Toxicological Advisory Committee

AIR TOXICS REPORT

Prepared for

State of Vermont

Agency of Natural Resources

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List of Acronyms

ACGIH - American Conference of Governmental Industrial Hygienists

AEGL - Acute Exposure Guideline Level

Agency - Vermont Agency of Natural Resources

Category I - A contaminant that is known or suspected to be a carcinogen.

Category II - A contaminant that is a noncarcinogen with potential/systemic effects due to long term exposure.

Category III - A contaminant that is a noncarcinogen considered to have primarily short term irritant effects.

CPF - Cancer Potency Factor

CPF_i - Inhalation Cancer Potency Factor

CPF_o- Oral Cancer Potency Factor

Committee - Toxicological Advisory Committee

HAAS - Hazardous Ambient Air Standard

HEAST - Health Effects Summary Tables, US EPA

IARC - International Agency for Research on Cancer

IRIS - Integrated Risk Information System database, US EPA

MDL - Method Detection Limit

NCI - National Cancer Institute

NIOSH - National Institute for Occupational Safety and Health

NTP - National Toxicological Program

OSHA - United States Department of Labor Occupational Safety and Health Administration

PAH - Polynuclear Aromatic Hydrocarbons

PCB - Polychlorinated Biphenyl

PCDD/PCDF - Polychlorinated Dibenzo-P-Dioxins/Furans

PEL - Permissible Exposure Limit, derived by OSHA

REL - Recommended Exposure Limit, derived by NIOSH

RfC - Inhalation Reference Concentration

TLV - Threshold Limit Value, derived by ACGIH

US EPA - United States Environmental Protection Agency

VOC - Volatile Organic Compound

I. Executive Summary

A. Introduction

During the 1980s, Vermonters expressed growing concern over the potential health and environmental effects of unregulated toxic air pollutants in the state's air-shed. Responding to this concern, the Vermont Agency of Natural Resources (hereafter referred to as "the Agency") proposed "Regulations for the Control of Hazardous Air Contaminants." One aspect of these regulations required a source to determine its own emissions and effect on air quality, and then to add these emissions to existing levels in order to determine if the source in question "caused or contributed to" a violation of the state ambient air quality standard for a hazardous air contaminant.

To address difficulties in implementation surrounding this provision, two actions were taken in 1993. First, after much public deliberation, the Agency amended the regulations and suspended the requirement to consider existing air quality. This suspension was for a five year period, pending a review of the air quality standards for hazardous air contaminants. This review was to be conducted based on the best available scientific information on health effects and risk, and the achievability of these ambient air standards. Section 5-261(6)(c) of the 1993 amended regulations states, "As expediously as practicable, but no later than January 1, 1998, the Secretary shall review each Hazardous Ambient Air Standard for the contaminants listed in Appendix C, Category I and the method for their derivation specified in Appendix D to assure that each such standard represents a level requisite to protect the public health." All parties agreed more information was needed on existing levels of air toxics throughout the State of Vermont.

In response to the need for more air toxic data in ambient air, the second action was the establishment of the Hazardous Air Contaminant Monitoring Program. Act 92 of the 1993 Adjourned Legislative Session directed the Agency to establish a Hazardous Air Contaminant Monitoring Program with the following goals:

(1) Measure the presence of hazardous air contaminants in ambient air;
(2) Identify sources of hazardous air contaminants;
(3) Assess human health and ecological risk to focus studies on those air contaminants which pose the greatest risk;
(4) Gather sufficient data to allow the Secretary to establish appropriately protective standards; and
(5) Ensure adequate data are collected to support the state's operating permit program.

Act 92 provided funding for this effort by placing a $1 surcharge on motor vehicle registrations beginning January, 1994, and a surcharge on industrial emissions based on the toxicity of chemicals emitted. These fees and the program will sunset if not re-authorized by July 1998.

B. Review of Hazardous Ambient Air Standards

To assist in the first action of reviewing the methodology used to derive the ambient air standards for toxic pollutants, the Agency appointed a Toxicological Advisory Committee ("the Committee"). The Committee consists of toxicologists and scientists from varying backgrounds, representatives of the State's Department of Health and Department of Environmental Conservation, and representatives of the major interest groups involved with the air toxic program including the Vermont Public Interest Research Group and the Associated Industries of Vermont. A complete list of the Committee members is found in Appendix E. The recommendations contained in this report reflect the general consensus of the Committee.

The Committee has reviewed the methodology for deriving the Hazardous Ambient Air Standards (HAAS) for Categories I, II and III compounds listed in Appendix C of the 1993 amended regulations. Per the current Regulations, Category I Compounds are those Hazardous Air Contaminants identified as potentially carcinogenic by the United States Environmental Protection Agency (US EPA) or International Agency for Research on Cancer (IARC) or reported to induce cancer in two or more tests performed by either the National Toxicological Program (NTP) or National Cancer Institute (NCI). Antimony trioxide and acrylamide were also placed in this Category due to their identification as potential human carcinogens by the American Conference of Governmental Industrial Hygienists (ACGIH) and Dr. Edward Calabrese of the University of Massachusetts. Category II Compounds are noncarcinogens with potential chronic/systemic effects due to long term exposure and Category III Compounds are noncarcinogens considered to have primarily short term irritant effects.

1. Category I Compounds

After reviewing Appendix D of the Regulations, the Committee is now recommending modifying the criteria for placing a chemical on Category I. The Committee now proposes that a chemical be placed on Category I if it is identified as potentially carcinogenic by the US EPA or IARC and may be considered for inclusion if positive tests in two or more species are reported by NTP. This change of requiring positive tests in two or more species is consistent with the EPA and IARC methodology for determining if a compound is potentially carcinogenic. The Committee has determined that both NCI and NTP need not be consulted in determining whether a compound is potentially carcinogenic. This is because the NCI carcinogenesis Bioassay Program has been part of the National Institute for Environmental Health Sciences since 1981 at which time NTP became responsible for conducting the carcinogenicity/toxicity studies.

There are currently 54 compounds on the Category I list. The Agency has determined that all but six compounds (arsine, 1,1-biphenyl, diazomethane, methyl bromide, methyl iodide and propylene imine) of the current Category I compounds meet the above proposed revised requirements for classification as a Category I contaminant. The Committee recommends that these six compounds be removed from Category I and be placed in Category II. Standards for the remaining 48 Category I compounds have been updated using the most current toxicity information.
The Agency has determined that 14 compounds currently classified as Category II or III meet the proposed revised criteria for classification as Category I. The majority of the Committee

proposes that these 14 compounds be placed on Category I and the standards be updated using the Category I methodology.

The Committee also proposes the following revisions to deriving Category I ambient standards:

(1) employing a conservative absorption factor of 100% or 1;

(2) if an inhalation potency factor is unavailable, employing an oral potency factor as a surrogate unless carcinogenicity is unique to ingestion route of exposure;

(3) if neither an inhalation nor an oral potency factor exists, employing an inhalation reference concentration divided by an uncertainty factor of 10 to account for carcinogenicity; and

(4) if a potency factor or reference concentration is not available, the Committee recommends retaining the default standard as the ambient standard.

2. Category II and III Compounds

There are currently 44 Category II and 192 Category III compounds. At present, the Regulations require that the HAAS for Category II and III compounds be based upon the ACGIH Threshold Limit Values (TLVs) which are work place guidelines. Ambient standards for these compounds are derived by dividing the TLV, when one is available, by one or more uncertainty factors.

Per the current Regulations, if a TLV is not available for a particular compound, then, if available, the Agency shall use an occupational standard established by either the National Institute for Occupational Safety and Health (NIOSH)(termed Recommended Exposure Limits or RELs) or the US Department of Labor Occupational Safety and Health Administration (OSHA)(termed Permissible Exposure Limits or PELs) as a surrogate in the ambient standard derivation process. If no occupational value is available from any of these sources, a standard is issued based on an evaluation of the toxicity information available for the compound in question.
The Committee recommends changing the above methodology to derive the standards for Category II and III compounds. Inhalation reference concentrations (RfCs) are proposed to be used as the basis for ambient standards. RfCs are designed to be protective of potential noncarcinogenic health effects given long term, continuous exposure.

If chemical specific RfCs are not available, the Committee recommends using the most conservative of the TLV, PEL, or REL as the basis of the HAAS. It is also recommended that a simple mechanism be developed to allow for the consideration of updated, scientific information that supports using an occupational standard other than the most conservative one as the basis of the HAAS.

If no RfC or occupational value is available, a standard will be issued based on an evaluation of the toxicity information available for the compound in question.

In addition, the proposed revised methodology for Category II and Category III compounds includes employing an extra uncertainty factor of 10 to derive the HAAS for those Category II and Category III compounds identified as a potential occupational carcinogen by NIOSH or as an A1: Confirmed Human Carcinogen; A2: Suspected Human Carcinogen; or A3: Animal Carcinogen by ACGIH.

Finally, the Committee recommends comparing the HAAS to short term exposure limits called Acute Exposure Guidelines (AEGLs), to determine if the standard is sufficiently protective. Future efforts should focus on developing short term ambient standards to protect against potential adverse health effects that may be associated with acute or short term exposures.

C. Hazardous Air Contaminant Monitoring Program

In order to compare ambient air levels to the standards, the Agency has reviewed air monitoring data from 1993 to 1995. The Air Monitoring Data shows that there are nine compounds whose air concentrations are consistently above the current standards. Those compounds are benzene, 1-3 butadiene, carbon tetrachloride, chloroform, formaldehyde, methyl chloride, methylene chloride, tetrachloroethylene, and 1,2,4-trimethyl benzene. Due to public concern, the Agency is also concerned about styrene, acrolein and mercury. Based on updated toxicity information, current air concentrations of tetrachloroethylene and 1,2,4-trimethyl benzene would no longer exceed the proposed revised standards, however, acrolein's concentrations would now exceed the proposed revised standard. Out of the eight compounds that would consistently exceed the proposed revised standard, five are considered to be generated locally, and three are considered to be transported from other areas. The local pollutants are benzene, 1,3-butadiene, formaldehyde, methylene chloride and acrolein. The transported pollutants are carbon tetrachloride, chloroform, and methyl chloride. Of the locally generated compounds listed above, benzene, 1,3-butadiene, formaldehyde and acrolein are all byproducts of combustion.

D. Discussion of Risk

Since the Agency is responsible for ensuring public health protection from outdoor exposures to toxic air pollutants, the Agency believes it is prudent to establish ambient air standards adequate to protect public health with an ample margin of safety. For carcinogens, where it is assumed there is no absolutely risk-free level of exposure, it becomes necessary for the Agency to establish a maximum allowable level of incremental lifetime carcinogenic risk (hereafter incremental lifetime carcinogenic risk is referred to as "risk"). This maximum allowable level of risk represents a negligible increase in potential risk over background risk over a lifetime of exposure, for the population of concern. Various federal and state agencies employ different levels of maximum allowable risk or negligible risk. Values between one and one million (1×10^{-6}) to one in ten thousand (1×10^{-4}) are typically used. The maximum allowable level of risk is a risk management decision that must be made by the Agency of Natural Resources, in consultation with the Department of Health, and therefore the Committee is not recommending any specific risk level at which ambient standards should be established. However, the Committee does agree that $1x10^{-6}$ is a negligible risk.

Deciding what amount of potential risk is to be considered negligible is a complex task. However, whether or not it is acceptable to allow additional emissions even at this negligible level should depend on a consideration of existing ambient air quality. For example, if risk associated with inhalation of existing ambient air is estimated to already be highly elevated, above $1x10^{-4}$ for example, it may not be appropriate to allow additional emissions, even at the $1x10^{-6}$ level, without further investigation.

The Committee agrees that it may be prudent to define a total ambient air quality goal that should not be exceeded. Individual point sources would then be required to control emissions so that the total risk associated with inhalation of ambient air is below the total ambient air quality goal.

E. Recommendations for Future Actions

1. The Committee recommends the continued reliance on EPA and IARC for the purposes of classification of known or potential carcinogens (Category I compounds). The Committee further recommends that a compound may be considered for inclusion into Category I if positive cancer tests in two species are reported by NTP.

2. The Committee recommends the current methodology for establishing risk-based standards for known or potential carcinogens be retained using the most recent cancer potency factors developed by EPA. The one suggested change to the methodology is to use a conservative absorption factor of 100 percent for all Category I compounds. The Committee recommends the Agency allow for the consideration of the use of a factor other than 100 percent where toxicological information is presented to support an alternative value.

3. For known or potential carcinogens without EPA cancer potency factors, the Committee recommends revising the current methodology to allow the use of inhalation reference concentrations (RfCs) divided by an additional uncertainty factor of 10. In the absence of cancer potency factors and RfCs, the Committee recommends that a default concentration of 0.01 micrograms per cubic meter (ug/m^3) be retained.

4. For those non-cancer compounds (Category II & III) where RfCs have been developed by EPA, the Committee recommends using RfCs as the basis for air standards instead of modified occupational levels. For those Category II and III compounds where an RfC has not been established, the Committee recommends using the lower of the TLV, PEL, or REL value as the basis for the modified occupational standard. In addition, if a Category II or III compound is considered an occupational carcinogen by NIOSH or ACGIH, the Committee recommends dividing the standard by an additional uncertainty factor of 10.

5. The Committee suggests continuing the air monitoring program for air toxics to determine ambient levels and to observe any trends as regulatory actions are implemented. The air monitoring data allows the Agency to compare ambient air levels to the standards to determine those compounds that consistently exceed the standard.

6. The Committee recommends developing a Toxic Air Action Plan to discuss methods of reducing emissions of those locally generated compounds that consistently pose a public health concern.

7. The Committee recommends conducting a review of standards every five years so that standards can be updated based on the most recent toxicological information. The Committee recommends using the proposed revised methodologies for reviewing the standards. If during the five year periodic update it is determined that a change in toxicity information will significantly impact the standard, the Agency should consider a single regulatory amendment to revise the standard (s).

8. The Committee recommends developing short term air standards for some Hazardous Air Contaminants to protect against potential adverse health effects that may be associated with acute or short term exposures. In the interim, as a temporary measure, the Committee recommends establishing the environmental level for these contaminants as the occupational ceiling limit.

9. The Committee suggests considering other endpoints in addition to carcinogenic endpoints in order to determine if the current standards are protective. The Committee suggests considering other endpoints such as endocrine disruptors and environmental respiratory disease in future regulatory decisions.

II. Review of Hazardous Ambient Air Standards

A. Background

In November 1981, the Vermont Air Pollution Control Regulations were amended to adopt a definition for hazardous air contaminants and adopt Regulation 5-261 which specifically deals with the control of such compounds. In June of 1985, the Air Pollution Control Division issued a draft proposal entitled "Vermont Hazardous Air Contaminant Guideline", the goal of which was to offer guidance in the implementation of the relatively new Regulation 5-261. The 1985 Guideline (hereafter referred to as "Guideline") included a list of compounds referred to as Hazardous Air Contaminants, that, when present at elevated concentrations in outdoor (ambient) air, may be of concern for public health. This set of compounds was simply all the chemicals listed in the 1984 issue of "Threshold Limit Values for Chemical Substances and Physical Agents" produced by the American Conference of Governmental Industrial Hygienists (ACGIH).
An ambient standard was originally derived for each Hazardous Air Contaminant by dividing its work place (occupational) air standard, called a Threshold Limit Value or TLV, by 100 to account for some model uncertainties and then by a time factor of 4.2 to extrapolate from a standard designed to protect the average healthy worker from adverse health effects to an outdoor air standard designed to protect the general public from adverse effects (i.e., to go from 8 hours per day, 5 days per week to continuous exposures of 24 hours per day, 7 days per week) (ACGIH, 1997). No distinction was made between compounds based on their toxic endpoint, i.e., compounds with known carcinogenic potential were treated the same as compounds known to be short term irritants.

Over a number of years and with the help of several air quality and public health specialists, the original Guideline were revised and eventually became incorporated into the Air Pollution Control Regulations in March of 1989. The major revisions included:

(1) Compounds not reasonably expected to be used in Vermont or used solely as pesticides and thus regulated by the Department of Agriculture, Farm and Markets were eliminated from the list of Hazardous Air Contaminants;

(2) The remaining compounds were divided into three categories by type of toxic endpoint (carcinogens versus noncarcinogens):

(a) Category I Contaminants: known or suspected carcinogens;

(b) Category II Contaminants: Noncarcinogens with potential chronic/systemic effects due to long term exposure;

(c) Category III Contaminants: Noncarcinogens considered to have only short term irritant effects; and

(3) Distinct procedures were established for setting ambient air standards for each Category of Hazardous Air Contaminant.

In 1993, the Regulation was revised to address difficulties in interpretation and implementation. Changes were only made in how the Regulation was to be applied, not how the standards were to be derived or the values of the standards themselves. This version of the Regulation exists today. A five year review period was concurrently set aside to review the basis for each ambient standard and the impacts of regulating individual emitters without considering the levels of contaminants in the existing air. This report presents the findings and recommendations resulting from this review.

B. Category I

1. Current Methodology

Per the current Regulations, those Hazardous Air Contaminants identified as potentially carcinogenic by the United States Environmental Protection Agency (US EPA) or International Agency for Research on Cancer (IARC) or reported to induce cancer in two or more tests performed by either the National Toxicological Program (NTP) or National Cancer Institute (NCI) are classified as Category I Compounds: known or suspected carcinogens. Antimony trioxide and acrylamide were also placed in this Category due to their identification as potential human carcinogens by the ACGIH and Dr. Edward Calabrese of the University of Massachusetts.

Because it is generally assumed that no threshold level of exposure exists for potential carcinogens, i.e., an increase in the probability of developing cancer (over the background cancer rate) is assumed to be associated with any exposure greater than zero, the current Regulations require that the ambient air standard for each Category I compound be set at a concentration estimated to correspond to a one in one million (1×10^{-6}) increase in the probability of developing cancer (over and above the background rate) over a lifetime of exposure. Each standard then represents the estimated level of that compound in ambient air to which one could be exposed to 24 hours per day, 365 days per year for 70 years and experience only a one in one million increase in the risk of developing cancer from that exposure.

In order to estimate the concentration of a compound in outdoor air that corresponds to a maximum allowable risk of 1×10^{-6}, it is necessary to first have a quantitative estimate of the compound's carcinogenic potential. A Cancer Potency Factor, also referred to as a Cancer Slope Factor, is a measure of a chemical's carcinogenic toxicity. The higher the value, the greater the compound's carcinogenic potential.

Because the ability of a compound to cause cancer may vary depending on the way it enters the body, estimates of carcinogenic potency via inhalation and oral exposure have been derived for a number of compounds. A number of different mathematical models are available for deriving such estimates. For example, several cancer potency factors currently available on the US EPA

Integrated Risk Information System (IRIS) database were derived using the Linearized Multistage Model. The Committee assumes that the various models employed by the US EPA to derive the factors cited on IRIS are both reasonable and appropriate.

The ambient air standard for each Category I compound was derived by combining an estimate of the compound's cancer causing potential (Cancer Potency Factor) with an estimate of potential human exposure (24 hours per day, every day for 70 years). The greater the Cancer Potency Factor, the more restrictive the standard derived.

The following hierarchy was employed in the derivation process:

(1) An ambient air standard corresponding to an excess lifetime carcinogenic risk of one in one million was calculated based on an available inhalation Cancer Potency Factor;

(2) In the absence of an inhalation Cancer Potency Factor, an ambient air standard corresponding to an excess lifetime carcinogenic risk of one in one million was calculated based on an oral Cancer Potency Factor;

(3) If no Cancer Potency Factor was available for either the inhalation or oral route of exposure, a default value of 0.01 micrograms/cubic meter was employed as the ambient standard.

The algorithm employed in options (1) and (2) is presented in Equation 1. A detailed description of the variables involved is provided in Appendix A, Figure 1.

Equation 1:

Hazardous Ambient Air Standard (micrograms/cubic meter) =

$$\frac{\ln [1 - (1 \times 10^{-6})] \times 1000 \text{ micrograms per milligram}}{- [(\text{Cancer Potency Factor} \times \text{Daily Inhalation Rate} \times F) \backslash \text{Body Weight}]}$$

The above algorithm incorporates a factor, referred to as the F factor, originally intended to account for the fact that, in some instances, less than 100 percent of the compound inhaled into the lungs is actually absorbed into the blood stream. At present, a value other than 100 percent is employed for only three compounds: carbon tetrachloride, tetrachloroethylene and trichloroethylene.

For those Category I contaminants with neither an inhalation nor oral potency factor, a default maximum acceptable concentration of 0.01 ug/m^3 was employed as an ambient standard. This value was derived in 1989 in the following manner:

(1) For each Category I compound adopted into the Regulations in 1989 that had a cancer potency factor (CPF), the potency factor was multiplied by the compound's molecular weight (MW) to produce a Potency Index (PI) [CPF X MW = PI];

(2) The median value of all the Potency Indices generated (100) and the median of all the Molecular Weights (285) were plugged into the above equation to yield a calculated Cancer Potency Factor [CPF x 285 = 100 thus CPF = .35];

(3) The calculated Cancer Potency Factor was plugged into the algorithm used to derive an annual ambient standard for a potential carcinogen. A value of 0.01 ug/m^3 resulted.

Thus, an annual average ambient concentration of 0.01 ug/m^3 was estimated to correspond to a one in one million excess lifetime cancer risk given a cancer potency factor of 0.35 $(mg/kg\text{-}day)^{-1}$.

An annual averaging period was employed for all Category I compounds.

2. Recommended Revisions

For the purposes of this report, the Cancer Potency Factors employed for each Category I contaminant were reviewed, researched and where appropriate updated. Toxicity information was obtained from the US EPA IRIS database and Health Effects Summary Tables (HEAST) as well as a summary of carcinogenic evaluations presented in the IARC monographs. A compendium of NTP abstracts issued between 1972 and 1993 was also searched (EHP, 1993). In addition, a meeting of the Toxicological Advisory Committee (hereinafter "Committee") in November 1997 generated several recommendations for alterations in the current assessment process. Proposed changes in assessment protocol along with updated toxicity information are detailed in the paragraphs which follow.

Although the regulations currently state that both NTP and NCI studies are to be reviewed for evidence of potential carcinogenicity, a review of the NTP abstracts compendium is sufficient because the NCI Carcinogenesis Bioassay Program has been part of the National Institute for Environmental Health Sciences since 1981 at which time NTP became responsible for conducting the carcinogenicity /toxicity studies (Felter, 1997). While it is possible that a pre-1981 study may be noted as NCI/NTP or just NCI, it is likely to be included in the compendium reviewed.

As of this writing, listings of NTP abstracts issued since 1993 (and any older NCP studies not included in the compendium) had not been located. For future efforts, a mechanism for periodically obtaining NTP reports will need to be established so that this source may be used to help identify potential Category I compounds. However, it is important to recognize that the research reports issued by this entity are not routinely and regularly updated as are the US EPA and IARC sources. It is possible that a research report indicating a shift in the status of a compound may be issued the day after the report prepared by the Committee is issued. Because

research reports are produced by a dynamic process, a dynamic mechanism for reviewing such information and making adjustments in the categorization of compounds should be developed.

Considering the aforementioned factors and the fact that EPA and IARC already have reviewed such studies in their determination of potential carcinogens, the Committee recommends that the Regulation be reworded to indicate that a compound **may be considered for inclusion** as a Category I contaminant if positive tests in two species are reported by NTP as opposed to **must be included** if reported to induce cancer in two or more tests (perhaps even of the same species) performed by either NTP or NCI. In this way, in the event that a particular chemical of concern for the state has not been classified or reviewed by either EPA or IARC, the Agency will have the option of reviewing the raw NTP studies to try and make some determination of potential carcinogenicity.

Cancer classification status for each Category I compound is presented in Appendix A, Table 1. In some instances, it was not possible to determine exactly why the compound had originally been placed in this category. A plausible explanation for this lapse is that perhaps the EPA and/or IARC classification has been revised since the compound was added to the list. Thus, it is possible that a compound identified as a Class C by EPA in 1989 has since been reclassified as a D. As of this writing, designation rationale was not clear for the following six compounds: arsine, 1,1-biphenyl, diazomethane, methyl bromide, methyl iodide and propylene imine. Therefore, the Agency recommends reclassifying these compounds as Category II and deriving their ambient air standards using Category II methodology. The proposed revised methodology for Category II compounds includes employing an extra uncertainty factor of 10 to derive the HAAS for those Category II compounds identified as a potential occupational carcinogen by NIOSH or as an A1: Confirmed Human Carcinogen; A2: Suspected Human Carcinogen; or A3: Animal Carcinogen by ACGIH. All of the above compounds, except for 1,1-biphenyl, have been noted as potential occupational carcinogens by NIOSH or ACGIH. The proposed revisions are presented in Appendix A, Table 2.

The derivation of and need for F factors was also investigated by the Committee. After much discussion, the Committee recommends that the assessment procedure be revised and that a conservative absorption factor of 100 percent, meaning an F factor of 1, be employed in the derivation of ambient standards for all Category I compounds. This value assumes 100 percent absorption across the lungs, similar absorption in humans under environmental conditions as seen in the studies which generated the toxicity values and if not so, that the inhalation potency factor has already taken this into account. It further assumes that absorption via inhalation will be similar to that via ingestion for those Category I compounds where an oral potency factor is used as a surrogate in the absence of an inhalation potency factor. The Committee also recommends that a simple mechanism be developed to allow for the presentation of information that may lead to the use of a factor other than 100 percent. For example, the situation may exist where metabolic differences between species or between experimental and ambient conditions dictate than an adjustment be made. It is possible that one species may have enzymes that rapidly deactivate a potentially toxic compound while another species does not.

Where appropriate, the Committee still recommends that, when available, an oral potency factor be employed as a surrogate in the absence of an inhalation potency factor with the following exceptions:

1. Those instances where the type of cancer produced is directly related to the route of exposure, e.g., ingestion results in cancer of the stomach; and

2. Those instances where ingestion and the digestive process causes the parent compound to be converted to a carcinogenic intermediary that would not have occurred if the compound had been inhaled across the lungs.

A simple mechanism allowing for the presentation of information that may lead to the use of an F factor other than 100 percent should be developed for those instances where oral values are used as surrogates.

For several Category I compounds, neither an inhalation nor oral cancer potency factor is available. However, in some instances an inhalation reference concentration (RfC), which is designed to be protective of potential noncarcinogenic health effects that may be associated with long term, continual exposure, has been developed. For these situations, three options were considered:

(1) continue to use the default of 0.01 ug/m^3 as the ambient standard;

(2) employ the RfC as the ambient standard; or

(3) divide the RfC by an additional uncertainty factor to account for the fact that the compound's carcinogenic potential has not been quantified and use this adjusted value as the ambient standard.

The Committee recommends that option (3) be employed using an additional uncertainty factor of ten. The rationale behind this recommendation is that because cancer may be the more sensitive endpoint, an ambient concentration protective of cancer will tend to be more restrictive than one designed to be protective solely of noncarcinogenic effects. Thus, dividing the RfC by an uncertainty factor of ten attempts to decrease the maximum allowable ambient concentration to a level that will be protective of potential carcinogenic effects. Although, precedence exists for using uncertainty factors between three and ten to attempt to account for various model uncertainties, the Committee recommends using ten in an attempt to be adequately health protective. This approach is recommended over using the default value described above in that it is based in part on some chemical specific knowledge. As with the F factor discussion, a simple mechanism allowing for the presentation and consideration of alternative values should be developed for those compounds where this procedure would be employed. A more complete discussion of inhalation reference concentrations may be found in Section II.C.2. For those Category I compounds with no inhalation or oral cancer potency factor or inhalation reference

concentration, the Committee recommends that a default concentration of 0.01 ug/m^3 still be employed as the HAAS. The above hierarchy is depicted in Figure 2.1.

During the course of this review, several changes were noted in the toxicity values associated with several Category I compounds. In some instances, updated inhalation Cancer Potency Factors were available, in other instances, Potency Factors were available for compounds that previously had none. Inhalation reference concentrations are also available for some Category I compounds. A summary of the updated toxicity information is presented in Appendix A, Table 3.
For ease of comparison, compounds are grouped by the type of change noted. Those compounds with no changes are also listed.

Provisional inhalation cancer potency factors and weight of evidence classifications were obtained from the US EPA Technical Support Center for both tetrachloroethylene and trichloroethylene. These values are cited in the Risk Assessment Issue Paper for Tetrachloroethene (NYS, 1996a) and Risk Assessment Issue Paper for Trichloroethene (NYS, 1996b) respectively.

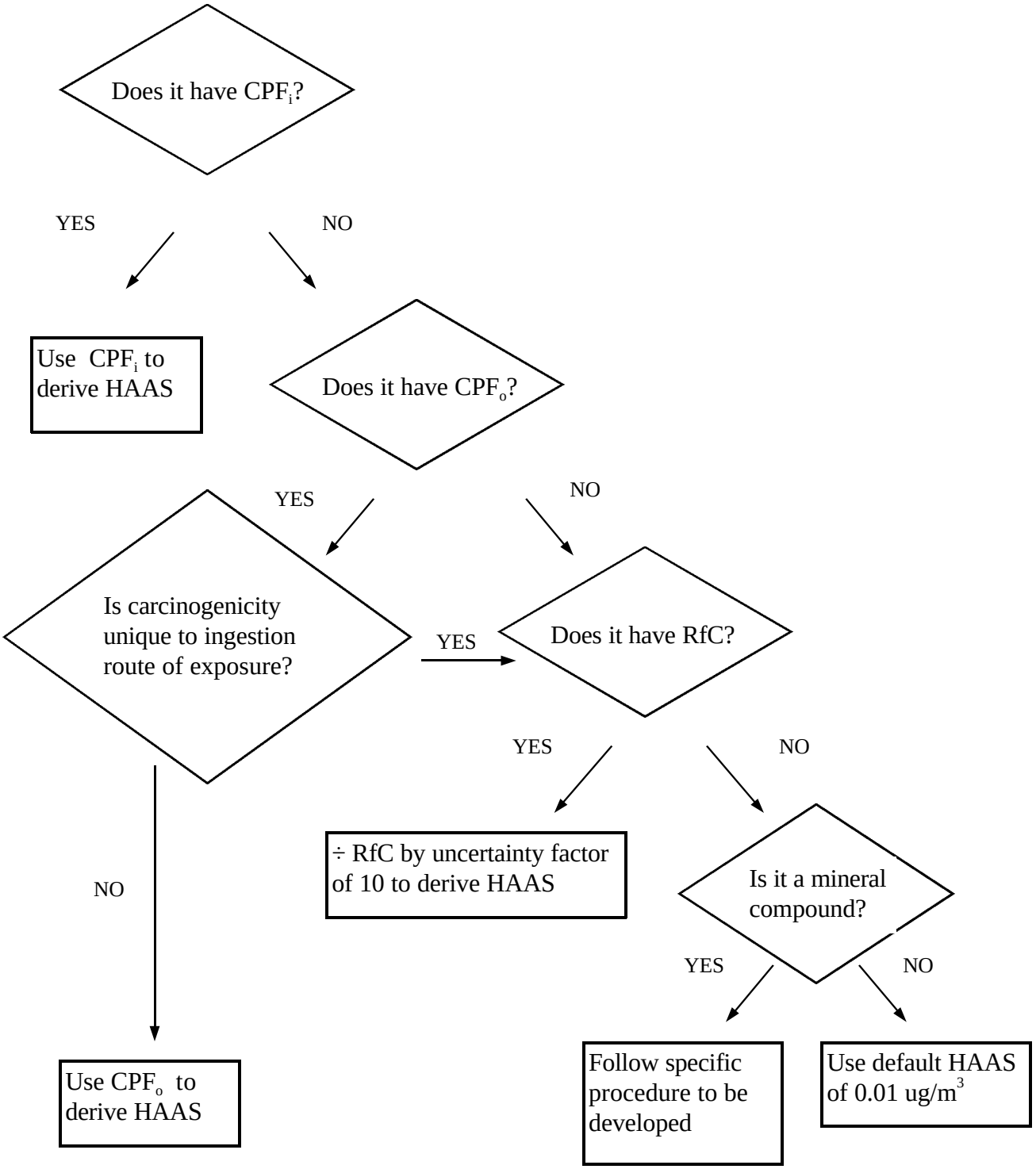
Although the inhalation cancer potency factor noted for benzo(a)pyrene was removed from IRIS by EPA in 1993, the value is still employed by the majority of the risk assessment community because a new value has not yet been released and no clear consensus exists as to why this value should not be used.

While inhalation potency factors are still not available for aniline, dioxane and propylene dichloride, oral cancer potency factors are now available for all three. Thus, the Committee recommends the ambient standard for each should be revised from the current default of 0.01 ug/m^3 using the new oral factor as an inhalation factor surrogate.

The existing standards for allyl chloride and 2,4-dinitrotoluene are based on oral cancer potency factors which are no longer used. No updated oral potency factors were located for either compound. Inhalation potency factors are still not available for these two compounds. However, an RfC is now available for allyl chloride. Per the Committee's recommendation, the standard for allyl chloride should be derived by dividing the RfC by an uncertainty factor of 10. In the case of 2,4-dinitrotoluene, research revealed that several experimental tests have been conducted with a mixture of 2,4 and 2,6 dinitrotoluene and that an oral cancer slope factor of 0.68 $(mg/kg\text{-}day)^{-1}$ has been generated for this combination. Although the 2,6 isomer is thought to be primarily responsible for the carcinogenic action noted, it is recommended that the oral potency factor for the mixture be employed to derive the standard for what is now listed as solely the 2,4 isomer and that the Regulation be revised to note that the compound of concern is a mix of both isomers. The basis of this recommendation is that in real life a combination of the 2,4 and 2,6 isomers, not just one isomer or the other, will be encountered.

RfCs are now available for two compounds (antimony trioxide, and chloroprene) which still do not have associated inhalation or oral potency factors. The standards for these compounds should be revised to one-tenth the appropriate RfC.

Methodology for Deriving Hazardous Ambient Air Standards for Category I Compounds



CPF_i = Inhalation Cancer Potency Factor
CPF_o = Oral Cancer Potency Factor
RfC = Inhalation Reference Concentration
HAAS = Hazardous Ambient Air Standards

Figure 2.1 - See Section II.B.2.

This leaves three Category I compounds (dimethyl sulfate, nickel carbonyl, and o-toluidine) with no inhalation or oral potency factors, or inhalation reference concentration. The standard for these three should remain at the default of 0.01 ug/m^3.

A summary of proposed revised ambient air standards for Category I compounds is presented in Appendix A, Table 4.

There are some compounds which are identified as carcinogenic which would not fit this standard model of derivation because the compound initiates a carcinogenic response due to a unique physical property of the compound. An example would be asbestos. Asbestos dust causes fibrosing inflammation of the lung tissue and cancers. This is thought to be triggered due to the length and shape of the fiber itself, not its ability to diffuse into the blood stream like most of the organic compounds studied for cancer potential. For this reason, the standard methodology discussed above for establishing ambient standards is not appropriate. The Committee recommends establishing ambient standards for asbestos and other mineral compounds, such as crystalline silica, by a different method, first relying on other organizations such as EPA to establish a standard appropriate for the compound, or, in the absence of a standard adopted by EPA, develop a distinct procedure for mineral compounds.

C. Category II and Category III

1. Current Methodology

Per the current Regulations, those Hazardous Air Contaminants not identified as potentially carcinogenic by the US EPA or IARC or reported to induce cancer in two or more tests performed by either NTP or NCI are divided into two categories:

(1) Category II - Noncarcinogens with potential chronic/systemic effects due to long term exposure; and

(2) Category III - Noncarcinogens considered to have primarily short term irritant effects.

For noncarcinogenic health effects, it is generally assumed that some threshold level of toxicity exists i.e., there is some level of exposure below which no adverse health effects are likely to occur. A particular effect is assumed to arise only after a certain minimum fraction of given target molecules have been exposed to the chemical in question. Because the actual threshold level of exposure will vary from individual to individual, assessment of noncarcinogens focuses on estimating a population threshold level.

Several techniques for estimating population thresholds are employed by various entities. Experimentally determined subthreshold doses or estimates of subthreshold doses derived from occupational (work place) studies are commonly relied upon for use in assessing health effects associated with exposure to noncarcinogenic compounds.

At present, the Regulations require that the HAAS for Category II and Category III compounds be based upon ACGIH TLVs which are work place air guidelines. Ambient standards for these compounds are derived by dividing the TLV, when one is available, by one or more uncertainty factors designed to account for some model uncertainties and by a time factor which accounts for the accumulation potential of the compound. Uncertainty factors totaling 10, 100 and in a few instances 1000 are employed depending on the strength and type of exposure data available for each compound.

Because potential chronic/systemic effects are associated with long term exposure to all Category II contaminants, the TLV for each such compound is also divided by a time factor of 4.2 to extrapolate from a standard designed to protect the average healthy worker from adverse health effects to an outdoor air standard designed to protect the general public from adverse effects (i.e., to go from 8 hours per day, 5 days per week to continuous exposures of 24 hours per day, 7 days per week) (ACGIH, 1997). The resulting value is used as an estimate of the level of daily exposure thought to present no significant increase in the likelihood of developing adverse noncarcinogenic health effects over a lifetime even for sensitive subpopulations. Emitters are held to such standards on an annual average basis.

Short term (transient), non-cumulative irritant effects are associated with the majority of Category III contaminants. In these instances, it is not appropriate to adjust the TLV for continuous exposures and rather a time factor of 1 is employed along with the appropriate uncertainty factors. Sources are held to these standards on an eight hour average basis.

A few Category III compounds are associated with both short term irritant effects and also some type of extended, but not chronic, effect. For example, the effect noted may dissipate a few days after exposure has stopped. In view of this, a conservative, health protective approach has been taken and along with the appropriate uncertainty factors, a time factor of 4.2 is employed to extrapolate the TLV to a continuous level as described above. For these Category III compounds, emitters are held to the standard on a twenty-four hour average basis.

The current Regulations dictate that if no ACGIH TLV is available for a particular compound then, if available, an occupational standard established by either the National Institute for Occupational Safety and Health (NIOSH) (termed Recommended Exposure Limits or RELs) or the US Department of Labor Occupational Safety and Health Administration (OSHA)(termed Permissible Exposure Limits or PELs) shall be used as a surrogate in the ambient standard derivation process. If no occupational value is available from any of these sources, a standard is issued based on an evaluation of the toxicity information available for the compound in question.

2. Recommended Revisions

For the purposes of this report, the occupational standards employed for each Category II and III contaminant were reviewed, researched and where appropriate updated. Current occupational standards were obtained from ACGIH, NIOSH and OSHA. In addition, toxicity information was

obtained from the US EPA IRIS database and HEAST. A summary of carcinogenic evaluations presented in the IARC monographs and NTP abstracts were also searched for toxicity information.

During the course of this review, several changes were noted in the occupational standards and toxicity values available for several Category II and Category III compounds. For example, of the hundreds of compounds in these two categories, over a dozen now meet the criteria for classification as a Category I contaminant and should therefore be reclassified as Category I contaminants. Once reclassified, a HAAS for each such compound should be derived using the procedure recommended for all Category I contaminants outlined in Section II.B.2 above. A list of all such compounds is presented in Appendix B, Table 1.

Crystalline silica has been identified by IARC as a potential occupational human carcinogen. Like asbestos, cancer caused by occupational exposure to crystalline silica does not fit the basic cancer models used to derive standards for the other potential carcinogens. For this reason, the Committee recommends an alternative procedure be developed to derive a standard protective of cancer for mineral compounds if EPA has not already done so. In the case of crystalline silica, the EPA has not identified crystalline silica as a potential carcinogenic compound and therefore no appropriate standard currently exist. Until the Agency is able to develop an appropriate derivation process for mineral compounds, the Committee recommends the current standard be retained for crystalline silica.

Inhalation reference concentrations (RfCs), which are designed to be protective of potential noncarcinogenic health effects given long term, continuous exposure, are now available on IRIS and in HEAST for several Category II and Category III compounds. In general, an RfC estimates the level of a particular compound in ambient air to which one could be exposed on a twenty-four hour a day basis and not experience a significant increase in the likelihood of developing adverse noncarcinogenic health effects over a lifetime, even for sensitive subpopulations.

WHAT IS AN RfC?

Inhalation reference concentrations are designed to be protective of potential noncarcinogenic health effects that may be associated with long term, continuous exposure. An RfC estimates the level of a particular compound in ambient air to which one could be exposed on a 24 hour a day basis and not experience a significant increase in developing adverse noncarcinogenic health effects over a lifetime, even for sensitive populations. For noncarcinogenic health effects, it is assumed that some threshold level of toxicity exists i.e., there is some level of exposure below which no adverse health effects are likely to occur.

