Sierra Club Great Lakes Program
Airborne Toxicant Education Series

An Introduction to the Evaluation and Regulation of Airborne Toxicants

Published by the
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Forward

This document is one in a series produced for the Sierra Club Great Lakes Program in order to facilitate and increase public understanding of toxic substance issues and the connection between toxic air pollution, Great Lakes water pollution and effects on human health and the environment.

In this document, we brief the reader on fundamental aspects of air quality engineering, toxicology and regulatory approaches used for addressing the problem of airborne toxicants and their impacts on public health and environment. The reader will hopefully come away with an introduction to the language and concepts of airborne toxicant regulation, emission control and risk assessment.

The Sierra Club Great Lakes Program hopes that these educational materials will assist citizens in their understanding of state and federal airborne toxicant regulations and stimulate discussion about potential changes in state regulatory policy to more fully protect public health and the environment. Other documents in this series describe state airborne toxicant regulations in detail, provide activist's checklists and discuss policy agenda's for proposed changes in state airborne toxicant programs.

The Sierra Club Great Lakes Program wishes to acknowledge a generous grant from the Joyce Foundation of Chicago to make these efforts possible.

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1 Introduction

This briefing paper is intended to give the reader a brief introduction to the most fundamental principles involved in evaluating and regulating airborne toxicants and their effects on health and environment. We've included some basic principles of air pollution engineering, air quality modeling and toxicology to help the reader begin to understand key policy and factual questions that arise when dealing with toxic air contaminants, their environmental fate and transport and their subsequent effects.

Only a portion of concepts presented here might be contained in a given state's airborne toxicant control program. Some states have no rules or program to control airborne toxicants. Citizens concerned about airborne toxicants can use the concepts presented here as a guidepost to better protection of public health and environment.

2. A Brief History on Hazardous Pollutants

In the mid-60s, modern air pollution control programs first focused on what came to be known as criteria pollutants. These common pollutants include particulate matter, sulfur dioxide, nitrogen oxides, carbon monoxide, photochemical oxidants and lead. The criteria pollutants were named after long "criteria documents," that the U.S. Environmental Protection Agency published describing the health and community welfare effects of these pollutants. The Agency found that excessive levels of the criteria pollutants in the air caused harmful effects on public health, vegetation, agriculture and community welfare.

EPA developed legally enforceable national ambient air quality standards to protect public health and community welfare from the effects of these common pollutants. The states developed plans to meet the federal standards by controlling these pollutants with emission rules and permit emission limitations.

In the late seventies and early eighties, some state air pollution control officials began to address "non-criteria" pollutants, which were the large universe of specific chemical compounds and toxic metals emitted by air pollution sources. These officials had to make decisions about new or modified industrial facilities emitting these non-criteria pollutants. Then-existing standards and emission rules to control solid and gaseous pollutants didn't account for the toxic effects of most "non-criteria" pollutants.

At the same time, public concern about the human health and environmental effects of toxic pollutants was increasing. Through the late 1970s and into the 1980s, the public learned that polychlorinated biphenyls (PCB) and mercury were contaminating fish. In 1984, a catastrophic release of methyl isocyanate by a Union Carbide chemical plant in Bhopal, India immediately killed over 2500 people and injured many others. In 1987, EPA published the first toxic release inventory (TRI) under the Superfund law. The TRI showed that U.S. industry discharged relatively massive amounts of 313

different toxic chemicals that were only regulated as particulate matter and/or as volatile organic compounds with no attention to their individual toxic nature. Prior to release of the TRI, EPA had given very little regulatory attention to toxicity issues in emission regulations. At the time, EPA had only issued about 7 standards under a Clean Air Act program for National Emission Standards for Hazardous Pollutants.

The release of the TRI enabled the public to confront state and federal air pollution control officials and industrial parties with questions on whether toxic chemical emissions from new and/or existing air pollution sources constituted a public health or environmental hazard.

By 1990, Congress legislated control technology and risk assessment requirements covering a variety of industrial sectors and 189 specifically designated toxic compounds.

3. The Public Policy Basis for Controlling Airborne Toxicants

Established public policy objectives motivate state and federal efforts to evaluate and control airborne toxicant emissions. These objectives include the public health paradigm of prevention, the precautionary principle on protection of the environment and the public trust doctrine for natural resources. Citizens working to evaluate and control airborne toxicants should always measure how well government agency rules and legislative proposals on airborne toxicants support these public policy objectives.

3.1 The Public Health Paradigm of Prevention

The public health prevention paradigm stresses health promotion and disease prevention. The function of directed public health programs is to anticipate potential harm and to take actions through education and government intervention to prevent these hazards from actually causing increased illness or death. For example, the foreword to the 1979 report of the Surgeon General of the United States stated boldly that its purpose was to:

> "encourage a second public health revolution in the history of the United States.... let us make no mistake about the significance of this document, it represents an emerging consensus among scientists and the health community that the Nation's health strategy must be dramatically recast to emphasize the prevention of disease."

Public health officials who take the prevention paradigm to heart take prudent and reasonable measures to effectively avoid sickness and death. Scientific investigations of environmental health risks from toxic substances help target priority setting. Experience

and lessons learned in one context, such as protection of occupational health, are translated for use in other contexts, such as controlling community air contamination. Physical and biological monitoring, epidemiology and disease surveillance is used to monitor the effectiveness of these prevention efforts and also serve as the basis for imposing additional controls..

