P R O C E E D I N G S

EPA REGION 9 AIR TOXICS CONFERENCE

September 13 and 14, 1983
Sheraton Palace Hotel
San Francisco, CA

TABLE OF CONTENTS

EPA Region 9 presents an

AIR TOXICS CONFERENCE

September 13 and 14, 1983

Sheraton Palace Hotel

San Francisco, California

Co-Sponsors

United States Environmental Protection Agency
California Air Pollution Control Officers Association
California Air Resources Board
League of Women Voters of California

FINAL AGENDA

Conference moderator: David Howekamp
Director, Air Management Division
EPA, Region 9

September 13, 1983

8:30 Welcome and introduction
John Wise - Acting Regional Administrator
EPA, Region 9

AIR TOXICS PROGRAMS AND POLICIES

8:45 EPA's regulatory process for air toxics
David Patrick - Chief, Pollutant Assessment Branch
EPA, Research Triangle Park, NC

9:30 California Air Resources Board's framework for air toxics
Michael Scheible - Chief, Office of Program Planning, Evaluation, & Coordination
California Air Resources Board

10:15 Break

10:30 California Department of Health Services carcinogen policy
Dr. Kim Hooper - Research Scientist
California Department of Health Services

11:15 Lunch

AIR TOXICS CONFERENCE

September 13, 1983

BACKGROUND INFORMATION ON AIR TOXICS

12:30 Ambient monitoring for air toxics in Region 9
Dr. Hanwant Singh - Director, Atmospheric Chemistry Program
SRI International

1:15 Sources of air toxics in California
G. C. Hass - Chief, Haagen-Smit Laboratory Division
California Air Resources Board
Terry McGuire - Assistant Division Chief, Stationary Source Control Division
California Air Resources Board

2:00 Break

2:15 Air toxics in the indoor environment
Dr. David Grimsrud - Co-Leader, Building Ventilation and Indoor Air Quality Program
Lawrence Berkeley Laboratory
Dr. Ken Sexton - Director, Indoor Air Quality Program
California Department of Health Services

3:30 A study of the relationship between cancer incidence and air pollution in Contra Costa County, California
Dr. Donald Austin - Chief, Resource for Cancer Epidemiology and California Tumor Registry
California Department of Health Services

September 14, 1983

AIR TOXICS CASE STUDIES

8:30 Air emissions from a former disposal site
Kathleen Shimmin - Chief, Field Operations Branch
EPA, Region 9

9:15 Emissions from the ASARCO copper smelter in Tacoma, Washington
Alexandra Smith - Director, Air & Waste Management Division
Mike Johnston - Chief, Air Operations Section
Dana Davoli - Environmental Scientist
EPA, Region 10

10:00 Break

AIR TOXICS CONFERENCE

September 14, 1983

10:15 Toxic air pollution in Henderson, Nevada
Michael Naylor - Director, Air Quality
Clark County Health District

11:00 Emissions from the semiconductor industry
Milton Feldstein - Air Pollution Control Officer
Bay Area Air Quality Management District

11:45 Lunch

1:00 Volatile organic emissions from landfills
Ed Camarena - Director, Enforcement Division
South Coast Air Quality Management District

1:45 Asbestos decontamination in Globe, Arizona
David Chelgren - Manager, Compliance Section
Arizona Bureau of Air Quality Control

2:30 Break

PANEL DISCUSSION

2:45 Panel moderator: Dr. Herschel Griffin - San Diego State University

Panel members:
Milton Feldstein - California Air Pollution Control Officers Association
Jeanne Harvey - League of Women Voters of California
Maureen Lennon - Atlantic Richfield Company
David Patrick - Environmental Protection Agency
Michael Scheible - California Air Resources Board

4:30 Closing remarks-EPA, Region 9

EPA'S REGULATORY PROCESS FOR AIR TOXICS

David Patrick
Chief, Pollutant Assessment Branch
U.S. Environmental Protection Agency
Research Triangle Park, NC

EPA'S TOXIC AIR POLLUTANT REGULATORY PROCESS

The principal authority under the CAA for control of toxic air pollutants is section 112, entitled National Emission Standards for Hazardous Air Pollutants. Section 112 defines "hazardous pollutant" as an "air pollutant to which no ambient air quality standard is applicable and which in the judgment of the Administrator causes, or contributes to, air pollution which may reasonably be anticipated to result in an increase in mortality or an increase in serious irreversible, or incapacitating reversible, illness." Regulations under section 112 must be established at a level to protect the public health with an "ample margin of safety." Section 111 (New Source Performance Standards) also provides for control of pollutants which adversely affect human health or welfare but are not "hazardous" as defined under section 112. Seven pollutants have been listed as hazardous under section 112 and to date emission regulations have been promulgated to control four. Regulations for the remaining three hazardous air pollutants have been proposed. One additional pollutant has been regulated under section 111 for health reasons.

EPA's activities in dealing with these pollutants have been, as I am sure you know, a matter of considerable debate both within the Agency and outside. We attempted to articulate a decision-making policy in 1979 by proposing the Airborne Carcinogen Policy. Recently, we have been attempting to develop a more general Air Toxics Policy. While there is clear agreement that resolution in the near future is important, the issues and options remain broad and complex. Let me describe some of them first.

In evaluating the extent of the toxic air pollutant problem, we must first determine whether pollutants emitted to the ambient air pose significant risks to public health. Available ambient exposure and health effects information and the scientific interpretation of that information do not allow a clear-cut, absolute determination of the extent of the health risks associated with toxic air pollutants for the nation as a whole. The magnitude of the toxic air pollutant problem in terms of number of pollutants and sources of emissions also is difficult to determine with precision. A 1976 EPA survey of the organic chemical industry identified over six hundred commercially important chemicals, of which about 50 were identified from preliminary health information or production volume as possible toxic air pollutants requiring more detailed assessment. Many source categories other than those in the organic chemical industry also may be significant emitters of toxic air pollutants. These sources include mining, smelting, refining, manufacture and end-use of minerals and other inorganic chemicals; combustion; petroleum refining, distribution, and storage; solvent usage and disposal; mining, processing, use and disposal of radioactive substances and radioactive by-products; waste treatment, storage and disposal facilities; and various sources of non-toxic emissions which are chemically transformed into toxic air pollutants in the atmosphere. Notwithstanding, while the existence of significant widespread risks resulting from exposure to ambient concentrations of toxic air pollutants is the subject of considerable scientific debate, clearly there are individuals that are at increased risk from exposure to relatively high concentrations of air pollutants that may be toxic and are emitted from uncontrolled or partially controlled sources. In addition, there is the concern that exposures to these pollutants at low

levels may result in chronic adverse effects which may not become evident for many years.

EPA is concerned with all human health effects that could result from exposure to toxic air pollutants, although cancer is of special concern because of the high incidence of mortality associated with it. While the total number of cancer deaths each year is well known, the contribution of air pollution to this total is uncertain. Ambient air pollutants generally are believed to rank well below smoking, occupational exposure, and diet as an incremental cause of cancer, although the risk associated with voluntary personal habits, such as diet and smoking, tends to be of lesser concern than that resulting from involuntary exposures to air and water pollution.

As I mentioned earlier, there are two principal alternatives provided under the Clean Air Act for dealing with emissions of toxic air pollutants from stationary sources: section 112 and section 111.

Section 112 has been considered in the past to be the primary statutory mechanism for controlling toxic air pollutants. However, a major issue complicating its implementation is the establishment of toxicity to humans based on uncertain mathematical extrapolation from high-dose animal tests or occupational exposure to low-dose public exposure at ambient air concentrations. Another is identification of the appropriate level of emission controls for pollutants for which health effects thresholds have not been demonstrated. In other words, what is an ample margin of safety for a carcinogen?

There also is considerable uncertainty with exposure estimation because of the difficulty in obtaining precise data on long-term emission rates, atmospheric dispersion patterns and population concentrations around individual sources, and because of the lack of information on short-term

and long-term movement (migration) of people and indoor versus outdoor toxic air pollutant concentration patterns. Further, ambient monitoring data are limited and would be both very costly and time consuming to obtain for use in exposure assessment. Finally, there are uncertainties concerning exposure to multiple pollutants and to a single pollutant from multiple sources, and the possibility of synergistic actions and heightened susceptabilities to some cancers by some population groups. These factors make it difficult if not impossible to determine, or even estimate with any confidence, the real magnitude of the risk to human health based on the available data or to establish any epidemiological association between cancer and public exposure to ambient concentrations of a specific substance.

Finally, section 112 does not mention economics. Thus, a literal interpretation of section 112 would require zero emissions to achieve zero exposure to non-threshold pollutants. As I am sure you know, zero emissions requirements would likely result in widespread industry shutdown. We do not believe Congress intended that.

Principally because neither the language nor the legislative history of the Clean Air Act provide any specific Congressional intent on these issues, it has been difficult to establish definitive criteria for the evaluation and control of toxic air pollutants under section 112. Administrative, legal, and legislative requirements, coupled with a lack of acceptable criteria for decision-making, have resulted in an evaluation process that can take from 5 to 7 years from initial identification to promulgation of regulations. As a result of this lengthy process, while many substances are under evaluation as possible toxic air pollutants, few have reached the final decision stage.

The air toxics policy, that I mentioned earlier is under development, attempts to respond to these issues and concerns in the following way. In general, it would continue use of section 112 to control air toxics which are clearly "hazardous" in the sense that exposure at ambient levels may reasonably be anticipated to result in an increase in mortality or serious illness. However, where population exposure, the number and location of sources, or the estimated health risks warrant consideration of other sections of the Clean Air Act, or where the use of other legislative authorities or nonregulatory control options are clearly indicated, the process provides the flexibility to use these alternatives. In order to ensure that public health concerns are dealt with in the most timely and efficient manner, and in order to optimize resource use, the process provides for several levels of increasingly detailed analysis. At each level, decisions are made to ensure that the pollutants which receive the most detailed and resource intensive analysis are the most important to public health.

More specifically, candidate pollutants would be identified periodically and then ranked using available health and source information. The highest ranked candidates would be screened to assess their potential for health risks at ambient exposures. One of the following actions then would be taken: (1) Where information is not adequate to determine the appropriate next step, that information would be gathered. (2) Where there clearly is no significant risk to public health, the candidate would be dropped from further active consideration. (3) Where source categories of a toxic pollutant clearly pose a health concern but can be dealt with more efficiently with using a statute (e.g., Federal or State) other than the Clean Air Act,

jurisdiction would be transferred to the appropriate program. (4) Where information warrants, the candidate would undergo comprehensive health and exposure assessment.

Development of the comprehensive health assessment document is a detailed and resource intensive process that normally leads to formal Science Advisory Board review in public meetings and closure by the Board when it is satisfied that the document is scientifically sound and adequately represents the latest scientific knowledge. Following closure, this document along with other relevant information is provided to the Administrator and all feasible control options are identified.

Several responses are possible at this point: (1) Where Federal regulatory action is appropriate, the necessary legislative authority would be implemented. (2) Where State/local, voluntary or other non-Federal actions are appropriate, EPA would provide necessary technical or support information. (3) Where public health needs indicate that further action at this time is not required, activities would be halted and the candidate placed in a category for periodic reassessment.

Under the Clean Air Act, the process for developing standards under §112 begins with the listing of a hazardous air pollutant. Source categories that result in significant risks would be evaluated to determine those for which proposal of regulation is appropriate and those for which proposal is not. The basis for control using this approach would be best available technology, or BAT, with additional control applied if the risk remaining after application of best available technology is determined to be unreasonable.

By BAT, EPA means the best control available, considering economic, energy, and environmental impacts. BAT may be different for new and existing sources within a source category and may be equal to or more stringent than the best technology defined for New Source Performance Standards under §111. Whether a source category is estimated to cause a significant risk would be decided in light of the estimated risks to individuals, and the estimated cumulative risks to populations affected by that source category. Whether the estimated risks remaining after application of BAT are unreasonable would be decided in light of a judgmental evaluation of the estimated maximum lifetime risk and cancer incidences per year remaining after application of BAT, the impacts, including economic impacts, of further reducing those risks, the readily available benefits of the substance or activity producing the risk and the availability of substitutes and possible health effects resulting from their use. In all cases where estimated risks are used, the significant uncertainties associated with those numbers would be weighed carefully in reaching the final decision.

In this approach, the use of risk numbers generally is confined to areas of broad comparisons, e.g., in selecting source categories to evaluate and in assessing the incremental change in risk that results from application of various control options. The use of risk numbers in an absolute sense is avoided because of the many uncertainties.

Obviously, there are legitimate concerns with this approach, particularly in its limited use of risk assessment and predominant use of technology and cost. However, this approach has been generally followed for several reasons: (1) we did not have to rely on very uncertain risk estimates, (2) we are able generally to precisely quantify technology and cost, and (3) it

provides consistency with section 111. Then, in June the Administrator spoke before the National Academy of Sciences and issued a call for a more rational system for assessing and managing risks to the American public. Mr. Ruckelhaus stated, among other things, that risk assessment should be improved, that risks should be weighed against the benefits of continued use and the risk of substitutes and environmental transfer, and that the public should be involved in risk management to a greater extent.

In response to this, we have begun to explore other decision-making criteria and procedures. Our goal is to expand the use of risk assessment in order to relate regulation more directly to public health concerns, add across pollutants and sources more consistency to regulations and their effects, and provide more balance in benefits and costs. Clearly these concerns must be addressed quickly since regulations for benzene, radionuclides and arsenic now have been proposed.

Some options being considered are the following:

1. Specify risk number cutoffs to eliminate source categories from consideration for regulation.

2. Adopt target after control risk number levels.

3. Use population density around sources to assist in determining the extent and level of control.

4. Orient regulation more specifically to individual sources.

Several other aspects of our toxic air pollutant program should also be of interest to you. First, EPA is beginning to take a more active role in working with EPA Regional Offices and State/local air pollution control agencies on toxic air pollutants. This is appropriate because of the widespread and growing interest in toxic pollutants at the State/local

level. For example, regional workshops like this one for State and local officials were held last year in Boston, Atlanta, and Philadelphia to consider a wide range of toxics issues and problems. Others are being planned. We have also begun to develop an Air Toxics Clearinghouse to provide pertinent information to State and local agencies on sources, emissions and control information, health summaries, exposure assessment methodologies, monitoring information and regulatory progress in other State and local areas. We are working closely with STAPPA and ALAPCO in developing this Clearinghouse. Next, EPA recently completed a detailed assessment of the eight most active State and local air pollutant control programs, and this report was circulated to State and local program offices. A follow-up questionnaire was sent out by STAPPA and a final report is expected from them soon summarizing its results. I think a major new step is the recent start-up of the first of 10-12 planned air toxics monitoring centers. It is located in Philadelphia and will provide our first opportunity to begin obtaining long-term toxics trend data and to develop and test sampling and analytical mehtods for potentially toxic air pollutants.

Research also is on-going by EPA in several other areas. These include basic health effects research, such as the evaluation of the mechanisms and effects of potential air toxics on humans, atmospheric fate and transport studies, development of control technologies, sampling and analytical techniques and ambient monitoring.