RfCs are considered to take into account both potential respiratory and non-respiratory effects and address both pharmacodynamics and pharmacokinetics. Some RfCs have been derived based on studies of occupationally exposed groups of people, while others are based upon results of experimental studies with laboratory animals extrapolated to humans. As with all toxicity values on the US EPA IRIS database and HEAST document, RfCs are only included after a

"...comprehensive review of chronic toxicity data [is conducted] by work groups composed of US EPA scientists..." (IRIS, 1997). These values are typically derived by the US EPA Reference Dose/Reference Concentration (RfD/RfC) Workgroup (EPA, 1994).

The RfC methodology and RfCs themselves were not widely available at the time the methodologies for Category II and Category III compounds were developed in 1989. Therefore, the adjusted occupational standard approach was a default, more subjective approach developed to try and approximate the same ambient level that is now estimated in a more scientifically defensible manner by the RfCs.

Considering the above information, for Category II and Category III contaminants, it is recommended that where available, the RfC be used as the HAAS rather than employing an adjusted occupational standard. In those instances where using an RfC would result in a greater HAAS than the current approach, an investigation will be conducted to ensure that the RfC takes into account the same uncertainties and toxic endpoints addressed by the occupational standard.

For those Category II compounds without an RfC, the Committee recommends that the existing adjusted occupational standard methodology described above be used to derive an appropriate ambient standard. However, it is recommended that the Regulation be revised to require that the **most conservative** (lowest) available occupational standard (ACGIH TLV, NIOSH REL or OSHA PEL) for a chemical be used in the equation to derive an appropriate HAAS. It is also recommended that a simple mechanism be developed to allow for the consideration of updated, scientific information that supports using an occupational standard other than the most conservative one as the basis of the HAAS. The current Regulation allows a PEL or REL to be used only in the absence of a TLV.

Once an adjusted work place value is derived, it is recommended that it be compared to the Acute Exposure Guideline Level 2 (AEGL2) for the contaminant in question. The AEGL2 represents an airborne concentration of a substance at or above which it is predicted that the general population, including susceptible but not hyper-susceptible individuals, could experience irreversible or other long lasting effects or impaired ability to escape (FR, 1997). For example, exposure to a certain elevated amount of ammonia (a very strong eye irritant) while not producing a long lasting effect, could impair vision to the point where the ability to escape would be hindered. AEGLs are derived by the National Advisory Committee on Acute Exposure Guideline Levels and information on these values is readily available through the State of Vermont Department of Health.

If the adjusted work place value is at least ten times greater than the corresponding AEGL2, the Committee recommends that the basis of the occupational standard itself undergo further investigation to ensure that it is adequately protective of public health.

If no occupational value (TLV, PEL and/or REL) is available then, as before, a standard should then be established based on an evaluation of the toxicity information available for the compound in question.

The above hierarchy is depicted in Figure 2.2.

A similar type of hierarchy is recommended for Category III contaminants. As described for Category II contaminants, it is recommended that, where available, inhalation reference concentrations (RfCs) be employed as twenty-four hour ambient standards. Unlike Category II compounds, because the majority of chemicals in this category are considered to be short-term irritants, it is recommended that in the absence of an RfC, a corresponding AEGL1 be employed as an eight hour ambient standard. An AEGL1 represents an airborne concentration at or above which it is predicted that the general population, including susceptible but not hyper-susceptible individuals, could experience notable discomfort (FR, 1997). Airborne concentrations below the AEGL1 represent exposure levels that could produce mild odor, taste or sensory irritation.

In the event that no AEGL1 is available for a compound, the Committee recommends that, if available, a corresponding AEGL2 be employed as the HAAS. If no RfC, AEGL1 or AEGL2 is available, then the ambient standard should be set using the adjusted occupational method described above for Category II compounds.

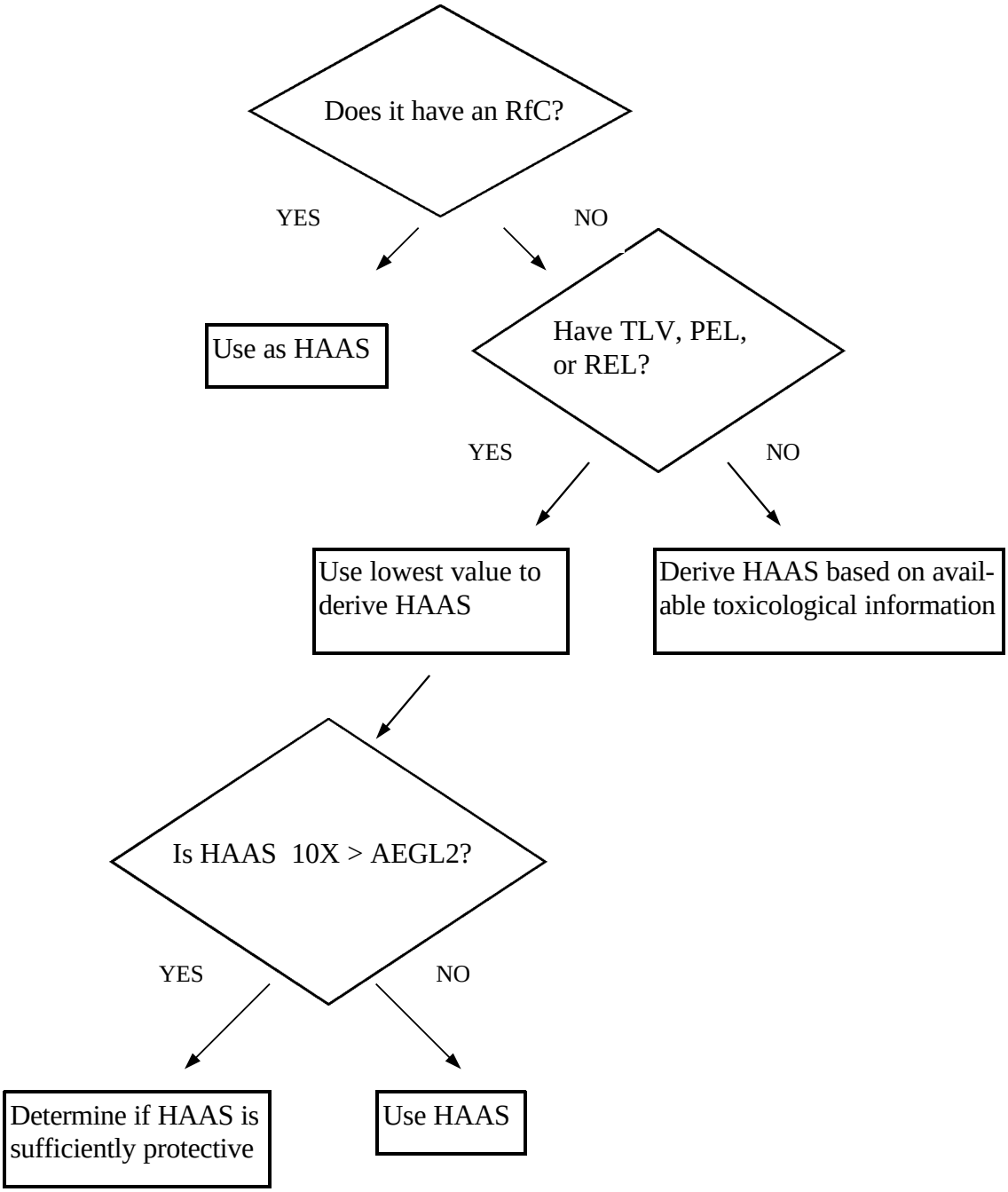
If no TLV, PEL or REL is available then, as before, the ambient standard should then be issued based on an evaluation of the toxicity information available for the compound in question.

The hierarchy for Category III contaminants is depicted in Figure 2.3.

The one caveat to the proposed revised methodologies described above is that as a conservative, health protective measure, an extra uncertainty of ten was applied to derive the HAAS for those Category II and Category III compounds identified as a potential occupational carcinogen by NIOSH or as A1: Confirmed Human Carcinogen; A2: Suspected Human Carcinogen; or A3: Animal Carcinogen by ACGIH. Proposed standards derived in this manner are so noted on the tables provided in Appendix A and Appendix B. Of particular note, and as depicted in Appendix A, Table 2, are arsine, diazomethane, methyl bromide, methyl iodide, and propylene imine.

A summary of all Category II and Category III compounds, updated toxicity information, and occupational standards and how the corresponding ambient standard would change based on the proposed revised methodologies is presented in Appendix B, Tables 2 through 5.

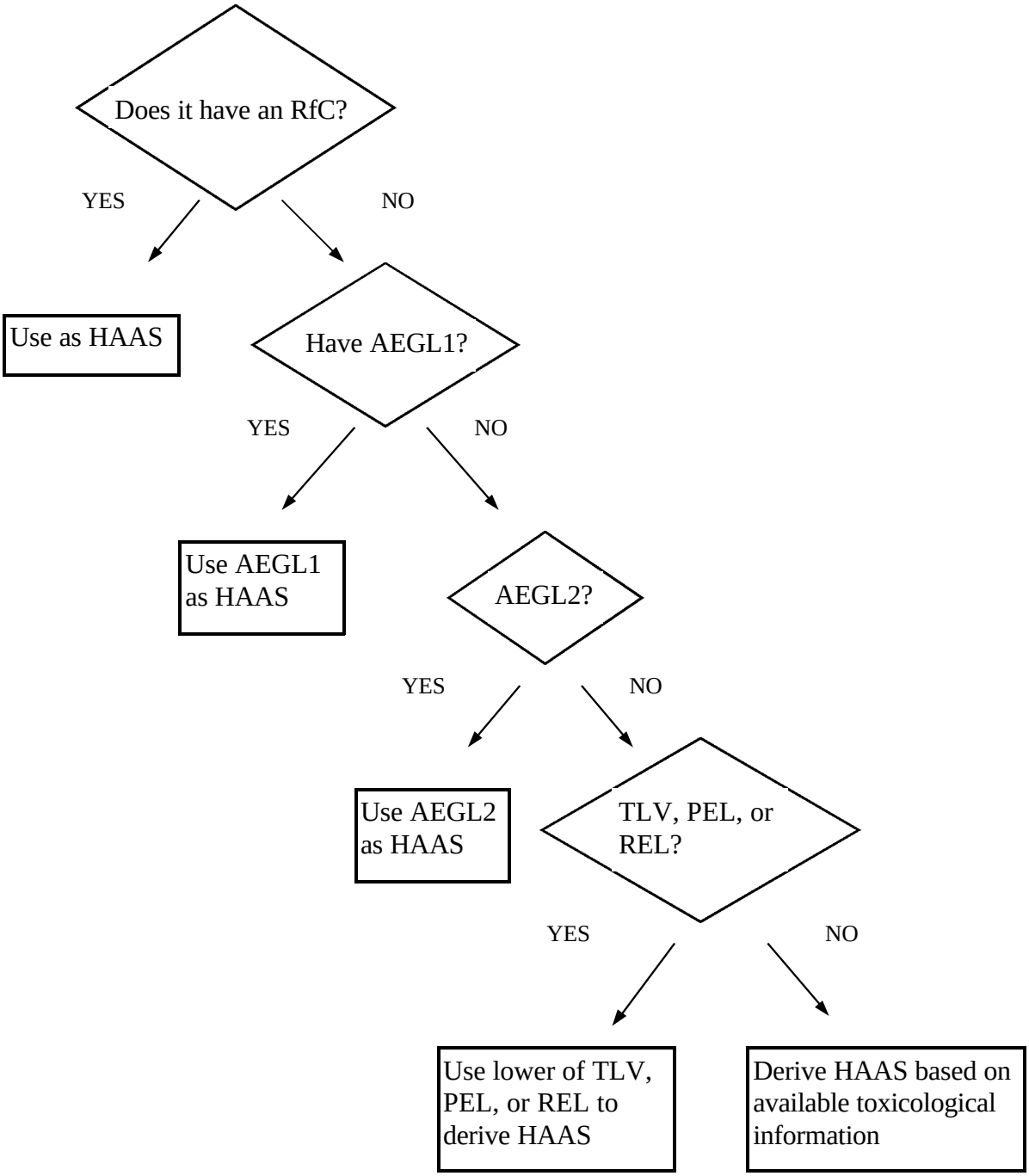
Methodology for Deriving Hazardous Ambient Air Standards for Category II Compounds



RfC = Inhalation Reference Concentration
HAAS = Hazardous Ambient Air Standard
TLV = Threshold Limit Value
PEL = Permissible Exposure Limit
REL = Recommended Exposure Limit
AEGL2 = Acute Exposure Guideline Level

Figure 2.2 - See Section II.C.2.

Methodology for Deriving Hazardous Ambient Air Standards for Category III Compounds



RfC = Inhalation Reference Concentration
HAAS = Hazardous Ambient Air Standard
AEGL1 and AEGL2 = Acute Exposure Guideline Level
TLV = Threshold Limit Value
PEL = Permissible Exposure Limit
REL = Recommended Exposure Limit

Figure 2.3 - See Section II.C.2.

III. Measurements of Hazardous Air Contaminants in Ambient Air

A. Background

The Committee has reviewed the ambient air monitoring data from 1993 to 1995 in order to compare the ambient levels to the ambient air standards. The ambient air monitoring data is collected as part of the Agency's Air Monitoring Program. This section discusses the air monitoring program and includes information regarding (1) compounds monitored, (2) statistics of the data, (3) detection limit issues, (4) compounds exceeding the ambient air standards, and (5) local compounds versus transported compounds. A more detailed discussion of compounds exceeding the standard and compounds of public concern is found in Section IV.

1. Compounds

The air toxics monitoring program was developed by the Hazardous Air Contaminant Monitoring Committee in order to determine the ambient levels of numerous air toxics in accordance with the objectives of Act 92 of the 1993 legislative session, section 575. The program began in 1993 with collection of data on Volatile Organic Compounds (VOCs) and has since expanded to include carbonyl compounds and semi-volatile compounds (Polychlorinated Biphenyls (PCBs), Polynuclear Aromatic Hydrocarbons (PAHs), Polychlorinated Dibenzo-P-Dioxins/Furans (PCDDs/PCDFs)). The VOC and carbonyl compounds monitored are listed in Appendix C, Table 1 (VOC and Carbonyl Compounds Monitored).

2. Sites

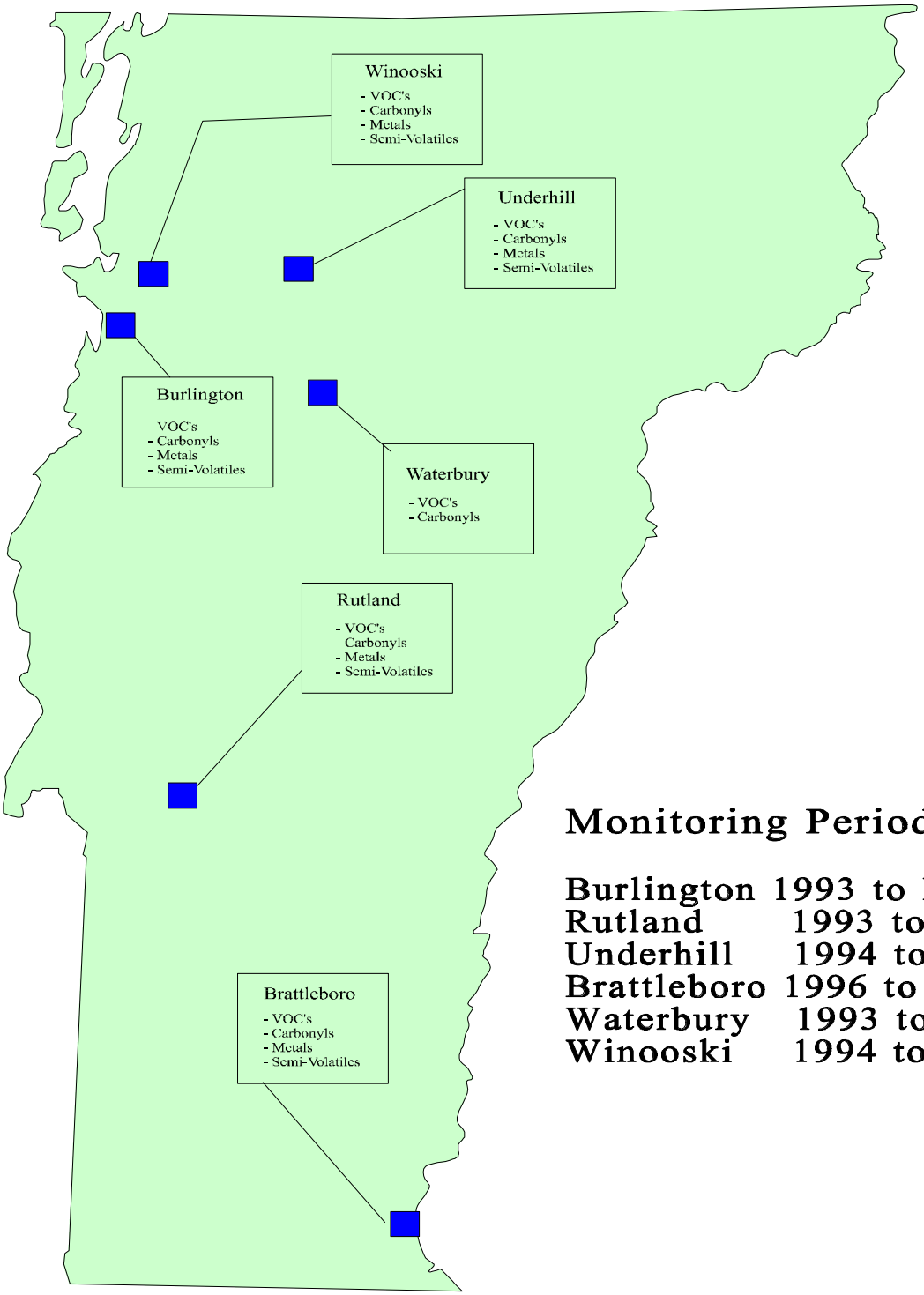
Monitoring sites were selected by the Agency with the concurrence of the Hazardous Air Contaminant Monitoring Committee to provide information on a variety of locations, from urban to rural. Burlington, Brattleboro and Rutland represent urban locations with high population exposure. Winooski is a suburban location and Waterbury represents a village location. The Underhill site provides background data from a rural location on the side of Mt. Mansfield (see Figure 3.1). Sources of air toxics in urban areas include industry, motor vehicles as well as a residential contribution.

3. Methods

Samples were collected by different methods for each class of contaminants.

VOC samples were collected over 24 hours in evacuated stainless steel canisters (EPA Method TO-14) and subsequently analyzed by gas chromatography/mass spectrometry. Carbonyl samples were collected over 24 hours on a treated silica gel (EPA Method TO-11) and then analyzed by high pressure liquid chromatography. Samples for both VOCs and carbonyls were collected every 12 days.

State of Vermont
Air Contaminant Monitoring Sites
Figure 3.1 (see Section III.A.2)



Monitoring Period

Burlington 1993 to Present
Rutland 1993 to Present
Underhill 1994 to Present
Brattleboro 1996 to Present
Waterbury 1993 to 1994
Winooski 1994 to Present

The three classes of semi-volatiles (PCB, PCDF/PCDD and PAH) were collected simultaneously over 72 hours using modified EPA toxic organic methods (TO-4/EPA Modified 680 (PCB), TO-9/EPA 8290 (PCDF/PCDD) and TO-13/Modified CARB 429 (PAH)) and analyzed by high resolution gas chromatography/high resolution mass spectrometry (HRGC/HRMS) and high resolution gas chromatography/low resolution mass spectrometry (HRCG/LRMS) . The semi-volatile data was collected quarterly.

Mercury samples were collected by the University of Vermont every Wednesday and every sixth day (to follow national EPA and IMProVE [Interagency Monitoring of Protected Visual Environments] and regional NESCAUM [Northeast States for Coordinated Air Use Management] particulate sampling schedules. Vapor and particulate samples were collected for 24 hours (8 am cycle) on gold coated sandpaper traps and glass-fiber particulate filters, respectively (Scherbatskoy et al; 1996).

B. Discussion of Data

1. Summary of Statistics and Non-detects

Summary statistics for VOC and carbonyl compounds have been developed for the1993-1995 data. The summary statistics present the mean, median, standard deviation, maximum, minimum and range for 41 of the 72 compounds monitored. The VOC and carbonyl summary statistics for 1993-1995 are located in Appendix C, Tables 2 and 3 **(**VOC Summary Statistics and Carbonyl Summary Statistics). For VOCs and carbonyls the annual average concentration was determined by averaging the 30 samples collected each year. A summary of the semi-volatile data is shown in Appendix C, Table 4. The Agency will continue to develop summaries of the air toxics data collected. These summaries are available to the public and will be updated annually.

Numerous VOC and carbonyl compounds were not present in the atmosphere at sufficient levels to be detectable during sample analysis. These samples are considered “non-detects” because their concentration is less than their respective method detection limit (MDL). Summary statistics were only calculated for the 41 compounds with an average percent non-detect less than 95%. A list of the compounds and their average percent non-detects is shown in Appendix C, Table 1. When calculating the summary statistics, individual sample values less than the detection limit were assigned a numerical value of one half the detection limit (½ MDL) for that compound. Additional statistics, high and low mean, were calculated to represent the uncertainty due to the non-detects. The high mean uses the detection limit for all non-detects and the low mean uses zero for all non-detects, thus bracketing the true mean value.

2. Non-Gaussian or Non-Parametric Statistics

Analysis of the frequency distribution of the VOC and carbonyl data showed that the data did not follow a normal distribution, nor did they consistently follow any other particular pattern. This lack of normalcy requires the use of non-parametric statistics rather than the typical gaussian or normal statistics. Non-parametric statistics differ from gaussian statistics in that they have less power,

requiring more data in order to reach a statistical conclusion. However, non-parametric statistics have the capability of identifying differences in highly variable data. Different statistical techniques are required for analysis on non-parametric data, limiting the analysis tools available to the researcher. Finally, the median is a better indicator of the central tendency of the data set then the mean for non-parametric data. It is important to consider the type of statistics.

3. Limitations and Uncertainties

There are limitations and uncertainties inherent in the collection and analysis of any statewide ambient air monitoring program. These are due to the number of monitoring sites, monitoring frequency and duration as well as analytical limitations, such as the detection limit. Monitoring every 12 days for 24 hours at discrete locations cannot provide a complete picture of ambient air toxic concentrations for the whole state. The 12 day sampling frequency would not detect isolated infrequent emissions. Twenty four hour composite samples can not represent peak values due to the averaging inherent in a 24 hour composite sample. The finite number of monitoring sites provides a limited picture of contaminant concentration across Vermont.

The detection limit for each compound can also create uncertainty in the data collected. Non-detects in the data generate uncertainty as their true values cannot be determined. This uncertainty can be reduced by using the high and low mean to bracket the true mean of the data set. Using the median, rather than the mean, as an indicator of the central tendency of the data set also reduces the uncertainty due to non-detects. For some compounds the state standard is less than the detection limit which makes it difficult to determine if the compounds exceed the standard. These compounds are listed in Table 3.1.

There is also uncertainty inherent in the process of collection and laboratory analysis of the air toxics samples.

compound name	CAS #	HAAS	averaging	ESE MDL	Radian MDL	Percent
			time	7/93 - 3/95	8/95 - 12/95	Non-Detect
benzene	71-43-2	0.12	annual average	0.10	0.77	11.52
bromoform	75-25-2	0.01	annual average	1.45	0.83	100
1,3-butadiene	106-99-0	0.035	annual average	0.15	0.33	41.01
carbon tetrachloride	56-23-5	0.067	annual average	0.19	0.44	14.75
chloroform	67-66-3	0.043	annual average	0.24	0.29	89.86
chloroprene	126-99-8	0.01	annual average	0.18	0.18	100
ethylene dichloride	107-06-2	0.038	annual average	0.24	1.05	100
hexachlorobutadiene	87-68-3	0.045	annual average	0.53	na	100
methyl bromide	74-83-9	0.01	annual average	0.27	0.70	98.62
methyl chloride	74-87-3	0.01	annual average	0.21	0.81	7.37
propylene dichoride	78-87-5	0.01	annual average	0.18	0.18	99.54
1,1,2,2-tetrachloroethane	79-34-5	0.017	annual average	1.99	1.10	100
1,1,2-trichloroethane	79-00-5	0.061	annual average	0.33	0.27	99.54
1,2,4-trimethyl benzene	95-63-6	0.15	annual average	0.49	na	35.63
vinyl chloride	75-01-4	0.20	annual average	0.18	0.28	100

Table 3.1-See Section III.B.3
Compounds with HAAS Less Than Detection Limit

C. Observations and Conclusions

Analysis of the VOC and carbonyl data shows that 9 compounds exceed the state standard for annual average concentration (see Table 3.2). These compounds are: formaldehyde, benzene, 1,3-butadiene, carbon tetrachloride, methylene chloride, chloroform, tetrachloroethylene, methyl chloride and 1,2,4-trimethyl benzene (see Figure 3.2). Section IV of this report provides greater detail regarding the ambient levels of these compounds. Some compounds appear to be locally generated while others appear to be regional or transported. Locally generated compounds are characterized by fairly short atmospheric persistence and show increased concentrations with increased urbanization (see Figure 3.3). The regional or transported compounds persist for longer periods in the atmosphere and show no statistically significant variation in median values between all sites, both urban and rural (see Figure 3.4).

Compound	HAAS	Max. Annual Average			% Data Sets
	ug/m^3	ug/m^3	Site	Year	Exceeding HAAS
benzene	0.12	4.05	Burlington	1994	100%
benzo-a -pyrene	0.0003	0.00053	Burlington	1996	50%
1,3-butadiene	0.035	0.95	Burlington	1994	100%
carbon tetrachloride	0.067	0.72	Rutland	1994	100%
chloroform	0.043	0.22	Burlington	1995	100%
formaldehyde	0.08	10.16	Winooski	1995	100%
methyl chloride	0.01	1.39	Burlington	1994	100%
methylene chloride	2	8.41	Rutland	1994	75%
PCBs	0.00081	0.00082	Winooski	1996	14.29%
tetrachloroethylene	0.41	0.62	Burlington	1994	8.33%
1,2,4-trimethyl benzene	0.15	2.8	Burlington	1994	100%

Table 3.2-See Section III.C.
Compounds Exceeding Vermont Standard

The semi-volatile data (Appendix C, Table 4) shows that some compounds approach or exceed the Vermont standard (benzo-a-pyrene, and PCBs). These results indicate the need for the development of a comprehensive monitoring program for these compounds. The current program has collected one integrated sample per year for the semi-volatile compounds, providing useful information regarding which compounds are present in the atmosphere at levels near the state standard. The results of this initial screening indicate which compounds need to be monitored more closely by collecting numerous samples throughout the year with collection methods specific to the compound being analyzed. The results of the proposed comprehensive, compound specific monitoring program would provide data for comparison to the state standard.

Figure 3.2, Compounds Exceeding Vermont Hazardous Ambient Air Standard (HAAS) Burlington, Vermont 1994 Ambient Data (See Section III.C)

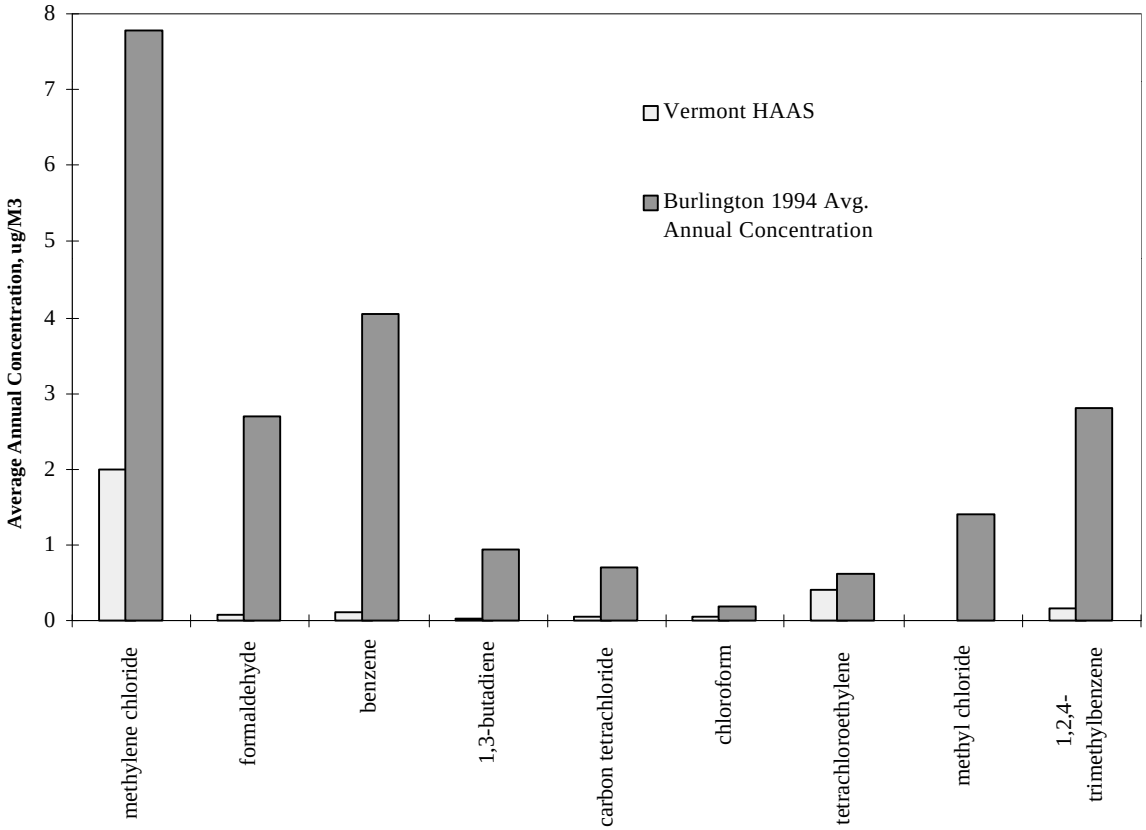


Figure 3.3, Example of Locally Generated Compounds
1994 Vermont Ambient Data, (See Section III.C)
Concentrations increase with urbanization.

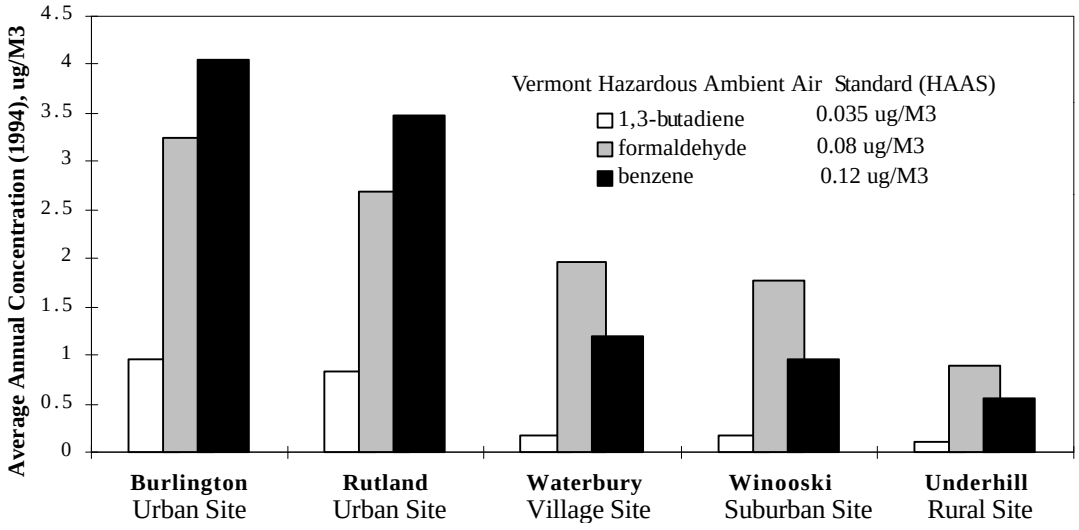
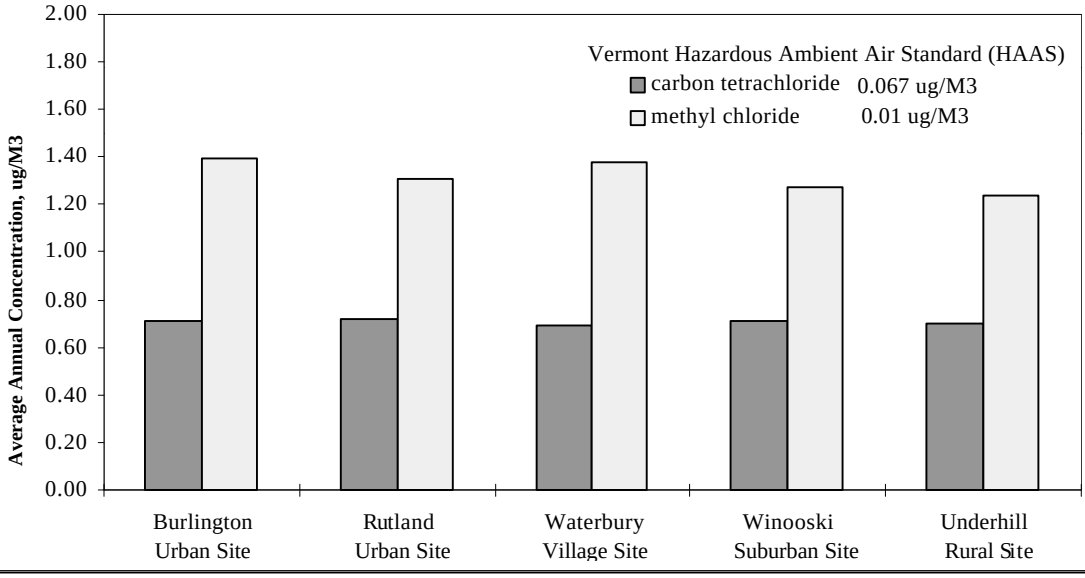


Figure 3.4, Example of Regional or Transported Compounds
1994 Vermont Ambient Data, (See Section III.C)
Concentrations do not increase with urbanization.



D. Recommendations

The Hazardous Air Contaminant Monitoring Committee recommends continued monitoring of the VOC and carbonyl compounds to detect changes due to new sources and to determine the effectiveness of reduction programs for target compounds. Some VOC and carbonyl compounds could be eliminated from the program as they exist at low concentrations far below their respective standards. Elimination of these compounds will be addressed if it results in a cost savings for the program. Those compounds that exceed or approach their standards should continue to be monitored. Changes in the monitoring program are recommended for those compounds that can be harmful during short, acute exposures. These compounds have eight hour standards and the current 24 hour sampling duration is not effective for determination of eight hour peak values. This would require either sampling for shorter periods of time or changing the standard to reflect the current monitoring capability.

Monitoring of VOCs and carbonyls is currently done at five sites (Burlington, Brattleboro, Rutland, Underhill and Winooski). Four (Burlington, Brattleboro, Underhill and Rutland) of these sites should continue to collect data that would show changes over time. The fifth site would be a roving site, being moved to other locations in the state as needed to address areas of concern and to better characterize a cross section of the state. Monitoring at the roving sites would be conducted for a minimum of 12 months to provide sufficient data for analysis.

The current methodologies for analysis and collection of semi-volatiles need to be reconsidered due to quality assurance issues with the extraction and analysis of the samples. It is appropriate to consider different sampling methodologies due to problems with the current approach. The current method collects a composite sample of all three semi-volatile groups simultaneously. This leads to analysis problems caused by reduced sample amounts and interference between the compounds collected. A standard collection method will collect samples of the three classes of semi-volatiles independently, eliminating the problems associated with the current composite method. Upon development of a revised collection and analysis method, monitoring of semi-volatile compounds will be continued to more accurately determine the levels of these compounds in the ambient air.

Further work needs to be done to develop lower detection limits for those compounds with standards less than the detection limit. This will require consideration of other analytical techniques, as they may have lower detection limits for some compounds.

IV. Priority Compounds

The following twelve compounds were identified as priority compounds because they either exceed the ambient air standard or they are of concern to the public. For each compound, the committee has provided a detailed discussion of ambient air levels, sources of the compound, whether the compound is locally generated or transported from other areas, and uncertainties of the data. In addition, a discussion is provided on how the ambient air standard will change based on the proposed revised methodology. Finally, in order to determine if the revised standard is health protective, the committee discusses the biological activities and health concerns to illustrate the nature and risk from different exposures, and the populations of concern. Section V provides a discussion on management options for reducing emissions from these compounds.

A. Category 1

1. Benzene

a. Discussion

Benzene was selected as a priority compound because the annual average concentrations always exceed the current standard at every site, every year. The current HAAS for benzene is 0.12 ug/m^3 (annual average). The highest annual average concentrations were observed in Burlington: 2.83 ug/m^3, 4.05 ug/m^3 and 2.62 ug/m^3 for 1993, 1994 and 1995. Annual average concentrations of benzene in Rutland were less than those in Burlington (1.76 ug/m^3 in 1993, 3.48 ug/m^3 in 1994 and 1.74 ug/m^3 in 1995). Waterbury and Winooski annual average concentrations ranged from 0.94 ug/m^3 to 1.19 ug/m^3. Underhill had the lowest annual average concentrations (0.54 ug/m^3 to 0.56 ug/m^3)(see Figure 4.1). Average annual concentrations of benzene in US urban areas range from 4 to 7 ug/m^3 (EETE, 1995). Benzene levels were too low to be detected in 12% of the samples collected. This is not significant in relation to the standard as all the low means for benzene exceed the HAAS.

Benzene in the atmosphere comes from numerous sources. Motor vehicles are considered a significant source of benzene. Benzene is present in both evaporative emissions and in exhaust emissions from motor vehicles (EETE, 1995). Refueling of motor vehicles is another source of benzene (ATSDR, 1996a). Benzene is also released by industries in the state and is a component of fossil fuel emissions, including wood stoves. Benzene in Vermont appears to be locally generated as the highest concentrations are observed in urban areas and the concentrations decrease as the sites become more rural. Benzene has an atmospheric half life of 10-12 days (Kao, 1994).

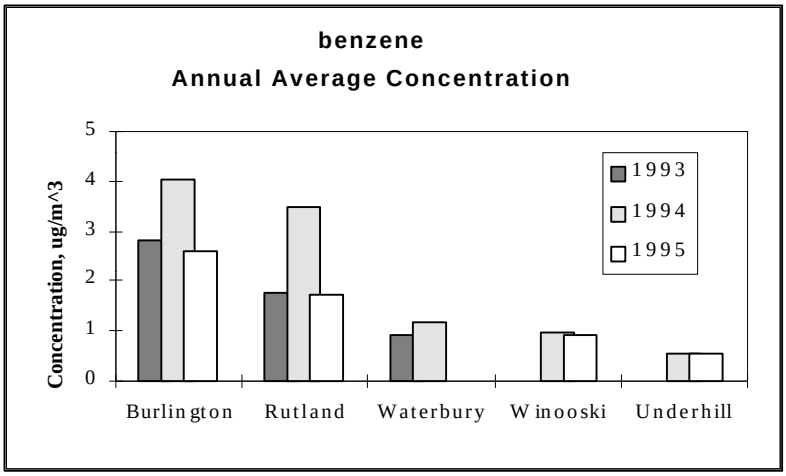


Figure 4.1-Annual average ambient air concentrations of benzene in micrograms per cubic meter (ug/m^3) at Vermont Monitoring Sites from 1993 to 1995. The Hazardous Ambient Air Standard (HAAS) is .12 ug/m^3. See section IV.A.1.

b. Limitations and Uncertainties

There are no significant uncertainties regarding non-detects for benzene. Benzene has numerous sources and the proportion each contributes to the total benzene concentration is not known. The proximity of some monitoring sites to area sources creates some uncertainty regarding the ambient concentration. Both Burlington and Rutland have gas stations in close proximity to the monitoring sites. Refueling operations could affect the ambient concentrations at these sites.

c. Implications of Revised Standard

The proposed revised standard for benzene will remain the same at 0.12 ug/m^3. Emissions will continue to exceed the standard unless measures are taken to reduce emissions. As stated above, sources of benzene emissions include automobiles, refueling at the gas station, industry, and wood stoves.

d. Health Concerns:

One way benzene may enter the body is by inhalation of contaminated air into the lungs. Studies with human volunteers indicate that inhaled benzene vapors are rapidly absorbed from the lungs into the bloodstream where they can then be transported throughout the body. The highest absorption (up to eighty percent) has been noted during the first few minutes of exposure (ATSDR, 1996a). Studies where human volunteers were exposed to high levels of benzene vapors for a few hours have noted that approximately half of the vapors inhaled are retained and absorbed from the lungs (EM, 1995).