3.2 The Precautionary Principle

The United States is a party to the world-wide Rio Declaration which provides, in part:

> "In order to protect the environment, the precautionary approach shall be widely applied by States according to their capabilities. Where there are threats of serious or irreversible damage, lack of full scientific certainty shall not be used as a reason for postponing cost-effective measures to prevent environmental degradation."[1]

We know that many toxic chemical compounds and elements have the potential to cause serious damage to the environment. However, we frequently do not have adequate data, or sometimes even any data at all, to ensure that the consequences of decisions about emissions and economic development will not cause serious and/or irreversible environmental harm.

In the absence of a complete understanding of potential risks, the Rio Declaration's embrace of the precautionary principle mandates that effective and reasonably available measures will be imposed in an attempt to avoid serious and irreversible damage. Such measures include the use of available emission control technologies, environmental and public health impact evaluation measures and efforts to control human and ecological exposures.

With particularly significant known hazards, such as those posed to the Great Lakes from persistent bioaccumulative toxicants, a "reverse onus" can be imposed under the precautionary principle in dealing with decisions covering emissions or process use of known dangerous materials. Under this concept, the burden of proof is reversed so that the party wishing to continue using or emitting such dangerous materials bears the burden of showing acceptability. The presumption under "reverse onus" emphasizes virtual elimination of hazardous emissions of particularly dangerous materials and potential bans on the industrial and consumer process uses of such materials. U.S. regulation banning poly-chlorinated biphenyls in most industrial and consumer process uses is an example of this approach.

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[1] Rio Declaration on Environment and Development, U.N. Conference on Environment and Development, Agenda 21, Principle 15 (1992)

3.3 Public Trust Doctrine for Publicly Owned Natural Resources

The public trust doctrine is an acknowledgment that air, water, fish and wildlife are publicly owned natural resources to which all should have free and equal access. It states that these resources are entrusted to the care of resource management agencies, whose duty is to manage them as a public trust, prohibiting conduct by any party that would make exclusive and/or near exclusive use of these resources. Language articulating this public trust doctrine is found in a number of state constitutional enactments.

Citizen participation and intervention frequently plays an important role in protecting the public trust in natural resources and monitoring the quality of decisions by resource management agencies. Where these agencies fail to carry out their duties, many environmental statutes that emphasize the public trust empower citizens with the ability to go to court to protect public resources.

4 Translating Public Policy Concerns Into Environmentally Protective Controls on Airborne Toxicants

To bridge the gap between public policy concerns and the adoption of laws and regulations to control the impact of airborne toxicants, scientists and regulators must conduct detailed review and evaluation of the physical and biological aspects of airborne toxicants. This type of evaluation will then serve as the basis for making statutory and regulatory decisions on how airborne toxicants will be regulated, the form of agency rulemaking that must be adopted and the provisions of permits that will apply to new, modified and existing industrial and other sources of airborne toxicants.

Physical evaluation emphasizes an understanding of airborne toxicant emissions that are or will occur based on emission control engineering decisions, the fate in the environment of these pollutants and the level of exposure to the community. Biological evaluation develops knowledge about the dose to humans and animals associated with environmental exposure, the likely harmful effects that will result and the development of assessment measures whose purpose is to prevent adverse biological and health effects.

5. Physical Review and Assessment of Airborne Toxicants

5.1 Emission Characterization and Monitoring

Knowing what toxic chemicals are emitted from existing sources or which are likely to be emitted from proposed new or modified sources is an essential feature of the airborne toxicant evaluation process.

Emission estimates are frequently developed for making permit applications or for making reports to state and federal agencies. Such estimation methods are based on engineering calculations and the use of certain assumptions. Published "emission factors" relate the amount of emissions expected without the application of emission control devices to the amount of production in an a piece of industrial or chemical process equipment. Assumptions about the efficiency of air pollution control equipment can then be used to estimate actual emissions.

Although the estimation methods are useful and provide insight for facilities that are new and/or modified sources, estimation can still yield results that are not truly reflective of actual source emissions. Variations in process equipment, operating methods and emission control equipment can lead to defects in the emission estimates.

For sources presently in operation, stack testing can determine the actual emissions of selected airborne toxicants. Stack tests are frequently done for three testing periods each of which typically last two and a half to three hours long. The emission results are then averaged across the three runs to determine whether the facility is complying with any emission limitation to which it is subject.

Unfortunately, stack tests cannot be done on an unannounced basis. In practice, the vast majority of stack tests are done by contractors hired by the owners or operators of the emission sources in question. In general, it is accepted industrial practice that such facilities are "on their best behavior" during such a test, which may not necessarily reflect emissions occurring at other times and under other conditions.

At the present time, continuous stack emission analyzers exist only for common pollutants like sulfur dioxide and carbon monoxide and not for toxic emissions. Although continuous stack emission analyzers are beginning to be used for measuring toxic heavy metals in Europe, they have not yet been finally accepted for use in the United States by the U.S. Environmental Protection Agency.

5.2 Emission Inventories

One goal of a government program to regulate toxic air pollution is the creation of an emission inventory system. EPA's Toxic Release Inventory (TRI) system is one such attempt at an emission inventory. This data is collected nationally by U.S. EPA from certain source categories.

For over ten years, public availability of TRI data has stimulated public interest the performance of local industrial emitters at limiting emissions and accounting for potential health and environmental effects from such industrial operations. In addition to air

emissions, pollutants discharged to other environmental media are covered and waste generation and pollution prevention are monitored as well.