In a related area, study and control of air toxics must mesh properly with other environmental control programs. Of principal interest are the interfaces with the toxic water pollutant program under the Clean Water Act, the hazardous waste program under the Resource Conservation and Recovery Act, and the hazardous substance control efforts under the Superfund

legislation. In each of these programs, there is an air pollution component, particularly with volatile organic compounds, and we are beginning to interface more closely with these programs.

In conclusion, we have a growing toxic air pollutant evaluation and control program. ~~We are~~ EPA is proceeding to streamline the process for evaluation and control of air toxics and to articulate this process to the public, Congress, industry, and environmental groups so that everyone will understand how ~~we~~ they intend to fulfill ~~our~~ their mandate to protect the public health from toxic air pollutants. We take this mandate very seriously, and I appreciate the opportunity to discuss this important program. This conference provided ~~with you today~~.

CALIFORNIA AIR RESOURCES BOARD'S

FRAMEWORK FOR AIR TOXICS

Michael Scheible
Chief, Office of Program Planning, Evaluation, and Coordination
California Air Resources Board
Sacramento, CA

Summary of Health and Safety Code Section 39650 et seq (AB 1807 of 1983) Relating to Toxic Air Contaminants

Section 39650. INTENT. Establish a program to identify and control toxic air contaminants so that the public health is protected.

Section 39655. DEFINITIONS.
1) Toxic air contaminant = air pollutant which may cause or contribute to increased mortality or serious illness or pose present or potential health threat. Includes NESHAP substances.
2) Airborne toxic control measure = recommended methods to be used by districts to reduce emissions of toxic air contaminants.

Section 39660-62. SUBSTANCE IDENTIFICATION PHASE (risk assessment).
1) ARB requests DOHS evaluation of substance's health effects.
2) DOHS submits written evaluation and recommendations within 90 days (30 day extension).
3) ARB prepares report with DOHS participation.
4) Scientific Review Panel considers report and submits written findings to ARB within 45 days (15 day extension).
   a) If panel finds deficiencies, ARB has 30 days to revise and resubmit.
   b) ARB prepares hearing notice and proposed regulation within 10 days after panel reports.
5) Public hearing.
6) ARB determines that substance is a TAC and specifies threshold if applicable.
7) DFA in charge of pesticide health effects evaluation and designation.

Section 39665-67. CONTROL DECISION PHASE (risk management).
1) ARB reports on need and degree of emission control; districts participate, affected parties and public consulted.
   a) If threshold, control to threshold; if no threshold, reduce risks.
   b) ARB must consider technology, cost, risk and adverse environmental impacts.
2) Public hearing (45 day notice).
3) ARB to use existing rulemaking authority for controlling mobile sources or motor vehicle fuels.
4) ARB adopts airborne toxic control measure to guide district action on stationary sources.
5) Districts propose regulations within 120 days of ARB adoption of measures.
6) Districts adopt rules within 6 months of step 4).
7) Seasonal food/fiber processors exempt from district NSR rules until 1987.
8) DFA in charge of pesticide controls.

Section 39670. SCIENTIFIC REVIEW PANEL.
1) Consists of 9 members: 5 appointed by the Secretary of Environmental Affairs, 2 by Senate Rules, 2 by Assembly Speaker, from list supplied by the President of U.C.
2) Areas of expertise, qualifications, terms (3 years) and disclosure requirements specified.
3) ARB, DOHS, DFA provide staff support.

Section 39674. PENALTIES. $10,000/day if violation of emission or other condition.

A14 The Sacramento Bee • Thursday, October 28, 1982

Sharp Debate on ARB's Toxic-Air Proposal

Board Says Hearing 'Only A First Step,' Calls For Further Discussion

By Max Miller
Bee Staff Writer

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Jobs Versus Cancer

What Cost a Life? EPA Asks Tacoma

L.A. Times

By ELEANOR RANDOLPH
Times Staff Writer

TACOMA, Wash.

Does bad air cause cancer?

By ROBERT LOCKE
AP Science Writer

LOS ANGELES (AP) — The California Air Resources Board will take its first formal step next month toward controlling now-unregulated cancer-causing chemicals mixed in the air of urban air, says the chairman. said Monday during a

and carcinogens to the federal Environmental Protection Agency, told the hearing "there is a sound scientific reason for public concern about carcinogens in the air."

e urged the ARB "to set priorities for identifying carcinogens so the worst will be dealt with ." But, he added, "the proposed regulations do conform to scientific methods of identifying carcinogens."

Dr. Roy Albert, professor of en medicine at New York Univer the EPA's Carcinoge urged the board to group and to gi considerin said

He urged that the ARB re include criteria consiste concepts.

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worked with asbestos decades ago are now dying of lung cancers and other maladies.

the said targeted chemicals clude not only those to cause cancer also any sub- strongly,

necessary risk of cancer people die caused by substances are breathing in the want to minimize the she said. "This is controversial millions of dollars Her news confe after weekend re an ARB study concentrations cinogens in Los Angeles Cour times gre

Panel OKs Bill That Targets Airborne Toxic Chemicals

By Thorne Gray
Bee Capitol Bureau

A bill that would launch a long-delayed attack on airborne toxic chemicals won approval Tuesday in the Senate Committee on Govern-

scientists to identify some of them four years ago after deciding the Environmental Protection Agency was moving too slowly on the problem.

The toxics in question include arsenic from wood pre- smelters, cadmi- smelters,

LA study finds many cancer agents in air

News Enterprise 9/28/82

By ROBERT LOCKE

LOS ANGELES (AP) — Contending urban air is laced with myriad cancer-causing chemicals, hairman of the state said yester-

lem until incontrovertible evidence is in. "We don't want to wait like we did with asbestos workers in the shipyards and (take no action) until they are dying of cancer" — as thousands of people who worked with asbestos decades ago are now dying of and other maladies

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Air Board Moves to Bar Carcinogens

L.A. Times 5/28/82

By RICHARD O'REILLY,
Times Staff Writer

NAPA, Calif. — Despite opposition from the chemical and petroleum industries, the state Air Resources Board on Thursday ordered its staff to prepare a program aimed at eliminating atmospheric emissions of

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State air board prepares to regulate toxic chemicals

By Don Bruning
Herald Examiner staff writer

DAVIS — To fill the vacancy left by diminished federal regulations, the California Air Resources Board is preparing to begin regulation of chemicals put into the air and water that are known to cause cancer and birth defects in humans. A board spokesman

so many chemicals known to cause cancer in animals that it will never be possible to do long-term studies on all of them in people. These scientists, pointing to the fact that cancer is a disease that proceeds in complex stages, said that a human being could go on for years enduring all the insults of the environment to his body

limited or even reversed.

"This conference at UC Davis will probably set the tone for what the board does."

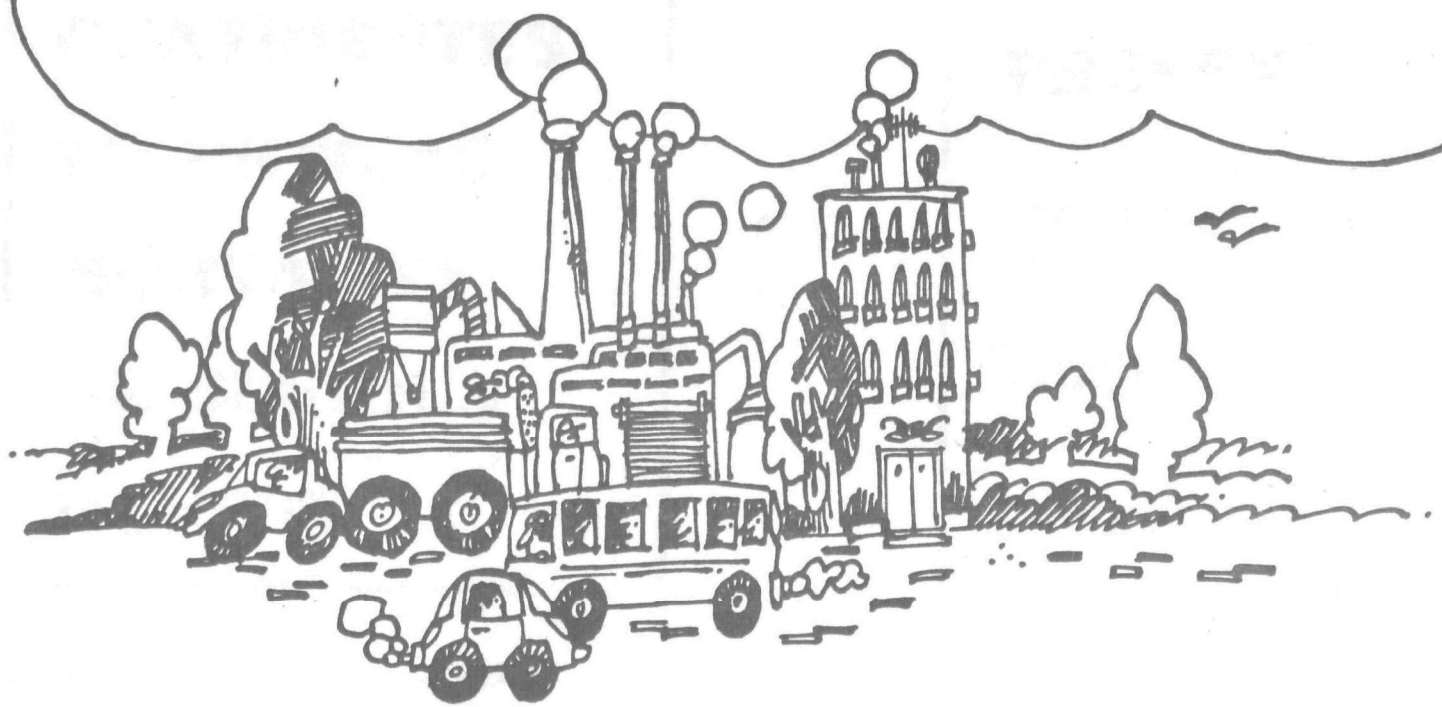
The board has the overview of air and water pollu

* **Why are toxic air contaminants important?**

* **What is the ARB approach?**

* **What is happening now?**

TOXIC AIR CONTAMINANTS
AND
TRADITIONAL POLLUTANTS



TOXIC AIR CONTAMINANT

CAUSES OR CONTRIBUTES TO INCREASED MORTALITY OR SERIOUS ILLNESS

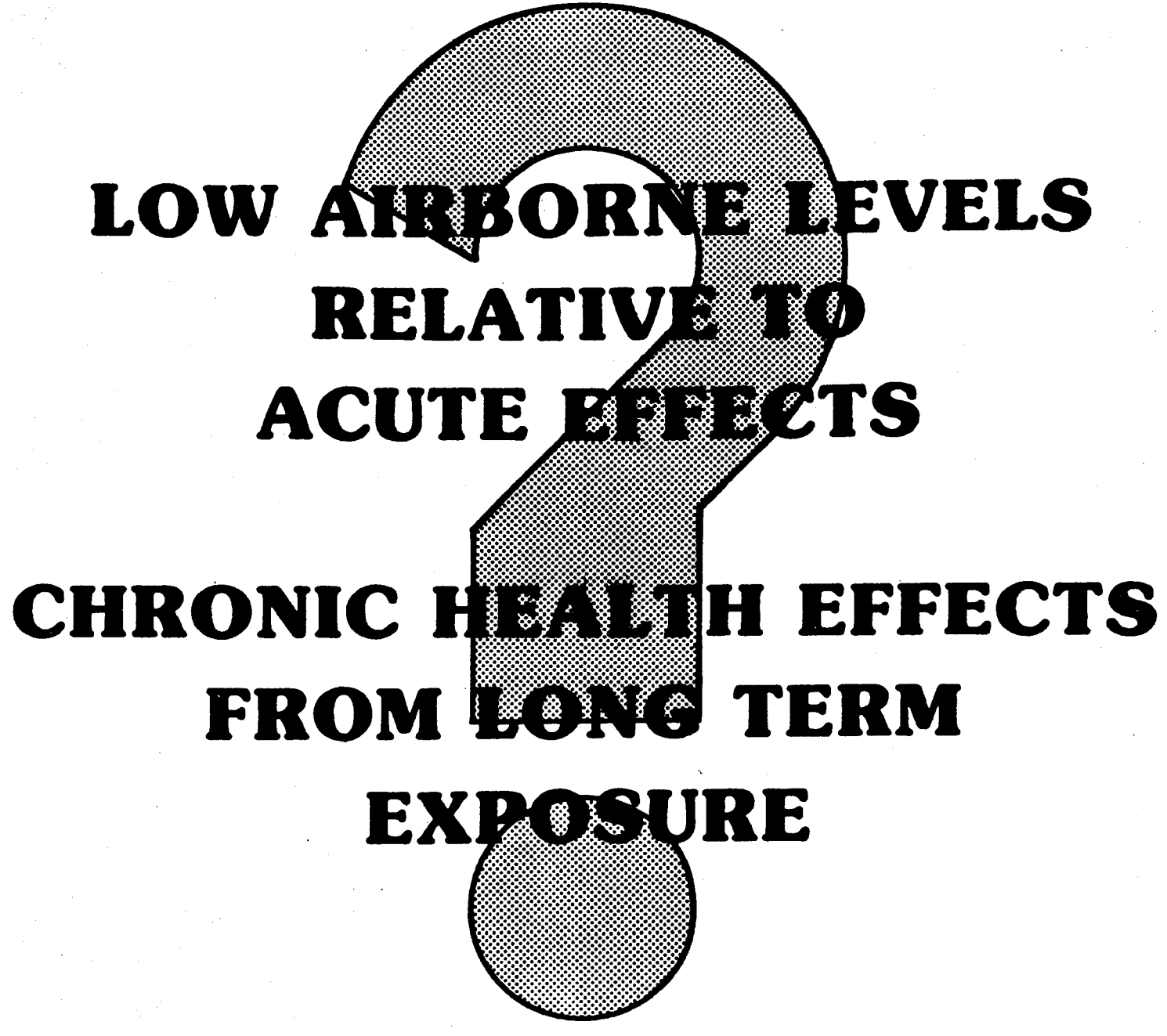
OR

MAY POSE PRESENT OR POTENTIAL HUMAN HEALTH HAZARD

INCLUDES NESHAP's POLLUTANTS

Traditional Pollutants*	Toxic Air Contaminants
Few (6)	Potentially Numerous
Not Bioaccumulated	Some may Bioaccumulate
Lung Primary Target Organ (Except CO)	Many Target Organs
Readily Available Human Health Effects Data	Dose-Response Data For Humans Rarely Available
Effects Generally Occur From Minutes to Months	Effects Generally Occur After Long Latent Period (Years)