Benzene is soluble in fat. Absorbed benzene can temporarily be stored (accumulate) in bone marrow and fat and slowly be re-released to the blood stream. The amount of body fat and degree of physical

activity influence how quickly stored benzene will be re-released.

About half of the benzene absorbed into the blood stream leaves the body unchanged in exhaled air within about 36 hours after exposure has stopped (EM, 1995). Here again, timing is influenced by the amount of body fat and degree of physical activity (EM, 1995). The remaining fifty percent is broken down into other compounds (metabolites) in the liver and bone marrow. Some of these metabolites are believed to be responsible for some of the adverse blood effects associated with long term inhalation of high levels of benzene vapors (EM, 1995). Most of the metabolites leave the body through the urine within 48 hours after short term exposure stops (ATSDR, 1996a).

The majority of information on potential health effects that may be associated with inhalation of benzene vapors comes from studies of workers who were exposed to high levels of benzene vapors for extended periods of time. The general public is not expected to experience such high exposures.

Brief exposure (less than 10 minutes) to highly elevated levels of benzene vapors (about 20,000 ppm, 64,000,000 ug/m^3) can result in death (ATSDR, 1996a). Inhalation of between 700 and 3,000 ppm (2,200,000 to 9,600,000 ug/m^3) can significantly depress the central nervous system and result in dizziness, drowsiness, rapid heart rate, headaches, tremors, confusion and unconsciousness (ATSDR, 1996a; EM, 1995). In many instances, recovery from such central nervous system effects has been noted once exposure stops and a person starts to breathe fresh air (ATSDR, 1996a). However, recovery time will vary depending on the amount of benzene inhaled and stored in the body.

Studies of people, especially workers, indicate that inhalation of elevated levels of benzene for long periods of time may damage the tissues that form blood cells, especially the bone marrow. Anemia, excessive bleeding and leukemia (cancer of the blood forming organs) may result. Blood processes may return to normal if exposure has been limited and the person returns to breathing fresh air (ATSDR, 1996a). Prolonged exposure to elevated levels of benzene may also weaken the immune system thus decreasing the body's ability to fight infection and perhaps ward off cancer. Exposure to benzene has also been associated with damage to the body's genetic material (chromosomes).

Limited studies of women exposed to elevated levels of benzene, and a mix of other volatile chemicals, in the work place suggest that such exposure may effect the reproductive organs and perhaps impair fertility. However, because exposure was to more than one chemical at a time, it is not known which chemical or combination of chemicals may be responsible for the health effects noted. The impact of such exposure on a developing human fetus is not known.

Ingested benzene is also rapidly absorbed into the blood stream. Ingestion of food and/or drink containing high levels of benzene may result in vomiting, stomach irritation, dizziness, sleepiness, convulsions, rapid heart rate, coma, and death (ATSDR, 1996a). The potential health effects that may be associated with long term consumption of foodstuffs containing lower levels of benzene have not been identified. However, long term experimental ingestion studies with laboratory animals have noted damage to the blood and immune system and in some instances, result in cancer (ATSDR, 1996a).

Dermal contact with benzene can cause skin irritation and result in redness and sores. Benzene can also cause eye irritation and corneal damage if it comes in contact with the eyes.

Long term experimental studies with laboratory animals ingesting food and drink with elevated levels of benzene noted damage to the blood and immune systems and in some instances increased incidences of cancer (ATSDR, 1996a). Experimental studies with pregnant laboratory animals breathing in large amounts of benzene for long periods of time have noted damage to the developing fetus. It is not known if human fetuses may be similarly effected.

Benzene has been classified as Class A: Known Human Carcinogen by the United States Environmental Protection Agency and as Group 1: Human Carcinogenic by the International Agency for Research on Cancer.

2. 1,3-Butadiene

a. Discussion

1,3-butadiene was classified as a priority compound because the annual average exceeded the HAAS at all sites, every year. The current Vermont HAAS for 1,3-butadiene is 0.035 ug/m^3 (annual average). Burlington has the highest levels with annual average concentrations of 0.64 ug/m^3, 0.95 ug/m^3 and 0.47 ug/m^3 for 1993, 1994 and 1995, respectively. Rutland had annual average concentrations of 0.32 ug/m^3, 0.84 ug/m^3 and 0.34 ug/m^3 for 1993-1995. Waterbury and Winooski annual average concentrations were less than those in Burlington and Rutland, ranging from 0.17 ug/m^3 to 0.20 ug/m^3. Underhill had the lowest concentrations, 0.11 ug/m^3 in 1994 and 0.13 ug/m^3 in 1995 (see Figure 4.2). Annual average concentrations of 1,3-butadiene in urban areas range from 0.2 to 1.0 ug/m^3 (EETE, 1995). Although 1,3-butadiene concentrations were below the detection limit for 41% of the samples collected, non-detects are not a significant issue for 1,3-butadiene. An examination of the low mean (calculated by entering a zero for all non-detects) shows that the low mean exceeds the state standard for every site, every year, except Underhill 1995.

The primary source of 1,3-butadiene is motor vehicles. 1,3-Butadiene is formed in vehicle exhaust due to incomplete combustion of fuel (EETE, 1995). Other sources of 1,3-butadiene are waste incinerators and wood fires (ATSDR, 1992). The 1,3-butadiene concentrations observed appear to be locally generated as the concentrations are highest in the urban areas of Burlington and Rutland with levels decreasing as the sites become more rural. 1,3-Butadiene has a short atmospheric half-life of 4-6 hours (Kao, 1994), which also indicates that observed levels are locally generated, as opposed to transported from outside the state.

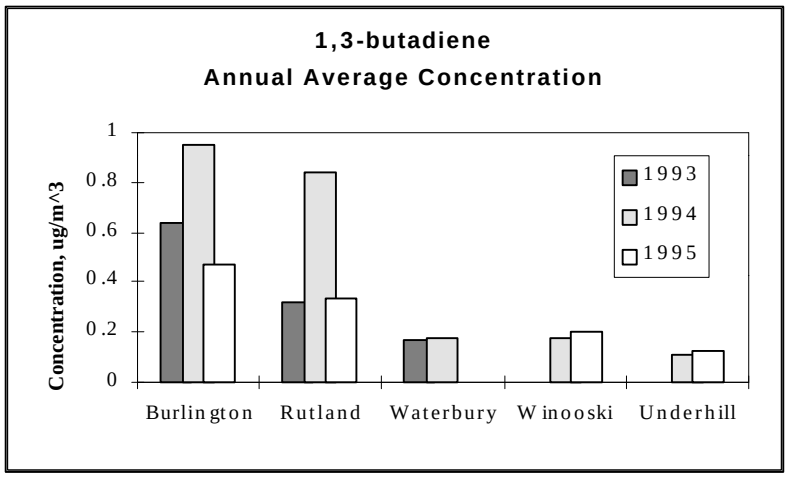


Figure 4.2-Annual average ambient air concentrations of 1,3-butadiene in micrograms per cubic meter (ug/m^3) at Vermont Monitoring Sites from 1993 to 1995. The Hazardous Ambient Air Standard (HAAS) is .035 ug/m^3. See section IV.A.2.

b. Limitations and Uncertainties

There are no significant uncertainties associated with non-detect values and the HAAS in relation to the standard for 1,3-butadiene. The compound has an average percent non-detect of 41% but, as the low mean consistently exceeds the standard, the non-detects are not significant in relation to the standard. The HAAS (0.035 ug/m^3) is less than the detection limits (0.15 ug/m^3 and 0.33 ug/m^3) for 1,3-butadiene but, because the compound is generally above the detection limit, this is not significant. The relative contribution of the various sources of 1,3-butadiene is not known.

c. Implications of Revised Standard

The proposed revised standard for 1,3-butadiene will decrease from .035 ug/m^3 to 0.0019 ug/m^3. At this lower standard, the ambient concentrations will continue to exceed the revised standard unless measures are taken to reduce emissions. As stated above, the primary source of 1,3-butadiene is motor vehicles. 1,3-Butadiene is formed in vehicle exhaust due to incomplete combustion of fuel. Other sources of 1,3-butadiene are waste incinerators and wood fires.

d. Health Concerns

The primary way 1,3-butadiene may enter the body is by inhalation of contaminated air into the lungs. Due to a lack of adequate human epidemiologic data with respect to this compound, much of what we know about 1,3-butadiene has been obtained from experimental studies of very high exposures with laboratory animals. Some of the information is helpful in estimating how this chemical may behave in humans. For example, although no studies are currently available as to how much of the 1,3-butadiene vapors inhaled into the lungs by humans is actually absorbed into the blood stream,

experimental studies with laboratory animals have noted rapid absorption of such vapors from the lungs into the blood (ATSDR, 1992). Once in the blood stream, the absorbed compound can be distributed throughout the body (whether it be the body of a laboratory animal or a human). Animal studies also indicate that absorbed 1,3-butadiene may be broken down into other compounds (metabolites) which leave the body through the urine and in air exhaled from the lungs (ATSDR, 1992).

It is known that brief inhalation of elevated levels of 1,3-butadiene by humans can irritate the eyes, nose and throat. Such exposures can also damage the central nervous system, cause blurred vision, nausea, fatigue, headache, decreased blood pressure and pulse rate and unconsciousness (ATSDR, 1992). It is believed that inhalation of **very** high levels of this compound could produce symptoms such as drunkenness, unconsciousness or in very extreme instances, death (ATSDR, 1992). Fortunately, no human exposure to such high levels has been reported to date.

Increased incidence of heart disease, lung disease, blood disease and cancer have been noted in studies of workers who have inhaled low levels of 1,3-butadiene, in a mix of other volatile chemicals, for long periods of time (ATSDR, 1992). Because exposure was to more than one chemical at a time, it is not known which chemical or combination of chemicals may be responsible for the health effects noted.

Dermal contact with 1,3-butadiene can cause skin irritation and frostbite in humans (ATSDR, 1992). No information is available on potential health effects in humans from ingestion of food and/or drink that may contain low levels of 1,3-butadiene.

Experimental studies with laboratory animals exposed to high levels of 1,3-butadiene vapors for even short periods of time has resulted in damage to blood producing organs, nasal tissues and at extreme levels, death. Increased birth defects were noted in pups of laboratory rodents that had been exposed to elevated levels of 1,3-butadiene vapors during pregnancy. Kidney, liver, lung and reproductive organ damage have been noted in lab rodents experimentally exposed to low levels of 1,3-butadiene vapors for long periods of time. Long term exposure to even small amounts of this compound in the air has resulted in cancers in laboratory rodents (ATSDR, 1992). No information is available on potential health effects in laboratory animals from ingestion of food and/or water containing 1,3-butadiene.

1,3-Butadiene has been classified as Class B2: Probable Human Carcinogen by the United States Environmental Protection Agency and as a Group 2A: Probable Human Carcinogen by the International Agency for Research on Cancer.

3. Carbon Tetrachloride

a. Discussion

Carbon tetrachloride was selected as a priority compound because it exceeded the standard at all sites, every year. The current HAAS for carbon tetrachloride is 0.067 ug/m^3 (annual average).

Annual average concentrations of carbon tetrachloride range from a high of 0.71 ug/m^3 (Burlington, 1994 and Winooski 1994) to a low of 0.48 ug/m^3 (Waterbury, 1993)(see Figure 4.3). The transported ambient air levels are believed to be about 0.1 ppb (0.6 ug/m^3) around the world and 0.2-0.6 ppb (1 to 4 ug/m^3) in cities (ATSDR, 1994). Carbon tetrachloride concentrations were below the detection limit in 15% of the samples collected. The low means were all greater than the standard.

Carbon tetrachloride in the atmosphere in Vermont is probably due to transported pollution. The compound was used extensively in degreasers, cleaning fluids and fire extinguishers until it was withdrawn from the market in the 1960s (ATSDR, 1994). Carbon tetrachloride is still used as a refrigerant and an aerosol propellant but, these uses are being phased out (ATSDR, 1994). Carbon tetrachloride appears to be a regional or transported pollutant as the concentrations do not vary significantly from urban sites to rural sites. The atmospheric half life of carbon tetrachloride is 50-100 years (Kao, 1994), indicating that it will take a long time for current concentrations to decrease.

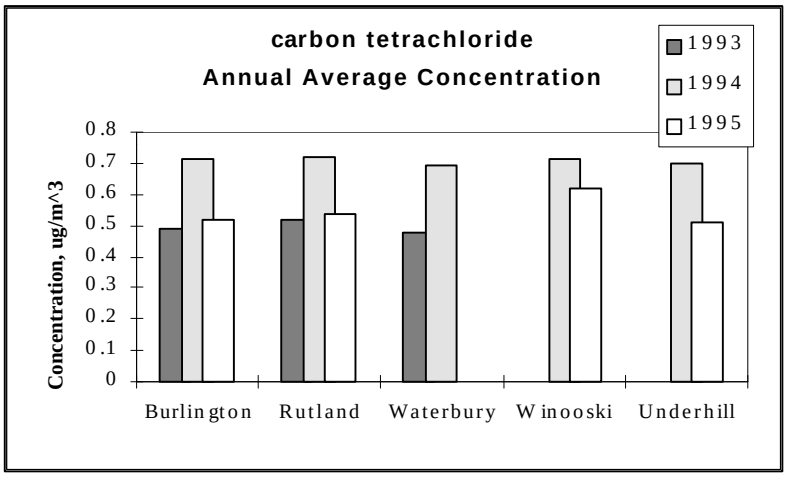


Figure 4.3-Annual average ambient air concentrations of carbon tetrachloride in micrograms per cubic meter (ug/m^3) at Vermont Monitoring Sites from 1993 to 1995. The Hazardous Ambient Air Standard (HAAS) is .067 ug/m^3. See section IV.A.3.

b. Limitations and Uncertainties

There are no uncertainties associated with the detection limit for carbon tetrachloride. The percent non-detect is low (15%) and the low means all exceed the standard.

c. Implications of Revised Standard

Since the proposed revised standard for carbon tetrachloride will remain the same at .07 ug/m^3, emissions will probably continue to exceed the revised standard. As stated above, carbon

tetrachloride in the atmosphere in Vermont is probably due to transported pollution.

d. Health concerns:

Carbon tetrachloride enters the body through the lungs, stomach, intestines and skin. About 60% of the carbon tetrachloride inhaled by humans is believed to be absorbed into the body. It is not known whether this relationship is applicable to low level exposures such as is found in the ambient air.

Most of the inhaled carbon tetrachloride that enters the body is temporarily accumulated in body fat. Some of the carbon tetrachloride can enter the kidney, liver, brain, lungs and skeletal muscle. Once it is transported to the liver by the blood it is transformed through metabolic processes to the toxic form. Because of this biotransformation in the body, toxic responses to carbon tetrachloride can be severely increased by drugs and chemicals. Chemical interactions between carbon tetrachloride and alcohol can be fatal.

Much of the carbon tetrachloride that enters the body through inhalation quickly leaves the body through exhaled air. Animal studies also suggest that it may take weeks for the remainder of the compound in the body to be eliminated, especially that which has entered the body fat. Although most of the carbon tetrachloride is eliminated from the body unchanged, some may change to other chemicals (for example, chloroform, hexachloroethane, and carbon dioxide). Chloroform and hexachloroethane may themselves cause harmful effects (ATSDR, 1994).

Most of the information on health effects of carbon tetrachloride in humans comes from cases where people have been exposed to relatively high levels of carbon tetrachloride, either only once or for a short period of time. Exposure to carbon tetrachloride in the environment may produce effects on the liver, kidney and brain. In severe cases, liver cells may be damaged or destroyed, leading to a decrease in liver function. Kidney failure often was the main cause of death in people who died after very high exposure to carbon tetrachloride. After exposure to high levels of carbon tetrachloride, the nervous system, including the brain, is affected. Such exposure can be fatal. The immediate effects are usually signs of intoxication, including headache, dizziness, and sleepiness perhaps accompanied by nausea and vomiting. In severe cases, stupor or even coma can result, and permanent damage to nerve cells can occur. In animals, the compound has produced cancer in various organs. The effects of carbon tetrachloride are reversible over periods of several days to a week or more. Repeated exposures would be expected to increase the toxicity experienced over a short term. Therefore, children, the elderly, and persons with liver or kidney disease are especially at risk from exposures.

Carbon tetrachloride is also found in drinking water supplies and household products. The effects of these multiple exposures would be additive to that found in the air and may be greater.

Carbon tetrachloride has been classified as Class B2: Probable Human Carcinogen by the United States Environmental Protection Agency and as a Group 2B: Possible Human Carcinogen by the International Agency for Research on Cancer.

4. Chloroform

a. Discussion

Chloroform was selected as a priority compound because the concentration exceeds the standard at every site, every year. The current HAAS for chloroform is 0.043 ug/m^3(annual average). Annual average concentrations range from a high of 0.22 ug/m^3 (Burlington 1995) to a low of 0.13 ug/m^3 (Burlington, 1993)(see Figure 4.4). Chloroform has an average percent non-detect of 90%. This may be significant as the low means do not exceed the standard for 7 out of 12 data sets.

The amount of chloroform normally expected to be present in air ranges from 0.02 to 0.05 ppb (0.1 to 0.2 ug/m^3). Chloroform has been found in the air from all areas of the United States. Sources of chloroform in the atmosphere are pulp and paper mills and water and wastewater plants that use chlorine as a disinfectant (ATSDR, 1995). Chloroform appears to be a transported pollutant. The concentrations do not vary significantly from urban to rural sites and the compound has an atmospheric half life of 2-3 months (Kao, 1994).

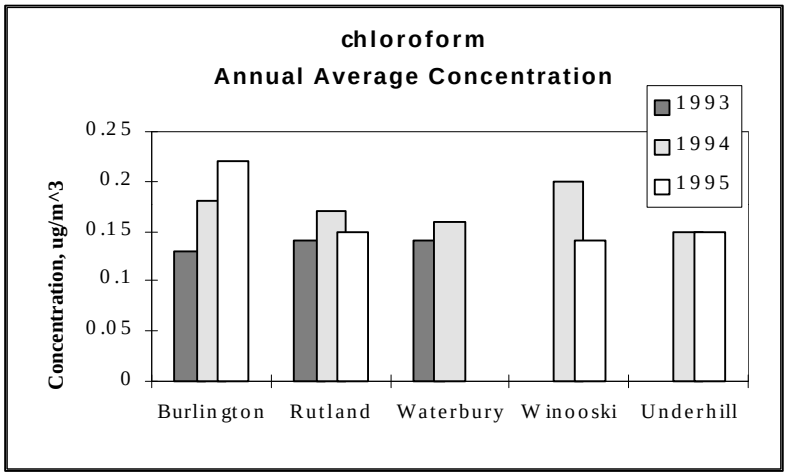


Figure 4.4-Annual average ambient air concentrations of chloroform in micrograms per cubic meter (ug/m^3) at Vermont Monitoring Sites from 1993 to 1995. The Hazardous Ambient Air Standard (HAAS) is .043 ug/m^3. See section IV.A.4.

b. Limitations and Uncertainties

There is uncertainty regarding the actual ambient concentration of chloroform in Vermont due to the high percentage of non-detect values (90%). The HAAS of 0.043 ug/m^3 is significantly less than the current detection limit of 0.29 ug/m^3, making it difficult to be certain how close the ambient concentration is to the standard.

d. Implications of Revised Standard

Since the proposed revised standard for chloroform will remain the same at .043 ug/m^3, emissions will probably continue to exceed the standard. The current detection limit of 0.29 ug/m^3 needs to be lowered in order to determine concentrations near the standard. As stated above, chloroform appears to be a transported pollutant since the concentrations do not vary significantly from urban to rural sites.

c. Health concerns

One way chloroform enters the body is by inhalation of contaminated air into the lungs. Chloroform is absorbed readily into the body when inhaled. Chloroform can quickly enter the blood stream from the lungs or intestines. Once in the blood, it is carried to all parts of the body, such as the liver or kidneys. Chloroform usually collects in body fat and is metabolized in the kidney and excreted through the lungs (unchanged) or through the urine and feces. Some of the chloroform that enters the body is broken down into other chemicals. These chemicals, or breakdown products, can attach to other chemicals inside the cells of the body and may cause harmful effects if they collect in high enough amounts in the body. Some of the breakdown products can also leave the body through exhaled air. Only a small amount of the breakdown products leaves the body in the urine or feces (ATSDR, 1995).

Exposure to chloroform in the environment from the ambient air is a health concern because of a potential to induce cancer from chronic exposure and possible additivity with other sources of chloroform in daily life. In humans, large amounts of inhaled chloroform can affect the central nervous system (brain), liver, and kidneys. At very high concentrations chloroform has been used as an anesthetic agent in man. This use was discontinued as safer agents became available. Breathing about 900 ppm (4000 ug/m^3) for a short time causes fatigue, dizziness, and headache. At lesser concentrations over a long period of time, chloroform may damage the liver and kidneys. At lesser concentrations chloroform has produced reproductive effects in animals such as birth defects and abnormal sperm. It is not known whether these effects would occur in humans.

Most chloroform in the air eventually breaks down, but this process is slow. The breakdown products in air include phosgene, which is more toxic than chloroform, and hydrogen chloride, which is also toxic (ATSDR, 1995).

Studies of people who drank chlorinated water showed a possible link between chloroform formed in the water and cancer of the colon and urinary tract. Animals which received similar exposures for longer periods of time developed liver and kidney tumors.

Because chloroform is absorbed rapidly and eliminated relatively slowly there is concern for chronic and periodic exposures from ambient air. The presence of chloroform in other media such as food and water raises concern for multi pathway exposures. The total dose from all sources of chloroform would need to be taken into account when considering the health hazard. Because the compound is metabolized before removal from the body some persons could be more sensitive to chloroform than the general public. Those of particular concern would include the young, the elderly, those who are

pregnant and those with liver disease or taking drugs that could affect the liver.

Chloroform has been classified as Class B2: Probable Human Carcinogen by the United States Environmental Protection Agency and as a Group 2B: Possible Human Carcinogen by the International Agency for Research on Cancer.

5. Formaldehyde

a. Discussion

Formaldehyde was selected as a priority compound because the annual average concentrations exceed the standard at every site, every year. The current HAAS for formaldehyde is 0.08 ug/m^3(annual average). Annual average concentrations observed in Burlington were 3.24 ug/m^3 (1994) and 5.23 ug/m^3 (1995). Rutland had annual average concentrations of 2.69 ug/m^3 (1994) and 5.92 ug/m^3 (1995). Annual average concentrations observed in Winooski were 1.77 ug/m^3 (1994) and 10.16 ug/m^3 (1995). Underhill showed the lowest concentrations: 0.89 ug/m^3 (1994) and 1.19 ug/m^3 (1995) (see Figure 4.5). Formaldehyde was detectable in 100% of the samples collected.

Formaldehyde has numerous atmospheric sources. The compound is a byproduct of combustion, which covers a wide range of sources from internal combustion engines to wood stoves. Formaldehyde is also generated by wood processing plants and glues (EHC, 1997). Formaldehyde is produced by the atmospheric reactions of other pollutants, including 1,3-butadiene (EETE, 1995). Formaldehyde in Vermont follows the locally generated pattern. Concentrations are highest in urban sites and decrease at rural locations (except for Winooski 1995). The atmospheric half life of formaldehyde is short, 4-10 hours (Kao, 1994).

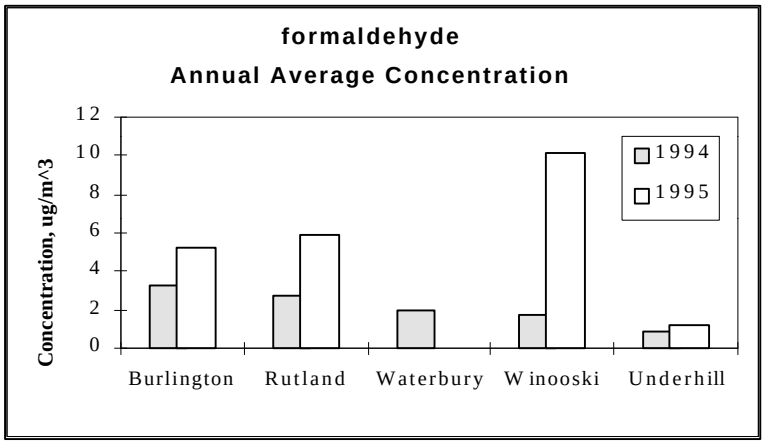


Figure 4.5-Annual average ambient air concentrations of formaldehyde in micrograms per cubic meter (ug/m^3) at Vermont Monitoring Sites from 1994 and 1995. The Hazardous Ambient Air Standard (HAAS) is .08 ug/m^3. See section IV.A.5.

b. Limitations and Uncertainties

Formaldehyde levels were always above the detection limit, eliminating any uncertainty regarding non-detects. The proportionate contribution of the various sources of formaldehyde is not known.

c. Implications of Revised Standard

Since the proposed revised standard for formaldehyde will remain unchanged at 0.078 ug/m^3, the ambient data will most likely continue to exceed the standard unless measures are taken to reduce emissions. As stated above, formaldehyde has numerous atmospheric sources including combustion sources, wood processing plants and glues. In addition, formaldehyde is produced by the atmospheric reactions of other pollutants, including 1,3-butadiene.

d. Health Concerns

The primary way formaldehyde may enter the body is by inhalation of contaminated air. Experimental studies indicate that most of the formaldehyde inhaled into the lungs is rapidly broken down into other compounds (metabolized) at the site of contact and quickly absorbed into the body through the lining of the nose or from the upper part of the lungs (ATSDR, 1997). Due to this rapid local metabolism, inhalation exposure to even moderately high ambient concentrations of this compound has not been found to effect the amount of formaldehyde present in the blood. In fact, rapid local metabolism is why "... little if any intact formaldehyde can be found in the blood..." at any time (ATSDR, 1997). Rapid local metabolism also results in toxicity primarily at the site of contact.

Formaldehyde is a normal metabolic product of animal cell metabolism. Thus, almost all the tissues in the body are able to metabolize this compound. Formaldehyde is primarily metabolized to formate which can be incorporated into other essential molecules or pass from the body in the urine or be further metabolized to carbon dioxide which leaves the body in exhaled air. If metabolism to formate is inhibited or the metabolic mechanism overloaded, internal levels of formaldehyde may increase to the point where it can form bonds between proteins or between proteins and deoxyribonucleic acid (DNA). Formaldehyde and formate are both part of routine animal cell metabolic processes and neither is stored to any extent in the body (ATSDR, 1997).

Inhalation of ambient levels of formaldehyde vapors between .4 and 3 ppm (490 ug/m^3 to 3700 ug/m^3), even for short periods of time, can irritate the eyes, nose, and throat and cause increased tearing and itching. Upper respiratory tract symptoms are believed to predominate because rapid local metabolism may prevent much formaldehyde from reaching the lower respiratory tract (ATSDR, 1997). Some studies indicate that long-term exposure to similar levels might adversely impact respiratory function while other studies do not support this contention. Short-term exposure to very high ambient levels may result in coughing wheezing, chest pains and bronchitis.

Some people are known to be more sensitive to formaldehyde than others and repeated exposure, including via inhalation of vapors, is believed to cause an increase in sensitivity in some individuals. Although one large study suggests that those with asthma may be particularly sensitive to formaldehyde vapors, many other studies have not made this finding.

Experimental studies with laboratory rats have found that long-term inhalation of highly elevated ambient levels of formaldehyde can cause nasal cancer (squamous cell carcinoma) in the rats. There is limited evidence that long-term inhalation of low levels of this compound might be associated with an increase in cancer in humans (ATSDR, 1997).

Formaldehyde has been shown to be a contact irritant, regardless of the route of exposure. Limited studies indicate that formaldehyde is rapidly absorbed from the gastrointestinal tract and meets the same metabolic fate as inhaled formaldehyde vapors (ATSDR, 1997). Ingestion of elevated amounts of this compound can irritate the mouth, esophagus and gastric mucosa (ATSDR, 19997). Lesions of the gastric mucosa have been noted in laboratory animals experimentally exposed to formaldehyde in drinking water for various periods of time. Human consumption of very large amounts of formaldehyde (i.e., suicide attempts) can result in severe abdominal pain, acidosis, central nervous system depression, coma and death (ATSDR, 1997).

Two studies indicate that an increase in the amount of formaldehyde in the diet of some milk producing animals such as cows, sheep and goats, can increase the amount of formaldehyde present in the milk produced. The reason for this is not clear. However, it is postulated that perhaps fundamental differences exist between the way humans and ruminants metabolize formaldehyde.

Although very small amounts of formaldehyde can be absorbed into the body through intact skin, dermal contact with this compound can result in sensitization. Dermal contact with liquid formaldehyde can irritate the skin. Allergic reactions of the skin and in extreme cases, anaphylaxis are reported in the literature (ATSDR, 1997).

Formaldehyde has been classified as Class B1: Probable Human Carcinogen by the United States Environmental Protection Agency and as a Group 2A: Probable Human Carcinogen by the International Agency for Research on Cancer.

6. Methyl Chloride

a. Discussion

Methyl chloride was selected as a priority compound because the annual average concentration exceeds the standard at every site, every year. The current HAAS for methyl chloride is 0.01 ug/m^3 (annual average). Annual average concentrations range from a high of 1.39 ug/m^3 (Burlington, 1994) to a low of 1.04 ug/m^3 (Rutland, 1995)(see Figure 4.6). Methyl chloride has an average percent non-detect of 7%. Non-detects are not significant in relation to the standard as the low means all exceed the standard at every site, every year.

Outside air contains less than 0.001 ppm (2 ug/m^3) and city air contains up to 0.001 ppm methyl chloride. These levels are much lower than the levels shown to have toxic effects . Methyl chloride is naturally released into the atmosphere from oceans and biomass, producing low ambient concentrations. Other sources of methyl chloride are wood burning and chlorinated swimming pools. The methyl chloride in the outdoor environment, however, is almost totally from natural sources

(ATSDR, 1990a). Methyl chloride appears to be a transported pollutant. The variation in concentrations between urban and rural sites is not significant and the atmospheric half life is fairly long: 1-2 years (Kao, 1994).

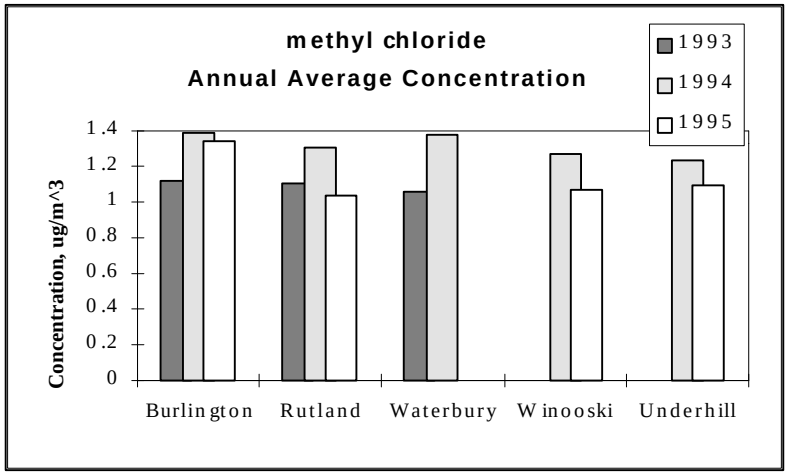


Figure 4.6-Annual average ambient air concentrations of methyl chloride in micrograms per cubic meter (ug/m^3) at Vermont Monitoring Sites from 1993 to 1995. The Hazardous Ambient Air Standard (HAAS) is 0.01 ug/m^3. See section IV.A.6.

b Limitations and Uncertainties

The methyl chloride data is not significantly affected by non-detects (7% non-detect). Although the HAAS is less than the detection limit, ambient concentrations are generally well above both the standard and the detection limit.

d. Implications of Revised Standard

Even though the proposed revised HAAS for methyl chloride will increase from 0.01 ug/m^3 to 0.56 ug/m^3, the ambient data still exceeds the proposed revised standard at all sites, every year. As stated above, methyl chloride appears to be a transported pollutant.

e. Health Concerns

Methyl chloride enters the body by inhalation of contaminated air into the lungs and by swallowing contaminated water into the digestive tract. Methyl chloride is rapidly absorbed from the air and from drinking water. It is taken up by the blood and breakdown products are distributed to the liver, brain and kidneys. A small part of the methyl chloride leaves through the lungs with the remainder changed to other breakdown products and removed through the urine. This removal may take from a few hours to several days.

Exposure to methyl chloride in the environment may produce effects on the nervous system and reproductive systems. Exposure to methyl chloride can also harm the liver and kidney, or have an effect on the heart rate and blood pressure (ATSDR, 1990a). Exposures to very high levels of methyl chloride in homes occurred in the past when the chemical was used as a refrigerant. These exposures which were frequently fatal show how the chemical is taken up into the body and metabolized but are not predictive of the effects of the lower level exposures found in the ambient air. Animal studies at lower levels (one hundred thousand to one million times higher than background levels) over a long period of time (weeks to months) have demonstrated effects on growth, reproduction and fetal development. Male mice that breathed air containing methyl chloride (one million ppb, two million ug/m^3) for 2 years developed tumors in the kidneys, but female mice and male and female rats did not develop tumors (ATSDR, 1990a).

Because the compound is metabolized prior to removal from the body and because the metabolites may be toxic to the brain or kidney it is necessary to consider sensitive populations. Such concern would include the elderly, the young, especially with respect to development, and the chronically ill with liver or kidney disease.

Because there are other sources of exposure such as water it is necessary to consider potential for cumulative exposures.

Methyl chloride has been classified as Class C: Possible Human Carcinogen based on limited evidence of carcinogenicity in animals by the United States Environmental Protection Agency and as a Group 3: Not classifiable due to limited human or animal data by the International Agency for Research on Cancer.

7. Methylene Chloride

a. Discussion

Methylene chloride was selected as a priority compound because the annual average concentration exceeds the standard at some sites, some years. The current HAAS for methylene chloride is 2.0 ug/m^3 (annual average). Annual average concentrations observed in Burlington were 1.80 ug/m^3 in 1993, 7.79 ug/m^3 in 1994 and 5.20 ug/m^3 in 1995. Rutland showed levels in the same range, 1.82 ug/m^3 in 1993, 8.41 ug/m^3 in 1994 and 3.66 ug/m^3 in 1995. Annual average concentrations observed in Winooski were 6.75 ug/m^3 in 1994 and 2.08 ug/m^3 in 1995. Waterbury had annual average concentrations of 3.19 ug/m^3 in 1993 and 3.66 ug/m^3 in 1994. Annual average concentrations observed in Underhill were 4.74 ug/m^3 in 1994 and 1.95 ug/m^3 in 1995 (see Figure 4.7). Methylene chloride in US urban air was typically 6.7 ug/m^3 (ATSDR, 1993). Methylene chloride concentrations were below the detection limit in 18% of the samples collected.

Methylene chloride comes from both transported and locally generated sources. Local sources are regulated industrial sources and area sources. Area sources include businesses where methylene chloride is used as a general solvent such as garages and the compound is also found in spray cans and furniture strippers (ATSDR, 1993a). Methylene chloride has a fairly long atmospheric half life, 3-4 months (Kao, 1994), indicating the fairly long persistence typical of transported pollutants.

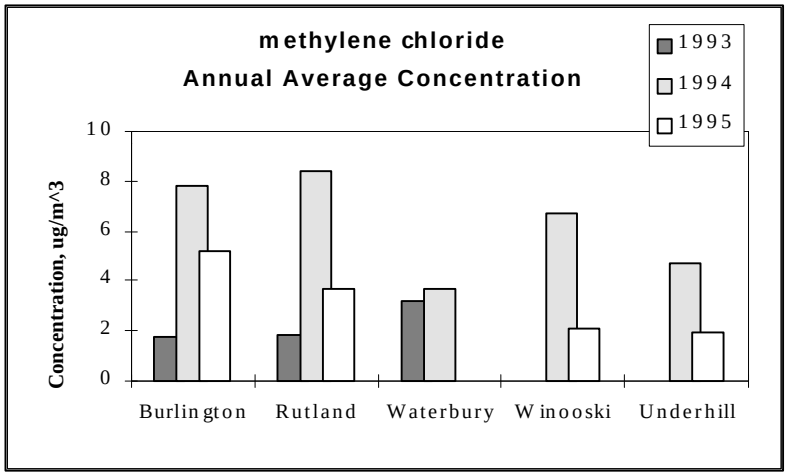


Figure 4.7-Annual average ambient air concentrations of methylene chloride in micrograms per cubic meter (ug/m^3) at Vermont Monitoring Sites from 1993 to 1995. The Hazardous Ambient Air Standard (HAAS) is 2.0 ug/m^3. See section IV.A.7.

b. Limitations and Uncertainties

Methylene chloride was detected in over 80% of the samples collected, limiting uncertainty regarding non-detects. The compound has numerous sources and the proportionate contribution of each to the ambient concentration is not known.

c. Implications of Revised Standard

Since the proposed revised HAAS for methylene chloride will remain the same at 2.0 ug/m^3, ambient air concentrations will continue to exceed the standard unless measures are implemented to reduce the emissions. As stated above, methylene chloride comes from both transported and locally generated sources. Local sources include industrial sources and area sources such as garages where methylene chloride is used as a general solvent. The compound is also found in spray cans and furniture strippers.

d. Health Concerns

The primary way methylene chloride may enter the body is by inhalation of contaminated air into the lungs. Inhaled methylene chloride vapors are rapidly absorbed into the blood stream from the lungs. The amount absorbed and retained by the body is influenced by the amount of chemical in the air, how long a person is exposed, the amount of body fat a person has and the degree of physical activity while being exposed (EM, 1995).

Studies indicate that approximately seventy percent of methylene chloride vapors inhaled into human lungs are rapidly absorbed into the bloodstream and distributed throughout the body primarily to the

liver, brain and fat tissue. Lesser amounts are distributed to the kidneys and lungs (ATSDR, 1993; EM, 1995). At a certain point, the blood can become saturated with methylene chloride. Thus, even if the concentration of chemical in the air increases, the concentration of chemical in the blood has leveled off (plateaued) (ATSDR, 1993; EM, 1995).

Once in the body, methylene chloride may be broken down into other compounds (metabolites). This occurs in primarily the liver and to a lesser extent in the kidney and lungs (EM, 1995). Two different sets of break down products may be formed in the liver: at low levels of exposure mostly carbon dioxide and carbon monoxide are formed; at higher levels of exposure formaldehyde and formic acid are also formed (EM, 1995).

Methylene chloride is very fat soluble and may temporarily be stored in the liver, brain and fat tissue. Accumulated (stored) methylene chloride may be re-released to the blood stream over a period of several hours. This can cause internal exposure to methylene chloride and its metabolites to continue for a few hours even after a person has stopped breathing contaminated air.

About half of the methylene chloride vapors absorbed from the lungs into the blood leave the body within an hour after exposure has stopped. The majority of both unchanged methylene chloride and its metabolites leave the body in air exhaled from the lungs. At low levels of exposure, more of the methylene chloride vapors absorbed are metabolized and are exhaled as carbon monoxide (EM, 1995). At greater levels of exposure, more of the unchanged methylene chloride itself is exhaled. A small percentage of unchanged methylene chloride and its metabolites pass from the body in the urine and feces (EM, 1995). Total elimination from the body usually occurs within 48 hours after exposure has stopped (ATSDR, 1993).

Short term inhalation of large amounts of methylene chloride is reported to depress the central nervous system. The level of methylene chloride vapors in the air as well as the length of time one is exposed influence the severity of the effects experienced. Brief inhalation of greater than 8,000 ppm ($2x10^8$ ug/m^3) has been shown to depress the central nervous system and may result in narcosis, unconsciousness. At extreme levels of methylene chloride between 8,000 and 20,000 ppm ($8x10^8$ ug/m^3), death may result due to depression of the respiratory system (EM, 1995; ATSDR, 1993). Inhalation of between 300 and 800 ppm ($7x10^6$ to $20x10^6$ ug/m^3) can interfere with psychomotor function and cause dizziness, nausea, tingling and numbness of the fingers and toes, and a feeling of drunkenness (EM 1995, ATSDR, 1993). Vision and hearing may be impaired at the lower end of this range (EM, 1995; ATSDR, 1993).

In many cases, recovery from the central nervous system effects associated with short term exposure to moderate levels of methylene chloride vapors may occur once a person begins to breath fresh air again. These effects are believed to be due to the action of either methylene chloride or methylene chloride and carbon monoxide in combination, not carbon monoxide alone (EM, 1995).