At the present time, the TRI system provides a lot of information but it does contain several gaps. Not all source categories that emit airborne toxicants are covered. Some sources are not required to report emissions that are created as intermediate products in their process. The reporting thresholds for certain bioaccumulative pollutants are far too high presently or such pollutants do not appear on the TRI reporting list.

Other toxic emission inventories are being created by some states and regions. For example, both Michigan and Wisconsin conduct their own inventories of airborne toxicants from industrial sources.

In the Great Lakes area, a major effort has been put into the Regional Air Pollutant Inventory Development System (RAPIDS) to develop basin-wide estimations of airborne toxicants. This most recent effort of the Great Lakes states is unique because it attempts to identify airborne toxicants from all sources and not just those that come from point sources --- industrial pollution smokestacks and vents. Area sources of airborne toxicants (small sources from commercial, residential or light industrial activities) and mobile sources (cars, trucks, railroad engines, ORVs) are emerging as very significant from a regional standpoint. For example, residential trash disposal by burning and wood burning for heating will likely be regionally significant sources of poly-cyclic aromatic hydrocarbons and other pollutants of concern.

5.3 Air Quality Dispersion Modeling

Once emissions of a toxic air contaminant are known in the form of a mass emission rate (such as grams per second), mathematical computer models can be used to estimate "ambient" concentrations of the pollutant in air at ground level. These ambient concentration predictions are then used in efforts to develop inhalation exposure estimates for risk assessment purposes.

"Air quality dispersion models" come in a wide variety of forms. The most basic air quality models are designed to estimate maximum ambient, ground level concentrations of air contaminants from a single air pollution source within a couple of miles of a facility. The simplest models are most frequently used in permitting new or modified sources of toxic air contaminants. The model outputs can be used to derive predictions of maximum average ambient ground-level air concentrations occurring over a ten minute, one hour, 8 hour, 24 hour or annual sampling time period. The different time sampling periods are used to develop exposure assessments appropriate to the toxic effect of a particular airborne toxicant in question.

Significantly more complex models must be used to address multiple point and area sources, multiple metropolitan areas and large regions. EPA is presently undertaking such large-scale modeling for toxic exposures in its "Cumulative Exposure" program. These large scale models have also been used to develop predictions of urban smog exposure over large sections of the Eastern United States.

5.4 Environmental Fate, Transport, Chemical Transformation and Deposition Analysis

Although determining the inhalation exposure from airborne toxicants through use of the most basic air quality models is important, it is important to track in detail and model what happens to toxicants after they are emitted, transported and deposited because these patterns will sometimes affect non-inhalation exposure. The need for other types of exposure assessment helps drive the development of the most sophisticated environmental modeling that examines the fate of emitted air contaminants.

Real world toxic air contaminants may be transported hundreds of miles, chemically transformed and deposited in land and water. In order to develop multi-pathway exposure analysis, significantly more complex modeling efforts are needed.

Environmental fate and transport modeling together with ecological risk assessment begins to allow us to understand and predict exposures from many different pathways. For example, atmospheric dispersion analysis together with extended techniques can estimate the dry deposition of mercury to inland or the Great Lakes. Once the deposition analysis is done, ecological exposure and risk assessment can be used to estimate mercury uptake by fish and other flora and fauna in the water column.

5.5 Ambient Airborne Toxicant Monitoring

It is possible with advanced analytical techniques to measure atmospheric concentrations of airborne toxicants, and deposition of toxicants to the land, in communities and in the environment. However, in practice, these efforts are relatively limited because of expense and difficulty. Most such ambient monitoring is related to scientific investigations of atmospheric contamination and Great Lakes deposition and related health assessment determinations. The use of ambient toxic air contaminant monitoring in programs to regulate toxic emissions is virtually non-existent. Instead, modeling efforts are used for regulatory programs to determine whether acceptable ambient concentrations of airborne toxicants will be ensured with the permitting and regulation of point sources of toxicants.

6. Biological Evaluation of Airborne Toxicants

6.1 The Effects of Toxic Substances on Human Health – a Very Brief Introduction to Chemical Toxicology and Risk Assessment

The health effects of human exposure to chemical substances are evaluated through the science of toxicology, the study of the nature and action of chemical substances and poisons on humans and animals. Toxicological studies can be supplemented by the science of human epidemiology, which uses statistical analysis to relate the incidence of disease among groups of people to potential causative factors, such as chemical exposures.

6.2 Potential Health Effects of Chemical Toxicants

Exposure to toxic substances can lead to one or more types of harmful effects on humans and animals, depending on the dose and other factors. These effects are described in the following table:

Category of Toxicant	**Description of Biological Effect on Living Systems (The Existence and Magnitude of any Effects is Dependant on Exposure and Dose-Response Characteristics)**
Pulmonary Irritants	A pulmonary irritant causes inflamation of tissues in the lung and/or airways. The most common effect of pulmonary irritants is to increase secretions from mucous membranes and to change the mechanics of lung function (i.e. decrease the ability of the lung to take in air and exchange oxygen with the circulatory system). More severe exposures to pulmonary irritants can cause the lungs to fill with fluid, cause pulmonary bleeding or destroy lung tissues.
Pulmonary Fibrotic Agents	Materials, such as silica, that cause development of fibrous, inflexible tissue in human lungs.
Sensitizer	A substance that can cause exposed individuals to develop mild to severe allergic reactions after repeated exposures.
Mutagen	A substance that can react with cell nuclear material at the gene and/or chromosome level
Carcinogen	A substance that can either initiate development of cancer cells and/or promote the action of cancer cell development by other agents or causes.