* As regulated under Clean Air Act, except Lead


LOW AIRBORNE LEVELS
RELATIVE TO
ACUTE EFFECTS
CHRONIC HEALTH EFFECTS
FROM LONG TERM
EXPOSURE

SUSPECTED AND KNOWN TOXIC SUBSTANCES

CATEGORY	APPROX. NO. OF SUBSTANCES
NIOSH REGISTRY OF TOXIC EFFECTS OF CHEMICAL SUBSTANCES (1979)	40,000*
IARC LIST OF ANIMAL CARCINOGENS WITH SUFFICIENT EVIDENCE OF CARCINOGENICITY (1982)	180
IARC LIST OF KNOWN AND PROBABLE HUMAN CARCINOGENS (1982)	80
NESHAP SUBSTANCES	6
SUBSTANCES PROPOSED FOR NESHAP REVIEW	30
POTENTIAL TOXIC AIR CONTAMINANTS	40-50

* Plus 2000-5000 per year

HISTORY

* AB 1005 — 1981/82

* ARB REGULATIONS — 1982/83

* AB 1807 — 1983

RATIONALE FOR CONTROL OF TOXIC AIR CONTAMINANTS

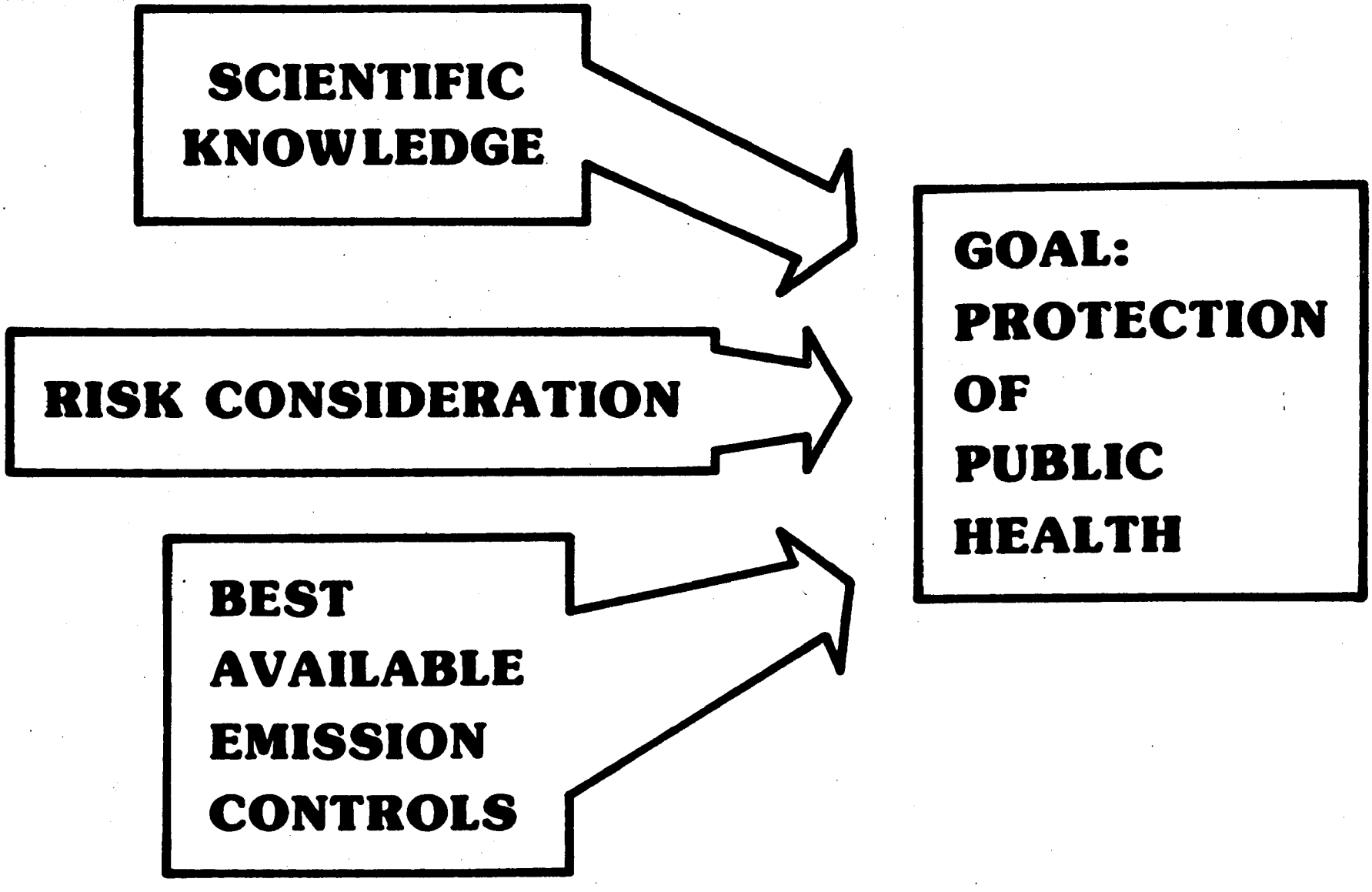
* AMBIENT AIR DATA

* PUBLIC EXPOSURE DATA

* EXISTENCE OF FEASIBLE CONTROL TECHNOLOGIES

* NEEDS OF LOCAL AIR POLLUTION CONTROL DISTRICTS

PROCESS


SCIENTIFIC KNOWLEDGE
RISK CONSIDERATION
BEST AVAILABLE EMISSION CONTROLS
GOAL: PROTECTION OF PUBLIC HEALTH

SUBSTANCE IDENTIFICATION

CONTROL DECISION

1 SUBSTANCE IDENTIFICATION

---- ARB ----

---- DOHS ----

2 CONTROL DECISION

---- APCD's ----

---- ARB ----

SCIENTIFIC REVIEW PANEL

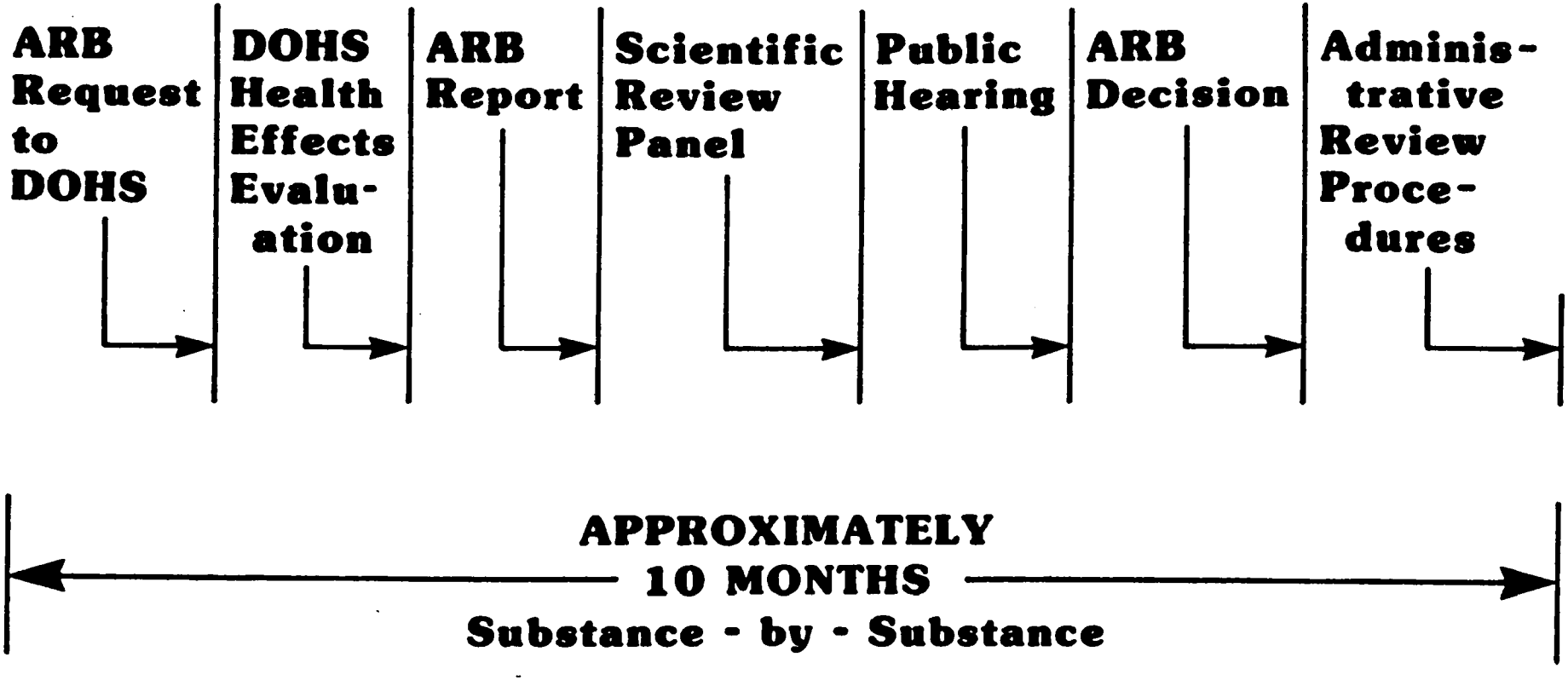
SUBSTANCE IDENTIFICATION

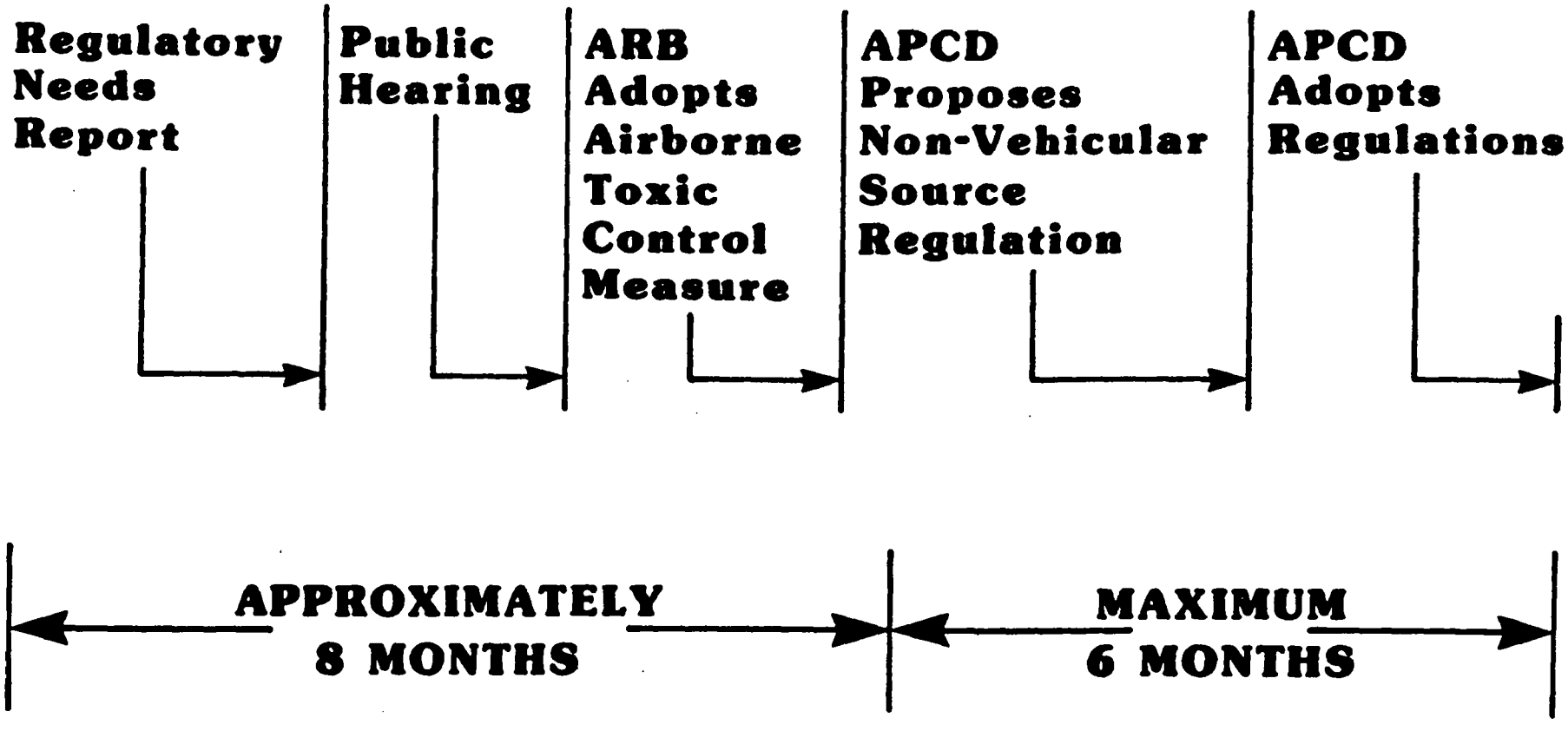
RISK ASSESSMENT

CONTROL DECISION

RISK MANAGEMENT

1 SUBSTANCE IDENTIFICATION PROCESS





2 CONTROL DECISION PROCESS

FACTORS TO BE ADDRESSED IN DEVELOPING AIRBORNE TOXIC CONTROL MEASURES:

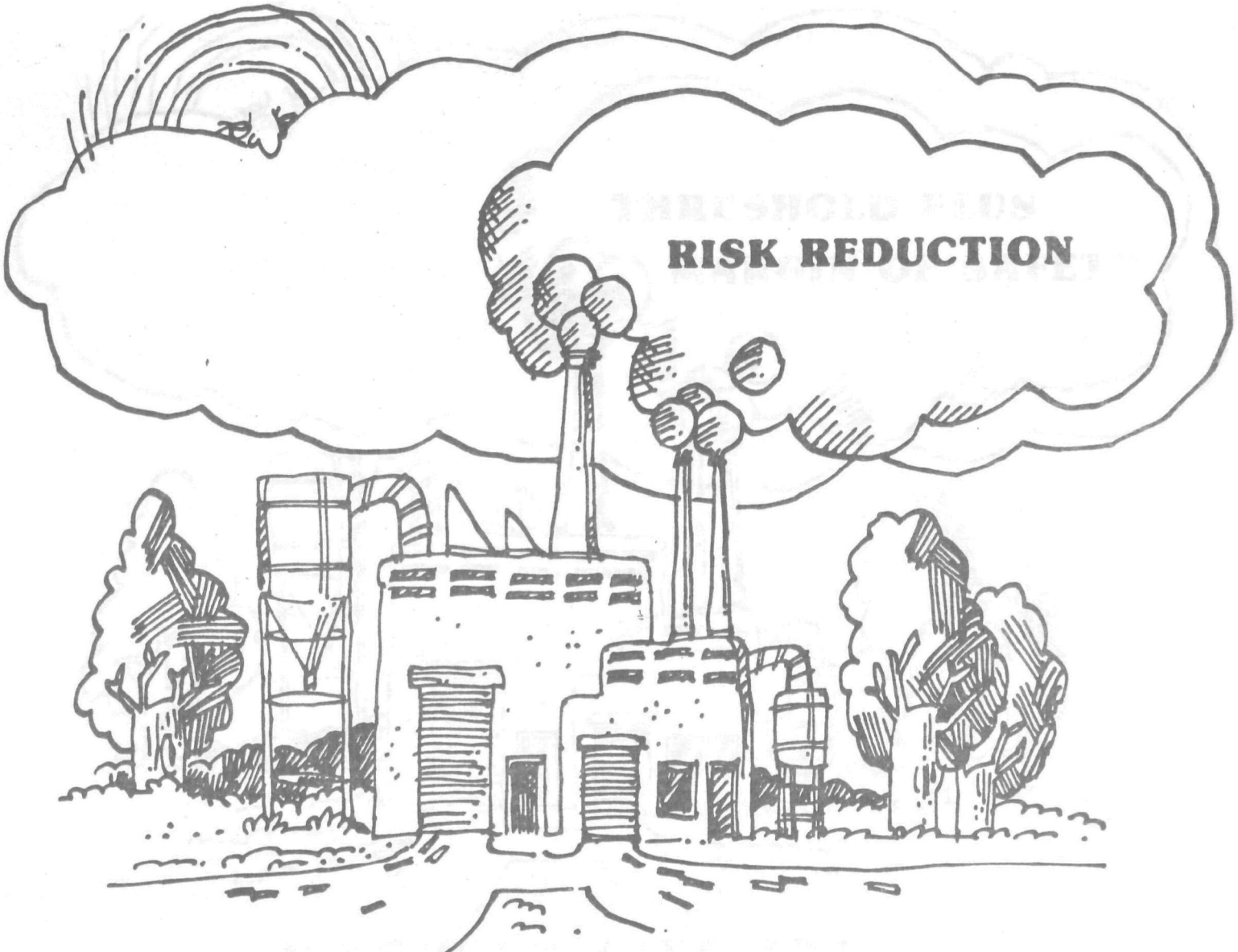
* SOURCES, SOURCE CATEGORIES, EMISSION LEVELS

