following paragraph (Radian 2000a; p. 3-4): On seven occasions between January and April 1999, the ambient air monitoring station at the GEMB site reported 24-hour hydrochloric acid concentrations near or above the	The acid gas samples (hydrogen chloride or hydrochloric acid) were collected using an annular denuder. This method works by first pulling the ambient air through a 2.5 micron impactor which removes all particles greater than 2.5 microns. At this point, the air stream can contain fine particulate salts (<2.5 microns), fine aerosols, and of course gas phase HCl. The gas phase HCl is absorbed on one of two glass denuders coated with sodium carbonate, that absorbs the acid gases. Some, but not all, of the aerosol phase HCl will be absorbed on these tubes, but none of the fine particulate phase chloride will be captured here. The final stage of the apparatus is a Teflon filter, which captures the remaining aerosol phase HCl and all of the particulate phase chloride. Each of the three stages were recovered and analyzed separately. Sample

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#	FTIR system's minimum detection limit of 7 µg/m³. The FTIR system did not detect hydrochloric acid on any of these occasions, and these results were carefully checked. As mentioned in Section 3.1 above, the FTIR only detects chemicals in the form of a gas. Hydrochloric acid in the form of aerosols or particles is not detected. It must be assumed that the hydrochloric acid measured by conventional means on the seven occasions consisted largely of aerosols and particles since the chemical was not detected in the form of a gas by the FTIR system. However, the following is stated about possible interference by other chloride compounds, on the basis of denuder measurements (Radian 2000a; p. 2-14): Therefore, particles captured on the filter should only contain negligible amounts of HCl and HF, but could contain an interferent such as sodium chloride NaCl (metallic chloride salts). Chloride salts on the filter would cause a high bias in the estimate of HCl. In fact, for most of the highest HCl values reported, the major contribution was found on the filter, with much smaller amounts on the denuder sections. Because of the potential presence of interfering chloride salts, the subcommittee believes that some discussion of analytic interference of chloride ion with hydrogen chloride is required; and the assumption that the hydrogen chloride measured with conventional means was from aerosols and particles should be reconsidered.	recovery consisted of rinsing each denuder tube with distilled water and desorbing the filter in distilled water. Each sample was then analyzed by ion chromatography for the fluoride, chloride, and sulfate. Due to chemical kinetics, the chloride found on the denuder tubes could only be HCl and no other chloride salts. However, for the filter samples, the original form of the anion cannot be determined with certainly, because these ions are water soluble, and they disassociate when water extracted and only the chloride (or other anion) determined. At the beginning of the study, it was expected that most of the chloride would be found on the first denuder. Because of the presence of a nearby source of ammonia (pig farm) that could neutralize the HCl by forming ammonium chloride in the atmosphere, it was decided to include chloride results from all three fractions in the calculation of HCl concentration. The final program results show that the chloride on the filter contributed, on average, 30% of the total chloride concentration. The chloride measured on the filter results from chloride salts and aerosol HCl. In cases when the filter fraction represents a substantial portion (>60%) of the overall chloride concentration, the HCl concentration values may be biased high, but we also cannot rule out that the source was the incinerator. In all but one instance, when the filter chloride concentration was high, the site was downwind of the incinerator a significant portion of the time and the upwind concentration of HCl was not elevated (indicating that outside sources of chloride salts were not causing the increased filter chloride concentration). While it is possible that sea salts were causing some of the elevated values, Spengler, et al. (JAPCA 33, 12:1162-1171, 1983.) found that the majority of chloride salts, either emitted by incinerators or formed by secondary atmospheric reactions, tend to be fine particles while sea-salts and road salts generally reside in a larger fraction. Therefore, the 2.5-

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		micron impactor should have removed the majority of the sea salts. If fine particle phase chloride salts were forming either in the incinerator itself, or due to secondary reaction in the atmosphere, the FTIR could not measure these salts while the denuder could. A number of the highest values resulted from elevated chloride in the particle fraction during periods when the site was downwind of the incinerator a significant portion of the sampling period. Because the corresponding upwind data from these sampling periods did not have elevated particulate phase chloride, long-range transport of chloride salts can be ruled out, and the incinerator must be considered to be the major source.
Page 28	The subcommittee also recommends quantification of "numerous visual observations" (p. 4) and a discussion of the uncertainty relevance of the statement "the eggs and produce may represent a possible source of variation from U.S. background" (p. 45) (NEHC 2000).	Since this is anecdotal information and counting these observations was not part of the scope of this project for determining the risk, it is not possible to quantify numerous visual observations. The statement "the eggs and produce may represent a possible source of variation from U.S. background" (p. 45) (NEHC 2000), has been deleted from the NEHC report.
Page 28	On p. 64, NEHC (2000) states that "there were few days in which these conditions were actually met; therefore, the analysis itself has low power." The subcommittee recommends including a discussion of the uncertainty caused by that fact and of how that uncertainty was factored into the analysis.	A discussion of the uncertainties associated with the upwind vs. downwind analysis has been added to the PIONEER and NEHC Reports.
	APPENDIX D Soil Trend Analysis	
Page 28	Although sufficient information on the soil-trend analysis was provided in a Radian report (Radian 1998b), the NEHC riskassessment report does not provide sufficient information. The subcommittee recommends including more of the details and	More of the details and description provided in the Radian report (1998b) have been included in the NEHC revised report. Additional information on soil sampling for trend analysis, including this paragraph has been added to the revised NEHC report.

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π	description provided in the Radian report (1998b), including the following:	
	In the first paragraph on p. 29 of the NEHC report, the second and third sentences should be replaced with the following, based on the Radian report (1998b; p. 1-15):	
	To determine the deposition trends across NAF Atsugi, the base was divided into areas defined by seven radii starting at the Jinkanpo Incineration Complex and extending to the north, with transects at arbitrary distances of less than 300 m, 300-800 m, 800-1,500 m, and greater than 1,500 m from the complex. For trend-analysis purposes, samples were collected from locations where the soil had not been recently disturbed (such as by construction activities). Also, samples were collected, where possible, from areas of potential sediment accumulation, areas of observed vegetation stress, and areas lacking evidence of erosion or ground cover.	
	The narrative in the Radian report (1998b) is more factual and demonstrates that the soil-trend samples were taken in a logical manner.	
Page 28	The second sentence in the second paragraph on p. 29 of the NEHC report should be replaced with the following based on the Radian report (1998b, p. 4-43):	Text replacement has been made in the NEHC revised report.
	It is evident that subsurface soils from the soil trend data set are less	

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	contaminated than the surface soils.	
Page 29	In the second paragraph on p. 29 of the NEHC report, "A definite footprint TEQ's exceeded RBCs" (NEHC uses "RBC" for "risk-based concentrations") should be deleted. The following, based on the Pioneer report (2000; p. 92), should be inserted in its place as a new paragraph: The soil trend analysis indicates a spatial correlation between concentration and distance from the SIC for total 2,3,7,8-TCDD TEQs (see Figure 7-1). Consequently, concentrations of total 2,3,7,8-TCDD TEQs in soil samples on the base typically increase as the distance from the SIC decreases. The soil-trend analysis also indicates that the concentrations of total 2,3,7,8-TCDD TEQs exceeded RBSCs (risk-based screening concentrations) throughout the base for soil samples at up to 3 in. (7.6 cm) and about half the base for soil samples at 3-12 in. (7.6-30.5 cm).	Text replacement has been made in the NEHC revised report.
Page 29	In the second paragraph on p. 29 of the NEHC report, the last sentence should be replaced with the following, based on the Radian report (1998b; p. 4-46): On the basis of the lack of spatial trends, and the generally isolated occurrence of the SVOCs, their presence in soils does not appear to be associated with the Jinkanpo Incineration Complex.	Text replacement has been made in the NEHC revised report.
Page 29	The subcommittee recommends the following: • Reporting averages and ranges of concentrations detected and the RBC in Table 2.6 (NEHC 2000) rather than the RME and average concentration. The geometric mean and geometric	NEHC has edited Table 2.6 to include more descriptive statistics such as the range of detected values, the range of detection limits for non-detected samples, the median, the mean, and the standard deviation the upper 95% confidence limit.

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	 standard deviation (or the 5-95% range of observed or calculated values) should also be included. Showing the reference areas (background soil areas) on Figure 1-2 in the NEHC report (the figure of the layout at NAF Atsugi). Showing the wind rose on any plots of the trend-analysis results that NEHC presents 	 The reference areas are already shown in figure 2-1 of the NEHC report. Since the wind rose patterns are variable it would be difficult to select a single wind rose to be placed on these trend analysis plots.
	APPENDIX E Air Monitoring	
Page 29	The subcommittee has reviewed the air-monitoring data and quality-assurance audits and has confidence in them. In general, the techniques used for air sampling and meteorologic monitoring appear to be adequate and to represent the state of the art. The subcommittee's main concerns are with the planning, the analysis of the data collected, and the connection between the analysis and the sample-collection strategies. Although the techniques used for air sampling are appropriate, there should be more discussion of the limitations and of possible alternative methods. Some minor comments on the air monitoring are presented below.	Monitoring techniques, methods and particular analysis appropriate for meeting the objectives of the health risk assessment are limited. The NEHC report does not describe rationale for using the particular monitoring techniques that were used because NEHC believes that the risk managers for whom the report was written, are mostly interested in the results of the risk assessment, rather than more discussion of the limitations and of possible alternative methods. More details on planning, including the analysis of the data collected, have been added to the revised NEHC report to help the reader in making the connection between the analysis and the sample-collection strategies
	Comparison with U.S. Cities	
Page 29	The second column in Table 2-2, titled "U.S. Data" (NEHC 2000; p.18), is confusing because it is not stated whether the values are means and, if so, of which cities. If they are means, it is not appropriate to use them as a basis for comparison with the highest or second-highest concentration in the National Ambient Air Quality Standards (NAAQS). It would also be helpful to include the NAAQS in this table.	The "U.S. Data" column has been deleted from Table 2-2 in the revised NEHC report. A column for the NAAQS has been added to this Table.

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	Fourier-Transform Infrared Monitoring	
Page 30	The documentation (such as, Radian 1998d) mentions that open-path FTIR was used at some time but does not clarify when it was used or reports on the results of using it. For example, it is unclear what type of FTIR monitoring is being referred to in the statement that "the FTIR monitoring found high (ppm) levels of hydrogen chloride in the SIC plume exiting the stack" (Radian 1998d; p. 5-5).	The open-path FTIR mentioned in the Radian report refers to the FTIR instrument used in a separate air monitoring project designed for compliance purposes to monitor the stack emissions.
	Other Monitoring	
Page 30	Although the NEHC report does not mention monitoring of the incinerator facility with a video camera or the use of optical pyrometers, a supporting document (Radian 1998a) does (referring to an infrared pyrometer initially and in the equipment inventory). The results obtained with those monitors are almost undocumented in any of the reports.	The pyrometer was initially used for determining whether the incinerator was on or off. Weather conditions deteriorated the pyrometer and could no longer be used. Video cameras were used to monitor the plume for compliance purposes and to verify wind direction if needed.
Page 30	The one documented result is the on-off status of the incinerator, which is recorded for each sample in one of the data files provided to the subcommittee and in Table 2-3 of the Radian report (2000a). In addition to indicating the status for each sample, it would be more appropriate to indicate the status hour by hour to correlate with the continuous air-monitoring data. In addition to indicating the status for each sample, it would be more appropriate to indicate the status hour by hour to correlate with the continuous air-monitoring data.	Reporting the status of the incinerator for each sample hour by hour was not included because it was well known that the incinerator operated 24 hours per day, 6 days a week, Monday through Saturday, and was off on Sundays, except during Japanese Holidays. Therefore during sampling times, the incinerator was either on or off, regardless of the time of the day. The information was kept readily available in case questions arose about specific days of sampling. The effort to review the video tapes hour by hour for 14 months would be extremely labor intensive and unwarranted.
Page 30	How the monitors (camera, pyrometer, and so on) were used to determine on-off status and any uncertainty involved in that determination should also be described. The subcommittee	On and off conditions prior to the use of the pyrometers and after they were discontinued were determined by visual observation for the presence of smoke from the stacks and predictable start time of operations.

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	recommends adding information on how on-off status of the stacks was determined before the optical pyrometer began operating in October 1998 (Radian 1998d, 1999c), including information on the reliability of the method(s).	
Page 30	There did not seem to be any analysis of the time-lapse video records. That is particularly surprising in light of the emphasis in the planning stage on the analysis of the tapes to obtain information on plume behavior and fumigation conditions (Radian International, unpublished data, January 27, 2000, July 1998).	Video cameras were used to monitor the plume for compliance purposes and to verify wind direction if needed. Time-lapse video records were used as evidence for the legal case against the Shinkampo Incinerator Complex by the U.S. Department of Justice. No analysis of the time-lapse video records was made because information on wind direction through plume behavior was not needed for the air monitoring for risk assessment purposes.
Page 30	The comparison of the Atsugi mean contaminant levels with the US mean values in Table 2.5 (NEHC 2000; pp. 25-26) is not valid. The values used for comparison in that table are presented, as US mean values but are not. They represent data collected in a small survey in California or very old exposure estimates reported by Shah and Singh (1988) that are not representative of average US exposures to the substances in question but are averages of all reported indoor-exposure measurements.	NEHC does not agree with the subcommittee that the comparison of the Atsugi mean contaminant levels with the US mean values in Table 2.5 (NEHC 2000; pp. 25-26) is not valid. There are no other studies as comprehensive as the Cal EPA and the studies reported by Shah and Singh on the EPA TEAM studies. Although the Cal EPA study may be limited to homes in California cities and counties, the U.S. TEAM study is an extensive and comprehensive review of numerous papers on indoor air data collected all over the United States.
		Table 2-5 has been improved. We have replaced the heading "U.S. Mean" with "U.S. Studies Mean" and ensured that the reported results from the TEAM studies include only average concentrations of chemicals found in residential indoor air. This is the best available data found in the literature. The subcommittee made no recommendation of other studies to use for comparison purposes. Since comparison with U.S. data is not an objective of the risk assessment, Table 2-5 has also been moved in the revised NEHC report to Section 4, Health Evaluation to add another perspective to the

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		health evaluation.
	APPENDIX F Health Risk Assessment Human Health Risk Assessment Results	
Page 30	Without a complete description of the population at risk, however, it is difficult to evaluate the relevance of those exposure scenarios. (For example, are subgroups of the population at greater risk, such as military personnel and their families who have repeated, but nonconsecutive and therefore not limited, tours of duty at NAF Atsugi?)	A subsection in Section 3 has been added to the revised NEHC report to provide better descriptions on populations at risk, such as children in elementary school, tower residents etc. More information from the exposure pathway analysis has also been added to the revised report. The following text has been added to Section 1: "The NAF Atsugi population consists of military personnel and their families who live and work on the facility, civilian personnel, and Navy contractors who work on the facility. Military personnel are typically stationed at NAF Atsugi for 3 years (1 Tour of Duty), however the tour can be extended to 6 years (2 Tours of Duty) or more. The NAF Atsugi population is approximately 7,500 when sailors, residents, and workers are present, of which 81.1% is composed of active duty members and their dependents, 1.22% are Department of Defense employees such as teachers and their dependents, 5.02% are Civil Service employees and their dependents and 12.65% are Master Labor Contractors including Japanese Nationals. Seventy-five percent of the population lives on base and 25% off base. It is estimated that approximately 6,000 are adults. There are approximately 446 dependents under 6 years of age and about 916 dependents between 6 and 18 years old living on base versus 129 and 180 respectively living off-base. The military sailors are typically out to sea for 4.5 months per year. Additional months may also be spent off base as needed (e.g. sailors were deployed to Persian Gulf for 6 months during 1998). Middle school and high school students (12 – 18 years) are

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		bussed daily during school (September – June) to Camp Zama, which is located several miles from the SIC. The normal tour of duty is 3 years, but it can be extended for the same length several times. Some military and or civilian members, particularly those with Asian spouses or Asian backgrounds have chosen to extend the tour many times."
Page 31	Various direct exposure pathways are introduced and discussed. Indirect exposures are not considered, because most food is assumed to be supplied from the United States and the drinking water is assumed not to be contaminated by incinerator fallout. Those assumptions should be better documented with supporting data.	Although this information is provided in the Radian supporting documentation on Exposure Pathways Analysis, the NEHC report has been revised to include information contained in this document and about results of drinking water testing performed routinely on base drinking water. Regarding the food pathway, NEHC, as well as the subcommittee has to believe that food supplied from the U.S. is safe. This is not an unreasonable assumption.
Page 31	In addition, the fact that drinking water is not affected by the incinerator facility does not obviate the assessment of drinking-water contaminants to determine the overall health effects of residing at NAF Atsugi.	Drinking water contaminants have been assessed. Not only the multipathway analysis determined that the groundwater pathway was incomplete, but recent drinking water sampling results conducted to ensure that it meets U. S. drinking water standards indicated that the groundwater has not been impacted by incinerator operations (Dames & Moore 1999).
Page 31	As is appropriate, the risks of cancer and noncancer effects are discussed in the NEHC report. It states that the cancer risk for children is "slightly higher" than the EPA benchmark (10 ⁻⁴), as is the risk of noncancer effects. Table 3-2 of the NEHC report indicates that 60% of the exposure scenarios for children have cancer risk estimates that exceed 10 ⁻⁴ , compared with 15% of adult exposure scenarios. Higher noncancer-hazard indexes are also observed for children than for adults for every exposure scenario except recreational golfers. The source of the apparently larger risks for children should be clearly identified by NEHC. For example, are the	Throughout the revised NEHC report, sources of larger risks for children are identified, such as physiological, biochemical, and behavioral differences from adults that make children more sensitive to chemical compounds during similar exposure scenarios.

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	differences from adult risks due to higher soil-ingestion rates, breathing rates per unit body weight, and so on?	
Page 31	With respect to the risk of non-cancer effects, the document gives the impression that risk is related largely to respiratory effects and that those effects are reversible, but both impressions might be overstated.	Since compounds contributing to the majority of the non-carcinogenic effects have toxicity studies showing inhalation as the primary route of entry and that acute effects are generally reversible, it is unreasonable to say that these are understatements. This is discussed in the subsection on the health effects of various chemicals of the NEHC report.
Page 31	Several of the chemicals of concern are reported to be reproductive and developmental toxicants; those types of toxicity should be given more consideration.	The revised NEHC report includes more consideration for the reproductive and developmental effects of the chemicals of concern, as discussed in the subsection on the health effects of various chemicals.
Page 31	In addition to listing the hazard index in Table 3-2 (p. 37; NEHC 2000), it would be useful to indicate whether any hazard quotients exceed 1.	NEHC has edited the table to indicate which Hazard Indices exceeded 1.
Page 31	The meaning of notes to Table 3-3 (p. 40; NEHC 2000) is not clear.	Footnotes have been corrected in the revised NEHC report.
	Health Evaluation	
Page 31	Overall, much of this section is repetitive of earlier sections of the report and not central to its stated purpose.	Repetition of earlier sections was necessary as an introduction to the interpretation of the risks; however, the text has been revised to minimize repetition in the revised NEHC report.
Page 31	Basic questions for persons residing at NAF Atsugi are how the incinerator is affecting their health and how certain NEHC is about the effects; for example, "How many studies have been completed on Jinkanpo and who did the studies?" (see "Frequently Asked Questions" in Appendix B, NEHC 2000). Those questions are not	This background information has been added more appropriately to the background subsection in Section 1 of the revised NEHC report, instead of the health evaluation section.

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	adequately answered in the health-evaluation section.	
Page 31	The text pertaining to children's risks (NEHC 2000; p. 55) is vague and superficial and does not consider potentially susceptible populations. Given NEHC's emphasis on using EPA methods, it is surprising that it does not follow EPA's increasing focus on childhood risks.	The revised NEHC report includes an expanded discussion of children's risks in the subsection on considerations for children.
Page 31	NEHC also does not address the potential initiation, exacerbation, or persistence of asthma due to chemical or particle exposures (see reviews by Jones 2000; D'Amato 1999; Goldsmith and Kobzik 1999; Linn and Gong	The effect of chemical or particle exposures on asthma is discussed in the subsections on considerations for children and health effects of various chemicals in the revised NEHC report.
Page 31	The calculated cancer risk estimate is an upper bound on lifetime probability of developing cancer under defined exposure conditions. NEHC uses the RME to estimate an upper bound on the estimates. If a number of upper-bound estimates of exposures are used to estimate risk, then on the basis of simple joint-probability calculations for independent events, the estimated risk will most likely be much higher than the actual risk. The same logic applies to the average-exposure scenario; the probability outcome of multiple mean estimates is unlikely to be an average result and more likely (in these types of risk assessments) to be an upper percentile, depending on the number of separate variables and on details of the distributions. The nature of the cancer risk and exposure scenarios should be taken into account in the risk assessment and its interpretation. An alternative approach would be to use a more sophisticated distributional analysis that could incorporate both individual variability and uncertainty.	As recognized by the subcommittee, NEHC uses the RME to estimate an upper bound on the estimates. The Public Health Summary indicates that the calculated cancer risk fro the child resident (less than 6 years of age) could result in "as much as" 1.1 additional cases of cancer in a population of 10,000. In the conclusions, the risk is addressed as the "reasonable maximum cancer risk." Both indicate that the calculated risk is an "upper bound" risk. Risk exposures are presented in the NEHC and Pioneer reports as separate upper bound estimates for different and independent exposure scenarios. Therefore, since we are not presenting the results as joint probability calculations, there should be no concern about overestimation of the risk beyond the reasonable maximum exposure. A more sophisticated distributional analysis that could incorporate both individual variability and uncertainty would be an interesting academic project. However, no resources are available to conduct such an analysis

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	Cullen and Frey (1999) present more information on that type of analysis.	that although interesting would not add any information that hasn't been already gathered to assist the risk managers in their risk management decisions.
Page 32	The NEHC report (p. 69) suggests that RMEs were used as input into the IEUBK model for this risk assessment, implying the use of 95th percentile upper confidence bounds (UCL95) on the means for air and soil concentrations, and the upper end estimates for other parameters as selected for the RME in the risk assessment; that would not be appropriate. The Pioneer (2000) report, however, suggests that estimates of UCL95 on the means for air and soil concentrations were used as input into the model, and that the IEUBK default values were used for other parameters; these also are not ideal inputs for the model. A preferred approach is to use median estimates for exposure concentrations as point-estimate inputs into the IEUBK. Evaluation of the variability among individuals would require a convolution of the variability distributions for the exposure-point concentrations with the lognormal variability distribution included with the IEUBK to estimate variation among individuals exposed to fixed input concentrations. The subcommittee also notes that the value of 3.9 μg/m³ used in the risk assessment (Pioneer 2000) as the UCL95 on the sitewide mean air lead concentration is incorrect by a factor of about 10: the UCL95 on the mean is close to 0.4 μg/m³, although the estimate depends somewhat on the assumptions made about the distribution.	The IEUBK model runs have been re-evaluated to ensure that they are correct. There was a typographical error in this section. The site-wide RME lead air concentration should read 0.39 ug/m3 NOT 3.9 ug/m3. Since 0.39 ug/m3 is below the NAAQS value for lead the note will not be added. These upper-bound concentrations were used to provide a conservative estimate of blood-lead levels at NAF Atsugi, Japan. If the mean or median concentrations were evaluated using the IEUBK model then the results would be lower than the RME results – which are below levels of concern.
Page 32	On p. 69, NEHC's discussion of lead measurements states that "this value is well below the Centers for Disease Control and Prevention (CDC) benchmark of greater-than-5 percent probability." The CDC has no such benchmark. As NEHC correctly indicated in the previous	We disagree with the subcommittee on this comment. The EPA and the Center for Disease Control and Prevention (CDC) have determined that childhood blood lead concentrations at or above 10 ug/dl present risks to children's health. ATSDR specifically points out in their health

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	paragraph on p. 69, the 5% probability is a guidance level from EPA.	consultations that this is the level of concern for CDC, and therefore it is a CDC benchmark.
Page 32	Page 54 of the NEHC report states "typically, RfCs are one-thousandth of a NOAEL; therefore, a hazard index of 10 would be acceptable in these cases because there would still be a safety margin of exposure of 100." That statement confuses the basis of uncertainty and modifying factors and of their relationship to the hazard index. Uncertainty and modifying factors are used to extrapolate to safe exposure levels for humans, accounting for uncertainties resulting from differences between studied exposures and possible human exposures. The average human might be ten times more susceptible than the average member of the most susceptible animal species studied; a highly susceptible human might be ten times as sensitive as the average; and human exposure can be ten times higher than the longest exposure observed in the laboratory. Similarly, the term "safety factors" as used by NEHC (p. 54) is not appropriate. It is also unclear whether NEHC is attempting to define a universal value for the hazard index that would correspond to a point where health effects might be expected or to define an acceptably low value of the hazard index to dismiss all concerns about health effects. Clarity is critical to the question of what the overall goal of the risk-assessment project is. Is it attempting to show that there is no problem, or is it attempting to see whether there is a problem?	In this comment, the subcommittee is alluding to a reference mentioned in the NEHC report from the Risk Assessment and Risk Management in Regulatory Decision-Making by The Presidential/Congressional Commission, Volume 2, 1997. The following is the paragraph from this document that NEHC referred to, from the Chapter on Recommendations for Specific Regulatory Agencies and Programs, which evaluated the EPA risk assessment approach for assessing hazardous air pollutant sources to implement section 112 of the Clean Air Act: "The 1990 amendments do not set a threshold for considering health risks other than cancer, which the Commission believes to be a serious omission. We chose a threshold hazard index of 10 because there are few hazardous air pollutants with RfCs that are within a factor of 10 of their no-observed-adverse-effect-levels (NOAELs). Typically, RfCs are one-thousandth of a NOAEL, so a hazard index of 10 in these cases would still leave a margin of exposure of 100. Analogous screening risk assessments that have been performed at Superfund sites might provide useful information about the extent to which screening risk assessments generally identify hazards above and below 10." As illustrated in this paragraph it doesn't appear to be any confusion between the uncertainty factors and their relationship to the hazard index. The safety factor equates to the margin of exposure and not to the uncertainty factors. By making a reference to this paragraph from the Risk Assessment and Risk
		Management in Regulatory Decision-Making NEHC's intent was just to

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		present the Presidential/Congressional Commission's perspective on their evaluation of the EPA risk assessment approach for assessing hazardous air pollutant sources, not define any values for acceptable Hazard Index.
Page 32	In the first paragraph on p. 75, citations should be added for all the concentrations and locations presented	Sources of information concerning concentrations of acetaldehyde at various locations in the U.S. have been added to the NEHC revised report.
Page 32	In the second and third paragraphs on p. 75, the information on EPA and the RfC for acetaldehyde is repetitive and contradictory. The RfC given in the second paragraph is incorrect. The correct value is given in the third paragraph.	The subsection on health effects of various chemicals, including information on acetaldehyde, has been revised in the NEHC report.
Page 33	The discussion of acrolein on p. 76 is simplistic and confusing.	The subsection on health effects of various chemicals, including information on acrolein, has been revised in the NEHC report
Page 33	On pp. 69-77, the brief descriptions of the health effects of various chemicals are not clear and do not add to the document. It would be preferable to include an evaluation that incorporates known and suspected adverse human health effects.	The subsection on health effects of various chemicals has been revised and includes a discussion of known and suspected adverse human health effects for each compound that contributes to the majority of carcinogenic and non-carcinogenic risks.
	Pioneer Risk Assessment Document	
Page 33	On p. 7 of the Pioneer (2000) report, it is stated that hole 9 of the golf course "frequently receives emissions from the incinerator stacks." However, the wind rose indicates that the wind is from west, west-southwest, or west-northwest about 2.7% of the time (average, about 10 d/yr).	This statement has been revised in the PIONEER report to read "Hole 9 is closest to the SIC and occasionally receives emissions from the incinerator stacks."
Page 33	The statement on p. 8 of the Pioneer (2000) report that "these assessments do not address risks from other sources of exposure (e.g.,	Lack of clarity in stating the objectives of the risk assessment has led the subcommittee to believe that the objective of the risk assessment is to

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#	dietary exposures) or risks from other constituents that are not associated with the site under evaluation" is also not consistent with the first objective of estimating the potential human health risks at NAF Atsugi.	estimate the potential human health risk resulting from living at NAF Atsugi and not just the risk related to the incinerator. As clarified in the revised NEHC and supporting documents, the objective is to estimate the potential total human health risk at NAF Atsugi resulting from exposure to constituents of concern (CoCs) in soil, ambient air, indoor air, and indoor dust, focusing solely on CoCs that are likely to be associated with ambient air emissions and/or subsequent deposition from point and non-point sources impacting the air quality at NAF Atsugi. With this clarification it is easier to understand that, as indicated in the NEHC and Pioneer reports, dietary exposure was not considered a pathway because food consumed by the base population comes from the U.S. and not from locations near the incinerator.
Page 33	On p. 12 of the Pioneer (2000) report, it is stated that a 0- to 3-in. deep (0-7.6 cm deep) soil sample was used "because it is representative of the portion of the soil column that most people routinely contact." However, people do not routinely come into contact with soil below the surface layer down to a depth of 3 in. (7.6 cm). Soil samples up to 3 in. (7.6 cm) deep might provide the closest available surrogate for the soils that people actually come into contact with. In some circumstances (such as longer exposures), if there is sufficient mixing of surface soil through this depth range for the concentrations in the entire depth range to be of relevance, those soil samples might be appropriate. Discussion of the potential mixing rate of surface soils, its effect on the soil-contact scenario, and the collection of surface-only samples (the top millimeter or so) should be considered in the planning of future studies.	The PIONEER report has been revised to state: "Soil depth is an important consideration because airborne COPCs are deposited on the surface of the soil. They can migrate deeper into the soil by mixing, tilling, digging, or, to a limited extent, natural processes. COPC concentrations in soil generally decrease with depth – due to dilution resulting from mixing with clean soil. Therefore, collecting a soil sample deeper than humans will come in contact with may underestimate the risks by diluting the sample, and collecting a soil sample at a shallower depth than people regularly come in contact with may overestimate the risks. The samples collected from 0 to 3 inches were evaluated in the risk assessment. This depth interval was selected because it is representative of the portion of the soil column that most people routinely contact." Additionally, the Agency for Toxic Substances and Disease Registry (ATSDR) from the U.S. Public Health Service has defined surface soil as the top 3 inches.

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Page 33	As discussed on p. 14 of the Pioneer (2000) report, duplicate field results are treated as independent observations when calculating summary statistics and exposure-point concentration estimates. Such treatment doubles the weight placed on the concentration at a single place and time, and is not appropriate. Field or laboratory duplicates should be averaged before summary statistics are calculated.	The statement in the Pioneer report has been revised. Duplicate air samples were only collected at the GEMB for all chemical groups except for mercury, which was collected at every site. For ambient air sampling, duplicates were used to determine precision of sampling and they were not included in the determination of exposure concentrations. Therefore there was no double weight treatment placed on the concentration at a single place and time. Duplicate soil samples were collected at every area of concern and used as discreet samples. However, since the risk contribution was 95% from air, double weighing caused by the use of duplicates as discreet samples does not contribute to a significant change in the total risk.
Page 33	NEHC should include a justification for the use of the QL/2 method to strengthen the discussion of the risk assessment with mean concentrations.	The PIONEER report has been revised to state: "Per USEPA Risk Assessment Guidance for Superfund, if a constituent was detected in a sample, then one-half the sample quantitation limit was substituted as the concentration for all of the non-detected values when calculating exposure point concentrations (U.S. EPA, 1989)."
Page 33	Page 14 of the Pioneer (2000) report mentions a procedure called "Compound Rules of Decision". The procedure is neither described nor referenced, and it is not stated whether the circumstances under which it is supposed to be invoked ever occurred in the risk assessment. Some description of the procedure, the specific circumstances under which it was invoked in this risk assessment, and a citation should be included.	The steps in the CROP decision rule have more clearly described in the revised report. In instances where analytical overlap occurred (i.e., results for a constituent were reported by different analytical methods for the same sample), a set of decision rules, called Compound Rules of Precedence (CROP), was applied to the data to select the concentration that should be used for risk assessment purposes (i.e., development of exposure point concentrations). CROP prioritizes the selection based on the sensitivity of the analytical methods involved in the overlap. However, other factors, such as the whether or not the analyte was positively detected by both methods, are also considered. The CROP rules used to reduce the analytical data and develop

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π		the exposure point concentrations presented in Section 2.5 are described below.
		Analytical overlap was identified only in ambient air data for constituents in the following methods:
		 Gas Chromatography/Mass Spectroscopy (GC/MS; EPA Method TO-15) [CROP Level of Precedence: 1] and Semi- Volatile Organic Compound (SVOC; SW8270) [CROP Level of Precedence: 2]
		 GC/MS (EPA Method TO-15) [CROP Level of Precedence: 1] and Aldehydes/Ketones (EPA Method TO-11) [CROP Level of Precedence: 2]
		3. Mercury (Gold foil amalgamation) [CROP Level of Precedence: 1] and Hopcolite-Resin Mercury [CROP Level of Precedence: 2]
		A conditional level or precedence was used so that results with a higher level of precedence (indicated by the lower number) were used to develop EPCs in all cases except in instances where the result of a constituent with a higher level of precedence was not detected and the result for the lower level of precedence was detected. In these instances the lower level of precedence result was used to develop the EPC.
Page 33	Section 2.3 (Pioneer 2000; p. 15) describes the initial screening of chemicals of concern (COCs). Such a screening, if carried out as stated, would prevent the risk assessment from addressing its first objective, because the overall risks of the site would include those due to background concentrations. It appears that the screening was carried out for the soil measurement but not for the air measurements.	Since we have further clarified that our objectives only relate to impacts from ambient air only, background soil screening is appropriate and it fits the objective because we are screening for chemicals normally found in soil, not in air.

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	The implications of that should be discussed.	
Page 34	On p. 16 (Pioneer 2000), the descriptions of the calculation of the UCL95 estimates of mean concentration are not clear for any distribution, an estimate of the UCL95 of the mean is required. How a distribution is tested for normality or lognormality is not specified, nor are the criteria applied to the results of any such test. If the distribution is neither normal nor lognormal, further analysis might be desirable before an approach based on normality is accepted. The description is also inadequate in that the estimates adopted for the UCL95 of the mean are not given. Many estimation procedures are available (such as, analytic estimates based on the t-distribution for normals and on Land's procedure for lognormals, minimum variance unbiased estimates, likelihood-based estimates, and bootstrap and jackknife estimates applied to any of these or others), and the procedure used should be stated.	The PIONEER Report was revised to state: "Analytical data summary statistics for each AOC, media, and constituent are presented in Appendix A. The exposure point concentrations calculated for this assessment are presented in Appendix B. The underlying distribution for each COC was determined using either the Shapiro-Wilk test (in cases when there were less than 50 data points) or D'Agostino's test (when there were more than 50 data points). The alpha level for each test was 0.05. Results identified in Appendix A as "Unknown" mean that the distribution is not normal or lognormal at the 0.05 significance level." Also, a new section on Statistical Formulas use to calculate summary statistics was added to Section 2 of the PIONEER Report.
Page 34	Page 22 (Pioneer 2000) describes the term MF (defined by Pioneer as exposure-pathway- and constituent-specific modifying factors, such as percutaneous absorption rate) in the first equation is described as having "variable units". With the definitions given for the other variables in the equations, MF is dimensionless.	The units for MF have been changed to read "MF = Exposure Pathway and Constituent Specific Modifying Factors (e.g., percutaneous absorption rate) (unitless, unless the units of C or HIF vary from units listed above)."
Page 34	Page 22 (Pioneer 2000), has the following explanation of how the exposure parameters were chosen for the RME case: Each variable in this equation has a range of possible values associated with it. The intake variable values for a given pathway are selected so that the combination of all intake variables results in a realistic upper bound estimate (or RME) of	For the purposes of this risk assessment, the Navy decided that it was important to clearly evaluate the impact of the exposure point concentrations on the risk assessment. Therefore, all of the exposure assumptions, except the exposure point concentrations, and the toxicity values used to calculate the risks were consistent between the average and RME cases. Consequently, the Navy can directly compare the long-term average and long-term upper bound risks when making risk management

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	the possible exposure by that pathway. The same values, however, were used for the average case. In the risk assessment, the average case appears to use RME estimates for all exposure parameters except the exposure-point concentration, where the difference between average and RME cases is the difference between an estimate of mean concentration and a UCL95 on the mean concentration (see, for example, Tables 3-2 through 3-6, particularly their footnote c). That is not the usual meaning of average for such exposure scenarios and is misleading. The typical approach for estimating an average or central-tendency case is to obtain an average for the whole population that is exposed by using exposure parameters that represent central-tendency values (such as means or medians). The ranges or confidence limits around the central-tendency values should also be presented.	decisions. This approach may not be considered "typical" but it does provide the Navy with information regarding the range of risks at NAF Atsugi, Japan.
Page 34	In Table 3-2, footnoted confuses the "fraction from contaminated source" with "outdoor and indoor exposure to soils". Although those concepts might overlap in some circumstances, they are distinct and do not overlap in this case. The formula presented appears to have been adapted in such a way that the "fraction from a contaminated source" represents the "fraction of time indoors". The explanation in the table should explain that better.	The Text and footnote in Table 3-2 has been revised and the FI now reads "Fraction of ingested soil/dust from outdoor source."
Page 34	In Table 3-3 (p. 23), footnote e does not explain how 150 mg/d is "the midpoint" between 50 and 200 mg/day.	The footnote has been revised to read "Based on professional judgment, the adolescent incidental soil ingestion rate was chosen as the midpoint between the residential child and adult values (i.e., 200 mg/day and 100 mg/day). It was assumed that an adolescent would potentially receive more soil contact than an adult, but that this contact was likely to be less than a child under age 6."

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Page 34	Pages 33 and 68 (Pioneer 2000) mention an inhalation RfD, the second time in the context of EPA's IRIS database. That RfD was probably derived from an RfC. The term "inhalation RfD," however, is not used by EPA and is confusing.	A note stating the inhalation RfDs were derived from RfCs was added to the report.
Page 34	Page 84-85 (Pioneer 2000), in the context of a comparison between the golf-course site and the GEMB site, states that "the only difference between the airborne concentrations, and consequently risk, at the GEMB and the airborne concentrations at the Golf Course should be emissions associated with the SIC." That would be correct only if the "Background + Other Point and Non-Point Sources (emissions)" affect the two sites equally. That hypothesis was not established or tested at any point in the project.	This section of the revised PIONEER report has been updated to present more detailed information on the "Upwind vs. Downwind" analysis. A formal test of the hypothesis that the only difference between airborne concentrations at the GEMB are emissions from the SIC (i.e., "Background + Other Point and Non-Point Sources (emissions)" affect the GEMB and Golf Course equally) was not performed as part of this assessment. However, the total risks, and the risks calculated for the vast majority of analytes assessed in the "Upwind vs. Downwind" analysis, support the hypothesis.
Page 34	On pp. 84-85 (Pioneer 2000), the methods adopted for the comparison between the golf-course and GEMB sites are not adequately explained. For example, there is no information in the documentation as to which particular days were used for the comparison. Even if the days were correctly selected, the results presented in Table 5-10 cannot be interpreted without further information on the method because some approaches to producing such values are statistically invalid.	In the revised PIONEER report the analytical data for the days evaluated in the Upwind vs. Downwind Analysis are presented in Appendix D. Also, the summary statistics for the Upwind vs. Downwind Analysis are presented in Tables A-10 and A-11 of Appendix A of the PIONEER 2000 Report.
Page 35	The committee can conceive of several ways of generating the values in Table 5-10. 1. Take the concentrations measured at each site for each chemical on the selected days, and find the average and an upper 95th percentile estimate on that average for the concentration of each	In an earlier section of their peer review document the subcommittee disagreed with NEHC's upwind downwind approach to determine the risk contribution from the incinerator and recommended using dispersion modeling combined with correlation/regression analysis. The approach recommended in this comment is different than the dispersion modeling

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	chemical at each site. Calculate an "average" and "RME" risk	approach. It uses the NEHC's upwind downwind approach, but
	estimate for each site on the basis of the two sets of concentration	recommends a different way of calculating the average and the RME
	estimates. The entries in Table 5-10 could then be the differences	concentrations for the upwind and the downwind locations. To obtain
	between sites. Although the "average" estimate so generated for a site is meaningful, the "RME" site differences so obtained have no	clarification on which approach would be the preferred approach, NEHC sent a written request to the subcommittee for instructions on how to use
	statistically valid meaning. This method appears to be the closest	either approach. The subcommittee chose not to reply to our request for
	approximation to what was meant by "RME" in the rest of the	instructions in writing, but in a phone conference that included only two
	document; but the differences between such "RME" values cannot be	members of the subcommittee (perhaps the reviewers that provided the
	interpreted.	initial recommendation). During the phone conference, the two reviewers
	2. Take the concentrations measured at each site for each	withdrew their recommendation to use a dispersion modeling approach and
	chemical on the selected days, calculate day-by-day concentration	did not articulate instructions for neither approach. Instead they
	differences for each chemical, and compute the average of these	recommended a third approach, but indicated that they could not ensure that
	differences in daily concentrations for each chemical over all days	the approach would provide valid results. As the subcommittee admits that
	selected and upper 95th percentiles on such average differences.	the third approach includes just as much uncertainty as the NEHC approach,
	Calculate the risk estimate differences for Table 5-10 on the basis of	NEHC chose to retain the approach that was initially presented to the
	the two measures ("average" and "RME") of concentration	subcommittee for review. The approach used by NEHC was actually the
	differences. The "average" so obtained will be the same as for	approach recommended by the previous Committee on Toxicology that
	approach 1, but the "RME" value will be different and will have no	reviewed the 1998 screening risk assessment and stated the following:
	statistically valid meaning.	
	3. For each selected day, calculate at each site a risk-	"Another approach that might be useful for getting a rough estimate of the
	weighted sum of concentrations of all the chemicals in question,	contribution of incinerator emissions to ambient air, relative to the
	selecting the risk weighting so that summing over all days would give a risk estimate (roughly speaking, a risk estimate for that day for that	background, would be to compare results from Location 1 (upwind site) with those from downwind locations on days when the wind direction is out
	site). Take the difference between the values for each site to obtain a	of the south-southwest and relatively constant."
	series of daily risk-weighted differences. Obtain the sum and the	of the south-southwest and relatively constant.
	upper 95th percentile estimate on the sum of the risk-weighted	
	differences as "average" and "RME" estimates. The "average" value	
	so obtained will be the same as approaches 1 and 2, but the "RME"	
	will be a statistically valid estimate that can be interpreted.	
	The statistical uncertainty associated with the "average" column in	

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THE STATE OF THE S	Table 5-10 is not presented—the differences might not be statistically distinguishable from zero. The third of the approaches just summarized provides a series of daily values that would allow calculation of statistics on differences between sites, including the statistical significance of such differences. In contrast, the first and second approaches (and many other possible ones) cannot provide such information. In any case, the values in Table 5-10 cannot be used to draw unequivocal conclusions about the contribution of the incinerator without an evaluation of the hypothesis that there is no difference in the absence of the incinerator. Moreover, such values as those in the table would allow an estimate only of the contribution of the incinerator to the differences between the GEMB and golf-course sites, not of the average contribution to actual populations or individuals.	
Page 35	It is pointed out on pp. 85-86 (Pioneer 2000) that the majority of the hazard-index estimates is contributed by acetaldehyde, acetonitrile, acrolein, and PM ₁₀ , but it is not pointed out that of those major contributors, only PM ₁₀ could be associated with incinerator emissions in the analyses presented. The "upwind" hazard index or risk estimate is higher than the "downwind" for several chemicals in Tables 5-11 and 5-12; that situation would not be possible (except for the inherent uncertainties) if the hypothesis that there are no differences in the absence of the incinerator is correct. Although such effects could be due to the uncertainties involved, the uncertainties are not discussed.	A discussion of the uncertainties associated with the "Upwind vs. Downwind" Evaluation has been added to the revised PIONEER report. The comment that "only PM ₁₀ could be associated with the incinerator" is not correct. PM ₁₀ was identified in the correlation analysis performed by Radian as being associated with the incinerator. However, the correlation analysis only identified 6 analytes (i.e., hydrochloric acid, dioxin 2,3,7,8-TCDD-TEQ, lead, cadmium, arsenic, and PM10) as having a statistically significant relationship between concentration and percentage downwind of the SIC. The rationale for the decision to use the Upwind vs. Downwind method was based on experience from previous risk assessments performed on municipal waste incinerators that indicate that multiple chemicals (i.e., 50 – 100s), some of which are highly toxic, are likely being emitted from the SIC (USEPA, 1998a). There are uncertainties with the Upwind vs. Downwind (primarily associated with the small sample size) approach, which are demonstrated by the fact that a few analytes have risks higher at

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#		the upwind site rather than the downwind site. However, the risks for most analytes were higher at the downwind site that the upwind site and the total risks were significantly higher at the downwind site than the upwind site – which is consistent with the hypothesis that formed the basis of the Upwind vs. Downwind evaluation.
Page 36	The recommendations section of the Pioneer risk-assessment document (Pioneer 2000) contains many recommendations that are not based on the findings and conclusions presented in the report.	Risk Management recommendations have been deleted from both the NEHC and the PIONEER reports.
	The primary recommendation (recommendation 1, p. 92) mentions specific periods (32 and 98 months) that are not mentioned in the report.	The majority of the recommendations previously stated in the NEHC and Pioneer reports were based on the level of concern that the Navy and EPA would have in the U.S. that would trigger policy actions to protect our military and civilian personnel. They were common sense administrative and public health practices.
		Findings and conclusions in the NEHC report address the calculated cancer risk by EPA methodology, based on policy. The reasonable maximum cancer risk for children who are residents at the base for one tour of duty (36 months) is 1.1 X 10 ⁻⁴ . Therefore to reduce the cancer risk level to 1 X 10 ⁻⁴ the recommended tour length should be 32 months for children and 98 months for adults. Although NEHC is well aware that the calculated risk is not an exact number because of the uncertainties associated with the risk
		assessment process, in the U.S. this is a level of concern that either would shut down an incinerator such as the Shinkampo incinerator complex and any other sources of air pollution, or cause action to be taken to reduce emissions. Since the U.S. government depends on the Government of Japan to enforce their environmental regulations regarding uncentralled.
		to enforce their environmental regulations regarding uncontrolled incinerator emissions, the easiest way to protect U.S. citizens was to recommend shorter exposure duration by decreasing the tour lengths to reduce risk levels to less than 10 ⁻⁴ for children and adults.

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UNITED STATES ENVIRONMENTAL PROTECTION AGENCY

OFFICE OF RESEARCH AND DEVELOPMENT

National Center for Environmental Assessment Washington, DC 20460

NCEA Washington Office (8623D)

October 28, 1998

MEMORANDUM

SUBJECT: Review of, "Results of March 1998 Soil Sampling NAF Atsugi, Japan", dated

August, 1998

FROM: Matthew Lorber

National Center for Environmental Assessment Office of Research and Development, EPA

TO: Yvonne Walker

Navy Environmental Health Center

THRU: William H. Farland, Director

National Center for Environmental Assessment Office of Research and Development, EPA

Dwain Winters, Director Dioxin Policy Project

Office of Prevention, Pesticides, and Toxic Substances, EPA

At your request, I am providing comments on above noted report produced by Radian for the Navy Environmental Health Center. I will restrict my comments to the technical content of the report and not on any assessment of the potential health risks to Navy personnel posed by exposure to 2,3,7,8-TCDD and other dioxin-like compounds (referred to simply as dioxin for the remainder of this memorandum) in the air or soil as measured in this study (with the exception of my initial comment below on the use of risk-based soil levels). Also, I am only going to comment at this time on the results for dioxin. At a later date, NCEA may be able to assist in the evaluation of some of the other data in this report. I hope these comments are useful and avail myself for any further assistance on this or other Navy sites.

I can make the following overall observations based on this soil study as well as some air

concentration data which you recently transmitted to me. These observations are similar to observations we made in the study of an incinerator known to have been a significant source of dioxin release in the US - the waste-to-energy municipal solid waste incinerator in Columbus, OH. I have sent you information on this site and can provide further details if you need them.

From the soil study and the air monitoring data, I have observed:

- 1) high soil concentrations of dioxin, over 100 ppt dioxin toxic equivalents (abbreviated hereafter as TEQs), near to and north of the incinerator (trend samples TRND-S004, S005, S008, S009, S010);
- 2) the similarity in congener profile of these very high samples among themselves and with samples of lower concentration downwind;
- 3) the further similarity of this characteristic profile with a characteristic profile from the Columbus site; and
- 4) the finding in the summer 1998 air sampling program which showed that, on nearly all sampling dates, that the air sampler with the highest air concentration the ground electronics site, was the sampling site in the most downwind direction from the Atsugi incinerator as compared to the other air sampling sites the golf course, residential towers, elementary school, and the criteria site.

Based on these observations, I would conclude that, unless another very obvious source of dioxin emissions can be located, the Atsugi incinerator is the source of elevated dioxin concentrations found in the air and the elevated dioxin concentrations found in the soil near the incinerator.

Here are my more detailed comments on this report:

1) Use of RBSLs to evaluate the impact of dioxin soil concentrations is misleading. Radian focused its evaluation on the use of EPA Region 3 "Risk Based Soil Levels", or RBSLs. These are preliminary screening concentrations and correspond to a lifetime cancer risk of 10⁻⁶ assuming traditional exposure scenarios for contaminated soils. The RBSL for 2,3,7,8-TCDD is 4.3 ppt. In the Radian report, this has also been applied to the toxic equivalent concentration of the 16 other dioxin-like dioxins and furans. While the RBSLs may be useful for other contaminants, they are certainly not as useful and perhaps misleading when used for dioxins. This is because the rural background concentration of dioxins in soil in the US is in the neighborhood of 5 ppt TEQ, and in urban settings, the background is more in the range of 5 to 30 ppt TEQ. These RBSLs are probably more useful for contaminants which, unlike dioxins, are not widespread in the environment at low levels. It is important to note that none of the soil concentrations exceed the soil concentration which EPA's Superfund Office uses for residential land uses in its clean-up decision making - a level of 1 ppb TEQ. This level is also supported by the Agency for Toxic Substances and Disease Registry (ATSDR). I have sent you

documentation on this 1 ppb level from these two sources. In summary, I would not rely on the 4.3 ppt RBSL for 2,3,7,8-TCDD for <u>any</u> interpretation of the results of this study.