Exposure to elevated levels of methylene chloride may be particularly worrisome for pregnant women because it is believed the carbon monoxide produced when methylene chloride is metabolized could potentially effect a developing fetus. Increased exposure to carbon monoxide may also be harmful

to those people with existing coronary artery disease. Their risk would be increased even further by exercising while inhaling methylene chloride vapors. People with already elevated levels of carbon monoxide in their blood, such as smokers and those who work with internal combustion engines, may also be at increased risk of developing adverse health effects due to methylene chloride exposure.

Exposure to methylene chloride in the air may also irritate the mucous membranes of the eyes, nose and throat.

Potential health effects that may be associated with long term inhalation of elevated levels of methylene chloride vapors have not been fully identified. However, limited studies of workers exposed to elevated levels of methylene chloride vapors, in a mix of other volatile organic compounds, have noted increased incidence of liver, biliary tract and pancreatic cancers. However, it is unknown what role methylene chloride itself may play in these effects. Long term inhalation of low levels of this compound are believed to be associated with some mild liver effects in humans (EM, 1995).

Low levels of methylene chloride are found in chlorinated drinking water, spice extracts and decaffeinated coffee. Health effects that may be associated with long term ingestion of such low levels have not been identified (EM, 1995).

Prolonged dermal contact with methylene chloride may cause skin irritation and in extreme instances, chemical burns.

Experimental studies with laboratory animals inhaling very high levels of methylene chloride for very long periods of time have noted increased incidence of noncancerous mammary tumors and cancerous lung and liver tumors. However, it is known that at least one of the species tested processes methylene chloride differently than humans.

Methylene chloride has been classified as Class B2: Probable Human Carcinogen by the United States Environmental Protection Agency and as a Group 2B: Possible Human Carcinogen by the International Agency for Research on Cancer.

8. Tetrachloroethylene

a. Discussion

Tetrachloroethylene was selected as a priority compound because it has exceeded the standard, although rarely. The current HAAS for tetrachloroethylene is 0.41 ug/m^3 (annual average). The highest annual average concentration was 0.62 ug/m^3, observed in Burlington in 1994. The other sites/years had annual averages that ranged from 0.10 ug/m^3 to 0.24 ug/m^3. The highest levels were observed at the urban sites, Burlington and Rutland (0.17-0.62 ug/m^3). Winooski and Waterbury showed lower concentrations (0.10-0.20 ug/m^3) and Underhill had the lowest concentrations (0.12-0.13 ug/m^3)(see Figure 4.8). Tetrachloroethylene concentrations were below the detection limit in 80% of the samples.

Sources of tetrachloroethylene are local area and point sources. Tetrachloroethylene is used in many industries as a general solvent. The compound is used extensively in dry cleaning and is also produced by waste incinerators (ATSDR, 1993b). Tetrachloroethylene concentrations are highest at urban sites and decrease at rural sites, indicating that the compound is locally generated. The atmospheric half life is 70-100 days (Kao, 1994).

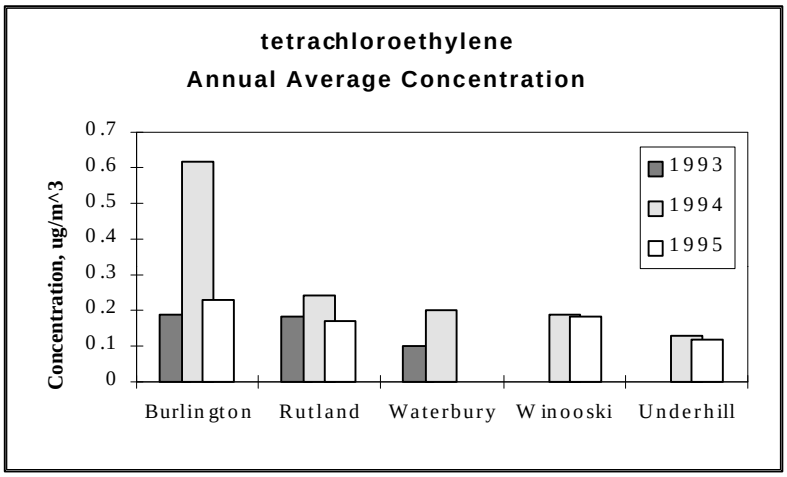


Figure 4.8-Annual average ambient air concentrations of tetrachloroethylene in micrograms per cubic meter (ug/m^3) at Vermont Monitoring Sites from 1993 to 1995. The Hazardous Ambient Air Standard (HAAS) is .41 ug/m^3. See section IV.A.8.

b. Limitations and Uncertainties

The percentage of non-detect values for tetrachloroethylene is fairly high, 80%, creating some uncertainty as to the true ambient concentration. Examination of the low means show only the Burlington 1994 data set exceeding the standard.

c. Implications of Revised Standard

Since the revised standard for tetrachloroethylene will increase from .41 ug/m^3 to 1.8 ug/m^3, the air concentration will no longer exceed the proposed revised standard, and therefore the compound should be removed from the priority list.

d. Health Concerns

One way tetrachloroethylene (perc) may enter the body is by inhalation of contaminated air into the lungs. Although this compound is rapidly absorbed by the lungs, the **amount** that is absorbed from the lungs into the bloodstream is influenced by how quickly a person is breathing (inhalation rate), the amount of chemical in the air, how long the person is exposed, the person's body mass and degree of physical activity while being exposed (NYS 1996; ATSDR, 1996b).

Studies with human volunteers indicate that absorption of tetrachloroethylene vapors from the air across the lungs may be greatest during the first few minutes of exposure. Perc absorbed into the blood stream is transported throughout the body and may be temporarily stored (accumulated) in fat tissue and slowly re-released to the blood stream over a period of several days. Absorbed perc can cross the blood-brain and placental-fetus barriers and has been found in fat rich tissues such as the brain, liver and breast milk (NYS, 1996).

The majority of perc absorbed from the lungs into the blood stream leaves the body unchanged in air exhaled from the lungs (EM, 1995). A small percentage of perc that is absorbed into the blood stream is broken down into other compounds (metabolites) in the liver (EM, 1995; ATSDR, 1996b). One metabolite in particular, trichloroacetic acid, is thought to be associated with some of the adverse health effects noted with prolonged inhalation of elevated levels of perc (ATSDR, 1996b). These metabolites leave the body in the urine. Experimental studies indicate that, once exposure has stopped, total elimination from the body may take a few days to a few weeks, depending on the amount of perc that has accumulated in body tissues.

Brief inhalation of large amounts of tetrachloroethylene can adversely effect the central nervous system and result in dizziness, headache, sleepiness, confusion, nausea, difficulty in speaking and walking, unconsciousness and, in extreme instances, death (ATSDR, 1996b). The mucous lining of the eyes, nose and throat may also become irritated (EM, 1995).

Recovery from the adverse central nervous system effects associated with inhalation exposure to perc vapors, in some cases even moderate term exposures, is possible once exposure has stopped (EM, 1995). However, the potential effects of long-term inhalation of relatively low levels of perc vapors are not currently known (ATSDR, 1996b).

Some liver and kidney effects may be associated with inhalation of elevated levels of perc. One instance of a reversible adverse cardiac effect associated with inhalation of tetrachloroethylene vapors is noted in the literature.

Limited studies of women exposed to elevated levels of perc in the work place, in a mix of other volatile chemicals, indicate that such exposure may effect the reproductive system and perhaps be related to an increased risk of spontaneous abortion (miscarriage) (ATSDR, 1996b). Because exposure was to more than one chemical at a time, it is not known which chemical or combination of chemicals may be responsible for the health effects noted.

Repeated or prolonged dermal contact with perc can cause skin irritation (ATSDR, 1996b). Potential health effects that may be associated with long term consumption of food and/or drink containing low levels of tetrachloroethylene are not known (ATSDR, 1996b).

Long term experimental studies with laboratory animals exposed to very high levels of perc vapors have noted skin, liver, kidney, cardiac effects and in some cases liver and kidney cancer. Not all such effects have been noted in people who have been exposed to tetrachloroethylene at much lower levels.

Tetrachloroethylene has been classified as Group 2A: Probably Carcinogenic to Humans by the International Agency for Research on Cancer and is generally considered to be an animal carcinogen and probable human carcinogen by the United States Environmental Agency.

B. Category II

1. Mercury

a. Discussion

Mercury was selected as a priority compound due to concerns surrounding this contaminant on the state and national level. The compound bioaccumulates in the environment and there are multiple pathways for exposure. The current HAAS for mercury is 0.12 ug/m^3 (annual average). Mercury has been monitored at the Underhill site since December 1992 by the University of Vermont. Annual average mercury vapor concentrations range from a high of 0.00183 ug/m^3 in 1993 to a low of 0.00157 ug/m^3 in 1994.

Sources of mercury include both transported and local emissions. Waste incineration is a local source of atmospheric mercury.

b. Implications of Revised Standard

Even though the revised standard for mercury will increase from .12 ug/m^3 to 0.3 ug/m^3 and current ambient air concentrations are below the proposed revised standard, concerns of mercury being deposited to the environment from the atmosphere and accumulating in lake sediments may need to be reflected in the ambient standard.

c. Health Concerns

Prolonged exposures to relatively high concentrations of mercury in the air produces damage to the nervous system and kidney, but rarely produce a fatal injury. However, accidental oral exposures have caused death (WHO, 1990; ATSDR ,1993c). The presence of mercury in the environment at lower levels also produces potential for human exposure directly through breathing mercury and indirectly through ingestion of fish contaminated with methyl mercury. Methyl mercury is about five times more dangerous than the inorganic and metallic forms of mercury (Casarett and Doull, 1990). Toxic actions of methyl mercury and mercury are different and should be considered separately.

I Elemental mercury. Inhalation of moderate levels of mercury for prolonged periods of time produces unique effects on the central nervous system and the kidney. The chemical form of mercury is important in the determination of the actual toxic effect (Casarett and Doull, 1990; ATSDR, 1993c). For example, elemental mercury is highly charged and does not readily pass through the blood brain barrier but directly reaches the kidney. Therefore, elemental mercury is more toxic to the kidney than the brain. Metallic mercury and organic forms of mercury pass easily through the protective blood brain barrier and may cause brain damage as well as kidney damage (ATSDR, 1993c). Once mercury enters the body it is retained in the kidney, brain, liver and the fetus. It may stay in the body for several months. When mercury is eliminated from the body it appears in the

breath, urine, feces, milk and hair. Different organs accumulate and retain mercury at different rates, with the brain and the kidney retaining mercury for periods of months to years. Enzymatic processes in the body transform mercury to the inorganic or metallic form.

Toxic effects of mercury are found in the brain, kidney, skin and liver. At low exposures the toxic effects of mercury are found mainly in the brain. The effects are much more serious in the infant and the fetus because of interference with development of the motor and cognitive functions (ATSDR, 1993c; Casarett and Doull, 1990). There is a latent period between the exposures to mercury and the onset of effects of the poisoning. This delay complicates the diagnosis of the damage and the implementation of the treatment (Clarkson,1992).

At first, low concentrations of mercury seem to have no health effects but signs of toxicity become noticeable with continued exposures (ATSDR 1993c). Toxic signs include loss of feeling or burning sensations in the legs, paralysis, congenital malformations, kidney toxicity and, on rare occasions, death.

Environmental exposures do not produce the more severe effects but subtle toxic effects are possible, especially in the fetus. These changes would not be apparent in the newborn but would become apparent after later development of motor functions such as walking (Casarett and Doull 1990). Unsteadiness and tremors may develop after long periods of exposure. Psychological effects such as insomnia, loss of appetite, shyness, emotional instability and memory loss are also reported in the literature. These actions are partially reversible with termination of exposures to mercury. Cancer is not a significant component of mercury toxicity (ATSDR, 1993c).

While the toxicity of mercury itself is an important concern, the toxic effects of methyl mercury are dangerous because of the higher potency of methyl mercury and its effects on development of the fetal brain.

The occupational safety and health Administration has set a limit of 50 ug/m^3 for metallic mercury in the work place. EPA has set limits for inorganic mercury in drinking water and surface water.

II. Methyl mercury. Human health effects of methyl mercury nearly all involve the ingestion of methyl mercury in fish, or, in one case in grain (Spyker Cranmer J., 1996). Methyl mercury in food is nearly all absorbed into the body and distributed throughout the body to the brain and fetus. Mercury absorbed into the blood stream may be metabolized to the inorganic forms in the liver and removed from the body or reabsorbed from the intestine.

Methyl mercury in the brain and the fetus can be transformed and concentrated. Thus, when the blood levels are decreasing overall the mercury in the brain and fetus may remain high or even increase. Mercury is transferred to hair where its measurement provides a historical indication of past exposures. Equally if not more important, methyl mercury is also excreted into the mother's milk where it is transferred to the nursing infant. Damage has been found in infants and in the fetus even when the mother has shown no toxic effects of mercury exposure.

There is a large body of information on the toxic effects of the chronic ingestion of fish containing methyl mercury by different populations starting before Minimata in Japan in the 1950s to present day exposures in the Amazon river basin. Relatively widespread human poisonings have been documented from these episodes. The actual levels of exposures at which injury begins to occur in the fetus, infants and children remains controversial. The findings from chronic exposures have been used to identify reference doses for estimating the human health hazards (Sea Food Safety, 1991). The reference doses from these different studies and exposures have converged on values in the 1 to 5 ug/m^3 range. Because of uncertainties in the conditions of the different exposures and in the potential for exposure from eating fish, there is a tendency to rely more on the lower end of the risk of 1 ug/kg/day in fish consumption advisories.

Over 39 states have issued fish consumption advisories to reduce the potential hazards of sport fish high in methyl mercury. It is not certain what the actual hazard is from periodic ingestion of fish which have high levels of methyl mercury, therefore public health agencies have adopted a risk reduction strategy to limit potential exposures to mercury in the more sensitive members of the population.

Deposition of mercury into lakes, streams and watersheds is the first step in contamination of fish (EPA, 1996). However the ultimate level of methyl mercury found in the fish is determined by several factors related to the age and type of fish and the nature of other biota in the water. Bioaccumulation appears to increase as the ecological food webs of the water body become more complex. Thus some ponds and lakes and rivers that appear to be pristine often have surprisingly high levels of mercury in the fish. Other lake which have become acidified also can have fish with high levels of mercury.

2. Styrene

a. Discussion

Styrene was selected as a priority compound due to local public concern regarding the adequacy of the current standard. The current HAAS for styrene is 512 ug/m^3 (annual average). Ambient concentrations for individual samples range from a low of 0 (non-detect) to a high of 5.41 (Underhill, 1994). For annual average concentrations for all sites see Figure 4.9. Styrene concentrations were below the detection limit (currently 0.34 ug/m^3) in 69% of the samples. Concentrations of styrene in the air range from 0.3 to 64 ug/m^3 in US cities. (Howard, 1989). Styrene is proposed to be classified as a Category I contaminant.

Styrene sources include local industrial point sources and area sources such as vehicle exhaust and auto body shops. Styrene has a fairly short atmospheric half life, 6-7 hours (Kao, 1994), indicating that it is probably a locally generated, rather than a transported pollutant.

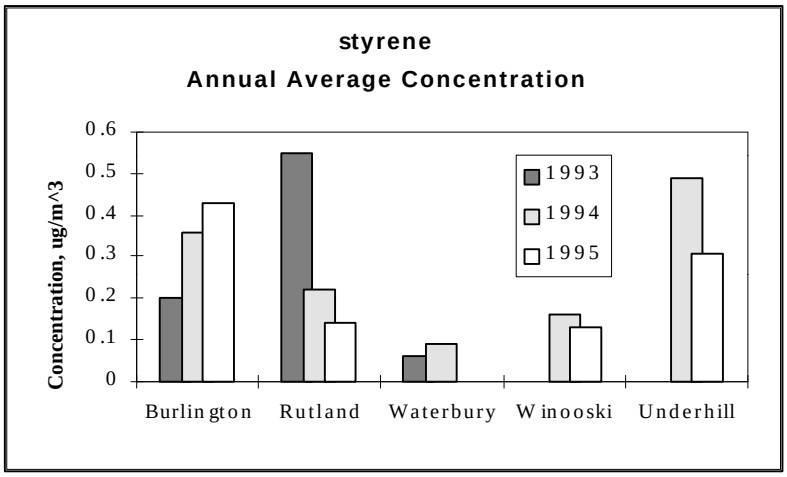


Figure 4.9-Annual average ambient air concentrations of styrene in micrograms per cubic meter (ug/m^3) at Vermont Monitoring Sites from 1993 to 1995. The Hazardous Ambient Air Standard (HAAS) is 512 ug/m^3. See section IV.B.2.

b. Limitations and Uncertainties

Styrene levels were below the detection limit in 69% of the samples collected, generating some uncertainty as to the true ambient concentration. The maximum concentration measured was 5.41 ug/m^3 (Underhill, 1994) which is far below the standard of 512 ug/m^3. So although the true concentration is uncertain, the ambient levels are well below the standard, making the uncertainty due to the non-detects not significant in relation to the standard.

c. Implications of Revised Standard

Styrene is recommended to be classified as a Category I contaminant. The proposed revised HAAS for styrene will decrease to 100 ug/m^3. Although ambient air concentrations do not exceed the proposed revised standard, some monitoring locations may be moved to better characterize ambient concentrations. As stated above, styrene sources include local industrial point sources and area sources such as vehicle exhaust and auto body shops.

d. Health Concerns

The primary way styrene may enter the body is by inhalation of contaminated air into the lungs. Studies indicate that approximately two-thirds of the styrene inhaled into human lungs is actually retained (ATSDR, 1991). The majority of retained styrene is rapidly absorbed from the lungs into the bloodstream where it can then be transported throughout the body.

A small portion of retained styrene leaves the body unchanged through exhaled air. The majority of absorbed styrene is rapidly broken down into other compounds (metabolites) and leaves the body

through the urine. Experimental studies indicate that, once exposure has stopped, total elimination of styrene from the body may take a few days up to a few weeks.

Short term inhalation of large amounts of styrene is reported to adversely impact the central nervous system. Depression, concentration problems, muscle weakness, tiredness, and nausea have been reported in people, especially workers, who have inhaled large amounts of styrene for short periods of time (ATSDR, 1991). Exposure to styrene in the air may also irritate the mucous membranes of the eyes, nose and throat.

Some studies of female workers indicate that occupational exposure to elevated air levels of styrene, in a mixture with other potentially hazardous chemicals, may be associated with lower birth weight babies and an increased risk of spontaneous abortions (miscarriages) (ATSDR, 1991). However, it is unknown what role styrene itself may play in these effects.

Rapid recovery from the adverse effects associated with short-term inhalation exposure to styrene vapors has been noted. However, the potential effects of long-term human exposure to low levels of styrene vapors are not currently known.

Experimental studies indicate that animals are also impacted by inhalation of styrene vapors. Changes in the lining of the nose of experimental animals have been noted up to several weeks after exposure has stopped. Although long-term inhalation of high levels of styrene has been associated with adverse liver effects in animals, this effect has not been noted in humans (ATSDR, 1991).

Scant information is available regarding adverse health effects associated with human ingestion of or dermal contact with styrene. Liver, kidney, blood, immune system and nervous system effects have been noted in experimental styrene ingestion studies with laboratory animals. Irritation of the skin and eyes has been noted in experimental dermal studies with rabbits.

Styrene has been classified as possibly carcinogenic to humans by the International Agency for Research on Cancer.

3. 1,2,4-Trimethyl Benzene

a. Discussion

1,2,4-Trimethyl benzene was selected as a priority compound because it always exceeds the standard. The current HAAS for 1,2,4-trimethyl benzene is 0.15 ug/m^3 (annual average). Burlington showed the highest annual average concentrations: 2.15 ug/m^3 in 1993, 2.80 ug/m^3 in 1994 and 2.11 ug/m^3 in 1995. Rutland had annual average values of 1.20 ug/m^3 in 1993, 2.72 ug/m^3 in 1994 and 1.56 ug/m^3 in 1995. Waterbury and Winooski had annual average concentrations from 0.33 ug/m^3 to 0.44 ug/m^3. Underhill had the lowest annual average values: 0.30 ug/m^3 in 1994 and 0.25 ug/m^3 in 1995 (see Figure 4.10). Concentrations of 1,2,4-trimethyl benzene in the US range from 3 ug/m^3 in rural areas to 6 ug/m^3 in urban areas (EPA, 1988). 1,2,4-Trimethyl benzene concentrations were below the detection limit in 36% of the samples. The low average did not exceed the standard in 2 out of 12 data sets.

1,2,4-Trimethyl benzene appears to be generated by local sources. These sources include area sources (paints, gasoline and surface coatings) and point sources, such as printing presses. The variation of the concentration levels follows the locally generated pollutant pattern: highest at urban sites and then decreasing as sites become more rural. The atmospheric half life is fairly short, 6-12 hours (Chemfate, 1994; EPA, 1987).

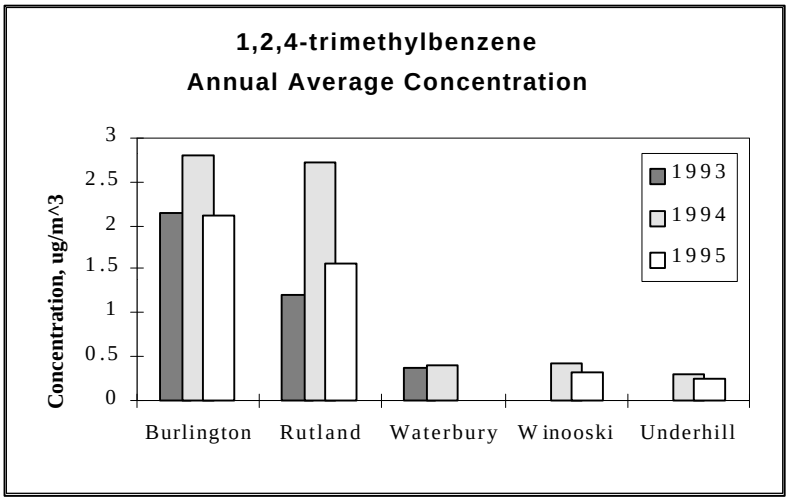


Figure 4.10-Annual average ambient air concentrations of 1,2,4-trimethyl benzene in micrograms per cubic meter (ug/m^3) at Vermont Monitoring Sites from 1993 to 1995. The Hazardous Ambient Air Standard (HAAS) is .15 ug/m^3. See section IV.B.3.

b. Limitations and Uncertainties

1,2,4-Trimethyl benzene concentrations were below the detection limit in 36% of the sample collected, creating uncertainty regarding the true ambient concentration of the compound. The standard (0.15 ug/m^3) is also less than the detection limit (0.49 ug/m^3) which generates uncertainty as to whether the compound truly exceeds the standard. This uncertainty is limited by examination of the low means which did exceed the standard in 10 of the 12 data sets. 1,2,4-Trimethyl benzene has not been monitored since March 1995 so current ambient levels are not known.

c. Implications of Revised Standard

Since the revised standard for1,2,4-trimethyl benzene will increase from 0.15 ug/m^3 to 297.6 ug/m^3, the air concentrations will no longer exceed the proposed revised standard and therefore the compound should be removed from the priority list.

d. Health Concerns

The primary way 1,2,4-trimethyl benzene, commonly referred to as pseudocumene, may enter the body is by inhalation of contaminated air into the lungs. Studies indicate that approximately two-thirds of the 1,2,4-trimethyl benzene inhaled into human lungs is actually retained.

About one-third of the retained 1,2,4-trimethyl benzene leaves the body unchanged through exhaled air. The remaining two-thirds is absorbed from the lungs into the bloodstream where it can be transported throughout the body (Jarnberg et al., 1996; Kostrzewski et al., 1997).

The majority of absorbed 1,2,4-trimethyl benzene is broken down into other compounds (metabolites) and leaves the body through the urine. Experimental studies in human volunteers suggest that this compound may significantly accumulate in adipose (fat) tissue (Jarnberg, 1996). This would imply that once exposure has stopped, total elimination of this compound from the body may take a few days up to a few weeks.

Inhalation of elevated levels of 1,2,4-trimethyl benzene, even for short periods of time, may irritate the eyes, respiratory tract and mucosal membranes. Headache, fatigue, nausea, irritation of the skin, eyes and mucous membranes, central nervous system depression asthmatic bronchitis, chemical pneumonitis or pulmonary edema have been reported in people, especially workers, who have inhaled elevated amounts of this compound for various amounts of time (HSDB, 1997; NIOSH, 1987). Short-term exposure to highly elevated levels may also produce hypothermia. Anxiety and nervousness were particularly noted in those who had been exposed for extended periods of time (HSDB, 1997). Long-term exposure may also result in disturbances in the blood forming organs and in the development of hypochromic anemia.

While experimental studies with laboratory animals indicate that 1,2,4-trimethyl benzene can cross the placenta, it was not found to be teratogenic in the specie studied (HSDB, 1997). Central nervous system depression, mucous membrane and respiratory irritation have also been noted in experiments with laboratory animals (NIOSH, 1987).

1,2,4-Trimethyl benzene may also be absorbed into the body across the skin. Dermal contact can cause skin irritation. Ingestion of elevated amounts of this compound may also produce nausea, vomiting, gastrointestinal or esophageal irritation (HSDB, 1997).

1,2,4-Trimethyl benzene does not have a United States Environmental Protection Agency weight of evidence or International Agency for Research on cancer classification.

C. Category III

1. Acrolein

a. Discussion

Acrolein was selected as a priority compound due to public concerns about the adequacy of the current standard. The current HAAS is 2.5 ug/m^3 (8 hr standard). Ambient levels range from a high of 0.31 ug/m^3 for one sample in Rutland in 1995 to a low of 0 (non-detect). Annual average concentrations are shown in Figure 4.11. Acrolein concentrations were below the detection limit in 90% of the samples collected.

In several large cities, acrolein has been measured at 9 ppb (21 ug/m^3)**.** Acrolein appears to be generated by local sources. Acrolein is a product of combustion and is also produced in secondary atmospheric chemical reactions from compounds such as 1,3-butadiene (EETE, 1995). Atmospheric half life is 10-17 hours (Kao, 1994).

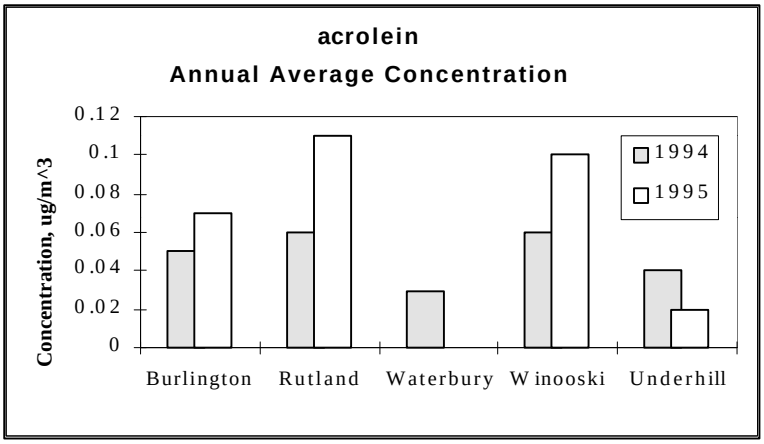


Figure 4.11-Average annual ambient air concentrations of acrolein in micrograms per cubic meter (ug/m^3) at Vermont Monitoring Sites from 1994 to 1995. The Hazardous Ambient Air Standard (HAAS) is 2.5 ug/m^3. See section IV.C.1.

b. Limitations and Uncertainties

The current standard is an 8 hour standard and as the samples are collected over 24 hours there is no way to really know if the standard is exceeded. The current monitoring program is not adequate to determine if an 8 hour standard has been exceeded. The high percentage of non-detect samples, 90%, adds to uncertainty regarding the true ambient concentration of acrolein. The detection limit for acrolein is a function of the sample volume and varies from sample to sample.

c. Implications of Revised Standard

Acrolein is recommended to be classified as a Category I contaminant. The proposed revised HAAS for acrolein would decrease from 2.5 ug/m^3 to 0.002 ug/m^3. At the proposed revised standard, current emissions would exceed the HAAS.

As stated above, acrolein is a product of combustion and is also produced in secondary atmospheric chemical reactions from compounds such as 1,3-butadiene. As stated earlier in this report, sources of 1,3-butadiene emissions include automobiles, waste incinerators, and wood fires.

d. Health Concerns

One way acrolein enters the body is through inhalation of contaminated air. Acrolein enters the lungs rapidly and a portion is taken up in the blood where it is metabolized and excreted through

the kidneys. When exposure ends acrolein levels in the lungs fall rapidly and the effects are terminated.

Exposure to acrolein in the environment produces irritation in the lung, eyes, nose and throat as the exposure increases from 170 to 430 ppb (390 to 990 ug/m^3)(ATSDR, 1990b). Extreme concentrations can produce severe lung damage and can be fatal. However at levels of exposure found in the ambient environment the toxic effects of acrolein are reversible when exposure stops.

Long term or repeated exposures have not been well studied in humans. Animal studies suggest that there is a potential for chronic lung toxicity at moderate levels of exposure. Certain persons may be more sensitive to acrolein. This could include the very young, the elderly and persons with respiratory diseases such as asthma.

Acrolein's actions in the presence of other irritants is not known but combined exposures should be expected to increase the toxicity.

Minimal risk levels for acrolein have been proposed to be 0.05 ppb (0.1 ug/m^3) for short term exposures and 0.009 ppb (0.02 ug/m^3) for longer term exposures (ATSDR, 1990b).

Acrolein has been classified as Class C: Possible human carcinogen by the United States Environmental Agency and Group 3: Unclassifiable as to Carcinogenicity to Humans by the International Agency for Research on Cancer.

V. Management Options

A. Local Emissions

As stated in Section IV, local emissions exceeding the proposed revised standards are benzene, 1,3-butadiene, formaldehyde, methylene chloride, and acrolein. Sources of emissions from these compounds are automobiles, gas stations, industry including incinerators and wood processing plants, wood stoves, furniture strippers, and garages.

Additional local emissions of concern but not exceeding the revised proposed standards are mercury and styrene. As stated in Section IV, the concern from mercury is the indirect risk from fish consumption. Vermont currently has a fish advisory in place concerning fish consumption.
Appendix D of this report contains an update from the University of Vermont School of Natural Resources on the research being performed on the effects of mercury in Lake Champlain.

Although the current air data for styrene does not show any emissions greater than the proposed revised standard, the Agency recommends relocating monitors periodically in order to get better representation of the air quality. The Agency recommends continuation of monitoring for mercury and styrene to observe any trends.

To address local contaminants exceeding the standard or causing public concern, the Agency recommends developing a Toxic Action Plan to propose methods of reducing emissions. In addition, the Agency recommends continuing monitoring to determine ambient levels and to observe any trends as regulatory actions are implemented.

B. Transported Emissions

Transported emissions exceeding the proposed revised standards are carbon tetrachloride, chloroform, and methyl chloride. As stated in Section IV**,** carbon tetrachloride was used extensively until it withdrawn from the market in the 1960s. Although it still has limited uses, it is being phased out. Since the atmospheric half life is 50-100 years, it will take a long time for current levels to decrease significantly. Chloroform has been found in the air from all areas of the United States and sources include pulp and paper mills, and water and wastewater plants that use chlorine as a disinfectant. The methyl chloride in the outdoor environment is almost totally from natural sources such as oceans and biomass. The atmospheric half-life is 1-2 years.

To address these transported emissions, the Agency recommends continuing monitoring to determine ambient levels and to observe any trends in the data. The ambient air concentrations for these transported compounds are not as high as the concentrations for the local emissions exceeding the standard.

C. Fine Particle Emissions

Many hazardous air contaminants are associated with the fine particle fraction of atmospheric samples. Organic compounds can be absorbed onto the surfaces of fine particles and deposited deep into the lungs. The U.S. EPA has recently established national standards for fine particulate matter (defined as particles less than 2.5 microns). Over the next three years, Vermont will be establishing monitoring sites throughout the state to determine current fine particulate levels in the ambient air.

Because the toxicity of fine particles is likely dependent on the individual chemical species comprising the particle, it is important to recognize the source and origin of these fine particle. All combustion sources generate fine particles, either directly as fly ash, or indirectly as combustion gases cool in the atmosphere and absorb onto the surfaces of particles. Emissions from fossil fuel combustion, especially diesel fuel and wood, all contribute to the fine particle concentrations found in ambient air. Future regulatory efforts to control airborne toxics will need to focus on these sources of fine particles if the state is to be successful in reducing the levels of hazardous contaminants in our air.

VI. Risk Issues

A. Allowable Risk Level in Vermont

1. Background

The state of Vermont Air Pollution Control Regulations as amended in March 1989 established a maximum allowable incremental risk level of one in one million. Consequently, the Regulations dictated that the ambient air standard for each Category I contaminant (known or suspected carcinogens) be set at a concentration estimated to correspond to a one in one million (1×10^{-6}) increase in the probability of developing cancer (over and above the background cancer rate) over a lifetime of exposure. The 1989 version of the Regulations required that this risk be implemented as a total ambient air quality standard not to be exceeded. Thus, no source was allowed to cause or contribute to an exceedance of any ambient air standard. For example, a source would not be allowed to discharge any compound if the potential incremental lifetime carcinogenic risk (hereafter referred to as "risk") associated with inhalation of existing ambient air was estimated to already be greater than1×10^{-6} or if emissions from that point source would cause the total risk associated with inhalation of ambient air to then exceed 1×10^{-6}.

When the 1989 Regulations were originally adopted, there was no available data on local levels of hazardous air contaminants in ambient air. Once the Agency began receiving such data, it became apparent that there were compounds present in outdoor air that already exceeded the established standards. Since the Regulation did not have a mechanism to address emissions in localities where the existing ambient air quality was found to exceed an established ambient air standard, the Agency had to deny permits to modify facilities emitting these compounds even in circumstances where the modification would have improved overall air quality.

In response to this dilemma, the Air Pollution Control Regulations were revised in 1993. A five year review period was set aside to review the scientific basis for each ambient standard and the impacts of regulating individual emitters without considering the existing levels of contaminants in ambient air. Therefore, during the past five years industry has been regulated by requiring that no point source emission result in greater than a 1×10^{-6} risk without taking the existing ambient air quality into account. No limit was set as to the maximum allowable risk associated with inhalation of outdoor ambient air.

The Agency is now at the end of this five year period. A decision must be made regarding what level of risk is to be considered negligible, and how this value is to be applied, i.e., whether the Agency should re-establish a total ambient air quality goal not to be exceeded, or continue to apply the standards at individual point sources without consideration of existing ambient air concentrations.

2. Discussion of Allowable Risk Level

It has traditionally been assumed that no threshold level of exposure exists for potential carcinogens. Consequently, an increase in the theoretical probability of developing cancer, over and above the background cancer rate, is assumed to be associated with any exposure greater than zero. This is a conservative assumption that in some instances may result in risk estimates greater than zero, when the actual value is zero. While the Committee is aware of this, it is agreed that the assumption of no threshold level of exposure is a conservative, public health protective measure to make in the assessment of potential carcinogens. Given this assumption and because the Agency is responsible for ensuring public health protection from outdoor exposures to toxic air pollutants, the Agency believes it is prudent to establish ambient air standards adequate to protect public health with an ample margin of safety. For carcinogens, where it is assumed there is no absolutely risk-free level of exposure, it becomes necessary for the Agency to establish a maximum allowable level of risk.

When selecting a level of maximum allowable risk, it is important to think about what this value represents in real life. In the vast majority of cases, a quantitative estimate of a compound's ability to cause cancer in humans is predicted by extrapolating data obtained from high dose experiments with laboratory animals to low dose environmental exposures in humans. More so than specifically bred laboratory animals, a great deal of heterogeneity exists in the human population. The actual risk experienced by individuals in a population thus varies between individuals as well as within an individual, depending on the state of the body at the time of exposure. At one particular point in time, one person may be particularly susceptible to developing an adverse effect even with highly restricted exposure, while another may be particularly resistant to developing an adverse effect despite long term elevated exposure. Because it is not possible to identify exactly which individual at which time is at greatest risk, a maximum allowable level of risk is designed to be protective of sensitive, but not hyper sensitive, individuals.

Various federal and state agencies employ different levels of maximum allowable risk. Values between one and one million (1×10^{-6}) to one in ten thousand (1×10^{-4}) are typically used. The objective of each agency is to choose a level of risk believed to represent a negligible increase in potential risk over background risk, for the population of concern. The same agency may employ different levels of maximum allowable risk in different situations, for example, less at residential sites than industrial sites.

Typically, risks estimated to be associated with individual compounds are assumed to be additive. This means the estimated risk associated with exposure to each compound are summed together to yield one estimate of total risk. For example, if the carcinogenic risk associated with inhalation of chemical X is estimated to be 1×10^{-7} and for chemical Y is estimated to be 5×10^{-7}, then the total risk estimated to be associated with inhalation of these two chemicals in ambient air is 6×10^{-7}. It should be noted this is a conservative approach due to the conservative, health protective nature of each individual risk estimate. Total risk of 1×10^{-6} from combined exposure to a number of potential carcinogens is typically assumed to be negligible and not of concern for public health. Many in the risk assessment community typically consider a total risk of 1×10^{-5} or 1×10^{-4} to represent a potential level of increased concern for public health.

3. Application of Maximum Allowable Level of Risk to Existing Air

Once the Vermont Agency of Natural Resources chooses a maximum allowable level of risk, it is necessary to determine how this value will be applied. As stated above, from 1989 to 1993, the Agency applied the risk level of $1x10^{-6}$ as a total ambient air goal not to be exceeded. This meant that no source was allowed to cause or contribute to ambient concentrations in excess of any standard. Thus, new sources were precluded from emitting those compounds where existing ambient air quality was estimated to be above the total ambient air goal of $1x10^{-6}$. This was regardless of whether the potential risk associated with inhalation exposure to emissions from the new source was negligible, i.e., less than 1×10^{-6}.

From 1993 to the present, the maximum allowable risk level has been applied as a point source emission limit without considering the existing concentrations of toxic pollutants in outdoor air. During this period, no total ambient quality goal was established.

The Agency has examined its risk assessment methodology with the assistance of the Toxicological Advisory Committee. It now must choose a maximum allowable level of risk and decide how it is to be applied. Various options are available. For example, some federal and state agencies have chosen to establish a tiered approach to setting maximum allowable risk. For example, risk associated with emission of an individual compound from a point source may not exceed 1×10^{-6}, but the total risk associated with inhalation exposure to all compounds being emitted from a source may not exceed a risk of 1×10^{-5}. Some agencies have chosen to use an iterative approach to risk management. This involves using screening assessments to identify those point sources of greatest potential concern for public health. Maximum estimates of potential emissions and exposure factors are used to determine whether a facility should undergo a more detailed assessment. For example, if the total risk derived using such elevated values is below the established risk level, no further assessment would be required and a permit could be issued. If the total risk is above the established risk level, then a refined analysis would be required.

Regardless of the methodology employed by these various agencies, those sources with total estimated risk less than 1×10^{-6} are routinely not considered to be of significant concern for public health. Those sources with total estimated risk between 1×10^{-6} and 1×10^{-4} are often identified as requiring further investigation.

The Committee agrees that $1x10^{-6}$ is a negligible risk, however, whether or not it is acceptable to allow additional emissions even at this negligible level should depend on the existing air quality of the area in question. If total risk associated with inhalation of existing ambient air is estimated to already be highly elevated, above 10^{-4} for example, it may not be appropriate to allow additional emissions, even at the $1x10^{-6}$ level**,** without further investigation.

The Committee agrees that it may be prudent to define a total ambient air quality goal that should not be exceeded. Individual point sources would then be required to control emissions so that the total risk associated with inhalation of ambient air is below the total ambient air quality goal.

B. Air Toxic Programs in Other States

The Committee has obtained information from other northeastern states and the State of Washington to determine how standards are developed from these states to control emissions from air toxics and how they are applied to ambient air. It is the Committee's understanding that air standards are applied only as point source limits and none of the northeastern states employ a total ambient air quality goal. It is also the Committee's understanding that existing levels of air toxics in the northeast are not considered when reviewing specific source emissions.

New Jersey

For cancer effects, New Jersey uses a $1x10^{-6}$ risk level for screening acceptable impacts; but the acceptable risk can go as high as $1x10^{-4}$ under a refined analysis. For noncancer effects, they use a Hazard Index (HI) of 1 for screening; but may go higher for refined analysis.