* PHYSICAL/CHEMICAL CHARACTERISTICS OF SUBSTANCE IN AIR

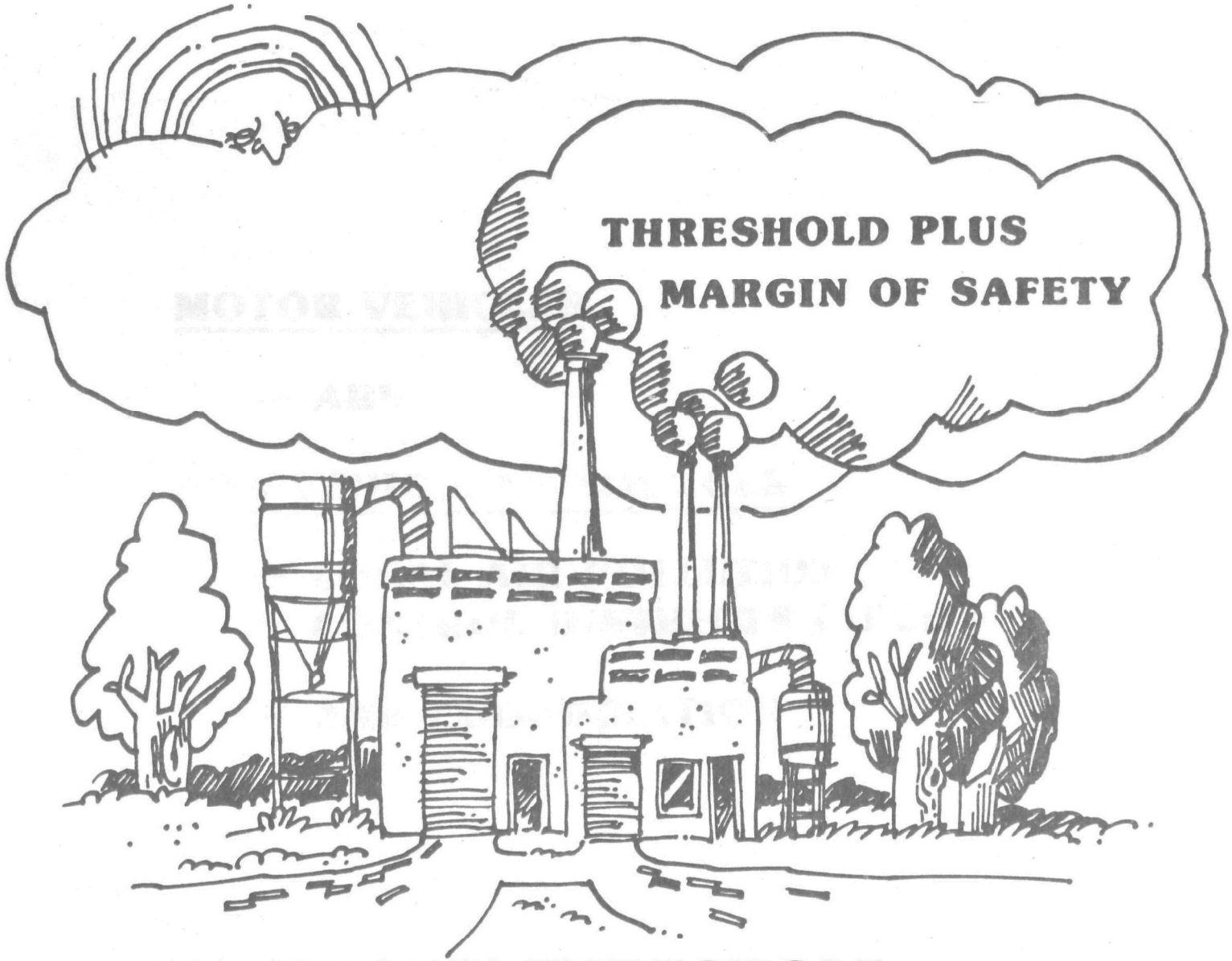
* PUBLIC HEALTH EFFECTS OF EXPOSURE

* AVAILABILITY AND COSTS OF CONTROLS AS RELATED TO RISK LEVELS

* SUITABILITY OF LESS HAZARDOUS SUBSTANCES



NO KNOWN THRESHOLD


THRESHOLD PLUS
MARGIN OF SAFETY
KNOWN THRESHOLD

MOTOR VEHICLES

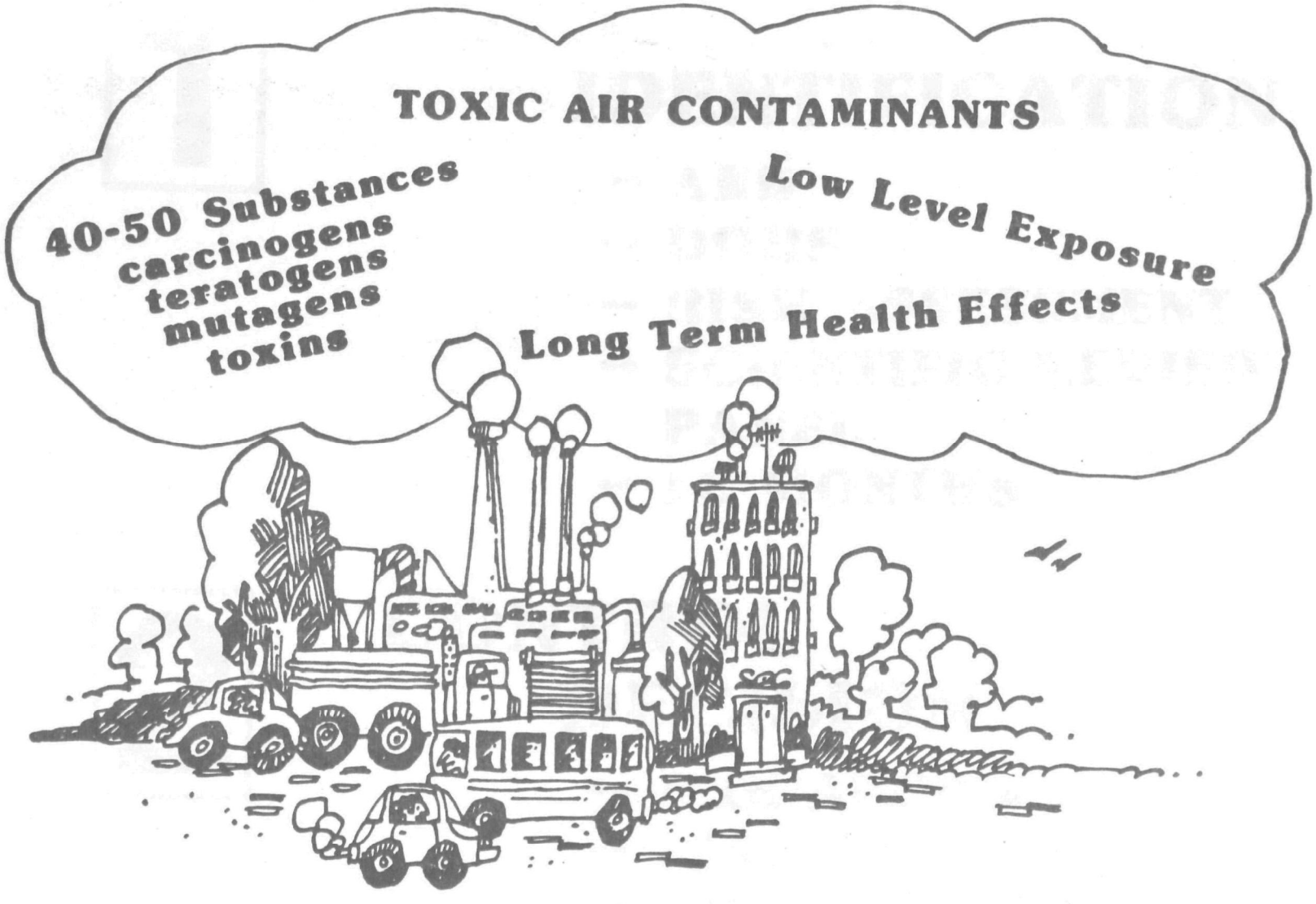
— **ARB**

NON-VEHICULAR SOURCES

— **LOCAL AIR POLLUTION CONTROL DISTRICTS (APCD's)**

— **ARB COORDINATION**

SUMMARY


TOXIC AIR CONTAMINANTS
40-50 Substances
carcinogens
teratogens
mutagens
toxins
Low Level Exposure
Long Term Health Effects

1 SUBSTANCE IDENTIFICATION

- -- ARB
- -- DOHS
- -- RISK ASSESSMENT
- -- SCIENTIFIC REVIEW PANEL
- -- 10 MONTHS

2 CONTROL DECISION

- -- ARB/APCD's
- -- RISK MANAGEMENT
- -- AIRBORNE TOXIC CONTROL MEASURES
- -- 14 MONTHS

CALIFORNIA DEPARTMENT OF HEALTH SERVICES CARCINOGEN POLICY

Dr. Kim Hooper
Research Scientist
California Department of Health Services
Berkeley, CA

Epigenetic Carcinogens: Problem with Identification and Risk Estimation

There is recent interest in the broad classification of carcinogens into two categories based on their mechanism of action: those that act through genetic mechanisms by interacting with DNA, causing gene mutation or duplication, or change in chromosome structure or number; and those that do not interact with DNA, but may cause changes in methylation patterns or tertiary structure of DNA, and are termed epigenetic carcinogens (IARC, 1983). Carcinogens which produce a consistent response in short-term tests for mutagenicity are designated as acting by a genetic mechanism, and are frequently called initiators or early stage carcinogens, indicating that they affect one of the early stages of the multi-step process of carcinogenesis. Carcinogens that do not produce responses in assays for mutation, cell transformation, chromosome aberration, or DNA binding or damage, are described as producing their carcinogenic effect by epigenetic mechanisms. Evidence for mechanism may be supplemented by initiation/promotion studies in specific organ systems, including mouse skin, rat liver, and urinary bladder to identify initiators or promotors.

The mechanisms of carcinogenesis are just beginning to be understood, and recent advances in the techniques of molecular biology have enabled us to describe and speculate on the actions and regulation of "oncogenes" (role of enhancers, promoter insertion models, role of various growth factors, etc.) and hold the promise to unravel the manner by which normal cells are transformed into the neoplastic state. It is likely that carcinogenesis is

complex, and that there are be many ways in which the cell's normal functioning can be disrupted and lead to neoplasia. Thus, in large part the proposed simple dichotomy of genetic and epigenetic carcinogens arises from and reflects our present limited knowledge of carcinogenesis. As we develop our ability to determine the mechanisms of action of individual carcinogens, such general terms as genetic and epigenetic will likely be replaced by more specific and meaningful terms. This view has been expressed by the International Agency for Research on Cancer (IARC), which concluded that "at present, no classification of carcinogens according to mechanism could be exhaustive or definitive. On the other hand, classification of mechanisms has considerable value for particular scientific purposes." (IARC, 1983)

A further significant proposal is that epigenetic carcinogens have "thresholds," dose levels at or below which no carcinogenic effects are produced. Consequently, risks of cancer from exposures to epigenetic carcinogens at low doses are presumed to be much lower than those for carcinogens which act by genetic mechanisms. A risk assessment method has been proposed which produces low estimates of cancer risks from exposures to epigenetic carcinogens. When applied to data from several cancer tests, this method would, in effect, permit public exposure to epigenetic carcinogens at levels 100-300 fold higher than would be permitted for genetic carcinogens using a standard method for estimating cancer risks. Such a proposal has enormous public and occupational health significance because several large volume industrial chlorinated carcinogens (e.g. DDT, dieldrin, PCBs, perchloroethylene, and trichloroethylene) have been described as acting by epigenetic mechanisms (Weissburger, 1983).

There are several problems with this proposal. First, there is at present no direct and validated means of identifying epigenetic carcinogens, except those that are active in an assay for promotors. Whereas genetic carcinogens may be directly identified by positive responses in DNA-binding studies or in a battery of short-term tests, epigenetic carcinogens are identified only indirectly by their failure to produce a response in one of the above short-term assays. For us to feel confident of an identification based on negative results, the frequency of false negatives in these tests must be known and be quite low. Unfortunately, the sensitivity of several of these assays is suspect and may lead to false negative results. In DNA-binding assays, for example, several thousand molecules of a carcinogen may be adducted to DNA per cell and not produce a significant positive response, even when radioactive carcinogens of the highest available specific activity are used. In fact, short-term test systems failed to detect most (all but direct-acting) carcinogens before metabolic activation was introduced. Under the proposed system, these would have been mistakenly classified as epigenetic carcinogens, even though they would be shown subsequently to directly interact with DNA after metabolic activation and be correctly classed as genetic carcinogens. Similarly, classic carcinogens which have been detected only after test systems were improved (e.g. large insoluble polycyclic aromatic hydrocarbons, aromatic amines, conjugated compounds and agents which presumably act through free-radical or oxidative mechanisms) would have been incorrectly designated as "epigenetic" agents because of the lack of response of the earlier test systems.

In summary, designating a substance an epigenetic carcinogen by the absence of response in a short-term test for mutagenicity or DNA-damage has obvious

shortcomings. At present, we can only tentatively identify agents as epigenetic carcinogens. Given this uncertainty, it appears unwise to permit exposure to 1. 300 fold higher levels than would be permitted for genetic carcinogens.