- 2) The focus on RBSLs to present the results for dioxin in this report made it difficult to easily evaluate the material. It would have been helpful to have provided tables showing TEQ concentrations for all soil sampling points. I have calculated TEQ as well as Total concentrations, and they are on the attached table. The "Total" concentrations here are defined as the sum of the concentrations of 17 dioxin-like congeners. I developed these by hand inputting them into a spreadsheet. If you have these numbers calculated elsewhere, please let me know if your TEQ concentrations are the same as mine, so that I can correct my spreadsheet. I note that Radian used the International Toxicity Equivalency Factors (I-TEFs), and I have done the same in my calculations. The WHO has recently proposed changes to this scheme including reassignment of the TEF for 1,2,3,7,8-PCDD to 1.0 (from 0.5), and for OCDD and OCDF to be reassigned to 0.0001 (from 0.001). The TEQs calculated with these TEFs are virtually the same as those calculated using the I-TEFs. For example, the following are TEQs for samples calculated with I-TEFs versus the newer proposed WHO-TEFs: Sample S001 - 47 (I-TEFs) vs. 50 (WHO-TEFs) ppt, S002 - 80 (I) vs. 84 (WHO), S003 - 66 (I) vs. 70 (WHO), S004 - 609 (I) vs 643 (WHO), S005 - 250 (I) vs 268 (WHO), S006 - 642 (I) vs 663 (WHO), S007 - 98 (I) vs 104 (WHO). As seen, TEQs calculated with the WHO TEFs are a bit higher, but not significantly, than TEQs calculated with the I-TEFs. I have also constructed congener profiles, and several of these are attached as well to support comments I make below. These are constructed as the fraction of the concentration that each congener makes to the total concentration, with "total" defined as I defined it earlier in this comment.
- 3) The sample, TRND-S006, appears to be an anomalous result warranting further evaluation. One important observation I can make up front is that the dioxin found in Trend sample TRND-S006 appears different than the dioxins found in any other sample. It was very high and drove the observed trend, as seen in the trend analysis figures, of a high soil level located to the east of the incinerator. It is not clear from the data supplied that this is a real trend or a trend driven by an anomalous soil sample. The three important clues that this soil sample is different from others is:
 - a) The first clue of concern for TRND-S006 can be easily seen in the gray and white trend figure for TEQs shown in Appendix G. There, a peak is easily seen directly on top of TRND-S006, at TEQ = 642 ppt, reducing to all its neighboring soil sample sites, including TRND-S001 (TEQ=47 ppt), TRND-S017 (TEQ=14 ppt), TRND-S018 (TEQ=21 ppt), TRND-S007 (TEQ=98 ppt), and TRND-S002 (TEQ=80 ppt).
 - b) The second clue is from an examination of congener profiles. I've attached the congener profiles from the next five highest soil samples, including TRND-S004 (TEQ = 609 ppt), TRND-S009 (TEQ=326 ppt), TRND-S008 (TEQ=312 ppt), TRND-S005 (TEQ=250 ppt), and TRND-S007 (TEQ=98 ppt). The congener profile for these all look similar (just eyeballing the figures not statistically evaluating them), with OCDD dominating the profile, explaining from just under 40% to above 60% of the profile. For

TRND-S006, both 1,2,3,4,6,7,8-HpCDF and OCDF explain more of the profile than OCDD, and the OCDD fraction is 0.16.

c) The third clue is from the other high soil samples. Besides all having a similar profile, these are all located more in the historical downwind direction from the incinerator, North/South (no samples could be taken to look for a high trend south of the incinerator). TRND-S006 is in the easterly direction from the incinerator, a direction in which the wind blows very infrequently. As I will discuss further, this trend of finding high concentrations downwind from an incinerator source with that characteristic profile (i.e., high OCDD with smaller peaks for the hepta dioxin, the hepta furan, and OCDF) was exactly what we found in our site investigation of the Columbus site.

We can look also to our study of the Columbus site to find a fourth clue as to why TRND-S006 may be a sample not to consider further for trend analysis. There were 5 soil samples which we did not use from our Columbus study to evaluate the overall trend of depositions of dioxins from emissions from the incinerator. Three were samples taken on-site - within the incinerator property. All these were high concentrations with a congener profile similar to stack gas emissions and distinctly different than the "deposition" profile (i.e., the profile typified by high OCDD). We speculated these high concentrations were due to ash drift from open storage pits or from their proximity to the road used to transport ash away from the incinerator. The other two were located about a kilometer away from the incinerator with also high concentrations and an atypical congener profile. We went back to those two sample sites to resample them. For one of them, we could locate the exact spot and a nearby (a few feet away) sample continued to show very high concentrations. Based on the color of the soil, we suspected that the site had been impacted by ash from a nearby sewage sludge incinerator. For the second, we couldn't find the exact same spot, but a location as close as we could ascertain showed more typical depositional characteristics - lower concentrations and the high-OCDD profile.

I would recommend further evaluation of TRND-S006. It is possible that a trend of high concentrations may exist east of the incinerator, but I would guess that it is doubtful. One thing to ask first is to confirm that the analytical results were accurate. If, in fact, the OCDD concentration for TRND-S006 were 25200 ppt instead of 2520 ppt (as listed in Appendix H), then you would have a congener profile consistent with all the other congener profiles of this study and a more real possibility of a trend of high soil concentrations east of the incinerator. The next thing I would do is take more samples east of the incinerator. I would resample the same spot as TRND-S006 (if it can be found), a second sample maybe twenty feet or so from the spot to evaluate how immediate the possible contamination is, and perhaps a handful of other sites between TRND-S006 and its neighbors TRND-S001, S017, S018, S007, and S002.

4) The cluster of soil samples with high dioxin concentrations just north of the incinerator, and the similarity of the congener profile for these samples and others of lower concentration on the site as well as with congener profiles from the Columbus incinerator site, strongly suggests that our experience with the Columbus incinerator is similar to the situation at NAF Atsugi. Also, like NAF Atsugi, we found the highest air concentrations at an air sampling

station located downwind of the Columbus incinerator. When we discussed the merit of a soil sampling program with yourselves several months ago, we said that if in fact the incinerator was the major source of dioxins in the NAF Atsugi environment, then you would likely find a "footprint" of high soil concentrations marching away from the incinerator in the downwind direction. This is what we found in our site investigation of the Columbus incinerator, an incinerator found to be emitting large amounts of dioxin. Not only was this same soil trend found, but you also found the highest air concentrations just downwind of the incinerator. While these observations indicate that the source of dioxin in the NAF Atsugi environment is the incinerator, one cannot imply a level of dioxin emissions from this incinerator with the data. It is important to note that the Columbus incinerator was a tall stack emitting to a flat terrain, while the incinerator near NAF Atsugi has a shorter stack emitting from a river valley. Unfortunately, a comparison of the rate of emissions between the Columbus incinerator and the one at Atsugi cannot be made because you are unable to sample the stack.

This cluster of soil samples with high concentrations includes trend samples TRND S-004, 005, 008, 009, and 010 with concentrations ranging from 221 to 609 ppt TEQ. Sample TRND-S007 was also high with a TEQ concentration of 98 ppt TEQ. No other samples exceeded 100 ppt TEQ (except TRND-S006 discussed above). The other identified trend is that the high samples have a very similar profile to lower samples on-site. This profile is typified by elevated OCDD, with secondary peaks found for dioxin congeners 1,2,3,4,6,7,8-HpCDD, 1,2,3,4,6,7,8-HpCDF, and OCDF. This is remarkably similar to the trend we found at the Columbus site. The typical soil profile there had perhaps more OCDD, above 60%, as compared to these typical profiles, 40% to greater than 60%. Attached are the profiles for these high soil samples, as well as for some lower trend samples TRND - S021, 28, 30, 31, and 33. As seen, the total and TEQ concentrations drop but the same four congeners dominate the profile, with perhaps some proportional elevation of OCDD in the lower soil samples as compared to the samples nearer the incinerator. A discussion of the soil profiles at the Columbus site is in the literature article we published on our site investigations, which you have.

5) There appears to be a trend of lower surface soil concentrations associated with lower sand content and bare soil conditions. Some of the sites showed unusually low concentrations, such as S-004 and -007 of the child development center, S-001, 002, 005, and 006 of the elementary school, and others. These had total concentrations around 50 ppt or so, and TEQ concentrations near 1 ppt. The photographs of some of these sites indicated bare soil conditions, near heavily trafficked area such as playgrounds. Surface vegetation serves as an effective capture mechanism for dioxins depositing from the air. If the areas had imported sand and/or had been recently developed in other ways, than they also would not be good sites for evaluating long term atmospheric depositions. Finally, low surface soil concentrations may also be associated with sandy soil conditions, which would encourage leaching below the surface 3 inches. There is perhaps some evidence in this report of low dioxin concentrations associated with sandy top soil. There were soil characteristics derived for several of the soil samples. The following table identifies soil sites where sand/silt/clay measurements were reported, and the associated total and TEQ concentrations. As seen here, there may be a trend of lower surface soil concentrations associated with higher sand contents. With sand contents higher than 80% (n=5), the average

total and TEQ concentrations were 246 and 5 ppt, between 60 and 80% (n=4), the average total and TEQ concentrations were 1420 and 70 ppt, and at less than 60% sand (n=3), the average total and TEQ concentrations were 2933 and 75 ppt. There is also evidence in the open literature of dioxin leaching below sandy surface soils to peak, in fact, up to 30 cm below the surface.

Site Identification	Percent Sand	Total, ppt	TEQ, ppt
Child development: S004	81	18	<1
Child development: S006	85	469	7
Elementary School: S002	82	40	<1
Elementary School: S004	81	518	10
Reference Site 1: S001	72	1540	62
Reference Site 2: S005	66	490	140
Residential Tower: S003	59	4310	87
Residential Tower: S005	86	186	7
Trend Sample: S001	57	1460	47
Trend Sample: S012	52	3030	92
Trend Sample: S020	63	2430	68
Trend Sample: S029	66	1220	9

6) Another trend that can be identified is that soil concentrations on the bare fields near the runway on the eastern half of the base are lower than concentrations found on the western half of the base, in areas of development. This is not a trend that can be associated with atmospheric depositions from the incinerator. The TEQ soil concentrations for the trend samples on the eastern half of the base include 14, 21, 23, 8, 19, 9, 24, and 35 for samples S-017, 018, 019, 024, 025, 029, 031, and 035, for an average of 19 ppt TEQ. TRND-S001 and 023 on the eastern half are higher at 47 and 51 ppt TEQ - these are discussed below in the next bullet. A second cluster we have noted above includes the very high samples in the predominant downwind direction near the incinerator. These include TRND S-002, 003, 004, 005, 007, 008, 009, 010, 012, and 013. The average TEQ concentration for these samples is 210 ppt. A final set of trend samples are ones on the western part of the base not discussed so far including TRND S-11, 14, 15, 16, 20, 21, 22, 26, 27, 28, 30, and 32. The average TEQ concentration for these westerly samples is about twice that of easterly samples, 36 ppt. Maybe this trend is strong statistically, but we have also found generally that higher soil concentrations in urban developed

soils as compared to rural, less developed soils. Some possible causes, not including tall stack emissions (which do not appear to explain the difference here), are the use of herbicides which contain trace dioxins (2,4,-D, for example), the use of pentachlorophenol for wood treatment (dioxins are found at significant concentrations in PCP; there is evidence of high dioxin concentrations near the base of PCP-treated utility poles), vehicular traffic (emissions are associated primarily with diesel vehicles), wood burning for home heating, and so on. In any case, high concentrations such as 83 ppt (TRND -S026), 57 ppt (TRND - S016), 90 ppt (Elementary School S-007), and 87 ppt (Residential Towers S-003) do not appear to be due to deposition from the incinerator, but maybe other base activities.

7) The slightly higher concentrations of 47 and 51 ppt TEQ at TRND S-001 and S-023 may or may not be indicative of another source of contamination near the southeastern base of the facility. The suggestion of another sources arises because of the findings there of high PAHs in soil. The evidence for another possible source is actually weak. The wind rose diagram does suggest that TRND S-001 and -023 are in a much more downwind direction as compared to all of the other bare field runway samples on the eastern part of the base. Also, the elevated PAH findings are only true for TRND S-001, not TRND S-023. Still, more investigation may be warranted, if only to be able to explain the PAH findings for TRND S-001.

Key for Trend Figures

Congener #	Congener
1	2378-TCDD
2	12378-PCDD
3	123478-HxCDD
4	123678-HxCDD
5	123789-HxCDD
6	1234678-HpCDD
7	OCDD
8	2378-TCDF
9	23478-PCDF
10	12378-PCDF
11	123478-HxCDF
12	123678-HxCDF
13	123789-HxCDF
14	234678-HxCDF
15	1234678-HpCDF
16	1234789-HpCDF
17	OCDF

For these trend figures, the "total" concentration of dioxin in the soil is calculated as the sum of the concentrations of the 17 dioxin-like congeners. The individual congener concentrations are absolute concentrations - they are not adjusted to a 2,3,7,8-TCDD equivalent concentration. The "fraction of total" on the y-axis is the fraction each congener's concentration contributes to the total concentration.

TOXICOLOGICAL EVALUATION OF CHEMICALS OF CONCERN AT NAF ATSUGI LACKING KNOWN TOXICITY VALUES

This appendix presents the details of a comprehensive analysis of the potential toxicity associated with 86 chemicals involved in the Human Health Risk Assessment at NAF Atsugi, for which published toxicity values were not available. An extensive search of all available scientific peer-reviewed databases was conducted to determine if sufficient toxicological information existed to calculate an interim Inhalation Reference Dose (RfDi) or Cancer Slope Factor (CSF). In cases where sufficient toxicological information was available, an RfDi was estimated using the same methodology recommended by the National Academy of Sciences and later adopted by the EPA. The objective of this study was to derive missing toxicity values so chemicals with missing values would not be excluded from the risk assessment resulting in an underestimation of risk.

Ten different U.S. regulatory or governmental agency databases and twenty toxicological databases were extensively queried. Over 11,000 abstracts were reviewed and over 300 of these were collected and further reviewed to identify quantitative toxicological information for deriving toxicity values. It was possible to develop interim RfDi for a subset of the 86 chemicals. The interim RfDi values were used to estimate a Hazard Index. There was no evidence for suspecting potential carcinogenic effects from any of the chemicals. Based on these estimations, it was determined that the 86 chemicals do not likely pose unacceptable threats to human health at the concentrations detected at NAF Atsugi.

NAF ATSUGI TOXICOLOGICAL EVALUATION

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1 EXECUTIVE SUMMARY

This study is a comprehensive analysis of the potential toxicity associated with 86 chemicals that the Navy Environmental Health Center (NEHC) identified as not currently having USEPA toxicity values. An exhaustive search of all available databases and scientific peer-review databases was conducted to determine whether sufficient toxicological information exists to calculate an interim Inhalation Reference Dose (RfDi) or cancer slope factor (CSF). For those chemicals where it was determined that sufficient toxicological information was available, an RfDi was estimated using the identical toxicological methodology recommended by the National Academy of Sciences and later adopted by USEPA. This methodology is currently used to develop toxicity values that are presented in USEPA Integrated Risk Information System.

During the course of this toxicity assessment approximately 10 different U.S. regulatory or governmental agency databases and 20 toxicological databases were extensively queried. Over 11,000 abstracts from peer-reviewed scientific publications were first reviewed and over 300 of these were collected and further reviewed to identify useful quantitative toxicological information. Based on careful review of these published studies, it was possible to develop an interim RfDi for a subset of the 86 chemicals.

In the final analysis, the interim RfDi values calculated in this report were used to estimate a Hazard Index for the three areas at NAF Atsugi. Based on these estimates, it was determined that the 86 chemicals do not likely pose unacceptable threats to human health at the concentrations detected in each of the three areas.

2 INTRODUCTION

The primary purpose of this document is to evaluate whether toxicological information exists on 86 chemicals (identified by NEHC) that have no USEPA-verified toxicity values to answer the following question:

Should the Navy be concerned about exposures to 86 chemicals that were characterized as having "no available toxicity information" and were not evaluated further in the NAF Atsugi risk assessment?

The information provided in this report will be used to determine whether it is possible the Navy has underestimated carcinogenic risks and systemic (noncarcinogenic) human health hazards associated with exposure to chemicals that were detected at NAF Atsugi. These chemicals were not quantified in the risk assessment report because no USEPA-verified toxicity values have yet been developed.

The question of whether these chemicals could unknowingly pose unacceptable threats to human health risks has been raised by the National Academy of Sciences (NAS) in their "Review of the U.S. Navy's Human Health Risk Assessment of the Naval Air Facility at Atsugi, Japan," published by the National Research Council and delivered to the Navy Environmental Health Center (NEHC) in January 2001. On page 12, the following NAS review comments are made:

According to the Pioneer report (2000); p.34, toxicity values for 86 of the 246 chemicals of concern were not available from the secondary sources consulted (US EPA's Integrated Risk Information system, IRIS; US EPA's Health Effects Assessment summary Tables, HEAST; US EPA's National Center for Environmental Assessment, EPA/NCEA; and California EPA). Those 86 chemicals were not evaluated further (see Table 4-4 of Pioneer 2000 for list). It is inaccurate to characterize all those chemicals as having "no available toxicity information". Primary literature and many useful secondary sources should be consulted for toxicity information that could be used in some cases to determine whether exposures to those chemicals at Atsugi might be of concern.

In response to this comment, a comprehensive evaluation of current scientific information and databases was conducted to determine if there is any existing toxicological information in the following:

- Environmental regulatory agency toxicological databases regarding exposure to the general public;
- Occupational databases that may provide information on exposure guidelines for workers; and
- Peer-reviewed toxicological publications that provide sufficient information on the inherent toxicity of these chemicals to develop chemical-specific RfDi values.

The primary risk assessment guidance documents used in this toxicity study are as follows:

- U.S. EPA. 1994b. Methods for Derivation of Inhalation Reference Concentrations and Application of Inhalation Dosimetry, EPA/600/8-90/066F, dated October 1994.
- ➤ U.S. EPA. 1995c. Use of the Benchmark Dose Approach in Health Risk Assessment, EPA/630/R-94/007, dated February 1995.
- U.S. EPA. 1996a. Proposed Guidelines for Carcinogen Risk Assessment: Notice dated April 23, 1996. Fed. Reg. 61, No. 79: 17960-18011.

3 SOURCES OF TOXICOLOGICAL INFORMATION

3.1 Background

For risk assessment purposes, chemicals are categorized as either carcinogenic or noncarcinogenic (it should be noted that chemicals classified as carcinogens also produce noncarcinogenic adverse health effects). USEPA's approach to assessing the health threats associated with systemic toxicity is different from its approach to assessing the risks associated with carcinogenicity. The differences are largely due to the (presumed) toxicological mode of action thought to be involved in the two classifications.

As a risk management policy, USEPA has developed the "default" assumption that a small number of molecular events initiated by carcinogenic chemicals can evoke changes in a single cell that can lead to uncontrolled cellular proliferation (neoplasm) and ultimately to cancer. Accordingly, tumorigenesis is referred to as a "nonthreshold" biological response since the Agency assumes that carcinogens theoretically have no level of exposure that does not pose a small, but finite, probability of generating a carcinogenic response. Furthermore, USEPA does not distinguish between benign and malignant tumors. Additionally, no distinction is made between the *mode of action* of different types of carcinogenic chemicals, such as tumor initiators, promoters, and cocarcinogens. Outside the regulatory process, distinguishing between initiators and promoters is generally recognized as important when determining the exposure steps necessary to produce a tumor. Likewise, the vast majority of toxicologists agree that thresholds do exist for many carcinogens. For example, it is estimated that on average more than a million DNA point mutations (involving chemical binding to DNA) occur in an average human daily. DNA repair processes and immunosurveillance are very effective in repairing cellular damage and most toxicologists consider these processes evidence of a threshold. These must be surmounted before neoplastic changes leading to tumor formation can occur. Nevertheless, the Agency has made a policy decision to view all potential carcinogens as lacking a threshold.

In the case of noncarcinogens, which produce systemic toxicity, biological homeostatic, compensating, and adaptive mechanisms exist that must be overcome before a toxic response becomes manifest. For example, there is redundancy in most organs where a large number of cells perform the same or similar function. A significant population must be significantly depleted or compromised before a toxic response is observed. Consequently, systemic toxicity is

assumed to have an identifiable exposure threshold (both for the individual and for populations) below which there are no observable adverse effects and it is this threshold phenomenon that distinguishes a carcinogenic from a noncarcinogenic chemical.

USEPA has developed a verifiable toxicity database: the *Integrated Risk Information System* (IRIS). IRIS presents both noncarcinogenic and carcinogenic toxicological information and toxicity values. While this is the primary source of toxicity values used in Human Health Risk Assessment, the absence of toxicological information should not be interpreted to mean exposures to those chemicals would not produce carcinogenic or noncarcinogenic effects. The lack of toxicological information in IRIS is typically due to internal Agency priorities, lack of funding (for the workgroup), or the absence of published scientifically tenable toxicological studies for particular chemicals.

There is a common misconception that USEPA conducts toxicity studies and develops toxicity values based on these studies. However, with very few exceptions, most USEPA-derived toxicity values are developed from primary peer-review scientific literature database and are not developed from internal Agency studies. The information in IRIS has been developed for use in the context of risk assessment as outlined by the National Academy of Sciences (NAS). The same peer-reviewed toxicological database accessed by USEPA has been exhaustively reviewed for this study. Moreover, the procedures for gathering the information and methods ultimately used to derive RfDi values is identical to USEPA IRIS workgroups. No information was available to develop CSF values.

The primary qualitative and quantitative health hazard information presented in IRIS for noncarcinogenic effects associated with inhaling chemicals in their vapor state is the RfDi. As of January 1991, IRIS and USEPA National Center for Exposure Assessment (NCEA) databases no longer present RfDs for the inhalation pathway. The RfD has been replaced with Reference Concentrations (RfC). However, in order to conduct a risk assessment, RfDi values must be derived (either *de novo* or be converted from RfC values). RfDi values represent the level of environmental exposure at or below which no adverse effect is expected to occur. The RfD is interpreted as an estimate (with uncertainty spanning perhaps an order of magnitude) of daily exposure to the human population (including sensitive subgroups) that is likely to be without appreciable risk of deleterious effects during a lifetime. A chronic RfDi is based on an

assumption of lifetime exposure. It should be evaluated for appropriateness when used to evaluate less-than-lifetime exposure situations. For example, when estimating health hazards associated with human exposure durations significantly *shorter* than a lifetime, the chronic RfD may *overestimate* the health hazard.

3.2 Sources of Toxicological Information and Data

A toxicity assessment begins with a qualitative weight-of-evidence judgment as to the likelihood that a chemical may produce a toxic response. This is termed a hazard identification (sometimes referred to as a cause-effect relationship), which is made independent of consideration of the toxic potency of the chemical. The second step is to quantify the dose-response relationship. That is, once a cause-effect relationship is established between a chemical and a specific toxic response, it must be determined *how much* (or dose) of the chemical is necessary to produce the toxic response. The hazard identification step for each of the 86 chemicals was conducted during an exhaustive review of numerous regulatory databases and review of more than 10,000 abstracts from peer-review scientific publications. The following databases were reviewed for any existing toxicological information or safe exposure limits derived by regulatory agencies, governmental, and scientific organizations.

3.2.1 Environmental Regulatory Databases

- ➤ IRIS: This database was developed and currently updated by USEPA Office of Research and Development (ORD) and USEPA National Center for Environmental Assessment (NCEA). IRIS is a database of human health effects that may result from exposure to various substances found in the environment. Information in IRIS includes the following:
 - 1. Oral reference doses and inhalation reference concentrations (RfDs and RfCs, respectively) for chronic noncarcinogenic health effects; and
 - 2. Hazard identification, oral slope factors, and oral and inhalation unit risks for carcinogenic effects.

- EPA Region 9 Toxicity Information: Region 9 has developed a stand-alone document that presents numerous toxicity values and Preliminary Remediation Goals (PRGs). Some toxicity values presented in this database should be considered interim. For example, some values are presented for chemicals that have been withdrawn from IRIS during the normal review process. PRGs have been developed as tools for evaluating and cleaning up contaminated sites. They are risk-based concentrations derived from standardized equations, combining exposure information assumptions and EPA toxicity data. The PRGs contained in the Region 9 PRG database are generic; they are calculated without site-specific information. PRGs should be viewed as Agency guidelines, not legally enforceable standards. They are used for site "screening" and as initial cleanup goals if applicable. PRGs are not de facto cleanup standards and should not be applied as such.
- California Office of Environmental Health Hazard (OEHHA)/Air Resources
 Board (ARB) Database: OEHHA/ARB has developed California approved
 toxicity values and Recommended Exposure Levels (REL). This database
 includes both carcinogenic and noncarcinogenic toxicity values for the inhalation
 pathway.
- ➤ CalEPA Department of Toxic Substance Control (DTSC): DTSC has developed a database of California Potency Factors. Included are Inhalation Slope Factors, Inhalation Unit Risks and Oral Slope Factors.

3.2.2 Governmental Agency Databases

Occupational Safety and Health Administration (OSHA)/ National Institute for Occupational Safety and Health (NIOSH): The OSHA/NIOSH database is a compendium of toxicological information presented in the NIOSH Pocket Guide to Chemical Hazards. It is intended as a source of general industrial hygiene information for workers, employers, and occupational health professionals. The Pocket Guide presents key information and data in abbreviated tabular form for 677 chemicals or substance groupings that are found in the work environment. The chemicals or substances contained in the pocket

guide include all substances for which NIOSH has developed recommended exposure levels (RELs) and those with permissible exposure limits (PELs) developed by OSHA.

- by the Department of Energy, National Institutes of Environmental Health Sciences (NIEHS), and University of California Berkeley. It has been published in the following handbook: Handbook of Carcinogenic Potency and Genotoxicity Database edited by L.S. Gold and E. Zeiger, CRC Press, Inc. 1997. It provides a single, standardized and easily accessible database. Both qualitative and quantitative information on positive and negative experiments are given, including all bioassays from the National Cancer Institute/National Toxicology Program (NCI/NTP). Analyses of 5152 experiments on 1298 chemicals are presented. For each experiment, information is included on the species, strain, and sex of test animal; features of experimental protocol such as route of administration, duration of dosing, dose level(s) in mg/kg body weight/day, and duration of experiment; histopathology and tumor incidence; carcinogenic potency (TD50) and its statistical significance; shape of the dose-response curve.
- The Registry of Toxic Effects of Chemical Substances (RTECS): This is a database of toxicological information compiled, maintained, and updated by NIOSH. RTECS is a congressionally mandated activity established by Section 20(a)(6) of the Occupational Safety and Health Act of 1970 (PL 91-596). RTECS contains toxicity information on over 130,000 chemicals. Six types of toxicity data are included in the file: (1) primary irritation; (2) mutagenic effects; (3) reproductive effects; (4) tumorgenic effects; (5) acute toxicity; and (6) other multiple dose toxicity effects.

3.2.3 National Library of Medicine Databases and Toxicological Information

➤ MICROMEDEX: This database includes the TOMES Plus System, a computerized library of proprietary and government databases providing information on chemical hazards, environmental, and medical information. The following databases were queried for this study:

- 1. **HAZARDTEXT:** Provides information needed for the initial response to chemical spills, leaks, and fires.
- CHRIS: Chemical Hazard Response Information System from the U.S.
 Coast Guard presents information useful for initial response to aquatic incidents involving hazardous materials. It lists information on approximately 1,300 chemicals.
- 3. **HSDB:** Hazardous Substance Data Bank-has detailed information on more than 4,000 hazardous chemical substances.
- 4. New Jersey Fact Sheets from the New Jersey Department of Health: Provides information on more than 700 chemicals.
- 5. **POISINDEX:** Contains information on more than 1,000 chemicals regarding clinical effects, range of toxicity, and medical treatment for exposures.
- Chemical Carcinogenesis Research Information System (CCRIS): This database provides information on carcinogenicity, mutagenicity, tumor promotion, and tumor inhibition data and has been developed by the National Cancer Institute (NCI).
- TOXLINE: This database is the National Library of Medicine's extensive collection of biochemical, pharmacological, physiological, and toxicological effects of chemicals. It contains more than 2.5 million bibliographic citations, almost all with abstracts and/or indexing terms and CAS Registry Numbers. The information in TOXLINE is taken from secondary sources, including the following:
 - 1. Developmental and Reproductive Toxicology (DART)
 - 2. Environmental Mutagen Information Center File (EMIC)

- 3. Environmental Teratology Information Center File (ETIC)
- 4. Epidemiology Information System (EPIDEM)
- 5. Federal Research in Progress (FEDRIP)
- 6. Hazardous Materials Technical Center (HMTC)
- 7. International Labour Office (CIS)
- 8. International Pharmaceutical Abstracts (IPA)
- 9. NIOSHTIC (NIOSH)
- 10. Pesticides Abstracts (PESTAB)
- 11. Poisonous Plants Bibliography (PPBIB)
- 12. Swedish National Chemicals Inspectorate (RISKLINE)
- 13. Toxic Substances Control Act Test Submissions (TSCATS)
- 14. Toxicity Bibliography (TOXBIB)
- 15. Toxicological Aspects of Environmental Health (BIOSIS)
- 16. Toxicology Document and Data Depository (NTIS)
- 17. Toxicology Research Projects (CRISP)

Table 1 presents a summary of all the abstracts from peer-reviewed scientific publications reviewed during this toxicity assessment. The abstracts from the initial review were carefully evaluated to determine whether dose-response information could be culled to derive an RfDi

value. As shown, some chemicals have been extensively studied while toxicologists have ignored others.

TABLE 1
SUMMARY OF ALL TOXICOLOGICAL STUDIES ANALYZED

CHEMICAL	CASRN	NUMBER OF ABSTRACTS REVIEWED	NUMBER OF ABSTRACTS CONTAINING SOME TOXICOLOGICAL INFORMATION
Hydrofluoric Acid	7664-39-3	63	7
Hexanal	66-25-1	234	7
Isovaleraldehyde	590-86-3	109	5
n-Butyraldehyde	123-72-8	434	0
Propionaldehyde	123-38-6	120	12
Tolualdehyde	529-20-4	257	4
Valeraldehyde	110-62-3	257	7
Chloride	16887-00-6	7	0
Fluoride	16984-48-8	9	0
Sulfate	NA	2	0
1,2,3-Trimethylbenzene	526-73-8	230	10
1-Decene	872-05-9	47	0
1-Heptene	592-76-7	63	2
1-Hexene	592-41-6	175	6
1-Nonene	124-11-8	50	0
1-Octene	111-66-0	92	5
1-Pentene	109-67-1	97	0
1-Undecene	821-95-4	19	0
2,2,3-Trimethylpentane	564-02-3	12	0
2,2,5-Trimethylhexane	3522-94-9	39	1
2,3,4-Trimethylpentane	565-75-3	82	3
2,3-Dimethylbutane	79-29-8	313	2
2,3-Dimethylpentane	565-59-3	70	0
2,4,4-Trimethyl-1- Pentene	107-39-1	21	4
2,4-Dimethylpentane	108-08-7	71	0
2,5-Dimethylhexane	592-13-2	38	2
2-Ethyl-1-Butene	760-21-4	9	0
2-Methyl-1-Pentene	763-29-1	37	0
2-Methyl-2-Pentene	625-27-4	37	0
2-Methylheptane	592-27-8	4	0
3-Methyl-1-Butene	563-45-1	47	0
3-Methylheptane	589-81-1	59	2
3-Methylhexane	589-34-4	114	0
3-Methylpentane	96-14-0	244	5
4-Methyl-1-Pentene	691-37-2	23	0
3-Methylphenol & 4-Methylphenol	108-39-4 & 106-44-5	197	10
4-Nonene	2198-23-4	2	0
a-Pinene	80-56-8	562	4
b-Pinene	127-91-3	266	0
Butyl Acrylate	141-32-2	295	17

CHEMICAL	CASRN	NUMBER OF ABSTRACTS REVIEWED	NUMBER OF ABSTRACTS CONTAINING SOME TOXICOLOGICAL INFORMATION
c-2-Butene	590-18-1	103	0
c-2-Hexene	7688-21-3	0	0
c-2-Octene	7642-04-8	5	0
c-2-Pentene	627-20-3	48	0
c-3-Hexene	7642-09-3	9	0
c-3-Methyl-2-Pentene	922-61-2	10	0
Chlorodifluoromethane	75-45-6	200	1
Cyclohexane	110-82-7	267	14
Cyclohexene	110-83-8	155	2
Cyclopentane	287-92-3	157	3
Cyclopentene	142-29-0	100	1
Dichlorofluoromethane	75-43-4	212	9
Ethanol	64-17-5	616	30
Freon 114	76-14-2	49	9
Heptanal	111-71-7	160	1
Indan	496-11-7	511	3
Indene	95-13-6	10	4
Isobutane	75-28-5	52	10
Isobutene	115-11-7	102	7
1-Butene	106-98	47	7
Isobutylbenzene	538-93-2	104	0
Isoheptane	591-76-4	5	1
Isohexane	73513-42-5	22	0
Isopentane	78-78-4	47	5
Isoprene	78-79-5	47	15
m-Diethylbenzene	141-93-5	64	1
Methylcyclopentane	96-37-7	272	5
Methylcyclopentene	27476-50-2	8	0
n-Butane	106-97-8	107	4
n-Decane	124-18-5	860	4
Neohexane	75-83-2	15	3
Neopentane	463-82-1	4	1
n-Nonane	111-84-2	58	4
n-Octane	111-65-9	234	5
n-Undecane	1120-21-4	412	5
p-Diethylbenzene	105-05-5	49	2
p-Isopropyltoluene	99-87-6	267	5
Propane	74-98-6	6	4
p-Xylene	106-42-3	226	17
m-Xylene	108-38	220	13
t-2-Butene	624-64-6	116	2
t-2-Hexene	4050-45-7	345	5
t-2-Pentene	646-04-8	15	0
Isodrin	465-73-6	151	0
PM2.5	NA	2	0
1-Naphthylamine	134-32-7	90	8
2-Methylnaphthalene	91-57-6	101	8
2-Nitrophenol	88-75-5	77	0

CHEMICAL	CASRN	NUMBER OF ABSTRACTS REVIEWED	NUMBER OF ABSTRACTS CONTAINING SOME TOXICOLOGICAL INFORMATION
TOTAL		11,544	333

4 METHODOLOGY FOR DERIVING TOXICITY VALUES

The goal of this toxicity assessment was to either identify RfDi values applicable to environmental exposures in existing databases or develop interim values based on peer-review scientific publications. For the latter, methods developed by NAS (*Risk Assessment in the Federal Government: Managing the Process*, Washington, DC: NAS Press, 1983) and adopted by USEPA, as detailed in risk assessment guidance, were applied.

4.1 Carcinogens

In the analysis of data regarding the potential human carcinogenicity of chemical agents, the U.S. EPA uses the approach described in its *Guidelines for Carcinogen Risk Assessment* (51 FR 33992-34003, Sept. 24, 1986). Detailed examples of how the Guidelines can be applied are found in two documents: (1) *Health Assessment Document for Epichlorohydrin* (EPA-600/8-83-032F, Dec., 1984, p. 7-32 to 7-48); and (2) *OTS Assessment of Health Risk of Garment Workers and Certain Home Residents from Exposure to Formaldehyde*, Appendix 4 (Apr., 1987).

As mentioned previously, the first step of the toxicity assessment involves a hazard identification, in which a determination is made as to whether human exposure to the chemical has the potential to be a human carcinogen.

The primary toxicological evidence used to make this determination are: (1) human studies of the association between cancer incidence and exposure to the agent; and (2) long-term animal studies under controlled laboratory conditions. Supporting evidence such as short-term tests for genotoxicity, metabolic and pharmacokinetic properties, toxicological effects other than cancer, structure-activity relationships, and physical/chemical properties of the agent are also evaluated. For example, the molecular structure of a chemical may be evaluated to determine if there is a potential to form epoxide intermediates that can result in DNA adducts and transcription errors. Based on the weight of evidence a chemical is classified as one of the following:

- **Category A:** Known Human Carcinogen;
- **Category B:** Probable Human Carcinogen;

- 1. B1-Iindicates limited human evidence.
- 2. B2 indicates sufficient evidence in animals and inadequate or no evidence in humans.
- **Category C:** Possible Human Carcinogen;
- **Category D:** Not Classifiable As To Human Carcinogenicity; and
- **Category E:** Evidence Of Noncarcinogenicity.

For the purpose of the current study, there is no need to further discuss the USEPA methodology for developing a slope factor (low dose extrapolation mathematical approaches) or unit inhalation value because there was no indication in any of the numerous databases analyzed or scientific papers reviewed that any of the 86 chemicals can be classified as human carcinogens. That is, all 86 chemicals should be considered Category D carcinogens (which indicates a lack of toxicological information).

4.2 Noncarcinogens

RfDi are concentrations that are considered safe under chronic exposure conditions(e.g., 30 years is USEPA's *Reasonable Maximum Exposure* duration for a resident living at a single residence). Empirical toxicological observations have generally revealed that as the dose of a toxic chemical is increased, the toxic response (in terms of severity and/or incidence of effect) also increases. This dose-response relationship is well founded in the theory and practice of toxicology and pharmacology. In evaluating a dose-response curve, a threshold (dose level) exists at which no toxic response is produced. In experimental studies on laboratory animals, this threshold is termed a *no-observed-adverse-effect-level* or NOAEL. This is defined experimentally as the dose at which no statistically or biologically significant indication of a toxic effect exists. In an experiment with several NOAELs, the regulatory focus is normally on the highest one. Thus, the term NOAEL represents the highest experimentally determined dose that does not produce a statistically or biologically significant adverse effect. The NOAEL for the critical toxic effect is sometimes referred to simply as the NOEL. In cases in which a NOAEL has not been derived experimentally, the term *lowest-observed-adverse-effect level* or LOAEL is used.

There are myriad uncertainties involved in developing toxicity values for human exposures based on experimental animal toxicity studies, including the following:

- Extrapolations from animal data to humans and from high experimental doses to lower environmental exposures;
- The organs affected and the type of adverse effect resulting from chemical exposure may differ between study animals and humans;
- Differences between toxicological mechanisms in humans and animals: and
- Extrapolating results from short-term animal experiments to represent chronic human exposures.

Chemicals can produce different toxic effects at different dose levels. The toxic response may range from gross effects, such as death, to subtler biochemical, physiologic, or pathologic changes. Typically, all toxic endpoints from all available studies are first considered; although primary attention usually is given to the "critical effect" exhibiting the lowest NOAEL. Where limited toxicological data is available (as in the current study), all studies are evaluated and the "best" study is selected. This is sometimes referred to as the Principal Study. Principal studies are those that are primarily used to quantify the toxicity of a chemical. For the most part, the toxicological studies identified as pertinent for evaluating the toxicity of the 86 chemicals in this study were based on animal studies in rats, mice, rabbits, guinea pigs, hamsters, dogs, or monkeys. Studies that were conducted *in vitro* were ignored, if *in vivo* studies could not be identified to support the *in vitro* results.

The primary route of exposure for the Atsugi chemicals was the inhalation pathway. For this reason, only inhalation toxicity values RfDi were sought or developed from peer-reviewed toxicological studies. It should be noted, the magnitude, frequency, and duration of exposure varied considerably in these studies, as did the overall quality of the studies. This introduced significant uncertainty into the derived RfDi. Dosing regimens followed acute, subchronic, and chronic dosing schedules and some were single, intermittent, or continuous dosing. In some studies it was impossible to identify the dosing regime and interpretations were made. All studies were evaluated on the basis of the study's hypothesis, design, execution, and interpretation. Not

all studies were conducted for use in environmental risk assessment applications. Some parameters evaluated to determine the appropriateness of the studies included the type of laboratory animal species, similarities and differences between the test species and humans (e.g., chemical absorption and metabolism), the number of individuals in the study groups, the number of study groups, the spacing and choice of dose levels tested, the types of observations and methods of analysis, the nature of pathologic changes, the alteration in metabolic responses, the sex and age of test animals, and the route and duration of exposure. However, the most important information was the derivation of a NOAEL. After the studies were evaluated, and the NOAEL identified, the RfDi was derived by following a 3-step process:

Step1: Dose conversion (NOAEL to an exposure level);

Step 2: Dose adjustment for discontinuous exposure dosing regimens; and

Step 3: Dose adjustment (based on uncertainty and modifying factors) to derive the final RfDi.

4.2.1 Step1: Dose Conversion

To derive the RfDi, the first step was to identify a study that observed and quantified a NOAEL. This represents the highest level tested in which "no adverse effect" was observed. With few exceptions, the units of the reported NOAEL are in parts per million (ppm). This concentration must be converted to milligrams per cubic meter (mg/m³). The following equation was used to make this adjustment:

NOAEL [mg/m³] = NOAEL [ppm] * g-mole/22.4 L * MW/gm-mole * 273°/T * P/760 mm Hg * 1E3L/m³ *1E3mg/gm

where:

NOAEL [adj] = mg/m^3

MW = Molecular Weight

22.4 L = the volume occupied by 1 gm-mol of any chemical in the gaseous state

at 0°C and 760 mm Hg

T = actual temperature in degrees Kelvin

P = actual pressure in mm Hg

4.2.2 Step 2: Dose Adjustment for Discontinuous Exposure Dosing Regimens

Many inhalation toxicity studies are conducted with a discontinuous exposure. Since the RfDi represents continuous exposure to receptors in a human health risk assessment, the NOAEL must be modified. The following equation is used to normalize the NOAEL (derived under the toxicological study conditions) to a NOAEL [adj] to represent chronic and continuous exposures:

NOAEL [adj]
$$(mg/m^3-day) = E(mg/m^3) * D(hours/day/24 hours) * W(days/7 days)$$

where:

E = experimental exposure level
D = number of hours exposed per day

W = number of days per week

4.2.3 Step 3: Dose Adjustment (Based on Uncertainty and Modifying Factors)

It should be noted that as of January 1991, IRIS and NCEA databases no longer present RfDs or SFs for the inhalation route. These criteria have been replaced with reference concentrations (RfC) for noncarcinogenic effects and unit risk factors (URF) for carcinogenic effects. However, for purposes of estimating risk and calculating risk-based concentrations, RfDi and inhalation slope factors (SFi) are used.

To calculate an RfDi from an RfC, the following equation and assumptions are used:

RfDi (mg/kg-day) =
$$\frac{\text{RfC}(\text{mg/m}^3) * \text{IR}}{\text{BW}}$$

where:

IR = inhalation rate BW = body weight

All studies were carefully evaluated for NOAEL levels. However, while the NOAEL level was preferred, in some cases the LOAEL was the only toxicological endpoint reported in the study and was used to derive RfDi values (an adjustment was subsequently made with an uncertainty factor). The RfDi is a benchmark dose operationally derived from the NOAEL [adj] (or LOAEL) by consistent application of generally order-of-magnitude uncertainty factors (UFs) that reflect various types of data sets used to estimate an RfDi. In addition, a modifying factor (MF) was incorporated which is based on a professional judgment. For example, a lower MF is assigned to well designed studies. Thus, the final RfDi is determined by use of the following equation:

$$RfDi = NOAEL / (UF \times MF)$$

The RfDi should be considered an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. The RfDi is expressed in units of *milligrams per kilogram of bodyweight per day* (mg/kg-day). In these units the RfDi is used directly in the risk assessment to evaluate the potential of toxic effects associated with exposure to a chemical. Doses (exposure) less than the RfDi are not likely to produce adverse effects.

The uncertainty factors used to derive an RfDi from an experimental NOAEL [adj] are as follows:

Population Heterogeneity: A 10-fold factor is used to extrapolate from valid experimental results in studies using prolonged exposure to average healthy

humans. This factor is intended to account for the variation in sensitivity among the members of the human population and is referenced as "10H".

- Animal to Human Exposure: A 10-fold factor is used when extrapolating from valid results of long-term studies on experimental animals when results of studies of human exposure are not available or are inadequate. This factor is intended to account for the uncertainty involved in extrapolating from animal data to humans and is referenced as "10A".
- Exposure Duration: A 10-fold factor is used when extrapolating from less than chronic results on experimental animals when there are no useful long-term human data. This factor is intended to account for the uncertainty involved in extrapolating from less than chronic NOAELs to chronic NOAELs and is referenced as "10S".
- Extrapolating a LOAEL to NOAEL: An additional 10-fold factor is used to derive an RfD from a LOAEL, instead of a NOAEL. This factor is intended to account for the uncertainty involved in extrapolating from LOAELs to NOAELs and is referenced as "10L".
- Modifying Factor (MF): The MF is an additional uncertainty factor that is greater than zero and less than or equal to 10. The magnitude of the MF represents the uncertainty of the study and database not explicitly accounted for by the uncertainty factors. The default value for the MF is 1.

It should be stressed that the use of uncertainty and modifying factors represents risk policy and not necessarily science. That is, uncertainty is always addressed by introducing an *increasing* amount of conservatism without determining whether it is warranted or scientifically valid. For example, if the laboratory species used in toxicological experiments was actually *more* sensitive to a chemical than humans (such as dioxin appears to be), introducing uncertainty factors into the RfDi would be ultraconservative, since the unaltered NOAEL already represents a conservative estimate of the toxic potential of the chemical with respect to human exposures. To simply divide the NOAEL by a factor of 10 (when the NOAEL already is a conservative estimate of the health hazard in humans) would compound the conservatism.

In the final analysis, the RfDi should not be interpreted to solely represent the inherent toxicity of chemicals. Rather the RfDi ultimately represents science and science policy incorporating the following two aspects:

- The inherent toxicity of a chemical (as actually defined by toxicity studies);
- The state and quality of the scientific data base (fewer scientific studies or lower quality toxicity information automatically translates into a more "toxic" chemical, or lower RfDi) where policy is introduced to increase safety.

In the current study, an upper bound value for the UF-MF was 3,000. When the combined UF-MF exceeded this brightline, it was concluded the RfDi had an unacceptable amount of uncertainty and it was deleted from this analysis.

4.2.4 Example: Deriving an RfDi

This hypothetical example illustrates how peer-reviewed scientific studies are used to develop an RfDi value. A toxicology study was conducted on Chemical X (molecular weight: 44.5) and published in a peer-review toxicological journal. After careful review of the study, it was concluded that the toxicological design of the experiment was adequate to develop the uncertainty factors for an RfD. Four different groups of rats (250 rats per dose level) were exposed to increasing vapor concentrations of Chemical X. All animals were dosed (exposed to Chemical X vapor for a period of 3 months). Table 2 presents the dosing regime, toxicological observations, and severity of toxic effect.

TABLE 2
EXAMPLE EXPERIMENTAL RESULTS FOR CHEMICAL X

Chemical X Vapor Concentration (Dose: ppm [8hr])	Toxicological Observation	Severity Of Toxic Effect
Control Animals (250 rats)	 Sex and aged-matched control animals were not exposed to chemical. No adverse effects observed. 	
1 (250 rats)	No statistically or biologically significant differences between treated and control animals.	NOEL
150 (250 rats)	 Two percent decrease in body weight gain (not toxicologically significant) increased ratio of liver weight to body weight. No histopathological changes in the liver. Slight increase in blood liver enzyme levels. 	NOAEL
1,000 (250 rats)	 Significant decrease in body weight gain. Increased ratio of liver weight to body weight. Periportal hepatic damage. Significant blood liver enzyme levels. 	LOAEL
10,000 (250 rats)	Fifty percent of the animals die within a 24-hour post-dose period.	LC50

Although the study was well designed (exposures were 8 hours per day 5 days a week for 3 months) and the necessary toxicological endpoints have been determined, the toxicity endpoints must be modified to develop an RfC.

The NOAEL was determined to be 150 ppm. The experimental NOAEL was first converted to mg/m^3 (assuming 25°C and 760 mmHg):

NOAEL(mg/m³) = (150 ppm) (44.5/22.4) (273°/298°) (760/760)(10^3 L/ m³) (10^3 mg/ gm)

The NOAEL [adj] was calculated as:

$$NOAEL[adj] = 273 \text{ mg/m}^3 * 8 \text{ hours/24 hours-day} * 5 \text{ days/7 days} = 65 \text{ mg/m}^3$$

Because the study animal was a rat and of subchronic duration, the uncertainty factor is:

$$UF = 10H *10A * 10S = 1000$$

However, with the high number of animals (250) per dose group:

$$MF = 0.8$$

These factors then give a safety margin UF *MF = 800, so that

RfC = NOAEL/(UF x MF) =
$$65/800 = 0.081 \text{ (mg/m}^3)$$
.

This RfC value is then converted to an RfDi value with the following equation:

The RfDi is used in the risk assessment to calculate a Hazard Index (HI). The chronic daily intake (CDI) is estimated with the following equation:

CDI
$$(mg/kg-day) = (CA * IR * ET * EF * ED)/(BW * AT)$$

where:

CA	= Chemical Concentration in Air
IR	= Inhalation Rate (m ³ /day; 20 m ³)
ET	= Exposure Time (hours/day; 24 hrs)
EF	= Exposure Frequency (days/year; 350)
ED	= Exposure Duration (years)
BW	= Body Weight (kg; 70 kg)
AT	= Averaging Time (days)

To derive the HI, the estimated chronic daily intake (CDI) is simply divided by the RfDi. If the HI is less than 1.0, it can be confidently concluded that the chemical poses no health threat. However, when the HI exceeds 1.0, the RfDi should be further evaluated, (particularly with

regard to the uncertainty and modifying factors used to develop the RfD). The exceedance could be the result of excessive conservatism (excessive uncertainty factors) introduced into the RfDi.

5 RESULTS

Prior to evaluating peer-review scientific publications, a detailed review of regulatory databases was conducted to determine if toxicity values had been developed after the NAF Atsugi risk assessment was conducted or if toxicity values have been developed by other regulatory agencies. Table 3 presents the result of the analysis where the following databases were queried:

- ➤ IRIS;
- **EPA** Region 9 Toxicity Information;
- California Office of Environmental Health Hazard (OEHHA)/Air Resources
 Board (ARB) Database; and
- Carcinogenic Potency Database (CPD).

As indicated in the table, none of the chemicals is considered carcinogenic by any regulatory agency. A cursory structure activity analysis was performed and there was little justification for developing cancer slope factors based on toxicity of a chemical structure.