Teratogen & Reproductive Toxicant	Teratogens are substances that kill, damage or cause defects in embryos and fetuses. Reproductive toxicants interfere with the ability of the body to produce and process healthy eggs and sperm for reproduction.
Endocrine Disruptor	A substance that interferes with the body's hormone systems for regulating growth, development and reproduction through the action of chemicals which mimic, block or trigger biological hormonal responses.
Immunotox-icant	Agents which destroy, degrade or interfere with the body's immune system by killing or damaging immune system cells.
Chemical Asphyxiant	A chemical asphyxiant interferes with the body's ability to absorb oxygen.
Anesthetic	Anesthetics cause a depressant effect on the central nervous system and the brain.
Hepatotoxic Agent	Hepatotoxicants damage the liver and or alter the availability of liver enzymes involved in breaking down chemical toxicants.
Nephrotoxic Agent	Nephrotoxic agents damage the kidneys and their ability to clean toxicants out of the blood for elimination.
Neurotoxic Agent	Neurotoxic agents damage and/or degrade the ability of the nervous system and the brain to function properly.
Hematopoie-tic Agent	These toxicants damage blood cells or the bone marrow that /forms blood cells.
Corrosives	A corrosive substance destroys and/or causes irreversible damage to living cells

As can be seen from the table, the many categories of individual chemical toxicants can cause many different types of harmful effects in humans and animals. Each chemical toxicant may cause one or more of such different effects. As a result, it can be difficult to develop standards to adequately protect human and animal health from the effects of chemical toxicants.

Some of the toxic effects of chemical exposure are manifest as prompt or "acute" effects which come relatively quickly after exposure to generally larger amounts of a chemical toxicant. For example, human exposure to high concentrations of chlorine dioxide, phosgene gas or hydrogen sulfide will cause immediate acute illness and potential for death. Because acute effects occur relatively quickly after chemical exposure, they are frequently easier to document in epidemiology studies. The relatively

short time between chemical exposure and adverse effect makes it easier to draw a connection between the two. Sometimes with acute exposures taking place over relatively short times, more information is available about the actual dose to which a person is exposed.

Other effects, such as the formation of cancers, neurological damage and disruption of the endocrine system, are regarded as delayed or "chronic" exposure effects. Such chronic effects generally have a latency period in which biological effects are less apparent or not immediately detectable. For example, human exposure to certain asbestos fibers can cause cancer of the lining of the lung (known as mesothelioma) up to 20-30 years after exposure.

6.3 Dose-Response Relationships and Toxic Effects

A primary goal of toxicological investigation is to determine the dose of a chemical, administered over a specific period of time, that will cause a specific adverse biological effect. Agencies use this information, a "dose-response" relationship, to develop recommendations and/or regulations designed to protect people and animals from the harmful effects of chemical toxicants.

Dose-response relationships are most often determined from studies on laboratory test animals. A second source of dose-response information is the epidemiological investigation done on workers exposed to chemicals. Finally, there are a few epidemiological studies on people who were subjected to accidental chemical releases, food contaminated with toxic chemicals, or other unfortunate occurrences.

6.4 Testing for Acute Toxicity

The objective of dose-response analysis is to determine the level of chemical exposure that results in a verifiable level of biological effect. In animal studies, groups of animals are exposed to different amounts of chemicals over a known period of time. The most basic animal toxicity test is to determine an LD50 (oral dose at which 50% of the test animals die, on average) or an LC50 (inhalation dose at which 50% of the test animals die, on average). These acute toxicity tests begin to describe the toxicity of the chemical; however, they do not tell you if non-lethal harmful effects are likely to occur.

More sophisticated tests of acute toxicity involve feeding or inhalation animal tests to determine an oral or inhalation No Observable Effects Level (NOEL). In NOEL tests, animals are exposed over a significant period of time to a range of doses lower than those expected to cause death. The animals are then examined for any evidence of tissue damage or disease. NOEL tests can only detect tissue damage and other effects that can

be revealed with biological examination, behavioral observations and chemical analysis. Sometimes the results of this kind of testing is alternatively expressed as a Lowest Observable Effects Level (LOEL), which is the lowest dose that shows an adverse effect.

The test animal still cannot tell the toxicologist about potential negative effects not measurable by the NOEL method – the more subtle and/or elusive influences of toxic chemical exposure. For example, the laboratory animal is unable to tell the researcher that it had a headache, learning problems or other neurological effects. Such subtle effects are problematic to detect and accepted laboratory methodologies may not always be available to determine all harmful effects that can occur.

6.5 Testing for Chronic Toxicity

Long term chronic animal tests are used to determine whether a substance is a carcinogen (cancer-causing chemical compound or element). In a long term test, laboratory animals are exposed by inhalation or by gavage methods (ingestion to the stomach) or by application to the test animal's skin over several weeks or months. The intent of these tests is to expose the laboratory animals to amounts and rates of exposure which would not otherwise be fatal, but which constitute a maximum tolerated dose. The objective in carcinogenicity bioassays is to measure the potential for tumor development from longer term exposure to a chemical agent.