Second, even if epigenetic carcinogens could be conclusively identified, there is not clear evidence that "threshold" dose levels exist for either genetic or epigenetic carcinogens. For genetic carcinogens, the Food Safety Council plotted dose-response curves for the available multi-dose (greater than 3 dose levels) cancer bioassays (Report of the Food Safety Council, 1978). In tests of four genetic carcinogens (aflatoxin, vinyl chloride, dimethylnitrosamine, and bis-dichloromethylether) there was no evidence of a threshold. Instead, there are dose levels at which less than one tumor is expected to appear based on the dose response curve, and no tumors appear. Multi-dose studies using large numbers of animals with 2-acetylaminofluorene (6 doses; 20,000 mice) (Littlefield, 1979) and dimethylnitrosamine (4 doses; 5,000 rats) (Peto, personal communication, 1983) give no indication of thresholds. The dose response curve for bladder cancer in the 2-AAF experiment gives the impression of a threshold, but a re-plot of this data on an enlarged scale at improved resolution indicated that the tumor rates increase with dose even at the lower doses (Gaylor, 1980). The large study with DMN in rats produced a dose-response relationship that is linear in the low dose range (Peto, personal communication, 1983).

The Food Safety Council Report also contains dose-response plots for three chemicals which have been designated as promotors or epigenetic carcinogens (DDT, dieldrin, and saccharin) and there is no evidence for threshold dose

levels. Instead, there are dose levels where no tumors are expected, and no tumors appear. Promotion studies using saccharin (Nakanishi, et al., 1980) or phenobarbital (Peraino, et al. 1977; Kunz, et al., 1983), are cited as demonstrating thresholds, but provide no such evidence.

A cancer bioassay of nitrilo-triacetic acid (NTA) (Food Safety Council, 1978) at 5 dose levels has been cited as the best documentation of a threshold or "no effect" level. In fact, no threshold is evident even though there are no tumors at the two lowest dose levels. The number of kidney tumors "expected" in these two low-dose groups is much less than 1. Thus, the fact that no tumors (or less than 1 tumor) appear in these dose groups is a reasonable finding. The NTA case is representative of other examples where the "no effect" or threshold level" appears to be confused with the "no sensitivity" level. The apparent "no effect" level is, in fact, the dose level at which the study lacks the sensitivity to detect the expected response.

These above findings support the positions taken by the proposed DOHS Carcinogen Policy: that present knowledge is inadequate to justify separate risk assessment methods for genetic and epigenetic carcinogens; and that unless convincing evidence is presented to the contrary, quantitative estimates of carcinogenic risk will be made using non-threshold models.

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AMBIENT MONITORING FOR AIR TOXICS IN REGION 9

Dr. Hanwant Singh
Director, Atmospheric Chemistry Program
SRI International
Menlo Park, CA

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Distribution of Selected Gaseous Organic Mutagens and Suspect Carcinogens in Ambient Air

Hanwant B. Singh,* Louis J. Salas, and Robin E. Stiles

SRI International, Menlo Park, California 94025

■ An on-site field data collection program, based on short-term studies, was conducted in seven U.S. cities. Atmospheric concentrations, variabilities, and diurnal behaviors of 20 gaseous organic bacterial mutagens or suspect carcinogens are described. Except for benzene and formaldehyde, average concentration levels for all chemicals measured were in the 0–1-ppb range. Benzene and formaldehyde average levels were in the 1–6 and 10–20-ppb range, respectively. Typical diurnal profiles show highest concentrations during nighttime or early morning hours, with minimum concentrations in the afternoon hours; chemistry plays only a nominal role in defining this diurnal behavior in most cases. It is concluded that organic mutagens have always existed in the atmosphere (and the ocean), although at relatively low background concentrations. Our measurements for this group of 20 chemicals show that in the cleanest environments the present exposure is more than twice the natural background, whereas in the U.S. cities we studied exposure may be 15–30 times greater.

Introduction

In a recent report the surgeon general stated that "Toxic chemicals are adding to the disease burden of the United States in a significant, although as yet not precisely defined way" (*1*). Estimates suggesting that 50–90% of human cancer may be of chemical origin persists (*1*, *2*). The degree to which synoptic and macro- and microenvironments individually contribute to human cancer is a matter of ongoing research and debate (*3*, *4*). Although the risks may be highly uncertain, there is little doubt that significant quantities of a growing number of synthetic organic chemicals have been released into the ambient enviornment during recent decades. In many cases, virtually the entire quantity of the chemical manufactured is released into the environment as a necessary outcome of use (*5*, *6*). A key parameter in assessing risk from ambient exposure entails the characterization of ambient atmospheres in which the affected population resides. Because of the relatively recent interest in ambient hazardous chemicals, the atmospheric abundance, sources, and sinks of this group of pollutants are poorly understood. Although environmental episodes (e.g., the "Love Canal" incident) have received considerable attention (*1*), the extent of human exposure to chemicals in normal ambient atmospheres remains relatively poorly determined.

The present study was initiated to measure selected organic chemicals in several U.S. cities. Although we measured 44 organic chemicals, results presented here are limited to 20 bacterial mutagens and suspect carcinogens. Table I lists chemicals that have been defined as bacterial mutagens or suspect carcinogens. References 8–14, shown in the last column of Table I, often refer to additional studies that support their findings. Other chemicals for which concurrent ambient data were collected but not included here are fluorocarbons F 12, F 11, F 113, and F 114, ethyl chloride, 1,1-dichloroethane, 1,1,1,2-tetrachloroethane, 1,2-dichloroethylene, monochlorobenzene, *o*-dichlorobenzene, *m*-dichlorobenzene, 1,2,4-trichlorobenzene, toluene, ethylbenzene, *m*- and *p*-xylenes, *o*-xylene; 4-ethyltoluene, 1,2,4-trimethylbenzene, 1,3,5-trimethylbenzene, acetaldehyde, phosgene, peroxyacetyl nitrate, and peroxypropionyl nitrate. These excluded chemicals are not considered to be mutagenic or carcinogenic at the present time. The data can be found in ref 15. Empirical tests have shown that nearly 90% of tested animal carcinogens are also bacterial mutagens, while an equal percentage of noncarcinogens are nonmutagens (*7*). Bacterial mutagenicity tests are simple and direct and provide a useful screening test for carcinogenicity. The carcinogenicity information is based on tests involving epidemiology and a critical and comprehensive evaluation of carcinogenicity, mutagenicity, and other toxicological data (*8–10*). The terms "bacterial mutagens" (BM) and "suspect carcinogens" (SC) as used here do not imply that a proven human health hazard exists; however, these chemicals are

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Table I. Sources, Sinks, Background Levels, and Toxic Effects of Chemicals of Interest

chemical	major source[a]	dominant removal mechanism[b]	daily loss,[c] %	surface level bckgrnd concn[d] ppt	surface level bckgrnd concn[d] ng/m³	toxicity (ref)[e]
methyl chloride	N(O), MM	HO	0.4	650	1340	BM (8, 9)
methyl bromide	N(O), MM	HO	0.4	20	78	BM (8, 9)
methyl iodide	N(O)	$h\nu$(T)	12.2	2	12	BM, SC (8, 9, 12)
dichloromethane	MM	HO	1.3	50	173	BM (8, 9)
chloroform	MM	HO	0.9	20	97	BM, SC (8, 10, 11)
carbon tetrachloride	MM	$h\nu$(S)	~0.0	135	848	NBM, SC (8, 9, 10)
1,2-dichloroethane	MM	HO	1.9	40	168	BM, SC (8, 9, 10)
1,2-dibromoethane	MM	HO	2.2	2	15	BM, SC (8, 10)
1,1,1-trichloroethane	MM	HO	<0.1	180	981	weak BM (8, 9)
1,1,2-trichloroethane	MM	HO	2.8			NBM, SC (8, 9, 13)
1,1,2,2-tetrachloroethane	MM	HO	<0.1			BM, SC (8)
1,2-dichloropropane	MM	HO	10.2			BM (8)
1,1-dichloroethylene	MM	HO	29.2			BM, SC (8, 9, 10)
trichloroethylene	MM	HO	17.2	15	80	BM, SC (8, 9, 10)
tetrachloroethylene	MM	HO	1.5	50	337	SC (8, 10, 14)
3-chloro-1-propene	MM	HO	91.1			SC (8, 10)
hexachloro-1,3-butadiene	MM	HO				
α-chlorotoluene	MM	HO	22.8			BM, SC (8)
benzene	MM	HO	11.4			SC (8, 10)
formaldehyde	N, MM	HO, $h\nu$(T)	88.2	400	490	BM, SC (8, 10)

[a] N, natural; O, oceanic; MM, man-made. [b] HO, hydroxyl radical; $h\nu$, photolysis; T, troposphere; S, stratosphere. [c] Within the boundary layer (12 sunlit h); calculated based on estimated daytime (12 h) average HO abundance of 2×10^6 molecules/cm³ and mean temperature of 300 K. [d] At 40° N. [e] BM, bacterial mutagen (positive Ames test); NBM, not bacterial mutagen (negative Ames test); SC, suspect carcinogen.

considered to be of present and future atmospheric interest for both the environment and human well-being.

Experimental Program

Chemicals were measured on site and in real time by using an instrumented mobile environmental laboratory. All compounds listed in Table I (excluding benzene and formaldehyde) were measured with electron capture (EC) gas chromatography (GC). Benzene was measured with a flame ionization GC system. Formaldehyde was measured by the chromotropic acid method as well as by the analysis of its 2,4-dinitrophenylhydrazone (DNPH) derivate with high-performance liquid chromatography (*15*). For the analysis of all chemicals (except formaldehyde) listed in Table I, a 400-mL air sample was preconcentrated on a $^1/_{16}$ in. o.d. loop filled with glass wool (4-in. length) and held at liquid oxygen temperature. Sampling volume for formaldehyde analyses was approximately 120 and 60 L (2-h sampling time) for the chromotropic acid and the DNPH methods, respectively. A 24-h around-the-clock measurement schedule was followed for a period of 1–2 weeks at selected sites (Table II), allowing us to collect a body of data to study mean diurnal variations. Primary standards were generated by using a complex array of 40 permeation tubes. For approximately 15 of the 20 chemicals listed in Table I, high concentration standards (5–10 ppm) were also stored. (The high concentration was chosen to ensure long-term stability). These were obtained commercially from Scott-Marrin Inc. (Riverside, CA). Field calibrations were performed by pressurizing a large volume of urban air to 40 psi in a 35-L electropolished cylinder. After allowing the air to stabilize for a few days, it was calibrated against the primary standard, which then became a secondary field standard, routinely analyzed two to three times a day. In addition, 10–15-ppb secondary standards, prepared from the ppm standards, were carried aboard and also routinely analyzed. On the basis of limited intra- and interlaboratory comparisons, we estimate the overall accuracy of these measurements to be within ±15%. The measurements for formaldehyde might be accurate to within ±30%. Additional measurement details can be found in ref 15.

Results and Discussion

Table I summarizes the estimated daily loss rate of chemicals, their major source, toxicity, and background concentrations. The loss rates are estimated on the basis of hydroxyl (HO) radical reactivity and photolysis. Methyl iodide and formaldehyde are the only two chemicals for which photolytic loss is important. The background concentrations are based on surface level measurements conducted around the globe, but especially at a Pacific marine site at Point Arena, CA (39.9° N) (*5*, *16–18*). Reliable formaldehyde measurements are not available from remote sites, but a 400-ppt mixing ratio is not inconsistent with either limited measurements or estimates from photochemical models. Concentration data are provided both as mixing ratios and in nanograms per cubic meter (ng/m³). This redundancy is provided for convenience because exposures are invariably expressed in mass concentration units. Table II summarizes field data on the 20 bacterial mutagens and suspect carcinogens. Measured average (arithmetic averages) concentrations and the corresponding standard deviations are presented in units of ppt and ng/m³. Maximum and minimum concentrations are provided in ppt units.

Methyl halides constitute a unique group of bacterial mutagens that are ubiquitously distributed in atmospheric as well as oceanic environments (*16*, *18*, *19*). There is currently no doubt that these three methyl halides are bacterial mutagens with a relative mutagenic potential (revertants per unit weight) of CH_3Cl = 1, CH_3Br = 30, and CH_3I = 3 (*9*). Evidence suggests that methyl chloride, methyl bromide, and methyl iodide are dominant natural chlorine, bromine, and iodine carriers in the atmosphere (*16*). Their biogeochemical roles, however, are not yet fully understood; the possibility exists that these chemicals regulate the burden of stratospheric ozone. We also speculate that such chemical mutagens of natural origin may have played a hitherto undefined role in the processes