TABLE 3
RESIDENTIAL REGULATORY RISK-BASED ACCEPTABLE CONCENTRATIONS AND EXISTING
TOXICITY VALUES

CHEMICAL	CASRN	USEPA ⁽¹⁾ IRIS RfCi/CSF	EPA REGION 9 ⁽²⁾ TOXICITY VALUES (RfDi;mg/kg-day)	EPA REGION 9 ⁽³⁾ PRGs (μg/m3)	CALIFORNIA ⁽⁴⁾ OEHHA REL (µg/m3)	CARCINOGENIC ⁽⁵⁾ POTENCY DATABASE
Hydrofluoric Acid	7664-39-3	NA	NA	NA	5.9	NA
Hexanal	66-25-1	NA	NA	NA	NA	NA
Isovaleraldehyde	590-86-3	NA	NA	NA	NA	NA
n-Butyraldehyde	123-72-8	NA	NA	NA	NA	NA
Propionaldehyde	123-38-6	NA	NA	NA	NA	NA
Tolualdehyde	529-20-4	NA	NA	NA	NA	NA
Valeraldehyde	110-62-3	NA	NA	NA	NA	NA
Chloride	16887-00- 6	NA	NA	NA	NA	NA
Fluoride	16984-48- 8	NA	NA	NA	NA	NA
Sulfate	NA	NA	NA	NA	NA	NA
1,2,3-Trimethylbenzene	526-73-8	NA	NA	NA	NA	NA
1-Decene	872-05-9	NA	NA	NA	NA	NA
1-Heptene	592-76-7	NA	NA	NA	NA	NA
1-Hexene	592-41-6	NA	NA	NA	NA	NA
1-Nonene	124-11-8	NA	NA	NA	NA	NA
1-Octene	111-66-0	NA	NA	NA	NA	NA
1-Pentene	109-67-1	NA	NA	NA	NA	NA

CHEMICAL	CASRN	USEPA ⁽¹⁾ IRIS RfCi/CSF	EPA REGION 9 ⁽²⁾ TOXICITY VALUES (RfDi;mg/kg-day)	EPA REGION 9 ⁽³⁾ PRGs (μg/m3)	CALIFORNIA ⁽⁴⁾ OEHHA REL (µg/m3)	CARCINOGENIC ⁽⁵⁾ POTENCY DATABASE
1-Undecene	821-95-4	NA	NA	NA	NA	NA
2,2,3-Trimethylpentane	564-02-3	NA	NA	NA	NA	NA
2,2,5-Trimethylhexane	3522-94-9	NA	NA	NA	NA	NA
2,3,4-Trimethylpentane	565-75-3	NA	NA	NA	NA	NA
2,3-Dimethylbutane	79-29-8	NA	NA	NA	NA	NA
2,3-Dimethylpentane	565-59-3	NA	NA	NA	NA	NA
2,4,4-Trimethyl-1-Pentene	107-39-1	NA	NA	NA	NA	NA
2,4-Dimethylpentane	108-08-7	NA	NA	NA	NA	NA
2,5-Dimethylhexane	592-13-2	NA	NA	NA	NA	NA
2-Ethyl-1-Butene	760-21-4	NA	NA	NA	NA	NA
2-Methyl-1-Pentene	763-29-1	NA	NA	NA	NA	NA
2-Methyl-2-Pentene	625-27-4	NA	NA	NA	NA	NA
2-Methylheptane	592-27-8	NA	NA	NA	NA	NA
3-Methyl-1-Butene	563-45-1	NA	NA	NA	NA	NA
3-Methylheptane	589-81-1	NA	NA	NA	NA	NA
3-Methylhexane	589-34-4	NA	NA	NA	NA	NA
3-Methylpentane	96-14-0	NA	NA	NA	NA	NA
4-Methyl-1-Pentene	691-37-2	NA	NA	NA	NA	NA
3-Methylphenol	108-39-4	NA	5E-2	1.8E+2	1.8E+2	NA
4-Methylphenol	106-44-5	NA	5E-3	1.8E+1	1.8E+2	NA
4-Nonene	2198-23-4	NA	NA	NA	NA	NA
a-Pinene	80-56-8	NA	NA	NA	NA	NA
b-Pinene	127-91-3	NA	NA	NA	NA	NA
Butyl Acrylate	141-32-2	NA	NA	NA	NA	NA
c-2-Butene	590-18-1	NA	NA	NA	NA	NA
c-2-Hexene	7688-21-3	NA	NA	NA	NA	NA

CHEMICAL	CASRN	USEPA ⁽¹⁾ IRIS RfCi/CSF	EPA REGION 9 ⁽²⁾ TOXICITY VALUES (RfDi;mg/kg-day)	EPA REGION 9 ⁽³⁾ PRGs (μg/m3)	CALIFORNIA ⁽⁴⁾ OEHHA REL (µg/m3)	CARCINOGENIC ⁽⁵⁾ POTENCY DATABASE
c-2-Octene	7642-04-8	NA	NA	NA	NA	NA
c-2-Pentene	627-20-3	NA	NA	NA	NA	NA
c-3-Hexene	7642-09-3	NA	NA	NA	NA	NA
c-3-Methyl-2-Pentene	922-61-2	NA	NA	NA	NA	NA
Chlorodifluoromethane	75-45-6	50	50	5.1E+4	5.0E+4	NA
Cyclohexane	110-82-7	NA	NA	NA	NA	NA
Cyclohexene	110-83-8	NA	NA	NA	NA	NA
Cyclopentane	287-92-3	NA	NA	NA	NA	NA
Cyclopentene	142-29-0	NA	NA	NA	NA	NA
Dichlorofluoromethane	75-43-4	NA	5.7E-2	2.1E+2	7.0E+2	NA
Ethanol	64-17-5	NA	NA	NA	NA	NA
Freon 114	76-14-2	NA	NA	NA	NA	NA
Heptanal	111-71-7	NA	NA	NA	NA	NA
Indan	496-11-7	NA	NA	NA	NA	NA
Indene	95-13-6	NA	NA	NA	NA	NA
Isobutane	75-28-5	NA	NA	NA	NA	NA
Isobutene	115-11-7	NA	NA	NA	NA	NA
1-Butene	106-98	NA	NA	NA	NA	NA
Isobutylbenzene	538-93-2	NA	NA	NA	NA	NA
Isoheptane	591-76-4	NA	NA	NA	NA	NA
Isohexane	73513-42- 5	NA	NA	NA	NA	NA
Isopentane	78-78-4	NA	NA	NA	NA	NA
Isoprene	78-79-5	NA	NA	NA	NA	NA
m-Diethylbenzene	141-93-5	NA	NA	NA	NA	NA
Methylcyclopentane	96-37-7	NA	NA	NA	NA	NA

CHEMICAL	CASRN	USEPA ⁽¹⁾ IRIS RfCi/CSF	EPA REGION 9 ⁽²⁾ TOXICITY VALUES (RfDi;mg/kg-day)	EPA REGION 9 ⁽³⁾ PRGs (μg/m3)	CALIFORNIA ⁽⁴⁾ OEHHA REL (µg/m3)	CARCINOGENIC ⁽⁵⁾ POTENCY DATABASE
Methylcyclopentene	27476-50-	NA	NA	NA	NA	NA
n-Butane	106-97-8	NA	NA	NA	NA	NA
n-Decane	124-18-5	NA	NA	NA	NA	NA
Neohexane	75-83-2	NA	NA	NA	NA	NA
Neopentane	463-82-1	NA	NA	NA	NA	NA
n-Nonane	111-84-2	NA	NA	NA	NA	NA
n-Octane	111-65-9	NA	NA	NA	NA	NA
n-Undecane	1120-21-4	NA	NA	NA	NA	NA
p-Diethylbenzene	105-05-5	NA	NA	NA	NA	NA
p-Isopropyltoluene	99-87-6	NA	NA	NA	NA	NA
Propane	74-98-6	NA	NA	NA	NA	NA
p-Xylene	106-42-3	NA	2.0E-1	7.0E2	7.0E2	NA
m-Xylene	108-38	NA	2.0E-1	7.0E2	7.0E2	NA
t-2-Butene	624-64-6	NA	NA	NA	NA	NA
t-2-Hexene	4050-45-7	NA	NA	NA	NA	NA
t-2-Pentene	646-04-8	NA	NA	NA	NA	NA
Isodrin	465-73-6	NA	NA	NA	NA	NA
PM2.5	NA	NA	NA	NA	NA	NA
1-Naphthylamine	134-32-7	NA	NA	NA	NA	NA
2-Methylnaphthalene	91-57-6	NA	NA	NA	NA	NA
2-Nitrophenol	88-75-5	NA	NA	NA	NA	NA

- 1) USEPA Integrated Risk Information Service; Reference Concentration (RfCi), Cancer Slope Factor (CSF)
- 2) EPA Region 9 Toxicity Values: Reference Dose-Inhalation
- 3) EPA Region 9 Preliminary Remediation Goals

- 4) California Office of Environmental Health Hazard Assessment (OEHHA): Recommended Exposure Level (REL)
- 5) Carcinogenic Potency Database (National Institutes of Environmental Health)

Table 4 presents acceptable or safe levels of exposure under an occupational scenario (8 hours per day, 5 days per week, typically for 250 days per year for 25-30 years). Although the primary receptors at NAF Atsugi are residential receptors, the occupational standards are simply presented as a point of reference.

TABLE 4
OCCUPATIONAL EXPOSURE LEVELS

CHEMICAL	CASRN	OCCUPATIONAL SAFETY AND HEALTH (ppm)	AMERICAN CONFERNCE OF GOVERNMENTAL HYGIENISTS (ppm)	NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH (ppm)
Hydrofluoric Acid	7664-39-3	PEL = 3; IDLH = 30	TLV = 3; $STEL = 6$	
Hexanal	66-25-1			
Isovaleraldehyde	590-86-3			
n-Butyraldehyde	123-72-8			
Propionaldehyde	123-38-6			
Tolualdehyde	529-20-4			
Valeraldehyde	110-62-3		TLV = 50	REL =50
Chloride	16887-00-6			
Fluoride	16984-48-8			
Sulfate	NA			
1,2,3-Trimethylbenzene	526-73-8		TLV = 25	REL = 25
1-Decene	872-05-9			
1-Heptene	592-76-7			
1-Hexene	592-41-6		TLV = 30	
1-Nonene	124-11-8			
1-Octene	111-66-0			
1-Pentene	109-67-1			
1-Undecene	821-95-4			
2,2,3-Trimethylpentane	564-02-3			
2,2,5-Trimethylhexane	3522-94-9			
2,3,4-Trimethylpentane	565-75-3			
2,3-Dimethylbutane	79-29-8		TLV = 500; STEL = 1000	REL = 500; Ceiling = 1000
2,3-Dimethylpentane	565-59-3			

CHEMICAL	CASRN	OCCUPATIONAL SAFETY AND HEALTH (ppm)	AMERICAN CONFERNCE OF GOVERNMENTAL HYGIENISTS (ppm)	NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH (ppm)
2,4,4-Trimethyl-1-Pentene	107-39-1		TLV = 600	
2,4-Dimethylpentane	108-08-7			
2,5-Dimethylhexane	592-13-2			
2-Ethyl-1-Butene	760-21-4			
2-Methyl-1-Pentene	763-29-1			
2-Methyl-2-Pentene	625-27-4			
2-Methylheptane	592-27-8			
3-Methyl-1-Butene	563-45-1			
3-Methylheptane	589-81-1			
3-Methylhexane	589-34-4			
3-Methylpentane	96-14-0			
4-Methyl-1-Pentene	691-37-2			
3-Methylphenol	108-39-4		IDLH = 250	
4-Methylphenol	106-44-5	PEL = 5	PEL = 5	REL = 2.3
4-Nonene	2198-23-4			
a-Pinene	80-56-8			
b-Pinene	127-91-3			
Butyl Acrylate	141-32-2	TLV = 2	PEL = 10	REL = 10
c-2-Butene	590-18-1			
c-2-Hexene	7688-21-3			
c-2-Octene	7642-04-8			
c-2-Pentene	627-20-3			
c-3-Hexene	7642-09-3			
c-3-Methyl-2-Pentene	922-61-2			
Chlorodifluoromethane	75-45-6		TLV = 1000	REL = 1000
Cyclohexane	110-82-7	PEL = 300	TLV=200; STEL = 400	REL = 300; IDLH = 1300
Cyclohexene	110-83-8	PEL = 300	TLV = 300	REL = 300; IDLH = 2000
Cyclopentane	287-92-3		TLV = 600	REL = 600
Cyclopentene	142-29-0			
Dichlorofluoromethane	75-43-4	PEL = 1000	TLV = 10	REL = 10; IDLH = 5000
Ethanol	64-17-5	PEL = 1000	TLV = 1000	REL = 1000; IDLH = 3300

CHEMICAL	CASRN	OCCUPATIONAL SAFETY AND HEALTH (ppm)	AMERICAN CONFERNCE OF GOVERNMENTAL HYGIENISTS (ppm)	NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH (ppm)
Freon 114	76-14-2	PEL = 1000	TLV = 1000	REL = 1000; IDHL = 15K
Heptanal	111-71-7			
Indan	496-11-7			
Indene	95-13-6	PEL = 10	TLV = 10	REL = 10
Isobutane	75-28-5			REL = 800
Isobutene + 1-Butene	115-11-7/106-98			
Isobutylbenzene	538-93-2			
Isoheptane	591-76-4			
Isohexane	73513-42-5			
Isopentane	78-78-4	PEL = 1000	TLV=600; STEL = 610	REL = 120
Isoprene	78-79-5			
m-Diethylbenzene	141-93-5			
Methylcyclopentane	96-37-7			
Methylcyclopentene	27476-50-2			
n-Butane	106-97-8	PEL = 800	TLV = 800	REL= 800
n-Decane	124-18-5			
Neohexane	75-83-2		TLV = 500	
Neopentane	463-82-1			
n-Nonane	111-84-2		TLV = 200	REL = 200
n-Undecane	1120-21-4			
p-Diethylbenzene	105-05-5			
p-Isopropyltoluene	99-87-6			
Propane	74-98-6	PEL = 1000	TLV = 2500	REL = 2100
p-Xylene	106-42-3	PEL = 100	TLV=100; STEL = 150	REL = 100; IDLH = 900
m-Xylene	108-38	PEL = 100	TLV = 100	REL = 100; IDLH = 900
t-2-Butene	624-64-6			
t-2-Hexene	4050-45-7			
t-2-Pentene	646-04-8			
Isodrin	465-73-6			
PM2.5	NA			
1-Naphthylamine	134-32-7			

CHEMICAL	CASRN	OCCUPATIONAL SAFETY AND HEALTH (ppm)	AMERICAN CONFERNCE OF GOVERNMENTAL HYGIENISTS (ppm)	NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH (ppm)
2-Methylnaphthalene	91-57-6		TLV = 10	REL = 10
2-Nitrophenol	88-75-5			

PEL: Permissible Exposure Limit – Established by OSHA; the permissible concentration in air of a substance to which nearly all workers may be repeatedly exposed 8 hours a day, 40 hours a week, for 30 years without adverse effects.

TLV: Threshold Limit Value – Copyrighted by ACGIH; airborne concentration of a substance to which it is believed nearly all workers can be exposed repeatedly without adverse effects.

STEL: Short-term Exposure Limit – Exposure that should not be exceeded for any 15-minute period.

REL: Recommended Exposure Limit – Recommended by NIOSH; occupational exposure limit that is protective of worker health and safety over a working lifetime.

IDLH: Immediately Dangerous to Life and Health – Maximum level to which a healthy individual can be exposed to a chemical for 30 minutes and escape without suffering irreversible health effects or impairing symptoms.

Table 5 presents LC50 values, which represent the airborne concentration at which 50 percent of the animals died. This is important in the toxicological evaluation to determine the margin of safety associated with a chemical. (For example, when the LC50 is close to the NOAEL the margin of safety for the chemical is narrow. For those chemicals with a high LC50 value and a low NOAEL there is a large amount of safety in the RfDi.) The animal species, dosing regimen, and reference that are presented were developed from a variety of toxicological sources.

TABLE 5
LC50 LEVELS – CONCENTRATIONS THAT CAUSE DEATH IN 50 PERCENT OF EXPERIMENTAL
ANIMALS

CHEMICAL	CAS NUM.	ANIMAL SPECIES	LC50 & LD50 LETHAL CONCENTRATION IN 50 PERCENT OF ANIMALS	REFERENCE
		Rat	1278 ppm, 1 hr	HSDB
Hydrofluoric Acid	7664-39-3	Mouse	500 ppm, 1 hr	HSDB
		Monkey	1780 ppm, 1 hr	HSDB
Hexanal	66-25-1			
Isovaleraldehyde	590-86-3	Rat	42700 mg/m3	RTECS
150 valet alueny ue	370-00-3	Mouse	57700 mg/m3	RTECS
n-Butyraldehyde	123-72-8	Mouse	44 g/m3, 2 hours	RTECS
• • • • • • • • • • • • • • • • • • • •		Rat	8000 ppm, 4 hours	RTECS
Propionaldehyde	123-38-6	Mouse	21 g/m3, 2 hours	RTECS
Tolualdehyde	529-20-4	Rat		
Valeraldehyde	110-62-3		4000 ppm, 4 hours	HSBD
Chloride	16887-00-6			
Fluoride	16984-48-8			
Sulfate	NA			
1,2,3-Trimethylbenzene	526-73-8			
1-Decene	1-Decene 872-05-9		8.5 g/m3, 1 hour	RTECS
1-Heptene	592-76-7			
1-Hexene 592-41-6		Rat	32,000 ppm, 4 hours	RTECS
1-Nonene	124-11-8			
1-Octene	111-66-0			
1-Pentene	109-67-1			
1-Undecene	821-95-4			

CHEMICAL	CAS NUM.	ANIMAL SPECIES	LC50 & LD50 LETHAL CONCENTRATION IN 50 PERCENT OF ANIMALS	REFERENCE
2,2,3-Trimethylpentane	564-02-3			
2,2,5-Trimethylhexane	3522-94-9	Rat	10 g/kg, 4 weeks	RTECS
2,3,4-Trimethylpentane	565-75-3	Rat	8 ml/kg, 2 weeks	RTECS
2,3-Dimethylbutane	79-29-8		-	
2,3-Dimethylpentane	565-59-3	Rat	10 g/kg, 4 weeks	RTECS
2,4,4-Trimethyl-1-Pentene	107-39-1		-	
2,4-Dimethylpentane	108-08-7			
2,5-Dimethylhexane	592-13-2			
2-Ethyl-1-Butene	760-21-4			
2-Methyl-1-Pentene	763-29-1	Rat 115 g/m3, 4 hours Mouse 127 g/m3, 4 hours		RTECS RTECS
2-Methyl-2-Pentene	625-27-4			
2-Methylheptane	592-27-8			
3-Methyl-1-Butene	563-45-1			
3-Methylheptane	589-81-1			
3-Methylhexane	589-34-4			
3-Methylpentane	96-14-0			
4-Methyl-1-Pentene	691-37-2			
3-Methylphenol	108-39-4	Rat 710 mg/m3		RTECS
4-Methylphenol	106-44-5	Rat 710 mg/m3		RTECS
· •				
4-Nonene	2198-23-4			
		Rat	625 ug/m3, 4 hours	RTECS
a-Pinene	80-56-8	Guinea Pig	572 ug/m3, 4 hours	RTECS
		Mouse	364 ug/m3, 4 hours	RTECS
b-Pinene	127-91-3			
		Rat	1000 ppm, 4 hours	OHM/TADS
Butyl Acrylate	141-32-2	Rat	2730 ppm, 4 hours	RTECS
		Mouse	7800 mg/m3, 2 hours	RTECS
c-2-Butene	590-18-1			

CHEMICAL	CAS NUM.	ANIMAL SPECIES	LC50 & LD50 LETHAL CONCENTRATION IN 50 PERCENT OF ANIMALS	REFERENCE
c-2-Hexene	7688-21-3			
c-2-Octene	7642-04-8			
c-2-Pentene	627-20-3			
c-3-Hexene	7642-09-3			
c-3-Methyl-2-Pentene	922-61-2			
Chlorodifluoromethane	75-45-6			
Cyclohexane	110-82-7			
Cyclohexene	110-83-8			
Cyclopentane	287-92-3			
Cyclopentene	142-29-0			
Dichlorofluoromethane	75-43-4	Rat	49,000 ppm, 4 hours ACGIH	
Ethanol	64-17-5	Rat Mouse	20,000 ppm, 10 hours 39 g/m3, 4 hours	RTECS RTECS
Freon 114	76-14-2	Rat Mouse Rabbit	72 ppm, 30 minutes 70 ppm, 30 minutes RTE 75 ppm, minutes RTE	
Heptanal	111-71-7			RTECS
Indan	496-11-7	10,100 mg/m2, 1 month		
Indene	95-13-6	Rat 14,000 mg/m3		RTECS
		Mouse	52 mg/m3, 1 hour	HSDB
Isobutane	75-28-5	Rat	57 ppm, 15 min	RTECS
Isobutene + 1-Butene	115-11-7/106-98			RTECS
Isobutylbenzene	538-93-2	3-3 8		
Isoheptane	591-76-4			
Isohexane	73513-42-5			
Isopentane	78-78-4			
Isoprene	78-79-5	, 6		RTECS RTECS
m-Diethylbenzene	141-93-5			
Methylcyclopentane	96-37-7			

CHEMICAL	CAS NUM.	ANIMAL SPECIES	LC50 & LD50 LETHAL CONCENTRATION IN 50 PERCENT OF ANIMALS	REFERENCE
Methylcyclopentene	27476-50-2			
n-Butane	106-97-8	Rat 658 g/m3 (280,000 ppm), 4 hours Mouse 680 g/m3 , 4 hours		urs RTECS RTECS
n-Decane	124-18-5	Mouse	72300 mg/m3/2 h	RTECS
Neohexane	75-83-2			
Neopentane	463-82-1			
n-Nonane	111-84-2	Rat 3200 ppm, 4 hours		RTECS
n-Octane	111-65-9	Rat 118 g/m3, 4 hours		RTECS
n-Undecane	1120-21-4	Rat 442 ppm, 8 hours		RTECS
p-Diethylbenzene	105-05-5			
p-Isopropyltoluene	99-87-6			
Propane	74-98-6			
p-Xylene	106-42-3	Rat 4550 ppm, 4 hours Mouse 3900 ppm, 6 hours		RTECS ACGIH
m-Xylene	108-38	Rat	6670 ppm	RTECS
m-Ayiene		Mouse	3900 ppm, 6 hours	RTECS
t-2-Butene	624-64-6			
t-2-Hexene	4050-45-7			
t-2-Pentene	646-04-8			
Isodrin	465-73-6			
PM2.5	NA			
1-Naphthylamine	134-32-7			
2-Methylnaphthalene	91-57-6			
2-Nitrophenol	88-75-5			

Table 6 presents toxicological summaries regarding NOAEL or LOAEL values that were quantified in peer-reviewed toxicological sources. This information was gathered as part of an exhaustive query of the National Library of Medicine databases. These studies were further reduced to a subpopulation of studies that were used to derive RfDi values. Note, only those studies that presented detailed information (number of hours per day, days per week, and number of weeks) could be used to develop toxicity values.

TABLE 6

NOAEL AND LOAEL SUMMARIES

COMPENDIUM OF CURRENT TOXICOLOGICAL PEER-REVIEW PUBLICATIONS

CHEMICAL	CASRN	SPECIES	DOSING REGIMEN	TOXICOLOGICAL ENDPOINT	REFERENCE
Hydrofluoric Acid	7664-39-3	Human Guinea Pig	Chronic Chronic	1.03 ppm - NOAEL 8.6 ppm - NOAEL	ACGIH, 1992 ACGIH, 1991, 6th ed.
Hexanal	66-25-1				
Isovaleraldehyde	590-86-3				
n-Butyraldehyde	123-72-8				
Propionaldehyde	123-38-6	Rat Rat	6 hr 20 days 6hr/day, 7day/wk for 7 wk	90 ppm - NOAEL 151 ppm - NOAEL	Patty's 3rd ed., 1981 Tokanova et al 1982
Tolualdehyde	529-20-4				
Valeraldehyde	110-62-3				
Chloride	16887-00-6				
Fluoride	16984-48-8				
Sulfate					
1,2,3-Trimethylbenzene	526-73-8				
1-Decene	872-05-9				
1-Heptene	592-76-7				
1-Hexene	592-41-6	Rat	5 d/w, 6 hr/day, 13 wk	1000 ppm - NOAEL	Gingell et al, 1999
1-Nonene	124-11-8				
1-Octene	111-66-0				
1-Pentene	109-67-1				
1-Undecene	821-95-4				
2,2,3-Trimethylpentane	564-02-3				
2,2,5-Trimethylhexane	3522-94-9				
2,3,4-Trimethylpentane	565-75-3				
2,3-Dimethylbutane	79-29-8				

CHEMICAL	CASRN	SPECIES	DOSING REGIMEN	TOXICOLOGICAL ENDPOINT	REFERENCE
2,3-Dimethylpentane	565-59-3				
2,4,4-Trimethyl-1- Pentene	107-39-1				
2,4-Dimethylpentane	108-08-7				
2,5-Dimethylhexane	592-13-2				
2-Ethyl-1-Butene	760-21-4				
2-Methyl-1-Pentene	763-29-1				
2-Methyl-2-Pentene	625-27-4				
2-Methylheptane	592-27-8				
3-Methyl-1-Butene	563-45-1				
3-Methylheptane	589-81-1				
3-Methylhexane	589-34-4				
3-Methylpentane	96-14-0	Rat	14 wks	1500 ppm - NOAEL	Ono et al, 1981
4-Methyl-1-Pentene	691-37-2				
3-Methylphenol	108-39-4				
4-Methylphenol	106-44-5				
4-Nonene	2198-23-4				
a-Pinene	80-56-8				
b-Pinene	127-91-3				
Butyl Acrylate	141-32-2	Rat	6hr/day, 5 day/wk, 2 yr	15ppm - LOAEL	Reinghaus, W et al, 1991
c-2-Butene	590-18-1				
c-2-Hexene	7688-21-3				
c-2-Octene	7642-04-8				
c-2-Pentene	627-20-3				
c-3-Hexene	7642-09-3				
c-3-Methyl-2-Pentene	922-61-2				
Chlorodifluoro- methane	75-45-6	Rat	10 months	2000 ppm - NOAEL	ACGIH, 1991

CHEMICAL	CASRN	SPECIES	DOSING REGIMEN	TOXICOLOGICAL ENDPOINT	REFERENCE
Cyclohexane	110-82-7	Monkey Rat Human Rabbit Rat	6 hr/day 50 days 10h/day, 6day/wk, 30 wks 2.8 yrs, 6hr/day, 5day/wks 10 wks, continuous	1243 ppm - NOAEL 2500 ppm - NOAEL 5-200 ppm - NOAEL 434 ppm - NOAEL 500 ppm - NOAEL	Clayton 1981 Frontali et al, 1981 Yuasa et al, 1996 Fairhurst et al, 1990 Krechman et al, 1998.
Cyclohexene	110-83-8	Rat	6hr/day, 5day/wks, 6 months	150 ppm - LOAEL	ACGIH, 1991
Cyclopentane	287-92-3	Rat	6 hr/day, 5day/wk 12 wks	8110 ppm - LOAEL	Clayton and Clayton 1994
Cyclopentene	142-29-0	Rat	6hr/day, 5 day/wks, 12 wks	1139 ppm - NOAEL	Kimmerle et al, 1975
Dichlorofluoro-methane	75-43-4	Guinea Pig Guinea Pig Rat	2 hr 2 hr 90-120 days	52000 ppm - LOAEL 12,000 ppm – NOAEL 150 ppm – NOAEL	ACGIH, 1971 ACGIH, 1971 EPA FYI-OTS-0779-0045
Ethanol	Rat		7hr/day, 6 wks 8hr 6 hr	6,000 ppm – NOAEL 6400 ppm – NOAEL 3260 ppm – NOAEL	Nelson et al, 1985 Verschueren et al, 1983 Verschueren et al, 1983
Freon 114	76-14-2	Dog Rat	6 Hr/D, 90 Days 6 H/D, 90 Days	5000 ppm - NOAEL 1000 ppm - NOAEL	Hathaway, 1996 Hathaway, 1996
Hepta l	111-71-7		, <u> </u>	• •	• /
Indan	496-11-7				
Indene	95-13-6	Rat Rat	105 Days 6 H/Day, 13 Weeks	0.6 mg/m3 - NOAEL 5 ppm - NOAEL	Dyshinevich NE Bevan et al, 1992
Isobutane	75-28-5	Human Human Human	8 Hr 8hr/Day, 10 Days	250-1000 ppm - NOAEL 500 ppm - NOAEL LOAEL1000 ppm - NOAEL	Synder et al, 1987 Stewart,et al, 1977 Clayton 1982 (Patty's)
Isobutene	115-11-	Rat	14 wks	8000 ppm - NOAEL	NTP, 1999
1-Butene	7/106-98	Mice	6hr/day, 5 day/wk, 105 wks	500 ppm - NOAEL	NTP, 1999
Isobutylbenzene	538-93-2				
Isoheptane	591-76-4				
Isohexane					
Isopentane				500 ppm - NOAEL	Clayton, 1892

CHEMICAL	CASRN	SPECIES	DOSING REGIMEN	TOXICOLOGICAL ENDPOINT	REFERENCE
Isoprene	78-79-5	Rat Rat	2 weeks 13 weeks	1400 ppm - NOAEL 7000 ppm - NOAEL 700 ppm - NOAEL	Mast et al, 1990 Melnick et al, 1996 Taalman et al, 1996
m-Diethylbenzene	141-93-5	Rat	8 hrs/day, 5 day/wks, 6 months	2200 ppm - NOAEL	Patty's, 1963
Methylcyclo-pentane	96-37-7				
Methylcyclo-pentene	27476-50-2				
n-Butane	106-97-8	Human	8hr/day/2 wks	1000 ppm – NOAEL	ACGIH, 1986
n-Decane	124-18-5	Rat	18hr/day, 123 days	540 ppm LOAEL - NOAEL	Snyder et al, 1983
Neohexane	75-83-2	Dogs		100,000 ppm LOAEL - NOAEL	Synder et al, 1987
Neopentane	463-82-1				
n-Nonene	111-84-2	Rat	6hrs/day, 5day/wks, 13 wks	590 ppm - NOAEL	Carpenter et al, 1978
n-Octane	111-65-9				
n-Undecane	1120-21-4	Mice		25,000 ppm - NOAEL	Kristiansen et al, 1988
p-Diethylbenzene	105-05-5	Monkeys Rat	8hr/day, 6 months 186 Days	2200 ppm – NOAEL 400 ppm - NOAEL	Patty's, 1963 Patty's, 1963
p-Isopropyltoluene	99-87-6	Human Rats	4 Weeks	200-500 ppm - LOAEL 50 ppm - LOAEL	Lam et al, 1996
Propane	74-98-6	Monkey	8 hr/day, 90 days	750 ppm - NOAEL	Snyder 1987
p-Xylene					
m-Xylene	106-42- 3/108-38				
t-2-Butene	624-64-6				
t-2-Hexene	4050-45-7				
t-2-Pentene	646-04-8				
Isodrin	465-73-6				
PM2.5					
1- Naphthylamine	134-32-7				
2-Methylnaphthalene	91-57-6	Rats	4 hr	67 mg/m3 - LOAEL	Korsak et al, 1998
2-Nitrophenol	88-75-5				

Table 7 presents the information used to derive RfDi values for the chemicals where sufficient toxicological information exists. The information in the studies was used to first convert the units of the NOAEL, calculate the RfC value, and ultimately calculate an RfDi. As the table shows, there was a high degree of uncertainty in the original NOAEL. For example, the combined uncertainty-modifying factors (UMF) for some chemicals were 10,000. It was determined that no RfDi would be developed for chemicals having an UMF of > 3,000. Note, only those studies that presented detailed information (number of hours per day, days per week, and number of weeks) shown in Table 6 were advanced to Table 7.

TABLE 7 CALCULATED RFC AND RFDI VALUES FROM TOXICOLOGY STUDIES

CHEMICAL	CASRN	MOLE- CULAR WEIGHT	NOAEL (ppm)	NOAEL DOSE CONVERSION (mg/m³)	NOAEL DOSE ADJUSTED		UNCE MODIF		FACT		RfC (mg/m ³)	RfDi (mg/kg- day)
						10H	10A	10S	10 L	MF		
Hydrofluoric Acid	7664-39-3											
Hexanal	66-25-1											
Isovaleraldehyde	590-86-3											
n-Butyraldehyde	123-72-8											
Propionaldehyde	123-38-6	59	151	365	91	10	10	10	-	1	9.1E-02	2.6E-02
Tolualdehyde	529-20-4											
Valeraldehyde	110-62-3											
Chloride	16887-00- 6											
Fluoride	16984-48- 8											
Sulfate	NA											
1,2,3- Trimethylbenzene	526-73-8											
1-Decene	872-05-9											
1-Heptene	592-76-7											
1-Hexene	592-41-6	84	1000	3436	614	10	10	10	-	1	6.2E-01	1.8E-01
1-Nonene	124-11-8											
1-Octene	111-66-0											

CHEMICAL	CASRN	MOLE- CULAR WEIGHT	NOAEL (ppm)	NOAEL DOSE CONVERSION (mg/m³)	CONVERSION DOSE ADJUSTED ADJUSTED		IODIF	RTAIN		ORS	RfC (mg/m³)	RfDi (mg/kg- day)
						10H	10A	10S	L	MF		
1-Pentene	109-67-1											
1-Undecene	821-95-4											
2,2,3- Trimethylpentane	564-02-3											
2,2,5- Trimethylhexane	3522-94-9											
2,3,4- Trimethylpentane	565-75-3											
2,3-Dimethylbutane	79-29-8											
2,3-Dimethylpentane	565-59-3											
2,4,4-Trimethyl-1- Pentene	107-39-1											
2,4-Dimethylpentane	108-08-7											
2,5-Dimethylhexane	592-13-2											
2-Ethyl-1-Butene	760-21-4											
2-Methyl-1-Pentene	763-29-1											
2-Methyl-2-Pentene	625-27-4											
2-Methylheptane	592-27-8											
3-Methyl-1-Butene	563-45-1											
3-Methylheptane	589-81-1											
3-Methylhexane	589-34-4											
3-Methylpentane	96-14-0											
4-Methyl-1-Pentene	691-37-2											
3-Methylphenol	108-39-4											
4-Methylphenol	106-44-5											
4-Nonene	2198-23-4											

CHEMICAL	CASRN	MOLE- CULAR WEIGHT	NOAEL (ppm)	NOAEL DOSE CONVERSION (mg/m³)	NOAEL DOSE ADJUSTED			ERTAING YING 10S			RfC (mg/m³)	RfDi (mg/kg- day)
a-Pinene	80-56-8											
b-Pinene	127-91-3											
Butyl Acrylate	141-32-2	128	15	79	14	10	10	-	10	1	1.4E-02	4.0E-03
c-2-Butene	590-18-1											
c-2-Hexene	7688-21-3											
c-2-Octene	7642-04-8											
c-2-Pentene	627-20-3											
c-3-Hexene	7642-09-3											
c-3-Methyl-2-Pentene	922-61-2											
Chlorodifluoro- methane	75-45-6											
Cyclohexane	110-82-7	84	2500	8589	3074	10	10	10	-	1	3.1E-01	8.9E-02
Cyclohexene	110-83-8	82	150	503	90	10	10	10	10	5	1.8E-03	5.1E-04
Cyclopentane	287-92-3	70	8110	23,219	4155	10	10	10	10	5	8.3E-02	2.4E-02
Cyclopentene	142-29-0	68	1139	3,168	19	10	10	10	-	1	1.9E-02	5.4E-03
Dichlorofluoro- methane	75-43-4											
Ethanol	64-17-5	46	6000	11,289	2357	10	10	10	-	3	7.9E-01	2.3E-01
Freon 114	76-14-2											
Heptanal	111-71-7											
Indan	496-11-7											
Indene	95-13-6											
Isobutane	75-28-5											
Isobutene 1-Butene	115-11- 7/106-98	56	500	1,146	205	10	10	10	-	3	6.8E-02	2.0E-02
Isobutylbenzene	538-93-2											

CHEMICAL	CASRN	CASRN MOLE- CULAR WEIGHT	NOAEL (ppm)	NOAEL DOSE CONVERSION (mg/m³)	NOAEL DOSE ADJUSTED			RTAII YING	FACT		RfC (mg/m³)	RfDi (mg/kg- day)
						10H	10A	10S	10 L	MF		
Isoheptane	591-76-4											
Isohexane	73513-42- 5											
Isopentane	78-78-4											
Isoprene	78-79-5											
m-Diethylbenzene	141-93-5	134	2200	12,058	2877	10	10	10	-	3	9.6E-01	2.7E-01
Methylcyclo-pentane	96-37-7											
Methylcyclo-pentene	27476-50-											
n-Butane	106-97-8											
n-Decane	124-18-5											
Neohexane	75-83-2											
Neopentane	463-82-1											
n-Nonane	111-84-2	128	590	3,089	604	10	10	10	-	1	6.0E-02	1.7E-02
n-Octane	111-65-9											
n-Undecane	1120-21-4											
p-Diethylbenzene	105-05-5											
p-Isopropyltoluene	99-87-6											
Propane	74-98-6											
p-Xylene	106-42-3											
m-Xylene	108-38											
t-2-Butene	624-64-6											
t-2-Hexene	4050-45-7											
t-2-Pentene	646-04-8											
Isodrin	465-73-6											
PM2.5	NA											

CHEMICAL	CASRN	MOLE- CULAR WEIGHT	NOAEL (ppm)	NOAEL DOSE CONVERSION (mg/m³)	NOAEL DOSE ADJUSTED	N	UNCERTAINTY AND MODIFYING FACTORS							RfDi (mg/kg- day)
			, ,		10H	10A	10S	10 L	MF		5 /			
1-Naphthylamine	134-32-7													
2-Methylnaphthalene	91-57-6													
2-Nitrophenol	88-75-5													

Note: The RfDi values are based on information presented in Table 6. Not all toxicological information presented in Table 6 was used to derive an RfDi in this table. Modifying factors were based on professional judgment regarding duration of the toxicological study, number of experimental animals, survival rates, toxicological endpoints, etc. The derivation of NOAEL, NOAEL conversion and NOAEL dose adjustment is presented in section 4.2.4.

Tables 8, 9, and 10 present the calculated hazard quotients and hazard indices for the Residential Towers site, GEMB, and School sites under a 30, 6, and 3 year exposure duration. Note, the CDI was calculated for a 30 year exposure. It was adjusted for a 6 and 3 year exposure by multiplying the CDI by 1/5 and 1/10, respectively. The chronic daily intake (CDI) was calculated for 30, 6, and 3 year exposures. The following equation, as defined on page 24 of this report, was used:

CDI (mg/kg-day) =
$$(CA * IR * ET * EF * ED)$$

(BW * AT)

To account for 6 years as a child and 24 years as an adult during the 30 year exposure period, the following equation from EPA Region 9 "Preliminary Remediation Goals" (http://www.epa.gov/region09/waste/sfund/prg), was used to calculated the adjusted inhalation factor (InhF_{adj}):

$$InhF_{adj} (m^{3}\text{-year/kg-day}) = \underbrace{(ED_{c}*IRA_{c})}_{BW_{c}} + \underbrace{(ED_{r}-ED_{c})*(IRA_{a})}_{BW_{a}}$$

 $ED_c = Exposure duration in child = 6 years$

 $IRA_c = Inhalation rate in child = 12 \text{ m}^3/\text{day}$

 $BW_c = Body$ weight in child = 15 kg

 ED_r = Exposure duration – residential = 30 years

 $IRA_a = Inhalation rate in adult = 20 \text{ m}^3/\text{day}$

 $Bw_a = Body$ weight in adult = 70 kg

Incorporating InhF_{adj} into the CDI equation, it becomes:

CDI
$$_{30 \text{ years}} = \frac{\text{(CA * ET * EF * InhF}_{adj})}{\text{AT}}$$

CA = Chemical concentration in air = RME = Reasonable Maximum Exposure = mg/m³

ET = Exposure Time = 24 hours per day

EF = Exposure Frequency = 350 days per year

AT = Averaging Time = 365 days per years * 30 years = 10,950 days

RFD_i values from Table 7 were advanced to Tables 8, 9, and 10 and used in the calculations of the Hazard Quotient (HQ) for 30, 6, and 3 year exposures.

TABLE 8 HAZARD QUOTIENTS AND INDICES— RESIDENTIAL TOWERS

CHEMICAL	CASRN	RfDi (mg/kg-day)	RME	CHRONIC DAILY INTAKE 30YEARS (mg/kg-day)	HAZARD QUOTIENT 30 YEARS	CHRONIC DAILY INTAKE 3/6 YEARS (mg/kg-day)	HAZARD QUOTIENT 3/6 YEARS
Hydrofluoric Acid	7664-39-3						
Hexanal	66-25-1						
Isovaleraldehyde	590-86-3						
n-Butyraldehyde	123-72-8						
Propionaldehyde	123-38-6	2.60E-02	7.54E-04	2.87E-04	1.10E-02	5.81E-04	2.23E-02
Tolualdehyde	529-20-4						
Valeraldehyde	110-62-3						
Chloride	16887-00-6						
Fluoride	16984-48-8						
Sulfate	NA						
1,2,3-Trimethylbenzene	526-73-8						
1-Decene	872-05-9						
1-Heptene	592-76-7						
1-Hexene	592-41-6	1.80E-01	4.96E-04	188E-04	1.05E-03	3.82E-04	2.12E-03
1-Nonene	124-11-8						
1-Octene	111-66-0						
1-Pentene	109-67-1						
1-Undecene	821-95-4						
2,2,3-Trimethylpentane	564-02-3						
2,2,5-Trimethylhexane	3522-94-9						
2,3,4-Trimethylpentane	565-75-3						
2,3-Dimethylbutane	79-29-8						
2,3-Dimethylpentane	565-59-3						
2,4,4-Trimethyl-1-Pentene	107-39-1						

CHEMICAL	CASRN	RfDi (mg/kg-day)	RME	CHRONIC DAILY INTAKE 30YEARS (mg/kg-day)	HAZARD QUOTIENT 30 YEARS	CHRONIC DAILY INTAKE 3/6 YEARS (mg/kg-day)	HAZARD QUOTIENT 3/6 YEARS
2,4-Dimethylpentane	108-08-7						
2,5-Dimethylhexane	592-13-2						
2-Ethyl-1-Butene	760-21-4						
2-Methyl-1-Pentene	763-29-1						
2-Methyl-2-Pentene	625-27-4						
2-Methylheptane	592-27-8						
3-Methyl-1-Butene	563-45-1						
3-Methylheptane	589-81-1						
3-Methylhexane	589-34-4						
3-Methylpentane	96-14-0						
4-Methyl-1-Pentene	691-37-2						
3-Methylphenol	108-39-4	5.00E-02		ND			
4-Methylphenol	106-44-5	5.00E-03		ND			
4-Nonene	2198-23-4						
a-Pinene	80-56-8						
b-Pinene	127-91-3						
Butyl Acrylate	141-32-2	4.00E-03		ND			
c-2-Butene	590-18-1						
c-2-Hexene	7688-21-3						
c-2-Octene	7642-04-8						
c-2-Pentene	627-20-3						
c-3-Hexene	7642-09-3						
c-3-Methyl-2-Pentene	922-61-2						
Chlorodifluoro-methane	75-45-6	5.00E+01	5.07E-03	1.93E-03	3.85E-05	3.90E-03	7.81E-05
Cyclohexane	110-82-7	8.80E-01	2.54E-03	9.65E-04	1.10E-03	1.96E-03	2.22E-03
Cyclohexene	110-83-8	5.10E-04	2.04E-04	7.75E-05	1.52E-01	1.57E-04	3.08E-01
Cyclopentane	287-92-3	2.40E-02	5.92E-04	2.25E-04	9.37E-03	4.56E-04	1.90E-02
Cyclopentene	142-29-0	5.40E-03	1.16E-04	4.41E-05	8.16E-03	8.93E-05	1.65E-02
Dichlorofluoro-methane	75-43-4	5.70E-02	1.22E-04	4.64E-05	8.13E-04	9.39E-05	1.65E-03
Ethanol	64-17-5	2.30E-01	9.56E-01	3.63E-01	1.58E+00	7.36E-01	3.20E+00
Freon 114	76-14-2						

CHEMICAL	CASRN	RfDi (mg/kg-day)	RME	CHRONIC DAILY INTAKE 30YEARS (mg/kg-day)	HAZARD QUOTIENT 30 YEARS	CHRONIC DAILY INTAKE 3/6 YEARS (mg/kg-day)	HAZARD QUOTIENT 3/6 YEARS
Heptanal	111-71-7						
Indan	496-11-7						
Indene	95-13-6						
Isobutane	75-28-5						
Isobutene 1-Butene	115-11-7/106-98	2.00E-01		ND			
Isobutylbenzene	538-93-2						
Isoheptane	591-76-4						
Isohexane	73513-42-5						
Isopentane	78-78-4						
Isoprene	78-79-5						
m-Diethylbenzene	141-93-5	2.70E-01	3.36E-04	1.28E-04	4.73E-04	2.59E-04	9.58E-04
Methylcyclo-pentane	96-37-7						
Methylcyclo-pentene	27476-50-2						
n-Butane	106-97-8						
n-Decane	124-18-5						
Neohexane	75-83-2						
Neopentane	463-82-1						
n-Nonane	111-84-2	1.70E-01	1.32E-03	5.02E-04	2.95E-03	1.02E-03	5.98E-03
n-Octane	111-65-9						
n-Undecane	1120-21-4						
p-Diethylbenzene	105-05-5						
p-Isopropyltoluene	99-87-6						
Propane	74-98-6						
p-Xylene	106-42-3	2.00E-1		ND			
m-Xylene	108-38	2.00E-1		ND			
t-2-Butene	624-64-6						
t-2-Hexene	4050-45-7						
t-2-Pentene	646-04-8						
Isodrin	465-73-6						
PM2.5	NA						

CHEMICAL	CASRN	RfDi (mg/kg-day)	RME	CHRONIC DAILY INTAKE 30YEARS (mg/kg-day)	HAZARD QUOTIENT 30 YEARS	CHRONIC DAILY INTAKE 3/6 YEARS (mg/kg-day)	HAZARD QUOTIENT 3/6 YEARS
1-Naphthylamine	134-32-7						
2-Methylnaphthalene	91-57-6						
2-Nitrophenol	88-75-5						
HAZARD INDEX					1.77E+00		3.58E+00

Note: Shaded RfDi cells were developed in this report based on toxicological information in peer-reviewed sources.

Unshaded RfDi cells were derived by EPA Region 9 (presented in PRG tables). For cells indicating ND for the CDI, the chemical was either non-detect or was not analyzed. Hazard Quotients and Indices were calculated under the assumption of a 30-, 6-, and 3-year year exposure duration.

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TABLE 9 HAZARD QUOTIENTS AND INDICES— GEMB SITE

CHEMICAL	CASRN	RfDi (mg/kg-day)	RME	CHRONIC DAILY INTAKE 30 YEARS (mg/kg-day)	HAZARD QUOTIENT 30 YEARS	CHRONIC DAILY INTAKE 3/6 YEARS (mg/kg-day)	HAZARD QUOTIENT 3/6 YEARS
Hydrofluoric Acid	7664-39-3						
Hexanal	66-25-1						
Isovaleraldehyde	590-86-3						
n-Butyraldehyde	123-72-8						
Propionaldehyde	123-38-6	2.60E-02	7.62E-04	2.90E-04	1.11E-02	5.87E-04	2.26E-02
Tolualdehyde	529-20-4						
Valeraldehyde	110-62-3						
Chloride	16887-00-6						
Fluoride	16984-48-8						
Sulfate	NA						
1,2,3-Trimethylbenzene	526-73-8						
1-Decene	872-05-9						
1-Heptene	592-76-7						
1-Hexene	592-41-6	1.80E-01	7.84E-04	2.98E-04	1.66E-03	6.04E-04	3.35E-03
1-Nonene	124-11-8						
1-Octene	111-66-0						
1-Pentene	109-67-1						
1-Undecene	821-95-4						
2,2,3-Trimethylpentane	564-02-3						
2,2,5-Trimethylhexane	3522-94-9						
2,3,4-Trimethylpentane	565-75-3						
2,3-Dimethylbutane	79-29-8						
2,3-Dimethylpentane	565-59-3						

CHEMICAL	CASRN	RfDi (mg/kg-day)	RME	CHRONIC DAILY INTAKE 30 YEARS (mg/kg-day)	HAZARD QUOTIENT 30 YEARS	CHRONIC DAILY INTAKE 3/6 YEARS (mg/kg-day)	HAZARD QUOTIENT 3/6 YEARS
2,4,4-Trimethyl-1-Pentene	107-39-1						
2,4-Dimethylpentane	108-08-7						
2,5-Dimethylhexane	592-13-2						
2-Ethyl-1-Butene	760-21-4						
2-Methyl-1-Pentene	763-29-1						
2-Methyl-2-Pentene	625-27-4						
2-Methylheptane	592-27-8						
3-Methyl-1-Butene	563-45-1						
3-Methylheptane	589-81-1						
3-Methylhexane	589-34-4						
3-Methylpentane	96-14-0						
4-Methyl-1-Pentene	691-37-2						
3-Methylphenol	108-39-4	5.00E-02		ND			
4-Methylphenol	106-44-5	5.00E0-03		ND			
4-Nonene	2198-23-4						
a-Pinene	80-56-8						
b-Pinene	127-91-3						
Butyl Acrylate	141-32-2	4.00E-03		ND			
c-2-Butene	590-18-1						
c-2-Hexene	7688-21-3						
c-2-Octene	7642-04-8						
c-2-Pentene	627-20-3						
c-3-Hexene	7642-09-3						
c-3-Methyl-2-Pentene	922-61-2						
Chlorodifluoro-methane	75-45-6	5.00E+01	4.00E-03	1.52E-03	3.04E-05	3.08E-03	6.16E-05
Cyclohexane	110-82-7	8.80E-01	1.57E-03	5.97E-04	6.78E-04	1.21E-03	1.37E-03
Cyclohexene	110-83-8	5.10E-04	2.33E-04	8.85E-05	1.74E-01	1.79E-04	3.52E-01
Cyclopentane	287-92-3	2.40E-02	3.50E-04	1.33E-04	5.54E-03	2.70E-04	1.12E-02
Cyclopentene	142-29-0	5.40E-03	1.35E-04	5.13E-05	9.50E-03	1.04E-04	1.93E-02
Dichlorofluoro-methane	75-43-4	5.70E-02	2.59E-04	9.84E-05	1.73E-03	1.99E-04	3.50E-03

CHEMICAL	CASRN	RfDi (mg/kg-day)	RME	CHRONIC DAILY INTAKE 30 YEARS (mg/kg-day)	HAZARD QUOTIENT 30 YEARS	CHRONIC DAILY INTAKE 3/6 YEARS (mg/kg-day)	HAZARD QUOTIENT 3/6 YEARS
Ethanol	64-17-5	2.30E-01	4.22E-02	1.60E-02	6.97E-02	3.25E-02	1.41E-01
Freon 114	76-14-2						
Heptanal	111-71-7						
Indan	496-11-7						
Indene	95-13-6						
Isobutane	75-28-5						
Isobutene 1-Butene	115-11-7/106-98	2.00E-01		ND			
Isobutylbenzene	538-93-2						
Isoheptane	591-76-4						
Isohexane	73513-42-5						
Isopentane	78-78-4						
Isoprene	78-79-5						
m-Diethylbenzene	141-93-5	2.70E-01	3.89E-04	1.48E-04	5.47E-04	3.00E-04	1.11E-03
Methylcyclo-pentane	96-37-7						
Methylcyclo-pentene	27476-50-2						
n-Butane	106-97-8						
n-Decane	124-18-5						
Neohexane	75-83-2						
Neopentane	463-82-1						
n-Nonane	111-84-2	1.70E-01	1.47E-03	5.59E-04	3.29E-03	1.13E-03	6.66E-03
n-Octane	111-65-9						
n-Undecane	1120-21-4						
p-Diethylbenzene	105-05-5						
p-Isopropyltoluene	99-87-6						
Propane	74-98-6						
p-Xylene	106-42-3	2.00E-01		ND			
m-Xylene	108-38	2.00E-01		ND			
t-2-Butene	624-64-6						
t-2-Hexene	4050-45-7						
t-2-Pentene	646-04-8						

CHEMICAL	CASRN	RfDi (mg/kg-day)	RME	CHRONIC DAILY INTAKE 30 YEARS (mg/kg-day)	HAZARD QUOTIENT 30 YEARS	CHRONIC DAILY INTAKE 3/6 YEARS (mg/kg-day)	HAZARD QUOTIENT 3/6 YEARS
Isodrin	465-73-6						
PM2.5	NA						
1-Naphthylamine	134-32-7						
2-Methylnaphthalene	91-57-6						
2-Nitrophenol	88-75-5						
HAZARD INDEX					2.77E-01		5.62E-01

Note: Shaded RfDi cells were developed in this report based on toxicological information. Un-shaded RfDi cells were derived by EPA Region 9 (presented in PRG tables). For cells indicating ND for the CDI, the chemical was either non-detect or was not analyzed. Hazard Quotients and Indices were calculated under the assumption of a 30-, 6-, and 3-year exposure duration.