Chronic exposure to a chemical toxicant may also cause other types of harmful effects, such as pulmonary damage, endocrine disruption, learning disabilities, neurological damage, birth defects, etc. Some of these other effects may be more important than cancer induction or promotion or otherwise occur with less exposure. Established and accepted testing methodologies for some of these chronic health effects are just beginning to be developed. Many existing chemicals with wide environmental exposure have never been tested for many of these chronic effects.

Sometimes subsequent discoveries have shown chronic effects can occur at very low doses for certain chemicals. For example, it has recently been determined that environmental exposure in the Great Lakes to chlorinated dibenzo-dioxins/furans is the probable cause of the failure of Great Lakes lake trout to reproduce -- a phenomena with dramatic negative ecological and economic consequences. This type of experience with environmental toxicants further emphasizes the need for cautious and conservative methods for human and ecological risk assessment procedures and for insisting on prudent preventative controls on emissions and exposures.

6.6 Developing Dose-Response Relationships

After reviewing both animal and human data (if it is available), toxicologists use a variety of models and safety factors to make their best judgement about the relationship between human exposure and risk of harmful effects.

For cancer-causing chemicals, a computer model that makes assumptions about the initiation and promotion of cancer may be used. One such model is called the linearized multi-stage model of carcinogenesis. This model assumes that there is there is no "safe" level of human exposure to a carcinogen other than zero exposure and that any exposure will potentially cause and increased risk of the incidence of cancer. Such models are used to draw conclusions about the risk of potential human exposure to a carcinogen at relatively low doses based on a statistical analysis of information about health effects shown from toxicological information at higher doses. The source of the high dose information may come from long term animal toxicology carcinogenicity evidence or from human evidence of carcinogenicity that may be available from workplace or other exposures.

The ultimate objective of this analysis is to arrive at a predicted human oral or inhalation exposure number called the "unit risk value" that is equivalent to producing a one in a million risk level of cancer induction for a person exposed continuously for a 70 year lifetime. The risks associated with such exposures can then be compared with other societal risks that are voluntary or involuntary.

For risk assessment for non-carcinogens, the toxicologists is concerned about finding a safe level of exposure that will not cause either acute or chronic health effects. In general, it is thought that non-carcinogens that do not display other types of chronic toxicity will have a threshold for exposure. If humans are exposed to a chemical concentration below such a threshold, no adverse health effects will occur.

In developing risk assessments for such materials, there will frequently be incomplete information on the effects of different types of exposures. The toxicologist must then develop an exposure model and draw conclusions about doses delivered by a different exposure route. For example, if the toxicologist has data only for acute effects in an animal exposed to a chemical via ingestion, the toxicologist must develop a model and safety factors that determines an equivalent dose in the animal if were instead exposed by through the lungs. Assumptions will sometimes be made about differing adsorption efficiencies to the body through different exposure pathways (i.e. through the lungs instead of through the intestine.)

Another important concept is the time duration of the exposure and its relation to the 'total body burden' of a chemical exposure that will result. Time duration is

important because it must be compared to actual human exposures that might occur in an environmentally realistic situation.

Safety factors for exposure route differences, species differences, adsorption differences and general uncertainty are used to make a conservative prediction of an acceptable human ambient dose exposure.

6.7 Pharmacokinetic Analysis

In the past several years, a specialty of toxicology has arisen known as pharmacokinetic analysis. Pharmacokinetic analysis is sometimes used to make arguments that a prediction of an acceptable ambient concentration for human exposure or a unit risk value for a carcinogen should be modified from values predicted by traditional analytical means. With phamacokinetic analysis, it might be argued, for example, that a biological process within a test animal that leads to a biological effect when exposed to a given chemical compound is not present in humans, or the biological effect is related to a metabolic breakdown product rather than the actual compound. As such, the pharmacokinetic analysis is used to derive alternate exposure limits and dose response relationships or to show that a traditional modeled method for predicting an exposure or risk number is really not applicable to humans for that chemical.

Pharmacokinetic analysis will frequently be done by an industrial applicant seeking to allow more community exposure to a chemical toxicant than would be permitted under a more usual and traditional toxicology analysis. For example, the pulp bleaching industry in some states has used pharmacokinetic analysis to seek a ten-fold increase in permissible human exposure on screening concentrations in the community for chloroform emissions.

6.8 Exposure Analysis

In order to determine potential risks from chemical exposure, use of dose-response relationships must be combined with an assessment of potential human exposures. The exposure analysis is used to determine such factors as maximum dose and/or most likely estimated dose over a specific time interval of exposure.

6.8.1 Direct Inhalation Exposure

The most common type of exposure analysis and the one most frequently done by government regulators of airborne toxicant emissions is for direct inhalation exposure. This type of exposure analysis relies on physical air quality modeling to relate emissions

from an industrial source to the maximum ambient concentration in the air at ground level. These predictions from air quality modeling are done for a given averaging time appropriate to the toxicity of the chemical compound or element in question (see also the section on air quality modeling). This process provides for a determination of an inhalation-only dose that would be received if a person were continuously exposed at ground level outside a facility for the entire time of the sample averaging time in question.

For example, predictions of maximum annual average exposure is generally compared to risk screening values for airborne carcinogens. Predictions of maximum one hour, 8 hour or 24 hour average airborne concentrations are used to compare to screening values for acutely toxic chemical compounds and elements.

The predicted maximum ambient concentration of the airborne toxicant is then compared to screening levels derived from dose response information to determine if there is a cause for concern.