Table II. Concentrations of Chemicals in Urban Air

Chemical Compound	Houston--15-24 May 80 (Lat. 29°47' Long. 95°15')		St. Louis- 30 May-8 June 80 (Lat. 38°46' Long. 90°17')		Denver--16-26 June 80 (Lat. 39°43' Long. 104°59')		Riverside--2-12 July 80 (Lat. 33°59' Long. 117°18')		Staten Island--27 March-5 April 81 (Lat. 40°35' Long. 74°12')		Pittsburgh--8-16 April 81 (Lat. 7[illegible]° 26' Long. 79° 56')		Chicago--21-30 April 81 (Lat. 41° 45' Long. 87°42')	
	Concentration		Concentration		Concentration		Concentration		Concentration		Concentration		Concentration	
	ppt	ng/m³	ppt	ng/m³	ppt	ng/m³	ppt	ng/m³	ppt	ng/m³	ppt	ng/m³	ppt	ng/m³
Methyl chloride	955 (403)* 2284† 531‡	1968 (831)	732 (138) 1015 531	1509 (284)	763 (132) 1157 519	1573 (272)	703 (179) 1593 437	1449 (369)	701 (186) 1208 446	1445 (383)	665 (105) 852 450	1371 (216)	856 (168) 1311 575	1764 (346)
Methyl bromide	100 (58) 278 45	388 (225)	81 (25) 125 7	314 (97)	124 (51) 227 23	481 (198)	259 (167) 1033 43	1004 (648)	86 (108) 671 27	326 (419)	41 (6) 62 27	159 (23)	47 (17) 96 21	182 (66)
Methyl iodide	4 (2) 13 1	21 (13)	3 (2) 7 0	15 (9)	2 (1) 5 1	10 (6)	3 (1) 6 1	16 (7)	2 (1) 4 1	12 (6)	1 (1) 3 0	6 (6)	2 (2) 8 0	12 (12)
Dichloromethane	574 (533) 3404 49	1991 (1919)	421 (583) 6402 82	1461 (2023)	967 (926) 4876 108	3355 (3213)	1949 (1406) 9426 678	6762 (4878)	1605 (2947) 18476 226	5846 (10224)	390 (244) 1308 152	1353 (847)	1666 (6653) 56700 128	5780 (23082)
Chloroform	423 (749) 5112 38	2055 (3638)	73 (30) 191 25	335 (146)	185 (206) 1836 19	899 (1001)	703 (798) 4747 109	3415 (3876)	146 (117) 872 38	709 (568)	97 (41) 238 31	471 (199)	81 (26) 130 25	393 (126)
Carbon tetrachloride	404 (449) 2934 126	2539 (2822)	129 (6) 148 112	811 (38)	174 (19) 274 118	1094 (119)	175 (23) 267 151	1100 (165)	309 (202) 1200 125	1942 (1270)	331 (107) 691 131	2081 (673)	260 (52) 540 120	1634 (327)
1,2-Dichloroethane	1512 (1863) 7300 50	6110 (7528)	124 (101) 607 45	501 (408)	241 (297) 2089 54	974 (1200)	357 (325) 2505 63	1442 (1313)	256 (520) 4312 55	1034 (2101)	121 (35) 237 66	489 (141)	195 (360) 2820 22	788 (1374)
1,2-Dibromoethane	59 (72) 368 10	450 (550)	16 (4) 26 8	122 (31)	31 (15) 78 10	237 (114)	22 (7) 67 10	168 (53)	20 (6) 36 12	153 (46)	16 (10) 59 6	122 (76)	26 (37) 249 6	198 (282)
1,1,1-Trichloroethane	353 (263) 1499 134	1923 (1433)	235 (136) 896 132	1281 (741)	713 (553) 2699 171	3885 (3013)	747 (257) 1349 205	4070 (1400)	468 (248) 1427 221	2550 (1351)	486 (272) 1595 158	2648 (1482)	476 (158) 909 241	2594 (861)
1,1,2-Trichloroethane	32 (24) 129 <5	174 (131)	15 (6) 45 6	82 (33)	27 (10) 56 7	147 (54)	41 (21) 89 <5	223 (114)	7 (2) 11 3	38 (11)	6 (2) 11 3	33 (11)	7 (3) 14 3	38 (16)
1,1,2,2-Tetrachloroethane	11 (9) 77 2	75 (62)	6 (2) 12 4	41 (14)	10 (3) 17 3	69 (21)	12 (9) 77 5	82 (62)	- (-) - -	- (-)	4 (1) 6 3	27 (7)	3 (1) 6 2	21 (7)
1,2-Dichloropropane	81 (37) 253 22	374 (171)	53 (12) 88 22	244 (55)	48 (14) 99 20	221 (65)	57 (15) 88 11	263 (69)	26 (15) 79 10	120 (69)	23 (8) 50 4	106 (37)	29 (7) 40 10	134 (32)
1,1-Dichloroethylene	25 (36) 136 <4	99 (143)	9 (5) 34 <4	36 (20)	31 (49) 224 <4	123 (196)	9 (6) 56 <4	36 (24)	- (-) - -	- (-)	- (-) - -	- (-)	22 (19) 68 3	87 (75)
Trichloroethylene	144 (195) 980 3	773 (1047)	112 (154) 1040 8	80: (827)	198 (313) 2483 7	1063 (1680)	118 (55) 236 15	633 (295)	187 (199) 1005 26	896 (1068)	96 (93) 420 13	515 (499)	225 (282) 1386 18	1208 (1514)
Tetrachloroethylene	401 (598) 3215 34	2717 (4052)	326 (955) 7604 67	2209 (6471)	394 (158) 1130 99	2670 (1071)	484 (236) 1626 173	3279 (1599)	292 (200) 1034 79	1978 (1355)	409 (357) 1657 80	2771 (2419)	590 (452) 1787 90	3998 (3063)
3,Chloro-1-propene	<5 (-) <5 <5	<16(-)	<5 (-) <5 <5	<16 (-)	<5 (-) <5 <5	<16 (-)	<5 (-) <5 <5	<16 (-)	- (-) - (-) - (-)	- (-)	6 (7) 19 <1	64 (75)	- (-) - (-) - (-)	- (-)
Hexachloro-1,3 butadiene	11 (20) 154 1	117 (213)	3 (2) 10 1	32 (21)	2 (1) 7 0	21 (11)	4 (3) 16 1	43 (32)	- (-) - -	- (-)	- (-) - (-) - (-)	- (-)	- (-) - -	- (-)
o-Chlorotoluene	<5 (-) 58 <5	26 (-)	<5 (-) 25 <5	26 (-)	<5 (-) 111 <5	26 (-)	<5 (-) 39 <5	26 (-)	- (-) - -	- (-)	- (-) - -	- (-)	- (-) - -	
Benzene	5780 (5880) 37700 840	18402 (18720)	1410 (1190) 5820 110	4489 (3789)	4390 (3940) 23910 110	13976 (12544)	3950 (1910) 10980 520	12576 (6081)	4204 (4287) 19034 82	13384 (13648)	5003 (9818) 64619 392	15928 (31257)	2561 (1779) 8771 508	8153 (5664)
Formaldehyde	- (-) - -	- - (-)	11300 (4500) 18700 8100	13836 (5510)	12300 (5900) 28700 6600	15061 (7224)	19000 (7600) 41000 10400	23265 (9306)	14300 (9100) 45900 7000	17510 (11143)	20600 (5200) 35100 12900	25224 (6367)	12800 (3300) 17200 9100	15673 (4041)

* Arithmetic average (standard deviation). † Maximum concentration. ‡ Minimum concentration.

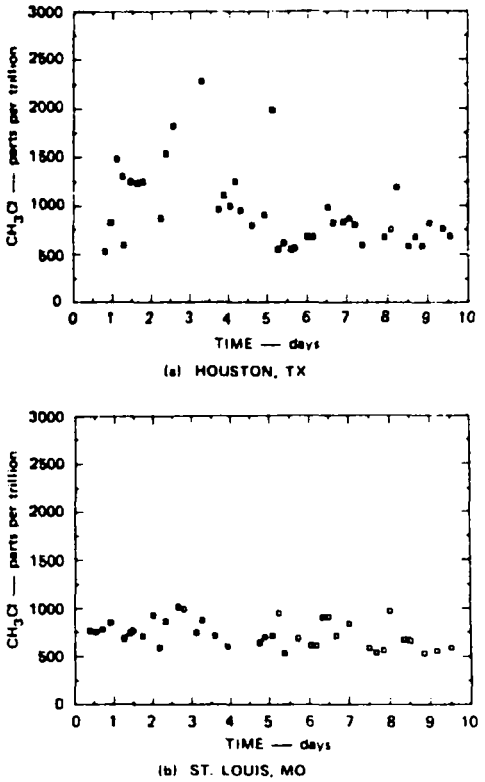

Figure 1. Methyl chloride in the ambient air of selected cities.

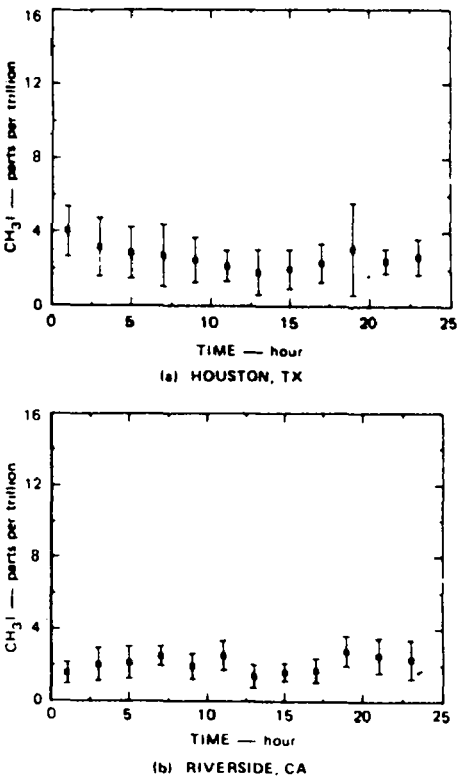

Figure 2. Mean diurnal variation of methyl iodide.

of biological evolution; chemical bacterial mutagens have been a part of our environment since prehistoric time.

Table II summarizes the urban methyl halide levels at seven sites in selected U.S. cities. Methyl chloride average levels of 0.66–0.96 ppb are close to or marginally above the background of 0.6–0.7 ppb. Clearly, local sources of methyl chloride in urban areas exist. Figure 1 shows elevated methyl chloride levels in Houston, while St. Louis levels are near background. It appears unlikely that primary methyl chloride emissions could account for this difference. We suspect secondary sources of methyl chloride (e.g., combustion) exist but have not yet been fully characterized. Methyl bromide levels (average of 0.04–0.26 ppb) were found to be well above background in all cities. Average methyl iodide levels of 1–4 ppt at all urban sites are slightly lower or indistinguishable from their backgrounds. Thus, methyl iodide remains a chemical of virtually exclusive natural origin. Its low abundance is, in part, attributable to its high reactivity (12% daily loss rate). Figure 2 shows a mean diurnal methyl iodide profile at two selected sites; the afternoon minimum is attributable to its high reactivity. Overall maximum methyl chloride, methyl bromide, and methyl iodide concentrations of 2.3 ppb (Houston), 1.0 ppb (Riverside), and 0.01 ppb (Houston) were measured, with minimum concentrations being indistinguishable from background levels.

Urban methyl halide data from the literature are scarce, partly because solid sorbents such as Tenax, which have been used extensively for routine data collection, do not appear to collect methyl halides (*20*, *21*) although the presence of methyl bromide was noted (*20*). Chameides and Davis' recently summarized methyl iodide data from clean as well as from polluted environments point to a great deal of variability (22). A substantial part of this variability, especially in urban areas, we believe to be associated with earlier measurement problems.

Dichloromethane (methylene chloride) is also a bacterial mutagen (*9*), but is only about a quarter as potent as methyl iodide. Average concentrations (Table II) were in the 0.4–2-ppb range, which are at least an order of magnitude higher than the background concentration of 0.05 ppb. Maximum concentrations of 18 and 9 ppb were measured at Staten Island and Riverside, respectively. Even though methylene chloride is a typical solvent likely to find most use during daytime, in virtually all cases (except Riverside) the highest values were encountered during nighttime, with afternoon lows. Figure 3 provides an example of this typical diurnal behavior at the Houston and Denver sites and the reverse behavior at Riverside. Although conclusive data interpretation is not possible in the absence of accurate daily emission inventories, the very high methylene chloride levels reported earlier from Los Angeles (average = 3.7 ppb) (*23*) and the downwind nature of the Riverside site can provide part of the explanation. The afternoon minimums cannot be attributed to chemical loss because of the relatively unreactive nature of this chemical (Table I). As we shall see, the afternoon minimum is a fairly general feature and can only be attributed to dilution caused by deep vertical mixing typical of afternoon hours.

Pellizzari and Bunch (*20*), using the Tenax collection procedures, have also reported methylene chloride concentrations from several industrial sites and show significantly greater variability as compared with our data. Although our data are not necessarily inconsistent with these measurements, certain discrepancies are evident. For example, concentrations significantly lower than geochemical background have been frequently reported, a phenomenon also found to be true in cases of carbon tetrachloride, 1,1,1-trichloroethane, 1,2-dichloroethane, trichloromethylene, and tetrachloroethylene (*20*). The causes of this problem are unclear. By and large, urban methylene chloride data are scarce.

Chloroform, a mutagen and a suspect carcinogen (*9–11*), has received considerable attention because of its high levels in drinking water (*24*). Average concentrations of about 0.7 ppb at Riverside, 0.4 ppb at Houston, and about 0.1 ppb at the other sites are clearly 1–2 orders of mag-

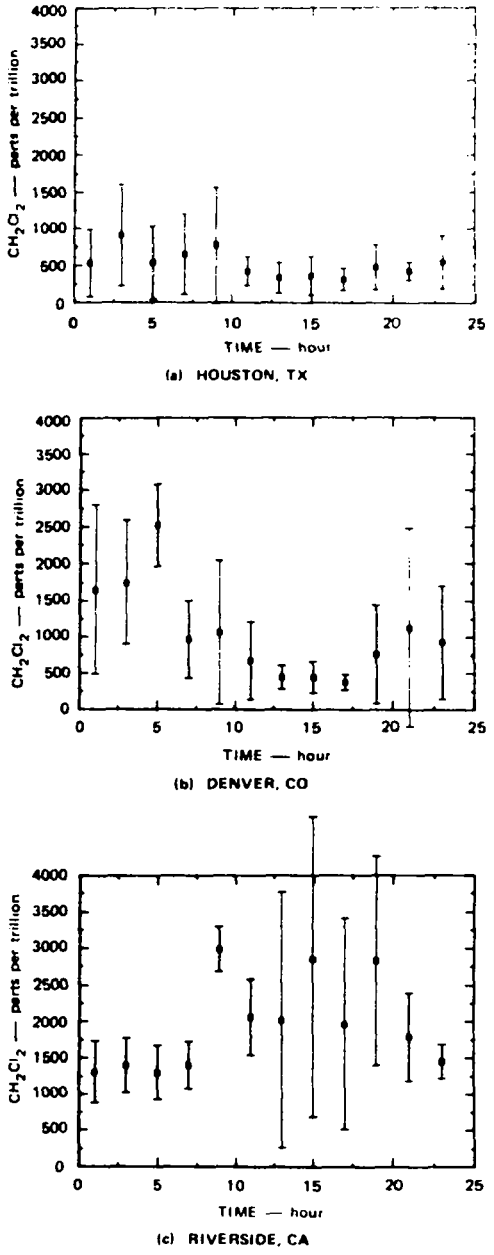

Figure 3. Mean diurnal variation of methylene chloride at selected sites.

nitude higher than background concentrations. Maximum concentrations of about 5 ppb were measured at more than one site. Figure 4 clearly points to the existence of urban sources. As shown earlier (*15*, *23*), the highest levels of chloroform are measured during the night. The direct sources of chloroform (U.S. emissions are ≤0.02 million tons/year) appear to be too small to account for its pervasiveness in urban environments. In a recent review (*25*), chlorination of water and possibly automobile exhaust were suggested as two important sources of chloroform. Pellizzari and Bunch (*20*) report concentrations that vary from unquantifiable levels to 7 ppb, a range comparable to that found in our study.

Carbon tetrachloride, a man-made chemical, is nearly uniformly distributed around the globe (*18*, *26*). Urban carbon tetrachloride levels are higher than background levels by a factor 1.5–3. At all sites (except Houston) average concentrations were between 0.2 and 0.3 ppb. At Houston the maximum and average concentrations were 2.9 and 0.4 ppb, respectively. The diurnal behavior of carbon tetrachloride was typical of other pollutants. The Staten Island mean diurnal behavior is shown in Figure

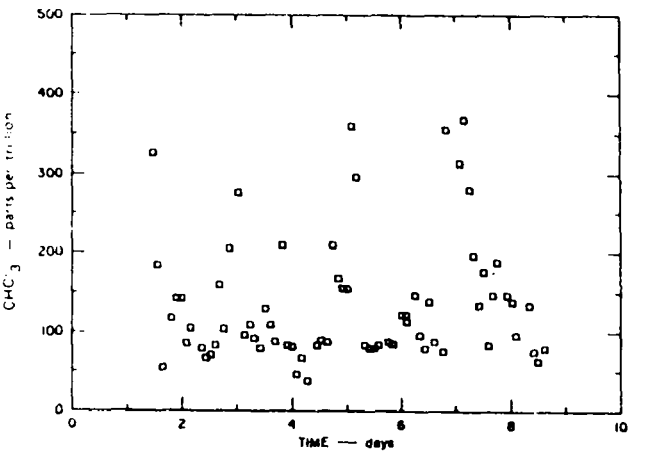

Figure 4. Atmospheric concentrations of chloroform at Staten Island, NY.