TABLE 10 HAZARD QUOTIENTS AND INDICES— SCHOOL SITE

CHEMICAL	CASRN	RfDi (mg/kg-day)	RME	CHRONIC DAILY INTAKE 30 YEARS (mg/kg-day)	HAZARD QUOTIENT 30 YEARS	CHRONIC DAILY INTAKE 3/6 YEARS (mg/kg-day)	HAZARD QUOTIENT 3/6 YEARS
Hydrofluoric Acid	7664-39-3						
Hexanal	66-25-1						
Isovaleraldehyde	590-86-3						
n-Butyraldehyde	123-72-8						
Propionaldehyde	123-38-6	2.60E-02	8.49E-04	3.23E-04	1.24E-02	6.54E-04	2.51E-02
Tolualdehyde	529-20-4						
Valeraldehyde	110-62-3						
Chloride	16887-00-6						
Fluoride	16984-48-8						
Sulfate	NA						
1,2,3-Trimethylbenzene	526-73-8						
1-Decene	872-05-9						
1-Heptene	592-76-7						
1-Hexene	592-41-6	1.80E-01	5.80E-04	2.20E-04	1.22E-03	4.47E-04	2.48E-03
1-Nonene	124-11-8						
1-Octene	111-66-0						
1-Pentene	109-67-1						
1-Undecene	821-95-4						
2,2,3-Trimethylpentane	564-02-3						
2,2,5-Trimethylhexane	3522-94-9	-	·				
2,3,4-Trimethylpentane	565-75-3						
2,3-Dimethylbutane	79-29-8						
2,3-Dimethylpentane	565-59-3	-	<u>-</u>				

CHEMICAL	CASRN	RfDi (mg/kg-day)	RME	CHRONIC DAILY INTAKE 30 YEARS (mg/kg-day)	HAZARD QUOTIENT 30 YEARS	CHRONIC DAILY INTAKE 3/6 YEARS (mg/kg-day)	HAZARD QUOTIENT 3/6 YEARS
2,4,4-Trimethyl-1-Pentene	107-39-1						
2,4-Dimethylpentane	108-08-7						
2,5-Dimethylhexane	592-13-2						
2-Ethyl-1-Butene	760-21-4						
2-Methyl-1-Pentene	763-29-1						
2-Methyl-2-Pentene	625-27-4						
2-Methylheptane	592-27-8						
3-Methyl-1-Butene	563-45-1						
3-Methylheptane	589-81-1						
3-Methylhexane	589-34-4						
3-Methylpentane	96-14-0						
4-Methyl-1-Pentene	691-37-2						
3-Methylphenol	108-39-4	5.00E-02		ND			
4-Methylphenol	106-44-5	5.00E-03		ND			
4-Nonene	2198-23-4						
a-Pinene	80-56-8						
b-Pinene	127-91-3						
Butyl Acrylate	141-32-2	4.00E-03	1.75E-04	6.65E-05	1.66E-02	1.35E-04	3.37E-02
c-2-Butene	590-18-1						
c-2-Hexene	7688-21-3						
c-2-Octene	7642-04-8						
c-2-Pentene	627-20-3						
c-3-Hexene	7642-09-3						
c-3-Methyl-2-Pentene	922-61-2						
Chlorodifluoro-methane	75-45-6	5.00E+01	4.15E-03	1.58E-03	3.15E-05	3.20E-03	6.39E-05
Cyclohexane	110-82-7	8.80E-01	9.45E-04	3.59E-04	4.08E-04	7.28E-04	8.27E-04
Cyclohexene	110-83-8	5.10E-04	2.42E-04	9.20E-05	1.80E-01	1.86E-04	3.65E-01
Cyclopentane	287-92-3	2.40E-02	3.46E-04	1.31E-04	5.48E-03	2.66E-04	1.11E-02
Cyclopentene	142-29-0	5.40E-03	1.30E-04	4.94E-05	9.15E-03	1.00E-04	1.85E-02
Dichlorofluoro-methane	75-43-4	5.70E-02	ND				

CHEMICAL	CASRN	RfDi (mg/kg-day)	RME	CHRONIC DAILY INTAKE 30 YEARS (mg/kg-day)	HAZARD QUOTIENT 30 YEARS	CHRONIC DAILY INTAKE 3/6 YEARS (mg/kg-day)	HAZARD QUOTIENT 3/6 YEARS
Ethanol	64-17-5	2.30E-01	3.71E-02	1.41E-02	6.13E-02	2.86E-02	1.24E-01
Freon 114	76-14-2						
Heptanal	111-71-7						
Indan	496-11-7						
Indene	95-13-6						
Isobutane	75-28-5						
Isobutene	115-11-	2.00E-01		ND			
1-Butene	7/106-98	2.00E-01		ND			
Isobutylbenzene	538-93-2						
Isoheptane	591-76-4						
Isohexane	73513-42-5						
Isopentane	78-78-4						
Isoprene	78-79-5						
m-Diethylbenzene	141-93-5	2.70E-01	3.72E-04	1.41E-04	5.24E-04	2.86E-04	1.06E-03
Methylcyclo-pentane	96-37-7						
Methylcyclo-pentene	27476-50-2						
n-Butane	106-97-8						
n-Decane	124-18-5						
Neohexane	75-83-2						
Neopentane	463-82-1						
n-Nonane	111-84-2	1.70E-01	1.29E-03	4.90E-04	2.88E-03	9.93E-04	5.84E-03
n-Octane	111-65-9						
n-Undecane	1120-21-4						
p-Diethylbenzene	105-05-5						
p-Isopropyltoluene	99-87-6						
Propane	74-98-6						
p-Xylene	106-42-3	2.00E-01		ND			
m-Xylene	108-38	2.00E-01		ND			
t-2-Butene	624-64-6						
t-2-Hexene	4050-45-7						
t-2-Pentene	646-04-8						

CHEMICAL	CASRN	RfDi (mg/kg-day)	RME	CHRONIC DAILY INTAKE 30 YEARS (mg/kg-day)	HAZARD QUOTIENT 30 YEARS	CHRONIC DAILY INTAKE 3/6 YEARS (mg/kg-day)	HAZARD QUOTIENT 3/6 YEARS
Isodrin	465-73-6						
PM2.5	NA						
1-Naphthylamine	134-32-7						
2-Methylnaphthalene	91-57-6						
2-Nitrophenol	88-75-5						
HAZARD INDEX					2.90E-01		5.88E-01

Notes: Shaded RfDi cells were developed in this report based on toxicological information. Un-shaded RfDi cells were derived by EPA Region 9 (presented in PRG tables). For cells indicating ND for the CDI, the chemical was either non-detect or was not analyzed. Hazard Quotients and Indices were calculated under the assumption of a 30-, 6-, and 3-year exposure duration.

6 CONCLUSIONS AND RECOMMENDATIONS

This toxicological evaluation of Atsugi chemicals represents an exhaustive review and analysis of all available current toxicological investigations and regulatory databases. Insufficient toxicity information exists for most of the chemicals in this study. Although it cannot be ascertained empirically, it is likely that the reason so few toxicological studies have been conducted on the chemicals of interest in this report is that their toxicological significance is of no consequence. That is they likely possess low inherent toxicity. For that reason, the chemicals lacking toxicity values may best be viewed as chemicals posing very low health hazards. Particularly at the low levels detected at NAF Atsugi.

For those chemicals where toxicity values have been identified in EPA Region 9 PRG Tables or have been developed based on careful review of the peer-review toxicological literature, the estimated health hazards are extremely low and can be considered *de minimus*. With the exception of ethyl alcohol, all hazard quotients and indices were below 1.0. The sole reason the hazard quotient for ethyl alcohol exceeded one was its high level of uncertainty in the toxicity database. It is highly unlikely that ethyl alcohol poses a real health hazard at NAF Atsugi at the concentrations detected.

Review of all available information regarding the toxicities of the 86 chemicals for which toxicity values are not available indicates that no additional risk can be attributed to these 86 chemicals.

7 REFERENCES

7.1 Hydrogen Fluoride

- Meldrum M. Toxicology of hydrogen fluoride in relation to major accident hazards. Regul Toxicol Pharmacol. 1999 Oct;30(2 Pt 1):110-6. Review.
- Dalbey W, Dunn B, Bannister R, Daughtrey W, Kirwin C, Reitman F, Wells M, Bruce J. Short-term exposures of rats to airborne hydrogen fluoride. J Toxicol Environ Health A. 1998 Oct 23;55(4):241-75.
- Dalbey W, Dunn B, Bannister R, Daughtrey W, Kirwin C, Reitman F, Steiner A, Bruce J. Acute effects of 10-minute exposure to hydrogen fluoride in rats and derivation of a short-termexposure limit for humans. Regul Toxicol Pharmacol. 1998 Jun;27(3):207-16.
- Cittanova ML, Lelongt B, Verpont MC, Geniteau-Legendre M, Wahbe F, Prie D, Coriat P, Ronco PM. Fluoride ion toxicity in human kidney collecting duct cells. Anesthesiology. 1996 Feb;84(2):428-35.
- Stavert DM, Archuleta DC, Behr MJ, Lehnert BE. Relative acute toxicities of hydrogen fluoride, hydrogen chloride, and hydrogen bromide in nose-and pseudo-mouth-breathing rats. Fundam Appl Toxicol. 1991 May;16(4):636-55.
- Braun J, Stoss H, Zober A. Intoxication following the inhalation of hydrogen fluoride. Arch Toxicol. 1984 Nov;56(1):50-4.
- Sadilova MS, Selyankina KP Shturkina, OK. Experimental Studies on th3e effect of hydrogen flhuoride on the central nervous system. 1965 Hyg. Sanit. 30, 155-160.

7.2 Hexanal

- Martelli A, Canonero R, Cavanna M, Ceradelli M Marinari UM Cytotoxic and genotoxic effects of five n-alkanals in primary cultures of rat and human hepatocytes Mutat Res; VOL 323, ISS 3, 1994, P121-6.
- Komsta E Chu I Secours VE Valli VE Villeneuve DC Results of a short-term toxicity study for three organic chemicals found in Niagara River drinking water. Bull Environ Contam Toxicol; VOL 41, ISS 4, 1988, P515-22.
- Brambilla G Cajelli E Canonero R Martelli A Marinari UM Mutagenicity in V79 Chinese hamster cells of n-alkanals produced by lipid peroxidation. Mutagenesis; VOL 4, ISS 4, 1989, P277-9.

- Kaneko T, Kaji K, Matsuo M. Cytotoxicities of a linoleic acid hydroperoxide and its related aliphatic aldehydes toward cultured human umbilical vein endothelial cells.. Chem Biol Interact. 1988;67(3-4):295-304.
- Sciaba L, Finollo R, Bassi AM, Brambilla G. DNA-damaging activity of biotic and xenobiotic aldehydes in Chinese hamster ovary cells. Cell Biochem Funct. 1984 Oct;2(4):243-8.
- Poli G, Chiarpotto E, Biasi F, Pavia R, Albano E, Dianzani MU Enzymatic impairment induced by biological aldehydes in intact rat liver cells. Res Commun Chem Pathol Pharmacol 1982 Oct;38(1):71-6.
- Chiarpotto E, Cecchini G, Biasi F, Pavia R, Poli G. Functional impairment of intact rat liver cells due to biological aldehydes. Boll Soc Ital Biol Sper. 1982 Sep 30;58(18):1189-94.

7.3 Isovaleraldehyde

- Auerbach C; Moutschen-Dahmen M; Moutschen J Genetic and cytogenetical effects of formaldehyde and related compounds. MUTAT RES; 39 (3-4). 1977 317-362.
- Wingard C; Hitchcock P; Teague RS A Survey Of Aldehydes With Respect To Their Action On The Blood Pressure. Archives Internationales de Pharmacodynamie et de Therapie, Vol. 102, No. 1-2, pages 65-84, 17 references, 1955.
- Safronkina EI; Kiseleva AV; Gafurova EV [Basis for the maximum permissible concentration of isovaleric aldehyde] Gig Tr Prof Zabol, ISS 5, 1983, P60-1 [Russian].
- Marshall AW; DeSouza M; Morgan MY Plasma 3-methylbutanal in man and its relationship to hepatic encephalopathy. Clin Physiol; VOL 5, ISS 1, 1985, P53-62.
- Steinhagen WH, Barrow CS Sensory Irritation Structure-Activity Study Of Inhaled Aldehydes InB6C3F1 And Swiss-Webster Mice. Toxicology and Applied Pharmacology, Vol. 72, No. 3, pages 495-503.

7.4 Propionaldehyde

- Anonymous Propionaldehyd TA:Beratergremium fuer umweltrelevante Altstoffe (BUA) PG:132 p YR:1997 IP: VI:195.
- Anonymous Propionaldehydy, TA:Toxikologische Bewertung. Heidelberg, Berufsgenossenschaft der chemischen IndustriePG:23 p YR:1991 IP: VI:207.
- Koerker RL; Berlin AJ; Schneider FH. The cytotoxicity of short-chain alcohols and aldehydes in cultured neuroblastomacells. Toxicol Appl Pharmacol; 37 (2). 1976 281-288.

- Bassi AM; Penco S; Canuto RA; Muzio G; Ferro M Comparative evaluation of cytotoxicity and metabolism of four aldehydes in twohepatoma cell lines. Drug Chem Toxicol; VOL 20, ISS 3, 1997, P173-87.
- Bassi Am; Penco S; Canuto Ra; Muzio G; Ferro M Comparative evaluation of cytotoxicity and metabolism of four aldehydes in twohepatoma cell lines. Drug And Chemical Toxicology An International Journal For Rapid Communication; 20 (3). 1997. 173-187.
- Garnier R; Chataigner D; Efthymiou ML Aldehydes Encyclopâedie mâedico-chirurgicale Intoxications, Pathologie du travail, 1990, 13p.
- Dillon D; Combes R; Zeiger E The effectiveness of Salmonella strains TA100, TA102 and TA104 for detectingmutagenicity of some aldehydes and peroxides. Mutagenesis; VOL 13, ISS 1, 1998, P19-26.
- Bombick Dw; Doolittle Dj The role of chemical structure and cell type in the cytotoxicity of low-molecular-weight aldehydes and pyridines. In Vitro Toxicology; 8 (4). 1995. 349-356.
- Babiuk C; Steinhagen WH; Barrow CS Sensory irritation response to inhaled aldehydes after formaldehyde pretreatment. Toxicol Appl Pharmacol; VOL 79, ISS 1, 1985, P143-9.
- Gage JC The Subacute Inhalation Toxicity of 109 Industrial Chemicals British Journal of Industrial Medicine, Vol. 27, pages 1-18.
- Tokanova ShE [Biological action and hygienic evaluation of propionaldehyde and propionic acidas air pollutants of populated sites] Gig Sanit, ISS 4, 1982, P10-3.
- Mel'nikova AP; Tokanova ShE [Biological action of propionaldehyde and propionic acid on the body of experimental animals] Gig Sanit, ISS 4, 1983, P74-.

7.5 Tolualdehyde

- Opdyke etal. Monographs on Fragrance Raw Materials, New York: Paergamon Press, p. 707. 1979.
- Anon BIBRA Toxicity Profile of tolualdehydes. Govt Reports Announcements & Index (GRA&I), Issue 19, 1996.
- Branton Pg; Gaunt If; Grasso P; Lansdown A Bg Short-term toxicity of tolualdehyde in rats; Gangolli Sd Food Cosmet Toxicol; 10 (5). 1972 637-647.
- BIBRA working group Tolualdehydes, Toxicity profile. The British Industrial Biological Research Association. PG:3 p YR:1990 IP.

7.6 Valeraldehyde

- Savolainen H; Pfaffli P; Elovaara E Blood And Brain n-Pentanol In Inhalation Exposure Acta Pharmacologica et Toxicologica, Vol. 56, No. 3, pages 260-264, 23 references, 1985.
- BIBRA working group Valeraldehyde Toxicity profile. The British Industrial Biological Research Association PG:4 p YR:1986 IP.
- Wingard C; Hitchcock P; Teague RS A Survey Of Aldehydes With Respect To Their Action On The Blood Pressure Archives Internationales de Pharmacodynamie et de Therapie, Vol. 102, No. 1-2, pages, 65-84, 17 references.
- Brambilla G; Cajelli E; Canonero R; Martelli A; Marinari UM Mutagenicity in V79 Chinese hamster cells of n-alkanals produced by lipid Peroxidation. Mutagenesis; VOL 4, ISS 4, 1989, P277-9.
- Damgêard Nielsen G; Kragh Hansen M; M²lhave L Arbejdstilsynet, Arbejdsmilj²instituttet, Rosenvaengets Allâe Toxicological evaluation of a number of substances that may pollute the workplace air 16-18, 2100 K²benhavn ¢, Denmark, Aug. 1982. 102p. Illus. 268 ref. 8
- ANON National Institute for Occupational Safety and Health NIOSH Current Intelligence Bulletin No.55 - Carcinogenicity of acetaldehyde and malonaldehyde, and mutagenicity of related low-molecular-weight aldehydes 4676 Columbia Parkway, Cincinnati, OH 45226, USA, Sep. 1991. 39p.
- Garnier R; Chataigner D; Efthymiou ML Aldehydes. Encyclopâedie mâedico-chirurgicale Intoxications, Pathologie du travail, 1990, 13p. Illus. 208.

7.7 1,2,3-Trimethylbenzene

- Korsak Z; Rydzynski K Neurotoxic Effects of Acute and Subchronic Inhalation Exposure to Trimethylbenzene Isomers (Pseudocumene, Mesitylene, Hemimellitene) in Rats International Journal of Occupational Medicine and Environmental Health, Vol. 9, No. 4, pages 341-349, 17 references, 1996.
- Anonymous, Information Profiles on Potential Occupational Hazards: Trimethylbenzenes Center for Chemical Hazard Assessment, Syracuse Research Corporation, Syracuse, New York, Report No. SRC TR 81-526, Contract No. 210-79-0030, 33 pages, 51 references, 1981.
- Wiaderna D; Gralewicz S; Tomas T Behavioural changes following a four-week inhalation exposure to hemimellitene (1,2,3-trimethylbenzene) in rats. Int J Occup Med Environ Health; VOL 11, ISS 4, 1998, P319-34.

- Jarnberg J; Johanson G; L Toxicokinetics of Inhaled Trimethylbenzenes in Man. Toxicology and Applied Pharmacology, Vol. 140, No. 2, pages 281-288, 21 references, 1996.
- Mikulski P; Wiglusz R; Galuszko E; Delag G Reciprocal metabolic effect of benzene and its methyl derivatives in rats: 1. Study in vivo. Bull Inst Marit Trop Med Gdynia; 30 (1). 1979. 77-88.
- Delic J; Gardner R; Cocker J; Widdowson EM; Brown R Trimethylbenzenes: Criteria document for an occupational exposure limit HMSO Books, P.O. Box 276, London SW8 5DT, United Kingdom, 1992. vi, 33p.
- Mikulski PI; Wiglusz R Toxicol The comparative metabolism of mesitylene, pseudocumene, and hemimellitene inrats. Appl Pharmacol; VOL 31, ISS 1, 1975, P21-31.
- Gajsinskaja SE; Daskina ZI Toxicological assessment of trimethylbenzenes Uzdavini ER; Gigiena truda i professional'nye zabolevanija Oct. 1984, No.10, p.54-55. 4 ref.
- ACGIH Trimethyl benzene isomers Anonymous TA:. Documentation of the threshold limit values and biological exposure indicesPG:1648-9 YR:1991 IP: VI:6th Ed.
- EPA working group Health effects assessment for Trimethylbenzenes Environmental Protection Agency PG:32 p YR:1987 IP: VI:EPA/600/8-88/060.

7.8 1-Heptene

- Damgêard Nielsen G Kragh Hansen M M²lhave L Arbejdstilsynet Toxicological evaluation of a number of substances that may pollute the workplace air, Arbejdsmilj²instituttet, Rosenvaengets Allâe 16-18, 2100 K²benhavn ¢, Denmark, Aug. 1982. 102p. Illus. 268 ref.
- Von Oettingen WF Toxicity and Potential Dangers of Aliphatic and Aromatic Hydrocarbons. A Critical Review of the Literature U.S. Public Health Bulletin, No. 255, 135 pages, 318 references, 19401940

7.9 1-Hexene

- Henderson VE; Smith AHR Propylene Impurities. Hexenes And Hexanes Journal of Pharmacology and Experimental Therapeutics, Vol. 58, No. 3, pages 319-327, 15
- Gingell R; Bennick JE; Malley LA Subchronic inhalation study of 1-hexene in Fischer 344 rats. Drug Chem Toxicol; VOL 22, ISS 3, 1999, P507-28 [TOXBIB]

- Gingell R; Daniel EM; Machado M; Bevan C Reproduction/developmental toxicity screening test in rats with orally-administered 1-hexene Drug Chem Toxicol 2000 May;23(2):327-38 [DART]
- James RC The Toxic Effects Of Organic Solvents Industrial Toxicology: Safety and Health Applications in the Workplace, Williams, P. L., and J. L. Burson, Editors; Van Nostrand Reinhold Company, New York, pages 230-259.
- Eide I; Hagemann R; Zahlsen K; Tareke E; Tornqvist M; Kumar R; Vodicka P; Hemminki K Uptake, distribution, and formation of hemoglobin and DNA adducts after inhalation of C2-C8 1-alkenes (olefins) in the rat CARCINOGENESIS (OXFORD); 16 (7). 1995. 1603-1609.
- ACGIH 1-Hexene, Documentation of the threshold limit values and biological exposure indices PG:3 p YR:1998 IP:Suppl. VI:6 th Ed.

7.10 1-Octene

- Eide I; Hagemann R; Zahlsen K; Tareke E; Tornqvist M; Kumar R; P; Hemminki K Uptake, distribution, and formation of hemoglobin and DNA adducts after inhalation of C2-C8 1-alkenes (olefins) in the rat. Carcinogenesis (Oxford); 16 (7). 1995. 1603-1609.
- Hempel-J²rgensen A; Kjaergaard SK; Mãolhave L; Hudnell HK Time course of sensory eye irritation in humans exposed to N-butanol and 1-octene. Arch Environ Health; VOL 54, ISS 2, 1999, P86-94
- ANON 1-Octene Instituto Nacional de Seguridad e Higiene en el Trabajo, Ediciones y Publicaciones, correlaguna 73, 28027 Madrid, Spain, 1991. 2p.
- ANON 1-Octene Official Publications of the European Communities, 2985 Luxembourg, Grand Duchy of Luxembourg; International Programme on Chemical Safety (IPCS), World Health Organization, 1211 Genáeve 27, Switzerland, 1993. 2p.
- Von Oettingen Toxicity and Potential Dangers of Aliphatic and Aromatic Hydrocarbons. A Critical Review of the Literature WF U.S. Public Health Bulletin, No. 255, 135 pages.]

7.11 2,2,5-Trimethylhexane

Halder Ca; Holdsworth Ce; Cockrell By; Piccirillo Vj Hydrocarbon nephropathy in female rats: Identification of the nephrotoxic components of unleaded gasoline Toxicol Ind Health; 1 (3). 1985 (Recd. 1986). 67-88.

7.12 2,3,4-Trimethylpentane

- Norton WN; Mattie DR The Cytotoxic Effects of Trimethylpentane on Rat Renal Tissue
- Scanning Microscopy, Vol. 1, No. 2, pages 783-790, 16 references, 1987.
- Olson CT; Hobson DW; Yu KO; Serve MP The Metabolism of 2,3,4-Trimethylpentane in Male Fischer-344 Rats Toxicology Letters, Vol. 37, No. 3, pages 199-202, 8 references, 1987.
- DelRaso NJ; Mattie DR; Godin CS Lack of Detectable Metabolism for Solubilized 2,3,4-Trimethylpentane by Rat Kidney Proximal Tubules Toxicology Letters, Vol. 54, No. 2, pages 337-344, 12 references, 1990.

7.13 2,3-Dimethylbutane

- Dutch expert committee on occupational standards Health-based recommended occupational exposure limit for 2-methylpentane, 3-methylpentane, 2,2-dimethylbutane, 2,3-dimethylbutane (hexane isomers) Directorate-General of Labour, the Netherlands PG:21 p YR:1993 IP: VI:RA 7/93.
- 2,3-Dimethylbutane. CAS# 79-29-8. Galvin JB; Panson R J Toxicol Environ Health; VOL 58, ISS 1-2, 1999, P111-8.

7.14 2,4,4-Trimethyl-1-Pentene

- ANON Workplace environmental exposure level guide Diisobutylene American Industrial Hygiene Association Journal Jan. 1982, Vol.43, No.1, p.B-83-B-84.
- Diisobutylene. Am Ind Hyg Assoc J; Vol 43, ISS 1, 1982, PB83-4 [TOXBIB]
- ANON 2,4,4-Trimethyl-1-penteneInstituto Nacional de Seguridad e Higiene en el Trabajo, Ediciones y Publicaciones, correlaguna 73, 28027 Madrid, Spain, 1991. 2p.
- ANON 2,4,4-Trimethyl-1-pentene Official Publications of the European Communities, 2985 Luxembourg, Grand Duchy of Luxembourg; International Programme on Chemical Safety (IPCS), World Health Organization, 1211 Geneva 27, Switzerland, 1993. 2p.

7.15 2,5-Dimethylhexane

Rabovsky J; Judy DJ The In Vitro Effects of Alkanes, Alcohols, and Ketones on Rat Lung Cytochrome P450-Dependent Alkoxphenoxyazone Dealkylase Activities Archives of Toxicology, Vol. 63, No. 1, pages 13-17, 28 references, 1989.

Serve MP; Bombick DD; Roberts J; McDonald GA; Mattie DR; Yu KO The Metabolism of 2,5-Dimethylhexane in Male Fischer 344 Rats Chemosphere, Vol. 22, Nos. 1-2, pages 77-84, 1991.

7.16 3-Methylheptane

- Rabovsky J; Judy DJ The In Vitro Effects of Alkanes, Alcohols, and Ketones on Rat Lung CytochromeP450-Dependent Alkoxphenoxyazone Dealkylase Activities Archives of Toxicology, Vol. 63, No. 1, pages 13-17.
- Serve MP; Bombick DD; Clemens JM; McDonald GA; Hixson CJ; Mattie DR The Metabolism of 3-Methylheptane in Male 344 Fischer Rats Chemosphere, Vol. 26, No. 9, pages 1667-1677, 42 references, 1993.

7.17 3-Methylpentane

- Brugnone F; Perbellini L; Grigolini L; Apostoli P Solvent exposure in a shoe upper factory: II. Methylcyclopentane, 2-methylpentane, and 3-methylpentane concentration in alveolar and in environmental air and in blood. Int Arch Occup Environ Health; 42 (3-4). 1979. 355-364.
- Frontali N; Amantini Mc; Spagnolo A; Guarcini Am; Saltari Mc Poly Neuropathy In Rats Treated With The Different Solvent Components Of The Glues Used In Shoe Factories 3rd European Neuroscience Meeting, Rome, Italy, Sept. 11-14, 1979. Neurosci Lett; Suppl. (3). 1979. S217.
- Nerve Ono Y; Takeuchi Y; Hisanaga N A Comparative Study on the Toxicity of n-Hexane and Its Isomers on the Peripheral Archives of Occupational and Environmental Health, Vol. 48, No. 3, pages, 289-294, 18 references, 1981.
- Galvin JB; Bond G J 3-Methylpentane. Toxicol Environ Health; VOL 58, ISS 1-2, 1999, P93-102.
- Dutch expert committee on occupational standards Health-based recommended occupational exposure limit for 2-methylpentane, 3-methylpentane, 2,2-dimethylbutane, 2,3-dimethylbutane (hexane isomers) -General of Labour, the Netherlands PG:21 p YR:1993.

7.18 4-Methylphenol/3-Methylphenol

Izard MK; George JD; Fail PA; Grizzle TB; Heindel JJ; Chapin RE Final report on the reproductive toxicity of meta-para-cresol (MPCRE) (CAS no. 1319-77-3) in CD-1 Swiss mice: volume 1. NTIS Technical Report (NTIS/PB92-191741) 1992 May;:295 pp. [DART]

- NTP working group Toxicity studies of cresols in F344/N rats and B6C3F1 mice (feed studies) National Toxicology Program PG:128 p YR:1992 IP: VI:TOX 9 [RISKLINE]
- Kavlock RJ Structure-activity relationships in the developmental toxicity of substituted phenols:in vivo effects. Teratology 1990 Jan;41(1):43-59 [DART]
- Thompson DC; Perera K; Fisher R; Brendel K Cresol Isomers: Comparison of Toxic Potency in Rat Liver Slices Toxicology and Applied Pharmacology, Vol. 125, No. 1, pages 51-58, 24 references, 1994.
- Anonymous Toxicological profile for cresols, o-cresol, p-cresol, m-cresol. Agency for Toxic Substances and Disease Registry U.S. Public Health PG:160 pYR:1992 IP: VI:ATSDR/TP-91/11 [RISKLINE]
- Dietz DD Toxicity Studies of Cresols (CAS Nos. 95-48-7, 108-39-4, 106-44-5) in F344 Rats and
- B6C3F1 Mice (Feed Studies) National Toxicology Program, U.S. Department of Health and Human Services, Research Triangle Park, North Carolina, NTP TOX 9, 128 pages, 148 references [NIOSH]
- Anonymous Reproductive toxicology. m-/p-cresol. Environ Health Perspect 1997 Feb;105 Suppl 1:295-6 [DART]

7.19 Cresol, all isomers

- ANONU.S. Department of Health and Human Services, Public Health Service, Center for Disease Control, National Institute for Occupational Safety and Health, Division of Standards Development and Technology Transfer, 4676 Columbia Parkway, Cincinnati, OH 45226, USA, 1988. 6p. Bibl. [CIS]
- EPA working group Health effects assessment for cresols. :Environmental protection agency PG:29 p YR:1984 IP: VI:EPA/540/1-86/050.
- NIOSH working group Occupational exposure to Cresol. Criteria for a recommended standard PG:117 p YR:1978 IP: VI:NIOSH 78-133.

7.20 a-Pinene

- BIBRA working groupToxicity alpha-Pinene profile. BIBRA Toxicology International PG:9 p YR:1992 IP: VI.
- Eriksson KA; Levin JO; Sandstrom T; Lindstrom-Espeling K; Linden G; Stjernberg NL Terpene Exposure and Respiratory Effects among Workers in Swedish Joinery Shops

- Scandinavian Journal of Work, Environment and Health, Vol. 23, No. 2, pages 114-120, 20 references, 1997.
- Filipsson AF Short term inhalation exposure to turpentine: toxicokinetics and acute effects in men. Occup Environ Med; VOL 53, ISS 2, 1996, P100-5.
- Anon BIBRA Toxicity Profile of alpha-pinene. Govt Reports Announcements & Index (GRA&I), Issue 19, 1996.

7.21 Butyl Acrylate

- BUA), n-Butyl acrylate (Aug 1992) Anonymous , Beratergremium fuer umweltrelevante Altstoffe (PG:81 p YR:1995 IP: VI:129.
- Anonymous, n-Butyl Acrylate, IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans: Some Chemicals Used in Plastics and Elastomers, International Agency for Research onCancer, Lyon, France, Vol. 39, pages 67-79, 50 references, 1986.
- Merkle J; Klimisch HJ n-Butyl acrylate: prenatal inhalation toxicity in the rat. Fundam Appl Toxicol; VOL 3, ISS 5, 1983, P443-7.
- Engelhardt G; Klimisch H-J n-Butyl Acrylate: Cytogenetic Investigations In The Bone Marrow Of Chinese Hamsters And Rats After 4-Day Inhalation. Fundamental and Applied Toxicology, Vol. 3, No. 6, pages 640-641, 6 references, 1983.
- ANON n-Butyl acrylate CAS No.141-32-2 European Centre for Ecotoxicology and Toxicology of Chemicals, Avenue E. Van Nieuwenhuyse 4, Bte.6, 1160 Bruxelles, Belgium, Aug. 1994. ii, 47p.
- Saillenfait AM; Bonnet P; Gallissot F; Protois JC; Peltier A; Fabri`es JF Relative developmental toxicities of acrylates in rats following inhalation exposure. Toxicol Sci 1999 Apr;48(2):240-54.
- BIBRA working group n-Butyl acrylate Toxicity profile. BIBRA Toxicology International PG:8 p YR:1991 IP: VI.
- Rats Sanders JM; Burka LT; Matthews HB Metabolism and Disposition of n-Butyl Acrylate in Male Fischer Drug Metabolism and Disposition, Vol. 16, No. 3, pages 429-434, 17 references, 1988.
- Reininghaus W; Koestner A; Klimisch HJ Chronic toxicity and oncogenicity of inhaled methyl acrylate and n-butyl acrylate in prague-Dawley rats. Food Chem Toxicol; VOL 29, ISS 5, 1991, P329-39.

- ANON Butyl acrylate Register of Safety Information of Chemical Products, National Board of Labour Protection, Box 536, 33101 Tampere, Finland, Feb. 1986. 2p. Original on microfiche.
- US Department of Health and Human Services, Buyl acrylate Public Health Service, Centers for Disease Control, National Institute for Occupational Safety and Health, Division of Standards Development and Technology Transfer, 4676 Columbia Parkway, Cincinnati, OH 45226, USA, 1992. 7p.
- Vodicka P; Gut I; Frantik E Effects of Inhaled Acrylic Acid Derivatives in Rats. Toxicology, Vol. 65, No. 1, pages 209-221.
- Chebotarev PA Establishment of the maximum permissible concentration in a single exposure to utylacrylate in the atmosphere. Sanit, ISS 9, 1981, P81-2.
- Kennedy G L Jr; Graepel Gj Acute toxicity in the rat following either oral or inhalation exposure. Toxicol Lett (Amst); 56 (3). 1991. 317-326.
- Chebotarev Pa Derivation Of A Single Occasion Maximum Permissibleconcentration For Butyl Acrylate In The Ambient Air Gig Sanit; 0 (9). 1981. 81-82.
- BIBRA Toxicity Profile of N-butyl acrylate. Govt Reports Announcements & Index (GRA&I), Issue 19, 1996.
- ACGIH. n-Butyl Acrylate Documentation of the threshold limit values and biological exposure indices PG:168 YR:1991 IP: VI:6th ed.

7.22 Chlorodifluoromethane

Tingston, D.J., Ls Chart, MJ Goodley etal, Chlorodifluoromethane (CFC22): Lont-term inhalation study in the rat. Report No. CTLP/548. Impoerial Chemical Industires Limited, Central Toxicology Laboratory, Alderly Park, Cheshire, UK, 1981.

7.23 Cyclohexane

- Kreckmann KH, Roberts LG Baldwin JK. Inhalation developmental toxicity studies of cyclohexane in rats and rabbits. Toxicologist 1998 Mar;42(1-S):256
- Kreckmann KH Roberts LG Staab RJ. Inhalation multigeneration reproduction study with cyclohexane in rats. Toxicologist 1998 Mar;42(1-S):105-6
- Fairhurst S.Cyclohexane. HSE Toxicity Review PG:6 p YR:1990 IP: VI:25.

- Health-based recommended occupational exposure limits for cyclohexane Dutch expert committee for occupational standards Directorate-General of Labour, the Netherlands PG:27 p YR:1990 IP: VI:RA 15/90
- ACGIH: Documentation of the Threshold Limit Values and Biological Exposure Indices, 6th ed Am. Conference of Govt Ind Hyg, Inc, Cincinnati, OH, 1991, pp 355-356.
- Bernard AM, DeRussis R, Normand, JC et al: Evaluation of the subacute nephrotoxicity of cyclohexane and other industrial solvents in the Sprague-Dawley rat. Tox Letters, 1989;45;271-280.
- Clayton GD & Clayton FE: Patty's Industrial Hygiene and Toxicology, Vol 2B, Toxicology, 3rd ed. John Wiley & Sons, NY, NY 1981.
- Frontali N, Amantini MC, Spagnolo A, et al: Experimental neurotoxicity and urinary metabolites of the C5-C7 aliphatic hydrocarbons used as glue solvents in shoe manufacture. Clin Toxicol 1981;18:1357-1367.
- Gosselin, RE, Smith RP, & Hodge, HC: Clinical Toxicology of Commercial Products, 5th ed. Williams and Wilkins, Baltimore, MD, 1984, p151.
- Hathaway, GJ, Proctor, NH, Hughs JP et al: Chemical Hazards in the workplace, 3rs ed. Van Nostrand Reinhold Company, NY, NY 1991, p197.
- Yuasa J, Kishi R, Eguchi T et al: Investigation on neurotoxicity of occupational exposure to cyclohexane a neurophysiological study. Occupational Environmental Med 1996, 53; 174-179.
- Cyclohexane ANON Indian Chemical Manufacturers Association, India Exchange, India Exchange Place, Calcutta 700 001, India, 1986. 1p.
- Cyclohexane ANON Institut national de recherche et de sâecuritâe, 30 rue Olivier-Noyer, 75680 Paris Cedex 14, France, 1988. 4p. Bibl.
- ACGIH. Cyclohexane Documentation of the threshold limit values and biological exposure indices PG:355-6 YR:1991 IP: VI:6th ed

7.24 Cyclohexene

- Laham S, Inhalation Toxicity Of Cyclo Hexene. Toxicol Appl Pharmacol; 37 (1). 1976 155-156.
- ACGIH, Cyclohexene Documentation of the threshold limit values and biological exposure indices, PG:362-3 YR:1991 IP: VI:6th ed

7.25 Cyclopentane

- Galvin JB; Marashi F Cyclopentane. CAS#287-92-3. J Toxicol Environ Health; VOL 58, ISS 1-2, 1999, P57-74.
- Kimmerle G; Thyssen J Acute, subacute and subchronic inhalation toxicity of cyclopentene. Int Arch Arbeitsmed; VOL 34, ISS 3, 1975, P177-84.
- ACGIH Cyclopentane Documentation of the threshold limit values and biological exposure indices. :371-2 YR:1991 IP: VI:6th ed.

7.26 Cyclopentene

Kimmerle G; Thyssen J Acute, subacute and subchronic inhalation toxicity of cyclopentene. Int Arch Arbeitsmed; VOL 34, ISS 3, 1975, P177-84.

7.27 Dichlorofluoromethane

- ECETOC working group Dichlorofluoromethane (HCFC-21) ECETOC Joint assessment of commodity chemicals PG:20 p YR:1990 IP: VI:16
- ANON, Dichlorofluoromethane (HCFC-21)
- European Chemical Industry Ecology and Toxicology Centre, Avenue Louise 250, B.63, 1050, Bruxelles, Belgium, Aug. 1990. 21p.
- Brandt-Rauf PW; Fallon LF Jr; Tarantini T; Idema C; Andrews L Health Hazards of Fire Fighters: Exposure Assessment British Journal of Industrial Medicine, Vol. 45, No. 9, pages 606-612, 30 references, 1988.
- Belej Ma; Aviado Dm, Acute Fluorocarbon Toxicity In Rhesus Monkey. Fed Proc; 32 (3 Part 1). 1973 814 [Heep]
- Edling C; Sèoderkvist P Criteria document for threshold limit values: Fluorocarbons
- Arbetarskyddsstyrelsen, 171 84 Solna, Sweden, 1982. 38p. 86 ref. [Swedish] [CIS]
- IARC, Chlorofluoromethane IARC monographs on the Evaluation of the Carcinogenic risk of chemicals to humans, PG:229-35 YR:1986 IP: VI:41.
- Dutch expert committee for occupational standards Health-based recommended occupational exposure limits for fluorcarbons (except FC11) Directorate-General of Labour, the Netherlands PG:70 p YR:1987 IP: VI:RA 15/87.

- ACGIH, Dichlorofluoromethane, ACGIH. Documentation of the threshold limit values and biological exposure indices PG:434-5 YR:1991 IP: VI:6th ed.
- EPA, Industrial Bio-test labotatories: Subacute Inhalation Toxicity Study with Genetron 21 in Albino Rats, 1979. EPA Doc number; FYI-OTS-0779-0045.

7.28 Ethanol

- Hau Km; Connell Dw; Richardson Bj Mechanism of acute inhalation toxicity of alkanes and aliphatic alcohols. Environmental Toxicology And Pharmacology; 7 (3). 1999. 159-167.
- Kennedy G L Jr; Graepel GJAcute toxicity in the rat following either oral or inhalation exposure. Toxicol Lett (Amst); 56 (3). 1991. 317-326.
- Creasia DA; Thurman JD Comparative Acute Inhalation Toxicity of a Saline Suspension and an Ethanol Solution of T-2 Mycotoxin in Mice. Govt Reports Announcements & Index (GRA&I), Issue 12, 1993.
- Poon R; Chu I; Bjarnason S; Vincent R; Potvin M; Miller RB; Valli VE Short-Term Inhalation Toxicity of Methanol, Gasoline, and Methanolasoline in the Rat. Toxicology and Industrial Health, Vol. 11, No. 3, pages 343-361, 48 references, 1995.
- Poon R; Chu I; Bjarnason S; Potvin M; Vincent R; Miller RB; Valli VE Inhalation Toxicity Study of Methanol, Toluene, and Methanololuene Mixtures in Rats: Effects of 28-Day Exposure Toxicology and Industrial Health, Vol. 10, No. 3, pages 231-245, 29 references, 1994.
- Gage JC The subacute inhalation toxicity of 109 industrial chemicals. Br J Ind Med; VOL 27, ISS 1, 1970, P1-18.
- 52-Week Inhalation Toxicity Study In Cynomolgus Monkeys EPA/OTS; Doc #878210650 [TSCATS]
- Cholakis JM; Steele DH; Peterson SD; Hagensen JH; Liu GK Chronic Inhalation Toxicity Study of 1,2-Dichloroethane (EDC) in Rats Treated with Disulfiram or Ethanol. Part 1. Toxicology and Pathology. Govt Reports Announcements & Index (GRA&I), Issue 13, 1987.
- Wang PY; Kaneko T; Tsukada H; Sato A Dose and route dependency of metabolism and toxicity of chloroform in ethanol-treated rats. Arch Toxicol; VOL 69, ISS 1, 1994, P18-23.
- Irwin RD; Chou BJ; Mellick PW; Miller RA; Mahler J; Roycroft J Toxicity of Furfuryl Alcohol to F344 Rats and B6C3F1 Mice Exposed by Inhalation Journal of Applied Toxicology, Vol. 17, No. 3, pages 159-169, 19 references, 1997.

- Nakaseko H; Teramoto K; Horiguchi S; Wakitani F; Yamamoto T; Adachi M; Tanaka H; Hozu [Toxicity of isopropyl alcohol (IPA). Part 2. Repeated inhalation exposures in rats] Sangyo Igaku; VOL 33, ISS 3, 1991, P200-1.
- Nelson BK; Brightwell WS; Krieg EF Jr Developmental toxicology of industrial alcohols: a summary of 13 alcohols administered by inhalation to rats. Toxicol Ind Health 1990 May-Jul;6(3-4):373-87.
- Finn DA; Crabbe JC Chronic ethanol differentially alters susceptibility to chemically induced convulsions in withdrawal seizure-prone and -resistant mice. J Pharmacol Exp Ther; VOL 288, ISS 2, 1999, P782-90.
- Moser VC; Balster RL The effects of inhaled toluene, halothane, 1,1,1-trichloroethane, and ethanol on fixed-interval responding in mice. Neurobehav Toxicol Teratol; VOL 8, ISS 5, 1986, P525-31.
- Ghosh TK; Copeland RL Jr; Alex PK; Pradhan SN Behavioral effects of ethanol inhalation in rats. Pharmacol Biochem Behav; VOL 38, ISS 4, 1991, P699-704.
- Pastino GM; Asgharian B; Roberts K; Medinsky MA; Bond JA A comparison of physiologically based pharmacokinetic model predictions and experimental data for inhaled ethanol in male and female B6C3F1 mice, F344 rats, and humans. Toxicol Appl Pharmacol; VOL 145, ISS 1, 1997, P147-57.
- Rogers JM; Mole ML Critical periods of sensitivity to the developmental toxicity of inhaled methanol in the CD-1 mouse. Teratology; VOL 55, ISS 6, 1997, P364-72.
- Nakaseko H; Teramoto K; Horiguchi S; Wakitani F; Yamamoto T; Adachi M; Tanaka H; Hozu S Toxicity Of Isopropyl Alcohol Ipa Part 2. Repeated Inhalation Exposures In Rats Jpn J Ind Health; 33 (3). 1991. 200-201.
- Marietta CA; Jerrells TR; Meagher RC; Karanian JW; Weight FF; Eckardt MJ Effects of long-term ethanol inhalation on the immune and hematopoietic systems of the rat. Alcohol Clin Exp Res; VOL 12, ISS 2, 1988, P211-4.
- Wing DR; Harvey DJ; Hughes J; Dunbar PG; McPherson KA; Paton WDM Effects Of Chronic Ethanol Administration On The Composition Of Membrane Lipids In The Mouse Biochemical Pharmacology, Vol. 31, No. 21, pages 3431-3439, 25 references, 1982.
- Coon RA; Jones RA; Jenkins LJ Jr; Siegel J Animal inhalation studies on ammonia, ethylene glycol, formaldehyde, dimethylamine, and ethanol. Toxicol Appl Pharmacol; VOL 16, ISS 3, 1970, P646-55.

- Keith LD; Crabbe JC Specific and nonspecific effects of ethanol vapor on plasma corticosterone in mice. Alcohol; VOL 9, ISS 6, 1992, P529-33.
- Wroânska-Nofer T; Rosin J; Bartosz G Interaction of ethanol and xylene in their effects on erythrocytes and other haematological parameters in the rat. J Appl Toxicol; VOL 11, ISS 4, 1991, P289-92.
- Massad E; Saldiva PHN; Saldiva CD; Caldeira MPR; Cardoso LMN; de Morais AMS Calheiros DF; da Silva R; Bohm GM Toxicity of Prolonged Exposure to Ethanol and Gasoline Autoengine Exhaust Gases. Environmental Research, Vol. 40, No. 2, pages 479-486, 18 reference, 1986.
- Goldin RD; Wickramasinghe SN Hepatotoxicity of ethanol in mice. Br J Exp Pathol; VOL 68, ISS 6, 1987, P815-24.
- Lieber CS; DeCarli LM; Sorrell MF Experimental methods of ethanol administration.
- Hepatology; VOL 10, ISS 4, 1989, P501-10 (REF: 87) [TOXBIB]
- Liopo AV; Omel'ianchik MS; Chumakova OV [Inhalation effect of low doses of ethanol during pregnancy on development of ratoffspring] Biull Eksp Biol Med 1996 Mar;121(3):265-7.
- Price NH; Yates WG; Allen SD; Waters SW Toxicity Evaluation for Establishing IDLH Values. Govt Reports Announcements & Index (GRA&I), Issue 24, 1987.
- Nelson, BK et al, Neurobehavior, Toxicology and Teratol. 7: 7790783, 1985
- Verschueren, K. Handbook of Environmental Data on Organic Chemicals, 2ed. P 619, 1983

7.29 Freon 114

- Desoille H; Truffert L; Girard-Wallon C; Ripault J; Philbert M Experimental Research of an Eventual Chronic Toxicity of Dichlorotetrafluoroethane. Archives des Maladies Professionnelles de Medecine du Travail et de Securite Sociale, Vol. 34, No. 3, pages 117-125, 14 references, 1973
- Niazi S; Chiou WL Fluorocarbon aerosol propellants. 6. Interspecies differences in solubilities in blood and plasma and their possible implications in toxicity studies J. Pharm. Sci.; VOL 64 ISS Sep 1975, P1538-1541.
- Toxicity of Propellants Aviado DM Progress Research, Vol. 18, pages 365-397, 61 references, 1974.

- Lardear PJ; Graham RC Toxicity Review. Freon 114 Haskell Laboratory, Dupont, 18 pages, 65 references, 1981.
- ACGIH Dichlorotetrafluoroethane Documentation of the threshold limit values and biological exposure indices PG:443-5 YR:1991 IP: VI:6th ed.
- Anonymous 1,2-Dichlor-1,1,2,2-tetrafluoräthan Toxikologisch-arbeitsmedizinische Begrundung von MAK-Werten (Maximale Arbeitsplatzkonzentrationen) PG:3 p YR:1971.
- Belej MA; Aviado DM Cardiopulmonary Toxicity of Propellants for Aerosols Journal of Clinical Pharmacology, Vol. 15, No. 1, Part 2, pages 105-115, 5 references, 1975.
- Aviado DM; Drimal J Five Fluorocarbons For Administration Of Aerosol Bronchodilators Journal of Clinical Pharmacology, Vol. 15, No. 1, pages 116-128, 21 references, 1975.
- Hathaway, GJ, Proctor, NH, Hughes, JP et al. Chemical Hazards of the Workplace, 4th ed. Van Norstand Reihold Company, New York, 1996.

7.30 Heptanal

Hoechst Celanese Corp. Letter concerning enclosed acute inhalation toxicity study and air quality dispersion modeling report with attachments; 11/22/91; EPA Doc. No. 86-920000443; Fiche No. OTS05344991.

7.31 Indan

- Serve MP Study of the nephrotoxicity and metabolism of tetralin and indan in fischer 344 rats. Govt reports announcements and index (GRA&I) issue 19, 1989
- Cozzi NV et al. Indan analogs of fenfluramine and norfenfluramine have reduced neurotoxic potential. Pharmacol Biochem Behav vol 59 ISS 3, 1998 p 709-15.
- Serve MP Study of the nephrotoxicity and metabolism of tetralin and indan in fischer 344 rats. Govt reports announcements and index (GRA&I) issue 16, 1988.

7.32 Indene

- Cameron GR; Doniger CR The Toxicity of Indene Journal of Pathology and Bacteriology, Vol. 49, pages 529-533, 2 references, 1939.
- Dyshinevich NE Hygienic Evaluation of Indene Coumarone Floor Tiles Gigiena Primeneniia, Toksikologiia Pestitsidov i Klinika Otravlenii, Vol. 9, pages 366-370, 1971.

- Bevan C; Snellings WM; Dodd DE; Egan GF Subchronic toxicity study of dicyclopentadiene vapor in rats. Toxicol Ind Health; VOL 8, ISS 6, 1992, P353-67.
- ACGIH: documentation of the threshold limit values and biological exposure indices, vol 1, 6th ed, Am Conf of Govt Ind Hyg, Inc, Cincinnati, OH, 1991.

7.33 Isobutane

- Friedman SA; Cammarato M; Aviado DM Toxicity of aerosol propellants on the respiratory and circulatory systems. Respiratory and bronchopulmonary effects in the rat Toxicology; VOL 1 ISS 4 1973, P345-355.
- Aviado DM Toxicity of aerosol propellants in the respiratory and circulatory system. 10. Proposed. classification Toxicology; VOL 3 ISS 3 1975, P321-332.
- Burkhart KK; Britt A; Petrini G; O'Donnell S; Donovan JW Pulmonary Toxicity following Exposure to an Aerosolized Leather Protector Journal of Toxicology, Clinical Toxicology, Vol. 34, No. 1, pages 21-24, 16 references, 1996.
- Aviado DM Toxicity of Propellants Progress Research, Vol. 18, pages 365-397, 61 references, 1974.
- ACGIH Butane Documentation of the threshold limit values and biological exposure indicesPG:160-1 YR:1991 IP: VI:6th ed.
- Galvin JB; Bond G Isobutane. CAS# 75-28-5. J Toxicol Environ Health; VOL 58, ISS 1-2, 1999, P3-22.
- Belej MA; Aviado DM Cardiopulmonary Toxicity of Propellants for Aerosols Journal of Clinical Pharmacology, Vol. 15, No. 1, Part 2, pages 105-115, 5 references, 1975.
- Snyder (ed.) Ethyl Browning, Toxicity and Metabolism of Industrial Solvents 2nd ed, vol 1, hydrocarbons, Elsevier 1987 p 273-278.
- Stewart RD et al. Acute and repetitive human exposure to isobutane. Scand J Work Environ Health 1977; 3:234-243.
- Clayton and Clayton (1981-1982) Patty's Industrial Hygiene and Toxicology vol 2A, 2B, 2C toxicology 3rd ed., John Wiley and Sons, p3183.