6.8.2 Multi-Pathway Exposure Assessment

In multi-pathway exposure assessment, all potential pathways for pollutant exposure are analyzed, not just the inhalation route. Multi-pathway exposure assessment allows for estimation of an integrated total dose of a pollutant absorbed by a human receptor based on inhalation, ingestion and dermal exposure from air, water, food, skin contact, secondary ingestion of contaminated soils and other pathways.

In multi-pathway exposure assessment, an attempt is made to identify certain demographic groups which may be at particular risk because of their lifestyle, occupation and/or diet. For example, people who depend on subsistence fishing or who otherwise consume fish from recreational activity, farmers who consume a large portion of their food from their own cattle fed from grazing lands or from local fish ponds or other groups may be targeted for special analysis to determine whether individuals in such groups may be “maximum exposed individuals.”

Multi-pathway exposure assessment depends extensively on the use of physical and biological techniques for determining the environmental fate and transport of pollutants. These techniques are discussed in a subsequent section.

The ultimate objective of multi-pathway exposure assessment is to ensure that risk determinations that rely on such exposure assessments reflect the cumulative risk arising from these exposures integrated across all exposure routes and to ensure that special populations are protected.

6.9 Assessing Human Health Risks

When information has been compiled and analyzed to determine the dose-response characteristics of a particular chemical or elemental pollutant, and after an exposure assessment is made to determine the likely dose received for a given sampling time, an assessment of overall risk is possible.

For carcinogens, the objective is to determine the probability of cancer incidence for an individual who receives a maximum lifetime dose of the carcinogen under the worst case condition. A typical worst case condition for an airborne carcinogen would be an assumption that a person was continuously exposed for a seventy year lifetime at a point outside the company fence line showing the maximum annual average impact from that point source. A worst case condition for food ingestion might be that a person had a meal multiple times per week of contaminated fish.

The use of a worst case assumption is intended to give the risk assessment determination a strong level of conservatism and protectiveness. In other words, the analysis would be considered to err on the side of determining the maximum amount of exposure that might occur in practice.

For non-carcinogens, the objective is to determine that adverse effects from exposure will be unlikely and that the resulting exposure will not likely be a cause of illness or death.

If the risk of exposure is considered to be too high, or if the predicted exposure would be considered high enough to cause adverse health effects, then measures can be taken which will lower exposures. These could include lowering permissible emissions, conducting environmental remediation measures where they are feasible and taking measures to avoid the consumption of contaminated fish, drinking water or food.

6.10 Ecological Risk Assessment

While most of the discussion above has focused on human health assessment from toxicant exposures, an important new area of assessment involves ecological risk assessment. This type of assessment focuses on the biological fate of environmental toxicants and the effects of these pollutants aquatic and terrestrial ecological systems and biodiversity. Chemical toxicants which bioaccumulate in these aquatic and terrestrial ecological systems are especially important. Bioaccumulation occurs when creatures higher on the food chain, such as humans and predator species, gain dramatically increased exposure to a persistent toxicant as a result of consuming food species that tend to accumulate chemical toxicants in their bodies. In general, in the food pyramid, each higher predator species will gain a greater concentration of a persistent chemical toxicant.

Aspects of ecological risk assessment may also be important to multipathway human exposure assessment, particularly for fish consumption. In addition, aspects of ecological risk assessment will be particularly important for determining the effect of pollutant release on threatened and endangered species.

7. Standard Setting Organizations for Toxic Assessment and Health Hazard Evaluation

A variety of public and private organizations are involved in review of toxicology and epidemiological data. These organizations can have significant effects on how airborne toxicants will ultimately be regulated.

For carcinogens, the National Toxicology Program (NTP) and the International Association for Research on Cancer (IARC) publish lists of known and suspected human carcinogens. NTP is an interagency office at the U.S. federal level with participation by U.S. EPA, the Centers for Disease Control, the Occupational Safety and Health Administration, U.S. Food and Drug Administration and other federal agencies. Publication of a chemical as a carcinogen on either the NTP and/or IARC list will trigger certain risk assessment and control technology requirements. NTP and IARC will sometimes publish "unit risk" values for airborne or ingestion exposure to such chemical carcinogens that constitute the "one in a million risk level" for human exposure to such chemical carcinogens.

U.S. EPA publishes influential guidelines for risk assessment for chemical carcinogens, neurotoxicant agents, reproductive toxicants and for ecological risk assessment. EPA also publishes recommendations called "Reference Doses" and "Reference Concentrations" which are deemed to be acceptable, long term exposure guidelines for the public which are thought to be considered safe exposure levels. EPA recently concluded a consensus stakeholder advisory committee process to develop testing and screening guidelines to assess endocrine disrupting effects for human chemical exposure, although this type of assessment has not yet had a significant effect on the development of acceptable human exposure regulations..

The American Conference of Governmental Industrial Hygienists (ACGIH) publish "threshold limit values (TLV), " which are airborne concentrations of chemical pollutants which are deemed to be safe for healthy workers to be exposed to for 8 hours per day for a full working life without adverse health effects. These TLV recommendations often serve as important recommendations for future regulation by the Occupational Safety and Health Administration. TLVs have frequent use in state airborne toxicant regulations. The National Institute for Occupational Safety and Health (NIOSH) which is part of the Centers for Disease Control (CDC) also publishes

recommended maximum workplace exposure guidelines which are also sometimes referenced in state air toxicant rules.