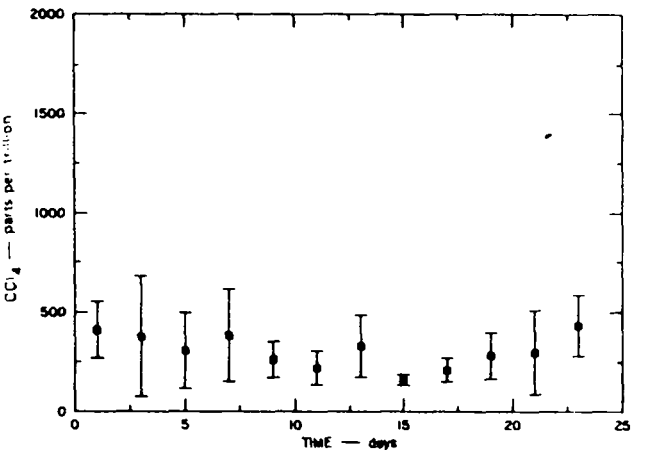

Figure 5. Mean diurnal variations of carbon tetrachloride at Staten Island, NY.

5. Concentrations during the afternoon minimum are comparable to background levels (0.14 ppb) of carbon tetrachloride, a condition caused by deep vertical mixing during afternoon hours. Very little urban data from other sources have been available, although its background appears well characterized (*17*, *18*, *26*). Limited urban measurements from Lillian et al. (*27*) and Simmonds et al. (*28*) are consistent with our measurements. Ohta et al. (*29*) reported significantly higher values (average = 1.4 ppb) from Tokyo.

1,2-Dichloroethane, a large-volume chemical (U.S. emissions approximately 0.2 million tons/year), is a bacterial mutagen and a suspect carcinogen (Table I). Average concentrations of 0.1–1.5 ppb point to a considerable difference in abundance at various sites. Maximum concentration of 7.3 ppb was measured at Houston; Figure 6 shows the mean diurnal profile of 1,2-dichloroethane at Houston and Riverside. Once again, the highest values are encountered during the night and early morning hours. Previous atmospheric data on 1,2-dichloroethane are extremely sparse. Bozzelli (*21*) could quantify only 2 samples from a total of 250 collected. Pellizzari and Bunch (*20*) provide abundant data that are well below the measured as well as the estimated background of about 30–50 ppt (*6*, *17*), although their higher concentrations are comparable to data presented here.

1,2-Dibromoethane is expected to be a potent carcinogen with a unit risk 50 times greater than 1,2-dichloroethane (*10*). This chemical is used primarily as a gasoline additive and a fumigant (U.S. production is 0.1 million tons/year). Average 1,2-dibromoethane concentration at no study site

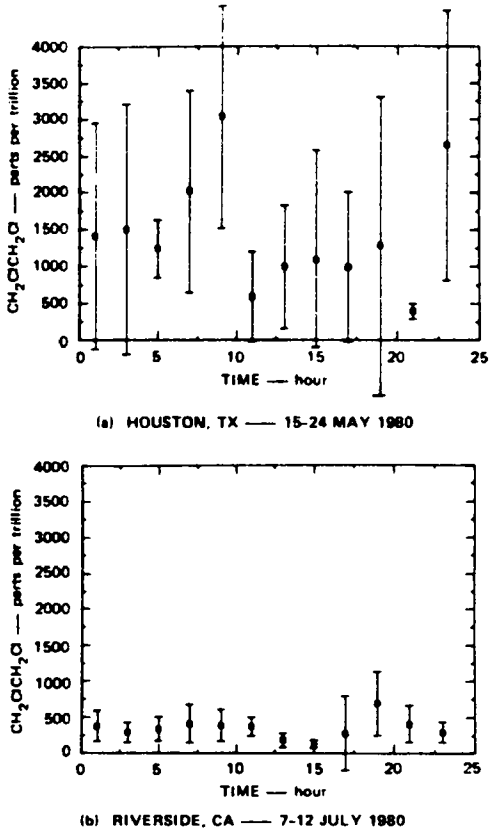

Figure 6. Mean diurnal variation of 1,2-dichloroethane.

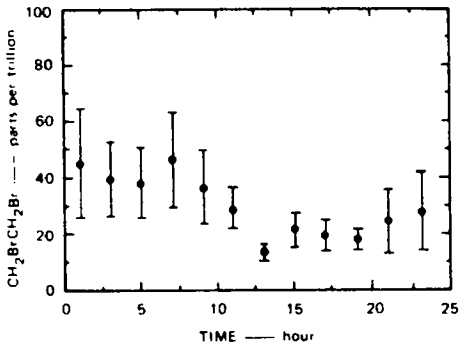

Figure 7. Mean diurnal variation of 1,2-dibromoethane at Denver, CO.

exceeded 0.06 ppb (average range 0.02–0.06 ppb), although concentrations as high as 0.37 ppb were measured. A typical mean diurnal profile for the Denver site is shown in Figure 7. The highest average levels were again encountered during night and early morning hours. Typical ambient concentration data available from the literature suggest a concentration range of 0–0.3 ppb (*20*, *30*, *31*). Bozzelli et al. (*21*) report some exceptionally high values from New Jersey.

1,1,1-Trichloroethane, a popular solvent in recent years, has undergone a rapid growth (*17*, *18*). It is weakly mutagenic, although considerable disagreement as to its health effects exists (*8*, *9*, *32*). Virtually all the manufactured amount is released into the environment. This chemical is quite stable in the atmosphere and a global residence time of about 8 years has been estimated (*18*). About 15% of this chemical could enter the stratosphere where it could interreact with the ozone layer in a manner similar to fluorocarbons. Typical average concentrations (Table II) were measured to be in the 0.25–0.75-ppb range. The highest concentration of 2.7 ppb was measured in Denver. Figure 8 shows a typical diurnal behavior in Houston and

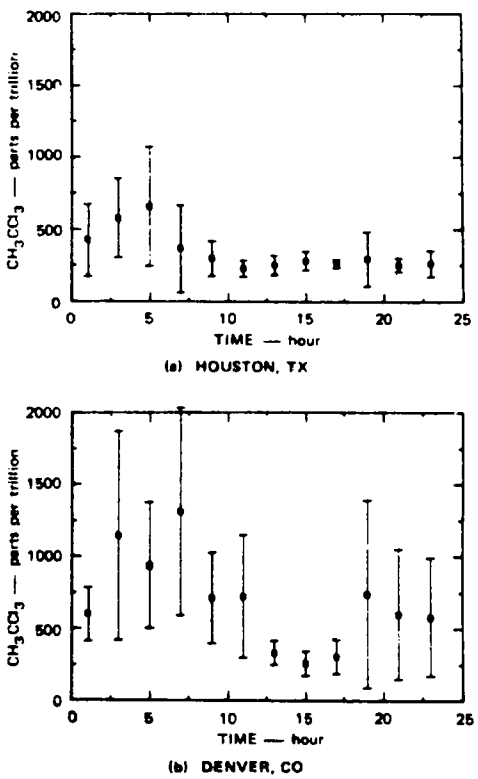

Figure 8. Mean diurnal variation of 1,1,1-trichloroethane.

Denver. Very little recent urban data have been published. Simmonds et al. (*28*) found an average concentration of 0.37 ppb in Los Angeles in 1973, which is in reasonable agreement with our Riverside data (average = 0.7 ppb), if we recognize that the emissions of 1,1,1-trichloroethane have more than doubled during the last 8 years. A great deal of 1,1,1-trichloroethane data from remote environments have been collected (*17*, *18*), even though urban measurements are sparse.

1,1,2-Trichloroethane was found at extremely low concentrations (average 0.01–0.04 ppb) at all sites. At no time did its concentration exceed 0.15 ppb. These data are not inconsistent with those reported by Pellizzari (*20*), who found levels of <0.01–2 ppb at sites in New Jersey, Texas, and Louisiana. This 1,1,2 isomer is nearly 30 times more reactive than the 1,1,1 isomer of trichloroethane (Table I). 1,1,2,2-Tetrachloroethane was measured at an average concentration of 0.01 ppb or less (Table II). Its highest concentration never exceeded 0.1 ppb. 1,2-Dichloroprane was also present at an average concentration of 0.02–0.07 ppb, and its highest measured concentration never exceeded 0.25 ppb. Compared with chloroethanes, this chloropropane is considerably more reactive, and a 10% daily loss rate is esimated (Table I). In the only data available (*20*), 1,1,2,2-tetrachloroethane and 1,2-dichloropropane levels of about 0.01 and 0.02 ppb, respectively, have been reported.

Five chloroalkenes were measured, and of these, allyl chloride (3-chloro-1-propene), a suspect carcinogen, could not be detected at concentrations exceeding 5 ppt. 1,1-Dichloroethylene (vinylidene chloride) was present at an average concentration of 0.01–0.03 ppb. However, it was below our detection limit of 5 ppt during 30–50% of the time of all sites. These values are quite consistent with the very low emissions (1–4 tons/year for 1,1-dichloroethylene and 500 tons/year for allyl chloride) and high reactivity (Table I). Although vinylidene chloride has been identified in Tenax air samples (*20*), quantification has not been possible for lack of sensitivity (detection limit of

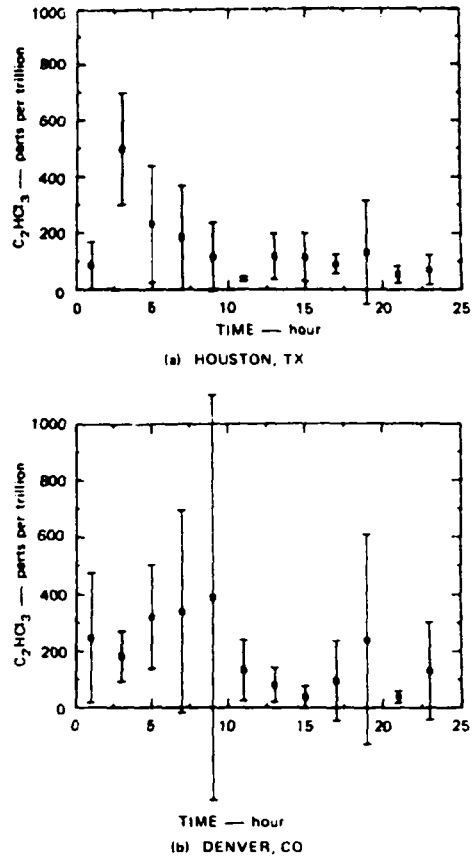

Figure 9. Mean diurnal variation of trichloroethylene.

0.05–0.1 ppb). Occasionally, however, scattered data in the concentration range of 0.01–0.6 ppb have been reported (*20*).

Of the chloroalkenes measured in this study, trichloroethylene (TCE) and tetrachloroethylene (called perchloroethylene, PCE) are two large-volume chemicals. Considerable debate on the potential carcinogenicity of these alkenes currently exists (*8*, *10*, *33*). Their annual U.S. emissions are estimated to be 0.15 and 0.3 million tons, respectively. As is clear from Table I, TCE is substantially more reactive in the atmosphere. TCE and PCE average concentrations range from 0.1 to 0.2 and 0.3 to 0.6 ppb, respectively. The concentration ratio (PCE/TCE) lies between 2 and 4. The highest PCE concentration, 7.6 ppb, is also about 3 times the highest TCE concentration of 2.5 ppb. In both instances a significant elevation above background levels is evident. Their diurnal behavior was very nearly identical; Figure 9 demonstrates a typical diurnal profile of TCE. PCE diurnal behavior is similar, but the nighttime–daytime gradients are somewhat less pronounced.

Although TCE and PCE have been measured by several investigators, data are sporadic. Lillian et al. (*27*) reported average concentrations of 0.1–0.9 ppb (maximum 18 ppb) for TCE and 0.1–4.5 ppb (maximum 8 ppb) for PCE from several Eastern coastal cities. Bozzelli et al. (*21*) could quantify only a small fraction of the collected samples and reported an average concentration range of 1–2 ppb of TCE and 0.3–4 ppb of PCE from six sites in New Jersey. Contrary to our findings, their average PCE/TCE concentration ratio was greater than 1 at only three of the six sites. Ohta et al. (*29*) reported from Tokyo average concentrations of 1.2 ppb for both. Other measurements by Singh et al. (*5*) have been reviewed (*14*). Unlike other chloroalkenes, hexachloro-1,3-butadiene (HCBD), a bacterial mutagen, is no longer manufactured in the U.S. HCBD, however, has been identified in the effluents of sewage treatment plants and as a byproduct of the combustion of plastics; secondary sources do exist. HCBD was measured at an average concentration of less than 0.01 ppb at all sites; its highest measured concentration was 0.15 ppb. No information is available on the reactivity of this chemical, but its structure would suggest that it is highly reactive. Limited measurements from Niagara Falls, Louisiana, and Texas (*20*) show a concentration range of 0–0.1 ppb.

Although several chloroaromatics were measured during this study (*15*), α-chlorotoluene (benzyl chloride) is the only member that shows clear evidence of bacterial mutagenicity (Table I). Ambient concentrations as high as 0.11 ppb were measured, but by and large this chemical was not detectable at 5-ppt levels. A daily loss rate of 23% (Table I) and an estimated U.S. emission strength of 45 tons/year are entirely consistent with its nondetectability in urban atmospheres (*15*). No abmient data could be found in the literature, although concentrations in the 1–2-ppb range have been reported near a Stauffer chemical plant in Edison, NJ (*20*).

Eight important aromatic hydrocarbons were measured; of these, benzene is a suspected carcinogen (*8*, *10*). Because the mutagenicity of toluene is strongly disputed (*1*, *10*), it is not included here. The average benzene concentrations at all sites were between 1.5 and 6 ppb, although concentrations as high as 65 ppb were measured. Toluene was typically 1–2 times more abundant than benzene. The diurnal behavior of benzene, shown in Figure 10, was representative of all aromatic hydrocarbons. The mean diurnal behavior of benzene is not atypical of other pollutants discussed here. Much of the literature data on benzene were obtained during the daytime (*34*), and these average concentrations are comparable to daytime concentrations reported here. No diurnal profiles of benzene could be found in the published literature.

Formaldehyde, a mutagen and a suspect carcinogen, is also a natural component of the global atmosphere (Table I). Its average urban concentrations of 10–20 ppb are significantly higher than an estimated background of 0.4 ppb (Table I). Despite its extremely high reactivity, formaldehyde was the most dominant bacterial mutagen found in the urban atmosphere, some 4 times (range of 3–8 times) more abundant than benzene. Formaldehyde was measured by two different methods, both with comparable results (±30%). Acetaldehyde, a nonmutagen, was also measured at an average concentration of 1 and 2 ppb (range of 0.2–3.4 ppb) at the Pittsburgh and Chicago sites, respectively. These data and those of others have been presented in greater detail in ref 15.