New Hampshire

New Hampshire has adopted regulations for standards for some contaminants and will be adopting new regulations for additional contaminants in March of 1998. The standards for the new contaminants will employ cancer risk values, RfCs, and modified occupational values. Modified occupational values include safety factors and time adjustment factors based upon reproductive toxicity, cancer effects, mutagenicity, acute toxicity and systemic noncancer effects. Acceptable cancer risks for specific source emissions will be set at the $1x10^{-5}$ level at the property line.

Connecticut

Connecticut adopted regulations in 1986 for hazardous air pollutants. The standards are based on occupational levels with various factors applied. These standards are applied at the stack (point of emission).

Maine

Maine repealed its air toxics program but Maine is interested in reinstating a program.

Rhode Island

Rhode Island adopted regulations for air toxics in 1988. For cancer effects, emissions are controlled at the $1x10^{-6}$ risk level, or at the$1x10^{-5}$ risk level if emission control technology is employed. In addition, Rhode Island uses an RfC based approach for noncarcinogens.

Rhode Island may be updating standards to take into account current toxicity information.

Massachusetts

Massachusetts' air toxics program is applied in limited circumstances for specific source categories such as municipal waste incinerators. Acceptable risks are set at $1x10^{-6}$ for single contaminants, and $1x10^{-5}$ for multiple contaminants.

New York

New York is currently in the process of adopting its air toxic guidelines into regulation. The New York approach uses modified occupational standards or chemical specific risk assessments to establish acceptable concentration levels which individual sources must attain at their property line. In the assessment of new pollution sources to be constructed, New York is considering restricting new source impacts to one-half the acceptable concentration level to account for background levels of the toxic pollutant and provide a further margin of safety.

Washington State

Washington State uses acceptable source impact levels set at the 1×10^{-6} risk level for review of new or modified sources of toxic air pollutants. If the source's impacts would exceed this level, then a refined risk assessment must be done to demonstrate the ambient air risk from the compound will be no greater than 1×10^{-5}. If the impacts would exceed the 1×10^{-5} level, a new source could still be permitted if it implemented an environmentally beneficial program such as replacing uncertified wood stoves, buying up older, polluting cars, paying for car/van pools, eliminating an existing source of toxic emissions at another factory, or a combination of these approaches in a effort to reduce the risk associated with outdoor air exposure in a community.

VII. Future Actions and Recommendations

A. Continue Monitoring

As stated in Section V, Management Options, the Agency should continue monitoring for air toxics to determine ambient levels and to observe any trends as regulatory actions are implemented. The air monitoring data allows the Agency to compare ambient air levels to the standards to determine those compounds that consistently exceed the standard.

B. Toxic Action Plan

As stated in Section V, the Agency should develop a Toxic Action Plan to address those contaminants that consistently exceed the standard and those contaminants such as mercury that provide concerns due to indirect health risks.

C. Review of Standards

The Agency recommends conducting a review of standards every five years so that standards can be updated based on the most recent toxicological information. Unless revisions are proposed, the reviews should follow the proposed methodologies outlined in Sections II.B and II.C. If during the five year periodic update it is determined that a change in toxicity information will significantly impact the standard, the Agency may consider requesting a line item regulatory amendment.

D. Short Term Standards

For several Hazardous Air Contaminants, it may be appropriate to develop two sets of ambient air standards, one to be protective of potential adverse health effects that may be associated with long term, chronic exposures and a second to protect against potential adverse health effects that may be associated with acute or short term exposures. Future efforts should focus on developing criteria by which to identify such compounds and identifying acceptable sources of short term standards. One potential source is the National Advisory Committee on Acute Exposure Guideline Levels for Hazardous Substances. As of this writing, short term standards, referred to as Acute Exposure Guideline Levels (AEGLs) are available for three current Category I compounds (aniline, arsine and ethylene oxide) and seven current Category III compounds (ammonia, chlorine, 1,2-dichloroethane, fluorine, hydrogen chloride, nitric acid and phosphine). Other potentially useful short-term exposure limits are occupational short-term exposure limits and/or ceiling limits derived for various contaminants by ACGIH, OSHA and NIOSH. In the interim, as a temporary measure, the environmental level of any contaminant should not be allowed to exceed an established work place ceiling limit.

E. Other Endpoints

Toxic air standards are designed to protect the public from unsafe exposures to compounds with defined human health effects. In order to establish an air standard it is necessary to identify the

human health effects and the exposure level which could cause an effect. As part of the process of setting protective ambient standards, the Committee recommends considering other health effects or endpoints in addition to cancer. The committee recommends considering other endpoints such as endocrine disruptors or environmental respiratory disease in future regulatory decisions.

1. Endocrine Disruptors

Endocrine disruption refers to an effect on the hormonal balance in humans. Endocrines are responsible for the long term biological control of growth and development. Growth and development are signaled and controlled by both the external environment and the internal hormonal balance. Chemicals that interfere with or block the function of the hormones would profoundly affect the development of the system.

Presently there is insufficient information about the endocrine disruptors to formulate a regulatory approach. However, it is important to be aware of and monitor this new area of investigation for future regulatory actions.

2. Environmental Respiratory Disease

Environmental respiratory disease refers to complex respiratory conditions that are exacerbated if not induced by the presence of toxics in the air. For example, air contaminants that provide a chronic irritant effect may increase the time it takes to recover from bacterial or viral induced colds and upper respiratory tract infections.

When the physiological conditions are sporadic or rare, such as colds or pneumonia, it is difficult to identify the contaminants that cause or exacerbate the health effect. However, there are conditions such as childhood and adult asthma which are endemic to a population and therefore benefits could be derived from reduction in exposure to airborne chemicals that precipitate or exacerbate the disease.

Some initial studies are being done which could provide another unique way to protect the public health. These studies do not yet yield definitive information but could in the near future identify an appropriate public health step for regulatory purposes.

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Appendix A

Category I

Algorithm and Tables

APPENDIX A
FIGURE 1
Discussed in Section II.B.1

CATEGORY I CONTAMINANTS:
ALGORITHM USED TO DERIVE HAZARDOUS AMBIENT AIR STANDARD

Goal: Estimate ambient concentration that corresponds to an excess lifetime carcinogenic risk of one in one million

Algorithm:

$$RISK = 1 - e^{-(CPF * CONCENTRATION * DAILY\ INHALATION\ RATE * F/BODY\ WEIGHT)}$$

Body Weight - 70 kilograms [kg]

Concentration - Ambient concentration (milligram chemical per cubic meter ambient air [ug/m^3]) (Hazardous Ambient Air Standard)

CPF - Cancer Potency Factor (milligram chemical\kilogram body weight-day)$^{-1}$ [$(mg/kg/d)^{-1}$]

Daily Inhalation Rate - 20 cubic meters per day [m^3/day]

F - Absorption Factor of 100 percent (1) employed for all compounds

Risk - Excess lifetime carcinogenic risk

SET RISK EQUAL TO 1 X 10^{-6} AND SOLVE FOR CONCENTRATION

$$CONCENTRATION\ (ug/m^3) = \frac{\ln [1 - (1 \times 10^{-6})] * CF}{- [\ CPF\ (mg/kg/d)^{-1} * Inhalation\ Rate\ (m^3/d) * F\ /\ Body\ Weight\ (kg)]}$$

CF - Conversion Factor 1000 micrograms per milligram

EQUATION REDUCES TO:

$$HAAS\ (ug/m^3) = .0035\ /\ (CPF * F)$$

APPENDIX A
TABLE 1
Discussed in Section II.B.2

CATEGORY I CONTAMINANTS: CLASSIFICATION STATUS

Category I Contaminant	CAS Number	US EPA Weight of Evidence	IARC Classification
Acrylamide	79-06-1	B2	2A
Acrylonitrile	107-13-1	B1	2A
Allyl chloride	107-05-1	C	3
Aniline	62-53-3	B2	3
Antimony trioxide	1309-64-4	No Data	2B
Arsenic, total	7440-38-2	A	1
Arsine	7784-42-1	No Data	No Data
Asbestos	1332-21-4	A	1
Benzene	71-43-2	A	1
Benzidine	92-87-5	A	1
Benzo(a)pyrene	50-32-8	B2	2A
Beryllium, total	7440-41-7	B2	1
1,1-Biphenyl	92-52-4	D	No Data
Bromoform	75-25-2	B2	3
1,3-Butadiene	106-99-0	B2	2A
Cadmium, total	7440-43-9	B1	1
Carbon tetrachloride	56-23-5	B2	2B
Chloroform	67-66-3	B2	2B
Chloroprene (a)	126-99-8	No Data	3
Chromium, total (b)	18540-29-9	A	1
Diazomethane	334-88-3	No Data	3
Dichloroethyl ether	111-44-4	B2	No Data
Dimethyl sulfate	77-78-1	B2	2A
2,4-Dintrotoluene	121-14-2	B2 (c)	2B
Dioxane	123-91-1	B2	2B
Epichlorohydrin	106-89-8	B2	2A
Ehtylene dibromide	106-93-4	B2	2A
Ethylene dichloride	107-06-2	B2	No Data
Ethylene oxide	75-21-8	B1	1
Formaldehyde	50-00-0	B1	2A
Hexachlorobenzene	118-74-1	B2	2B
Hexachlorobutadiene	87-68-3	C	3
Hexachloroethane	67-72-1	C	3
Methyl bromide	74-83-9	D	3
Methyl chloride	74-87-3	C	3
Methylene chloride	75-9-2	B2	No Data
Methyl iodide	74-88-4	No Data	3
Nickel carbonyl	13463-39-3	B2	No Data
Nickel (refinery dust) (d)		A	1
2-Nitropropane	79-46-9	B2	2B
PCDDs/PCDFs (e)	1746-1-6	B2	1
Polychlorinated Biphenyls	1336-36-3	B2	2A
Propylene dichloride	78-87-5	B2	No Data
Propyleneimine	75-55-8	No Data	No Data
Propylene oxide	75-56-9	B2	2B
1,1,2,2-Tetrachloroethane	79-34-5	C	3
Tetrachloroethylene	127-18-4	B2-C (f)	2A
o-Toluidine	95-53-4	No Data	2B
1,1,2-Trichloroethane	79-00-5	C	3
Trichloroethylene	79-1-6	B2-C (g)	2A
2,4,6-Trichlorophenol	88-06-2	B2	No Data
Vinyl chloride	75-01-4	A	1

Notes:

CAS - Chemical Abstracts Service

IARC - International Agency for Research on Cancer

US EPA - United States Environmental Protection Agency

(a) - Included as Category I based on information presented in National Toxicology Program abstract.

(b) - Values cited are for hexavalent chromium (Cr VI).

(c) - Based on evaluation of a mixture of 2,4 and 2,6 dinitrotoluene.

(d) - Nickel refinery dust is a mix of many nickel moieties and it is not certain what the carcinogenic species is in the refinery dust (IRIS, 1997).

(e) Information cited is for 2,3,7,8-tetrachlorodibenzo-p-dioxin.

(f) - Provisional information provided in Risk Assessment Issue paper for Tetrachloroethylene. Superfund Technical Support Center.

(g) - Provisional information provided in Risk Assessment Issue paper for Trichloroethylene. Superfund Technical Support Center.

APPENDIX A

TABLE 2

Discussed in Section II.B.2

CATEGORY I CONTAMINANTS PROPOSED TO BE RECLASSIFIED AS CATEGORY II CONTAMINANTS

Contaminant	US EPA Weight of Evidence	IARC Group Classification	CPFi [1/(mg/kg-day)]	CPFo [1/(mg/kg-day)]	RfC (ug/m^3)	TLV (ug/m^3)	PEL (ug/m^3)	REL (ug/m^3)	Time Factor	Uncertainty Factor (a)	Proposed Revised HAAS (ug/m^3)	Existing HAAS (ug/m^3)
Arsine	No Data	No Data			0.05	160	200	2 (c,d)		10 (e)	0.005	0.01
1,1-Biphenyl	D	No Data				1,300	1,000		4.2	100	2.38 *	0.01
Diazomethane	No Data	3				340 (a)	400	400	4.2	1000 (e)	0.081	0.01
Methyl bromide	D	3			5	3,900	80,000 (b)	(d)		10 (e)	0.5*	0.01
Methyl iodide	No Data	3				12,000	28,000	10,000 (d)	4.2	1000 (e)	2.38*	0.01
Propylene imine	No Data	No Data				4,700	5,000	5,000 (d)	4.2	1000 (e)	1.12*	0.01

Notes:

Shading indicates basis of proposed HAAS.

* - Indicates proposed revised HAAS differs from existing HAAS by at least an order of magnitude.

ACGIH - American Conference of Governmental Industrial Hygienists

CPFi - Inhalation Cancer Potency Factor

CPFo - Oral Cancer Potency Factor

HAAS - Hazardous Ambient Air Standard

IARC - International Agency for Research on Cancer

PEL - Permissible Exposure Limit established by federal Occupational Safety and Health Administration (OSHA), downloaded September 29, 1997.

REL - Recommended Exposure Limit established by federal National Institute for Occupational Safety and Health (NIOSH), June 1994.

RfC- Inhalation Reference Concentration as cited in US EPA Integrated Risk Information System database.

TLV - Threshold Limit Value as cited in 1997 Threshold Limit Values for Chemical Substances and Physicals Agents ad Biological Indices, ACGIH.

US EPA - United States Environmental Protection Agency

(a) - Identified as A2: Suspected Human Carcinogen by ACGIH.

(b) - Value represents a PEL ceiling level which is not to be exceeded at any time.

(c) - Value represents REL ceiling level not to be exceeded at any time.

(d) - Identified as potential occupational carcinogen by NIOSH.

(e) - Includes an extra uncertainty factor of 10 because compound is identified as potential carcinogen by ACGIH or NIOSH.

APPENDIX A
TABLE 3
Discussed in Section II.B.2

CATEGORY I CONTAMINANTS: UPDATED TOXICITY VALUES

Category I Contaminant:	Updated Cancer Potency Factor [(mg/kg/day)^-1)](a)		Toxicity Value Associated with Existing HAAS	
With Static Inhalation Cancer Potency Factor				
Acrylonitrile	2.4E-01	(b)	Same	
Benzene	2.9E-02	(b)	Same	
Benzidine	2.3E+02	(c)	Same	
Chromium, total	4.1E+01	(b,d)	Same	
Ethylene oxide	3.5E-01	(b)	Same	
Formaldehyde	4.5E-02	(b)	Same	
With Updated Inhalation Cancer Potency Factor				
Beryllium, total	8.4E+00	(b)	2.60E+00	
1,3-Butadiene	1.8E+00	(b,c)	1.00E-01	
Cadmium, total	6.3E+00	(e)	6.10E+00	
Carbon tetrachloride	5.3E-02	(b)	1.30E-01	
Nickel, total (f)	8.4E-01	(b)	1.05E+00	
Vinyl chloride	3.0E-01	(b)	1.75E-02	
Formerly with Only Oral Cancer Potency Factor, Now With Inhalation Cancer Potency Factor				
Arsenic, total	1.5E+01	(e)	1.50E+01	(Oral)
Benzo(a)pyrene	6.1E+00	(g)	1.15E+01	(Oral)
Chloroform	8.1E-02	(b)	8.10E-02	(Oral)
Dichloroethyl ether	1.1E+00	(b)	1.14E+00	(Oral)
Epichlorohydrin	4.2E-03	(b)	9.90E-03	(Oral)
Ethylene dibromide	7.6E-01	(b)	4.10E+01	(Oral)
Ethylene dichloride	9.1E-02	(b)	9.10E-02	(Oral)
Hexachlorobenzene	1.6E+00	(b)	1.67E+00	(Oral)
Hexachlorobutadiene	7.8E-02	(b)	7.75E-02	(Oral)
Hexachloroethane	1.4E-02	(b)	1.42E-02	(Oral)
Methylene chloride	4.7E-7 (unit risk)	(h,i)	1.40E-02	(Oral)
PCDDs/PCDFs	1.5E+05	(b,j)	1.56E+05	(Oral)
Polychlorinated Biphenyls	2.0E+00	(k)	4.34E+00	(Oral)
1,1,2,2-Tetrachloroethane	2.0E-01	(b)	2.00E-01	(Oral)
Tetrachloroethylene	2.0E-03	(l,m)	5.10E-02	(Oral)
1,1,2-Trichloroethane	5.7E-02	(b)	5.73E-02	(Oral)
Trichloroethylene	6.0E-03	(n)	1.10E-02	(Oral)
2,4,6-Trichlorophenol	1.0E-02	(b)	1.99E-02	(Oral)

APPENDIX A
TABLE 3
Discussed in Section II.B.2

CATEGORY I CONTAMINANTS: UPDATED TOXICITY VALUES

Category I Contaminant:	Updated Cancer Potency Factor [(mg/kg/day)^-1)] (a)		Toxicity Value Associated with Existing HAAS	
Formerly Without Toxicity Values, Now With Inhalation Cancer Potency Factor				
Acrylamide	4.50E+00	(b,o)		
Bromoform	3.9E-03	(e)		
Methyl chloride	6.3E-03	(b)		
2-Nitropropane	9.4E+00	(b)		
Propylene oxide	1.3E-02	(c)		
Formerly Without Toxicity Values, Now With Oral Cancer Potency Factor				
Aniline	.0057 (Oral)	(p)		
Dioxane	.011 (Oral)	(p)		
Propylene dichloride	.068 (Oral)			
Still With no inhalation or Oral Cancer Potency Factor or Inhalation Reference Concentration				
1,1-Biphenyl *				
Diazomethane *				
Dimethyl sulfate				
Methyl iodide *				
Nickel carbonyl				
Propylene imine *				
o-Toluidine				
With no inhalation or Oral Cancer Potency Factor but with Inhalation Reference Concentration				
Antimony trioxide	.2	(RfC)		
Arsine *	.05	(RfC)		
Chloroprene	7	(RfC)		
Methyl bromide *	5	(RfC)		
Allyl chloride	1	(RfC)	0.0119	(Oral)
With Only Updated Oral Cancer Potency Factor				
2,4-Dintrotoluene	.68 (Oral)	(q)	3.10E-01	(Oral)
Mineral Fiber				
Asbestos	.23 [(fibers/milliliter)]-1	(r)		

Notes:
CAS - Chemical Abstracts Service.
HAAS- Hazard Ambient Air Standard in ug/cubic meter.
Oral - Indicates that existing HAAS may be based upon an oral cancer slope factor
RfC - Inhalation Reference Concentration in micrograms of compound per cubic meter of ambient air
* - Compound proposed to be reclassified as Category II contaminant.
(a) - Value cited is inhalation cancer potency factor unless otherwise noted.
(b) - Value cited in HEAST, July 1997.
(c) - Cancer slope factor presented in IRIS citation.
(d) - Exposure was to both Chromium III and VI. Since only Cr VI has been found to be carcinogenic in animal studies, US EPA classified only Cr VI as a human carcinogen. US EPA assumed Cr VI to be 1/7th of total Chromium.
(e) Cancer slope factor derived from unit risk by assuming inhalation rate of 20 cubic meters/day and 70 kilogram body weight.
(f) - Values are for nickel refinery dust. Per IRIS citation 12/96, "Nickel refinery dust is a mix of many nickel species and it is not certain what the carcinogenic nickel species is in the refinery dust."
(g) Value removed from IRIS in 1993 but still conservatively employed by many in risk assessment community.
(h) - Because this unit risk was derived using a pharmacokinetic model, it is not suitable for use in the calculation of a cancer slope factor.
(i) - This unit risk may not be applicable to acute, high level exposures.
(j) - Information is representative of 2,3,7,8-Tetrachlorodibenzo-p-dioxin (1746-01-6).
(k) - Upper bound slope estimate for high risk and persistence. Recently recommended for use in evalualtion of inhalation of dust or aerosol exposures. US EPA, 1996. PCBs: Cancer Dose-Response Assessment and Application to Environmental Mixtures and adopted by IRIS 10/1/96.
(l) - Provisional Information. Risk Assessment Issue Paper for Tetrachloroethylene. Superfund Technical Support Center.
(m) - Based on geometric mean of unit risks reported in Risk Assessment Issue Paper for Tetrachloroethylene.
(n) - Provisional information. Risk Assessment Issue Paper for Trichloroethylene. Superfund Technical Support Center.
(o) - Inhalation unit risk is based on oral data.
(p) - Indicates compounds also with an noncarcinogenic inhalation reference concentration.
(q) - Value cited is oral cancer potency factor derived for a mixutre of 2,4 and 2,6 dinitrotoluene.
(r) - Unit risk. Additive combined risk of lung cancer and mesothelioma.
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APPENDIX A
TABLE 4
Discussed in Section II.B.2

CATEGORY I CONTAMINANTS: PROPOSED REVISED HAZARDOUS AMBIENT AIR STANDARDS

Category I Contaminant	CAS Number	Proposed Revised HAAS (ug/m^3)		Existing HAAS (ug/m^3)	
Acrylamide	79-06-1	0.00078	I	0.01	D
Acrylonitrile	107-13-1	0.015	I	0.015	I
Allyl chloride	107-05-1	0.10	R	0.29	O
Aniline	62-53-3	0.61	O	0.01	D
Antimony trioxide	1309-64-4	0.02	R	0.01	D
Arsenic, total	7440-38-2	0.00023	I	0.00023	O
Arsine	7784-42-1	(a)		0.01	D
Asbestos	1332-21-4	.000004 (b)		0.00012	
Benzene	71-43-2	0.12	I	0.12	I
Benzidine	92-87-5	0.000015	I	0.000015	I
Benzo(a)pyrene	50-32-8	0.00057	I	0.0003	O
Beryllium, total	7440-41-7	0.00042	I	0.0013	I
1,1-Biphenyl	92-52-4	(a)		0.01	D
Bromoform	75-25-2	0.90	I	0.01	D
1,3-Butadiene	106-99-0	0.0019	I	0.035	I
Cadmium, total	7440-43-9	0.00056	I	0.00057	I
Carbon tetrachloride	56-23-5	0.07	I	0.067	I
Chloroform	67-66-3	0.043	I	0.043	O
Chloroprene	126-99-8	0.70	R	0.01	D
Chromium, total (c)	18540-29-9 (d)	0.000085	I	0.000085	I
Diazomethane	334-88-3	(a)		0.01	D
Dichloroethyl ether	111-44-4	0.0032	I	0.0031	O
Dimethyl sulfate	77-78-1	0.01	D	0.01	D
2,4-Dintrotoluene	121-14-2	0.005 (e)	O	0.011	O
Dioxane	123-91-1	0.32	O	0.01	D
Epichlorohydrin	106-89-8	0.83	I	0.35	O
Ethylene dibromide	106-93-4	0.0046	I	0.000085	O
Ethylene dichloride	107-06-2	0.038	I	0.038	O
Ethylene oxide	75-21-8	0.01	I	0.010	I
Formaldehyde	50-00-0	0.078	I	0.08	
Hexachlorobenzene	118-74-1	0.0022	I	0.0021	O
Hexachlorobutadiene	87-68-3	0.045	I	0.045	O
Hexachloroethane	67-72-1	0.25	I	0.25	O
Methyl bromide	74-83-9	(a)		0.01	D
Methyl chloride	74-87-3	0.56	I	0.01	D
Methylene chloride	75-9-2	2.0 (b)		2.00	O
Methyl iodide	74-88-4	(a)		0.01	D
Nickel carbonyl	13463-39-3	0.01	D	0.01	D
Nickel, total (f)		0.0042	I	0.0033	I
2-Nitropropane	79-46-9	0.00037	I	0.01	D
PCDDs/PCDFs (g)		2.33e-08	I	2.00e-08	O
Polychlorinated Biphenyls	1336-36-3	0.0018 (h)	I	0.00081	O
Propylene dichloride	78-87-5	0.051	O	0.01	D
Propyleneimine	75-55-8	(a)		0.01	D
Propylene oxide	75-56-9	0.27	I	0.01	D
1,1,2,2-Tetrachloroethane	79-34-5	0.018	I	0.017	O
Tetrachloroethylene	127-18-4	1.8 (i)	I	0.41	O
o-Toluidine	95-53-4	0.01	D	0.01	D
1,1,2-Trichloroethane	79-00-5	0.061	I	0.061	O
Trichloroethylene	79-1-6	.58 (j)	I	0.42	O
2,4,6-Trichlorophenol	88-06-2	0.35	I	0.18	O
Vinyl chloride	75-01-4	0.012	I	0.20	I

Notes:

Indicates compound where proposed revised HAAS differs by at least one order of magnitude from existing HAAS.

CAS - Chemical Abstracts Service.
D - Indicates default standard of .01 ug/cubic meter.
HAAS- Hazard Ambient Air Standard in ug/cubic meter.
I - Indicates value derived using an inhalation cancer potency factor.
O - Indicates value derived using an oral cancer potency factor as a surrogate.
R - Indicates value derived using one-tenth an inhalation reference concentration.
(a) - Proposed to be reclassified as Category II compound. Please see Appendix A, Table 2.
(b) - Value cited in US EPA Integrated Risk Information System database as corresponding to an excess lifetime cancer risk of one in one million.
(c) - Exposure was to both Chromium III and VI. Since only Cr VI has been found to be carcinogenic in animal studies, US EPA classified only Cr VI as a human carcinogen. US EPA assumed Cr VI to be 1/7th of total Chromium.
(d) - CAS number is for Chromium VI.
(e) - Value cited is oral cancer potency factor derived for a mixutre of 2,4 and 2,6 dinitrotoluene.
(f) - Values are for nickel refinery dust. Per IRIS citation 12/96, "Nickel refinery dust is a mix of many nickel species and it is not certain what the carcinogenic nickel species is in the refinery dust."
(g) - Information is representative of 2,3,7,8-Tetrachlorodibenzo-p-dioxin (1746-01-6).
(h) - Derived using upper bound slope estimate for high risk and persistence. Recently recommended for use in evalualtion of inhalation of dust or aerosol exposures. US EPA, 1996. PCBs: Cancer Dose-Response Assessment and Application to Environmental Mixtures and adopted by IRIS 10/1/96.
(i) - Based on provisional information in Risk Assessment Issue Paper for Tetrachloroethylene. Superfund Technical Support Center.
(j) - Based on provisional information provided in Risk Assessment Issue Paper for Trichloroethylene. Superfund Technical Support Center.

Appendix B

Category II and Category III

Tables

APPENDIX B
TABLE 1
Discussed in Section II.C.2

CATEGORY II AND CATEGORY III CONTAMINANTS THAT NOW MEET CATEGORY I CRITERIA

Contaminant	Current Category	US EPA Weight of Evidence	IARC Group Classification	Inhalation CPF [1/(mg/kg-day)]	Oral CPF [1/(mg/kg-day)]	Inhalation RfC (ug/m^3)	Proposed Revised HAAS (ug/m^3)	Existing HAAS (ug/m^3)
Lead Compounds	II	B2	2B				0.01	0.25
Pentachlorophenol	II	B2	2B		1.2e-01		0.029	1.19
Silica, crystalline *	II		1				(a)	0.12
Styrene	II		2B			1000	100	512
Acetaldehyde	III	B2	2B	7.70e-03		9	0.45	1800
Acrolein	III	C	3			0.02	0.002	2.5
Bromodichloromethane	III	B2	2B		6.20e-02		0.056	42
Dibromochloromethane	III	C			8.40e-02		0.042	39
1,1-Dichloroethane	III	C					0.01	19300
Furfural	III	(b)				50 (c)	5	80
Isophorone	III	C			9.50e-04		3.68	1400
Nitrobenzene	III		2B			2 (c)	0.2	119
1,2,3-Trichloropropane	III	B2			7 (d)		0.0005	14.3
Vinyl Acetate	III		2B			200	20	350

Notes:

Indicates compound where proposed HAAS differs by at least one order of magnitude from existing HAAS.

CPF - Cancer Potency Factor

HEAST - US EPA Health Effects Assessment Summary Tables. July 1997.

IARC - International Agency for Research on Cancer

RfC- Reference Concentration

US EPA - United States Environmental Protection Agency

Unless otherwise noted, source of updated toxicity values is US EPA Integrated Risk Information System (IRIS) database.

*** - Please note that NTP has announced its intent to review Silica, crystalline for listing as a known human carcinogen in the 9th Edition of the NTP Report on Carcinogens. However, 3 members of tne Vermont Air Toxicological Advisory Committee do not agree that Silica, crystalline should be reclassified as a Category I Contaminant.**

(a) - Under review. To be determined at time of rule making because standard cancer models are not appropriate for assessing this compound.

(b) - National Toxicology Program abstract meets criteria for Category I.

(c) - Value presented in Table 2 of HEAST. Caveat: Derived from methodology that isn't current with the interim inhalation methodology used by the RfD/RfC workgroup.

(d) - Source: July 1997 US EPA Health Effects Assessment Summary Tables.

APPENDIX B
TABLE 2
Discussed in Section II.C.2

CATEGORY II CONTAMINANTS: UPDATED TOXICITY INFORMATION

Category II Contaminant	CAS Number	Basis of Proposed Revised HAAS (ug/m^3)		Threshold Limit Value Associated With Current HAAS (ug/m^3)	Time Factor	Uncertainty Factor
Barium, total	7440-39-3	0.5	RfC (a)	500	4.2	10
Bisphenol A. epichlorohydrin	25068-38-6	(b)		(b)		
4-Butyrolactone	96-48-0	(b)		(b)		
Chlorobenzene	108-90-7	20	RfC (a)	350,000	4.2	100 (h)
Chromium Compounds (c)		500	TLV	500	4.2	1000
Cumene	98-82-8	400	RfC	245,000	4.2	100
Cylcohexene	110-83-8	1,010,000	TLV	1015000	4.2	100
Dimethoxyethane	110-71-4	(b)		(b)		
Dimethylphthalate	131-11-3	5,000	TLV	5,000	4.2	10
Ethanolamine	141-43-5	6000	PEL	8,000	4.2	10
Fluoranthene	206-44-0	(b)		(b)		
Fluoride Compounds	7782-41-4	2,500	TLV	2,500	4.2	10
Lead Compounds	7439-92-1	(d)		NAAQS		
Manganese Compounds	7439-96-5	0.05	RfC	5,000	4.2	10
Mercury Compounds	7439-97-6	0.3	RfC	50	4.2	100
Mercury, Alkyl Compounds		10	TLV	10	4.2	100
2-Methoxyethanol	109-86-4	20	RfC	16000	4.2	10
Molybdenum Compounds	7439-98-7	5,000	TLV (e)	5,000	4.2	100
Naphthalene	91-20-3	50,000	PEL	50,000	4.2	100
Octachloronaphthalene	2234-13-1	100	TLV	100	4.2	100
Pentachloronaphthalene	1321-64-8	500	TLV	500	4.2	100
Pentachlorophenol	87-86-5	(d)		500	4.2	100
Phenanthrene	85-1-8	(b)		(b)		
Pyrene	129-0-0	(b)		(b)		
Pyridine	110-86-1	15,000	PEL	15,000	4.2	10
Selenium, total	7782-49-2	200	TLV	200	4.2	10
Silica, amorphous	61790-53-2	3,000	TLV (f)	10,000	4.2	10
Silica, crystalline	14808-60-7	(d)		50	4.2	100
Silica, fused	60676-86-0	100	TLV	100	4.2	100
Silcon tetrahydride	7803-62-5	6,600	TLV	7,000	4.2	100
Silver Compounds	7440-22-4	10	TLV	10	4.2	10
Sodium bromide	7647-15-6	(b)		(b)		
Stoddard solvent	8052-41-3	525,000	TLV	525,000	4.2	10
Styrene monomer	100-42-5	(d)		215,000	4.2	100
Tellurium Compounds	13494-80-9	100	TLV	100	4.2	10
Tetrachloronaphthalene	1335-88-2	2,000	TLV	2,000	4.2	100
Tin Compounds	7440-31-5	100	TLV (g)	2,000	4.2	100
Trichloronaphthalene	1321-65-9	5,000	TLV	5,000	4.2	100
Triethylamine	121-44-8	7	RfC	41,000	4.2	100
Triethylenetetramine	112-24-3	(b)		(b)		
Trifluorobromomethane	75-63-8	6,090,000	TLV	6,100,000	4.2	100
Trimethyl benzene	2551-13-7	123,000	TLV	123,000	4.2	100
1,2,4-Trimethyl benzene	95-63-6	125,000	REL	(b)	4.2	100
Xylene	1330-20-7	434,000	TLV	435000	4.2	100

Notes:

ACGIH - American Conference of Governmental Industrial Hygienists

CAS - Chemical Abstract System.

HAAS - Hazardous Ambient Air Standard in micrograms of compound per cubic meter of ambient air.

NAAQS - National Ambient Air Quality Standard

PEL - Permissible Exposure Limit established by federal Occupational Safety and Health Administration (OSHA), downloaded September 29, 1997.

REL - Recommended Exposure Limit established by federal National Institute for Occupational Safety and Health (NIOSH), June 1994.

RfC - Inhalation Reference Concentration. Unless otherwise noted, source is US EPA Integrated Risk Information System (IRIS) database.

TLV - Threshold Limit Value as cited in 1997 Threshold Limit Values for Chemical Substances and Physicals Agents ad Biological Indices, ACGIH.

(a) - Value presented in Table 2 of HEAST. Caveat: Derived from methodology that isn't current with interim inhalation methodology used by RfD/RfC workgroup.

(b) - HAAS based upon available toxicity information.

(c) - Excluding Cr VI.

(d) - Indicates compound now meets Category I criteria. Please see Appendix B Table 1 for updated information.

(e)- Value derived for soluble molybdenum compounds.

(f)- For inhalable (total) particulate matter containing no asbestos and lesss than 1% crystallline silica.

(g) Value represents TLV for organic compounds as SN.

(h) - Proposed revised HAAS includes extra uncertainty factor of ten because compound is identified as potential carcinogen by ACGIH or NIOSH.

APPENDIX B
TABLE 3
Discussed in Section II.C.2

CATEGORY II CONTAMINANTS: PROPOSED REVISED HAZARDOUS AMBIENT AIR STANDARDS

Category II Contaminant	CAS Number	Proposed Revised HAAS (ug/m^3)		Existing HAAS (ug/m^3) *
Barium, total	7440-39-3	.5	(a)	11.9
Bisphenol A. epichlorohydrin	25068-38-6	74	(b)	74
4-Butyrolactone	96-48-0	12	(b)	12
Chlorobenzene	108-90-7	2.0	(a,f)	833
Chromium Compounds (d)		0.12	(c)	0.12
Cumene	98-82-8	400	(a)	583
Cylcohexene	110-83-8	2,404.8	(c)	2,420
Dimethoxyethane	110-71-4	17	(b)	17
Dimethylphthalate	131-11-3	119	(c)	120
Ethanolamine	141-43-5	142.9	(c)	190
Fluoranthene	206-44-0	130	(b)	130
Fluoride Compounds	7782-41-4	59.5	(c)	59.5
Lead Compounds	7439-92-1	(e)		0.25
Manganese Compounds	7439-96-5	.05	(a)	119
Mercury Compounds	7439-97-6	.3	(a)	0.12
Mercury, Alkyl Compounds		0.024	(c)	0.024
2-Methoxyethanol	109-86-4	20	(a)	381
Molybdenum Compounds	7439-98-7	11.9	(c)	12
Naphthalene	91-20-3	119	(c)	120
Octachloronaphthalene	2234-13-1	0.24	(c)	0.24
Pentachloronaphthalene	1321-64-8	1.2	(c)	1.19
Pentachlorophenol	87-86-5	(e)		1.19
Phenanthrene	85-1-8	1.3	(b)	1.3
Pyrene	129-0-0	3.4	(b)	3.4
Pyridine	110-86-1	357.1	(c)	357
Selenium, total	7782-49-2	4.8	(c)	4.8
Silica, amorphous	61790-53-2	71.4	(c)	240
Silica, crystalline	14808-60-7	(e)		0.12
Silica, fused	60676-86-0	0.24	(c)	0.24
Silcon tetrahydride	7803-62-5	15.71	(c)	16.7
Silver Compounds	7440-22-4	0.24	(c)	0.24
Sodium bromide	7647-15-6	1,470	(b)	1,470
Stoddard solvent	8052-41-3	12,500	(c)	12,500
Styrene monomer	100-42-5	(e)		512
Tellurium Compounds	13494-80-9	2.4	(c)	2.4
Tetrachloronaphthalene	1335-88-2	4.8	(c)	4.8
Tin Compounds	7440-31-5	0.24	(c)	4.8
Trichloronaphthalene	1321-65-9	11.9	(c)	11.9
Triethylamine	121-44-8	7	(a)	98
Triethylenetetramine	112-24-3	16	(b)	16
Trifluorobromomethane	75-63-8	14,500	(c)	14,525
Trimethyl benzene	2551-13-7	293	(c)	293
1,2,4-Trimethyl benzene	95-63-6	297.6	(c)	0.15
Xylene	1330-20-7	1,033.3	(c)	1,040

Notes:

Indicates compound where proposed HAAS differs by an order of magnitude or more from existing HAAS.

ACGIH - American Conference of Governmental Industrial Hygienists

CAS - Chemical Abstract System

HAAS - Hazard Ambient Air Standard in micrograms of compound per cubic meter of ambient air.

NIOSH - National Institute for Occupational Safety and Health

* - Existing values were derived either by adjusting cccupational Threshold Limit Value or based on available toxicity information.

(a) - Value based on inhalation reference concentration.

(b) - Value derived based on available toxicity information.

(c) - Value based on adjusted occupational standard:uses most conservative of available compound specific Threshold Limit Value, Permissible Exposure Limit or Recommended Exposure Limit.

(d) - Excluding Cr VI.

(e) - Compound now meets Category I criteria. Please see Appendix B Table 1 for proposed revised HAAS.

(f) - Extra uncertainty factor of ten employed because compound's identified as potential carcinogen by either ACGIH or NIOSH.