Some state air pollution programs, such as California, Wisconsin, Texas and Michigan, also publish lists of acceptable ambient concentrations of airborne toxicants. Many of these efforts are supported by toxicology staff in the various state regulatory programs.

8 Statutory and Regulatory Tools to Control the Health and Environmental Effects of Airborne Toxicant Emissions

As noted above, control programs for airborne toxicants need to prevent detrimental public health impacts, to take precautions to avoid environmental damage in the face of incomplete information about environmental effects and to protect the public trust in the air and the natural environment. These goals can be carried out within the framework of existing federal and state legislation, but effective citizen environmental advocacy is needed to ensure the development of programs that are sufficiently protective.

8.1 The Federal Clean Air Act

In 1990, Congress adopted the most recent amendments to the Federal Clean Air Act. A major feature of the 1990 Amendments was to significantly strengthen the federal program to control airborne toxicants.

Section 112 of the Federal Clean Air Act provides the federal framework to control 189 federally designated airborne toxicants. Under Section 112(d) of the Act, EPA is required to issue regulations providing for Maximum Achievable Control Technology (MACT) for several emission source categories. MACT standards for existing sources of federal hazardous pollutants are supposed to reflect the level of emission control performance achieved by the average of the best performing 12% of existing sources in a given source category. For new sources, MACT regulations are supposed to provide a level of emission control performance equivalent to the best performing source. If EPA has not yet set a MACT standard for a source category, the Federal Act requires that states must impose the same requirement on a case by case basis for new and reconstructed sources of hazardous air pollutants during permit reviews..

Under Section 112, EPA has until 2008 to review risks to health and environment remaining after the imposition of MACT standards. If these risks exceed certain target levels, EPA is then empowered to issue health-risk-based emission limitations to control these residual risks.

8.2 State Airborne Toxicant Control Programs

Although the Federal Clean Air Act provides a significant control program for the 189 federally designated hazardous air pollutants (HAPs), other toxic air contaminants are not addressed by this federal effort. In addition, even for the federal HAPs, the only comprehensive environmental and health risk assessment that will take place is scheduled for many years in the future. State environmental and risk assessment of residual risks even for emissions controlled under the federal program is highly desirable given the long delay in federal risk assessment activities.

If an airborne toxicant is not on the federal list, it will be unregulated unless a state takes action to control it. Although airborne toxicants that are not on the federal HAP list may possibly be subject to controls for volatile organic compounds that contribute to smog and for particulate matter, they controls do not address the toxicity of the specific emitted pollutants. In relatively clean areas of many states, uncontrolled emissions of toxic air contaminants will be allowed for new and existing emission sources unless a state takes an initiative to impose control requirements, either via permit conditions or through rulemaking.

States that enact rules providing for minimum emission control technology requirements on airborne toxicants and comprehensive risk assessment and risk screening requirements can significantly reduce health and environmental risks from these otherwise unregulated toxic air contaminants.

8.3 Federal Clean Water Act Controls on Non-point Source Toxic Pollution; the Emergence of the Total Maximum Daily Load Program to Control Indirect Atmospheric Inputs to Impaired Water Bodies

Under the Clean Water Act, states and the EPA must go through a process of designation of impaired water bodies that do not meet water quality standards or have impaired uses from pollutants. After the impaired water bodies are listed, Total Maximum Daily Load (TMDL) plans must be developed to control both point and non-point sources of water pollutants.

In the Great Lakes region, pollutants such as mercury and chlorinated dibenzo-dioxins have caused impairments in inland lakes and in the Great Lakes. Most of the mercury problems have arisen as a result of non-point source atmospheric pollutant inputs from both nearby and distant pollution sources. These problems have resulted in extensive contamination of fish and wildlife that threatens public health and wildlife. The public hears about these problems as a result of extensive fish consumption advisories issued by state and federal governmental agencies.

TMDL plans to address these non-point, atmospheric inputs will need to be developed. Air quality deposition models and both local and regional atmospheric emission inventories will need to be developed. TMDLs for these impaired water bodies must ultimately set targets for rolling back these atmospheric inputs. The model results and the inventory efforts will be used to derive needed reductions in source stack emissions of mercury and other persistent, bioaccumulative toxicants. The TMDLs will then be used to enact permit emission limitation controls and/or new air pollution control rules to achieve such roll-back requirements.

Although TMDLs have been developed for some traditional water pollutants and impaired water bodies, relatively little activity has taken place to date on atmospheric input-related TMDL development. U.S. EPA is presently engaged in a pilot demonstration program for an air deposition-related TMDL for Devil's Lake near Madison WI for non-point source mercury control.

8.4 The Strategy of Using Technology-Based Emission Controls and Environmental/Public Health Assessment of Residual Risks

A number of approaches, each complimentary to the other, are frequently used to control toxic air contaminants. These involve emission standards, work practices, health based inhalation ambient air standards and standards that arise from the consequences of atmospheric deposition and consideration of multi-pathway risk assessment and analysis.

Under the public health "prevention paradigm," the precautionary principle and the public trust doctrine, environment and public health organizations generally advocate that pollution prevention measures and technology-based standards must first be imposed on airborne toxicant emission sources. Next, "residual risks" associated with the expected community, public health, long range transport, airborne deposition and environmental impacts are evaluated. If the "residual risks" are too high and/or if they exceed threshold criteria on environmental/public health effects or threaten sustainability, then additional, more stringent health/environment-related emission controls are imposed. In difficult cases, under the precautionary principle, a ban on emissions or process use of a material is imposed (i.e. the current ban on open process uses of poly-chlorinated biphenyls).