7.34 Isobutene/1-Butene

NIH publication no 99-3977. (1998) Toxicology and carcinogenicity studies of isobutene in F344/N rats and B6C3F1 mice. USDHHS, NTP, NIEHS RTP NC.

- NTP working group Toxicology and carcinogenesis studies of Isobutene in F344/N rats and B6C3F1 mice (inhalation studies) National Toxicology Program Technical Report Series PG:229p YR:1999 IP: VI:487.
- Henderson RF; Sabourin PJ; Bechtold WE; Steinberg B; Chang I-Y Disposition of Inhaled Isobutene in F344 Rats Toxicology and Applied Pharmacology, Vol. 123, No. 1, pages 50-61, 29 references, 1993.
- NTP Technical Report on the Toxicology and Carcinogenesis Studies of Isobutene (CAS No. 115-11-7) in F344 Rats and B6C3F1 Mice (Inhalation Studies). Govt Reports Announcements & Index (GRA&I), Issue 17, 1999.
- Cornet M; Rogiers V Metabolism and toxicity of 2-methylpropene (isobutene)--a review. Crit Rev Toxicol; VOL 27, ISS 3, 1997, P223-32.
- NTP working group NTP Technical Report on Toxicity studies of t-Butyl Alcohol. Administered byinhalation to F344/n rats and B6C3F1 mice. National Toxicology Program Toxicity Report Series PG:56 p YR:1997 IP: VI.
- Anonymous 2-Methylpropen Toxikologische Bewertung. Heidelberg, Berufsgenossenschaft der chemischen Industrie PG:26 p YR:1997 IP: VI:104.

7.35 Isoheptane

Hutcheson MS et al. Beyond TPH: health-based evaluation of petroleum hydrocarbon exposures. Regulatory Toxicology and Pharmacology 24 1996 p 85-101.

7.36 Isopentane

- Chiba S; Oshida S [Metabolism and toxicity of n-pentane and isopentane] Nippon Hoigaku Zasshi; VOL 45, ISS 2, 1991, P128-37.
- Galvin JB; Marashi F 2-Methylbutane (isopentane). CAS# 78-78-4. J Toxicol Environ Health; VOL 58, ISS 1-2, 1999, P23-33.
- ACGIH. Pentane (all isomers); Isopentane; Neopentane Documentation of the threshold limit values and biological exposure indices PG:11 p YR:1998 IP:Suppl. VI:6th Ed.
- Clayton and Clayton (1981-1982) Patty's Industrial Hygiene and Toxicology vol 2A, 2B, 2C toxicology 3rd ed., John Wiley and Sons, p3185.
- Author unknown, J of Am Col Tox 1(4):127-42 (1982).

7.37 Isoprene

- Melnick RL; Sills RC; Roycroft JH; Chou BJ; Ragan HA; Miller RA Inhalation toxicity and carcinogenicity of isoprene in rats and mice: comparisons with 1,3-butadiene. Toxicology 1996 Oct 28;113(1-3):247-52 [EMIC]
- Anonymous Isopren Toxikologische Bewertung. Heidelberg, Berufsgenossenschaft der chemischen Industrie PG:76 p YR:2000 IP: VI:105.
- NTP Toxicity studies of isoprene. Administered by inhalation to F344/N rats and B6C3F1 mice National Toxicology Program PG:100 p YR:1995 IP: VI:TOX 31.
- EPA Two-Year Chronic Inhalation Toxicity And Carcinogenicity Study Of Isoprene In Fischer 344 Rats With Cover Letter Dated 10/22/1997 Epa/Ots; Doc #Fyi-Ots-1197-1315.
- IARC, Isoprene IARC Monographs on the evaluation of the carcinogenic risk of chemicals to humans PG:215-32 YR:1994 IP: VI:60.
- Mast TJ; Rommereim RL; Weigel RJ; Stoney KH; Schwetz BA; Morrissey RE Inhalation developmental toxicity of isoprene in mice and rats. Toxicologist 1990 Feb;10(1):42
- Melnick RL NTP Technical Report on Toxicity Studies of Isoprene (CAS No. 78-79-5)
- Administered by Inhalation to F344 Rats and B6C3F1 Mice. Govt Reports Announcements & Index (GRA&I), Issue 17, 1995.
- Melnick RL; Roycroft JH; Chou BJ; Ragan HA; Miller RA Inhalation toxicology of isoprene in F344 rats and B6C3F1 mice following two-week exposures. Environ Health Perspect; VOL 86, 1990, P93-8.
- Placke ME; Griffis L; Bird M; Bus J; Persing RL; Cox LA Jr Chronic inhalation oncogenicity study of isoprene in B6C3F1 mice. Toxicology; VOL 113, ISS 1-3, 1996, P253-62.
- Cox LA Jr; Bird MG; Griffis L Isoprene cancer risk and the time pattern of dose administration. Toxicology; VOL 113, ISS 1-3, 1996, P263-72.
- Melnick RL; Elwell MR; Roycroft JH; Chou BJ; Ragan Ha; Miller Ra Toxicity of inhaled chloroprene (2-chloro-1,3-butadiene) in F344 rats and B6C3F1 mice. Toxicology; 108 (1-2). 1996. 79-91.
- Clayton and Clayton (1994) Patty's Industrial Hygiene and Toxicology vol 3 toxicology 4rd ed., John Wiley and Sons.
- Hong, HL, Devereux, T.R, et al Carcinogenesis, Vol 18, p783, 1997.

Taalman, RD, Isoprene, background and issues. Toxicology, Vol 113, p242, 1996.

Tice, RR et al, Mutagenesis 3 (2), 141, 1988.

7.38 m-Diethylbenzene

Patty's Ind Hyg and Tox vol II toxicology 2nd ed NY Interscience Publishers 1963, p1232.

7.39 Methylcyclopentane

Coleman EC et al. J Agric Food Chem 29:42-48 (1981)

Shahidi F et al. CRC Crit Rev Food Sci Nature 24:141-243 (1986).

Rappaport SM et al. Appl Ind Hyg 2:148-54 (1987).

- Brugnone F et al. Solvent exposure in a shoe upper factory: II. Methylcyclopentane, 2-methylpentane and 3-methylpentane concentration in alveolar and in environmental air and in blood. Int Arch Occup Environ Health 42(3-4). 1979. 355-364.
- Bartolucci GB et al. Occupational exposure to solvents: Field comparison of active and passive samplers and bi8ological monitoring of exposed workers. The Annuals of Occupational Hygiene, vol 30, no 3 pages 295-306, 39 references 1986.

7.40 n-Butane

- ACGIH: Documentation of the threshold limit values, 5th ed. Am Conf of Govt Ind Hyg. Inc. Cincinnati, OH 1986, pg 70
- Hau Km; Connell Dw; Richardson Bj Mechanism of acute inhalation toxicity of alkanes and aliphatic alcohols. Environmental Toxicology And Pharmacology; 7 (3). 1999. 159-167.
- Berzins T Health effects of selected chemicals 3. Butane NORD PG:10-26 YR:1995 IP: VI:28.
- Siegel E; Wason S Sudden death caused by inhalation of butane and propane N. Engl. J. Med.; VOL 323 ISS Dec 6 1990, P1638.

7.41 n-Decane

Snyder (ed.) Ethyl Browning, Toxicity and Metabolism of Industrial Solvents 2nd ed, vol 1, hydrocarbons, Elsevier 1987 p 273-278.

Verschueren K Handbook of Environmental Data of Organic Chemicals 2nd ed. NY Van Nostrand Reinhold Co (1983) p. 447.

Rappaport SM et al. Appl Ind Hyg 2:148-54 (1987).

Van der Wal JF et al Ann Occup Hyg 28:39-47 (1984)

7.42 Neohexane

- SDU Uitgeverij Plantijnstraat, Afdeling Verkoop Publikaties Arbeidsinspectie, Postbus Health-based recommended occupational exposure limits for 2-methylpentane, 3-methylpentane, 2,2-dimethylbutane, 2,3-dimethylbutane (hexane isomers) 20014, 2500 EA Den Haag, Netherlands, 1993. 21p.
- Galvin JB; Panson R 2,2-Dimethylbutane (neohexane). CAS 75-83-2. J Toxicol Environ Health; VOL 58, ISS 1-2, 1999, P103-10.
- Snyder (ed.) Ethyl Browning, Toxicity and Metabolism of Industrial Solvents 2nd ed, vol 1, hydrocarbons, Elsevier 1987 p 273-278.

7.43 Neopentane

Galvin JB; Panson R Neopentane (2,2-dimethylpropane). CAS# 463-82-1. J Toxicol Environ Health; VOL 58, ISS 1-2, 1999, P75-80.

7.44 n-Nonane

Carpenter et al., Toxicology and Applied Pharmacology, 44 (1), p53-62, 1978.

- Khan S; Pandya KP Hepatotoxicity in albino rats exposed to n-octane and n-nonane. J Appl Toxicol; VOL 5, ISS 2, 1985, P64-8.
- Khan S; Pandya Kp Biochemical studies on the toxicity on n-octane and n-nonane. Environ Res; 22 (2). 1980. 271-276.
- Eide I; Zahlsen K Inhalation experiments with mixtures of hydrocarbons. Experimental design, statistics and interpretation of kinetics and possible interactions. Arch Toxicol; VOL 70, ISS 7, 1996, P397-404.

7.45 n-Octane

Dahl AR The fate of inhaled octane and the nephrotoxicant, isooctane, in rats. Toxicol Appl Pharmacol; VOL 100, ISS 2, 1989, P334-41.

- Olson CT; Yu KO; Hobson DW; Serve MP The metabolism of n-octane in Fischer 344 rats. Toxicol Lett; VOL 31, ISS 2, 1986, P147-150.
- ACGIH Octane Committee on Threshold Limit Values (Unpublished), 1 page, 5 references, 1974.
- Khan S; Pandya Kp Biochemical studies on the toxicity on n-octane and n-nonane. ENVIRON RES; 22 (2). 1980. 271-276.
- NIOSH Criteria for a Recommended Standard... Occupational Exposure to Alkanes (C5-C8) Cincinnati, Ohio, HEW Publication No. (NIOSH) 77-151, 137 pages, 119 references, 1977.

7.46 n-Undecane

Jay K, et al. Chemosphere 30:1249-60 (1995)

Zielinska B et al. Sci Tot Environ 146/147:281-88 (1994)

- Kjaergaard S et al. Nordic expert group for documentation of occupational exposure limits 75. Ndecane and n-undecane. Arbetarskyddsstyrelsen, publikationservice, 171 84 solna, sweden, 1987 32p, 52 refs [in Danish]
- Kristiansen U et al. Activation of the sensory irritant recepotor by C7-C11 n-alkanes. Archives of Toxicology, vol 61, no6 pages 419-425, 10 ref. 1988.
- Kjaergaard S et al n-Decane and n-Undecane. Arbete och Halsa, vol. 40, criteria documents for the Nordic Expert Group. G Heimburger and P. Lundberg Editors. Pgs 45-73, 49 ref. 1987.

7.47 p-Diethylbenzene

Patty's Ind Hyg and Tox vol II toxicology 2nd ed NY Interscience Publishers 1963, p 1232.

Gagnaire F; Becker MN; Marignac B; Bonnet P; De Ceaurriz J. Diethylbenzene inhalation-induced electrophysiological deficits in peripheral nerves and changes in brainstem auditory evoked potentials in rats. J Appl Toxicol; VOL 12, ISS 5, 1992, P335-42

7.48 p-Isopropyltoluene

Lewis RJ Sax's Dangerous Properties of Industrial Materials, 9th ed. NY, 1998.

Cocheo V et al. Amer Ind Hyg Assoc J 44:521-527 (1983).

- Browning, E Toxicity and Metabolism of Industrial Solvents NY American Elsevier 1965p 106
- Lam HR; Ladefoged O; Ostergaard G; Lund SP; Simonsen Four weeks' inhalation exposure of rats to p-cymene affects regional and synaptosomal neurochemistry. Pharmacol Toxicol; VOL 79, ISS 5, 1996, P225-30 [TOXBIB]
- BIBRA Toxicity Profile of para-isopropyltoluene. Govt Reports Announcements & Index (GRA&I), Issue 19, 1996.

7.49 Propane

Stewart RD et al. US NTIS PB Rep ISS PB-279205: 1-95 (1977).

Stewart RD et al. Environ Health Perspect 26:275-285 (1978).

- Snyder R (ed) Ethyl Browning's Toxicity and Metabolism of Industrial Solvents 2nd ed. Volume 1: Hydrocarbons. Elsevier, 1987, p 263.
- Clayton and Clayton (1994) Patty's Industrial Hygiene and Toxicology vol 2B, toxicology 4th ed., John Wiley and Sons, NY,NY.

7.50 p-Xylene

- Lewis RJ Sax's Dangerous Properties of Industrial Materials, 9th ed. NY, 1998.
- Harper, C. Chronic inhalation exposure to rats did not cause leukopenia. (Proc. Int. Conference) 1977, 302-11. See HSDB.
- Ungvary, G. Toxicol. 18:61-74, 1980.
- Kennedy G L Jr; Graepel GJ Acute toxicity in the rat following either oral or inhalation exposure. Toxicol Lett (AMST); 56 (3). 1991. 317-326.
- Chassevant A; Garnier M The Toxicity of Benzene and Some Aromatic Hydrocarbon Homologs Comptes Rendu Societe de Biologie, Vol. 55, pages 1255-1257, 1903.
- Ungvåary G; Donâath T Effect of benzene and its methyl-derivatives (toluene, para-xylene) on postganglionicnoradrenergic nerves.
- Z Mikrosk Anat Forsch; VOL 98, ISS 5, 1984, P755-63.
- Sullivan FM; Watkins WJ; van der Venne MT; eds The toxicology of chemicals 2.

 Reproductive toxicity: Volume 1 Summary reviews of the scientific evidence Office

- for Official Publications of the European Communities, 2985 Luxembourg, Grand Duchy of Luxembourg, 1993. ix, 431p.
- Sociale Zaken en Werkgelegenheid, Directoraat-Generaal van de Arbeid), Postbus Health-based recommended occupational exposure limit for xylene Department of Social Affairs and Employment, Directorate-General of Labour (Ministerie van 90804, 2509 LV Den Haag, Netherlands, 1991.
- Browning E Xylene Toxicity and Metabolism of Industrial Solvents, Elsevier Publishing Company, New York, pages 77-89, 124-129, 139 references, 1965.
- Bell GM; Shillaker RO; Padgham MDJ; Standring P Xylenes
- HMSO Books, P.O. Box 276, London SW8 5DT, United Kingdom, 1992. 166p.
- Selgrade MK; Daniels MJ; Jaskot RH; Robinson BL; Allis JW Enhanced mortality and liver damage in virus-infected mice exposed to p-xylene. J Toxicol Environ Health; Vol 40, ISS 1, 1993, P129-44.
- Silverman DM; Schatz RA Pulmonary Microsomal Alterations following Short-Term Low Level Inhalation of p-Xylene in Rats Toxicology, Vol. 65, No. 3, pages 271-281, 38 references, 1991.
- IARC Xylene. ANONYMOUS Monogr Eval Carcinog Risks Hum 1989;47:125-56
- Ungvâary G; Tâatrai E; Hudâak A; Barcza G; Lèorincz M Investigation of the embryotoxic effect of p-xylene Egâeszsâegtudomany 1979, Vol.23, No.2, p.152-158.
- Padilla SS; Lyerly DP Effects of p-xylene inhalation on axonal transport in the rat retinal ganglion cells. Toxicol Appl Pharmacol; VOL 101, ISS 3, 1989, P390-8 [TOXBIB]
- Simmons JE; Allis JW; Grose EC; Seely JC; Robinson BL; Berman E Assessment of the hepatotoxicity of acute and short-term exposure to inhaled p-xylene in F-344 rats. J Toxicol Environ Health; VOL 32, ISS 3, 1991, P295-306 [TOXBIB]
- ASDR Toxicological profile for xylenes (Update) U.S. Department of Health and Human Services, Public Health Service, Agency for ToxicSubstances and Disease Registry, Division of Toxicology, Toxicology Information Branch, 1600 Clifton Road NE, E-29, Atlanta, GA 30333, USA, Aug. 1995. 270p.

7.51 m-Xylene

WinekCL Drug and Chemical Blood level data 1985 Pittsburgh, PA Allied Fischer Scientific

- Hayes WJ jr. et al. Handbook of pesticide toxicology vol 2 Classes of Pesticides NY, NY Acad Press Inc. 1991 p 644.
- Kawai T et al. Int Arch Occup Environ Health 63(1):69-76 (1991) NCI Monograph on human exposure to chemicals in the workplace: xylene p 4-2 July 1985.
- Kim SK; Kim YC Effect of a single administration of benzene, toluene or m-xylene on carboxyhaemoglobin elevation and metabolism of dichloromethane in rats. J Appl Toxicol; VOL 16, ISS 5, 1996, P437-44.
- Kobayashi H; Hobara T; Sakai T [Effects of inhalation of several organic solvents on left ventricular dp/dt] Sangyo Igaku; VOL 31, ISS 3, 1989, P136-41.
- Savolainen K; Riihimèaki V; Laine A; Kekoni J Short-term exposure of human subjects to m-xylene and 1,1,1-trichloroethane. Arch Toxicol Suppl; VOL 5, 1982.
- Sociale Zaken en Werkgelegenheid, Directoraat-Generaal van de Arbeid), Postbus Health-based recommended occupational exposure limit for xylene Department of Social Affairs and Employment, Directorate-General of Labour (Ministerie va 90804, 2509 LV Den Haag, Netherlands, 1991. iii, 89p.
- Korsak Z; Wiâsniewska-Knypl J; Swiercz R Toxic effects of subchronic combined exposure to n-butyl alcohol and m-xylene in rats. Int J Occup Med Environ Health; VOL 7, ISS 2, 1994, P155-66 [TOXBIB]
- Laine A; Savolainen K; Riihimèaki V; Matikainen E; Salmi T; Juntunen J Acute effects of m-xylene inhalation on body sway, reaction times, and sleep in man. Int Arch Occup Environ Health; VOL 65, ISS 3, 1993, P179-88.
- Hume As; Ho Ik Toxicity Of Solvents Basic Environmental Toxicology. Xi+627p. Crc Press, Inc.: Boca Raton, Florida, Usa London, England, Uk. Isbn 0-8493-8851-1.; 0 (0). 1994. 157-18
- ATSDR, Toxicological profile for total xylenes U.S. Department of Health and Human Services, Public Health Service, Centers for DiseaseControl, Atlanta, GA 30333, USA, Dec. 1990. xii, 191p.
- Elovaara E; Zitting A; Nickels J; Aitio A m-Xylene inhalation destroys cytochrome P-450 in rat lung at low exposure. Arch Toxicol; VOL 61, ISS 1, 1987, P21-6 [TOXBIB]
- Speck B; Moeschlin S The Effect of Toluene, Xylenes, Chloramphenicol, and Thiouracil on Bone Marrow Schweizerische Medizinische Wochenschrift, Vol. 98, pages 1684-1686, 13 references 1968

7.52 t-2-Butene

Conkle JP et al. Arch Environ Health 30:290-295 (1975).

Rappaport SM et al. Appl Ind Hyg 2:148-154 (1987).

7.53 t-2-Hexene

- Phillips et al. Variation in volatile organic compounds in the breath of normal humans. Journal of Chromatography B; 729 (1-2). 1999. 75-88
- Halder et al Hydrocarbon nephropathy in male rats: Identification of the nephrotoxic components of unleaded gasoline. Toxicol Ind Health 1 (3). 1985 (Recd 1986). 67-88.
- Ciccioli et al. Use of carbon adsorption traps combined with high resolution gas chromatographymass spectrometry fo rthe analysis of polar and non-polar C4-C14 hydrocarbons involved in photochemical smog formation. HRC (J High Resolut Chromatogr); 15 (2)/ 1992. 75-84.
- Ramnas et al. Assessment by gas chromatography-mass spectrometry of hexenes emitted to air from petrol. J Chromatogr; 638(1). 1993. 65-69.
- Fraser et al. Air quality model evaluation data for organics: 4 C2-C36 non-aromatic hydrocarbons. Environmental Science and Technology; 31(8). 1997. 2356-2367.

7.54 1 Naphthylamine

- IARC monograph Vol 4 pg 93, 1974
- Clayton and Clayton (1994) Patty's Industrial Hygiene and Toxicology vol 2B, toxicology 4th ed., John Wiley and Sons, NY,NY.
- Lewis RJ Sax's Dangerous Properties of Industrial Materials, 9th ed. NY, 1998.
- ITI: Toxic and Hazardous Industrial Chemicals Safety Manual. The International Technical Information Institute, Tokyo, Japan, 1988.
- Purchase IF; Kalinowski AE; Ishmael J; Wilson J; Gore CW; Chart IS Lifetime carcinogenicity study of 1- and 2-naphthylamine in dogs. Br J Cancer; VOL 44, ISS 6, 1981, P892-901.
- Radomski JL; Deichmann WB; Altman NH; Radomski T Failure of pure 1-naphthylamine to induce bladder tumors in dogs. Cancer Res; VOL 40, ISS 10, 1980, P3537-9.

- Iogannsen MG; Pliss GB; Vasil'eva IA [Effect of carcinogenic 2-naphthylamine on organ cultures of the urinary bladder of the rat] Vopr Onkol; VOL 29, ISS 4, 1983, P74-
- ATSDR alpha-Naphthylamine, potential human carcinogen US Department of Health and Human Services, Public Health Service, Centers for Disease Control, National Institute for Occupational Safety and Health, Division of Standards Development and Technology Transfer, 4676 Columbia Parkway, Cincinnati OH 45226, USA, 1988. 6p.

7.55 2-Methylnaphthylene

- Griffin KA; Johnson CB; Breger RK; Franklin RB Pulmonary Toxicity Of 2-Methylnaphthalene: Lack Of A Relationship Between Toxicity, Dihydrodiol Formation And Irreversible Binding To Cellular Macromolecules In DBA/2J Mice Toxicology, Vol. 26, No. 3, pages 213-230, 23 references, 1983.
- Murata Y; Denda A; Maruyama H; Nakae D; Tsutsumi M; Tsujiuchi T; Konishi Y Chronic Toxicity and Carcinogenicity Studies of 2-Methylnaphthalene in B6C3F1 Mice. Fundamental and Applied Toxicology, Vol. 36, No. 1, pages 90-93, 16 references, 1997.
- Rasmussen RE; Do DH; Kim TS; Dearden LC Comparative cytotoxicity of naphthalene and its monomethyl- and mononitro-derivatives in the mouse lung. J Appl Toxicol; VOL 6, ISS 1, 1986, P13-20.
- Honda T; Kiyozumi M; Kojima S Alkylnaphthalene: XI. Pulmonary toxicity of naphthalene, 2-methylnaphthalene, and isopropylnaphthalenes in mice. Chem Pharm Bull (Tokyo); 38 (11). 1990. 3130-3135. [BIOSIS]
- Korsak Z; Majcherek W; Rydzyânski K Toxic effects of acute inhalation exposure to 1-methylnaphthalene and 2-methylnaphthalene in experimental animals. Int J Occup Med Environ Health; VOL 11, ISS 4, 1998, P335-42.
- Dinsdale D; Verschoyle RD Pulmonary Toxicity of Naphthalene Derivatives in the Rat. Archives of Toxicology, Supplement 11, pages 288-291, 8 references, 1987.
- ATSDR, Toxicological profile for naphthalene:U.S. Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry, Division of Toxicology, Toxicology Information Branch, 1600 Clifton Road NE, E-29, Atlanta, GA 30333, USA, Aug. 1995. 200p.
- Griffin Ka; Johnson Cb; Breger Rk; Franklin Rb Pulmonary toxicity, hepatic and extrahepatic metabolism of 2-methylnaphthalene in mice. Toxicol Appl Pharmacol; 61 (2). 1981. 185-196.

ANALYTE	MIN 24 HOUR (MG/M3) /PPM	AVG 24 HOUR (MG/M3)	MAX 24 HOUR (MG/M3)/ PPM	ATSDR MRL* (PPM)	ACUTE HEALTH EFFECT/ REVERSIBLE	HEALTH EFFECT LEVEL	EFFECTS	SOURCE
1-Butanol (n-butyl alcohol)	0.001	0.02	0.27/ 0.0888		NO	200 PPM	developed increasing corneal inflammation associated with burning sensation, blurring of vision, lacrimation and photophobia, beginning at middle of the week & growing more severe toward the end of the week	Patty's Industrial Hygiene and Toxicology, 1981- 1982., p. 4576
						Above 200 PPM	slightly reduced erythrocyte count	International Labour Office. Encyclopedia of Occupational Health and Safety, 1983., p. 110
1-Decene	0.0002	0.00054	0.009		NO		No information found.	
1-Heptene	0.0002	0.00041	0.003		NO	70ug/m3 (20 ppb)		Texas Natural Resource Conservation Commission Short-term Effects Screening Level, On-line envirofacts chemical reference: Scorecard

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ANALYTE	MIN 24 HOUR (MG/M3) /PPM	AVG 24 HOUR (MG/M3)	MAX 24 HOUR (MG/M3)/ PPM	ATSDR MRL* (PPM)	ACUTE HEALTH EFFECT/ REVERSIBLE	HEALTH EFFECT LEVEL	EFFECTS	SOURCE
1-Hexene	0.0003	0.0005	0.002		NO	1000 PPM	depression, mucous membrane	Patty's Industrial Hygiene and Toxicology, Volume 2B, 1994, p. 1249
1-Nonene	0.00008	0.00025	0.001		NO	0.026 mg/m3		Texas Natural Resource Conservation Commission Short-term Effects Screening Level, Envirofacts
1-Octene	0.00006	0.0038	0.003		NO	0.02 mg/m3		Texas Natural Resource Conservation Commission Short-term Effects Screening Level, Envirofacts ANON
						10,400 and 18,000 mg/m3	Eye irritation in humans	Department of Environmental and Occupational Medicine, University of Aarhus, Denmmark (1999)
1-Pentene	0.0002	0.00061	0.002		NO		No information found.	

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						250 ml	Fatal dose by ingestion	Handbook of Poisoning. 12th ed., 1987., p. 178
							Mild irritation of eyes, nose, and throat	Patty's Industrial Hygiene and Toxicology, 1981- 1982., p. 4561
1-Propanol	0.003	0.007	0.02		NO		Persons with pre-existing skin disorders may be more susceptible to the effects of this agent. In persons with impaired pulmonary function, especially those with obstructive airway diseases, the breathing of propyl alcohol might cause exacerbation of symptoms due to its irritant properties.	National Institute of Occupational Safety and Health/Occupational Safety and Health Administration - Occupational Health Guidelines for Chemical Hazards. DHHS(NIOSH) Publication No. 81-123, Jan. 1981.
1-Undecene	0.0002	0.00036	0.005		NO		Not classified as a recognized or suspect hazard.	Envirofacts accessed through EPA's website (www.epa.gov)

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			0.0007/			650-1900 PPM		Environmental Protection Agency; Health Assessment Document: 1,1,1-Trichloroethane p.5-49 to 5-50, 1982, EPA-600/8-82-003
1,1-Dichloroethylene	0.00007	0.00012	0.0007/ 0.0002	Inh. Int = 0.02 (Hepatic)		Approximatel y 4,000 PPM	that may progress to unconsciousness if exposure is	Dow Chemical Co; Vinylidene Chloride Monomer: Safe Handling Guide (1980) as cited in USEPA; Phase I Document: Vinylidene Chloride p.76 (1981) EPA No. 68-01-6030
1,1,2-Trichloroethane	0.00005	0.00021	0.001/ 0.0002	1,1,1- Trichloroethane Inh. Acute = 2 (Neurol) Int = 0.7 (Neurol)	NO			
1,1,2,2-Tetrachloroethane	0.0002	0.00042	0.004/ 0.0006	Inh. Int. =0.4 (Hepatic)	NO	2.9 PPM @ 20 minutes	_	Agency for Toxic Substances and Disease Registry Tox Profile, 1996, pg. 16

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ANALYTE	MIN 24 HOUR (MG/M3) /PPM	AVG 24 HOUR (MG/M3)	MAX 24 HOUR (MG/M3)/ PPM	ATSDR MRL* (PPM)	ACUTE HEALTH EFFECT/ REVERSIBLE	HEALTH EFFECT LEVEL	EFFECTS	SOURCE
1,1,2,2-Tetrachloroethane	0.0002	0.00042		Inh. Int. =0.4	NO	13 PPM @ 10-30 min.	Mucosal irritation	Agency for Toxic Substances and Disease Registry Tox Profile, 1996, pg. 15
1,1,2,2-1 cu acmoroculane	0.0002	0.00042	0.0006	(Hepatic)		116 ppm & higher @ 10- 30 min.	Dizzy	Agency for Toxic Substances and Disease Registry Tox Profile, 1996, pg. 19
1,2-Dibromoethane	0.00008	0.00017	0.002		NO		No information found.	
1.2-Dichlorobenzene						1-44 PPM (avg 15 PPM)	No evidence in workers of organ injury or of any hematological effects	International Agency for Research on Cancer. Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man., V7 239, 1974
(this is the concentration for 1,3)	0.00001	0.00023	0.002/ 0.0003		NO	100 PPM	Eye and nose irritation	Patty's Industrial Hygiene and Toxicology, Volume 2B, 1994, p. 1458

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ANALYTE	MIN 24 HOUR (MG/M3) /PPM		MAX 24 HOUR (MG/M3)/ PPM	ATSDR MRL* (PPM)	ACUTE HEALTH EFFECT/ REVERSIBLE	HEALTH EFFECT LEVEL	EFFECTS	SOURCE
1,2-Dichloroethane (ethylene dichloride)	0.0002	0.00018	0.001/ 0.0003	Inh. Chr. = 0.6 (Hepatic)	NO	4-60 PPM	Symptoms including conjunctival congestion and burning sensation, weakness, bronchial and pharyngeal symptoms, metallic taste in mouth, headache, dermatographism, nausea, liver pain, tachycardia, and dyspnea.	Brzozowski J; Med Pracy 5: 89-98,1954
1,2-Dichloropropane (propylene dichloride)	0.0001	0.00012	0.001/ 0.0002	Inh. Acute = 0.05 (Resp) Inh. Int. = 0.007 (Resp)	NO			
1,2,3-Trimethylbenzene	0.0001	0.00045	0.002		NO	10-60 PPM		American Conference of Governmental Industrial Hygienists, Inc, Documentation of the Threshold Limit Value & Biological Exposure Indices., 1998.
1,2,4-Trimethylbenzene	0.0004	0.002	0.01		NO	10-60 PPM		American Conference of Governmental Industrial Hygienists, Inc, Documentation of the Threshold Limit Value & Biological Exposure Indices., 1998.

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ANALYTE	MIN 24 HOUR (MG/M3) /PPM	AVG 24 HOUR (MG/M3)	MAX 24 HOUR (MG/M3)/ PPM	ATSDR MRL* (PPM)	ACUTE HEALTH EFFECT/ REVERSIBLE	HEALTH EFFECT LEVEL	EFFECTS	SOURCE
1,3,5-Trimethylbenzene	0.0001	0.00059	0.003		NO		No information found.	
						2,000 - 18,000 PPM		Medical Toxicology - Diagnosis and Treatment of Human Poisoning., 1988., p. 968
						2,000-4,000 PPM	•	Patty's Industrial Hygiene and Toxicology, Volume 2B, 1994, p. 1251
						8,000 PPM @ 8 hours	respiratory tract	Patty's Industrial Hygiene and Toxicology, Volume 2B, 1994, p. 1251
1,3-Butadiene	0.0002	0.0006	0.006/ 0.0027		13()	8,000 PPM @ 8 hr	upper respiratory tract	Handbook of Environmental Data of Organic Chemicals. 2nd ed., 1983., p. 297
					NO 8,000 PPM @ Slight irritation of the eyes and Hupper respiratory tract 10,000 PPM @ Irritation of the human respiratory He system 2 10,000 PPM	Handbook of Environmental Data of Organic Chemicals. 2nd ed., 1983., p. 297		
						Above 18,000 PPM	fatigue	Medical Toxicology - Diagnosis and Treatment of Human Poisoning., 1988., p. 968
1,3-Dichlorobenzene	0.000005	0.00023	0.002/ 0.0003		NO		No information found.	

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ANALYTE	MIN 24 HOUR (MG/M3) /PPM	AVG 24 HOUR (MG/M3)	MAX 24 HOUR (MG/M3)/ PPM	ATSDR MRL* (PPM)	ACUTE HEALTH EFFECT/ REVERSIBLE	HEALTH EFFECT LEVEL	EFFECTS	SOURCE
1,4-Dichlorobenzene	0.0002	0.0014	0.005/ 0.0008	Inh. Acute = 0.8 Devlop) Int. = 0.2 (Hepatic) Chr = 0.1 (Hepatic)	NO			
					3 mg/m3 for 1 Respirate hour healthy h		Respiratory and eye irritation to healthy human volunteers	Inhalation Reference Exposure Level- California EPA, Office of Environmental Health Hazard Assessment
						200 PPM after 15 min	Irritating to human mucous membranes and conjuntivae	Clinical Toxicology of Commercial Products. 5th ed., 1984. II-185
1,4-Dioxane	0.0002	0.00095	0.01/ 0.028		NO	300 PPM	Irritation of eyes, nose & throat	Patty's Industrial Hygiene and Toxicology: Volume 2A, 2B, 2C, 1981-1982., p. 3956
						Short exposure to 500 PPM	Nausea & vomiting	Encyclopedia of Occupational Health and Safety., 1983., p. 1171
						1600 PPM @ 10 min	Immediate slight burning of eyes accompanied by lacrimation & slight irritation of nose & throat	Patty's Industrial Hygiene and Toxicology: Volume 2A, 2B, 2C, 1981-1982., p. 3956

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ANALYTE	MIN 24 HOUR (MG/M3) /PPM	AVG 24 HOUR (MG/M3)	MAX 24 HOUR (MG/M3)/ PPM	ATSDR MRL* (PPM)	ACUTE HEALTH EFFECT/ REVERSIBLE	HEALTH EFFECT LEVEL	EFFECTS	SOURCE
1,4-Dioxane	0.0002	0.00095	0.01/ 0.028		NO	Momentary Exposure to 33,000 & 100,000 PPM	Intolerable irritation of the eyes, nose, & throat	Patty's Industrial Hygiene and Toxicology: Volume 2A, 2B, 2C, 1981-1982., p. 4730
2-Ethyl-1-Butene	0.00005	0.00019	0.0003		NO		No information found.	
2-Methyl-1-Pentene	0.00008	0.00016	0.0006		NO		No information found.	
2-Methyl-2-Pentene	0.00005	0.00021	0.0007		NO		No information found.	
2-Methylheptane	0.0001	0.00044	0.01		NO		No information found.	
2-Nitrophenol	0.00003	0.00004	0.0002		NO		No information found.	
2-Propanol	0.003	0.015	1.96/ 0.7967		NO	7.586 mg/m3 (3.195 PPM)		Texas Natural Resource Conservation Commission Short-term Effects Screening Level

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ANALYTE	MIN 24 HOUR (MG/M3) /PPM	AVG 24 HOUR (MG/M3)	MAX 24 HOUR (MG/M3)/ PPM	ATSDR MRL* (PPM)	ACUTE HEALTH EFFECT/ REVERSIBLE	HEALTH EFFECT LEVEL	EFFECTS	SOURCE
2-Propanol	0.003	0.015	1.96/ 0.7967		NO	400 PPM	May cause slight irritation	American Conference of Governmental Industrial Hygienists, Inc. Threshold Limit Value's for Chemical Substances and Physical Agents in the Work Environment with Intended, p. 23, 1983
						400 PPM @ 3-5 minutes	Mild irritation of the eyes, nose, & throat	Patty's Industrial Hygiene and Toxicology: Volume 2A, 2B, 2C, 1981-1982., p. 4570
2,2,3-Trimethylpentane	0.00006	0.00019	0.001		NO		No information found.	
2,2,4-Trimethylpentane	0.0001	0.00049	0.008		NO		No information found.	
2,2,5-Trimethylhexane	0.00008	0.00035	0.0008		NO		No information found.	
2,3-Dimethylbutane	0.0001	0.00054	0.006		NO		No information found.	
2,3,4-Trimethylpentane	0.0005	0.00036	0.003		NO		No information found.	
2,4,4-Trimethyl-1- pentene	0.00007	0.00022	0.002		NO		No information found.	

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ANALYTE	MIN 24 HOUR (MG/M3) /PPM	AVG 24 HOUR (MG/M3)	MAX 24 HOUR (MG/M3)/ PPM	ATSDR MRL* (PPM)	ACUTE HEALTH EFFECT/ REVERSIBLE	HEALTH EFFECT LEVEL	EFFECTS	SOURCE
2,4-Dimethylpentane	0.0001	0.00021	0.001		NO		No information found.	
2,5-Dimethylhexane	0.00006	0.00022	0.001		NO		No information found.	
2-Methyl-2-Pentene	0.00005	0.00021	0.0007		NO		No information found.	
3-Methyl-1-Butene	0.0001	0.00013	0.0008		NO	0.715 mg/m3 (250 ppb)	Available data limited	Texas Natural Resource Conservation Commission Short-term Effects Screening Level, Envirofacts
3-Methylhexane	0.0005	0.002	0.06		NO		Moderate inducer of hyaline droplet neruropathy in fischer rats	
3-Methylpentane	0.0005	0.0017	0.009		NO		Not classified as a recognized or suspect hazard.	Envirofacts
4-Methyl-1-Pentene	0.0002	0.0002	0.0006		NO	3.5 mg/m3 (1000ppb)	Very limited data.	Texas Natural Resource Conservation Commission Short-term Effects Screening Level, Envirofacts
4-Methylphenol (p-cresol)	0.00002	0.00003	0.0001		NO	1.4 PPM	Upper respiratory tract irritation	American Conference of Governmental Industrial Hygienists, Inc, Documentation of the Threshold Limit Value & Biological Exposure Indices, 1998.

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3-Methylphenol (m-cresol)	0.00002	0.00003	0.0001		NO	1.4 PPM	Upper respiratory tract irritation	American Conference of Governmental Industrial Hygienists, Inc, Documentation of the Threshold Limit Value & Biological Exposure Indices, 1998.
4-Nonene	0.00007	0.00019	0.001		NO		No information found.	
a-Pinene	0.0001	0.00043	0.04			64 ug/m3 (11 ppb)	Suspected neurotoxicant, respiratory, skin ir sense organ toxicant. Toxicants were studied in humans exposed to 0,10, 225 and 450 mg/m3. 5 subjects complained of eye, nose and throat irritation.	Texas Natural Resource Conservation Commission Short-term Effects Screening Level, Envirofacts
Acetaldehyde	0.0008	0.028	0.28/ 0.1556			50 PPM @ 15 minutes	Eye irritation	American Conference of Governmental Industrial Hygienists, Inc, Documentation of the Threshold Limit Value & Biological Exposure Indices. 6th ed., Volume I, p. 3, 1991

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ANALYTE	MIN 24 HOUR (MG/M3) /PPM	AVG 24 HOUR (MG/M3)	MAX 24 HOUR (MG/M3)/ PPM	ATSDR MRL* (PPM)	ACUTE HEALTH EFFECT/ REVERSIBLE	HEALTH EFFECT LEVEL	EFFECTS	SOURCE
Acetaldehyde 0.						134 PPM 30 min	Irritation of respiratory tract	Handbook of Environmental Data of Organic Chemicals. 2nd ed., p. 141
	0.000	0.028	0.28/ 0.1556		NO	200 PPM 15 min	Irritation of nose and throat	Handbook of Environmental Data of Organic Chemicals. 2nd ed., p. 141
	0.0008					200 PPM	Red eyes and transient conjunctivitis, and a majority of the subjects suffered from nose and throat irritation	American Conference of Governmental Industrial Hygienists, Inc, Documentation of the Threshold Limit Value & Biological Exposure Indices. 6th ed., Volume I, p. 3, 1991
Acetone	0.002	0.03	0.28/ 0.2632	Inh Acute = 26 (Neuro) Inh. Int = 13 (Neuro) Inh. Chr=13 (Neuro)	NO	250 PPM @ 6 hours/day for 6 days	Irritation of nose and throat, delayed visual reaction time, headache, lack of energy, weakness	Agency for Toxic Substances and Disease Registry Toxicological Profile, 1994, p. 14 & 18
						500 PPM @ 3-5 min/day for 1 day		Agency for Toxic Substances and Disease Registry Toxicological Profile, 1994, p. 14

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				Inh Acute = 26 (Neuro) Inh. Int = 13 (Neuro) Inh. Chr=13 (Neuro)	NO	500 PPM @ 6 hours/day for 1 day	Increased white blood cell count, decreased phagocytic activity of neutrophils	Agency for Toxic Substances and Disease Registry Toxicological Profile, 1994, p. 15
	0.002		0.28/ 0.2632				Increased white blood cell count, decreased phagocytic activity of neutrophils	Agency for Toxic Substances and Disease Registry Toxicological Profile, 1994, p. 14
		0.03				901 PPM @ 8 hours/day for 2-3 days	Throat, eye and nose irritation	Agency for Toxic Substances and Disease Registry Toxicological Profile, 1994, p. 14
Acetone						1000 PPM for 4-8 hours	Subjective symptoms of tension, tiredness, complaints and annoyance	Agency for Toxic Substances and Disease Registry Toxicological Profile, 1994, p. 18
						1000 PPM @ 7.5 hours/day for 1 day	Shortened menstrual cycle	Agency for Toxic Substances and Disease Registry Toxicological Profile, 1994, p. 19
						1006 PPM @ 8 hours/day for 7 days	Irritation of nose, eye, and throat	Agency for Toxic Substances and Disease Registry Toxicological Profile, 1994, p. 15

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ANALYTE	MIN 24 HOUR (MG/M3) /PPM	AVG 24 HOUR (MG/M3)	MAX 24 HOUR (MG/M3)/ PPM	ATSDR MRL* (PPM)	ACUTE HEALTH EFFECT/ REVERSIBLE	HEALTH EFFECT LEVEL	EFFECTS	SOURCE
Acetone	0.002				NO	1200 PPM @ 2 min-4 hr/day for 1 day		Agency for Toxic Substances and Disease Registry Toxicological Profile, 1994, p. 15
		0.03	0.28/	Inh Acute = 26 (Neuro) Inh. Int = 13		2000 PPM @ 4-8 hours/day		Agency for Toxic Substances and Disease Registry Toxicological Profile, 1994, p. 55
		0.03	0.2632	(Neuro) Inh. Chr=13 (Neuro)		2 min-4	headache	Agency for Toxic Substances and Disease Registry Toxicological Profile, 1994, p. 17
						21049 PPM @1- 8 hours		Agency for Toxic Substances and Disease Registry Toxicological Profile, 1994, p. 17
						50 PPM	Irritation of the human eye	Toxicology of the Eye. 3rd ed., 1986., p. 32
Acetonitrile	0.0003	0.012	0.7/ 0.4179		NO	50 PPM after 15 minutes		American Conference of Governmental Industrial Hygienists, Inc, Documentation of the Threshold Limit Value & Biological Exposure Indices. 6th ed., Volume I, p. 3, 1991

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ANALYTE	MIN 24 HOUR (MG/M3) /PPM	HOUR	MAX 24 HOUR (MG/M3)/ PPM	ATSDR MRL* (PPM)	ACUTE HEALTH EFFECT/ REVERSIBLE	HEALTH EFFECT LEVEL	EFFECTS	SOURCE
Acetonitrile	0.0003	0.012	0.7/ 0.4179		NO	Greater than 100-200 PPM	on the upper respiratory tract	American Conference of Governmental Industrial Hygienists, Inc, Documentation of the Threshold Limit Value & Biological Exposure Indices. 6th ed., Volume I, p. 3, 1991
Acetophenone	0.0001	0.00012	0.0009		NO		No information found.	
						0.00019 mg/m3 for 1 hour	Eye irritation in healthy human volunteers	Inhalation Reference Exposure Level- California EPA, Office of Environmental Health Hazard Assessment
Acrolein	0.00008	0.00033	0.004/	Inh. Acute = .00005 (Occular)	YES/YES	0.17 PPM @ 40 minutes	Eye irritation	Agency for Toxic Substances and Disease Registry Toxicological Profile, 1990, p. 11
			0.0008	Inh. Int = .000009 (Resp)		0.26 PPM @ 40 minutes	Nose irritation	Agency for Toxic Substances and Disease Registry Toxicological Profile, 1990, p. 11
						0.43 PPM @ 40 minutes	Throat irritation	Agency for Toxic Substances and Disease Registry Toxicological Profile, 1990, p. 11

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Acrolein 0.				Inh. Acute = .00005 (Occular) Inh. Int = .000009 (Resp)		1 PPM at 5 min		International Agency for Research on Cancer. (IARC) Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man. V36 147, 1985
	Acrolein 0.00008 0.00033	0.00033	0.004/			3 PPM	Severe pulmonary irritant & powerful lachrymogen, greatly irritates the conjunctiva & mucous membranes of upper resp tract	IARC. Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man. V36 147, 1985
						10 PPM	May be fatal in a few minutes	Prudent Practices for Handling Hazardous Chemicals in Labs., 1981., p. 107
Acrylonitrile	0.0001	0.00026		Inh. Acute = 0.1 (Neuro)		5-20 PPM over 10 yr	weakness with symptoms of anemia, jaundice, conjunctivitis, and abnormal whole blood and serum specific gravity, cholinesterase, urobilinogen,	Sakarai H, Kusimoto M; Rodo Kagaku 48: 273 (1972) as cited in USEPA; Ambient Water Quality Criteria Doc: Acrylonitrile (Draft) p.C- 39 (1980)

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Acrylonitrile	0.0001	0.00026	0.002/ 0.0009	Inh. Acute = 0.1 (Neuro)	N()	16 - 100 PPM @ 20-45 minutes		Agency for Toxic Substances and Disease Registry Toxicological Profile, 1990, p. 13
Aldrin	0.0000006	0.00000017	0.000001		NO		No information found.	
a-BHC (a-benzene hexachloride)	0.0000003	0.00000025	0.000001		NO		No information found.	
a-Chlordane	0.00000006	0.00000039	0.000003	Inh. Int. = 0.0002 (Hepatic) Inh. Chr. = 0.00002 (Hepatic)	NO	100-160 PPM for over 1 hr	Dypsnea, tachycardia, headache, and dizziness	Noxious Gases, 2nd edition (1943) as cited in USEPA; Chemical Hazard Information Profile: Aniline (Draft) p.40, 1978
Antimony	0.0000002	0.000019	0.0004		NO	8.87 mg/m3	Inflammation of lungs	Agency for Toxic Substances and Disease Registry Toxic Profile, 1992, pg. 15
Arsenic	0.0000003	0.0000024	0.00006			0.00019 mg/m3 for 4 hours	Decreased fetal weight in mice	Inhalation Reference Exposure Level- California EPA, Office of Environmental Health Hazard Assessment

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b-Pinene	0.0006	0.0002	0.001		NO	64 ug/m3	No information available	Texas Natural Resource Conservation Commission Short-term Effects Screening Level, Envirofacts
Benz(a)anthracene	0.000001	0.0000035	0.000007		NO		No information found.	
	0.001					1.3 mg/m3 for 6 hours	Reproductive/development toxicity in the rat	Inhalation Reference Exposure Level- California EPA, Office of Environmental Health Hazard Assessment
		1 0.0038 1	0.02/ 0.0063	Inh. Acute = 0.05 (Immuno.) Inh. Int. = 0.004 (Neural)	NO	50-150 PPM @ 300 minutes	Headache, lassitude and weariness	Patty's Industrial Hygiene and Toxicology, Volume 2B, 1994, p. 13095
Benzene						60 PPM @ 3 weeks	Mucous membrane irritation, dyspnea, and skin irritation	Agency for Toxic Substances and Disease Registry Tox Profile, 1995, pg. 14 & 41
						300-3,000 PPM	Drowsiness, dizziness, headache, vertigo, tremor, delirium, & loss of consciousness	Agency for Toxic Substances and Disease Registry Tox Profile, 1995, pg. 45
						500 PPM @ 60 minutes	More exaggerated symptoms	Patty's Industrial Hygiene and Toxicology, Volume 2B, 1994, p. 1309

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Benzene	0.001	0.0038	0.02/ 0.0063	Inh. Acute = 0.05 (Immuno.) Inh. Int. = 0.004 (Neural)	NO	20,000 PPM @ 5-10 minutes	Fatal	Agency for Toxic Substances and Disease Registry Tox Profile, 1995, pg. 13
Benzo(b)fluoroanthene	0.000004	0.0000052	0.000004		NO		No information found.	
Benzyl chloride	0.002	0.002	0.002		NO	0.24 mg/m3 for 1 hour 31 PPM	Eye and respiratory irritation in rats and mice Unbearable eye irritation	Inhalation Reference Exposure Level- California EPA, Office of Environmental Health Hazard Assessment Toxicology of the Eye.
Belizyi Cilioride	0.002	0.002	0.002		NO	35 PPM	Nasal irritation	3rd ed., 1986., p. 144 American Conference of Governmental Industrial Hygienists, Inc, Documentation of the Threshold Limit Val, 98
Beryllium	0.00000001	0.0000002	0.000001		NO			
Bis(2- Ethylhexyl)phthalate	0.00001	0.000033	0.00009		NO	100 PPM @ 1-2 weeks	Headache, nausea, numbness, ataxia	Agency for Toxic Substances and Disease Registry Toxicological Profile, 1992., p. 22
						8600 to 60000 PPM	Fatal poisoning	Dangerous Properties of Industrial Materials. 6th ed., 1984., p. 531

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Butyl acrylate	0.0002	0.0002	0.0002		NO		No information found.	
Cis-1,3-Dichloropropene	0.00008	0.00022	0.001	Inh. Int. = 0.003 (Respiratory) Inh. Chr. = 0.002 (Respiratory)	NO		No information found.	
Cis-2-Butene	0.0001	0.00046	0.003		NO		Not recognized as a human health hazard - Lacking Data for Acute Toxicity	On-line Envirofacts, Scorecard
Cis-2-Hexene	0.0001	0.00011	0.0006		NO		May irritate or burn skin and eyes.	
Cis-2-Octene	0.0008	0.00055	0.0008		NO		No information found.	
Cis-2-Pentene	0.00007	0.00019	0.001			0.09 mg/m3 (30 ppb)		Texas Natural Resource Conservation Commission Short-term Effects Screening Level, Envirofacts
Cis-3-Hexene	0.00009	0.00016	0.0008		NO		No information found.	
Cis-3-Methyl-2-Pentene	0.0002	0.0002	0.0003		NO		No information found.	
Cadmium	0.0000007	0.000006	0.0003		NO			

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						1.9 mg/m3 for 7 hours	Reproductive/Developmental toxicity in rats	Inhalation Reference Exposure Level- California EPA, Office of Environmental Health Hazard Assessment
Carbon Tetrachloride	0.0002	0.00064		Inh. Acute = 0.2 (Hepatic) Inh. Int. = 0.05 (Hepatic)		Repeated exposure to 10 PPM		National Institute of Occupational Safety & Health/ OSHAGuidelines for Chemical Hazards. DHHS(NIOSH) Publication No. 81-123, 1981., p. 2.
						25 - 30 PPM	Nausea, vomiting, dizziness, drowsiness and headache	National Institute of Occupational Safety& Health/OSHA- Occupational Health Guidelines for Chemical Hazards. DHHS(NIOSH) Publication No. 81-123, 1981., p. 2.