Appendix B
Table 4
Discussed in Section II.C.2

CATEGORY III CONTAMINANTS: UPDATED TOXICITY INFORMATION

Contaminant	CAS Number	Basis of Proposed Revised HAAS (ug/m3)		Threshold Limit Value Associated With Existing HAAS (ug/m^3) (a)		Time Factor	Uncertainty Factor
Acetaldehyde	75-7-0	(b)		180,000	8	1	100
Acetic acid	64-19-7	25,000	TLV	25,000	8	1	100
Acetic anhydride	108-24-7	20,000	PEL	20,000	8	1	100
Acetone	67-64-1	1,188,000	TLV	1780000.0	8	1	10
Acetonitrile	75-05-8	50 (c)	RfC	70,000	8	1	10
Acrolein	107-02-8	(b)		250	8	1	100
2-Amino-2-methyl-1-propanol	124-68-5	(d)	TOX	(d)	24		
Ammonia	7664-41-7	100	RfC	18,000	8	1	10
Ammonium sulfamate	7773-06-0	10,000	TLV	10,000	24	4.2	100
n-Amyl acetate	628-63-7	525,000	PEL	530,000	8	1	10
s-Amyl acetate	626-38-0	650,000	PEL	665,000	8	1	10
Antimony Compounds	7440-36-0	500	TLV	500	8	1	10
1-2-Benzenedicarboxylic acid	88-99-3	(d)	TOX	(d)	24		
Benzyl alcohol	100-51-6	(d)	TOX	(d)	8		
Bisphenol A resin	80-5-7	(d)	TOX	(d)	24		
Bromodichloromethane	75-27-4	(b)		(d)	24		
2-Butoxyethanol	111-76-2	121,000	TLV	120,000	8	1	10
Butoxyethyl acetate	112-07-2	33,000	REL	(d)	8	1	100
2-(2-Butoxyethoxy)-ethanol	112-34-5	(d)	TOX	(d)	24		
n-Butyl acetate	123-86-4	710,000	PEL	710,000	8	1	100
s-Butyl acetate	105-46-4	950,000	TLV	950,000	8	1	10
t-Butyl acetate	540-88-5	950,000	TLV	950,000	8	1	10
n-Butyl alcohol	71-36-3	300,000	PEL	150,000	24	4.2	100
s-Butyl alcohol	78-92-2	305,000	REL	305,000	8	1	100
t-Butyl alcohol	75-65-0	300,000	PEL, REL	300,000	8	1	100

Appendix B
Table 4
Discussed in Section II.C.2

CATEGORY III CONTAMINANTS: UPDATED TOXICITY INFORMATION

Contaminant	CAS Number	Basis of Proposed Revised HAAS (ug/m3)		Threshold Limit Value Associated With Existing HAAS (ug/m^3) (a)		Time Factor	Uncertainty Factor
Butylamine	109-73-9	(d)	TOX	15000	8	1	100
Butyl propasol	5131-66-8	(d)	TOX	(d)	24		
p-t-Butyltoluene	98-51-1	6,100	TLV	60000	24	4.2	100
1,4-Butynediol	110-65-6	(d)	TOX	(d)	24		
Calcium oxide	1305-78-8	2,000	TLV	2,000	8	1	100
Carbon disulfide	75-15-0	700	RfC	30,000	24	4.2	10
Chlorine	7782-50-5	1,500	TLV	3,000	8	1	100
Chlorine dioxide	10049-04-4	0.2	RfC	300	8	1	100
2-Chloroethyl vinyl ether	110-75-8	(d)	TOX	(d)	24		
Cobalt Compounds	7440-48-4	20	TLV	50	24	4.2	100 (p)
Copper Compounds	7440-50-8	1,000 (e)	TLV	1,000	8	1	10
Cyanide Compounds	57-12-5	5,000	PEL	5,000	8	1	10
Cyclohexane	110-82-7	1,030,000	TLV	1,050,000	8	1	100
Cyclohexanol	108-93-0	206,000	TLV	200,000	8	1	100
Cyclohexanone	108-94-1	100,000	TLV	100,000	24	4.2	100
Cyclohexylamine	108-91-8	41,000	TLV	40,000	24	4.2	100
Decane	124-18-5	(d)	TOX	(d)	8		
Decaborane	17702-41-9	250	TLV	300	24	4.2	100
Diacetone alcohol	123-42-2	238,000	TLV	240,000	24	4.2	100
Dibenzoyl peroxide	95-36-0	(d)	TOX	5,000	8	1	100
Dibromochloromethane	124-48-1	(b)		(d)	24		
Dibutyl phthalate	84-74-2	5,000	TLV	5,000	8	1	10
o-Dichlorobenzene	95-50-1	200 (c)	RfC	300,000	8	1	100
Dichlorodifluoromethane	75-71-8	200 (c)	RfC	4,950,000	24	4.2	10
1,1-Dichloroethane	75-34-3	(b)		810,000	24	4.2	10
1,2-Dichloroethylene	540-59-0	790,000	PEL	790,000	8	1	10
s-Dichlorotetrafluoroethane	76-14-2	6,990,000	TLV	7,000,000	24	4.2	10
Diethanolamine	111-42-2	2,000	TLV	13,000	8	1	100
Diethylamine	109-89-7	15,000	TLV	30,000	24	4.2	100
Diethylamino ethanol	100-37-8	9,600	TLV	47,844.9	8	1	100
Diethylene glycol ethyl ether	111-90-0	(d)	TOX	(d)	8		
Dimethyl ammonium chloride	506-59-2	(d)	TOX	(d)	24		
Dimethoxymethane	109-87-5	3,100,000	PEL	3,100,000	24	4.2	100

Appendix B
Table 4
Discussed in Section II.C.2

CATEGORY III CONTAMINANTS: UPDATED TOXICITY INFORMATION

Contaminant	CAS Number	Basis of Proposed Revised HAAS (ug/m3)		Threshold Limit Value Associated With Existing HAAS (ug/m^3) (a)		Time Factor	Uncertainty Factor
Dimethylamine	124-40-3	9,200	TLV	18,000	24	4.2	100
n,n-Dimethyl dodecylamine	112-18-5	(d)	TOX	(d)	24		
Dimethylethanolamine	108-01-0	(d)	TOX	(d)	8		
2,6-Dimethyl-4-heptanone	108-83-8	145,000	TLV	145,000	24	4.2	100
n,n-Dimethyl octadecylamine	124-28-7	(d)	TOX	(d)	24		
1,3-Dioxolane	646-06-0	(d)	TOX	(d)	24		
Diphenylmethane diisocyanate	101-68-8	51	TLV	51	24	4.2	10
Dipropylene glycol	110-98-5	(d)	TOX	(d)	8		
Dipropylene glycol methyl ether	34590-94-8	606,000	TLV	600,000	8	1	100
Dodecylguanidine hydrochloride	13590-97-1	(d)	TOX	(d)	8		
Doxorubicin	23214-92-8	(d)	TOX	(d)	24		
1,2-Epoxy butane	106-88-7	20	RfC	(d)	8		
2-Ethoxyethanol	110-80-5	200	RfC	19,000	24	4.2	100
2-Ethoxyethyl acetate	111-15-9	27,000	TLV	27,000	24	4.2	100
Ethyl acetate	141-78-6	1,400,000	PEL	1,400,000	8	1	10
Ethyl alcohol	64-17-5	1,880,000	TLV	1,880,000	24	4.2	10
Ethylamine	75-04-7	9,200	TLV	18,000	24	4.2	100
Ethyl benzene	100-41-4	1,000	RfC	435,000	8	1	10
Ethyl bromide	74-96-4	22,000	TLV	890,000	8	1	100
Ethyl butyl ketone	106-35-4	230,000	PEL	230,000	8	1	100
Ethylene diamine	107-15-3	25,000	TLV	25,000	24	4.2	100
Ethyl-3-ethoxy propionate	763-69-9	(d)	TOX	(d)	24		
Ethylene glycol	107-21-1	(d)	TOX	125,000	8	1	100
Ethyl ether	60-29-7	1,200,000	PEL	1,200,000	8	1	10
2-Ethyl hexanol	104-76-7	(d)	TOX	(d)	24		
2-Ethylhexyl ester acrylic acid	103-11-7	(d)	TOX	(d)	8		
Ethyl mercaptan	75-8-1	1,300	TLV	1,268	8	1	10
Fluorine	7782-41-4	200	PEL	2,000	8	1	10
Formic acid	64-18-6	9,000	PEL	9,000	8	1	100
Furfural	98-1-1	(b)		8,000	8	1	100
Glutaraldehyde	111-30-8	(d)	TOX	820,000	8	1	100
Glyoxal	107-22-2	(d)	TOX	(d)	24		

Appendix B
Table 4
Discussed in Section II.C.2

CATEGORY III CONTAMINANTS: UPDATED TOXICITY INFORMATION

Contaminant	CAS Number	Basis of Proposed Revised HAAS (ug/m3)		Threshold Limit Value Associated With Existing HAAS (ug/m^3) (a)		Time Factor	Uncertainty Factor
Heptane	142-82-5	1,640,000	TLV	1,600,000	8	1	100
Hexamethylene-1-6-diisocyanate	822-6-0	0.01	RfC	34	24	4.2	100
n-Hexane	110-54-3	200	RfC	180,000	24	4.2	10
Hydrogen chloride	7647-1-0	20	RfC	7,000	24	4.2	100
Hydrogen fluoride	7664-39-3	2,450.2	PEL	2,500	24	4.2	10
Hydrogen peroxide	7722-84-1	1,400	TLV	1,500	8	1	100 (p)
Hydrogen sulfide	7783-6-4	1	RfC	14,000	24	4.2	100
Hydroquinone	123-31-9	2,000	TLV	2,000	24	4.2	100 (p)
Iodine	7553-56-2	(d)	TOX	1,000	8	1	100
Iron Compounds	1309-37-1	1,000 (f)	TLV	1,000	24	4.2	10
Isoamyl acetate	123-92-2	525,000	PEL	525,000	8	1	100
Isoamyl alcohol	123-51-3	360,000	PEL	360,000	8	1	100
Isobutyl acetate	110-19-0	700,000	PEL	700,000	8	1	100
Isobutyl alcohol	78-83-1	152,000	TLV	150,000	8	1	100
Isobutyl ester isobutyric acid	97-85-8	(d)	TOX	(d)	24		
Isophorone	78-59-1	(b)		140,000	8	1	100
Isopropyl acetate	108-21-4	950,000	PEL	950,000	8	1	100
Isopropyl alcohol	67-63-0	980,000	PEL	980,000	8	1	10
Isopropylamine	75-31-0	12,000	TLV	12,000	8	1	100
Isopropyl ether	108-20-3	1,040,000	TLV	1,050,000	8	1	100
Kerosene	8008-20-6	100,000	REL	(d)	24	4.2	100
Methoxyethoxyethanol	111-77-3	(d)	TOX	(d)	24		
o-Methoxyphenol	90-5-1	(d)	TOX	(d)	24		
1-Methoxy-2-propanol	107-98-2	(d)	TOX	360,000	8	1	100
Methyl acetate	79-20-9	606,000	TLV	610,000	24	4.2	100
Methyl alcohol	67-56-1	260,000	PEL	260,000	24	4.2	10
Methylamine	74-89-5	6,400	TLV	12,000	8	1	100
p-Methylaminophenol sulfate	55-55-0	(d)	TOX	(d)	24		
Methyl amyl ketone	110-43-0	233,000	TLV	233,000	8	1	100
Methylcyclohexanol	25639-42-3	234,000	TLV	235,000	24	4.2	100
Methyl ester salicylic acid		(d)	TOX	(d)	24		
Methyl ethyl ketone	78-93-3	1,000	RfC	590,000	8	1	100
Methyl ethyl ketone peroxide	1338-23-4	(d)	TOX	1,500	8	1	100

Appendix B
Table 4
Discussed in Section II.C.2

CATEGORY III CONTAMINANTS: UPDATED TOXICITY INFORMATION

Contaminant	CAS Number	Basis of Proposed Revised HAAS (ug/m3)		Threshold Limit Value Associated With Existing HAAS (ug/m^3) (a)		Time Factor	Uncertainty Factor
Methyl isoamyl ketone	110-12-3	234,000	TLV	234,000	8	1	100
Methyl isobutyl ketone	108-10-1	80 (c)	RfC	205,000	24	4.2	100
Methyl methacroylate	80-62-6	410,000	TLV	410,000	8	1	10
3-Methyl-2-oxazolidone	19836-78-3	(d)	TOX	(d)	8		
1-Methyl-2-pyrrolidone	872-50-4	(d)	TOX	403,200	24	4.2	100
Mineral Spirits	8030-30-6	(d)	TOX	(g)	24		
Morpholine	110-91-8	70,000	PEL	71,118.4	8	1	100
Nitric acid	7697-37-2	5,000	PEL	5,000	8	1	10
Nitric oxide	10102-43-9	30,000	PEL	30,000	24	4.2	100
Nitrobenzene	98-995-3	(b)		5,000	24	4.2	10
p-Nitrochlorobenzene	100-0-5	640	TLV	3,000	24	4.2	10 (p)
Nitroethane	79-24-3	307,000	TLV	310,000	24	4.2	100
Nitromethane	75-52-5	50,000	TLV	250,000	8	1	100
1-Nitropropane	108-3-2	90,000	PEL	90,000	8	1	100
1-Nitrotoluene	78-72-5	11,000	TLV	11,000	24	4.2	10
Oxalic acid	144-62-7	1,000	TLV	1,000	8	1	10
1-Pentanol	71-41-0	(d)	TOX	(d)	24		
2-Pentanone	107-87-9	700,000	PEL	705,000	24	4.2	100
Perchloric acid	7601-90-3	(d)	TOX	(d)	24		
Phenol	108-95-2	19,000	TLV	19,000	8	1	10
Phenoxyethanol	122-99-6	(d)	TOX	(d)	24		
Phenyl ether	101-84-8	7,000 (h)	TLV	7,000	8	1	10
1-Phenyl-3-pyrazolidone	92-43-3	(d)	TOX	(d)	24		
Phosgene	75-44-5	400	TLV	400	8	1	10
Phosphine	7803-51-2	0.3	RfC	400	8	1	10
Phosphoric acid	7664-38-2	10	RfC	1,000	8	1	10
Phosphorus pentachloride	10026-13-8	850	TLV	1,000	8	1	100
Phosporous pentasulfide	1314-80-3	1,000	TLV	1,000	8	1	100
Phosphorous trichloride	7719-12-2	1,100	TLV	1,500	8	1	100
Phthalic anhydride	85-44-9	120 (i)	RfC	6,000	8	1	10
Picric acid	88-89-1	100	TLV	100	24	4.2	100
Platinum Compounds	7440-06-4	2 (j)	TLV	2	24	4.2	100

Appendix B
Table 4
Discussed in Section II.C.2

CATEGORY III CONTAMINANTS: UPDATED TOXICITY INFORMATION

Contaminant	CAS Number	Basis of Proposed Revised HAAS (ug/m3)		Threshold Limit Value Associated With Existing HAAS (ug/m^3) (a)		Time Factor	Uncertainty Factor
Potassium hydroxide	1310-58-2	2,000	REL	2,000	8	1	100
1,2-Propanediol	57-55-6	(d)	TOX	(d)	24		
2-Propoyxethanol	2807-30-9	(d)	TOX	(d)	8		
Propoxypropanol	1569-1-3	(d)	TOX	(d)	24		
n-Propyl acetate	109-60-4	835,000	TLV	835,000	8 (o)	1	100
n-Propyl alcohol	71-23-8	492,000	TLV	500,000	8	1	10
1,2-Propylene carbonate	108-32-7	(d)	TOX	(d)	8		
Sebacic acid	11-20-6	(d)	TOX	(d)	8		
Sodium hydroxide	1310-73-2	50	PEL	2,000	8	1	100
Sodium tripolyphosphate	7758-29-4	(d)	TOX	(d)	24		
Sulfuric acid	7664-93-9	70 (k)	RfC	1,000	24	4.2	10
Sulfur monochloride	10025-67-9	6,000	PEL	6,000	8	1	10
1,1,2,2-Tetrachloro-1,2-difluoroethane	76-12-0	4,170,000	TLV	4,170,000	24	4.2	100
Tetrahydrofuran	109-99-9	590,000	TLV	590,000	24	4.2	10
Texanol	25265-77-4	(d)	TOX	(d)	24		
Titanium dioxide	13463-67-7	10,000	TLV	10,000	24	4.2	10 (p)
Toluene	108-88-3	400	RfC	375,000	24	4.2	10
Toluene-2,4-diisocyanate	584-84-9	0.07 (l)	RfC	40	24	4.2	100 (p)
p-Toluenesulfonic acid	88-20-0	(d)	TOX	(d)	24		
1,1,1-Trichloroethane	71-55-6	1,900,000	PEL	1,900,000	8	1	10
Trichlorofluoromethane	75-69-4	700 (c)	RfC	5,607,346.9	24	4.2	10
1,2,3-Trichloropropane	96-18-4	(b)		60,000	24	4.2	1000
1,1,2-Trichloro-1,2,2-trifluoroethane	76-13-1	30000 (i)	RfC	7,600,000	24	4.2	10
2,4,6-Tri(dimethyl aminomethyl)phenol	90-72-2	(d)	TOX	(d)	24		
Triethanolamine	102-71-6	5,000	TLV	(d)	24	4.2	100
Triethyl ester phosphoric acid	78-40-0	(d)	TOX	(d)	24		
Triethyl orthoformate	122-51-0	(d)	TOX	(d)	24		
s,s,s-Trimethyl ester phosphorotrithioic acid	150-50-5	(d)	TOX	(d)	24		
Triorthocresyl phosphate	78-30-8	100	TLV	100	24	4.2	100

Appendix B

Table 4

Discussed in Section II.C.2

CATEGORY III CONTAMINANTS: UPDATED TOXICITY INFORMATION

Contaminant	CAS Number	Basis of Proposed Revised HAAS (ug/m3)		Threshold Limit Value Associated With Existing HAAS (ug/m^3) (a)		Time Factor	Uncertainty Factor
Turpentine	8006-64-2	556,000	TLV	556,000	24	4.2	100
4-Undecanol,7-ethyl-2-methyl-hydrogen sulfate	139-88-8	(d)	TOX	(d)	24		
Vanadium Compounds		50 (m)	TLV	50	24	4.2	10
Vinyl acetate	108-5-4	(b)		35,138.8	8	1	100
Vinyl toluene	25013-15-4	242,000	TLV	240,000	24	4.2	10
VM & P naptha	8032-32-4	1,370,000	TLV	1,350,000	24	4.2	100 (p)
Zinc chloride	7646-85-7	1,000 (n)	TLV	1,000	24	4.2	100
Zinc Compounds	7440-66-6	(d)	TOX	5,000	24	4.2	100

Notes:
ACGIH - American Conference of Governmental Industrial Hygienists
CAS - Chemical Abstract System
HAAS - Hazardous Ambient Air Standard in micrograms compound per cubic meter of ambient air
HEAST - Health Effects Assessment Summary Tables. US EPA. July 1997.
NIOSH - National Institute for Occupational Safety and Health
PEL - Permissible Exposure Limit established by federal Occupational Safety and Health Administration (OSHA), downloaded September 29, 1997.
REL - Recommended Exposure Limit established by federal National Institute for Occupational Safety and Health (NIOSH), June 1994.
RfC - Inhalation Reference Concentration
TLV - Threshold Limit Value as cited in 1997 Threshold Limit Values for Chemical Substances and Physicals Agents ad Biological Indices, ACGIH.
TOX - Derived based upon available toxicity information.
(a) - Averaging period
(b) - Compound now meets Category I criteria. Please see Appendix B Table 1 for proposed revised HAAS.
(c) - Value presented in Table 2 of HEAST. Caveat: Derived from methodology that isn't current with the interim inhalation methodology used by the RfD/RfC workgroup.
(d) - HAAS derived based on available toxicity information.
(e) - ACGIH cites 1,000 ug/cubic meter for dusts and mists.
(f) - ACGIH cites 1,000 ug/cubic meter for soluble iron salts.
(g) - Due to similarities, values for VM & P naptha used as surrogates.
(h) - ACGIH value cited for phenyl ether vapor.
(i) - Value cited in Table 1 of HEAST July 1997.
(j) - ACGIH cites 2 ug/cubic meter for soluble salts.
(k) - Reported effects occured at portal of entry therefore value represents an acceptable air concentration.
(l) - Value derived for a mixture of 2,4 and 2,6 toluene diisocyanate.
(m) - ACGIH value for vanadium pentoxide respirable fume or dust.
(n) - ACGIH value cited for zinc chloride fumes.
(o) - Incorrect averaging period of 24 hours currently listed in Regulations.
(p) - Proposed revised HAAS includes extra uncertainty factor of ten because compound is identified as potential carcinogen by ACGIH or NIOSH.
C3TOXUP.WQ1 20-Feb-98

APPENDIX B
TABLE 5
Discussed in Section II.C.2

CATEGORY III CONTAMINANTS: PROPOSED REVISED HAZARDOUS AMBIENT AIR STANDARDS

Contaminant	CAS Number	Proposed Revised HAAS (ug/m3)		Existing HAAS * (ug/m^3)	(a)
Acetaldehyde	75-7-0	(b)		1,800	8
Acetic acid	64-19-7	250	(c)	250	8
Acetic anhydride	108-24-7	200	(c)	200	8
Acetone	67-64-1	118,800	(c)	178,000	8
Acetonitrile	75-05-8	50	(d)	7,000	8
Acrolein	107-02-8	(b)		2.5	8
2-Amino-2-methyl-1-propanol	124-68-5	65	(e)	65	24
Ammonia	7664-41-7	100	(d)	1,800	8
Ammonium sulfamate	7773-06-0	23.8	(c)	23.8	24
n-Amyl acetate	628-63-7	52,500	(c)	53,000	8
s-Amyl acetate	626-38-0	65,000	(c)	66,500	8
Antimony Compounds	7440-36-0	50	(c)	50	8
1-2-Benzenedicarboxylic acid	88-99-3	357	(e)	357	24
Benzyl alcohol	100-51-6	10	(e)	10	8
Bisphenol A resin	80-5-7	210	(e)	210	24
Bromodichloromethane	75-27-4	(b)		42	24
2-Butoxyethanol	111-76-2	12,100	(c)	12,000	8
Butoxyethyl acetate	112-07-2	330	(c)	270	8
2-(2-Butoxyethoxy)-ethanol	112-34-5	300	(e)	300	24
n-Butyl acetate	123-86-4	7,100	(c)	7,100	8
s-Butyl acetate	105-46-4	95,000	(c)	95,000	8
t-Butyl acetate	540-88-5	95,000	(c)	95,000	8
n-Butyl alcohol	71-36-3	714.3	(c)	360	24
s-Butyl alcohol	78-92-2	3,050	(c)	3,050	8
t-Butyl alcohol	75-65-0	3,000	(c)	3,000	8

APPENDIX B
TABLE 5
Discussed in Section II.C.2

CATEGORY III CONTAMINANTS: PROPOSED REVISED HAZARDOUS AMBIENT AIR STANDARDS

Contaminant	CAS Number	Proposed Revised HAAS (ug/m3)		Existing HAAS * (ug/m^3)	(a)
Butylamine	109-73-9	150	(e)	150	8
Butyl propasol	5131-66-8	142	(e)	142	24
p-t-Butyltoluene	98-51-1	14.5	(c)	143	24
1,4-Butynediol	110-65-6	0.1	(e)	0.10	24
Calcium oxide	1305-78-8	20	(c)	20	8
Carbon disulfide	75-15-0	700	(d)	714	24
Chlorine	7782-50-5	15	(c)	30	8
Chlorine dioxide	10049-04-4	0.2	(d)	3	8
2-Chloroethyl vinyl ether	110-75-8	1	(e)	1	24
Cobalt Compounds	7440-48-4	0.005	(c,g)	0.12	24
Copper Compounds	7440-50-8	100	(c)	100	8
Cyanide Compounds	57-12-5	500	(c)	500	8
Cyclohexane	110-82-7	10,300	(c)	10,500	8
Cyclohexanol	108-93-0	2,060	(c)	2,000	8
Cyclohexanone	108-94-1	238.1	(c)	240	24
Cyclohexylamine	108-91-8	97.6	(c)	95	24
Decane	124-18-5	1,300	(e)	1,300	8
Decaborane	17702-41-9	0.60	(c)	0.71	24
Diacetone alcohol	123-42-2	566.7	(c)	565	24
Dibenzoyl peroxide	95-36-0	50	(e)	50	8
Dibromochloromethane	124-48-1	(b)		39	24
Dibutyl phthalate	84-74-2	500	(c)	500	8
o-Dichlorobenzene	95-50-1	200	(d)	3,000	8
Dichlorodifluoromethane	75-71-8	200	(d)	118	24
1,1-Dichloroethane	75-34-3	(b)		19,300	24
1,2-Dichloroethylene	540-59-0	55,400	(f)	79,000	8
s-Dichlorotetrafluoroethane	76-14-2	166,429	(c)	167,000	24
Diethanolamine	111-42-2	20	(c)	130	8
Diethylamine	109-89-7	35.7	(c)	71.4	24
Diethylamino ethanol	100-37-8	96	(c)	480	8
Diethylene glycol ethyl ether	111-90-0	297	(e)	297	8
Dimethyl ammonium chloride	506-59-2	49	(e)	49	24
Dimethoxymethane	109-87-5	7,380	(c)	7,380	24

APPENDIX B
TABLE 5
Discussed in Section II.C.2

CATEGORY III CONTAMINANTS: PROPOSED REVISED HAZARDOUS AMBIENT AIR STANDARDS

Contaminant	CAS Number	Proposed Revised HAAS (ug/m3)		Existing HAAS * (ug/m^3)	(a)
Dimethylamine	124-40-3	21.9	(c)	42.9	24
n,n-Dimethyl dodecylamine	112-18-5	63	(e)	63	24
Dimethylethanolamine	108-01-0	27	(e)	27	8
2,6-Dimethyl-4-heptanone	108-83-8	345.2	(c)	345	24
n,n-Dimethyl octadecylamine	124-28-7	5.5	(e)	5.5	24
1,3-Dioxolane	646-06-0	92	(e)	92	24
Diphenylmethane diisocyanate	101-68-8	1.2	(c)	0.48	24
Dipropylene glycol	110-98-5	1,680	(e)	1,680	8
Dipropylene glycol methyl ether	34590-94-8	6,060	(c)	6,000	8
Dodecylguanidine hydrochloride	13590-97-1	0.6	(e)	0.6	8
Doxorubicin	23214-92-8	115	(e)	115	24
1,2-Epoxy butane	106-88-7	20	(d)	11	8
2-Ethoxyethanol	110-80-5	200	(d)	45.2	24
2-Ethoxyethyl acetate	111-15-9	64.3	(c)	64.3	24
Ethyl acetate	141-78-6	140,000	(c)	140,000	8
Ethyl alcohol	64-17-5	44,770	(c)	44,770	24
Ethylamine	75-04-7	21.9	(c)	42.9	24
Ethyl benzene	100-41-4	1,000	(d)	43,500	8
Ethyl bromide	74-96-4	220	(c)	8,900	8
Ethyl butyl ketone	106-35-4	2,300	(c)	2,300	8
Ethylene diamine	107-15-3	59.5	(c)	60	24
Ethyl-3-ethoxy propionate	763-69-9	230	(e)	230	24
Ethylene glycol	107-21-1	1,270	(e)	1,270	8
Ethyl ether	60-29-7	120,000	(c)	120,000	8
2-Ethyl hexanol	104-76-7	130	(e)	130	24
2-Ethylhexyl ester acrylic acid	103-11-7	29	(e)	29	8
Ethyl mercaptan	75-8-1	130	(c)	125	8
Fluorine	7782-41-4	20	(c)	200	8
Formic acid	64-18-6	90	(c)	90	8
Furfural	98-1-1	(b)		80	8
Glutaraldehyde	111-30-8	8,200	(e)	8,200	8
Glyoxal	107-22-2	130	(e)	130	24

APPENDIX B
TABLE 5
Discussed in Section II.C.2

CATEGORY III CONTAMINANTS: PROPOSED REVISED HAZARDOUS AMBIENT AIR STANDARDS

Contaminant	CAS Number	Proposed Revised HAAS (ug/m3)		Existing HAAS * (ug/m^3)	(a)
Heptane	142-82-5	16,400	(c)	16,000	8
Hexamethylene-1-6-diisocyanate	822-6-0	0.01	(d)	0.082	24
n-Hexane	110-54-3	200	(d)	4,290	24
Hydrogen chloride	7647-1-0	20	(d)	16.7	24
Hydrogen fluoride	7664-39-3	58.3	(c)	59.5	24
Hydrogen peroxide	7722-84-1	1.4	(c,g)	15	8
Hydrogen sulfide	7783-6-4	1.0	(d)	33.3	24
Hydroquinone	123-31-9	0.48	(c,g)	4.8	24
Iodine	7553-56-2	10	(e)	100 (i)	8
Iron Compounds	1309-37-1	24	(c)	24	24
Isoamyl acetate	123-92-2	5,250	(c)	5,250	8
Isoamyl alcohol	123-51-3	3,600	(c)	3,600	8
Isobutyl acetate	110-19-0	7,000	(c)	7,000	8
Isobutyl alcohol	78-83-1	1,520	(c)	1,500	8
Isobutyl ester isobutyric acid	97-85-8	580,780	(e)	580,780	24
Isophorone	78-59-1	(b)		1,400	8
Isopropyl acetate	108-21-4	9,500	(c)	9,500	8
Isopropyl alcohol	67-63-0	98,000	(c)	98,000	8
Isopropylamine	75-31-0	120	(c)	120	8
Isopropyl ether	108-20-3	10,400	(c)	10,500	8
Kerosene	8008-20-6	238	(c)	51,000	24
Methoxyethoxyethanol	111-77-3	595	(e)	595	24
o-Methoxyphenol	90-5-1	47	(e)	47	24
1-Methoxy-2-propanol	107-98-2	3,600	(e)	3,600	8
Methyl acetate	79-20-9	1,443	(c)	1,450	24
Methyl alcohol	67-56-1	6,190	(c)	6,190	24
Methylamine	74-89-5	64	(c)	120	8
p-Methylaminophenol sulfate	55-55-0	5,100	(e)	5,100	24
Methyl amyl ketone	110-43-0	2,330	(c)	2,330	8
Methylcyclohexanol	25639-42-3	557.1	(c)	560	24
Methyl ester salicylic acid		180	(e)	180	24
Methyl ethyl ketone	78-93-3	1,000	(d)	5,900	8
Methyl ethyl ketone peroxide	1338-23-4	15	(e)	15	8

APPENDIX B
TABLE 5
Discussed in Section II.C.2

CATEGORY III CONTAMINANTS: PROPOSED REVISED HAZARDOUS AMBIENT AIR STANDARDS

Contaminant	CAS Number	Proposed Revised HAAS (ug/m3)		Existing HAAS * (ug/m^3)	(a)
Methyl isoamyl ketone	110-12-3	2,340	(c)	2,400	8
Methyl isobutyl ketone	108-10-1	80	(d)	490	24
Methyl methacroylate	80-62-6	41,000	(c)	41,000	8
3-Methyl-2-oxazolidone	19836-78-3	57	(e)	57	8
1-Methyl-2-pyrrolidone	872-50-4	960	(e)	960	24
Mineral Spirits	8030-30-6	3,210	(e)	3,210	24
Morpholine	110-91-8	700	(c)	700	8
Nitric acid	7697-37-2	500	(c)	500	8
Nitric oxide	10102-43-9	71.4	(c)	71.4	24
Nitrobenzene	98-995-3	(b)		119	24
p-Nitrochlorobenzene	100-0-5	1.5	(c,g)	71.4	24
Nitroethane	79-24-3	731	(c)	738	24
Nitromethane	75-52-5	500	(c)	2,500	8
1-Nitropropane	108-3-2	900	(c)	900	8
1-Nitrotoluene	78-72-5	261.9	(c)	262	24
Oxalic acid	144-62-7	100	(c)	100	8
1-Pentanol	71-41-0	120	(e)	120	24
2-Pentanone	107-87-9	1,667	(c)	1,680	24
Perchloric acid	7601-90-3	50	(e)	50	24
Phenol	108-95-2	1,900	(c)	1,900	8
Phenoxyethanol	122-99-6	81	(e)	81	24
Phenyl ether	101-84-8	700	(c)	700	8
1-Phenyl-3-pyrazolidone	92-43-3	13	(e)	13	24
Phosgene	75-44-5	40	(c)	40	8
Phosphine	7803-51-2	0.3	(d)	40	8
Phosphoric acid	7664-38-2	10	(d)	100	8
Phosphorus pentachloride	10026-13-8	8.5	(c)	10	8
Phosporous pentasulfide	1314-80-3	10	(c)	10	8
Phosphorous trichloride	7719-12-2	11	(c)	15	8
Phthalic anhydride	85-44-9	120	(d)	600	8
Picric acid	88-89-1	0.24	(c)	0.24	24
Platinum Compounds	7440-06-4	0.005	(c)	0.005	24

APPENDIX B
TABLE 5
Discussed in Section II.C.2

CATEGORY III CONTAMINANTS: PROPOSED REVISED HAZARDOUS AMBIENT AIR STANDARDS

Contaminant	CAS Number	Proposed Revised HAAS (ug/m3)		Existing HAAS * (ug/m^3)	(a)
Potassium hydroxide	1310-58-2	20	(c)	20	8
1,2-Propanediol	57-55-6	1,300	(e)	1,300	24
2-Propoyxethanol	2807-30-9	4.3	(e)	4.3	8
Propoxypropanol	1569-1-3	210	(e)	210	24
n-Propyl acetate	109-60-4	8,350	(c)	8,350	24
n-Propyl alcohol	71-23-8	49,200	(c)	50,000	8
1,2-Propylene carbonate	108-32-7	1,260	(e)	1,260	8
Sebacic acid	11-20-6	268	(e)	268	8
Sodium hydroxide	1310-73-2	0.5	(c)	20	8
Sodium tripolyphosphate	7758-29-4	84	(e)	84	24
Sulfuric acid	7664-93-9	70	(d,h)	23.8	24
Sulfur monochloride	10025-67-9	600	(c)	600	8
1,1,2,2-Tetrachloro-1,2-difluoroethane	76-12-0	9,929	(c)	9,930	24
Tetrahydrofuran	109-99-9	14,048	(c)	14,050	24
Texanol	25265-77-4	207	(e)	207	24
Titanium dioxide	13463-67-7	2.38	(c,g)	240	24
Toluene	108-88-3	400	(d)	8,930	24
Toluene-2,4-diisocyanate	584-84-9	0.007	(d,g)	0.1	24
p-Toluenesulfonic acid	88-20-0	113	(e)	113	24
1,1,1-Trichloroethane	71-55-6	190,000	(c)	190,000	8
Trichlorofluoromethane	75-69-4	700	(d)	133,500	24
1,2,3-Trichloropropane	96-18-4	(b)		14.3	24
1,1,2-Trichloro-1,2,2-trifluoroethane	76-13-1	30,000	(d)	181,000	24
2,4,6-Tri(dimethyl aminomethyl)phenol	90-72-2	78	(e)	78	24
Triethanolamine	102-71-6	11.9	(c)	400	24
Triethyl ester phosphoric acid	78-40-0	970	(e)	970	24
Triethyl orthoformate	122-51-0	190	(e)	190	24
s,s,s-Trimethyl ester phosphorotrithioic acid	150-50-5	78	(e)	78	24
Triorthocresyl phosphate	78-30-8	0.24	(c)	0.24	24

APPENDIX B
TABLE 5
Discussed in Section II.C.2

CATEGORY III CONTAMINANTS: PROPOSED REVISED HAZARDOUS AMBIENT AIR STANDARDS

Contaminant	CAS Number	Proposed Revised HAAS (ug/m3)		Existing HAAS * (ug/m^3)	(a)
Turpentine	8006-64-2	1,324	(c)	1,300	24
4-Undecanol,7-ethyl-2-methyl-hydrogen sulfate	139-88-8	13	(e)	13	24
Vanadium Compounds		1.2	(c)	1.2	24
Vinyl acetate	108-5-4	(b)		350	8
Vinyl toluene	25013-15-4	5,762	(c)	5,710	24
VM & P naptha	8032-32-4	326	(c,g)	3,210	24
Zinc chloride	7646-85-7	2.4	(c)	2.4	24
Zinc Compounds	7440-66-6	12	(e)	12	24

Notes:
Indicates compound where proposed HAAS differs by an order of magnitude or more from existing HAAS.
ACGIH - American Conference of Governmental Industrial Hygienists
CAS - Chemical Abstract System
HAAS - Hazardous Ambient Air Standard in micrograms compound per cubic meter of ambient air
HEAST - Health Effects Assessment Summary Tables. US EPA. July 1997.
NIOSH - National Institute for Occupational Safety and Health
* - Existing values were derived either by adjusting cccupational Threshold Limit Value or based on available toxicity information.
(a) - Averaging period
(b) - Compound now meets Category I criteria. Please see Appendix B Table 1 for proposed revised HAAS.
(c) - Value based on adjusted occupational standard:uses most conservative of available compound specific Threshold Limit Value, Permissible Exposure Limit or Recommended Exposure Limit.
(d) - Value based on inhalation reference concentration.
(e) - Value derived based on available toxicity information.
(f) - Eight hour Acute Exposure Guideline Level of 55,400 ug/m^3 is used as proposed HAAS to proect some sensitive individuals that may experience dizziness if exposed to existing standard for a prolonged period of time.
(g) - Proposed revised HAAS includes extra uncertainty factor of ten because compound is identified as potential carcinogen by ACGIH or NIOSH.
(h) - Reported effects occured at portal of entry therefore value represents an acceptable air concentration (HEAST, July, 1997).
(i) - Value improperly calculated. Correct value should be 10.

Appendix C

Air Monitoring Data

Tables

APPENDIX C

TABLE 1

VOC and Carbonyl Compounds Monitored - See Section III.A.1

compound name	CAS #	annual average 1993-95 max.	state standard	averaging time	%Non-Detect	ESE MDL 7/93 - 3/95	Radian MDL 8/95 - 12/95
acetaldehyde	75-07-0	4.65	1800	8hr	0	c	c
acetone	67-64-1	6.26	178000	8hr	0.53	c	c
acetylene	74-86-2	4.94			4.65		0.13
acrolein	107-02-8	0.11	2.5	8hr	88.89	c	c
benzaldehyde	100-52-7	0.32			61.38	c	c
benzene	71-43-2	4.05	0.12	annual average	11.52	0.10	0.77
benzyl chloride	100-44-7	*			100	1.50	
bromochloromethane	74-97-5	*			100		0.37
bromodichloromethane	75-27-4	*	42	24hr	100	0.20	0.60
bromoform	75-25-2	*	0.01	annual average	100	1.45	0.83
1,3-butadiene	106-99-0	0.95	0.035	annual average	41.01	0.15	0.33
butyr/isobutyraldehyde	123-72-8	0.65			43.39	c	c
carbon tetrachloride	56-23-5	0.72	0.067	annual average	14.75	0.19	0.44
3-chloro-1-propene	107-05-1	*			98.85	0.09	
chlorobenzene	108-90-7	*	833	annual average	99.54	0.14	0.28
chlorodifluoromethane	75-45-6	8.02			2.87	0.32	
chloroethane	75-00-3	0.17			93.09	0.11	0.47
chloroform	67-66-3	0.22	0.043	annual average	89.86	0.24	0.29
chloroprene	126-99-8	*	0.01	annual average	100	0.18	0.18
crotonaldehyde	123-73-9	0.43			45.5	c	c
dibromochloromethane	124-48-1	*	39	24hr	100	0.43	0.43
1,2-dibromoethane	106-93-4	*			100	0.69	
1,2-dichlorobenzene	95-50-1	*	3000	8hr	100	0.12	0.48
1,3-dichlorobenzene	541-73-1	*			100	0.24	0.42
1,4-dichlorobenzene	106-46-7	*			99.54	0.24	0.36
dichlorodifluoromethane	75-71-8	5.60	118	24hr	1.15	0.35	
1,1-dichloroethane	75-34-3	*	19300	24hr	100	0.24	0.24
c-1,2-dichloroethylene	156-59-2	*			100	0.24	
t-1,2-dichloroethylene	156-60-5	*			100	0.36	0.89
c-1,3-dichloropropene	10061-01-5	*			100	0.27	0.23
t-1,3-dichloropropene	10061-02-6	*			100	0.32	0.36
2,5-dimethylbenzaldehyde	5779-94-2	0.19			91.53	c	c
ethylbenzene	100-41-4	1.81	43500	8hr	26.73	0.35	0.35
ethylene dichloride	107-06-2	*	0.038	annual average	100	0.24	1.05
formaldehyde	50-00-0	10.16	0.08	annual average	0	c	c
freon 113	76-13-1	67.16	181000	24hr	15.52	1.09	
freon 114	76-14-2	*			100	0.63	
heptane	142-82-5	1.52	16000	8hr	20.69	0.16	
hexachlorobutadiene	87-68-3	*	0.045	annual average	100	0.53	

APPENDIX C

TABLE 1

VOC and Carbonyl Compounds Monitored - See Section III.A.1

compound name	CAS #	annual average	state standard	averaging time	%Non-Detect	ESE MDL	Radian MDL
		1993-95 max.				7/93 - 3/95	8/95 - 12/95
hexanaldehyde	66-25-1	0.27			76.72	c	c
hexane	110-54-3	4.46	4290	24hr	12.64	0.18	
isovaleraldehyde	590-86-3	0.17			94.71	c	c
methyl bromide	74-83-9	*	0.01	annual average	98.62	0.27	0.70
methyl chloride	74-87-3	1.39	0.01	annual average	7.37	0.21	0.81
methylene chloride	75-09-2	8.41	2	annual average	17.51	0.24	0.56
a-methyl styrene	98-83-9	0.14			88.51	0.04	
octane	111-65-9	0.44			64.98	0.28	0.23
pentane	109-66-0	5.72			6.91	0.06	
propionaldehyde	132-38-6	0.87			21.16	c	c
propylene	115-07-01	4.59			6.91	0.07	0.15
propylene dichoride	78-87-5	*	0.01	annual average	99.54	0.18	0.18
styrene	100-42-5	0.55	512	annual average	69.12	0.13	0.34
1,1,2,2-tetrachloroethane	79-34-5	*	0.017	annual average	100	1.99	1.10
tetrachloroethylene	127-18-4	0.62	0.41	annual average	79.72	0.20	0.27
m-tolualdehyde	629-23-5	*			100	c	c
o-tolualdehyde	529-20-4	*			100	c	c
p-tolualdehyde	104-87-0	*			100	c	c
tolualdehydes		1.18			31.71		c
toluene	108-88-3	11.87	8930	24hr	1.38	0.26	0.15
1,2,4-trichlorobenzene	120-82-1	*			98.85	0.45	
1,1,1-trichloroethane	71-55-6	7.19	190000	8hr	15.21	0.11	1.80
1,1,2-trichloroethane	79-00-5	*	0.061	annual average	99.54	0.33	0.27
trichloroethylene	79-01-6	0.32	0.42	annual average	94.93	0.27	0.27
trichlorofluoromethane	75-69-4	1.81	133500	24hr	0.57	0.39	
1,2,4-trimethylbenzene	95-63-6	2.80	0.15	annual average	35.63	0.49	
1,3,5-trimethylbenzene	108-67-8	1.05			47.13	0.15	
valeraldehyde	110-62-3	0.13			76.19	c	c
vinyl chloride	75-01-4	*	0.2	annual average	100	0.18	0.28
vinylidene chloride	75-35-4	*			100	0.28	
m,p-xylene	108-38-3	5.81	See Xylene, total		13.82	0.43	0.48
o-xylene	195-47-6	2.23	See Xylene, total		20.74	0.13	0.26
xylene, total	1330-20-7	8.04	1040	annual average	n/a		

c - Denotes carbonyl compound. Detection limit (MDL) for carbonyls is a function of sample volume.