8.5 Technology-Based Toxic Emission Limitations

The most basic approach to limiting airborne toxicant emissions involves writing legally enforceable rules, permit limitations or agency orders to limit emissions from discharge stacks (known as "point sources") or from "fugitive sources" (emissions that don't come from discreet stacks). These limitations can be written in a couple forms – as controls on the amount of mass of a pollutant discharged in a given time (most commonly

pounds/hour), controls on the mass of emissions per volume unit of waste gas discharged, or finally controls on the amount of pollutant emitted per unit of production process output.

Technology-based emission limitations are generally written on the basis of what an industrial process emits, the efficiency of pollution control equipment available and whether there are opportunities for process changes that limit emissions – known as "pollution prevention" or "toxics use reduction."

These "technology based" emission limitations can range from lenient to stringent according to the legal basis of the standard, and how the setting of the emission standard depends on considerations of environmental risks, costs of control, the availability of "technology transfer," and the prevailing emission control practices in an industrial sector. The most stringent approaches to technology based emission standards involve "best available control technology" and "lowest achievable emission rate" approaches.

8.6 Work Practice Controls

Sometimes, as in the control of asbestos fibers during demolition, it isn't practical to write specific emission regulations. Then a "work practice standard" is enacted to ensure that a particular operation is done in a particular manner that is expected to limit emissions to the environment. Other examples of "work practice standards" include requirements for "good combustion control" in waste incinerators and requirements that solvent degreasing processes operate with closed tops.

8.7 Direct Inhalation Risk and Standards

The first type of health-based toxic air pollution standard involves setting limits to protect health from direct inhalation exposures outside company fence-lines to which the public has access. These so-called "ambient limits" are designed to ensure that the average ground-level air concentration of a toxic pollutant is not exceeded when measured or modeled over a certain time period, called an "averaging time." Averaging times are typically set as either one hour, eight hour, 24-hour or annual averages.

For most health-based ambient limits, such limitations are not directly enforced or monitored. Instead, a air quality models are used to make a prediction of the numerical relationship between emissions of a toxic pollutant (in grams per second) and the predicted maximum ambient toxic air pollution concentration at ground level under worst case weather conditions.

If the prediction shows that a health-based ambient limit is met when the source is discharging at maximum capacity under a technology-based limitation, then no further emission limitation is imposed. If the health-based limit is not met, however, then the emissions must be controlled more stringently and the source is subject to a more stringent health-based permit emission limitation to ensure that the ambient limit will not be exceeded.

The setting of the actual health-based ambient limit depends on the nature and toxicity of the particular hazardous pollutant in question. Many times air quality officials borrow the use of occupational health protection criteria known as threshold limit values (TLVs) that are published by the American Conference on Governmental Industrial Hygienists (ACGIH). For environmental carcinogens (materials known or suspected of causing cancer in human beings), a prediction of risk of cancer incidence is made using statistical models and human epidemiology and animal toxicity tests.

8.8 Environmental Fate and Transport Analysis Combined with Multi-Pathway Exposure and Cumulative Risk Assessment

Another type of risk assessment that may eventually lead to the development of standards of maximum permissible risk associated with multiple paths of exposure. Multi-pathway exposure assessment and cumulative risk assessments are particularly important for persistent, bioaccumulative airborne toxicants, such as chlorinated dibenzo-dioxins/furans, polychlorinated biphenyls, mercury, arsenic and other heavy metals. For these persistent, bioaccumulative toxicants, consideration of just inhalation risk is likely to significantly underestimate the cumulative health and environmental risk associated with such emissions.

Multi-pathway risk assessment for persistent, bioaccumulative toxicants is significantly more complex than just determining inhalation exposure and risks. Environmental fate and transport analysis must be used to examine any potential chemical conversion, transport and deposition in the environment of the particular toxicant in question. Any buildup in soils and water from subsequent atmospheric deposition is then determined over time. Risks from exposure from all pathways are analyzed, including ingestion of food, fish, incidental particulate/soil ingestion, etc. Sensitive receptor analysis is done for particular demographic groups, such as subsistence farmers using fish ponds or consuming local farm produced milk and/or beef. The cumulative risk and/or health effect is determined from all of the exposures and compared with policy guidance or screening levels. For environmental carcinogens, the goal is generally to show that expect cumulative risks will not exceed a range of one to ten in a million for cancer incidence as a result of exposure from all such pathways.

8.9 The Issue of Uncertainty and Incomplete Knowledge -- Safety Margins, Residual Risks and Sensitive Population Subgroups

Considerable uncertainty often occurs in developing risk assessments for potential emission sources. It is important for citizen environmental advocates to emphasize that risk assessment procedures be done in a conservative manner. Conservative assumptions should reflect worst case analysis if the risks assessment is intended to be used as a guide for setting emission limitations to protect public health and the environment.

Where state and federal rules countenance the use of risk assessment in making decisions that will ultimately affect emissions and the protection of public health and environment, the method employed should always encourage the submission of better, more refined and definitive toxicology and exposure assessment information. Where there is little or no information about the toxicity of a material, default presumptions should ensure stringent controls of emissions for health-based protections.

Stated goals for emission reduction should emphasize protection of sensitive population subgroups from excessive risks. Such population subgroups include children, pregnant women and their fetuses, the elderly and persons with compromised respiratory or immune systems.