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Chloroethane (ethyl chloride)	0.002	0.001/ Inh. Acute = 15		19,000 PPM		American Conference of Governmental Industrial Hygienists, Inc,, (ACGIH) Documentation for Threshold Limit Value's(TLVs), 1998		
			0.0004	(Developmental)		25,000 PPM	Incoordination	ACGIH, Documentation for TLV's, 1998
						36,000 PPM	Noisy, talkativeness, incoordination followed by cyanosis, nausea, and vomiting	Hygienists, Inc,, (ACGIH) Documentation for Threshold Limit Value's(TLVs), 1998 ACGIH, Documentation for TLV's, 1998 ACGIH Documentation for TLVs, 1998 Inhalation Reference Exposure Level-
Chloroform				Inh Acute = 0.1		0.15 mg/m3 for 7 hours		Exposure Level- California EPA, Office of Environmental Health
	0.00009	0.00023	0.0002	(Hepatic) Inh. Int. =0.05 (Hepatic) Inh. Chr = 0.02	NO	1000 PPM @ 7 min	Dizziness and GI upset	Toxicology.3rd ed.,
				(Hepatic)		Several hrs @ 1024 PPM.		ACGIH, Documentation of theThreshold Limit Values for Substances in Workroom Air. 3 rd Edition, 1971, p. 413

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Chloroform				Inh Acute = 0.1		FEW MIN @ 1475 PPM	Dizziness & Salivation	American Conference of Governmental Industrial Hygienists, Inc, Documentation of the Threshold Limit Values for Substances in Workroom Air. 3 rd Edition, 1971, p. 413	
	0.00009	0.00023	0.0002	(Hepatic) Inh. Int. =0.05 (Hepatic) Inh. Chr = 0.02 (Hepatic)		From 4096 PPM			
						14,000 PPM	Central nervous system depression	Casarett and Doull's Toxicology.3rd ed., 1986., p. 644	
Chloromethane	0.001	0.0017	0.003/ 0.0014	Inh Acute = 0.5 (Neurol) Inh. Int. = 0.2 (Hepatic) Inh. Chr = 0.05 (Neurol)	NO				

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ANALYTE	MIN 24 HOUR (MG/M3) /PPM	AVG 24 HOUR (MG/M3)	MAX 24 HOUR (MG/M3)/ PPM	ATSDR MRL* (PPM)	ACUTE HEALTH EFFECT/ REVERSIBLE	HEALTH EFFECT LEVEL	EFFECTS	SOURCE
Chromium	0.000002	0.0000065	0.0001	Inh. Int. = 0.001 (Respiratory)	NO			
Crotonaldehyde	0.0001	0.000054	0.0001/ 0.000035		NO	4.1 PPM @ 5 minutes Few seconds @ 45 PPM		American Conference of Governmental Industrial Hygienists, Inc, Documentation of the Threshold Limit Value & Biological Exposure Indices. 6th ed. Volumes I, 1991., p. 342 American Conference of Governmental Industrial
								Hygienists, Inc, Documentation of the Threshold Limit Value & Biological Exposure Indices. 6th ed. Volumes I, 1991., p. 342
Cyclohexane	0.0003	0.001	0.02/ 0.0058		NO	300 PPM	Detectable by odor and somewhat irritating to the eyes and mucous membranes. Suspected Neurotoxicant - More hazardthan most chemicals in 2 of 10 ranking systems. Carcinogenic test not available	Governmental Industrial Hygienists, Inc, Documentation of the

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ANALYTE	MIN 24 HOUR (MG/M3) /PPM	HIN IV	MAX 24 HOUR (MG/M3)/ PPM	ATSDR MRL* (PPM)	ACUTE HEALTH EFFECT/ REVERSIBLE	HEALTH EFFECT LEVEL	EFFECTS	SOURCE
Cyclohexene	0.0001	0.00019	0.001/ 0.0003		NO		Not recognized as a human health hazard - Data Lacking	
Cyclopentane	0.00008	0.0003	0.009/ 0.0031		NO		Suspected Neurotoxicant - Less hazard than most chemicals in 3 of 10 ranking systems. No tests available.	On-line Envirofacts, Scorecard
Cyclopentene	0.00008	0.0001	0.0005		NO	10-15 PPM	Tolerable level. Not recognized as a human health hazard - Less hazard than most chemicals in 1 ranking systems. No tests available.	Patty's Industrial Hygiene and Toxicology: Volume 2B, 1994, p. 1276
delta-BHC (benzene hexachloride)	0.0000005	0.00000012	0.0000009		NO		No information found.	
Dibromochloromethane	0.001	0.00033	0.001		NO		No information found.	
Dichlorodifluoromethane	0.002	0.0033	0.04/ 0.0081		NO		No information found.	
Dichlorofluoromethane	0.00004	0.0006	0.0003/ 0.0001		NO		No information found.	
Dieldrin	0.0000002	0.00000022	0.0000007		NO		No information found.	
Endosulfan I	0.0000001	0.00000014	0.0000005		NO		No information found.	
Endosulfan Sulfate	0.0000002	0.00000022	0.000001		NO		No information found.	

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ANALYTE	MIN 24 HOUR (MG/M3) /PPM	AVG 24 HOUR (MG/M3)	MAX 24 HOUR (MG/M3)/ PPM	ATSDR MRL* (PPM)	ACUTE HEALTH EFFECT/ REVERSIBLE	HEALTH EFFECT LEVEL	EFFECTS	SOURCE
Endosulfan II	0.0000004	0.00000017	0.0000004		NO		No information found.	
Endrin aldehyde	0.0000009	0.00000025	0.0000009		NO		No information found.	
Endrin ketone	0.0000004	0.00000019	0.0000004		NO		No information found.	
						100 PPM	Much irritation of eyes & nose	Toxicology of the Eye. 3rd ed, p. 438
						1380 PPM @ 33 min	Headaches & slight numbness	Patty's Industrial Hygiene and Toxicology: Volume 2D, 1994, p. 2621
						2000 PPM	Eye irritation & lacrimation are immediate and severe	Toxicology of the Eye. 3rd ed., 1986., p. 413
Ethanol	0.09	0.106	25.21/ 13.4096		NO	3340 PPM @ 100 min	Sensations of warmth and coldness, nasal irritation, headaches, and numbness	Patty's Industrial Hygiene and Toxicology: Volume 2D, 1994, p. 2621
			13.4090			5000 PPM	Intolerable irritation of the eyes and nose	Toxicology of the Eye. 3rd ed., 1986., p. 413
						5000 PPM	Causes intolerable irritation of eye, mucous membranes & nose	Industrial Hygiene and Toxicology: Vol II., 1963., p. 1232
						5000-10,000	Coughing, and smarting of eyes and nose. Suspected carcinogen.	Patty's Industrial Hygiene and Toxicology: Volume 2D, 1994, p. 2621

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ANALYTE	MIN 24 HOUR (MG/M3) /PPM	AVG 24 HOUR (MG/M3)	MAX 24 HOUR (MG/M3)/ PPM	ATSDR MRL* (PPM)	ACUTE HEALTH EFFECT/ REVERSIBLE	HEALTH EFFECT LEVEL	EFFECTS	SOURCE
Ethanol		8840 PPM @ M 64 min d c fi	difficulty in breathing,	Patty's Industrial Hygiene and Toxicology: Volume 2D, 1994, p. 2621				
	0.09	0.106	25.21/ 13.4096		NO	15,000 PPM	coughing	Patty's Industrial Hygiene and Toxicology: Volume 2D, 1994, p. 2621
					20,000 PPM		Patty's Industrial Hygiene and Toxicology: Volume 2D, 1994, p. 2621	
				Inh. Acute = 0.04		0.094 mg/m3 for 1 hour		Inhalation Reference Exposure Level- California EPA, Office of Environmental Health Hazard Assessment
Formaldehyde	0.0004	0.0025	0.02/	(Resp) Inh. Int = 0.03 (Resp)	NO	2-5 PPM	and throat	Patty's Industrial Hygiene and Toxicology: Volume 2A, 1993, p. 299
				Inh. Chr = 0.008 (Resp)		4 PPM	irritation on initial exposure	Health and Safety Executive Monograph: Formaldehyde p.8, 1981
						25-50 PPM		Patty's Industrial Hygiene and Toxicology: Volume 2A, 1993, p. 299

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ANALYTE	MIN 24 HOUR (MG/M3) /PPM	HOUR	MAX 24 HOUR (MG/M3)/ PPM	ATSDR MRL* (PPM)	ACUTE HEALTH EFFECT/ REVERSIBLE	HEALTH EFFECT LEVEL	EFFECTS	SOURCE
Freon 114	0.00007	0.00019	0.0003		NO		Suspected neurotoxicant. Less hazardous than most chemicals in 4 ranking systems. Lacks tests.	On-line Envirofacts, Scorecard
gamma-BHC (g-benzene hexachloride)	0.0000001	0.00000032	0.000004		NO		No information found.	
Heptachlor epoxide	0.0000003	0.00000021	0.0000007		NO		No information found.	
Heptachlor	0.0000002	0.00000023	0.000001		NO		No information found.	
Heptanal	0.002	0.0079	0.06		NO		No information found.	
Hexachloro-1,3- Butadiene (hexachlorobutadiene)	0.0001	0.00096	0.01		NO		No information found.	
Hexachlorobenzene	0.000005	0.0000039	0.000005		NO		No information found.	
Hexanal	0.00009	0.0066	0.08		NO		No information found.	
Hydrochloric acid	0.0008	0.0038	0.04/ 0.0267		NO	1 hour		Inhalation Reference Exposure Level- California EPA, Office of Environmental Health Hazard Assessment
•						35 PPM	Irritation of throat	Environmental Contaminant Reference Databook, Vol. 1, 1995., p. 740

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ANALYTE	MIN 24 HOUR (MG/M3) /PPM	AVG 24 HOUR (MG/M3)	MAX 24 HOUR (MG/M3)/ PPM	ATSDR MRL* (PPM)	ACUTE HEALTH EFFECT/ REVERSIBLE	HEALTH EFFECT LEVEL	EFFECTS	SOURCE
	0.000	0.0020	0.04/			50-100 PPM	Can be tolerated for 1 hr, Longer exposure may result in pulmonary edema and laryngeal spasm	Databook, V 1, 95., p.740
Hydrochloric acid 0.	0.0008	0.0038	0.0267		NO	1,000-2,000 PPM	Dangerous even for brief exposure. Can damage vision. Symps incl vomiting, diarrhea, thirst, & circulatory collapse.	Environmental Contaminant Reference Databook, Vol. 1, 1995., p. 740
						0.24 mg/m3 for 1 hour	Eye and respiratory tract membrane irritation of 20 healthy male volunteers	Inhalation Reference Exposure Level- California EPA, Office of Environmental Health Hazard Assessment
						20 PPM	Gas is potentially corrosive	Clinical Toxicology. 5th , 1972., p. 176
Hydrofluoric acid	0.00001	0.00032	0.003/		NO	30 PPM	Sour taste, smarting eyes	Environmental Contaminant Reference Databook, V1, 95., p. 751
			0.0039			50-250 PPM	Dangerous with short exposure	Environmental Contaminant Reference Databook V 1., 95, p. 751
						60 PPM	Burn pain may be delayed up to 1 hour. Apparent irritation of nose, and eyes	Environmental Contaminant Reference Databook, V1, 95., p. 751
						120 PPM	Irritation of skin, respiratory; vapors can cause ulcers of respiratory tract	Environmental Contaminant Reference Databook V1., 95, p. 751

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ANALYTE	MIN 24 HOUR (MG/M3) /PPM	AVG 24 HOUR (MG/M3)	MAX 24 HOUR (MG/M3)/ PPM	ATSDR MRL* (PPM)	ACUTE HEALTH EFFECT/ REVERSIBLE	HEALTH EFFECT LEVEL	EFFECTS	SOURCE
Indan	0.00006	0.00018	0.0007		NO		No information found.	
Indene	0.0003	0.00014	0.0003		NO		No information found.	
Indeno(1,2,3-cd)pyrene	0.000001	0.0000027	0.000001		NO		No information found.	
Isobutane (2-methyl propane)	0.0009	0.005	0.18/ 0.0756		NO	10,000 PPM @ 10 minutes		American Conference of Governmental Industrial Hygienists, Inc, Documentation of the Threshold Limit Values, 1998
Isobutene	0.001	0.0024	0.01		NO		No information found.	
1-Butene	0.001	0.0024	0.01		NO		No information found.	
Isobutylbenzene	0.00007	0.00016	0.00007		NO		No information found.	
Isodrin	0.0000003	0.00000015	0.000002		NO		No information found.	
Isoheptane	0.0003	0.0012	0.04		NO		No information found.	
Isohexane	0.0005	0.0023	0.02/ 0.0057	n-Hexane Inh. Chr. = 0.6 (Neurol.)	NO		No information found.	
Isopentane	0.002	0.0079	0.18		NO		No information found.	

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ANALYTE	MIN 24 HOUR (MG/M3) /PPM	AVG 24 HOUR (MG/M3)	MAX 24 HOUR (MG/M3)/ PPM	ATSDR MRL* (PPM)	ACUTE HEALTH EFFECT/ REVERSIBLE	HEALTH EFFECT LEVEL	EFFECTS	SOURCE
Isoprene	0.00008	0.00027	0.002		NO		No information found.	
Isovaleraldehyde	0.00009	0.000071	0.0005		NO		No information found.	
Lead	0.00002	0.00026	0.02		NO			
m-Diethylbenzene	0.00007	0.00028	0.002/ 0.0004		2000 PPM Caused irritation irrita		Eye irritation Caused immediate, severe irritation, lacrimation, and irritation of the mucous membranes of the nose	Patty, F., Industrial Hygiene and Toxicology: Volume II, 2nd ed., 1963., p. 1232 Patty, F., Industrial Hygiene and Toxicology: Volume II, 2nd ed., 1963., p. 1232
						5000 PPM	Caused intolerable irritation of eyes and nose	Patty, F., Industrial Hygiene and Toxicology: Volume II, 2nd ed., 1963., p. 1232
m-Ethyltoluene	0.0002	0.0012	0.006		NO		No information found.	
Mercury	0.000004	0.0000075	0.00009	Inh. Chr = 0.0002 (Neurol.)		0.0018 mg/m ³ for 1 hour	Behavioral deficits after in utero exposure to metallic mercury vapor	Inhalation Reference Exposure Level- California EPA, Office of Environmental Health Hazard Assessment

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ANALYTE	MIN 24 HOUR (MG/M3) /PPM	AVG 24 HOUR (MG/M3)	MAX 24 HOUR (MG/M3)/ PPM	ATSDR MRL* (PPM)	ACUTE HEALTH EFFECT/ REVERSIBLE	HEALTH EFFECT LEVEL	EFFECTS	SOURCE
Methylcyclopentane	0.0002	0.00076	0.02		NO		unavailable	
Methylcyclopentene	0.0003	0.0003	0.0003		NO	Above 75 to 100 PPM.		
Methylene Chloride	0.002	0.01	0.68/ 0.3301	Inh Acute =0.6 ppm (Neurol) Inh Int = 0.3 (Hepatic) Inh Chr = 0.3 (Hepatic)	NO	14 mg/m3 for 1 hour	Subtle impairment of the central nervous system in humans	Inhalation Reference Exposure Level- California EPA, Office of Environmental Health Hazard Assessment
						50-105 PPM @ 15-30 min	Provoked gastrointestinal disturbances and central nervous system impairment in a few workers	International Labour Office. Encyclopedia of Occ Health and Safety. Vols. I&II, 1983., p.1171
Methylisobutylketone	0.0005	0.0016	0.01/		NO	100 PPM	Some individuals developed headache and nausea, whereas another group complained only of respiratory tract irritation.	The Chemistry of Industrial Toxicology 2nd ed., 1959
		3.55.15	0.0024			200 PPM	Odor was objectionable and vapor was irritating to the eyes	American Conference of Governmental Industrial Hygienists, Inc, Documentation of the Threshold Limit Value & Biological Exposure Indices. 6th ed., Volumes I, p. 1020, 1991.

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ANALYTE	MIN 24 HOUR (MG/M3) /PPM	AVG 24 HOUR (MG/M3)	MAX 24 HOUR (MG/M3)/ PPM	ATSDR MRL* (PPM)	ACUTE HEALTH EFFECT/ REVERSIBLE	HEALTH EFFECT LEVEL	EFFECTS	SOURCE
n-Butane	0.002	0.0072	0.04/ 0.0168		NO	10,000 PPM for 10 min	Central nervous system depression	Patty's Industrial Hygiene and Toxicology:Volume 2B, 4th ed., 1994, p. 1230
n-Butyraldehyde	0.00009	0.0099	0.09		NO		No information found.	
n-Decane	0.0006	0.0024	0.08		NO		No information found.	
						1000 PPM @ 6 minutes	Slight dizziness	American Conference of Governmental Industrial Hygienists, Inc, Documentation of the Threshold Limit Values, 1998
n-Heptane	0.0003	0.0013	0.04/ 0.0098		NO	5000 PPM	Nausea, loss of appetite, and gasoline-like taste	American Conference of Governmental Industrial Hygienists, Inc, Documentation of the Threshold Limit Values, 1998
						16,000 PPM	Fatal	American Conference of Governmental Industrial Hygienists, Inc, Documentation of the Threshold Limit Values, 1998

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ANALYTE	MIN 24 HOUR (MG/M3) /PPM	AVG 24 HOUR (MG/M3)	MAX 24 HOUR (MG/M3)/ PPM	ATSDR MRL* (PPM)	ACUTE HEALTH EFFECT/ REVERSIBLE	HEALTH EFFECT LEVEL	EFFECTS	SOURCE
						450-650 PPM for as little as 2 months 880 PPM @ 15 min	May result in peripheral neuropathy, characterized by muscular weakness, loss of sensation, and impaired gait. Can cause eye and upper respiratory tract irritation in humans	Biological Monitoring Methods for Industrial Chemicals. 2nd ed., 1988., p. 172 Patty's Industrial Hygiene and Toxicology:Volume
n-Hexane	0.0005	0.0029	0.02/ 0.0057	Inh. Chr. = 0.6 (Neurol)	NO	5000 PPM	humans Caused dizziness & giddiness, slight nausea, headache, eye & throat irritation	2A, 2B, 2C, 3rd ed., 1981-1982., p. 3186 American Conference of Governmental Industrial Hygienists, Inc, Documentation of the Threshold Limit Value & Biological Exposure Indices.5th ed., p 305, 86.
						5000 PPM @ 10 min	Causes marked vertigo	Patty's Industrial Hygiene and Toxicology:Volume 2A, 2B, 2C, 3rd ed., 1981-1982., p. 3186
						10500g/m3 (2000ppb)		Texas Natural Resource Conservation Commission Short-term Effects Screening Level, Envirofacts

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ANALYTE	MIN 24 HOUR (MG/M3) /PPM	AVG 24 HOUR (MG/M3)	MAX 24 HOUR (MG/M3)/ PPM	ATSDR MRL* (PPM)	ACUTE HEALTH EFFECT/ REVERSIBLE	HEALTH EFFECT LEVEL	EFFECTS	SOURCE
n-Nonane	0.0004	0.0012	0.005		NO		No information found.	
n-Octane	0.0002	0.00075	0.02/ 0.0043		NO		No information found.	
n-Pentane	0.0007	0.005	0.29/ 0.0983		NO	130,000 PPM	Lethal concentration	Patty's Industrial Hygiene and Toxicology:Volume 2B, 1994, p. 1231
n-Propylbenzene	0.0001	0.00043	0.002		NO		No information found.	
n-Undecane	0.0002	0.0014	0.1		NO		No information found.	
			0.002/	Inh. Chr. = 0.002		15 PPM	Vapors can cause eye irritation	Kirk-Othmer Encyclopedia of Chemical Technology. 3rd ed., V15 713,1981
Naphthalene	0.00006	0.00032		(Resp)	NO	0.44 mg/m3 (440 ug/m3 or 85 ppb) for 1 hour		Texas Natural Resource Conservation Commission Short-term Effects Screening Level, Envirofacts
Neohexane	0.00005	0.0004	0.002		NO		No information found.	
Neopentane	0.00003	0.000082	0.0004		NO		No information found.	
o-Ethyltoluene (2- ethyltoluene)	0.0001	0.00054	0.003		NO		No information found.	

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ANALYTE	MIN 24 HOUR (MG/M3) /PPM	AVG 24 HOUR (MG/M3)	MAX 24 HOUR (MG/M3)/ PPM	ATSDR MRL* (PPM)	ACUTE HEALTH EFFECT/ REVERSIBLE	HEALTH EFFECT LEVEL	EFFECTS	SOURCE
				Total Xylenes		22 mg/m3 for 1 hour		Inhalation Reference Exposure Level- California EPA, Office of Environmental Health Hazard Assessment
o-Xylene	0.0006	0.0024	0.02	Inh. Acute = 1 (Neurol) Inh. Int = 0.7 (Develop) Inh. Chr = 0.1 (Neurol)	NO	110-460 PPM	Irritation to eyes, nose, and throat	Patty's Industrial Hygiene and Toxicology:Volume 2B, 4 th ed., 1994, p. 1333
						200 PPM @ 3-5 minutes	Irritation to eyes, nose, and throat	
						460 PPM	Eye irritation	Patty's Industrial Hygiene and Toxicology:Volume 2B, 4 th ed., 1994, p. 1336
						1000 PPM		Patty's Industrial Hygiene & Toxicology: Volume II, 2nd ed., 1963, p. 1232
p-Diethylbenzene	0.0003	0.00029	0.0009		NO	2000 PPM		Patty's Industrial Hygiene & Toxicology: Volume
						5000 PPM		Patty, F. , Industrial Hygiene & Toxicology: Volume II, 2nd ed., 1963, p. 1232
p-Ethyltoluene	0.0002	0.00064	0.003		NO		No information found.	

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ANALYTE	MIN 24 HOUR (MG/M3) /PPM	AVG 24 HOUR (MG/M3)	MAX 24 HOUR (MG/M3)/ PPM	ATSDR MRL* (PPM)	ACUTE HEALTH EFFECT/ REVERSIBLE	HEALTH EFFECT LEVEL	EFFECTS	SOURCE
p-Isopropyltoluene	0.00009		0.0007		NO		No information found.	
p-Xylene	0.002	0.0059	0.06/ 0.0138	Total Xylene Inh. Acute = 1 (Neurol) Inh. Int = 0.7	NO	22 mg/m3 for 1 hour	Eye irritation in healthy human volunteers	Inhalation Reference Exposure Level- California EPA, Office of Environmental Health Hazard Assessment
			0.0138	(Develop) Inh. Chr = 0.1 (Neurol)		460 PPM	Eye irritation	Patty, F. , Industrial Hygiene & Toxicology: Volume 2B, 4th ed., 1994, p. 1336
m-Xylene	0.002	0.0059	0.06/ 0.0138	Total Xylenes Inh. Acute = 1 (Neurol) Inh. Int = 0.7	NO	22 mg/m3 for 1 hour	Eye irritation in healthy human volunteers	Inhalation Reference Exposure Level- California EPA, Office of Environmental Health Hazard Assessment
			0.0138	(Develop) Inh. Chr = 0.1 (Neurol)		460 PPM	Eye irritation	Patty, F. , Industrial Hygiene & Toxicology: Volume 2B, 4th ed., 1994, p. 1336
Phenanthrene	0.00001	0.000026	0.0001		NO		No information found.	
PM-10	0.009	0.069	0.24		NO		No information found.	
PM-2.5		0.026	0.13		NO		No information found.	
Propane	0.003	0.014	0.09/ 0.0500		NO		No information found.	
Propionaldehyde	0.0002	0.00067	0.004		NO		No information found.	

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Propylene	0.0005	0.0054	0.24		NO		No information found.	
Sulfuric Acid	0.005	0.011	0.05			0.12 mg/m3 for 1 hour	Small changes in airway function tests, especially in asthmatics; respiratory irritation in humans	Inhalation Reference Exposure Level- California EPA, Office of Environmental Health Hazard Assessment
trans-2-Butene	0.0001	0.00052	0.003		NO		No information found.	
trans-2-Hexene	0.00004	0.00013	0.0006		NO		No information found.	
trans-2-Pentene	0.00008	0.00033	0.002		NO		No information found.	
						20 mg/m3 for 1 hour	irritation, headache and light- headedness.	Inhalation Reference Exposure Level- California EPA, Office of Environmental Health Hazard Assessment
Tetrachlorethylene (Perchlorethylene)	0.0001	0.0012	0.01/ 0.0015	Inh. Acute = 0.2 (Neurol) Inh. Chr = 0.04	NO	Above 75 to 100 PPM.	Mucous membrane & upper respiratory irritant	Medical Toxicology - Diagnosis and Treatment of Human Poisoning., 1988., p. 986
				(Neurol)		200 PPM	Early signs of central nervous system depression	American Conference of Governmental Industrial Hygienists, Inc, Documentation of the Threshold Limit Value & Biological Exposure Indices, 5th, p464, 1986.

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Tolualdehyde	0.0001	0.001	0.03		NO		No information found.	
Toluene	0.007	0.024	0.52/ 0.1383	Inh. Acute = 1 (Neurol) Inh. Chr = 0.08	NO	37 mg/m3 for 1 hour	Headache, dizziness, slight eye and respiratory irritation in humans	Inhalation Reference Exposure Level- California EPA, Office of Environmental Health Hazard Assessment
				(Neurol)		100 PPM	Transient irritation	Patty's Industrial Hygiene and Toxicology: Volume 2B, 1994, p. 1327
						200 PPM	Affected the central nervous system in humans	Patty's Industrial Hygiene and Toxicology: Volume 2B, 1994, p. 1327
						300-400 PPM	Irritation to human eyes	Toxicology of the Eye. 3rd ed, 1986.,p. 927
	0.007	0.024	0.52/	Inh. Acute = 1 (Neurol)	NO	400 PPM	Mild eye irritation, lacrimation	Patty's Industrial Hygiene and Toxicology: Volume 2B, 1994, p. 1327
Toluene	0.007	0.024	0.1383	Inh. Chr = 0.08 (Neurol)		600 PPM	Lassitude, hilarity, slight nausea	Patty's Industrial Hygiene and Toxicology: Volume 2B, 1994, p. 1327
						800 PPM	Irritation is slight	Toxicology of the Eye. 3rd ed, 1986., p. 927
						800 PPM	Metallic taste, headache, lassitude, slight nausea	Patty's Industrial Hygiene and Toxicology: Volume 2B, 1994, p. 1327

^{*}ATSDR MRL's are Minimal Risk Levels, which are screening levels to estimate the daily human exposure to a hazardous substance which will not cause any adverse noncancer health effect. **Inh** = Inhalation; **Acute** = Acute exposure duration (1-14 days); **Int.** = Intermediate exposure duration (14 - 365 days); **Chr.** = Chronic exposure (365 days or longer)

ANALYTE	MIN 24 HOUR (MG/M3) /PPM	AVG 24 HOUR (MG/M3)	MAX 24 HOUR (MG/M3)/ PPM	ATSDR MRL* (PPM)	ACUTE HEALTH EFFECT/ REVERSIBLE	HEALTH EFFECT LEVEL	EFFECTS	SOURCE
Total Dioxins/Furans (2,3,7,8-TCDD TEQs)	3.0E-10	1.6E-09	4.0E-08		NO			
Trichloroethylene	0.0003	0.0017	0.01/ 0.0019	Inh. Acute = 2 (Neurol) Inh. Int = 0.1 (Neurol)	NO			Handbook of Environmental Data of Organic Chemicals., 2nd ed., 1983., p. 1135
Valeraldehyde	0.00008	0.00023	0.004/ 0.0011		NO		No information found.	
Vinyl Acetate	0.0009	0.0054	0.04/ 0.0114	Inh Int.= 0.01	NO	19.4 – 71 PPM @ 0.5 – 4 hours	Respiratory tract irritation	American Conference of Governmental Industrial Hygienists, Inc, Documentation of Threshold Limit Values (TLVs), 1998
			0.0114	(Resp)		21.6 PPM	Irritating to eyes	American Conference of Governmental Industrial Hygienists, Inc, Documentation of TLVs, 1998
Vinyl Chloride	0.0001	0.0001	0.0006/ 0.0002	Inh. Acute = 0.5 (Develop) Inh. Int = 0.03 (Hepatic)	NO		Mild headache and irritation of eyes and respiratory tract in healthy human volunteers	Inhalation Reference Exposure Level- California EPA, Office of Environmental Health Hazard Assessment

^{*}ATSDR MRL's are Minimal Risk Levels, which are screening levels to estimate the daily human exposure to a hazardous substance which will not cause any adverse noncancer health effect. **Inh** = Inhalation; **Acute** = Acute exposure duration (1-14 days); **Int.** = Intermediate exposure duration (14 - 365 days); **Chr.** = Chronic exposure (365 days or longer)

ANALYTE	MIN 24 HOUR (MG/M3) /PPM	AVG 24 HOUR	MAX 24 HOUR (MG/M3)/ PPM	ATSDR MRL* (PPM)	ACUTE HEALTH EFFECT/ REVERSIBLE	HEALTH EFFECT LEVEL	EFFECTS	SOURCE
Vinyl Chlorido	0.0001	0.0001		Inh. Acute = 0.5 (Develop)		8000 – 20,000 PPM		American Conference of Governmental Industrial Hygienists, Inc, Documentation of TLVs, 1998
Vinyl Chloride	0.0001	0.0001	0.0002	Inh. Int = 0.03 (Hepatic)		· ·		American Conference of Governmental Industrial Hygienists, Inc, Documentation of TLVs, 1998

^{*}ATSDR MRL's are Minimal Risk Levels, which are screening levels to estimate the daily human exposure to a hazardous substance which will not cause any adverse noncancer health effect. **Inh** = Inhalation; **Acute** = Acute exposure duration (1-14 days); **Int.** = Intermediate exposure duration (14 - 365 days); **Chr.** = Chronic exposure (365 days or longer)

Pregnancy Loss at NAF Atsugi Japan (June 1995-May 1998)



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PUBLIC HEALTH SUMMARY

A study designed to describe the rate of miscarriage at NAF Atsugi and other naval facilities in Japan was conducted in the summer of 1998. The researchers examined hospital and clinic records for Navy personnel or their dependents who were pregnant and living in Japan at some point between June 1995 and May 1998. Information used to calculate the miscarriage rates came from three different sources, Delivery Logs at Naval Hospital Yokosuka (NHY), Pathology records at NHY and the Prenatal Log at the Atsugi Branch Medical Clinic.

Data were collected on the number of live births and the number of miscarriages. For the purposes of this study, a miscarriage was defined as an unintentional pregnancy loss at up to the 28th week of pregnancy. Multiple births were excluded from the analysis. The miscarriage or pregnancy loss rate was defined as the number of miscarriages divided by the total number of pregnancies examined (the number of babies born plus the number of miscarriages).

A total of 1862 pregnancies with known outcomes from NHY (including Atsugi, Yokosuka, Sasebo and Iwakuni) were examined. There were 1701 live births and 130 miscarriages between June 1995 and May 1998. The corresponding miscarriage rate for this period was 7.1%. The rate at NAF Atsugi, determined from review of the prenatal log during the same period, was 8.8%. This rate was based on the examination of 353 total pregnancies, with 322 live births and 31 miscarriages.

The miscarriage rate in the U.S. population varies based on how miscarriages are defined and identified. The average range is 10% to 15% for recognized pregnancies. The results of this study suggest that the occurrence of miscarriage at NAF Atsugi and other naval facilities within Japan is at the low end of the expected range described for the population of the United States. The miscarriage rate at NAF Atsugi cannot be directly compared to that of other naval facilities in Japan because different methods were used to gather the data.

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Pregnancy Loss in Women at NAF Atsugi Japan (June 1995 – May 1998)

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INTRODUCTION

Background

Naval Air Facility (NAF) Atsugi is located in Ayase City, Japan. It is about 16 km west of Yokohama and about 36 km south west of Tokyo. Atsugi's population currently includes about 456 active duty officers, 2950 active duty enlisted personnel, 1664 active duty Japanese Maritime Self Defense Force personnel, 1751 dependents, 1383 civilian employees, and a few retirees.

Japan's crowded conditions and small size makes it impossible to dispose of municipal wastes in landfills. As a result, seventy five percent of municipal waste is burned in more than 4,000 public and privately owned incinerators. Japanese residents living near incinerators have complained of a variety of health effects, including difficulty breathing, numbness, increased infant mortality, atopic dermatitis, and an increased cancer incidence.

The Shinkampo incineration complex is located about 150 meters south of NAF Atsugi. Air quality measurements conducted on the base have detected the presence of a number of compounds at levels above the EPA Region III Risk-Based Screening Concentrations (RBCS). The RBCs are very conservative health screening values, based on 30 years of exposure to various pollutants. These include dioxins and furans, hydrochloric acid and other acid gasses, a number of aldehydes, heavy metals including arsenic and cadmium, a number of volatile organic compounds including benzene and methyene chloride, and particulates.

The presence of the incinerator and the general level of air pollution in the area have led to concerns by base residents regarding the possible health effects of these exposures. A preliminary study was conducted in December 1994 to determine if a higher than expected

incidence of adverse pregnancy outcomes was occurring at Naval Hospital Yokosuka (NHY). A follow up visit to collect additional data took place in 1995.

Study Goals

In response to continuing concerns, the primary goal of this study was to describe the occurrence of pregnancy loss among beneficiaries living on or near NAF Atsugi and those living in Japan at other Navy facilities. Data collection took place during the summer of 1998.

MATERIALS AND METHODS

Study Design

The severe time constraint placed on the completion of this study dictated a retrospective design.

Study Population

The study population consisted of US Navy beneficiaries in the NHY catchment area who were pregnant at some point during the interval of June 1995 to May 1998. This includes women who lived at NAF Atsugi (either on or off base), Sasebo, Iwakuni, or Yokosuka.

Outcome Definition

The two study outcomes of interest were viable, singleton births and pregnancy losses (PL). PL was defined as "the unintentional termination of an intrauterine pregnancy at up to the 28th week of gestation". Multiple births were excluded from the analysis. The PL percentage was defined as the number of PLs divided by the number of singleton live births and PLs.

Data Sources

There were two methods of outcome ascertainment: (1) Delivery Logs/Pathology Records from NHY and (2) Atsugi Branch Medical Clinic Prenatal Log. Using the first method, pregnancies were identified from the NHY labor and delivery logs and PLs were identified from

NHY pathology records. Using the second method, both pregnancies and PLs were identified from the Atsugi Branch Medical Clinic Prenatal Log, which the clinic has maintained since January 1996. This log contained entries for 493 women when data collection for this study took place.

Data from the Atsugi housing office were used to determine on base/off base status and to identify women who left the Atsugi area prior to the end of their pregnancies.

Data were collected on a very limited number of potential confounding variables, including maternal age and race, and on or off base status at Atsugi.

The EpiInfo statistical software package was used for data entry and analysis.

RESULTS

As presented in Table 1, there were 1,701 viable singleton births during the 36-month study period at NHY, including those of women from Atsugi, Iwakuni, and Sasebo. The pathology department records for this period included 130 PLs, 14 cases of ectopic pregnancy, and 4 molar pregnancies. Using PLs ascertained from pathology records and singleton births, the overall PL percentage for the entire group was 7.1%. Information from the Atsugi Prenatal Log allowed identification of 315 births and pathology specimens from 12 PLs that occurred in women from Atsugi, resulting in a PL percentage of 3.7% at Atsugi by this method. Removal of Atsugi births and pathology reports from Table 1 results in a PL percentage of 7.8% for the remainder of the pregnancies at NHY (which include Iwakuni and Sasebo as well as Yokosuka).

The age and racial distribution of mothers are also presented in Table 1. Race was not recorded in the Atsugi prenatal log and was missing from 36% of the pathology reports, thus precluding an analysis of race as a confounding factor.

Data from the Atsugi Pregnancy Log are presented in Table 2. Using these data, the PL percentage among Atsugi residents was 8.8%. As noted above, 12 of the 31 PLs noted in the log were also found in the NHY pathology reports. Atsugi housing data was not available for 31% of births. This lack of data, combined with the low number of Atsugi PLs, prevented an on base-off base risk comparison.

There was also no evidence of seasonality or clustering in PLs.

Table 1. Known Pregnancy Outcomes At Naval Hospital Yokosuka From June 1995 And May 1998, Based On NHY Pathology And Delivery Logs (Includes Patients From Yokosuka, Atsugi, Sasebo, And Iwakuni)

Pregnancies with known outcomes	1862
Viable singleton births	1701
Pregnancy loss	130
Percent PL*	7.1%
Mean age-live delivery	28.5
Mean age-PL	28.8
Race of Mothers (with live births)	
White	32.7%
Black	7.2%
Filipino	28.3%
Other Asian	25.4%
Other	3.4%
Unknown	3.0%

^{*} Calculated as PL/(PL+Live Singleton Births)

Table 2. Atsugi Pregnancy Outcomes Between January 1996 And May 1998 (From The Atsugi Prenatal Log).

Total pregnancies	493
Lost to follow up	56
Known Outcomes	357
Not yet delivered	80
Live singleton births	322
PL	31
Percent PL*	8.8%
Twin sets	1
Still births	1
Elective termination	2

^{*} Calculated as PL/(PL+Live Singleton Births)

DISCUSSION

The extent of pregnancy losses in the U.S. population has been well described. Although reported losses vary between studies, generally about 10% to 15% of recognized pregnancies result in pregnancy loss. If early occult losses are considered, then up to one third of conceptions fail. Additional (unrecognizable) losses occur due to failure to implant. Despite this knowledge, studies of pregnancy loss are inherently difficult to design and conduct for a number of reasons.

First, outcomes from a given couple are statistically dependent and are related to differences in inherent risk. This becomes important when multiple pregnancies from a couple are included in a study. It is also preferable to study a woman's entire reproductive history to separate possible intrinsic risks from environmental ones. The optimal denominator for a reproductive study is women at risk for pregnancy, not those with clinically recognized pregnancies. None of these steps could be taken in this study.

When using a retrospective, hospital-based approach, there is the risk of selection bias, since not all PLs are found in a hospital record. That was certainly the case in this study, with pathology records available for only 12 of the 31 PLs identified in the Atsugi Prenatal Log – a substantial under-ascertainment that precludes valid statistical comparisons between Atsugi and NHY using this method. It is highly likely that women with symptoms indicating a threatened abortion or late pregnancy loss would prefer to seek urgent care at a hospital. The hospital most convenient to women who live near Yokosuka is Naval Hospital Yokosuka, while the hospital most convenient to women at remote bases, such as Atsugi, would be a local civilian hospital. Pathology specimens resulting from pregnancy losses evaluated at civilian hospitals would be less likely to be sent to NHY Pathology Department than those evaluated by the NHY Emergency Room. Thus, the apparent difference in PL percentage between Atsugi and the remainder of the NHY population can likely be explained by differential patient presentation and clinician referral patterns between a relatively remote outpatient facility (Atsugi) and a full service hospital with continuous Emergency Department coverage and in-house pathology services (NHY).

Since such a high percentage of pregnancy losses occur very early in the pregnancy it is very important to determine the gestational age at which the pregnancy was recognized. Women

who are concerned about pregnancy loss tend to recognize their pregnancies early and to do home pregnancy tests. This can result in a substantial bias, with the concerned group appearing to have a higher PL rate because early occult losses are classified as PLs due to earlier recognition of the pregnancy. Not having this data is a serious limitation.

This study was also limited by a relatively large percentage of pregnancies with no known outcome. This problem resulted from the high mobility found in a military population and the fact that a substantial percentage of the women are Japanese nationals. Many of these women chose to use civilian Japanese medical facilities for prenatal care and/or for delivery. No information was available on the outcome of these pregnancies. The mobility of this population also made the study subject to misclassification of exposure, since many of the women at Atsugi were not there for their entire pregnancy. We have no data on individual exposures to incineration products or other air pollutants.

Finally, there are many known risk factors for PL, including gravidity, parity, prior PL, maternal exposure to tobacco, alcohol, other drugs, history of pelvic inflammatory disease, chronic medical conditions, education, nutrition, occupation, as well as paternal risk factors. We were not able to adjust for these potential confounders.

In the face of these limitations, only very tentative conclusions can be drawn from these results. First, the use of pathology reports as a surrogate for pregnancy loss is problematic, particularly when populations being compared have substantial differences in patient presentation and referral patterns. The data from the Atsugi Prenatal Log, which we know to be more inclusive than the NHY pregnancy and pathology data, indicated the magnitude of the under-ascertainment that can result. Second, although the Atsugi log data are not necessarily comparable to data generated by other studies and inter-study comparisons must be viewed with skepticism, the 8.8% Atsugi PL percentage is lower than the expected 10-15% found in the United States civilian population.²⁻⁷ Since the military population is generally younger and healthier than the United States civilian population, we would expect their percentage of pregnancy loss to be lower than population norms.

Nonetheless, it is clear that that the time constraints placed on the completion of this study dictated a retrospective design using existing data that was wholly inadequate for this type of research. Therefore, any determination of no excess risk must be viewed as very tentative. If there is a need to reach firmer conclusions regarding the possibility of an elevated risk of adverse

pregnancy outcomes among women at Atsugi then it will be necessary to conduct a carefully designed prospective study using survival analytic methods that includes time to pregnancy, involves interviews with pregnant women and/or their husbands, and records a range of health outcomes. This will require careful study design and will probably need a dedicated research staff on site.

CONCLUSIONS

- Due to its retrospective nature, this study had a number of very serious limitations. A
 prospective study of this population would provide more definitive assessment of the risk of
 pregnancy loss.
- 2. Within the study constraints, the risk of pregnancy loss among women living at NAF Atsugi appears to be at the low end of the range of expected risks, compared to the U.S. civilian population.

REFERENCES

- Wilcox AJ, Weinberg CR, O'Connor JF, et al: Incidence of early pregnancy loss, N Engl J Med 1988 Jul 28;319(4):189-194
- 2. Sciarra JJ, editor: Gynecology and Obstetrics, Revised edition, J B Lippincott Company, 1995; Ch 69:1-5, 9-14
- 3. Goldstein SR: Embryonic death in early pregnancy: a new look at the first trimester, Obstet Gynecol 1994 Aug;84(2):294-7
- 4. Simpson JL, Gray RH, Queenan JT, et al: Risk of recurrent pregnancy loss for pregnancies discovered in the fifth week of gestation, Lancet 1994 Oct 1;344(8927):964
- 5. Goldhaber MK, Fireman BH: The fetal life table revisited: pregnancy loss rates in three Kaiser Permanente cohorts, Epidemiology 1991 Jan;2(1):33-9
- 6. Ellish NJ, PLoda K, O'Connor J, et al: A prospective study of early pregnancy loss, Hum Reprod 1996 Feb;11(2):406-12
- 7. Parazzini F, Chatenoud L, Tozzi L, et al: Determinants of pregnancy loss in the first trimester of pregnancy, Epidemiology 1997 Nov 8(6): 681-3

Is Air Pollution from the Shinkampo Incinerator Associated with Adverse Respiratory Effects among Children at NAF Atsugi?



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PUBLIC HEALTH SUMMARY

Between May 7, 1998 and June 5, 1998, 127 fifth and sixth grade children who attended the Atsugi or Yokosuka DOD schools volunteered to participate in a health study. This study was designed to determine if air pollutants from the Shinkampo incinerator were affecting their respiratory health.

There were two primary goals of this study. The first was to determine if there were differences in respiratory health between children who live or go to school at NAF Atsugi and similar children who live at Yokosuka. The second goal was to identify whether the children who live or go to school at Atsugi have more respiratory symptoms on days when they were exposed to higher levels of pollutants from the Shinkampo incinerator. To answer these questions, the volunteer study group consisted of eighty children who resided on base at NAF Atsugi, 17 who lived off base at Atsugi, and 30 who lived at the Yokosuka Naval Base. Each school day during the four-week study, the participants performed two lung function tests during their lunch period. These two tests, called the Forced Expiratory Volume 1 (FEV1) and the Peak Expiratory Flow Rate (PEF), assessed breathing ability as measured by a handheld spirometer. In addition, the participants kept daily records of the number of hours spent outdoors and respiratory and allergy related symptoms. Continuous air monitoring was conducted at the Shirley Lanham School at NAF Atsugi throughout the study period. This monitoring included hourly measurements of wind direction, wind speed, and the amounts of three pollutants present in the air that are known to be associated with respiratory health conditions. These three pollutants are particulate matter less than 10 microns in diameter (PM₁₀), sulfur dioxide and nitrogen oxides.

A review of the data collected during the study period indicates no unusual differences between the respiratory health of the Atsugi children and the children who live at Yokosuka, away from the Shinkampo incinerator. The breathing ability tests indicated that the children studied, including the Atsugi residents, have healthy lung function with average to above average FEV1 and PEF results. The only difference in respiratory symptoms between the groups was a higher number of runny noses reported by Atsugi children.

The results indicated that airborne levels of PM_{10} , sulfur dioxide and nitrogen oxides, measured at the Shirley Lanham School from May 7, 1998 through June 5, 1998 were similar to the annual average air levels of these pollutants in Washington D. C. and New York City. However, care must be taken when reviewing and summarizing the air sampling results used in this study for the following reasons:

- 1. atypical wind patterns may have resulted in lower concentrations of pollutants at the sampling sites;
- 2. the study was of brief duration and measured only a small number of pollutants;
- changes in operations at the Shinkampo incinerator may have also resulted in significantly lower levels of emissions in 1998 compared to prior years; and
- 4. a relationship between wind direction and measured levels of air pollutants from the Shinkampo incinerator could not be determined due to atypical and infrequent winds.

If incinerator pollution increased in the future, there could be a greater impact on Atsugi air quality and respiratory health. If the possible effects of exposure to the emissions of the Shinkampo incinerator continue to be of concern, longer-term respiratory health monitoring of Atsugi residents and air quality testing may be required.

EXECUTIVE SUMMARY

The Shinkampo incineration complex is located about 150 meters south of Naval Air Facility (NAF) Atsugi. Concerns about the possible effects of exposure to its incineration products led to the decision to conduct a series of epidemiological studies on the Atsugi population. This study was designed to investigate the possible impact of emissions from the Shinkampo incinerator on respiratory health. The volunteer study population consisted of 127 5th and 6th grade children who attended the Atsugi or Yokosuka DOD schools. Eighty of the children resided on base at NAF Atsugi, 17 lived off base at Atsugi, and 30 lived at the Yokosuka Naval Base.

Study goals

- 1. Determine if there are differences in respiratory symptoms, Forced Expiratory Volume 1 (FEV1), and Peak Expiratory Flow Rate (PEF) between children who live or go to school at NAF Atsugi and similar children who live at Yokosuka.
- 2. Determine if there is a decrease in FEV1 or PEF and an increase in respiratory symptoms among Atsugi residents and those going to school at Atsugi on days when they are exposed to higher levels of air pollution from the Shinkampo incinerator.

Study Procedures

The study took place between 7 May and 5 June 1998. During the lunch period on school days participants performed a PEF maneuver 3 times into a handheld spirometer, which recorded FEV1 and PEF. The participants also reported the number of hours spent outdoors, and a range of respiratory and pollution related symptoms. Air monitoring at the Shirley Lanham school was conducted throughout the study period and included hourly measurements of wind direction, wind speed, SO_2 , NO_X , NO_2 , NO, and 24 hour means of PM_{10} .

Results

 There was no difference in forced expiratory volume in one second (FEV1) between children living at Atsugi and those living in Yokosuka. Among Atsugi children, the off-base group had higher FEV1s then either the Yokosuka or Atsugi on-base groups. The vast majority of

- children at all three sites had mean FEV1 measurements that were above the predicted values.
- Peak expiratory flows among the on-base Atsugi children were about 14% lower than in the Yokosuka group, while off-base Atsugi children had PEFs that were about 1% below the Yokosuka results. The vast majority of children at all three sites had mean PEF measurements that were above their predicted values
- Children in Atsugi reported more runny noses than those living in Yokosuka, but otherwise did not differ in reported symptoms.
- Among Atsugi participants, there was no relationship between PM₁₀ tertile and FEV1 or PEF.
- There were strong temporal trends in the data reflecting an increase in both FEV1 and PEF
 and a decrease in most reported symptoms over the course of the study.
- There was no consistent relationship between pulmonary function and self-reported asthma and allergy. These findings suggest that these reports may contain some random misclassification.
- The levels of airborne PM₁₀, sulfur dioxide, and nitrogen dioxide that were measured at the Shirley Lanham school (Atsugi) during the study period are comparable to measurements taken in New York City and Washington, D.C. and are within EPA air quality standards for 24 hours average for PM₁₀ and 1hour average for SO2 and NO2.
- Due to atypical and infrequent winds during May 1998, when this study was conducted, no clear relationship between wind direction and the measured levels of airborne pollutants could be established.

Discussion

The reason for the PEF/FEV1 disparity is not known, but some possibilities include random error, a systematic difference in volunteer exhortations between Atsugi and Yokosuka, and a difference between the two measures in their sensitivity to air pollution related pulmonary effects. However, FEV1 is generally considered to be a more reproducible and more sensitive measure.

Care must be taken in generalizing the apparently weak relationship between pollutant concentrations and observed health effects beyond the study period and other specific pollutants. This study was of a relatively brief duration and measured a small number of pollutants.

In addition, changes in operations at the Shinkampo incinerator may have resulted in significantly lower levels of emissions this year compared to prior years. If the levels of incinerator emissions that were present during this study were to increase in the future, it is possible that this would be associated with worsening Atsugi air quality and respiratory health.

If the possible effects of exposure to the emissions of the Shinkampo incinerator continue to be of concern, longer term monitoring of the pulmonary health of Atsugi residents and continued air quality testing are recommended.

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Is Air Pollution from the Shinkampo Incinerator Associated with Adverse

Respiratory Effects among Children at NAF Atsugi?

Deleted: Among

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INTRODUCTION

Background

Naval Air Facility (NAF) Atsugi is located in Ayase City, Japan. It is about 16 kilometers west of Yokohama and about 36 kilometers southwest of Tokyo and is surrounded by residential areas and light industry. In recent years NAF Atsugi's primary mission has been to support fleet aviation units and the Seventh Fleet. The base is now home to a number of U.S. Navy tenant commands and Japanese Marine Self Defense Force (JMSDF) units.

Atsugi's population currently includes about 456 active duty officers, 2950 active duty enlisted personnel, 1664 active duty JMSDF personnel, 1751 family members, 1383 civilian employees, and a few retirees.