*- Compound concentration was below detection limit (MDL) in at least 95% of the samples collected.

All units are ug/M3

Table 2 (See Section III.B.1)

VOC Summary Statistics, 1993-1995

Laboratories: ESE from 1/93 to 3/95 and Radian from 8/95 to 12/95 Units: ug/M3
NND/N: Number of Non-Detects / Number of Samples
All Non-Detects = 1/2 MDL (except for high mean and low mean)
High Mean: All Non-Detects = MDL Low Mean: All Non-Detects = 0

1,1,1-trichloroethane state standard 190000 8hr CAS # 71-55-6
ESE MDL: 0.11 Radian MDL: 1.80

Site	Year	Average	Median	NND/N	High Mean	Low Mean	Min	Max	Range	Std. Dev.
Burlington	1993	1.20	1.09	1 / 14	1.21	1.20	0.05	3.27	3.22	0.71
Burlington	1994	1.04	1.04	1 / 31	1.05	1.04	0.05	1.58	1.53	0.34
Burlington	1995	1.69	0.90	8 / 18	2.09	1.29	0.82	9.49	8.68	2.06
Rutland	1993	1.03	1.04	0 / 13	1.03	1.03	0.76	1.36	0.60	0.17
Rutland	1994	1.15	1.04	0 / 30	1.15	1.15	0.65	4.20	3.55	0.67
Rutland	1995	0.98	0.90	9 / 17	1.46	0.50	0.71	2.51	1.80	0.41
Underhill	1994	0.77	0.71	0 / 13	0.77	0.77	0.60	1.75	1.15	0.30
Underhill	1995	1.15	0.90	9 / 18	1.60	0.70	0.65	3.71	3.06	0.85
Waterbury	1993	1.08	0.95	0 / 14	1.08	1.08	0.76	2.40	1.64	0.41
Waterbury	1994	1.36	1.15	0 / 18	1.36	1.36	0.82	3.76	2.95	0.67
Winooski	1994	7.19	2.56	0 / 13	7.19	7.19	0.82	28.16	27.34	9.95
Winooski	1995	5.22	1.77	5 / 18	5.47	4.97	0.87	31.81	30.94	7.97

1,2,4-trimethylbenzene state standard 0.15 annual average CAS # 95-63-6
ESE MDL: 0.49 Radian MDL:

Site	Year	Average	Median	NND/N	High Mean	Low Mean	Min	Max	Range	Std. Dev.
Burlington	1993	2.15	2.41	1 / 14	2.17	2.14	0.25	3.83	3.59	0.98
Burlington	1994	2.80	2.75	0 / 31	2.80	2.80	0.59	5.95	5.36	1.20
Burlington	1995	2.11	2.02	0 / 7	2.11	2.11	0.59	4.13	3.54	1.11
Rutland	1993	1.20	1.03	0 / 13	1.20	1.20	0.64	2.56	1.92	0.56
Rutland	1994	2.72	1.70	4 / 30	2.76	2.69	0.25	13.27	13.03	2.98
Rutland	1995	1.56	1.43	1 / 7	1.59	1.52	0.25	3.10	2.85	0.95
Underhill	1994	0.30	0.25	12 / 13	0.53	0.08	0.25	0.98	0.74	0.20
Underhill	1995	0.25	0.25	7 / 7	0.49	0.00	0.25	0.25	0.00	0.00
Waterbury	1993	0.37	0.25	10 / 14	0.55	0.20	0.25	1.13	0.88	0.25
Waterbury	1994	0.40	0.25	14 / 18	0.59	0.20	0.25	2.06	1.82	0.43
Winooski	1994	0.44	0.25	8 / 13	0.59	0.29	0.25	1.43	1.18	0.34
Winooski	1995	0.33	0.25	5 / 7	0.51	0.15	0.25	0.59	0.34	0.15

1,3,5-trimethylbenzene state standard none CAS # 108-67-8
ESE MDL: 0.15 Radian MDL:

Site	Year	Average	Median	NND/N	High Mean	Low Mean	Min	Max	Range	Std. Dev.
Burlington	1993	0.88	0.76	1 / 14	0.88	0.87	0.07	2.06	1.99	0.58
Burlington	1994	0.93	0.84	0 / 31	0.93	0.93	0.34	2.41	2.06	0.43
Burlington	1995	0.80	0.74	1 / 7	0.81	0.79	0.07	1.67	1.60	0.53
Rutland	1993	0.19	0.07	10 / 13	0.25	0.13	0.07	0.74	0.66	0.23
Rutland	1994	1.05	0.54	3 / 30	1.06	1.04	0.07	4.23	4.15	1.07
Rutland	1995	0.94	0.59	2 / 7	0.96	0.92	0.07	2.61	2.53	0.94
Underhill	1994	0.12	0.07	12 / 13	0.19	0.06	0.07	0.74	0.66	0.18
Underhill	1995	0.07	0.07	7 / 7	0.15	0.00	0.07	0.07	0.00	0.00
Waterbury	1993	0.18	0.07	11 / 14	0.24	0.12	0.07	0.88	0.81	0.23
Waterbury	1994	0.19	0.07	16/ 18	0.25	0.12	0.07	1.92	1.84	0.43
Winooski	1994	0.16	0.07	12 / 13	0.22	0.09	0.07	1.13	1.06	0.29
Winooski	1995	0.07	0.07	7 / 7	0.15	0.00	0.07	0.07	0.00	0.00

1,3-butadiene state standard 0.035 annual average CAS # 106-99-0
ESE MDL: 0.15 Radian MDL: 0.33

Site	Year	Average	Median	NND/N	High Mean	Low Mean	Min	Max	Range	Std. Dev.
Burlington	1993	0.64	0.67	2 / 14	0.65	0.63	0.08	1.39	1.32	0.37
Burlington	1994	0.95	0.84	0 / 31	0.95	0.95	0.44	2.04	1.59	0.38
Burlington	1995	0.47	0.28	9 / 18	0.55	0.39	0.17	1.24	1.07	0.37
Rutland	1993	0.32	0.27	5 / 13	0.35	0.29	0.08	0.82	0.74	0.27
Rutland	1994	0.84	0.49	0 / 30	0.84	0.84	0.15	4.03	3.87	0.88
Rutland	1995	0.34	0.17	9 / 17	0.43	0.25	0.17	0.88	0.72	0.23
Underhill	1994	0.11	0.08	10 / 13	0.17	0.05	0.08	0.35	0.28	0.08
Underhill	1995	0.13	0.17	18 / 18	0.26	0.00	0.08	0.17	0.09	0.04
Waterbury	1993	0.17	0.08	10 / 14	0.23	0.12	0.08	0.71	0.63	0.18
Waterbury	1994	0.18	0.12	9 / 18	0.22	0.14	0.08	0.80	0.72	0.17
Winooski	1994	0.18	0.18	6 / 13	0.22	0.14	0.08	0.53	0.45	0.13
Winooski	1995	0.20	0.17	11 / 18	0.30	0.10	0.15	0.35	0.20	0.07

a-methyl styrene state standard none CAS # 98-83-9
ESE MDL: 0.04 Radian MDL:

Site	Year	Average	Median	NND/N	High Mean	Low Mean	Min	Max	Range	Std. Dev.
Burlington	1993	0.12	0.02	12 / 14	0.14	0.10	0.02	0.85	0.83	0.26
Burlington	1994	0.14	0.02	22 / 31	0.15	0.12	0.02	0.72	0.70	0.21
Burlington	1995	0.02	0.02	7 / 7	0.04	0.00	0.02	0.02	0.00	0.00
Rutland	1993	0.09	0.02	11 / 13	0.10	0.07	0.02	0.64	0.62	0.18
Rutland	1994	0.07	0.02	26 / 30	0.09	0.05	0.02	0.51	0.49	0.13
Rutland	1995	0.02	0.02	7 / 7	0.04	0.00	0.02	0.02	0.00	0.00
Underhill	1994	0.08	0.02	12 / 13	0.10	0.06	0.02	0.77	0.75	0.21
Underhill	1995	0.02	0.02	7 / 7	0.04	0.00	0.02	0.02	0.00	0.00
Waterbury	1993	0.04	0.02	13 / 14	0.06	0.02	0.02	0.26	0.23	0.06
Waterbury	1994	0.02	0.02	18 / 18	0.04	0.00	0.02	0.02	0.00	0.00
Winooski	1994	0.05	0.02	12 / 13	0.07	0.03	0.02	0.34	0.32	0.09
Winooski	1995	0.02	0.02	7 / 7	0.04	0.00	0.02	0.02	0.00	0.00

acetylene state standard none CAS # 74-86-2
ESE MDL: Radian MDL: 0.13

Site	Year	Average	Median	NND/N	High Mean	Low Mean	Min	Max	Range	Std. Dev.
Burlington	1995	4.94	4.44	0 / 11	4.94	4.94	2.68	10.60	7.91	2.31
Rutland	1995	3.95	3.08	1 / 10	3.95	3.94	0.06	9.50	9.44	2.94
Underhill	1995	0.59	0.70	1 / 11	0.60	0.59	0.06	0.87	0.81	0.25
Winooski	1995	2.36	1.30	0 / 11	2.36	2.36	0.73	8.25	7.52	2.49

benzene state standard 0.12 annual average CAS # 71-43-2
ESE MDL: 0.10 Radian MDL: 0.77

Site	Year	Average	Median	NND/N	High Mean	Low Mean	Min	Max	Range	Std. Dev.
Burlington	1993	2.83	2.88	1 / 14	2.83	2.83	0.05	4.73	4.68	1.18
Burlington	1994	4.05	3.61	0 / 31	4.05	4.05	1.79	8.88	7.09	1.66
Burlington	1995	2.62	2.38	0 / 18	2.62	2.62	0.89	4.73	3.83	1.24
Rutland	1993	1.76	1.37	0 / 13	1.76	1.76	1.09	3.64	2.56	0.78
Rutland	1994	3.48	2.20	0 / 30	3.48	3.48	1.21	15.40	14.19	3.17
Rutland	1995	1.74	1.34	4 / 17	1.83	1.65	0.38	3.96	3.58	1.12
Underhill	1994	0.56	0.48	0 / 13	0.56	0.56	0.19	1.66	1.47	0.40
Underhill	1995	0.54	0.38	11 / 18	0.77	0.31	0.38	0.99	0.61	0.21
Waterbury	1993	0.94	0.65	0 / 14	0.94	0.94	0.38	3.00	2.62	0.71
Waterbury	1994	1.19	0.88	0 / 18	1.19	1.19	0.48	4.35	3.87	0.89
Winooski	1994	0.97	0.83	0 / 13	0.97	0.97	0.45	2.43	1.98	0.52
Winooski	1995	0.94	0.78	9 / 18	1.13	0.75	0.38	2.01	1.63	0.60

carbon tetrachloride state standard 0.067 annual average CAS # 56-23-5
ESE MDL: 0.19 Radian MDL: 0.44

Site	Year	Average	Median	NND/N	High Mean	Low Mean	Min	Max	Range	Std. Dev.
Burlington	1993	0.49	0.63	5 / 14	0.53	0.46	0.09	0.82	0.72	0.32
Burlington	1994	0.71	0.69	0 / 31	0.71	0.71	0.44	0.94	0.50	0.10
Burlington	1995	0.52	0.50	4 / 18	0.57	0.47	0.22	0.88	0.66	0.22
Rutland	1993	0.52	0.69	4 / 13	0.55	0.49	0.09	0.82	0.72	0.30
Rutland	1994	0.72	0.75	1 / 30	0.72	0.72	0.09	0.94	0.85	0.15
Rutland	1995	0.54	0.50	5 / 17	0.60	0.47	0.22	1.20	0.98	0.27
Underhill	1994	0.70	0.69	0 / 13	0.70	0.70	0.63	0.82	0.19	0.07
Underhill	1995	0.51	0.44	5 / 18	0.57	0.45	0.22	1.07	0.85	0.26
Waterbury	1993	0.48	0.60	5 / 14	0.52	0.45	0.09	0.82	0.72	0.31
Waterbury	1994	0.69	0.69	1 / 18	0.69	0.68	0.09	0.94	0.85	0.18
Winooski	1994	0.71	0.69	0 / 13	0.71	0.71	0.63	0.88	0.25	0.08
Winooski	1995	0.62	0.57	2 / 18	0.64	0.59	0.22	1.20	0.98	0.26

chlorodifluoromethane state standard none CAS # 75-45-6
ESE MDL: 0.32 Radian MDL:

Site	Year	Average	Median	NND/N	High Mean	Low Mean	Min	Max	Range	Std. Dev.
Burlington	1993	1.72	0.80	1 / 14	1.74	1.71	0.16	10.05	9.89	2.54
Burlington	1994	3.92	1.63	0 / 31	3.92	3.92	0.53	26.99	26.46	5.37
Burlington	1995	3.98	1.10	1 / 7	4.00	3.96	0.16	21.08	20.92	7.60
Rutland	1993	0.95	0.92	0 / 13	0.95	0.95	0.42	2.26	1.84	0.47
Rutland	1994	3.53	1.38	1 / 30	3.53	3.52	0.16	24.65	24.49	5.70
Rutland	1995	2.97	0.88	1 / 7	2.99	2.95	0.16	10.65	10.49	3.90
Underhill	1994	2.56	0.67	0 / 13	2.56	2.56	0.46	15.67	15.21	4.41
Underhill	1995	2.71	1.70	0 / 7	2.71	2.71	0.57	4.95	4.39	2.04
Waterbury	1993	3.61	0.83	1 / 14	3.62	3.60	0.16	33.00	32.84	8.57
Waterbury	1994	8.02	1.11	0 / 18	8.02	8.02	0.57	116.0	115.45	27.00
Winooski	1994	4.39	1.80	0 / 13	4.39	4.39	0.60	22.18	21.58	6.28
Winooski	1995	2.16	1.34	0 / 7	2.16	2.16	0.57	4.10	3.54	1.62

chloroethane state standard none CAS # 75-00-3
ESE MDL: 0.11 Radian MDL: 0.47

Site	Year	Average	Median	NND/N	High Mean	Low Mean	Min	Max	Range	Std. Dev.
Burlington	1993	0.05	0.05	14 / 14	0.11	0.00	0.05	0.05	0.00	0.00
Burlington	1994	0.07	0.05	27 / 31	0.12	0.02	0.05	0.24	0.18	0.05
Burlington	1995	0.17	0.24	18 / 18	0.33	0.00	0.05	0.24	0.18	0.09
Rutland	1993	0.07	0.05	12 / 13	0.12	0.02	0.05	0.24	0.18	0.05
Rutland	1994	0.08	0.05	24 / 30	0.12	0.04	0.05	0.26	0.21	0.06
Rutland	1995	0.17	0.24	16 / 17	0.33	0.01	0.05	0.24	0.18	0.09
Underhill	1994	0.07	0.05	11 / 13	0.11	0.02	0.05	0.18	0.13	0.04
Underhill	1995	0.17	0.24	18 / 18	0.33	0.00	0.05	0.24	0.18	0.09
Waterbury	1993	0.07	0.05	13 / 14	0.12	0.02	0.05	0.29	0.24	0.06
Waterbury	1994	0.05	0.05	18 / 18	0.11	0.00	0.05	0.05	0.00	0.00
Winooski	1994	0.05	0.05	13 / 13	0.11	0.00	0.05	0.05	0.00	0.00
Winooski	1995	0.17	0.24	18 / 18	0.33	0.00	0.05	0.24	0.18	0.09

chloroform state standard 0.043 annual average CAS # 67-66-3
ESE MDL: 0.24 Radian MDL: 0.29

Site	Year	Average	Median	NND/N	High Mean	Low Mean	Min	Max	Range	Std. Dev.
Burlington	1993	0.13	0.12	13 / 14	0.25	0.02	0.12	0.29	0.17	0.05
Burlington	1994	0.18	0.12	27 / 31	0.28	0.07	0.12	0.78	0.66	0.17
Burlington	1995	0.22	0.15	17 / 18	0.35	0.09	0.12	1.61	1.49	0.35
Rutland	1993	0.14	0.12	12 / 13	0.25	0.02	0.12	0.29	0.17	0.05
Rutland	1994	0.17	0.12	23 / 30	0.26	0.07	0.12	0.44	0.32	0.09
Rutland	1995	0.15	0.15	16 / 17	0.28	0.02	0.12	0.29	0.17	0.04
Underhill	1994	0.15	0.12	12 / 13	0.27	0.04	0.12	0.54	0.42	0.12
Underhill	1995	0.15	0.15	17 / 18	0.28	0.02	0.12	0.29	0.17	0.04
Waterbury	1993	0.14	0.12	13 / 14	0.25	0.02	0.12	0.34	0.22	0.06
Waterbury	1994	0.16	0.12	17 / 18	0.27	0.04	0.12	0.73	0.61	0.14
Winooski	1994	0.20	0.12	10 / 13	0.29	0.10	0.12	0.78	0.66	0.18
Winooski	1995	0.14	0.15	18 / 18	0.27	0.00	0.12	0.15	0.02	0.01

dichlorodifluoromethane state standard 118 24hr CAS # 75-71-8
ESE MDL: 0.35 Radian MDL:

Site	Year	Average	Median	NND/N	High Mean	Low Mean	Min	Max	Range	Std. Dev.
Burlington	1993	3.11	2.67	1 / 14	3.12	3.09	0.17	6.28	6.11	1.39
Burlington	1994	3.06	2.87	0 / 31	3.06	3.06	2.27	6.28	4.01	0.73
Burlington	1995	3.41	3.21	0 / 7	3.41	3.41	2.18	5.93	3.76	1.19
Rutland	1993	2.76	2.57	0 / 13	2.76	2.76	2.08	3.71	1.63	0.50
Rutland	1994	3.03	2.87	0 / 30	3.03	3.03	2.27	4.75	2.47	0.67
Rutland	1995	5.60	2.62	0 / 7	5.60	5.60	2.13	20.03	17.90	6.48
Underhill	1994	2.37	2.37	0 / 13	2.37	2.37	1.43	2.82	1.38	0.34
Underhill	1995	2.46	2.47	0 / 7	2.46	2.46	2.08	2.77	0.69	0.27
Waterbury	1993	2.42	2.60	0 / 14	2.42	2.42	0.84	3.41	2.57	0.59
Waterbury	1994	2.63	2.65	0 / 18	2.63	2.63	0.59	3.41	2.82	0.63
Winooski	1994	3.25	2.52	1 / 13	3.27	3.24	0.17	14.39	14.22	3.42
Winooski	1995	2.47	2.57	0 / 7	2.47	2.47	1.88	3.07	1.19	0.41

ethylbenzene state standard 43500 8hr CAS # 100-41-4
ESE MDL: 0.35 Radian MDL: 0.35

Site	Year	Average	Median	NND/N	High Mean	Low Mean	Min	Max	Range	Std. Dev.
Burlington	1993	1.24	1.24	1 / 14	1.25	1.23	0.17	2.13	1.95	0.54
Burlington	1994	1.54	1.56	0 / 31	1.54	1.54	0.56	2.78	2.21	0.54
Burlington	1995	1.21	1.09	0 / 18	1.21	1.21	0.52	2.30	1.78	0.59
Rutland	1993	1.11	0.96	1 / 13	1.13	1.10	0.17	2.34	2.17	0.74
Rutland	1994	1.46	0.93	1 / 30	1.46	1.45	0.17	6.43	6.25	1.40
Rutland	1995	0.76	0.61	3 / 17	0.79	0.73	0.17	2.04	1.87	0.52
Underhill	1994	0.25	0.17	11 / 13	0.39	0.10	0.17	0.91	0.74	0.21
Underhill	1995	1.81	0.17	11 / 18	1.92	1.71	0.17	8.55	8.38	2.69
Waterbury	1993	0.27	0.17	10 / 14	0.39	0.14	0.17	0.87	0.69	0.19
Waterbury	1994	0.30	0.17	12 / 18	0.41	0.18	0.17	1.13	0.96	0.25
Winooski	1994	0.52	0.43	2 / 13	0.55	0.49	0.17	1.00	0.83	0.26
Winooski	1995	0.50	0.48	6 / 18	0.56	0.44	0.17	2.30	2.13	0.48

freon 113 state standard 181000 24hr CAS # 76-13-1
ESE MDL: 1.09 Radian MDL:

Site	Year	Average	Median	NND/N	High Mean	Low Mean	Min	Max	Range	Std. Dev.
Burlington	1993	1.77	1.34	4 / 14	1.93	1.62	0.54	6.94	6.39	1.67
Burlington	1994	3.82	1.42	4 / 31	3.89	3.75	0.54	34.93	34.38	6.44
Burlington	1995	1.50	1.09	2 / 7	1.66	1.35	0.54	4.09	3.55	1.24
Rutland	1993	67.16	61.50	0 / 13	67.16	67.16	41.03	118.6	77.63	21.08
Rutland	1994	44.15	42.57	0 / 30	44.15	44.15	1.34	71.28	69.94	18.54
Rutland	1995	29.50	27.07	0 / 7	29.50	29.50	19.89	52.31	32.42	10.63
Underhill	1994	1.39	1.09	4 / 13	1.56	1.23	0.54	2.76	2.21	0.84
Underhill	1995	1.35	1.59	2 / 7	1.50	1.19	0.54	2.09	1.55	0.61
Waterbury	1993	2.89	1.71	5 / 14	3.09	2.70	0.54	15.96	15.42	4.17
Waterbury	1994	3.98	1.63	4 / 18	4.10	3.86	0.54	36.10	35.55	8.32
Winooski	1994	3.40	1.84	2 / 13	3.48	3.32	0.54	18.22	17.67	4.74
Winooski	1995	2.14	1.75	0 / 7	2.14	2.14	1.09	3.59	2.51	0.90

heptane state standard 16000 8hr CAS # 142-82-5
ESE MDL: 0.16 Radian MDL:

Site	Year	Average	Median	NND/N	High Mean	Low Mean	Min	Max	Range	Std. Dev.
Burlington	1993	0.68	0.61	1 / 14	0.69	0.68	0.08	1.43	1.35	0.34
Burlington	1994	1.27	1.23	0 / 31	1.27	1.27	0.37	2.66	2.30	0.54
Burlington	1995	1.02	1.11	1 / 7	1.03	1.01	0.08	1.60	1.52	0.49
Rutland	1993	0.38	0.29	5 / 13	0.42	0.35	0.08	0.94	0.86	0.31
Rutland	1994	1.52	0.96	1 / 30	1.52	1.52	0.08	9.92	9.84	1.88
Rutland	1995	0.93	0.94	0 / 7	0.93	0.93	0.45	1.80	1.35	0.44
Underhill	1994	0.16	0.08	10 / 13	0.22	0.09	0.08	0.57	0.49	0.16
Underhill	1995	0.25	0.29	1 / 7	0.26	0.24	0.08	0.33	0.25	0.09
Waterbury	1993	0.18	0.08	10 / 14	0.24	0.13	0.08	0.66	0.57	0.19
Waterbury	1994	0.39	0.31	3 / 18	0.40	0.37	0.08	1.27	1.19	0.30
Winooski	1994	0.54	0.37	3 / 13	0.56	0.52	0.08	2.83	2.75	0.72
Winooski	1995	0.40	0.37	1 / 7	0.41	0.39	0.08	0.66	0.57	0.19

hexane state standard 4290 24hr CAS # 110-54-3
ESE MDL: 0.18 Radian MDL:

Site	Year	Average	Median	NND/N	High Mean	Low Mean	Min	Max	Range	Std. Dev.
Burlington	1993	1.45	1.29	1 / 14	1.45	1.44	0.09	2.68	2.59	0.66
Burlington	1994	2.26	2.04	0 / 31	2.26	2.26	0.70	7.40	6.70	1.36
Burlington	1995	3.60	2.11	0 / 7	3.60	3.60	1.30	14.31	13.01	4.74
Rutland	1993	1.22	1.27	0 / 13	1.22	1.22	0.49	2.11	1.62	0.52
Rutland	1994	4.46	1.66	0 / 30	4.46	4.46	0.56	53.58	53.01	9.63
Rutland	1995	1.57	1.59	0 / 7	1.57	1.57	0.88	2.86	1.97	0.68
Underhill	1994	0.29	0.09	8 / 13	0.34	0.24	0.09	1.20	1.11	0.35
Underhill	1995	0.44	0.32	0 / 7	0.44	0.44	0.25	0.88	0.63	0.24
Waterbury	1993	0.44	0.19	7 / 14	0.48	0.40	0.09	2.08	1.99	0.54
Waterbury	1994	0.87	0.49	2 / 18	0.88	0.86	0.09	5.43	5.34	1.23
Winooski	1994	1.06	0.39	4 / 13	1.09	1.04	0.09	7.51	7.42	2.01
Winooski	1995	0.71	0.74	0 / 7	0.71	0.71	0.49	0.85	0.35	0.14

m,p-xylene state standard See Xylene, total CAS # 108-38-3
ESE MDL: 0.43 Radian MDL: 0.48

Site	Year	Average	Median	NND/N	High Mean	Low Mean	Min	Max	Range	Std. Dev.
Burlington	1993	4.01	4.34	1 / 14	4.03	3.99	0.22	7.16	6.95	1.77
Burlington	1994	5.81	5.86	0 / 31	5.81	5.81	1.56	11.07	9.51	2.16
Burlington	1995	4.78	4.56	0 / 18	4.78	4.78	2.17	9.73	7.56	2.14
Rutland	1993	2.48	2.39	0 / 13	2.48	2.48	1.26	4.21	2.95	0.98
Rutland	1994	5.67	3.67	0 / 30	5.67	5.67	1.00	21.45	20.45	5.49
Rutland	1995	3.17	2.91	1 / 17	3.18	3.15	0.24	8.99	8.75	2.15
Underhill	1994	0.49	0.22	9 / 13	0.64	0.34	0.22	2.30	2.08	0.60
Underhill	1995	0.70	0.24	10 / 18	0.83	0.58	0.22	2.17	1.95	0.64
Waterbury	1993	0.79	0.65	1 / 14	0.81	0.78	0.22	2.48	2.26	0.55
Waterbury	1994	0.93	0.83	5 / 18	0.99	0.87	0.22	3.91	3.69	0.87
Winooski	1994	1.08	1.00	2 / 13	1.11	1.05	0.22	3.00	2.78	0.75
Winooski	1995	1.44	1.00	1 / 18	1.45	1.43	0.22	8.21	7.99	1.74

methyl chloride state standard 0.01 annual average CAS # 74-87-3
ESE MDL: 0.21 Radian MDL: 0.81

Site	Year	Average	Median	NND/N	High Mean	Low Mean	Min	Max	Range	Std. Dev.
Burlington	1993	1.12	1.26	1 / 14	1.13	1.12	0.10	1.59	1.49	0.39
Burlington	1994	1.39	1.34	0 / 31	1.39	1.39	1.07	2.19	1.12	0.23
Burlington	1995	1.34	1.38	1 / 18	1.36	1.32	0.40	2.40	1.99	0.45
Rutland	1993	1.11	1.09	0 / 13	1.11	1.11	0.60	1.84	1.24	0.35
Rutland	1994	1.31	1.31	0 / 30	1.31	1.31	0.99	1.53	0.54	0.13
Rutland	1995	1.04	0.97	6 / 17	1.18	0.90	0.40	2.35	1.95	0.61
Underhill	1994	1.24	1.26	0 / 13	1.24	1.24	0.97	1.49	0.52	0.14
Underhill	1995	1.09	1.20	4 / 18	1.18	1.00	0.40	1.67	1.27	0.43
Waterbury	1993	1.06	1.19	0 / 14	1.06	1.06	0.31	1.69	1.38	0.41
Waterbury	1994	1.38	1.36	0 / 18	1.38	1.38	0.21	2.02	1.82	0.37
Winooski	1994	1.27	1.22	0 / 13	1.27	1.27	1.05	1.98	0.93	0.24
Winooski	1995	1.07	1.20	4 / 18	1.16	0.98	0.40	1.65	1.25	0.42

methylene chloride state standard 2 annual average CAS # 75-09-2
ESE MDL: 0.24 Radian MDL: 0.56

Site	Year	Average	Median	NND/N	High Mean	Low Mean	Min	Max	Range	Std. Dev.
Burlington	1993	1.80	1.08	2 / 14	1.82	1.78	0.12	7.61	7.49	2.03
Burlington	1994	7.79	2.85	1 / 31	7.80	7.79	0.12	101.4	101.32	18.41
Burlington	1995	5.20	0.71	8 / 18	5.33	5.08	0.28	71.91	71.63	16.72
Rutland	1993	1.82	1.08	0 / 13	1.82	1.82	0.49	7.99	7.50	2.00
Rutland	1994	8.41	1.55	1 / 30	8.42	8.41	0.12	125.4	125.29	23.32
Rutland	1995	3.66	1.49	5 / 17	3.74	3.58	0.28	12.92	12.65	4.15
Underhill	1994	4.74	1.95	0 / 13	4.74	4.74	0.49	18.52	18.03	5.80
Underhill	1995	1.95	0.28	10 / 18	2.11	1.80	0.28	10.32	10.04	2.99
Waterbury	1993	3.19	1.46	2 / 14	3.20	3.17	0.12	25.40	25.27	6.50
Waterbury	1994	3.66	1.20	0 / 18	3.66	3.66	0.24	19.04	18.79	5.34
Winooski	1994	6.75	2.29	0 / 13	6.75	6.75	0.56	39.95	39.40	10.99
Winooski	1995	2.08	0.64	9 / 18	2.22	1.94	0.28	11.39	11.12	2.90

o-xylene state standard See Xylene, total CAS # 195-47-6
ESE MDL: 0.13 Radian MDL: 0.26

Site	Year	Average	Median	NND/N	High Mean	Low Mean	Min	Max	Range	Std. Dev.
Burlington	1993	1.76	1.95	1 / 14	1.76	1.76	0.07	3.08	3.02	0.78
Burlington	1994	2.23	2.17	0 / 31	2.23	2.23	0.69	4.69	3.99	0.93
Burlington	1995	1.91	1.76	0 / 18	1.91	1.91	0.96	4.04	3.08	0.88
Rutland	1993	0.96	1.09	2 / 13	0.97	0.95	0.07	1.78	1.72	0.54
Rutland	1994	2.13	1.41	0 / 30	2.13	2.13	0.39	9.34	8.95	2.08
Rutland	1995	1.20	1.13	1 / 17	1.21	1.20	0.13	3.47	3.34	0.80
Underhill	1994	0.18	0.07	9 / 13	0.22	0.13	0.07	0.78	0.72	0.21
Underhill	1995	0.38	0.13	11 / 18	0.43	0.32	0.07	2.17	2.11	0.52
Waterbury	1993	0.25	0.07	8 / 14	0.29	0.21	0.07	1.04	0.98	0.28
Waterbury	1994	0.36	0.37	7 / 18	0.39	0.34	0.07	1.82	1.76	0.41
Winooski	1994	0.37	0.35	4 / 13	0.39	0.35	0.07	1.17	1.11	0.31
Winooski	1995	0.48	0.37	2 / 18	0.49	0.47	0.07	2.39	2.32	0.49

octane state standard none CAS # 111-65-9
ESE MDL: 0.28 Radian MDL: 0.23

Site	Year	Average	Median	NND/N	High Mean	Low Mean	Min	Max	Range	Std. Dev.
Burlington	1993	0.22	0.14	10 / 14	0.32	0.12	0.14	0.51	0.37	0.13
Burlington	1994	0.35	0.33	9 / 31	0.39	0.31	0.14	0.84	0.70	0.18
Burlington	1995	0.29	0.28	5 / 18	0.33	0.26	0.12	0.65	0.54	0.15
Rutland	1993	0.20	0.14	10 / 13	0.31	0.09	0.14	0.42	0.28	0.12
Rutland	1994	0.44	0.28	10 / 30	0.49	0.39	0.14	2.01	1.87	0.43
Rutland	1995	0.23	0.14	10 / 17	0.30	0.16	0.12	0.51	0.40	0.14
Underhill	1994	0.14	0.14	13 / 13	0.28	0.00	0.14	0.14	0.00	0.00
Underhill	1995	0.15	0.13	16 / 18	0.26	0.03	0.12	0.37	0.26	0.06
Waterbury	1993	0.15	0.14	13 / 14	0.28	0.02	0.14	0.28	0.14	0.04
Waterbury	1994	0.15	0.14	17 / 18	0.29	0.02	0.14	0.37	0.23	0.06
Winooski	1994	0.15	0.14	12 / 13	0.28	0.03	0.14	0.33	0.19	0.05
Winooski	1995	0.15	0.13	16 / 18	0.26	0.04	0.12	0.42	0.30	0.07

pentane state standard none CAS # 109-66-0
ESE MDL: 0.06 Radian MDL:

Site	Year	Average	Median	NND/N	High Mean	Low Mean	Min	Max	Range	Std. Dev.
Burlington	1993	4.04	3.64	1 / 14	4.04	4.03	0.03	8.62	8.59	2.32
Burlington	1994	5.72	4.34	0 / 31	5.72	5.72	2.33	22.57	20.24	4.10
Burlington	1995	3.46	3.10	0 / 7	3.46	3.46	2.04	5.58	3.54	1.32
Rutland	1993	3.39	2.27	0 / 13	3.39	3.39	1.42	9.24	7.82	2.43
Rutland	1994	5.52	3.69	0 / 30	5.52	5.52	1.65	20.60	18.94	4.92
Rutland	1995	3.09	2.71	0 / 7	3.09	3.09	1.27	6.34	5.08	1.64
Underhill	1994	0.62	0.44	3 / 13	0.62	0.61	0.03	2.10	2.07	0.66
Underhill	1995	0.79	0.65	0 / 7	0.79	0.79	0.53	1.77	1.24	0.44
Waterbury	1993	1.21	0.66	2 / 14	1.22	1.21	0.03	7.38	7.35	1.85
Waterbury	1994	2.92	1.11	0 / 18	2.92	2.92	0.35	31.57	31.22	7.20
Winooski	1994	1.46	1.27	1 / 13	1.46	1.46	0.03	4.63	4.60	1.27
Winooski	1995	1.55	1.68	0 / 7	1.55	1.55	0.91	1.98	1.06	0.41

propylene state standard none CAS # 115-07-0
ESE MDL: 0.07 Radian MDL: 0.15

Site	Year	Average	Median	NND/N	High Mean	Low Mean	Min	Max	Range	Std. Dev.
Burlington	1993	2.36	2.19	1 / 14	2.36	2.36	0.03	4.58	4.54	1.18
Burlington	1994	3.38	2.91	0 / 31	3.38	3.38	1.74	7.21	5.47	1.51
Burlington	1995	2.96	2.70	0 / 18	2.96	2.96	0.83	6.57	5.75	1.40
Rutland	1993	1.91	1.48	0 / 13	1.91	1.91	0.60	4.60	3.99	1.40
Rutland	1994	4.59	3.16	0 / 30	4.59	4.59	1.14	17.04	15.90	3.86
Rutland	1995	2.25	1.45	1 / 17	2.26	2.25	0.08	6.37	6.29	1.79
Underhill	1994	0.61	0.48	0 / 13	0.61	0.61	0.09	1.60	1.51	0.41
Underhill	1995	0.53	0.18	8 / 18	0.56	0.49	0.08	1.84	1.76	0.58
Waterbury	1993	1.19	0.61	1 / 14	1.19	1.19	0.03	4.51	4.47	1.36
Waterbury	1994	1.49	1.26	0 / 18	1.49	1.49	0.50	4.35	3.86	0.88
Winooski	1994	1.07	1.00	1 / 13	1.07	1.07	0.03	2.86	2.82	0.73
Winooski	1995	0.85	0.56	3 / 18	0.86	0.83	0.08	2.29	2.21	0.69

styrene state standard 512 annual average CAS # 100-42-5
ESE MDL: 0.13 Radian MDL: 0.34

Site	Year	Average	Median	NND/N	High Mean	Low Mean	Min	Max	Range	Std. Dev.
Burlington	1993	0.20	0.06	8 / 14	0.24	0.16	0.06	0.51	0.45	0.18
Burlington	1994	0.36	0.34	8 / 31	0.38	0.35	0.06	1.11	1.04	0.26
Burlington	1995	0.43	0.17	11 / 18	0.51	0.35	0.06	3.71	3.64	0.83
Rutland	1993	0.55	0.06	7 / 13	0.58	0.51	0.06	2.17	2.11	0.71
Rutland	1994	0.22	0.06	20 / 30	0.26	0.17	0.06	1.66	1.60	0.32
Rutland	1995	0.14	0.17	16 / 17	0.26	0.02	0.06	0.30	0.23	0.07
Underhill	1994	0.49	0.06	11 / 13	0.54	0.44	0.06	5.41	5.35	1.48
Underhill	1995	0.31	0.17	11 / 18	0.38	0.25	0.06	1.02	0.96	0.31
Waterbury	1993	0.06	0.06	14 / 14	0.13	0.00	0.06	0.06	0.00	0.00
Waterbury	1994	0.09	0.06	16 / 18	0.15	0.04	0.06	0.34	0.28	0.08
Winooski	1994	0.16	0.06	10 / 13	0.21	0.11	0.06	0.89	0.83	0.24
Winooski	1995	0.13	0.17	18 / 18	0.26	0.00	0.06	0.17	0.11	0.05

tetrachloroethylene state standard 0.41 annual average CAS # 127-18-4
ESE MDL: 0.20 Radian MDL: 0.27

Site	Year	Average	Median	NND/N	High Mean	Low Mean	Min	Max	Range	Std. Dev.
Burlington	1993	0.19	0.10	11 / 14	0.27	0.11	0.10	0.68	0.58	0.19
Burlington	1994	0.62	0.10	16 / 31	0.67	0.56	0.10	9.97	9.87	1.76
Burlington	1995	0.23	0.14	16 / 18	0.34	0.12	0.10	1.63	1.53	0.36
Rutland	1993	0.18	0.10	10 / 13	0.26	0.10	0.10	0.47	0.37	0.15
Rutland	1994	0.24	0.10	21 / 30	0.31	0.17	0.10	1.15	1.05	0.25
Rutland	1995	0.17	0.14	15 / 17	0.27	0.06	0.10	0.75	0.64	0.15
Underhill	1994	0.13	0.10	12 / 13	0.22	0.04	0.10	0.47	0.37	0.10
Underhill	1995	0.12	0.14	18 / 18	0.24	0.00	0.10	0.14	0.03	0.02
Waterbury	1993	0.10	0.10	14 / 14	0.20	0.00	0.10	0.10	0.00	0.00
Waterbury	1994	0.20	0.10	14 / 18	0.28	0.12	0.10	0.68	0.58	0.19
Winooski	1994	0.19	0.10	10 / 13	0.27	0.11	0.10	0.68	0.58	0.18
Winooski	1995	0.18	0.14	16 / 18	0.28	0.07	0.10	0.95	0.85	0.20

toluene state standard 8930 24hr CAS # 108-88-3
ESE MDL: 0.26 Radian MDL: 0.15

Site	Year	Average	Median	NND/N	High Mean	Low Mean	Min	Max	Range	Std. Dev.
Burlington	1993	7.25	7.03	1 / 14	7.26	7.24	0.13	13.46	13.32	3.36
Burlington	1994	11.87	9.87	0 / 31	11.87	11.87	3.17	58.04	54.88	9.47
Burlington	1995	6.26	5.56	0 / 18	6.26	6.26	2.71	12.66	9.95	2.75
Rutland	1993	5.11	3.96	1 / 13	5.12	5.10	0.13	12.48	12.34	3.24
Rutland	1994	9.49	6.90	0 / 30	9.49	9.49	2.19	34.90	32.71	7.67
Rutland	1995	4.54	3.20	1 / 17	4.55	4.54	0.08	10.70	10.63	2.94
Underhill	1994	1.46	0.64	0 / 13	1.46	1.46	0.34	6.48	6.14	1.76
Underhill	1995	1.13	0.79	0 / 18	1.13	1.13	0.34	2.86	2.53	0.76
Waterbury	1993	1.93	1.49	0 / 14	1.93	1.93	0.53	6.67	6.14	1.49
Waterbury	1994	2.68	1.79	0 / 18	2.68	2.68	0.75	11.80	11.04	2.90
Winooski	1994	2.85	2.86	0 / 13	2.85	2.85	0.75	6.29	5.54	1.55
Winooski	1995	2.50	1.71	0 / 18	2.50	2.50	0.72	8.48	7.76	2.03

trichloroethylene state standard 0.42 annual average CAS # 79-01-6
ESE MDL: 0.27 Radian MDL: 0.27

Site	Year	Average	Median	NND/N	High Mean	Low Mean	Min	Max	Range	Std. Dev.
Burlington	1993	0.13	0.13	14 / 14	0.27	0.00	0.13	0.13	0.00	0.00
Burlington	1994	0.14	0.13	30 / 31	0.27	0.01	0.13	0.38	0.24	0.04
Burlington	1995	0.13	0.13	18 / 18	0.27	0.00	0.13	0.13	0.00	0.00
Rutland	1993	0.32	0.13	12 / 13	0.44	0.19	0.13	2.52	2.39	0.66
Rutland	1994	0.19	0.13	29 / 30	0.32	0.06	0.13	1.83	1.69	0.31
Rutland	1995	0.13	0.13	17 / 17	0.27	0.00	0.13	0.13	0.00	0.00
Underhill	1994	0.13	0.13	13 / 13	0.27	0.00	0.13	0.13	0.00	0.00
Underhill	1995	0.17	0.13	17 / 18	0.30	0.04	0.13	0.81	0.67	0.16
Waterbury	1993	0.17	0.13	13 / 14	0.29	0.04	0.13	0.59	0.46	0.12
Waterbury	1994	0.13	0.13	18 / 18	0.27	0.00	0.13	0.13	0.00	0.00
Winooski	1994	0.27	0.13	9 / 13	0.37	0.18	0.13	0.70	0.56	0.22
Winooski	1995	0.17	0.13	16 / 18	0.29	0.05	0.13	0.48	0.35	0.10

trichlorofluoromethane state standard 133500 24hr CAS # 75-69-4
ESE MDL: 0.39 Radian MDL:

Site	Year	Average	Median	NND/N	High Mean	Low Mean	Min	Max	Range	Std. Dev.
Burlington	1993	1.54	1.63	1 / 14	1.55	1.52	0.20	2.02	1.83	0.43
Burlington	1994	1.81	1.69	0 / 31	1.81	1.81	1.40	3.54	2.14	0.40
Burlington	1995	1.57	1.52	0 / 7	1.57	1.57	1.07	2.25	1.18	0.36
Rutland	1993	1.56	1.57	0 / 13	1.56	1.56	1.24	2.08	0.84	0.24
Rutland	1994	1.71	1.69	0 / 30	1.71	1.71	1.35	2.36	1.01	0.23
Rutland	1995	1.59	1.46	0 / 7	1.59	1.59	1.07	2.70	1.63	0.51
Underhill	1994	1.59	1.52	0 / 13	1.59	1.59	1.35	2.70	1.35	0.34
Underhill	1995	1.57	1.46	0 / 7	1.57	1.57	1.40	1.85	0.45	0.18
Waterbury	1993	1.55	1.69	0 / 14	1.55	1.55	0.39	1.91	1.52	0.38
Waterbury	1994	1.81	1.66	0 / 18	1.81	1.81	0.45	3.32	2.87	0.60
Winooski	1994	1.58	1.57	0 / 13	1.58	1.58	1.40	2.14	0.73	0.19
Winooski	1995	1.57	1.57	0 / 7	1.57	1.57	1.46	1.74	0.28	0.09

xylene, total state standard 1040 annual average CAS # 1330-20-
ESE MDL: Radian MDL:

Site	Year	Average	Median	NND/N	High Mean	Low Mean	Min	Max	Range	Std. Dev.
Burlington	1993	5.77	6.36				0.28	10.25	9.97	2.55
Burlington	1994	8.04	8.12				2.26	15.20	12.94	3.05
Burlington	1995	6.68	6.27				3.13	13.77	10.64	3.01
Rutland	1993	3.44	3.56				1.63	5.99	4.36	1.47
Rutland	1994	7.80	4.99				1.48	30.79	29.31	7.49
Rutland	1995	4.37	4.04				0.37	12.46	12.09	2.95
Underhill	1994	0.66	0.28				0.28	3.08	2.80	0.81
Underhill	1995	1.08	0.37				0.28	3.95	3.67	1.10
Waterbury	1993	1.04	0.74				0.28	3.52	3.24	0.82
Waterbury	1994	1.29	1.15				0.28	5.73	5.45	1.27
Winooski	1994	1.45	1.39				0.28	4.17	3.89	1.06
Winooski	1995	1.93	1.37				0.28	10.60	10.31	2.23

Table 3 (See Section III.B.1)

Carbonyl Summary Statistics, 1994-1995

MDL for Carbonyl are a function of sample volume and vary from sample to sample.
Laboratories: ESE from 1/93 to 3/95 and Radian from 8/95 to 12/95
NND/N : Number of Non-Detects / Number of Samples
All Non-Detects = 1/2 MDL (except for high mean and low mean)
High Mean: All Non-Detects = MDL Low Mean: All Non-Detects = 0 Units: ug/M3

2,5-dimethylbenzaldehyde — state standard: no standard — CAS #: 5779-94-2

Site	Year	Average	Median	NND/N	High Mean	Low Mean	Min	Max	Range	Std. Dev.
Burlington	1994	0.15	0.15	34 / 34	0.30	0.00	0.12	0.16	0.04	0.01
Burlington	1995	0.17	0.15	12 / 18	0.26	0.09	0.06	0.44	0.37	0.10
Rutland	1994	0.17	0.16	36 / 36	0.34	0.00	0.08	0.47	0.38	0.05
Rutland	1995	0.17	0.16	14 / 17	0.29	0.05	0.08	0.32	0.24	0.07
Underhill	1994	0.11	0.11	13 / 13	0.22	0.00	0.10	0.13	0.03	0.01
Underhill	1995	0.08	0.09	13 / 17	0.14	0.03	0.02	0.32	0.30	0.07
Waterbury	1994	0.10	0.10	24 / 24	0.19	0.00	0.06	0.12	0.06	0.01
Winooski	1994	0.19	0.19	13 / 13	0.38	0.00	0.18	0.21	0.03	0.01
Winooski	1995	0.12	0.15	14 / 17	0.22	0.03	0.05	0.19	0.14	0.06

acetaldehyde — state standard: 1800 — 8hr — CAS #: 75-07-0

Site	Year	Average	Median	NND/N	High Mean	Low Mean	Min	Max	Range	Std. Dev.
Burlington	1994	1.80	1.73	0 / 34	1.80	1.80	0.74	2.94	2.20	0.57
Burlington	1995	3.93	2.94	0 / 18	3.93	3.93	0.80	23.07	22.27	5.10
Rutland	1994	1.66	1.73	0 / 36	1.66	1.66	0.33	3.41	3.07	0.71
Rutland	1995	4.65	3.41	0 / 17	4.65	4.65	0.83	19.73	18.89	4.71
Underhill	1994	0.57	0.45	0 / 13	0.57	0.57	0.15	1.17	1.03	0.36
Underhill	1995	1.59	0.60	0 / 17	1.59	1.59	0.18	9.26	9.08	2.31
Waterbury	1994	1.03	0.90	0 / 24	1.03	1.03	0.61	1.81	1.21	0.37
Winooski	1994	1.08	0.90	0 / 13	1.08	1.08	0.31	2.75	2.45	0.66
Winooski	1995	2.77	1.36	0 / 17	2.77	2.77	0.48	13.64	13.16	3.26

acetone — state standard: 178000 — 8hr — CAS #: 67-64-1

Site	Year	Average	Median	NND/N	High Mean	Low Mean	Min	Max	Range	Std. Dev.
Burlington	1994	2.15	2.14	0 / 34	2.15	2.15	0.83	3.77	2.94	0.88
Burlington	1995	4.92	4.60	0 / 18	4.92	4.92	0.97	11.04	10.07	3.56
Rutland	1994	2.18	2.11	0 / 36	2.18	2.18	0.59	4.83	4.24	1.11
Rutland	1995	6.26	4.56	0 / 17	6.26	6.26	0.84	16.56	15.71	5.15
Underhill	1994	1.15	1.01	0 / 13	1.15	1.15	0.21	2.33	2.12	0.61
Underhill	1995	1.85	1.15	0 / 17	1.85	1.85	0.46	5.04	4.58	1.38
Waterbury	1994	1.29	1.09	1 / 24	1.30	1.29	0.04	2.58	2.55	0.85
Winooski	1994	2.29	1.66	0 / 13	2.29	2.29	0.69	4.69	4.01	1.24
Winooski	1995	1.83	1.64	0 / 17	1.83	1.83	0.64	4.79	4.15	1.11

acrolein state standard 2.5 8hr CAS # 107-02-8

Site	Year	Average	Median	NND/N	High Mean	Low Mean	Min	Max	Range	Std. Dev.
Burlington	1994	0.05	0.05	33 / 34	0.10	0.01	0.05	0.18	0.13	0.02
Burlington	1995	0.07	0.05	13 / 18	0.09	0.04	0.01	0.24	0.23	0.06
Rutland	1994	0.06	0.06	35 / 36	0.11	0.00	0.01	0.15	0.15	0.02
Rutland	1995	0.11	0.06	11 / 17	0.14	0.09	0.01	0.31	0.30	0.10
Underhill	1994	0.04	0.04	13 / 13	0.07	0.00	0.03	0.04	0.01	0.00
Underhill	1995	0.02	0.01	17 / 17	0.04	0.00	0.00	0.05	0.05	0.02
Waterbury	1994	0.03	0.03	24 / 24	0.07	0.00	0.02	0.04	0.02	0.00
Winooski	1994	0.06	0.06	13 / 13	0.13	0.00	0.06	0.07	0.01	0.00
Winooski	1995	0.10	0.06	9 / 17	0.13	0.07	0.01	0.47	0.46	0.11

benzaldehyde state standard no standard CAS # 100-52-7

Site	Year	Average	Median	NND/N	High Mean	Low Mean	Min	Max	Range	Std. Dev.
Burlington	1994	0.19	0.20	10 / 34	0.21	0.16	0.08	0.35	0.27	0.08
Burlington	1995	0.32	0.31	5 / 18	0.35	0.29	0.09	0.83	0.74	0.20
Rutland	1994	0.15	0.10	27 / 36	0.22	0.07	0.08	0.47	0.39	0.10
Rutland	1995	0.26	0.15	10 / 17	0.32	0.20	0.08	0.79	0.71	0.24
Underhill	1994	0.07	0.06	13 / 13	0.13	0.00	0.05	0.10	0.05	0.01
Underhill	1995	0.07	0.05	13 / 17	0.10	0.03	0.02	0.26	0.25	0.06
Waterbury	1994	0.07	0.06	18 / 24	0.11	0.03	0.03	0.17	0.13	0.04
Winooski	1994	0.11	0.10	12 / 13	0.21	0.02	0.10	0.20	0.10	0.03
Winooski	1995	0.29	0.19	8 / 17	0.34	0.25	0.05	1.75	1.70	0.39

butyr/isobutyraldehyde state standard no standard CAS # 123-72-8

Site	Year	Average	Median	NND/N	High Mean	Low Mean	Min	Max	Range	Std. Dev.
Burlington	1994	0.15	0.15	9 / 34	0.16	0.13	0.06	0.33	0.28	0.07
Burlington	1995	0.50	0.50	5 / 18	0.51	0.48	0.06	1.23	1.18	0.40
Rutland	1994	0.14	0.15	15 / 36	0.17	0.11	0.06	0.30	0.24	0.07
Rutland	1995	0.65	0.64	6 / 17	0.67	0.62	0.06	1.68	1.62	0.60
Underhill	1994	0.05	0.04	11 / 13	0.09	0.01	0.04	0.09	0.06	0.02
Underhill	1995	0.10	0.04	8 / 17	0.12	0.09	0.00	0.39	0.38	0.10
Waterbury	1994	0.09	0.10	9 / 24	0.11	0.08	0.03	0.22	0.19	0.05
Winooski	1994	0.09	0.07	10 / 13	0.15	0.03	0.07	0.15	0.08	0.03
Winooski	1995	0.11	0.07	9 / 17	0.15	0.08	0.01	0.35	0.34	0.10

crotonaldehyde state standard no standard CAS # 123-73-9

Site	Year	Average	Median	NND/N	High Mean	Low Mean	Min	Max	Range	Std. Dev.
Burlington	1994	0.18	0.17	10 / 34	0.20	0.17	0.06	0.44	0.38	0.11
Burlington	1995	0.42	0.30	1 / 18	0.42	0.41	0.07	1.52	1.45	0.36
Rutland	1994	0.18	0.17	17 / 36	0.22	0.14	0.07	0.55	0.48	0.13
Rutland	1995	0.43	0.32	2 / 17	0.44	0.42	0.07	1.36	1.28	0.34
Underhill	1994	0.05	0.05	12 / 13	0.10	0.01	0.04	0.13	0.09	0.02
Underhill	1995	0.13	0.05	9 / 17	0.15	0.11	0.01	1.01	1.00	0.24
Waterbury	1994	0.09	0.05	15 / 24	0.11	0.06	0.03	0.25	0.22	0.07
Winooski	1994	0.08	0.08	13 / 13	0.16	0.00	0.08	0.09	0.02	0.00
Winooski	1995	0.24	0.08	7 / 17	0.27	0.21	0.02	1.30	1.28	0.37

formaldehyde			state standard		0.08	annual average		CAS #		50-00-0
Site	Year	Average	Median	NND/N	High Mean	Low Mean	Min	Max	Range	Std. Dev.
Burlington	1994	3.24	3.20	0 / 34	3.24	3.24	1.63	4.44	2.81	0.86
Burlington	1995	5.23	5.17	0 / 18	5.23	5.23	1.59	11.14	9.55	3.19
Rutland	1994	2.69	2.55	0 / 36	2.69	2.69	0.64	5.74	5.10	1.17
Rutland	1995	5.92	5.95	0 / 17	5.92	5.92	1.45	14.05	12.60	4.50
Underhill	1994	0.89	0.77	0 / 13	0.89	0.89	0.45	1.41	0.96	0.33
Underhill	1995	1.19	1.08	0 / 17	1.19	1.19	0.26	2.34	2.08	0.63
Waterbury	1994	1.97	1.90	0 / 24	1.97	1.97	0.75	3.85	3.11	0.63
Winooski	1994	1.77	1.86	0 / 13	1.77	1.77	0.57	3.39	2.82	0.85
Winooski	1995	10.16	9.14	0 / 17	10.16	10.16	0.86	44.28	43.43	11.38

hexanaldehyde			state standard			no standard		CAS #		66-25-1
Site	Year	Average	Median	NND/N	High Mean	Low Mean	Min	Max	Range	Std. Dev.
Burlington	1994	0.14	0.14	33 / 34	0.26	0.01	0.01	0.28	0.27	0.03
Burlington	1995	0.23	0.16	7 / 18	0.28	0.17	0.12	0.74	0.62	0.15
Rutland	1994	0.17	0.15	33 / 36	0.32	0.02	0.14	0.41	0.27	0.06
Rutland	1995	0.27	0.17	7 / 17	0.33	0.21	0.12	0.74	0.62	0.17
Underhill	1994	0.10	0.10	13 / 13	0.20	0.00	0.09	0.11	0.02	0.01
Underhill	1995	0.08	0.08	9 / 17	0.12	0.04	0.00	0.27	0.26	0.06
Waterbury	1994	0.10	0.09	22 / 24	0.18	0.02	0.06	0.19	0.13	0.03
Winooski	1994	0.19	0.17	12 / 13	0.35	0.03	0.16	0.40	0.23	0.06
Winooski	1995	0.12	0.13	9 / 17	0.19	0.05	0.01	0.17	0.16	0.05

isovaleraldehyde			state standard			no standard		CAS #		590-86-3
Site	Year	Average	Median	NND/N	High Mean	Low Mean	Min	Max	Range	Std. Dev.
Burlington	1994	0.07	0.07	34 / 34	0.15	0.00	0.07	0.08	0.01	0.00
Burlington	1995	0.17	0.08	10 / 18	0.20	0.13	0.03	0.94	0.92	0.23
Rutland	1994	0.08	0.08	36 / 36	0.17	0.00	0.04	0.22	0.18	0.02
Rutland	1995	0.06	0.07	17 / 17	0.13	0.00	0.04	0.09	0.05	0.02
Underhill	1994	0.05	0.06	13 / 13	0.11	0.00	0.05	0.06	0.01	0.00
Underhill	1995	0.03	0.04	17 / 17	0.06	0.00	0.01	0.05	0.05	0.02
Waterbury	1994	0.05	0.05	24 / 24	0.10	0.00	0.03	0.07	0.04	0.01
Winooski	1994	0.09	0.09	13 / 13	0.18	0.00	0.09	0.10	0.02	0.00
Winooski	1995	0.13	0.07	15 / 17	0.18	0.08	0.02	1.24	1.22	0.29

propionaldehyde			state standard			no standard		CAS #		132-38-6
Site	Year	Average	Median	NND/N	High Mean	Low Mean	Min	Max	Range	Std. Dev.
Burlington	1994	0.32	0.32	1 / 34	0.32	0.32	0.05	0.72	0.66	0.16
Burlington	1995	0.57	0.59	2 / 18	0.58	0.57	0.02	1.35	1.34	0.45
Rutland	1994	0.33	0.31	5 / 36	0.34	0.32	0.06	0.73	0.68	0.18
Rutland	1995	0.87	0.99	0 / 17	0.87	0.87	0.12	2.02	1.89	0.72
Underhill	1994	0.11	0.04	8 / 13	0.14	0.09	0.03	0.42	0.38	0.11
Underhill	1995	0.13	0.05	9 / 17	0.15	0.11	0.00	0.79	0.79	0.19
Waterbury	1994	0.18	0.18	2 / 24	0.18	0.17	0.04	0.41	0.38	0.08
Winooski	1994	0.18	0.16	6 / 13	0.21	0.15	0.06	0.39	0.33	0.13
Winooski	1995	0.19	0.14	7 / 17	0.21	0.17	0.01	0.71	0.70	0.20

tolualdehydes			state standard			no standard		CAS #		
Site	Year	Average	Median	NND/N	High Mean	Low Mean	Min	Max	Range	Std. Dev.
Burlington	1995	1.18	1.12	1 / 11	1.21	1.14	0.38	2.66	2.28	0.60
Rutland	1995	1.09	0.82	4 / 10	1.31	0.87	0.46	2.68	2.22	0.73
Underhill	1995	0.33	0.23	5 / 10	0.40	0.26	0.08	1.19	1.11	0.35
Winooski	1995	0.59	0.53	3 / 10	0.65	0.54	0.19	1.73	1.54	0.46

valeraldehyde			state standard			no standard		CAS #		110-62-3
Site	Year	Average	Median	NND/N	High Mean	Low Mean	Min	Max	Range	Std. Dev.
Burlington	1994	0.10	0.07	26 / 34	0.16	0.04	0.07	0.26	0.19	0.05
Burlington	1995	0.09	0.07	13 / 18	0.13	0.05	0.02	0.31	0.29	0.08
Rutland	1994	0.13	0.08	26 / 36	0.19	0.07	0.08	0.46	0.38	0.09
Rutland	1995	0.13	0.08	10 / 17	0.17	0.09	0.04	0.44	0.40	0.10
Underhill	1994	0.05	0.05	13 / 13	0.11	0.00	0.05	0.06	0.01	0.00
Underhill	1995	0.03	0.04	15 / 17	0.06	0.01	0.01	0.05	0.04	0.02
Waterbury	1994	0.12	0.06	13 / 24	0.15	0.09	0.04	0.32	0.28	0.09
Winooski	1994	0.10	0.09	12 / 13	0.18	0.01	0.09	0.19	0.10	0.03
Winooski	1995	0.06	0.03	16 / 17	0.11	0.01	0.02	0.25	0.23	0.06

Table 4

Semi-Volatile Data-See Section III.B.1

PAH (Polynuclear Aromatic Hydrocarbons) Data			Sample Date: October 24-26, 1995		
Compound	**Site**	**Concentration ug/m^3**	**HAAS ug/m^3**	**Exceeds HAAS**	**Flags**
Napthalene	Burlington	0.0295	120		S
	Winooski	0.0255	120		S
	Rutland	0.055	120		S
	Underhill	0.010	120		B
Phenanthrene	Burlington	0.0078	1.30		B
	Winooski	0.0062	1.30		B,PR
	Rutland	0.0178	1.30		none
	Underhill	0.0016	1.30		B
Flouranthane	Burlington	0.0016	130		none
	Winooski	0.0018	130		none
	Rutland	0.0030	130		none
	Underhill	0.0003	130		B
Pyrene	Burlington	0.0021	3.40		Q
	Winooski	0.0131	3.40		PR
	Rutland	0.0030	3.40		none
	Underhill	0.0003	3.40		B
Benzo(a)pyrene	Burlington	0.000048	0.0003		none
	Winooski	0.000006	0.0003		none
	Rutland	0.000347	0.0003	*	none
	Underhill	non-detect	0.0003		none

PAH (Polynuclear Aromatic Hydrocarbons) Data			Sample Date: February 27-29 1996		
Compound	**Site**	**Concentration ug/m^3**	**HAAS ug/m^3**	**Exceeds HAAS**	**Flags**
Napthalene	Burlington	0.4783	120		none
	Winooski	0.0771	120		none
	Rutland	0.2483	120		none
	Underhill	0.0193	120		S
Phenanthrene	Burlington	0.0140	1.30		none
	Winooski	0.0070	1.30		none
	Rutland	0.0134	1.30		none
	Underhill	0.0020	1.30		B
Flouranthane	Burlington	0.0045	130		none
	Winooski	0.0038	130		none
	Rutland	0.0035	130		none
	Underhill	0.0007	130		B
Pyrene	Burlington	0.0041	3.40		none
	Winooski	0.0085	3.40		none
	Rutland	0.0033	3.40		none
	Underhill	0.0006	3.40		B
Benzo(a)pyrene	Burlington	0.00053	0.0003	*	none
	Winooski	0.00034	0.0003	*	none
	Rutland	0.00046	0.0003	*	none
	Underhill	0.00006	0.0003		none

Table 4 con't

PCB (Polychlorinated Biphenyls) Data						
Sample Date	**Site**	**Concentration, ug/m^3**	**HAAS ug/m^3**	**Exceeds HAAS**	**Flags**	**Notes**
October 24-26 1995	Burlington	0.00134	0.00081		Q	
	Rutland	0.00058	0.00081		Q	
	Underhill	0.00057	0.00081		Q	
February 27-29 1996	Burlington	0.00018	0.00081		none	
	Winooski	0.00082	0.00081	*	none	98% of sample was EMPC
	Rutland	0.00013	0.00081		none	
	Underhill	0.00002	0.00081		none	

Dioxins/Furans (Chlorodibenzodioxins/chlorodibenzofurans) (a)					
Sample Date	**Site**	**Concentration pg/m3**	**HAAS pg/m3**	**Exceeds HAAS**	**Flags**
October 24-26 1995	Burlington	0.0072	0.02		B,PR
	Winooski	0.0188	0.02		none
	Rutland	0.0095	0.02		B,PR
	Underhill	0.0013	0.02		B,PR
February 27-29 1996	Burlington	0.0092	0.02		none
	Winooski	0.0052	0.02		none
	Rutland	0.0058	0.02		none
	Underhill	0.0015	0.02		none

Flag Definitions and Notes

S-Indicates that the signal exceeds the range.
The reported analyte concentration is a minimum estimate of the concentration.

B- Indicates that the analyte has been detected in the lab blank as well as the field blank.
This flag denotes possible contribution of background laboratory contamination to the field sample.

PR- Indicates a poorly resolved GC peak. The concentrations reported are most likely overestimated.

Q- Indicates quantitative interferences. Affected analytes may be overestimated or underestimated.

EMPC- (Estimated Possible Concentration) Calculated when peaks do not have appropriate ion mass ratios for a true PCB.

(a)-Dioxin/Furans are expressed as 2,3,7,8-tetrachlorodibenzo-p-dioxin

Appendix D

Research Status Report

Mercury Deposition and Effects in the Lake Champlain Basin

RESEARCH STATUS REPORT

Mercury Deposition and Effects in the Lake Champlain Basin

December 1, 1997

Tim Scherbatskoy
University of Vermont School of Natural Resources
Vermont Forest Ecosystem Monitoring Program
(802) 656-8336 ~ tscherba@zoo.uvm.edu

Co-Investigators:
Gerald Keeler, University of Michigan Air Quality Laboratory
Jamie Shanley, U.S. Geological Survey, Montpelier, VT
Mary Watzin, University of Vermont School of Natural Resources.

Background:

Since 1992, EPA, NOAA and other agencies have supported research and monitoring of atmospheric pollutant deposition to the Lake Champlain basin, a designated Great Water. Our work has focused on the deposition, ecosystem cycling, and fate of atmospheric mercury (Hg), as well as the wet and dry deposition of sulfur, nitrogen, other major ions and trace metals, and meso-scale modeling of pollutant deposition in the basin. Recent efforts have emphasized understanding Hg movement in forested watersheds, providing information on seasonal patterns of concentration and deposition, soil water transport mechanisms, winter accumulation in the snowpack, and input/output relations.

These studies are important to our efforts to understand Hg patterns, impacts and control in the Lake Champlain basin and the region. Our deposition monitoring is one of the longest running consistent Hg data base (5 years) in air and precipitation in the world, and is the only long-term year-around toxics deposition monitoring project in New England. These long-term data allow us to observe if changes in Hg emissions and controls on sources such as medical and municipal waste incinerators have impacts on Hg deposition in Vermont. Our site is also one of the few locations in North America where watershed deposition and ecosystem processing of Hg is being investigated. It is a critical site in the NESCAUM Hg monitoring program (EPA REMAP) started this past June. It is a critical location for comparisons and cooperation with the Canadian Networks. And finally, it allows us to connect the Lake Champlain and New England projects into the Great Lakes and Great Waters Hg research programs.

What we know:

Total Hg deposition (Figure 1) averages around 130 mg/ha per year (wet plus dry), and varies seasonally with 68% of annual deposition occurring during May-Sept. Dry deposition comprises approximately 38% of the total Hg deposition. About 85% of the total Hg deposition is retained in the terrestrial ecosystem (although some of this may return to the atmosphere by volatilization). Of the 15% exported in streamflow (Figure 2), about 1/3 is dissolved and 2/3 is associated with organic particulate matter. Our data suggest that a large proportion of total Hg flux in streamflow may occur during a very few large runoff events (spring snowmelt, floods). In the Lamoille River system, Hg concentrations are generally 3-10 ng/L, and tend to follow the pattern of the upland catchment, with elevated concentrations during higher flows (Figure 3). In the forest ecosystem, we know that Hg accumulates in foliage during the growing season, and the flux of Hg to the forest floor in autumn litterfall is large and comparable to the annual total wet plus dry atmospheric deposition. Concentrations of Hg in soil water (unpublished data) are much greater in the organic horizons (15-20 ng/L) than in the mineral horizons (1-2 ng/L), reflecting the similar behavior of dissolved organic carbon (DOC). At this time, we have begun analysis of the relationship between Hg in soil and stream water, with special attention to the role of DOC, and have also begun examination of the concentrations and pools of Hg in aquatic food web components (plankton, minnows, fish).

What we need to know:

We need to know atmospheric concentrations (gaseous Hg^0 and Hg^{2+}) and particulate phase Hg in its various forms) and deposition rates to (a) determine trends, and (b) understand atmospheric chemistry, transport and loading. Knowing the levels of atmospheric Hg, when determined with other trace elements (e.g., manganese, nickel, vanadium) and major ions (e.g. sulfate, nitrate, chloride) allows us to investigate the source(s) of the Hg in the environment. This requires continued monitoring of Hg in precipitation, vapor and aerosol phases.

We also need to refine our knowledge of the mechanisms controlling Hg transport within the forested watershed in order to identify risk factors and possible control points. Several lines of evidence indicate that soils - particularly highly organic forest soils - are very large sinks for Hg and can under certain conditions be significant sources for Hg. We need to continue work studying Hg transport and cycling in the forest, particularly the roles of particulate and dissolved organic carbon compounds in Hg transport. Lastly, the rates of

production of methyl-Hg in forested wetlands need to be investigated as this may control methyl-Hg levels in the ecosystem and in Lake Champlain.

We need to examine the pathways and mechanisms of Hg bioaccumulation in aquatic primary producers and consumers in order to complete our understanding of the movement of Hg from the atmosphere into the food web. This will require measurement of total and methyl-Hg in the water column, plankton and minnows.

To more accurately quantify total Hg deposition and loadings in the Lake Champlain basin, we need to refine models predicting spatial deposition patterns and dry deposition rates in the basin. This will require coordination among our group in Vermont (including the Vermont Air Pollution Control Division), the University of Michigan Air Quality Laboratory and the atmospheric modeling community (e.g., NOAA, EPA) to further develop and use these deposition models.

To calculate Hg loading to Lake Champlain we need information on Hg concentrations in soils and export to streams from important land use types. We currently have good information from one hardwood forested catchment, but need data from agricultural lands and other forest systems and catchments.

Therefore, our priorities for the next three years are to: (1) continue monitoring atmospheric Hg concentration and deposition, including cloud water and dew chemistry, (2) continue research on the role of organic matter in Hg transport in soil/stream systems, (3) expand the runoff and stream transport analysis to other watersheds in the basin (including both agricultural and other land uses), (4) measure Hg and methyl-Hg in aquatic food web components, and (5) assess regional air transport patterns, loadings to the basin and lake, and mass balance with the goal of identifying critical control points. We propose to address these five research areas through continuation of our collaborative research in the Lake Champlain basin utilizing experts in forest eco-physiology (Scherbatskoy), atmospheric chemistry (Keeler), watershed biogeochemistry (Shanley), and toxicology (Watzin). While this description focuses on Hg, most of these samples will also be analyzed by ICP-MS for other trace elements (Cd, As, Pb, Ni, V, Cr, Mn, etc.) as well, which will broaden our understanding of pollutant deposition and movement in the environment.

Relevant Publications:

Scherbatskoy, T. ,J.B. Shanley and G.J. Keeler, Factors controlling mercury transport in an upland forested catchment. Submitted to Water, Air, and Soil Pollution.

Rea, A.W., G.J. Keeler 1997. Microwave digestion and analysis of foliage for total mercury by cold vapor atomic fluorescence spectroscopy. In press, Biogeochemistry.

Rea, A.W., G.J. Keeler and T. Scherbatskoy. 1996. The deposition of mercury in throughfall and litterfall in the Lake Champlain watershed: a short-term study. Atmos. Environ. 30:3257-3263.

Scherbatskoy, T. , J.M. Burke, A.W. Rea and G.J. Keeler. 1997 Atmospheric mercury deposition and cycling in the Lake Champlain Basin of Vermont. pp 245-258 in: J.E. Baker (ed.) Atmospheric deposition of contaminants to the Great Lakes and coastal waters. SETAC Press, Pensacola, FL

Burke, J., M. Hoyer, G. Keeler and T. Scherbatskoy. 1995. Wet deposition of mercury and ambient mercury concentration at a site in the Lake Champlain Basin. Water Air Soil Pollut. 80:353-362.

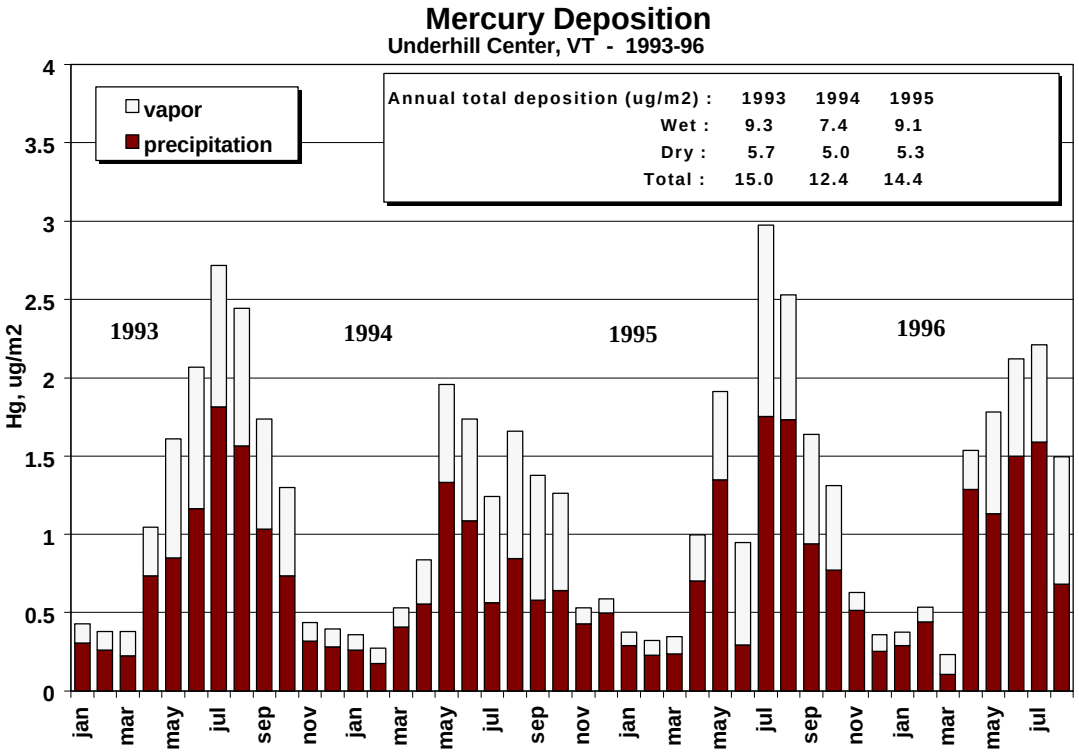

Figure 1. Monthly wet and dry deposition of Hg at the Underhill monitoring site.

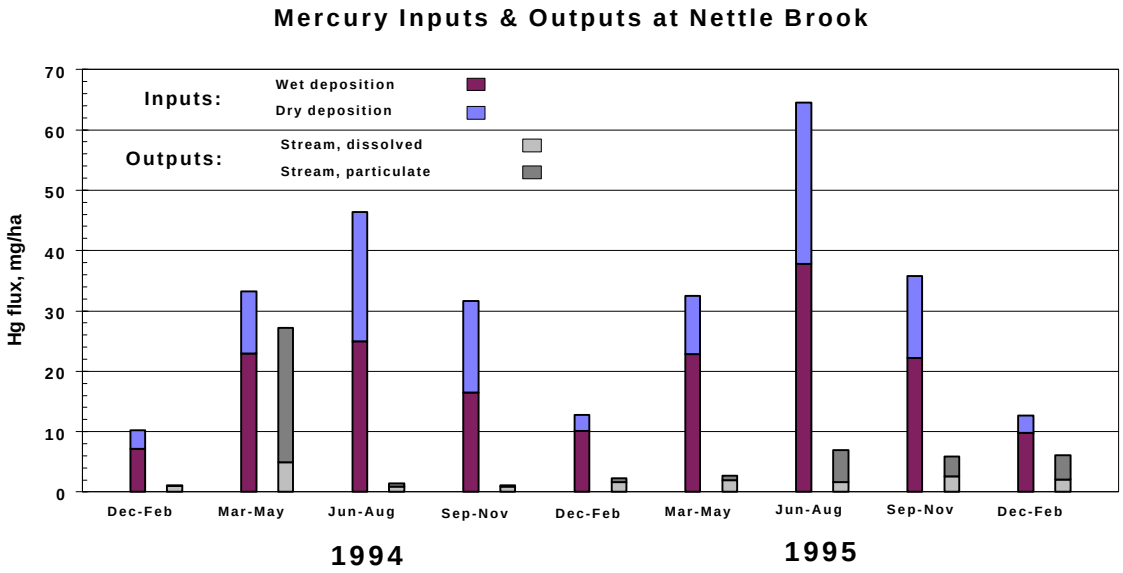

Figure 2. Hg input (wet and dry deposition) and output in streamflow (dissolved and particulate) in Nettle Brook, draining a small (11 ha) upland deciduous forest catchment in Underhill, VT.

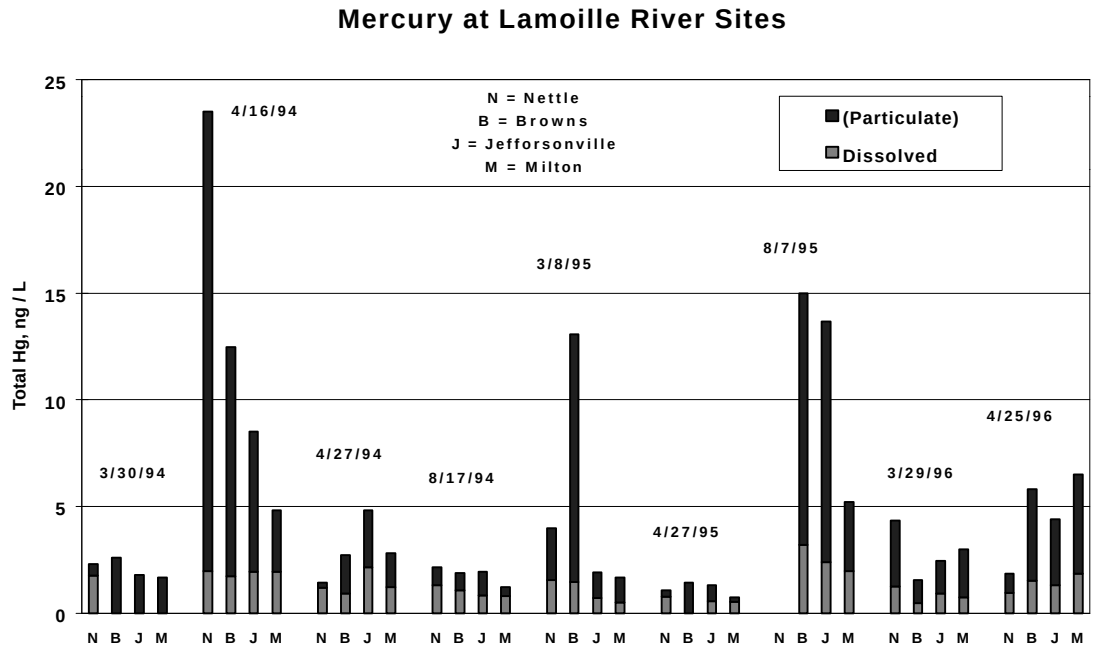

Figure 3. Hg concentration (dissolved and particulate phase) in Nettle Brook, Browns River, and two sites in the Lamoille River (Jeffersonville and Milton) during 1994-1996.

Appendix E

Toxicological Advisory Committee

Vermont Toxicological Advisory Committee

Member	Mailing Address	Phone Number
Dr. David Brown, Chair	NESCAUM 129 Portland St. Boston, MA 02114	(203) 259-5698 FAX (203) 256-8799
Dr. Bradley Schwab	Ogden Environmental & Energy Services 239 Littleton Rd., Suite 7C Westford, MA 01886	(978) 692-9090
Dr. John Morris	Univ. of Connecticut School of Pharmacy Box U-92 Storrs, CT 06269-2092	(860) 486-3590
Dr. William Graham	Fletcher Allen Hospital Pulmonary Lab 111 Colchester Ave. Burlington, VT 05401 or VT State Health Dept. P.O. Box 70 Burlington, VT 05402	(802) 425-3124
Dr. David Hemenway	Univ. of Vermont Dept. of Civil Engineering Votey Building Burlington, VT 05401	(802) 656-1935
Dr. William Bress	Vermont Health Dept. P.O. Box 70 Burlington, VT 05402	(802) 863-7220
Ms. Razelle Hoffman-Contois	Vermont Health Dept. P.O. Box 70 Burlington, VT 05402	(802) 863-7558 FAX (802) 863-7632
Ms. Linda Goldsmith	General Dynamics Lakeside Ave. Burlington, VT 05401-4985	(802) 657-6218

Ms. Sarah O'Brien	VPIRG 43 State St. Montpelier, VT 05602	(802) 223-6383
Mr. Brian J. Fitzgerald	Vermont ANR-APCD Bldg. 3 South, 103 South Main St. Waterbury, VT 05671-0402	(802) 241-3848 FAX (802) 241-2590
Ms. Mary Ann Abrahamson	Vermont ANR-APCD Bldg. 3 South, 103 South Main St. Waterbury, VT 05671-0402	(802) 241-3767 FAX (802) 241-2590