Japan's crowded conditions and small size makes it difficult to dispose of municipal wastes in landfills. As a result, seventy five percent of municipal waste is burned in 1,850 public and more than 3,300 privately owned incinerators. There has been increasing public awareness in Japan regarding dioxin and other emissions from these incinerators and their possible health effects. Japanese residents living near incinerators have complained of a variety of health effects, including difficulty breathing, numbness, increased infant mortality, atopic dermatitis, and an increased cancer incidence.¹

The Shinkampo incineration complex is located about 150 meters south of NAF Atsugi. This is a privately owned and operated facility known to receive a great variety of industrial wastes for combustion, including solid waste, plastics, and other unknown wastes. Previous air sampling conducted at NAF Atsugi has indicated the presence of various gases and particulates. Because of health concerns about the impact of the Shinkampo incineration emissions on the air quality of NAF Atsugi, three hundred Atsugi residents attended a 1997 town meeting, which was

held to address health issues related to the Shinkampo incinerator. They expressed concerns regarding possible increased risks of asthma, respiratory infections, headaches, eye irritation, sinus infections, rashes, coughing, and reproductive effects (spontaneous abortion and developmental defects).²

Air sampling conducted in 1997, at NAF Atsugi, indicated that PM_{10} (particulates up to 10 microns in size) levels as high as 340 micrograms per cubic meter (ug/m³) and a 6-hour average of 110 ug/m³ were detected at one housing area. Similar measurements yielded concentrations of 160 and 120, respectively, near the school, and 580 and 240, respectively, at the base fence near the incinerator. By comparison, the EPA has numerical scales for various pollutants for reporting air quality as related to health. The numerical scale used for PM_{10} is as follows:

EPA Health Effect Categories for Various Levels of Particulate Matter Less Than 10 Microns in Diameter (PM_{10})			
Health Effects Category PM ₁₀ Level			
Treatm Effects Category			
Good	Less than 50 ug/m ³		
Moderate	50 ug/m ³ to 150 ug/m ³		
Unhealthful	150 ug/m ³ to 350 ug/m ³		
Very Unhealthful	350 ug/m ³ to 420 ug/m ³		
Warning	420 ug/m ³ to 550ug/m ³		
Emergency	550 ug/m ³ and above		

Effects of Pollutants

The adverse respiratory effects of air pollution have been extensively studied. Increases in levels of ozone, nitrogen dioxide, and sulfur dioxide were found to be significantly associated with increased hospital admissions for cardiac and respiratory diseases.⁴ Longitudinal and cross sectional studies of children under age 15 found that acute respiratory morbidity was increased during periods of increased SO₂.⁵ A Japanese study of adults in the Hiroshima Prefecture yielded similar results, with significant associations between eye irritation, runny nose, coughing and SO₂.⁶

Non-specific urban air pollution has been shown to be associated with an increase in respiratory symptoms in children, including coughing, rhinitis, pneumonia, asthma, and respiratory infections. Indoor pollution is also an important cause of respiratory problems in children. Coughing, wheezing, pneumonia, bronchitis, and asthma were all more common among children living in damp houses. Mite dust was also a significant cause of respiratory symptoms.

Concerns about the possible effects of exposure to the incineration products of the Shinkampo incinerator led to the decision to conduct a series of epidemiological studies of the Atsugi population and a comprehensive health risk assessment. This study is a component of that series. It is designed to investigate the possible impact of emissions from the Shinkampo incinerator on the respiratory health of 5th and 6th grade students at Atsugi.

Study goals

- 1. Determine if there are differences in respiratory symptoms, Forced Expiratory Volume 1 (FEV1), and Peak Expiratory Flow Rate (PEF) between children who live or go to school at NAF Atsugi and similar children who live at Yokosuka.
- 2. Determine if there is a decrease in FEV1 or PEF and an increase in respiratory symptoms among Atsugi residents and those going to school at Atsugi on days when they are exposed to higher levels of air pollution from the Shinkampo incinerator.

MATERIALS AND METHODS

Population

The study population consisted of 127 volunteer 5^{th} and 6^{th} grade children whose parents are either civilian or military personnel. Eighty of the children resided on base at NAF Atsugi, 17 lived off base at Atsugi, and 30 lived at Yokosuka Naval Base.

The 97 children from Atsugi comprised the exposed groups, while the 30 children from Yokosuka represented the control population. There were another two children at Atsugi and one at Yokosuka who provided informed consent but did not participate in the study. Children were chosen for inclusion in this study because their airways tend to be more sensitive to

atmospheric pollutants and because child health is a major concern of the Atsugi active duty population

Recruitment and Eligibility

All 5th and 6th grade children at the Atsugi and Yokosuka DOD schools were eligible to participate. Permission to conduct the study was obtained from Commander Naval Forces Japan,, the commanding officers of NAF Atsugi and Yokosuka Naval Base, and the principals of the participating DOD schools. All eligible children were given letters to carry home to their parents describing the study and asking for written permission for their children to participate in the school based study. The letters included contact names and telephone numbers in case the parents had any questions regarding the study or if they did not want their children to volunteer for the study. A meeting was also held at the Shirley Lanham School on the Atsugi base to answer any questions that potential participants or their parents might have had.

Written informed consent was obtained from all participants and their parents or guardians. Each participant and their parents were also asked to complete and return a brief questionnaire providing demographic information, data on allergies, and possible household sources of pollutants or allergens. This study was approved by both the Naval Health Research Center Institutional Review Board and their Committee for the Protection of Human Subjects.

Training

Before data collection began every participant attended an assembly at their school where they were shown the peak flow meter and the mechanics of its use was explained and demonstrated. They were also shown how to fill out their daily logs. There was also a 1 hour training session for volunteer supervisors at each school where they received similar training. At the Shirley Lanham school the first day of the study was used as a training session and results from that day were not used in the data analysis. There was no corresponding training day at Yokosuka since they were not able to begin data collection until 12 May 1998.

Study Procedures

The study took place over a four-week period, from 7 May to 5 June 1998. During the lunch period on school days participants performed a PEF maneuver 3 times into an Airwatch Handheld Spirometer (Enact Health Management Systems) in the school cafeteria. This instrument recorded FEV1 and PEF. The spirometer used interchangeable mouthpieces and each participant had their own, with their name written on it in indelible ink. The mouthpieces were kept in individual sealed plastic bags. These in turn were kept in plastic boxes, with one box for each class. Every day participants found their mouthpiece and removed it from the bag for attachment to the spirometer.

The participants also turned in their daily data forms during the lunch period. These forms reported the number of hours spent outdoors, respiratory and pollution related symptoms, including coughing during the day, coughing at night, trouble breathing, feeling bad, runny nose, cold, headache, and irritated eyes. A daily symptom score was calculated from these responses. Participants were also asked to record Queries regarding eye irritation and headaches were included because Atsugi residents had previously reported frequent occurrence of these symptoms..

At the conclusion of the study, each participating child was given a non-monetary reward (MWR coupons) worth \$30.

Exposure Assessment

Air monitoring at the Shirley Lanham school was conducted by Radian International LLC (Austin, Texas) and included hourly measurements of wind direction, wind speed, SO₂, NO_x, NO₂, NO, and 24 hour means of PM₁₀. A Grasby-Anderson volumetric controlled PM High Volume air sampler with a 1.13 cubic meter per minute flow rate was used to measure particulates. The sampler's stagnation pressure, ambient temperature and the barometric pressure were recorded before and after each sampling run to determine flow rates from reference tables. Particulates were collected on equilibrated, pre-weighed 8x10 inch quartz fiber filters. After the completion of each sampling run, the filters were recovered and returned to the U.S. for filter equilibration and mass determination.

 SO_2 was monitored with an Advanced Pollution Instrumentation (API) Model 100 Fluorescent SO_2 analyzer. This instrument continuously measures ambient SO_2 concentrations by exposing the molecules to ultraviolet light in the reaction chamber of the analyzer. As they

are excited and return to their ground state they emit a characteristic fluorescence which is detected and converted into an analog signal. Nitrogen Oxides (NO_X and NO) were measured with an API Model 200A Analyzer that measures the light intensity of the chemiluminescent gas phase reaction of NO and O_3 . NO_2 concentration is determined by calculation based on the difference between NO_X and NO values.

Historical data indicated that the wind blows from the incinerator to NAF Atsugi as much as 50% of the time from the south-southeast, south, and south-southwest direction. We planned to determine the contribution of the Shinkampo incinerator to the observed pollution levels when the prevailing winds were within 45 degrees of the school. Days that met this condition during more then 25% of the measured hours were to be classified as "exposed" in a dichotomous variable. However, this classification could not be pursued because the wind did not blow emissions from the incinerator to the base as much as anticipated by historical data.

We also planned to classify pollution as medium when the 24 hour mean concentration of total particulates was 50 ug/m^3 or greater or the 24 hour mean concentration of SO_2 was 80 ug/m^3 or greater. Similarly, the threshold for a high pollution classification was to be 150 ug/m^3 for total particulates or 365 ug/m^3 of SO_2 . However this classification scheme had to be abandoned due to the observed levels of air pollution, which were lower then expected.

It was not possible to conduct air pollution measurements at the control site (Yokosuka).

Data Analysis

Data forms were designed for automatic scanning using the Teleform (Cardiff Software, San Marcos, California) software package. Since necessary hardware did not arrive in time the actual data entry was done using a combination of scanning and manual entry, with manual error checking. The analyses were conducted using the SPSS for Windows (SPSS, Inc. Chicago, Illinois) and STATA (Stata Corporation, College Station, Texas) statistical software packages.

Random effects linear regression models were used to assess differences related to site and to adjust for potential confounding variables and the repeated measure nature of the data. The exposure variable (location) was forced into the model and confounding variables were included if they were statistically significant, or if their removal caused an approximate 10% change in the exposure estimate of the exposure variable. Two similar models were presented so that the effects of different covariates could be observed.

The possible association between PM_{10} and respiratory function among Atsugi participants was assessed using a random effects linear regression model where daily variations from the individual participants' overall mean was the dependent variable. Unconditional logistic regression models were used to assess the impact of location on symptom occurrence.

Potential confounding variables included age, weight, height, sex, time at Atsugi, time in Japan, exposure to environmental tobacco smoke, hours spent outside, air conditioning, cooking with a gas stove, the presence of a pet in the home, having a cold, feeling bad, allergies, and prior treatment for asthma.

Daily reported respiratory symptoms were scored as 0 to 6, with one point each being given for reported eye pain, headache, coughing last night, coughing today, runny nose, and feeling bad. Colds were scored on a 1-3 scale, representing "no", "unsure", and "yes".

RESULTS

Demographics

Table 1 describes the demographics of the three study groups. The age difference reflects the fact that the Atsugi groups included both 5th and 6th grades while the Yokosuka group was all 5th grade students. Those living off base at Atsugi averaged less time stationed in Japan, used less air conditioning, and used more gas stoves than the other groups. A higher percentage of the Yokosuka group reported a prior diagnosis of asthma and treatment for allergies. However, as will be seen, there was little apparent correlation between self-reported allergy or asthma status and study outcomes.

Wind Direction and Pollutant Levels

Due to atypical and infrequent winds during May 1998 when this study was conducted, the relationship between wind direction and the measured levels of SO_2 , NO_X , NO_2 , or NO could

not be adequately assessed. The levels of PM_{10} , SO_2 , and NO_2 obtained at the Shirley Lanham School were compared with EPA measurements made during 1997 in Washington, D.C. and Manhattan (Table 2). Atsugi mean PM_{10} levels were comparable to Manhattan levels while the study maximum level (104 ug/m³) was higher then the 1997 Manhattan maximum (90 ug/m³).

Atsugi PM_{10} levels were also noticeably higher than Washington D.C. measurements. However, both SO_2 and NO_2 levels at Atsugi were much lower than were found in Washington, D.C. or New York and met the applicable EPA standards. It should be noted that these levels were based on a one-month average for NAF Atsugi whereas the statistics for the U. S. cities are annual averages.

Pulmonary Function - FEV1

The distribution of mean forced expiratory volume in one second (FEV1) and height for each participant is shown in Figure 1. Figure 2 illustrates the mean FEV1 and the ratio of mean to predicted FEV1s, where the reference values are those of Dickman. The horizontal dotted line represents a predicted/actual ratio of 0.8, which is the standard cutoff for diagnosing respiratory problems. Two Atsugi children fell below this level. The mean FEV1 of the vast majority of participants at all three study sites were well above their predicted values, although those of the on base group (1.42) were lower then those of the off base (1.68) and Yokosuka (1.66) groups (Figure 3).

There was a noticeable "learning curve" or trend towards higher FEV1 results over the course of the study. This encompassed a range of about 0.2 liters per second (Figure 4). However there was very little relationship between participants' gender and FEV1. Atsugi males recorded somewhat higher FEV1s than females, while at Yokosuka the females had slightly higher results (Figure 5).

Each study day participating children reported their cold status as "yes", "no", or "unsure". As seen in Figure 6, there was no correlation between cold status and FEV1 for either the on base or Yokosuka groups, with Yokosuka children actually having slightly higher FEV1s on the days when they reported having a cold. This pattern was different in the off base group, with the "no" cold group posting a much higher mean FEV1 then any of the other groups.

The mean daily FEV1 values were 2.45 for on base children, 2.82 for off base, and 2.51 for Yokosuka (Table 3). This pattern was also found in the linear regression models. The results of two representative models are presented in Table 4 and 5. After adjustment for potential confounders (including the learning effect and the repeated measure nature of the study, centered height, centered weight, and age), the predicted differences in FEV1 associated with residence

were very similar to the unadjusted results. Neither of these models demonstrated significant differences between FEV1s at the 3 study sites, although the on base-off base comparison for Model 1 (Table 4) approached significance.

Two models were presented to show the sensitivity of the relationship between site and pulmonary function to model selection in situations where that relationship is marginally significant.

Pulmonary Function - Peak Expiratory Flow

Figure 7 illustrates the relationship between mean peak flow and height for participants at each site. The relationship between observed and predicted values is shown in Figures 8 and 9. As was the case with FEV1 measurements, most participants at each site had peak flows above their predicted values. There were 3 on base children whose mean peak flows fell below 80% of the predicted value. There was also a strong learning curve effect associated with peak flow measurements over the course of the study (Figure 10). Girls at Yokosuka and off base Atsugi had slightly higher peak flows than boys (Figure 11). The relationship between cold status and peak flow is shown in Figure 12.

The on base participants had a mean peak flow that was 46.5 ml lower than that of the Yokosuka group, and 43.1 ml lower than the off base group (Table 6). Using the same two models as were employed with the FEV1 results (Tables 7 and 8), all of the comparisons achieved statistical significance.

Particulate Levels and Pulmonary Function

As seen in Figures 13 and 14, there was no evident relationship between 24-hour PM_{10} level and either FEV1 or peak flow among Atsugi on and off base residents. This remained true when the pulmonary function tests were tested against PM_{10} tertile and when daily deviations from individual mean PEF and FEV1 were regressed against 24 hour PM_{10} and potential confounders (Table 9).

Reported Symptoms

Figures 15 and 16 show that reported colds, runny noses, coughing (yesterday and today) all decreased substantially over the course of the study. Headache and eye pain also decreased, although the trend was less striking (Figure 17).

Figure 18 shows that when reported colds and respiratory symptoms were pooled and compared by location, Atsugi on base children reported more symptoms overall than either Atsugi off base children or children in Yokosuka. This difference was largely due to more reports of runny nose among children at Atsugi (Figure 19). There was no difference between on base and Yokosuka children in mean cold scores, with off base children reporting higher scores.

Tables 10, 11, 12, and 13 portray a series of logistic regression analyses comparing presence of symptoms and residence. Children at Atsugi reported more runny noses than those at Yokosuka, while those on base had lower cold scores than those living off base. Other than runny nose and cold symptoms, none of these differences achieved statistical significance.

Particulate Levels and Symptoms

Tables 14 and 15 show that there was no correlation between tertile of PM_{10} levels and the range of study symptoms among Atsugi on and off base residents.

Pulmonary Function and Self Reported Allergy and Asthma Status

As is seen in Tables 16 and 17, the mean pooled unadjusted FEV1 and PEF values of children at the three study sites were unrelated to reported asthma status. There was a decrease in unadjusted peak flow and FEV1 among Yokosuka children who reported allergies. There was also a large difference in the off base population, but this was based on 1 child.

DISCUSSION

In this study we were able to prospectively gather data on FEV1, peak expiratory flow, and respiratory symptoms from three populations of normal (primarily non-asthmatic) children while

simultaneously monitoring air pollution levels. This design provided the best available method to compare these populations and to investigate the possible effects of the Atsugi environment. The primary findings of this study were that:

- Due to atypical and infrequent winds during May 1998, no clear relationship between wind direction and levels of airborne pollutants could be established.
- The observed levels of airborne particulates, sulfur dioxide, and nitrogen dioxide that were found at Atsugi are comparable to measurements taken in New York City and Washington, D.C. and are within EPA air quality standards. However, it should be noted that air quality data for NAF Atsugi were based on a one month average and for the U.S. cities they were based on a yearly average.
- There was no difference in forced expiratory volume in one second (FEV1) between children living at Atsugi and those living in Yokosuka. Among those living at Atsugi, the FEV1 was lower in children who live on base then in those who live off base. The vast majority of children at all three sites had mean FEV1 measurements that were above the predicted values, suggesting that their environment is not having a deleterious effect on their pulmonary function.
- Pulmonary peak flows in children who live at Atsugi are lower than in those who live at Yokosuka and, among children at Atsugi, those living on base have lower peak flows than those living off base.
- Children in Atsugi reported more runny noses than those living in Yokosuka, but otherwise did not differ in reported symptoms.
- Neither symptoms nor pulmonary flow measurements were related to observed levels of airborne particulates.
- There were strong temporal trends in the data reflecting an increase in both FEV1 and PEF and a decrease in most reported symptoms over the course of the study.
- There was no consistent relationship between pulmonary function and self-reported asthma and allergy. These findings suggest that these reports may contain some random misclassification.

The differences in unadjusted mean FEV1s and PEFs between on base and off base participants were very similar, at 15.1% and 13.25% respectively. However, there was a large and unexpected disparity between the FEV1 and PEF results in the on base-Yokosuka comparison. Mean Yokosuka PEFs were 14.3% higher then those of Atsugi on base participants while the difference in FEV1s was only 2.4%... However, FEV1 is generally considered to be a more reproducible measure and has been shown to be more sensitive in identifying reactions to inhalation challenges. ¹²

The reason for the PEF/FEV1 disparity is not known, but some possibilities include random error, a systematic difference in volunteer exhortations between Atsugi and Yokosuka, and a difference between the two measures in their sensitivity to air pollution-related pulmonary effects. Since FEV1 has been shown to be more sensitive than PEF, this explanation is unlikely to account for the disparity.

Although FEV1 is effort dependent, it is much less so then PEF.¹³ There was one group of volunteer supervisors who worked at Atsugi and a separate group at Yokosuka. Although both groups received similar training, the extent of the training was limited (approximately 1 hour) and it was not possible to standardize the volunteers. The difficulty and time involved in traveling between Yokosuka and Atsugi (due to traffic congestion) also prevented us from rotating volunteers between the two sites. Empirically, it appeared that the Yokosuka volunteers encouraged the study participants to blow harder to a greater extent then the Atsugi volunteers did. The actual impact of this is unknown but it is possible that at least some of the PEF/FEV1 differences are related to this observation.

Despite the variations in PEF between Atsugi and Yokosuka, the vast majority of children at all study sites achieved mean FEV1 and PEF measurements that were greater than the predicted values for the general population. The reasons for this may include the Navy's overseas screening, better access to medical care than the general U.S. population enjoys, a mild climate, and abundant recreational facilities and organized activities. All of these factors could result in an unusually healthy population.

Both pulmonary function tests and reported symptoms exhibited strong time trends during this study. The increase in FEV1 and PEF is consistent with a "learning curve" effect. The reasons for the decrease in symptoms over time are more problematic. Possible causes include

more accurate reporting as the children became more comfortable with the study and a strong "end of school year" effect.

During this study we observed 24-hour PM₁₀ levels in the range of 21.6-104.8 ug/m³. While these levels may have resulted in some decreases in peak flow the effects were too small to be detected. Although we also observed essentially no relationship between PM₁₀ levels and reported symptoms or respiratory function at the relatively low levels of particulates detected during this study, the deleterious respiratory effects of PM₁₀ on PEFs of children have been well described by Dockery and Pope. ¹⁴ In addition, a study of 155 asthmatic children over a 3-year period found a significant relationship between PM₁₀ and PEF. This study also found that pollution-related PEF changes in children were larger then those of adults. ¹⁵ An Italian study found that decreases in peak flow were associated with particulates and NO₂. ¹⁶ Similar PEF changes have also been observed in non-asthmatic children. ¹⁷ However, in a study of the possible effects of an incinerator, Shy et al. also failed to detect a relationship between PEF rates in adults and daily variations in PM₁₀ during a 35 day study where PM₁₀ ranged from 18-37ug/m³. ¹⁸

Mr. E. Anderson of Radian International has suggested that changes in materials burned, variations in burning patterns according to wind direction, and more extensive operation of various anti-pollution devices at the Shinkampo incinerator has resulted in significantly lower levels of emissions this year compared to prior years.¹⁹ He has hypothesized that these actions may represent the incinerator operator's response to the Navy's ongoing emissions monitoring. Figure 20, which is adapted from the "NAF Atsugi, Japan Ambient Air Monitoring Summary June 1998" report by Radian International seems to lend support to this view. Measured PM₁₀ levels fell dramatically after the first week of monitoring and remained at a lower level throughout the rest of the monitoring period. Figure 20 also shows that PM₁₀ levels were not lower on a day when the incinerator was not in operation, which is consistent with our finding of a poor correlation between emissions and PM₁₀ levels.

If the relatively low levels of incinerator emissions recorded during this study were to return to their prior (higher) levels in the future it is very possible that this would be associated with a greater effect on respiratory function and reported symptoms. If this occurred the incinerator emissions could also have a greater impact on overall Atsugi air quality. Weather patterns and atmospheric inversions tend to increase air pollution levels in the Atsugi area during

the summer months. It was not practical to conduct this study after school ended, but it is possible that the observed effects would have been more severe later in the summer.

In general, the levels of the specific pollutants measured during this study appear to be consistent with an urban environment, and the measurements were similar to those observed in Manhattan and Washington, D.C.

It is not possible to quantify the contribution of the incinerator to the air pollution at Atsugi using only the data gathered in this study. If data on airborne pollutant levels, wind speed and wind direction are combined with quantitative data on incinerator emissions, then the impact of the incinerator at the monitoring site may be calculated using chemical mass balance receptor modeling. Unfortunately, it is impossible to remotely gather stack emissions data, and Shinkampo will not allow sampling on their property.

Every epidemiological study design suffers from at least some weaknesses that must temper our interpretation of the results. In addition to the limitations already discussed, the determination of exposure assessment in this type of study is of particular concern.

Our air monitoring station was placed at the Shirley Lanham School since this is where the students spend most of their day. However there are many factors influencing each student's individual exposure beyond those which we were able to measure.

The number of hours spent outdoors influences exposure since indoor pollutant concentrations are sometimes lower²⁰. This should be especially true at Atsugi, where housing units have been provided with air filtration devices. The daily symptom form contained a question asking for the number of hours spent outside. However this was not a significant predictor of either FEV1 or PEF and was not included in the final models.

Variations in wind speed and direction combined with the differing locations of the students' homes all increase the potential for misclassification of exposure to incinerator emissions. We were not able to incorporate the location of residences into this study beyond classifying them as on base or off base. There are a number of pollution point sources in the Atsugi area, and without setting up a monitoring station at each residence it would be impossible to estimate the contribution of the Shinkampo incinerator to the pollution at each house.

Indoor air quality can also be a major contributor to overall exposure and can vary greatly, depending on the presence of mold, dust, mites, pets, a smoker in the house, the use of air conditioning or a gas stove, and the ventilation of the building²¹. In addition to variations in

exposure, there are also variations in each individual's response to different pollutants. It is worthwhile to note that in general, random exposure misclassification tends to bias results towards the null.

In addition, we were unable to monitor the air quality at Yokosuka. This makes it impossible to attribute the observed differences in symptoms and PEFs to variations in pollution levels between Atsugi and Yokosuka.

Most of the Atsugi and Yokosuka participants had either one or two Asian parents. The race of the participants was not recorded and was not included in the analysis, primarily because we thought it was inappropriate to query the children about their parents. It is very unlikely that this omission could cause appreciable bias in our results.

The participants in this study were volunteers. While the offer of non-monetary rewards and an ice cream party was certainly a motivating factor, we don't know why some children chose to participate and others didn't. Although improbable, it is possible that our sample was in some way unrepresentative of the general population of children at Atsugi, and that responses of our participants materially differed from that of a different sample of children.

There is also the possibility that differences between participants at each site caused confounding that could not be properly adjusted. However the demographics of each group were similar. This, combined with our understanding of physiologic factors related to pulmonary function and the fact that observed PEF and FEV1 differences did not change substantially after adjustment, suggests that confounding was not a major issue.

If the effects of exposure to the emissions of the Shinkampo incinerator continue to be a source of concern, monitoring of the health of Atsugi residents at other times of the year and continued monitoring of the incinerator are recommended. More extensive environmental monitoring and modeling (which is now ongoing) should also further clarify the contribution of the incinerator to the air quality at NAF Atsugi.

REFERENCES

- 1. Mutsuko M, Tet-Sieu C, Japan is alarmed by excessive levels of dioxin. Asia Week, May 30, 1997.
- 2. Personal communication, LCDR C. Waggoner, NAF Atsugi.
- 3. Measuring Air Quality: The pollution standard index, EPA 451/K-94-001, July, 1996.
- 4. Burnett R, Cakmak S, Brook J, Krewski D, The role of particulate size and chemistry in the association between summertime ambient air pollution and hospitalization for cardiorespiratory diseases. Environmental Health Perspectives. 105:614-620, 1997.
- Pinter A, Rudnai P, Sarkany E, Goczan M, Paldy A, Air pollution and children's respiratory morbidity in the Tata area, Hungary. Central European Journal of Public Health 4: Supplement 17-20, 1996.
- 6. Setiani O, Trend of air pollution and it's effect on human health in Hiroshima prefecture. Hiroshima Journal of Medical Science. 45:2, 43-50, 1996.
- 7. Corbo G, Forastiere F, Dell Orco V, et.al., Effects of environment on atopic status and respiratory disorders in children. Journal of Allergy and Clinical Immunology. 92:616-23, 1993.
- 8. Yang CY, Chiu JF, Chiu HF, Kao WY, Damp housing conditions and respiratory symptoms in primary school children. Pediatric Pulmonology. 24(2):73-7, 1997.
- Leung R, Ho P, Lam CW, Lai CK, Sensitization to inhaled allergens as a risk factor for asthma and allergic diseases in Chinese population. Journal of Allergy and Clinical Immunology. 99(5):594-9, 1997.
- Rosner B, Munoz A, Conditional linear models for longitudinal data, in Dwyer J, Feinleib M, Lippert P, Hoffmeister H. "Statistical models for longitudinal studies of health" Oxford University Press 1992.
- 11. Dickman ML, Schmidt CD, Gardner RM, Spirometric standard for normal children and adolescents, age 5 years through 18 years. Am Rev Respir Dis. 104:680-687, 1971.
- Dahut P, Rachiele A, Martin R, Malo J, Histamine dose-response curves in asthma: reproducibility and sensitivity of different indices to assess response. Thorax. 38:516-22, 1983.

- 13. Berube D, Cartier A, L'Archeveque J, Ghezzo H, Malo J, Comparison of peak expiratory flow rate and FEV 1 in assessing bronchomotor tone after challenges with occupational sensitizers. Chest 99:831-836, 1991.
- 14. Peters A, Goldstein I, Acute effects of exposure to high levels of air pollution in eastern Europe. American Journal of Epidemiology. 144:570-81, 1996.
- 15. Baldacci S, Carrozzi L, Viegi G, Giunini C, Assessment of respiratory effect of air pollution: study design on general population samples. Journal of Environmental Pathology, Toxicology and Oncology. 16(2&3):77-83, 1997.
- 16. Personal communication, Professor Carl Shy, University of North Carolina.
- 17. Dockery DW, Pope CA, Acute respiratory effects of particulate air pollution. Atmos Environ. 21:407-418, 1987.
- 18. Shy C, Degnan D, Fox D, Mukergee S, et. al., Do waste incinerators induce adverse respiratory effects? an air quality and epidemiological study of six communities. Environmental Health Perspectives. 103:714-724, 1995.
- 19. Personal communication, Eric Anderson, Radian International.
- 20. Lewis CW, Sources of air pollutants indoors: VOC and fine particulate species. J Expos Anal Environ Epidemiol. 1:31-44, 1991.
- 21. Etzel RA, Indoor air pollution and childhood asthma: effective environmental interventions. Environmental Health Perspectives. 103 Suppl 6:55-8, 1995.

APPENDIX

List Of Tables

Table 1. Basic Demographics Of Atsugi And Yokosuka Study Participants

Variable	Atsugi On Base	Atsugi Off Base	Yokosuka
Number of participants	80	17	30
Mean samples per participant	18.7	17.4	12.9
Percent male	43.8%	35.3%	33.3%
Mean age (months)	137.3 (7.7)	139.1 (10.3)	131.9 (5.6)
Mean height (inches)	57.3 (3.4)	58.1 (4.1)	56.9 (3.9)
Mean weight (pounds)	87.7 (22.4)	85.9 (18.4)	89.3 (20.3)
Mean months in Japan	48.2 (30.1)	23.5 (35.9)	34.0 (24.2)
Treatment for allergies or hay fever	27.3%	5.9%	33.3%
Treated for asthma	9.7%	0%	20%
Dog or cat in the house	26.6%	23.5%	20%
Air conditioning in the house	98.7%	70.6%	96.7%
Smoker in the house	27.8%	35.3%	33.3%
Gas stove in kitchen	5.0%	82.4%	15.4%

Table 2. Comparison of Airborne Pollutant Levels Measured at the Shirley Lanham School to 1997 Levels in Washington D.C. and New York City

	PM10 ug/m ³	SO2 ppb	NO2 ppb
Atsugi 4 Week Mean*	48.6	2.90	18.8
• 1 Hour Max.	-	23	65
• 24 Hour Max.	104.8	-	-
New York Annual Mean+	51.1	12	40
• 1 Hour Max.		158	128
• 24 Hour Max.	90	-	-
D.C. Annual Mean+	23.3	7	25
• 1 Hour Max.	-	87	102
• 24 Hour Max.	49	-	-
EPA 24 hour Standard&	150	140	-
EPA Annual Standard&	50	30	53

^{*} Data collected between 5 May and 7 June, 1998

^{+ 1997} EPA data from Washington D.C. and New York City monitoring stations 19, 20.

[&]amp; From the July, 1997 National Ambient Air Quality Standards

Figure 1. Mean of Maximum Daily FEV1s by Height and Residence

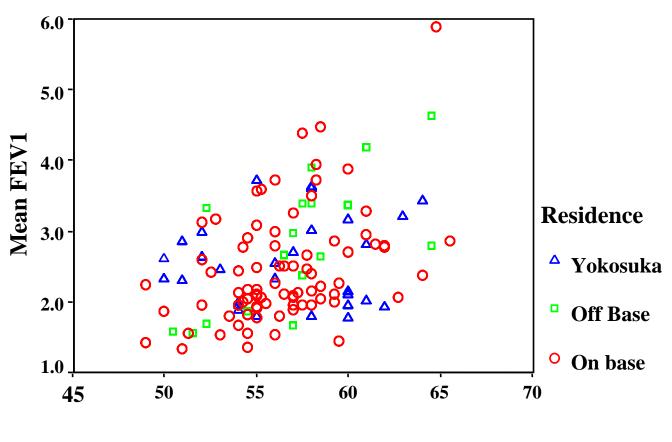


Figure 2. The Ratio of Observed to Predicted Mean FEV1 For Each Study Participant

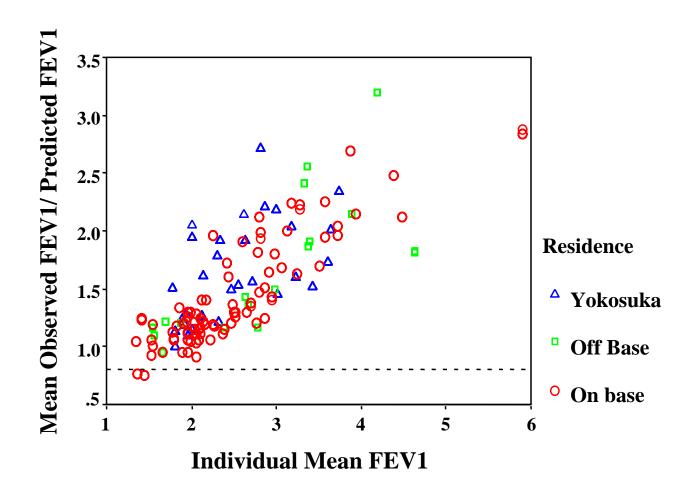


Figure 3. The Ratio of Observed to Predicted Mean FEV1 by Study Site

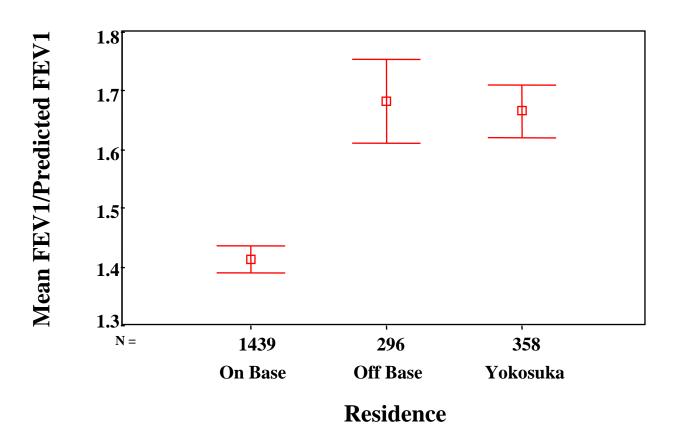


Figure 4. Temporal Trends in Daily Maximum FEV1 Versus Individual Means

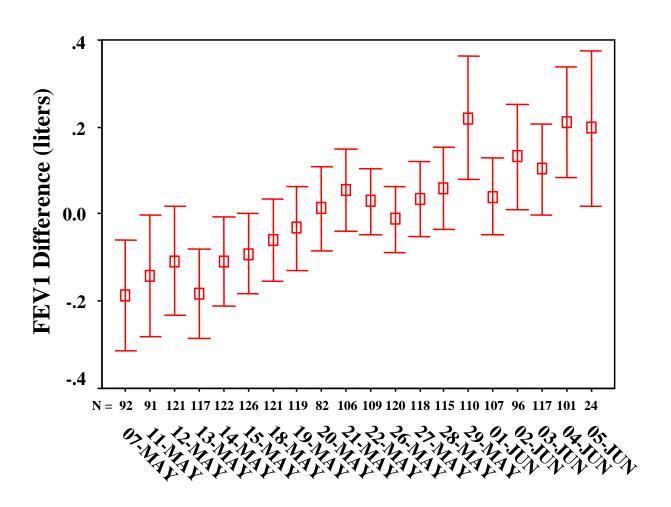


Figure 5. Maximum Daily FEV1 By Sex and Residence

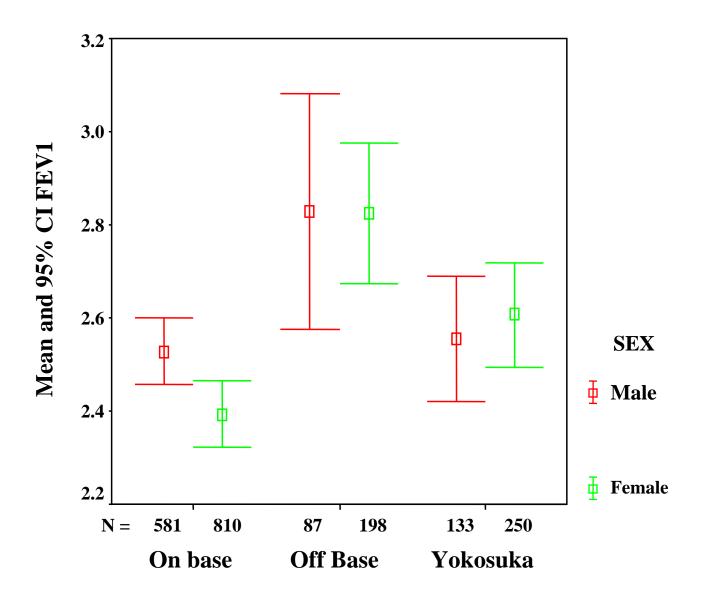


Figure 6. Maximum Daily FEV1 By Cold Status and Residence

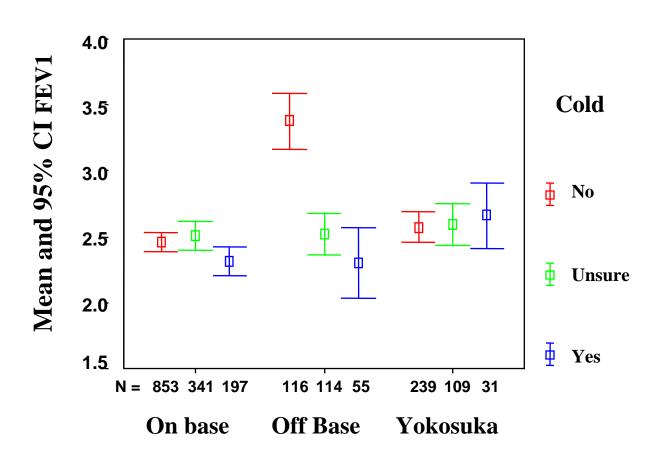


Table 3. Summary Measures of Daily Maximum FEV1 and Residence

Group	Mean Daily	Number of	Minimum FEV1	Maximum FEV1
	FEV1+	Observations		
On base	2.45	1391	0.67	7.94
Off base	2.82	285	1.25	6.28
All Atsugi	2.51	1676	0.67	7.94
Yokosuka	2.51	438	0.55	5.36

⁺ Unadjusted mean of all observations

Table 4. Daily Maximum FEV1 and Residence, Model 1+

Comparison	Mean FEV1	Adjusted FEV1 Difference	Significance	R^2++
	Difference*	and	Level&	
		95% CI**		
On base vs Yokosuka	0.06	0.12 (-0.22 - 0.47)	0.48	0.22
On base vs off base	0.37	0.39 (-0.10 – 0.78)	0.056	0.31
All Atsugi vs Yokosuka	0	0.071 (-0.28 - 0.42)	0.69	0.23

⁺ Random effects repeated measures linear regression model, adjusted for day, study week, individual, height, weight, age, months in Japan, and exposure to smokers.

Table 5. Daily Maximum FEV1 and Residence, Model 2+

Comparison	Mean FEV1	Adjusted FEV1 Difference	Significance	R^2++
	Difference*	and	Level&	
		95% CI**		
On base vs Yokosuka	0.06	0.11 (-0.22 - 0.50)	0.52	0.18
On base vs Off base	0.37	0.32 (-0.063 - 0.70)	0.10	0.29
All Atsugi vs Yokosuka	0	0.084 (-0.25 - 0.42)	0.62	0.22

⁺ Random effects repeated measures linear regression model, adjusted for day, study week, individual, height, weight, age, months in Japan, and having a cold.

^{*} From all observations at each residence (on base, off base, all Atsugi, Yokosuka).

^{**}From regression model.

[&]amp; P value from regression model.

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Figure 7. Mean of Daily Maximum Peak Flows by Height and Residence

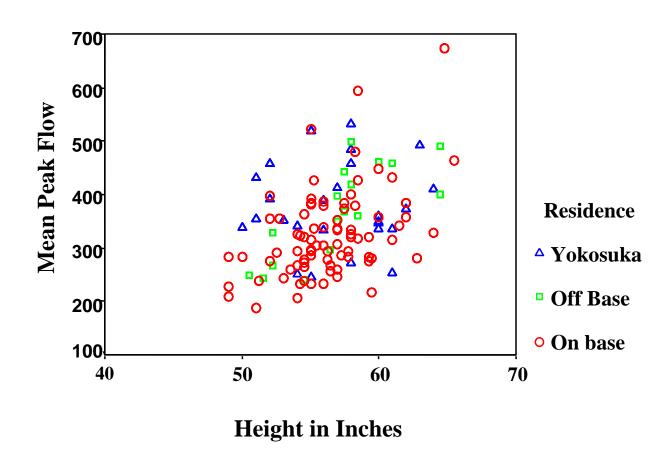


Figure 8. The Ratio of Observed to Predicted Mean Peak Flow For Each Study Participant

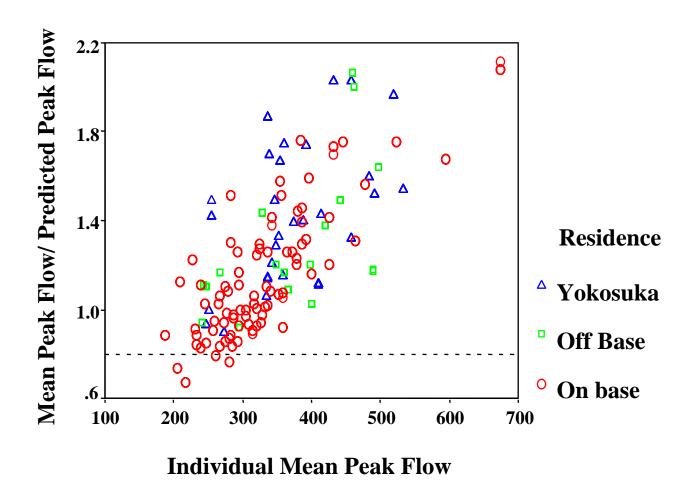


Figure 9. The Ratio of Observed to Predicted Mean Peak Flow by Study Site

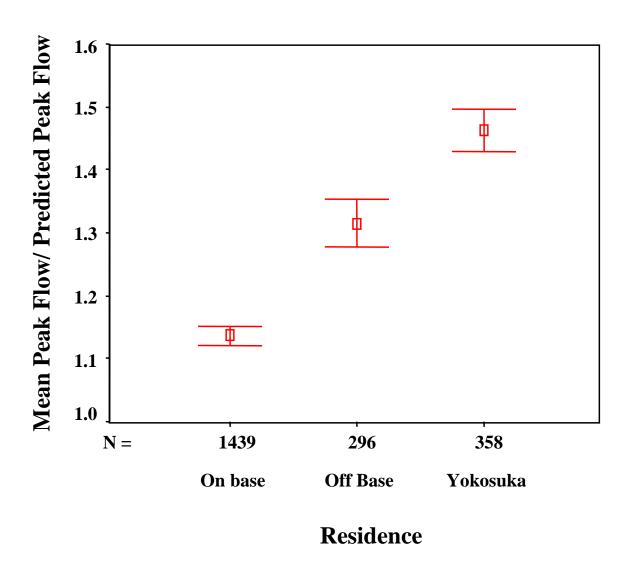


Figure 10. Temporal Trends in Daily Maximum Peak Flow Versus Individual Means

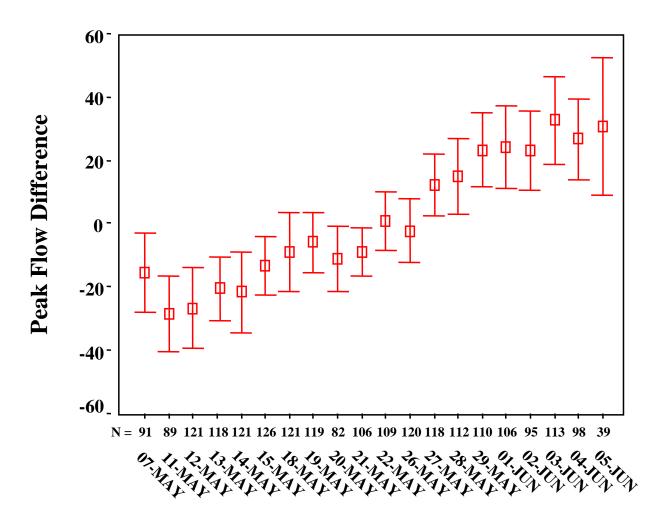


Figure 11. Maximum Daily Peak Flow By Sex and Residence

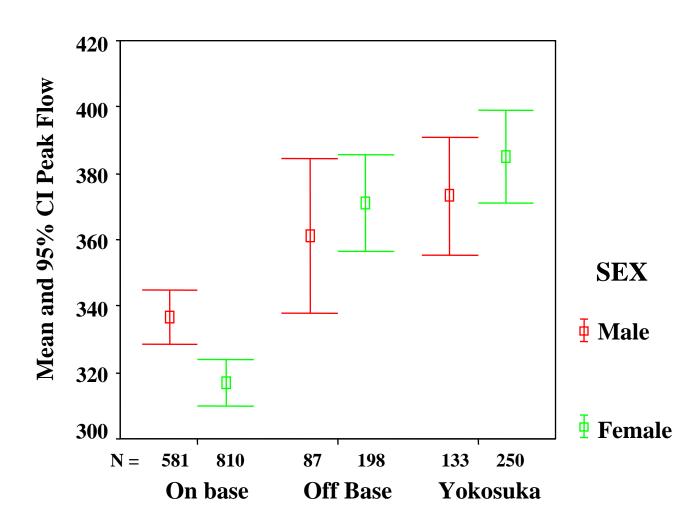


Figure 12. Maximum Daily Peak Flow By Cold Status and Residence

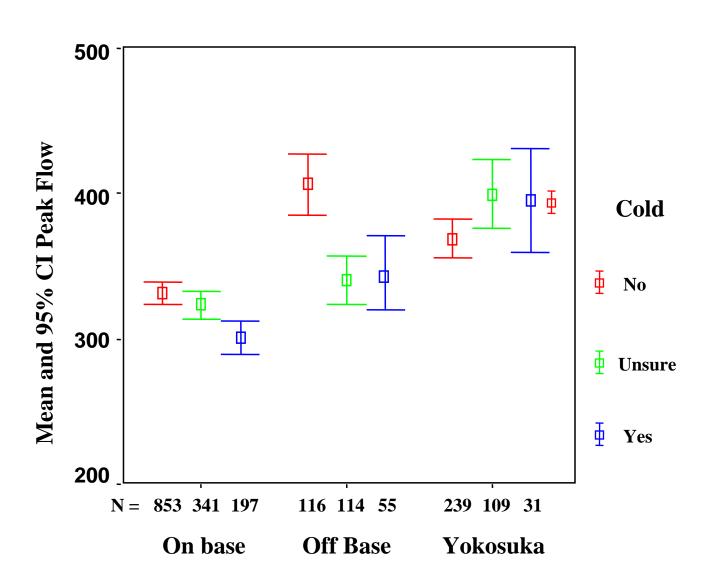


Table 6. Summary Measures of Daily Maximum Peak Flow and Residence

Group	Mean Daily Peak	Number of	Minimum Peak	Maximum Peak
	Flow+	Observations	Flow	Flow
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Off base	368.2	285	172	678
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Yokosuka	371.6	438	148	794

⁺ Unadjusted mean of all observations

Table 7. The Relationship Between Daily Maximum Peak Flow and Residence, Model 1+

Comparison	Mean PEF	Adjusted PEF Difference	Significance	\mathbb{R}^2++
	Difference*	and	Level&	
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On base vs off base	43.1	38.0 (0.5-75.5)	0.047	0.41
All Atsugi vs Yokosuka	39.2	43.6 (7.9-79.4)	0.017	0.30

⁺ Random effects repeated measures linear regression model, adjusted for day, study week, individual, height, weight, age, months in Japan, and exposure to smokers.

Table 8. The Relationship Between Daily Maximum Peak Flow and Residence, Model 2+

•	_			
Comparison	Mean PEF	Adjusted PEF Difference	P Significance	\mathbb{R}^2++
	Difference*	and	Level&	
		95% CI**		
On base vs Yokosuka	46.5	50.8 (16.0-85.7)	0.004	.28
On base vs off base	43.1	37.7 (2.3-73.2)	0.037	.40
All Atsugi vs Yokosuka	39.2	47.9 (14.1-81.8)	0.005	.30

⁺ Random effects repeated measures linear regression model, adjusted for day, study week, individual, height, weight, age, and having a cold.

^{*} From all observations at each residence (on base, off base, all Atsugi, Yokosuka)

^{**}From regression model.

[&]amp; P value from regression model.

⁺⁺R² between groups.

^{*} From all observations at each residence (on base, off base, all Atsugi, Yokosuka)

^{**}From regression model.

[&]amp; P value from regression model.

⁺⁺R² between groups.

Figure 13. Mean Daily Maximum FEV1 by 24 Hour Mean PM10

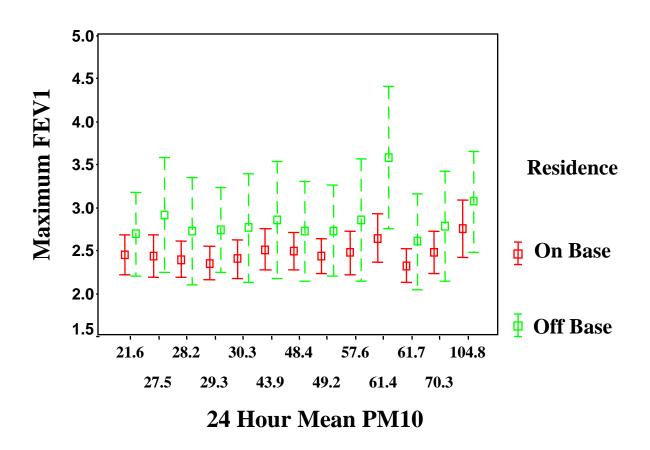


Figure 14. Mean Daily Maximum Peak Flow by 24 Hour Mean PM10

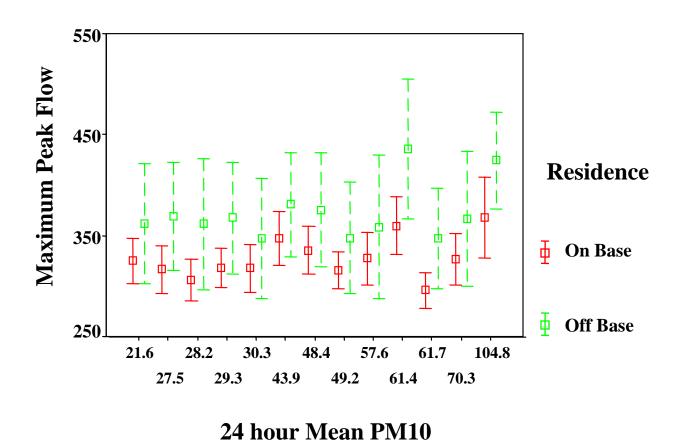


Table 9. Daily Mean PM₁₀ Levels and Daily Deviations From Individual Mean Peak Flows and FEV1s Among Atsugi On and Off Base Participants

Outcome	PM_{10}	PM_{10}	PM ₁₀ Tertile	PM ₁₀ Regression	P value+
Measure	Tertile 1*	Tertile 2**	3***	Coefficient and 95% CI+	
Peak Flow	324.1	343.6	330.9	0.032 (-0.15 0.20)	0.73
FEV1	2.46	2.57	2.51	0.0004 (-0.0014 0.0021)	0.66

⁺ From random effects repeated measures linear regression model, adjusted for on or off base, study week, individual, height, and having a cold.

^{* 21.6-30.3} ug/m³

^{**} $43.9 - 61.4 \text{ ug/m}^3$ *** >= 61.7 ug/m^3

Figure 15. Temporal Trends in Reported Colds and Runny Noses

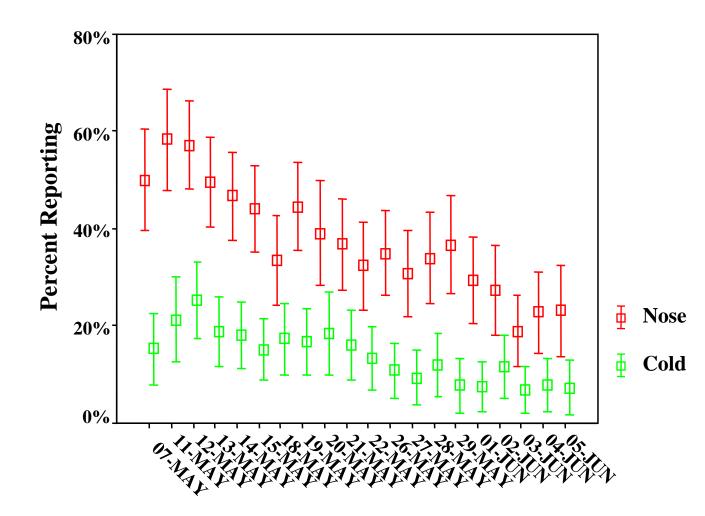


Figure 16. Temporal Trends in Reported Coughing Among Study Participants

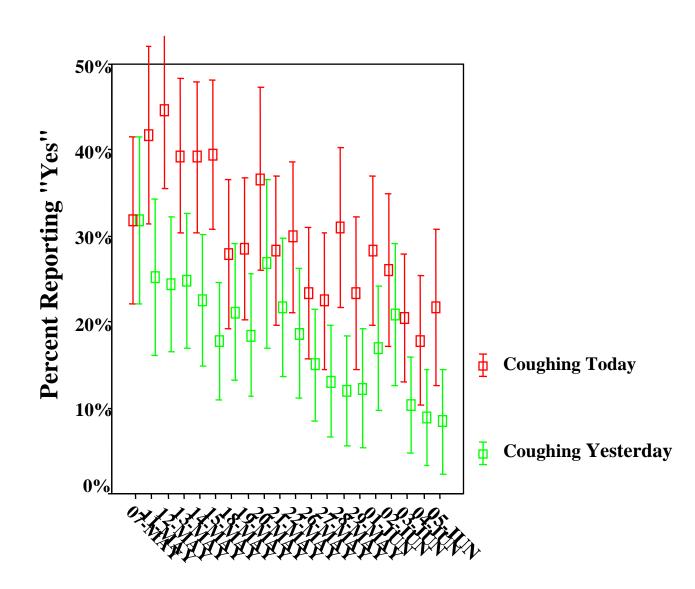


Figure 17. Temporal Trends in Reported Headache and Eye Pain Among Study Participants

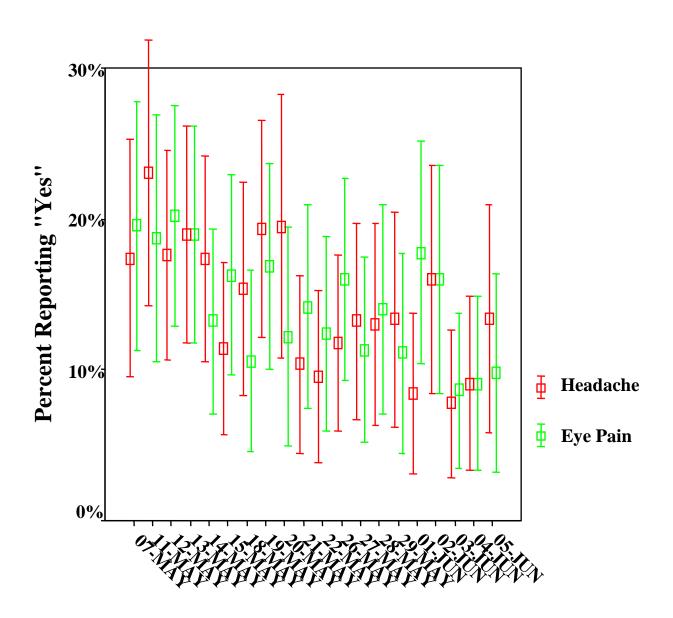


Figure 18. Mean Daily Respiratory Symptom (0-6) and Cold (1-3) Scores by Residence

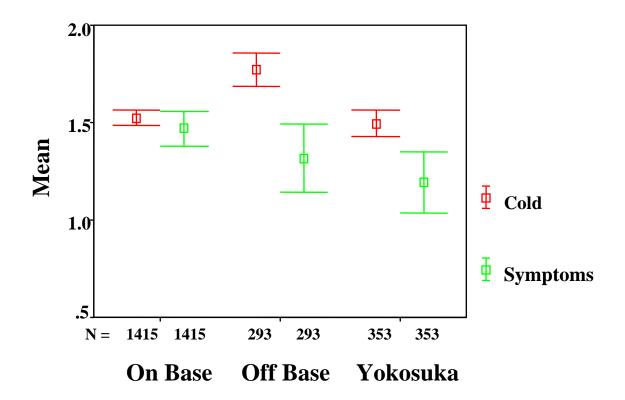


Figure 19. Percent of Days That Participants Reported Having a Runny Nose, Headache, or Eye Pain

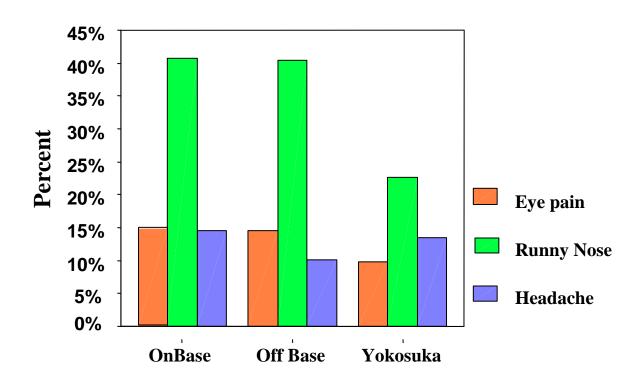


Table 10. Percent of Person Days that Participants Reporting Having Eye Pain, a Runny Nose, a Cold, Feeling Okay or Bad, or a Headache

Symptom	On base	Off base	Yokosuka	Relative Risk and (P	Relative Risk and
				value) – Yokosuka	(P value) - Off
				vs On base&	base vs On base&
Eye Pain	14.9%	14.5%	12.4%	0.73 (0.49)	0.95 (0.91)
Runny Nose	40.4%	40.2%	25.4%	0.52 (0.02)	0.99 (0.98)
Cold (yes)	14.0%	18.6%	10.7%	0.71 (0.52)	1.45 (0.46)
Headache	14.5%	10.1%	15.7%	1.25 (0.59)	0.61 (0.31)
Feeling Okay	58.1%	57.4%	39.1%	0.77 (0.47)	1.0 (1.0)
or Bad					

[&]amp; From logistic regression model, adjusted for study week, age, months in Japan, and individual.

Table 11. Mean Respiratory Symptom Score (0-6) and Mean Cold Score (1-3) for Participants at Each Site

Variable	On base	Off base	Yokosuka	P value – On	P value - On
				Base vs	Base vs Off
				Yokosuka&	Base&
Symptom	1.47 (1.70)	1.32 (1.53)	1.20 (1.50)	0.52	0.66
Score+					
Cold Score++	1.52 (0.73)	1.77 (0.74)	1.48 (0.68)	0.96	0.03

^{+ 1} Point each for eye pain, runny nose, coughing yesterday, coughing today, coughing last night, and trouble breathing

Table 12. Percent Distribution of Cold Status by Total Study Person Days

Do you have a cold?	On base	Off base	Yokosuka
Person days	1439	296	380
No	61.7%	41.9%	63.2%
Unsure	24.3%	39.5%	28.7%
Yes	14.0%	18.6%	8.2%

Table 13. Percent Distribution of Feeling Status by Total Study Person Days.

How do you feel?	On base	Off base	Yokosuka
Person days	1437	295	374
Good	43.7%	42.4%	58.0%
Okay	54.2%	54.9%	39.8%
Bad	4.0%	2.7%	2.1%

⁺⁺ Cold scale is no, unsure, yes

[&]amp; Random effects repeated measures linear regression model, adjusted for study week, day, individual, age, and months in Japan.

Table 14. The Relationship Between PM₁₀ Tertile and Reported Symptoms Among Atsugi Residents+

Symptom	Relative Risk and (P Value) Tertile 2 vs 1	Relative Risk and (P Value) Tertile 3 vs 1
Cold (Unsure vs No)	1.19 (0.31)	1.03 (0.87)
Cold (Yes vs No)	0.82 (0.30)	0.98 (0.94)
Feel (Okay vs Good)	0.99 (0.94)	1.03 (0.87)
Feel (Bad vs Good)	0.65 (0.23)	1.0 (1.0)

⁺Relative Risks from Polytomous Logistic Regression Models, adjusted for age and study week

Table 15. The Relationship Between PM₁₀ Tertile and Reported Symptoms Among Atsugi Residents+

Symptom	Relative Risk and (P Value)	Relative Risk and (P Value)
	Tertile 2 vs 1	Tertile 3 vs 1
Headache	0.90 (0.57)	1.08 (0.74)
Eye Pain	0.81 (0.25)	0.80 (0.32)
Runny Nose	0.95 (0.71)	0.95 (0.75)

⁺Relative Risks from Polytomous Logistic Regression Models, adjusted for age and study week

Table 16. Mean Daily Maximum Peak Flow and FEV1 by Residence and Self Reported Allergy Status (unadjusted for height or age)

Residence and Allergy	Observations	Peak Flow Mean and	FEV1 Mean and
Status	(Number of	STD	STD
	Children)		
On base-No	962 (57)	323.8 (97.4)	2.46 (0.99)
On base-Yes	340 (20)	320.9 (98.6)	2.36 (0.85)
On base-Unknown	89 (5)	354.8 (147.0)	2.65 (1.18)
Off base-No	267 (16)	376.3 (103.9)	2.91 (1.09)
Off base-Yes	18 (1)	248.0 (29.2)	1.57 (0.16)
Off base-Unknown	0 (0)	-	-
Yokosuka-No	272 (20)	389.8 (117.9)	2.63 (0.87)
Yokosuka-Yes	112 (9)	358.5 (81.3)	2.49 (0.82)
Yokosuka-Unknown	54 (4)	307.4 (124.5)	1.97 (0.67)

Table 17. Mean Daily Maximum Peak Flow and FEV1 by Residence and Self Reported Asthma Status (unadjusted for height or age)

Residence and Asthma	Observations	Peak Flow Mean and	FEV1 Mean and
Status	(Number of	STD	STD
	Children)		
On base-No	1187	322.6 (95.01)	2.44 (0.94)
	(70)		· · ·
On base-Yes	80 (5)	336.2 (100.1)	2.50 (0.97)
On base-Unknown	124 (7)	341.7 (152.3)	2.55 (1.29)
Off base-No	267 (16)	360.0 (100.9)	2.70 (1.01)
Off base-Yes	0 (0)	-	-
Off base-Unknown	18 (1)	489.8 (99.1)	4.63 (0.89)
Yokosuka-No	310 (23)	383.1 (116.5)	2.62 (0.85)
Yokosuka-Yes	74 (6)	370.1 (72.2)	2.45 (0.86)
Yokosuka-Unknown	54 (4)	307.4 (124.5)	1.97 (0.67)

Figure 20. Summary of 24 Hour PM10 Averages

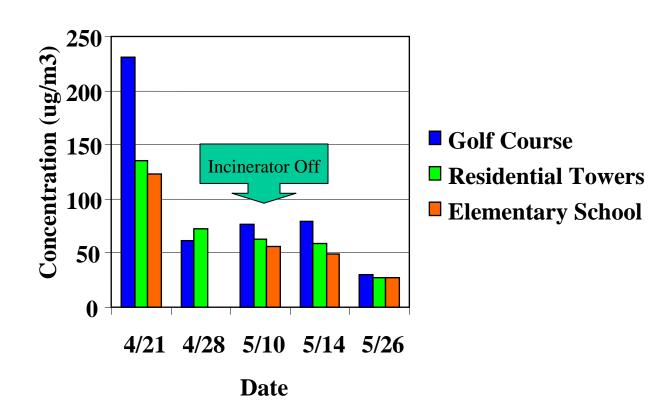
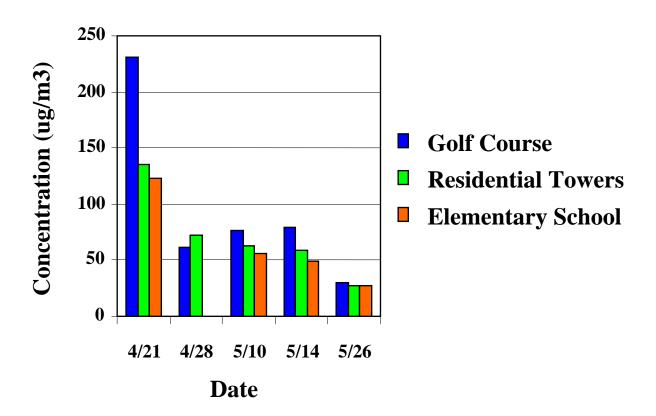


Figure 20. Summary of 24 Hour PM10 Averages



NOTICE

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Figure 20	Summary of 24 Hour PM10 Averages

Figure 1. Mean of Maximum Daily FEV1s by Height and Residence

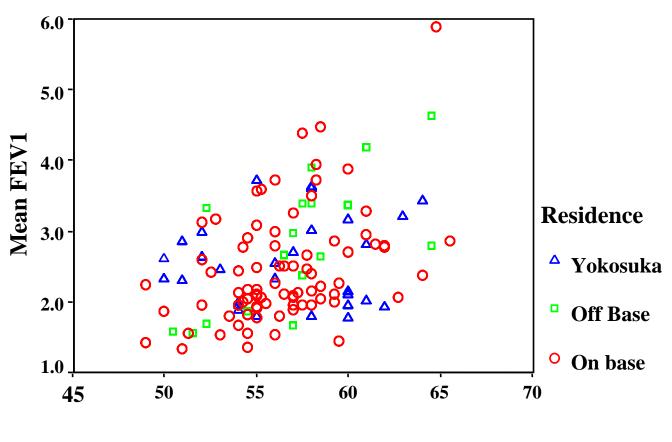


Figure 2. The Ratio of Observed to Predicted Mean FEV1 For Each Study Participant

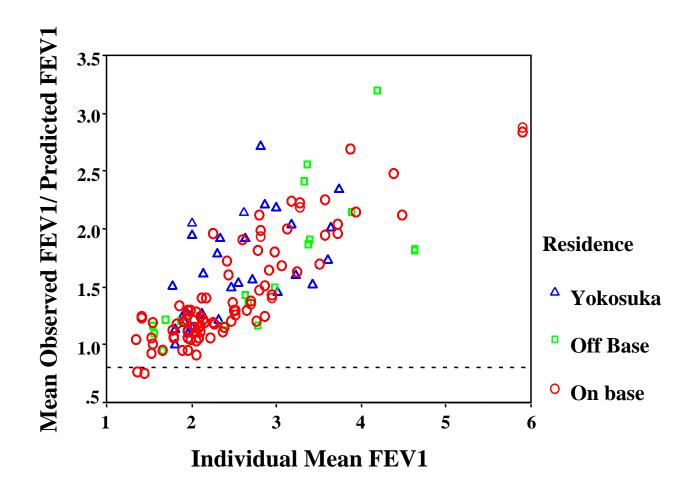


Figure 3. The Ratio of Observed to Predicted Mean FEV1 by Study Site

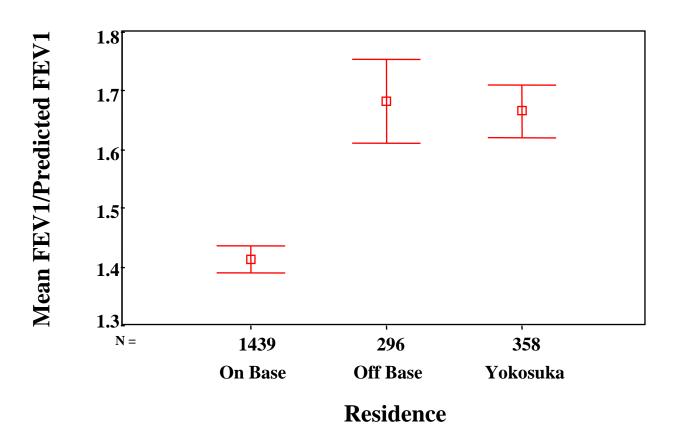


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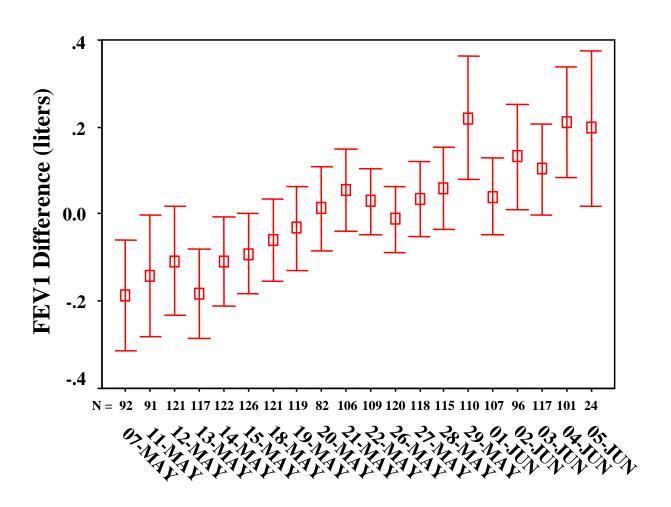


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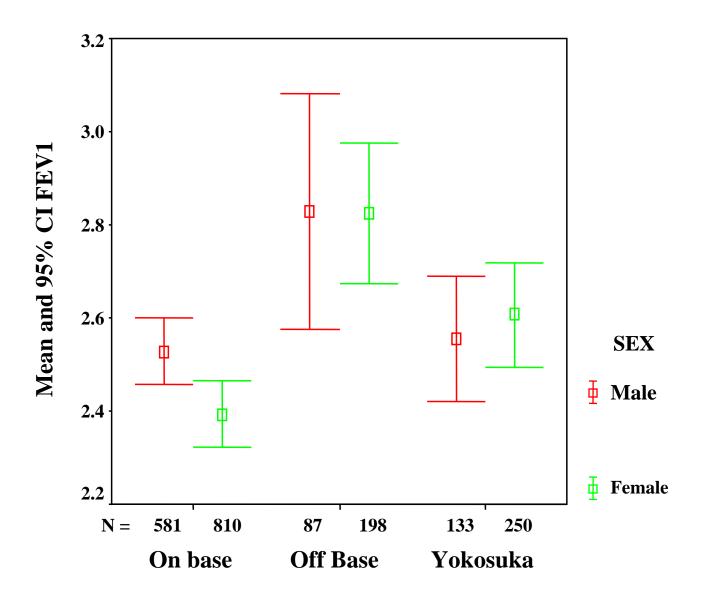


Figure 6. Maximum Daily FEV1 By Cold Status and Residence

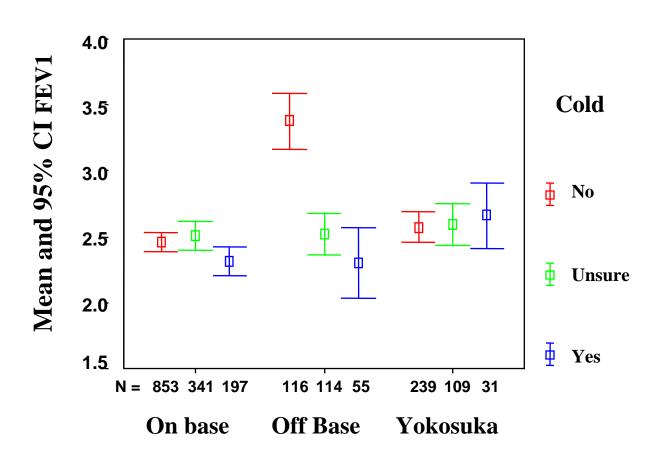


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Table 4. Daily Maximum FEV1 and Residence, Model 1+

Comparison	Mean FEV1	Adjusted FEV1 Difference	Significance	R^2++
	Difference*	and	Level&	
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On base vs Yokosuka	0.06	0.12 (-0.22 - 0.47)	0.48	0.22
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Table 5. Daily Maximum FEV1 and Residence, Model 2+

Comparison	Mean FEV1	Adjusted FEV1 Difference	Significance	R^2++
	Difference*	and	Level&	
		95% CI**		
On base vs Yokosuka	0.06	0.11 (-0.22 - 0.50)	0.52	0.18
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Figure 7. Mean of Daily Maximum Peak Flows by Height and Residence

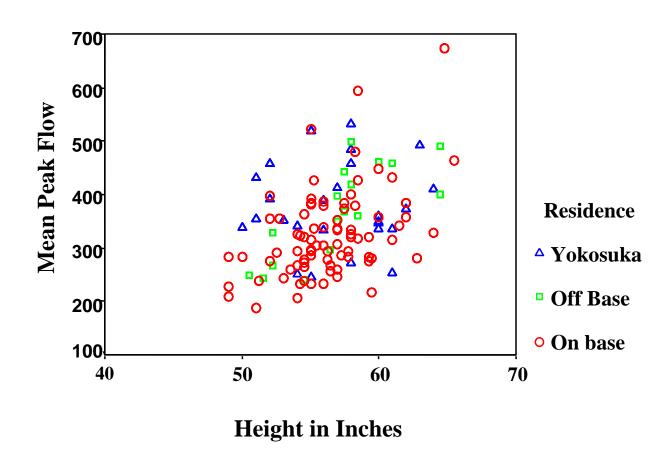


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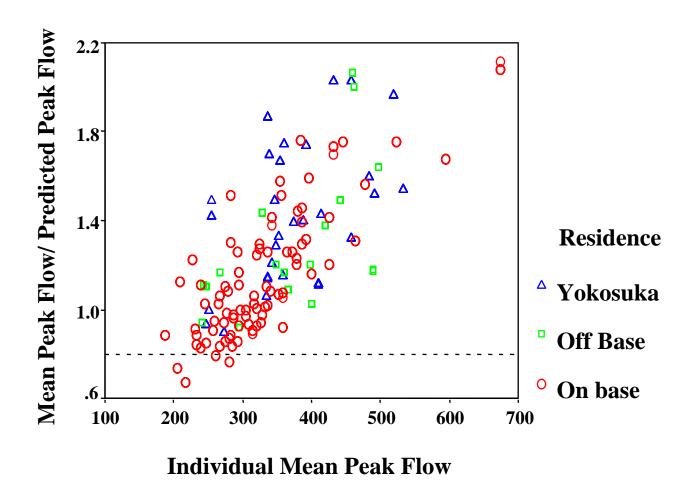


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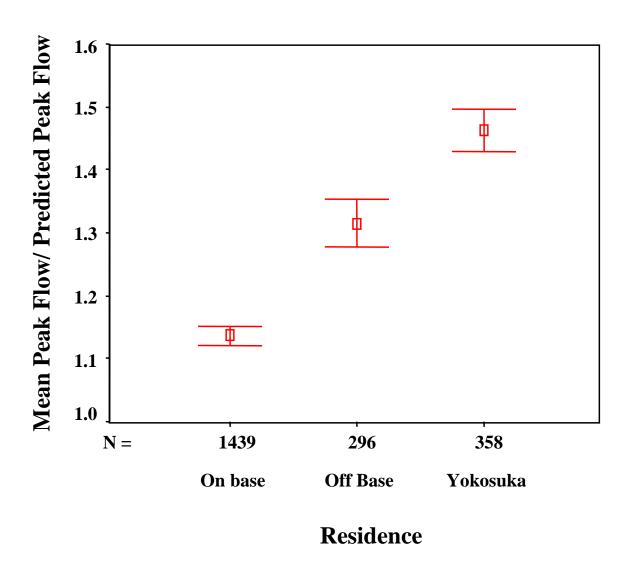


Figure 10. Temporal Trends in Daily Maximum Peak Flow Versus Individual Means

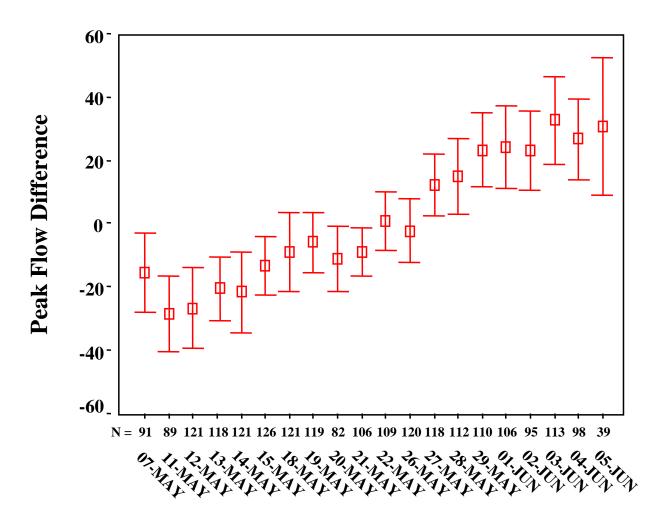


Figure 11. Maximum Daily Peak Flow By Sex and Residence

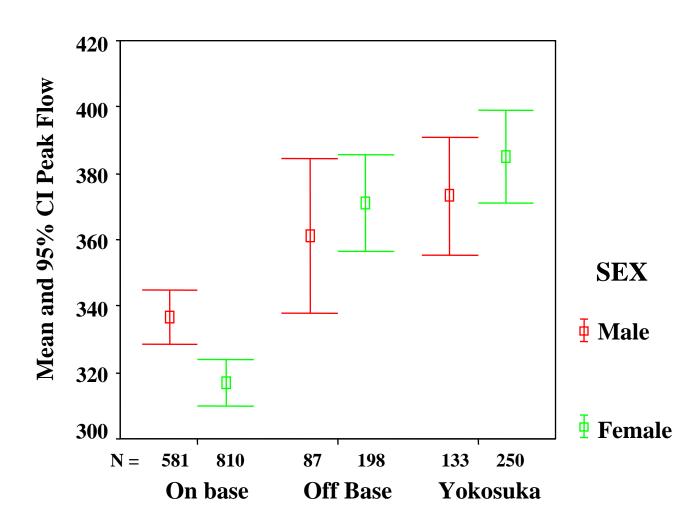


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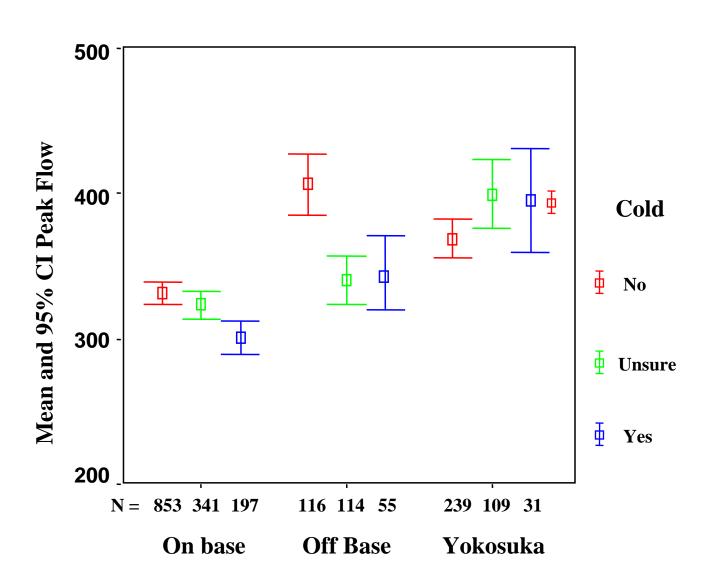


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Comparison	Mean PEF Difference*	Adjusted PEF Difference and 95% CI**	Significance Level&	R ² ++
On base vs Yokosuka	46.5	49.4 (12.2-86.5)	0.009	0.29
On base vs off base	43.1	38.0 (0.5-75.5)	0.047	0.41
All Atsugi vs Yokosuka	39.2	43.6 (7.9-79.4)	0.017	0.30

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On base vs Yokosuka	46.5	50.8 (16.0-85.7)	0.004	.28
On base vs off base	43.1	37.7 (2.3-73.2)	0.037	.40
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^{*} From all observations at each residence (on base, off base, all Atsugi, Yokosuka)

^{**}From regression model.

[&]amp; P value from regression model.

⁺⁺R² between groups.

^{*} From all observations at each residence (on base, off base, all Atsugi, Yokosuka)

^{**}From regression model.

[&]amp; P value from regression model.

⁺⁺R² between groups.

Figure 13. Mean Daily Maximum FEV1 by 24 Hour Mean PM10

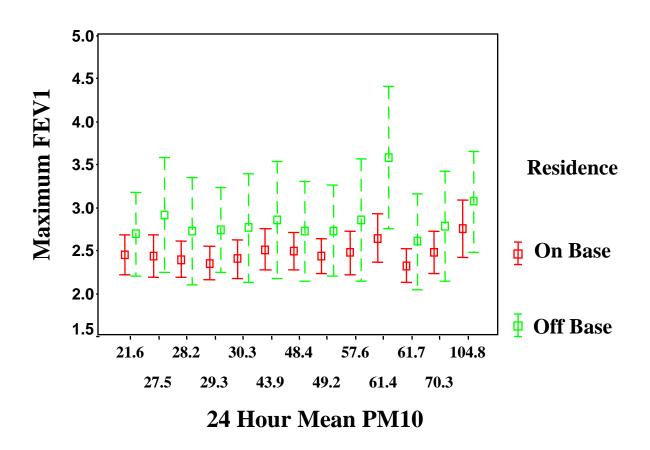


Figure 14. Mean Daily Maximum Peak Flow by 24 Hour Mean PM10

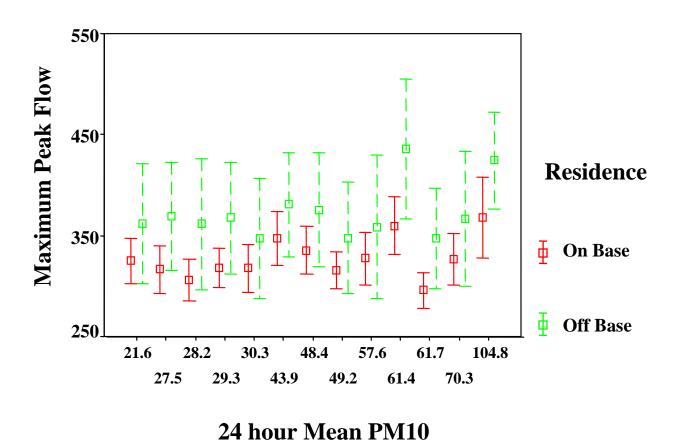


Table 9. Daily Mean PM₁₀ Levels and Daily Deviations From Individual Mean Peak Flows and FEV1s Among Atsugi On and Off Base Participants

Outcome	PM_{10}	PM_{10}	PM ₁₀ Tertile	PM ₁₀ Regression	P value+
Measure	Tertile 1*	Tertile 2**	3***	Coefficient and 95% CI+	
Peak Flow	324.1	343.6	330.9	0.032 (-0.15 0.20)	0.73
FEV1	2.46	2.57	2.51	0.0004 (-0.0014 0.0021)	0.66

⁺ From random effects repeated measures linear regression model, adjusted for on or off base, study week, individual, height, and having a cold.

^{* 21.6-30.3} ug/m³

^{**} $43.9 - 61.4 \text{ ug/m}^3$ *** >= 61.7 ug/m^3

Figure 15. Temporal Trends in Reported Colds and Runny Noses

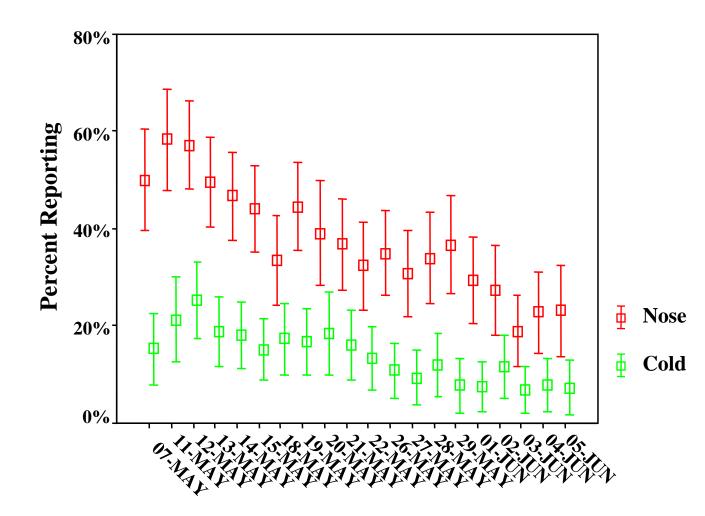


Figure 16. Temporal Trends in Reported Coughing Among Study Participants

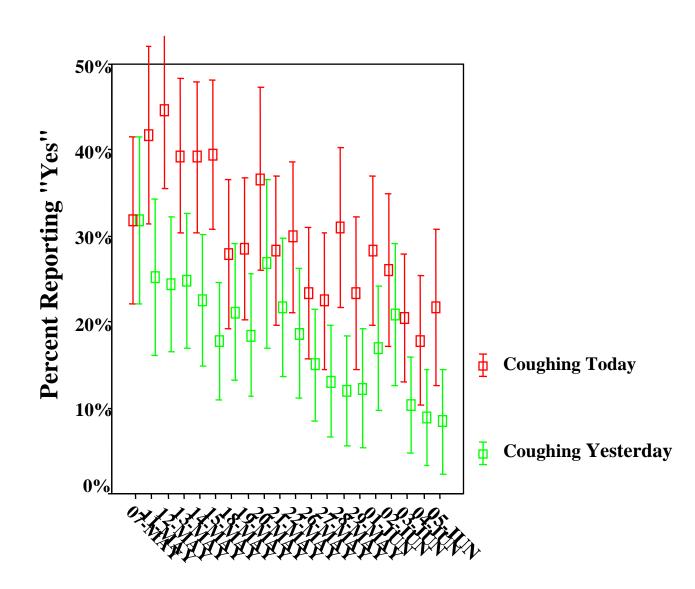


Figure 17. Temporal Trends in Reported Headache and Eye Pain Among Study Participants

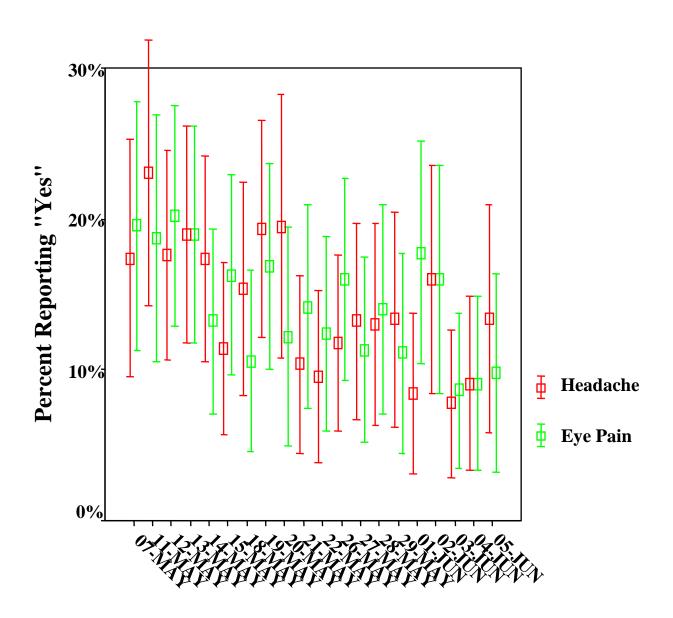


Figure 18. Mean Daily Respiratory Symptom (0-6) and Cold (1-3) Scores by Residence

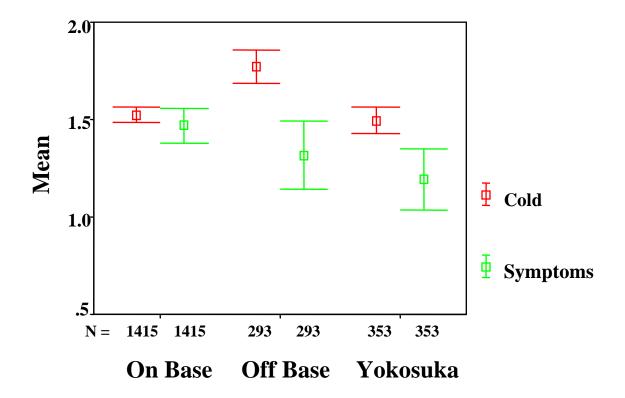


Figure 19. Percent of Days That Participants Reported Having a Runny Nose, Headache, or Eye Pain

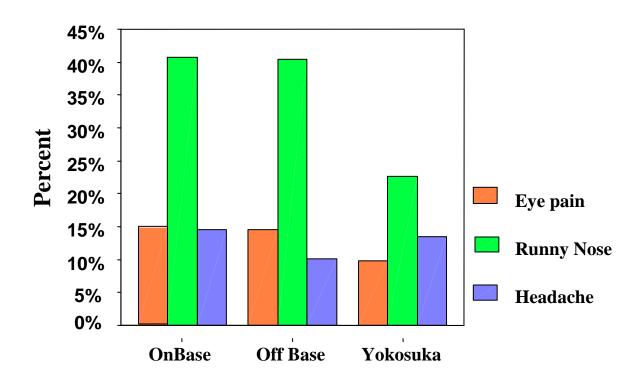


Table 10. Percent of Person Days that Participants Reporting Having Eye Pain, a Runny Nose, a Cold, Feeling Okay or Bad, or a Headache

Symptom	On base	Off base	Yokosuka	Relative Risk and (P	Relative Risk and
				value) – Yokosuka	(P value) - Off
				vs On base&	base vs On base&
Eye Pain	14.9%	14.5%	12.4%	0.73 (0.49)	0.95 (0.91)
Runny Nose	40.4%	40.2%	25.4%	0.52 (0.02)	0.99 (0.98)
Cold (yes)	14.0%	18.6%	10.7%	0.71 (0.52)	1.45 (0.46)
Headache	14.5%	10.1%	15.7%	1.25 (0.59)	0.61 (0.31)
Feeling Okay	58.1%	57.4%	39.1%	0.77 (0.47)	1.0 (1.0)
or Bad					

[&]amp; From logistic regression model, adjusted for study week, age, months in Japan, and individual.

Table 11. Mean Respiratory Symptom Score (0-6) and Mean Cold Score (1-3) for Participants at Each Site

Variable	On base	Off base	Yokosuka	P value – On	P value - On
				Base vs	Base vs Off
				Yokosuka&	Base&
Symptom	1.47 (1.70)	1.32 (1.53)	1.20 (1.50)	0.52	0.66
Score+					
Cold Score++	1.52 (0.73)	1.77 (0.74)	1.48 (0.68)	0.96	0.03

^{+ 1} Point each for eye pain, runny nose, coughing yesterday, coughing today, coughing last night, and trouble breathing

Table 12. Percent Distribution of Cold Status by Total Study Person Days

Do you have a cold?	On base	Off base	Yokosuka
Person days	1439	296	380
No	61.7%	41.9%	63.2%
Unsure	24.3%	39.5%	28.7%
Yes	14.0%	18.6%	8.2%

Table 13. Percent Distribution of Feeling Status by Total Study Person Days.

How do you feel?	On base	Off base	Yokosuka
Person days	1437	295	374
Good	43.7%	42.4%	58.0%
Okay	54.2%	54.9%	39.8%
Bad	4.0%	2.7%	2.1%

⁺⁺ Cold scale is no, unsure, yes

[&]amp; Random effects repeated measures linear regression model, adjusted for study week, day, individual, age, and months in Japan.

Table 14. The Relationship Between PM₁₀ Tertile and Reported Symptoms Among Atsugi Residents+

Symptom	Relative Risk and (P Value) Tertile 2 vs 1	Relative Risk and (P Value) Tertile 3 vs 1
Cold (Unsure vs No)	1.19 (0.31)	1.03 (0.87)
Cold (Yes vs No)	0.82 (0.30)	0.98 (0.94)
Feel (Okay vs Good)	0.99 (0.94)	1.03 (0.87)
Feel (Bad vs Good)	0.65 (0.23)	1.0 (1.0)

⁺Relative Risks from Polytomous Logistic Regression Models, adjusted for age and study week

Table 15. The Relationship Between PM₁₀ Tertile and Reported Symptoms Among Atsugi Residents+

Symptom	Relative Risk and (P Value)	Relative Risk and (P Value)	
	Tertile 2 vs 1	Tertile 3 vs 1	
Headache	0.90 (0.57)	1.08 (0.74)	
Eye Pain	0.81 (0.25)	0.80 (0.32)	
Runny Nose	0.95 (0.71)	0.95 (0.75)	

⁺Relative Risks from Polytomous Logistic Regression Models, adjusted for age and study week

Table 16. Mean Daily Maximum Peak Flow and FEV1 by Residence and Self Reported Allergy Status (unadjusted for height or age)

Residence and Allergy	Observations	Peak Flow Mean and	FEV1 Mean and
Status	(Number of	STD	STD
	Children)		
On base-No	962 (57)	323.8 (97.4)	2.46 (0.99)
On base-Yes	340 (20)	320.9 (98.6)	2.36 (0.85)
On base-Unknown	89 (5)	354.8 (147.0)	2.65 (1.18)
Off base-No	267 (16)	376.3 (103.9)	2.91 (1.09)
Off base-Yes	18 (1)	248.0 (29.2)	1.57 (0.16)
Off base-Unknown	0 (0)	-	-
Yokosuka-No	272 (20)	389.8 (117.9)	2.63 (0.87)
Yokosuka-Yes	112 (9)	358.5 (81.3)	2.49 (0.82)
Yokosuka-Unknown	54 (4)	307.4 (124.5)	1.97 (0.67)

Table 17. Mean Daily Maximum Peak Flow and FEV1 by Residence and Self Reported Asthma Status (unadjusted for height or age)

Residence and Asthma	Observations	Peak Flow Mean and	FEV1 Mean and
Status	(Number of	STD	STD
	Children)		
On base-No	1187	322.6 (95.01)	2.44 (0.94)
	(70)		
On base-Yes	80 (5)	336.2 (100.1)	2.50 (0.97)
On base-Unknown	124 (7)	341.7 (152.3)	2.55 (1.29)
Off base-No	267 (16)	360.0 (100.9)	2.70 (1.01)
Off base-Yes	0 (0)	-	-
Off base-Unknown	18 (1)	489.8 (99.1)	4.63 (0.89)
Yokosuka-No	310 (23)	383.1 (116.5)	2.62 (0.85)
Yokosuka-Yes	74 (6)	370.1 (72.2)	2.45 (0.86)
Yokosuka-Unknown	54 (4)	307.4 (124.5)	1.97 (0.67)

Figure 20. Summary of 24 Hour PM10 Averages

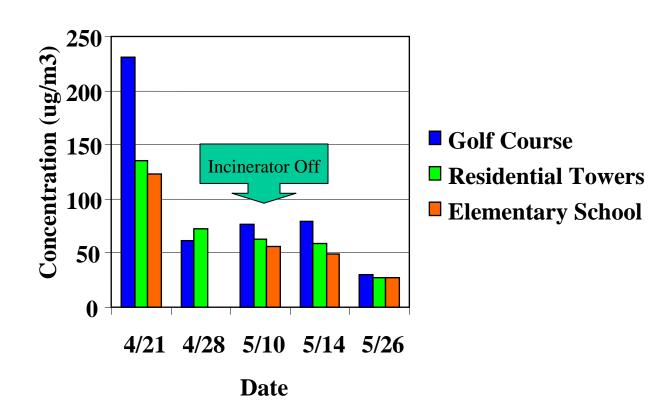
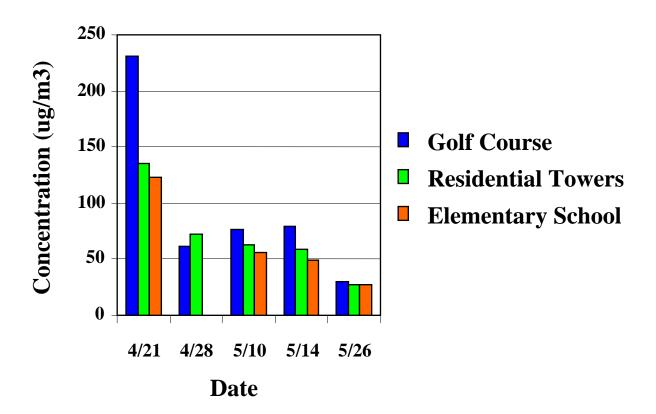


Figure 20. Summary of 24 Hour PM10 Averages



APPENDIX I

ACRONYMS, ABBREVIATIONS, AND SYMBOLS

AA Atomic Absorption

ACGIH American Conference of Governmental Industrial Hygienists

ADD Average Daily Dose

ADS Ambulatory Data System

AOC Area of Concern

ARARs Applicable or Relevant and Appropriate Requirements

ARB Air Resources Board

ASN Assistant Secretary of the Navy

ASN(E&S) Assistant Secretary of the Navy Environment and Safety

ASN(I&E) Assistant Secretary of the Navy Installations and Environment

ASN(M&R) Assistant Secretary of the Navy Manpower and Reserve Affairs

ASTM American Standards for Testing and Materials

ATSDR Agency for Toxic Substances and Disease Registry

BOO Bachelor Officer's Quarters

BRMED- Branch Medical Clinic

CLINIC

BUMED Navy Bureau of Medicine and Surgery

BUPERS Bureau of Naval Personnel

Cal EPA California Environmental Protection Agency

Cal DTSC California Department of Toxic Substances Control

CDC Child Development Center

CERCLA Comprehensive Environmental Response, Compensation and Liability Act

CFR Code of Federal Regulations

CHINFO Navy Chief of Information

CIH Certified Industrial Hygienist

CINCPAC-

Commander in Chief, U.S. Pacific Fleet

FLT

CLP Contract Laboratory Program

CNFJ Commander Naval Forces Japan

CNO Chief of Naval Operations

CO Commanding Officer

COC Constituent of Concern

COMNAV- Commander Naval Facilities Engineering Command

FACENG-

COM

CPDB Carcinogenic Potency Database

CRARM Presidential Commission on Risk Assessment and Risk Management

CRAVE Carcinogen Risk Assessment Verification Endeavor

CSF Carcinogenic Slope Factor

CVW 5 Carrier Air Wing FIVE

DNPH Dinitrophenylhydrazine

DOD Department of Defense

DSG Deputy Surgeon General

DTSC California EPA Department of Toxic Substances Control

EarthTech Earth Tech Environmental Corporation

EPA Environmental Protection Agency

FDA Food and Drug Administration

FOIA Freedom of Information Act

FTIR Fourier Transformed Infrared

GC/MS Gas Chromatography/Mass Spectrometry

GEMB Ground Electronics Maintenance Building

GOJ Government of Japan

HAPs Hazardous Air Pollutants

HEAST Health Effects Assessment Summary Tables

HI Hazard Index

HPLC High Performance Liquid Chromatography

HRA Health Risk Assessment

HSDB Hazardous Substances Data Bank

HSL 51 Helicopter Anti-Submarine Squadron Light FIVE ONE

HxCDF Hexachlorinated Dibenzo Furan

IA/AA Ratio of the maximum Indoor Air to maximum Ambient Air concentration

IARC International Agency for Research on Cancer

ICD International Classification of Disease

IEUBK EPA's Integrated Exposure Uptake Biokinetic Model for Lead

INCONUS In the continental United States

IRIS Integrated Risk Information System

ISCST3 USEPA Industrial Source Complex-Short Term model

IT IT Corporation

JMSDF Japan Maritime Self Defense Force

KPG Kanagawa Prefectural Government

LADD Lifetime Average Daily Dose

LANTDIV Naval Facilities Engineering Command, Atlantic Division

LOAEL Lowest-Observed-Adverse-Effect Level

MACT Maximum Achievable Control Technology

ManTech Environmental Technology, Inc.

MC Medical Corps

MRL Minimal Risk Levels

MS Master of Science Degree

MS Matrix Spikes

MSD Matrix Spike Duplicates

MSC Medical Service Corps

MSE Master of Science Engineering Degree

N/A Not Applicable

NAAQS National Ambient Air Quality Standards

NAF Naval Air Facility

NAS National Academy of Sciences

NAS COT National Academy of Sciences Committee on Toxicology

NAVPERS Naval Personnel

NCEA National Center for Environmental Assessment

NCEA CIN National Center for Environmental Assessment Cincinnati

NCP National Contingency Plan

NEHC Navy Environmental Health Center

NERL National Exposure Research Laboratory

NFESC Naval Facilities Engineering Service Center

NHY Naval Hospital, Yokosuka

NIEHS National Institute of Environmental Health Sciences

NIG Navy Inspector General

NIOSH National Institute for Occupational Safety and Health

NLM National Library of Medicine

NOAEL No-Observed-Adverse-Effect-Level

NRC National Research Council

NRDC Natural Resources Defense Council

OCDD Octachlorodibenzo-p-dioxin

OCONUS Outside the continental United States

OEHHA California Office of Environmental Health Hazard

OSHA Occupational Safety and Health Administration

PACDIV Naval Facilities Engineering Command, Pacific Division

PAMS Photochemcial Assessment Monitoring Stations

PCBs Polychlorinated Biphenyls

PCS Permanent Change of Station

PCDDs Polychlorinated Dibenzo Dioxins

PCDFs Polychlorianted Dibenzo Furans

PeCDF Pentachlorinated Dibenzo Furan

PEL Permissible Exposure Limit

PhD Doctor of Philosophy Degree

Pioneer Technologies Corporation

PLPP Pediatric Lead Poisoning Prevention

PM₁₀ Particulate Matter less than 10 micrometers in diameter

PM_{2.5} Particulate Matter less than 2.5 micrometers in diameter

PPBV Parts Per Billion by Volume

PRG EPA Region 9 Preliminary Remediation Goal

PSD Particle Size Distribution

QAPP Quality Assurance Project Plan

QA/QC Quality Assurance/Quality Control

RA Risk Assessment

Radian International LLC

RAGS Risk Assessment Guidance for Superfund

RBCs EPA Region III Risk Based Concentrations

RCRA Resource Conservation and Recovery Act

REL Reference Exposure Level

REM Registered Environmental Manager

RfC Reference Concentration

RfD Oral Reference Dose

RfDi Inhalation Reference Dose

RME Reasonable Maximum Exposure

RTECS Registry of Toxic Effects of Chemical Substances

RTI Research Triangle Institute

RTP Research Triangle Park

SF Carcinogenic Slope Factor

SF Standard Form

SIC Shinkampo Incineration Complex

SPM Suspended Particulate Matter

SVOCs Semivolatile Organic Compounds

TCDD 2,3,7,8 Tetrachlorodibenzo-p-dioxin

TEAM USEPA Total Exposure Assessment Method

TEQ Toxic Equivalent Concentration

TSP Total Suspended Particulates

TWA Time Weighted Average

UAI UAI Environmental, Inc.

UATMP Urban Air Toxics Monitoring Program

UCL Upper Confidence Limit

UF Uncertainty Factor

URI Upper Respiratory Illness

USC United States Code

USEPA U.S. Environmental Protection Agency

USFJ U.S. Forces Japan

USN United States Navy

VOCs Volatile Organic Compounds

WPAFB Wright-Patterson Air Force Base Toxicological Detachment

WTI Waste Technologies Industries

1 X 10⁻⁶ One in a million; also 1E-06