Although the chemicals listed in Table I can differ significantly as to their mutagenic and carcinogenic capacities (*9*, *10*), a simple summation of their mass concentration is of interest for comparison with background levels. Collectively, the aggregate daily mean exposure to all chemicals in Table II is found to lie between 27 and 59 $\mu g/m^3$ at all sites. (Formaldehyde data in Houston could not be collected because of technical difficulties, so the Houston data of Joshi (*35*) are used). The daily average exposure in an unpolluted environment from the four naturally occurring mutagens (methyl halides and formaldehyde) is determined to be about 1.9 $\mu g/m^3$ from data in Table I. Total exposure to all mutagens and suspect carcinogens (both natural and man-made) in the present remote environments is 4.4 $\mu g/m^3$ (Table I). Thus, even in a locally unpolluted environment, the present exposure

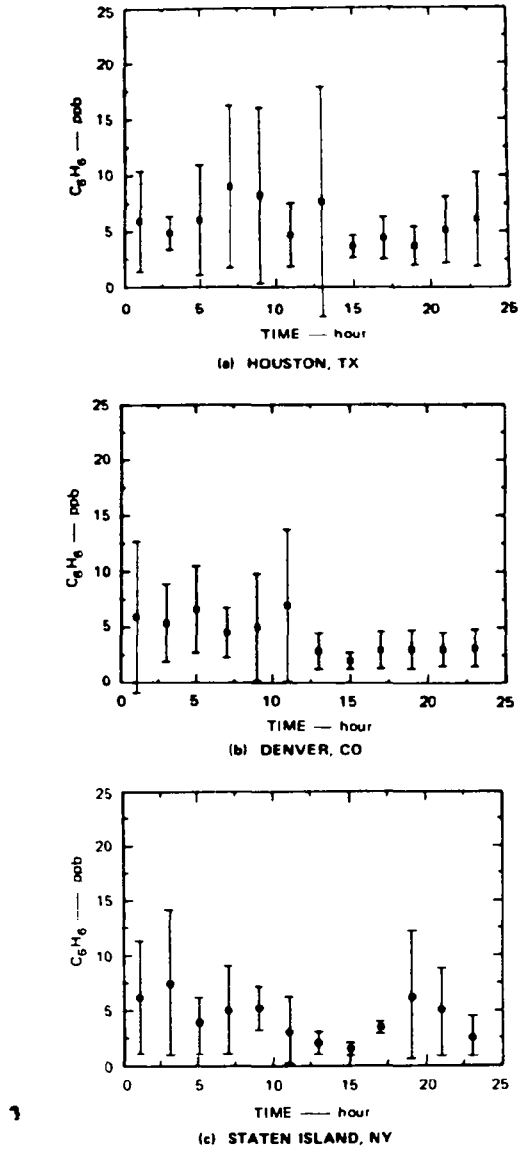

Figure 10. Mean diurnal variation of benzene.

to this group of chemicals has more than doubled. In urban environments this exposure is at least 15–30 times the natural background (1.9 vs. 27–59 $\mu g/m^3$).

The total exposure to mutagens and carcinogens from urban ambient air is, of course, much higher because of nongaseous species (e.g., polyaromatic hydrocarbons) (*36*) as well as other gaseous species for which either toxicity studies are inconclusive or measurement methods inadequate (e.g., oxygenated chemicals). More extensive measurements are clearly needed to refine further the quantitative relationships developed here.

Concluding Thoughts

A number of synthetic organic chemicals that are known to be toxic at concentrations much higher than those found in ambient air are present in the urban as well as remote atmospheres. The data base to define the abundance of such chemicals is currently very limited. Most synthetic chemicals listed in Table I came into major use after 1950, and since then their production and release have continued to grow exponentially, with a doubling time of about 6 years (*5*). Because of the long lag times (10–50 years) associated with the onset of cancer (*1*, *2*), a significant risk may not be identified until a future date. Continuous exposure to low levels of such chemicals could erode any human threshold that may exist or enhance the frequency of cancer's occurring from other primary causes such as cigarette smoking (*37*). The ubiquitousness of organic mutagens of natural origin in the air and oceans leads us to speculate that they may have played a role similar to that attributed to radiation in the processes of biological evolution. A comparison of the mutagenic activity of these natural organics with natural low-level radiation would help to understand better the part natural chemicals might have played in those processes.

Acknowledgments

Helpful discussions with L. Cupitt of the U.S. Environmental Protection Agency are appreciated.

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SOURCES OF AIR TOXICS IN CALIFORNIA

G. C. Hass
Chief, Haagen-Smit Laboratory Division
California Air Resources Board
El Monte, CA

Terry McGuire
Assistant Division Chief, Stationary Source Division
California Air Resources Board
Sacramento, CA

Summary of Comments on Ambient Air Monitoring for Toxic Compounds by the Air Resources Board

by
G. C. Hass, Chief
Haagen-Smit Laboratory Division
El Monte

The Air Resources Board began monitoring for toxic compounds in the vicinity of suspected sources of particular compounds; e.g., vinyl chloride, halogenated hydrocarbons, benzene, etc. It was standard practice to locate a control sampling point some distance removed from the source. Not infrequently, measurements at the control point exceeded those near the source. This led to the beginning of our current program to monitor the general urban air on a regular basis. Currently, we collect four 24-hour samples weekly at our El Monte lab headquarters. Three other locations, Riverside, downtown Los Angeles, and Dominguez are sampled on a three-day rotating schedule.

The samples are collected in Tedlar bags and taken to the El Monte facility for analysis within a few hours of the end of the collection period. An aliquot of the bag contents is transferred by syringe to the freeze out loop of a gas chromatograph equipped with an electron capture detector. Our standard procedure yields results for nineteen halogenated hydrocarbons (not all of interest as toxics). It is our intention to add a separate GC analysis for benzene.

The El Monte station has been in operation since November, 1982, and the satellites from one to three months later. Mean results from the stations are shown for six compounds in Table I. These six compounds are believed

to be of significant toxic interest. Some were found above the limit of detection in all samples, while the others were found often enough to yield an estimate of their mean concentration. Carbon tetrachloride behaves much like a "clean air" background compound in terms of spatial and temporal distribution. Our mean value, however, is only about one third the background value reported by Dr. Singh in the previous presentation.

This raises the question of the reliability of those measurements. The concentrations reported are about three orders of magnitude lower than those encountered in conventional air pollution work. Opportunities for error in sampling, sample transfer, standardizing procedures, and analysis are correspondingly amplified. Nevertheless, the numbers cannot be ignored pending attainment of the confidence limits to which we are accustomed. In the case of carbon tetrachloride cited above, which shows an apparent discrepancy of a factor of three, either number is of social concern when considered in the context of published cancer risk factors and the population at risk.

The ARB Haagen-Smit Laboratory has benefitted by being close neighbors to the South Coast Air Quality Management District facilities. Professional cooperation has been beneficial to both laboratories. The ARB now proposes to extend this cooperation to other governmental organizations in California by establishing in the near future a Toxic Substances Technical Advisory Committee (TOXTAC).

TABLE I

TOXIC ORGANIC SURVEY
MEAN VALUES OF COMPOUNDS
AT FOUR SAMPLING SITES
INCLUSIVE DATES: 11/14/82 TO 6/26/83

	SAMPLING SITE COMPOUNDS	EL MONTE CONC. PPb	DOLA CONC. PPb	DOMINGUEZ CONC. PPb	RIVERSIDE CONC. PPb
1.	DICHLOROMETHANE	1.47	0.89	1.62	1.09
2.	TRICHLOROMETHANE	0.05	0.17	0.05	0.05
3.	TETRACHLOROMETHANE	0.04	0.06	0.04	0.04
4.	TRICHLOROETHYLENE	0.32	0.63	0.34	0.25
5.	1,2-DIBROMOETHANE	0.01	0.01	0.01	0.01
6.	TETRACHLOROETHYLENE	1.20	1.38	1.36	0.44

Sources of Air Toxics in California

Terry McGuire - Assistant Division Chief
Stationary Source Division
California Air Resources Board

The California Air Resources Board is working in a number of areas to identify and inventory sources of potentially toxic substances in California. Current activities in this field include:

- Identification of substances to be inventoried
- Literature studies and preparation of preliminary inventories
- Extramural research
- Field evaluation and testing
- Coordination with air pollution control districts
- Utilization of existing organic gas emission data

Identification of Substances

Lists have been established by the EPA and the ARB of more than forty substances of concern because of their potential toxicity. To date, the EPA has identified seven substances as hazardous pollutants under Section 122 of the Clean Air Act (National Emission Standards for Hazardous Air Pollutants). A recently enacted law (AB 1807, Tanner) has added to the State Health and Safety Code, procedures for identifying substances as toxic. No substances have been identified to date.

Present inventory efforts are concerned with a number of substances of high current interest. The Emission Inventory Technical Advisory Committee (EITAC), composed of representatives from the EPA, the ARB, and six of the air pollution control districts in the state, is considering a statewide survey of the following 13 substances:

- Arsenic
- Benzene
- Carbon Tetrachloride
- Chloroform
- Ethylene Oxide
- Ethylene Dibromide
- Formaldehyde
- Methyl Bromide
- Mehtyl Chloroform
- Methylene Chloride
- Perchloroethylene
- Trichloroethylene
- Vinyl Chloride

Literature Studies and Preparation of Preliminary Inventories

A study has been in progress for more than a year to identify sources and compile a preliminary inventory of emissions of selected substances. The inventory is primarily based on information in the literature, but includes applicable information from current research studies and field evaluation and testing. The preliminary inventory is being revised and will include information on the properties, present uses, emission potential, and statewide emissions for twenty-five substances.

Information available in the literature has a number of limitations:

- Information is incomplete.
- Information available is not always for the same year. (Use of some substances has changed significantly in recent years.)
- There are sometimes conflicting information in different references.
- There is a lack of quantitative and source-specific data.

Typical sources of potentially toxic substances that have been identified from the literature and examples of the substances emitted are shown in Figure I.

Extramural Research

Several ARB sponsored research projects, either completed or are in progress, are providing data to support this inventory effort.

- "An Inventory of Carcinogenic Substances Released into the Ambient Air in California," Science Applications and KVB. The study investigated: Arsenic, Asbestos, Benzene, Cadmium, Carbon-tetrachloride, Chloroform, Ethylene Dibromide, Ethylene Dichloride, Nitrosamines, Perchloroethylene, Polycyclic Organic Matter. Tests were concluded on: lead smelters, a steel mill, an asbestos cement plant, and four organic chemical manufacturing plants.

- "Formaldehyde - A Survey of Airborne Concentrations and Sources," Science Applications, Inc.

- "Improvement of Emission Inventories for Reactive Organic Gases and Oxides of Nitrogen in the Sourth Coast Air Basin," Systems Applications. The study will provide improved organic speciation data.

- "Development and Improvement of Organic Compound Emissions," Science Applications, Inc.

Field Evaluation and Testing

The ARB staff has been active in the evaluation and testing of facilities known to emit potentially toxic substances. This activity includes designing test procedures and conducting material balances; conducting tests such as stack and ambient monitoring for Ethylene Oxide, tests on the incineration of waste solvents in the General Portland cement kiln, and monitoring at the BKK and Kettleman dump sites; and evaluating a wet air oxidation unit jointly with the Santa Barbara County Air Pollution Control District.

Coordination with Air Pollution Control Districts

A number of the air pollution control districts in the state have initiated programs to identify and inventory potentially toxic substances. This activity has been coordinated with the EPA and the ARB through the Emission Inventory Technical Advisory Committee.

- The South Coast Air Quality Management District is conducting a survey of approximately 1600 facilities for 20 substances.

FIGURE I

TYPICAL SOURCES OF POTENTIALLY TOXIC AIR CONTAMINANTS

INDUSTRIAL
- CHEMICAL INDUSTRY
  - BENZENE
  - CARBON TETRACHLORIDE
  - CHLOROFORM
  - 1,4 DIOXANE
  - TRICHLOROETHYLENE
- PETROLEUM INDUSTRY
  - BENZENE
  - FORMALDEHYDE
- CLEANING AND DEGREASING
  - METHYLENE CHLORIDE
  - METHYL CHLOROFORM
  - TRICHLOROETHYLENE
- FUEL COMBUSTION
  - ARSENIC
  - BERYLLIUM
  - CADMIUM
  - CHROMIUM
  - FORMALDEHYDE
  - LEAD
  - MERCURY
- METAL SMELTING
  - ARSENIC
  - CADMIUM
  - LEAD

AGRICULTURE
- PESTICIDES AND FUMIGANTS
  - ARSENIC
  - CARBON TETRACHLORIDE
  - ETHYLENE DIBROMIDE
  - ETHYLENE DICHLORIDE
  - METHYL BROMIDE

MOTOR VEHICLES
- EXHAUST AND EVAPORATION
  - BENZENE
  - ETHYLENE DIBROMIDE
  - ETHYLENE DICHLORIDE
  - FORMALDEHYDE
  - LEAD
  - POLYCYCLIC AROMATIC HYDROCARBONS

WASTE DISPOSAL
- HAZARDOUS WASTE LANDFILLS
  - BENZENE
  - CHLOROFORM
  - METHYL CHLOROFORM
  - TETRACHLOROETHYLENE
  - VINYL CHLORIDE
- SANITARY LANDFILLS
  - BENZENE
  - DICHLOROBENZENE
  - XYLENE
- INCINERATION FACILITIES
- CHEMICAL DESTRUCTION FACILITIES

- The Ventura County APCD is conducting a survey of dry cleaners, hospitals, plastic and electronic industries;

- The Santa Barbara County APCD is conducting a survey of 550 companies and is planning to test a dump site in Santa Maria.

- The Sacramento County APCD contracted with KVB to inventory emissions of potentially toxic substances within the county.

- The Bay Area Air Quality Management District has been obtaining inventory data for some potentially toxic substances as part of its normal inventory procedures.

Members of the EITAC are developing a uniform survey format that can be used by the districts to report emission data for potentially toxic substances to the state emission data system.

Utilization of Existing Organic Gas Emissions Data

A potential exists for locating and quantifying some potentially toxic emissions by using the present organic gas data base and speciation profiles. A speciation profile for a process shows what fractions of the total organic gas emissions are various organic compounds. Using the speciation profile and the total organic gas emitted by a process provides an estimate of the emissions for a specific organic substance. Existing speciation profiles include fourteen substances that have been identified as potentially toxic. A preliminary evaluation of benzene emissions using speciation profiles and the 1979 organic gas emission inventory indicates that more accurate speciation profiles are needed.

Work is in progress at the Air Resources Board to establish a data system to store in a consistent manner, the information on emissions of potentially toxic substances that is being compiled by the local air pollution agencies. The system will link to the existing data base of criteria pollutants to utilize existing organic emissions information.

AIR TOXICS IN THE INDOOR ENVIRONMENT

Dr. David Grimsrud
Co-Leader, Building Ventilation and Indoor Air Quality Program
Lawrence Berkeley Laboratory
Berkeley, CA

Dr. Ken Sexton
Director, Indoor Air Quality Program
California Department of Health Services
Berkeley, CA

Summary of Presentation of David Grimsrud
Region 9 Air Toxics Conference
September 13, 1983

Introduction

Prior to any discussion of indoor air quality I must point out that there are no indoor air quality standards that govern general public access buildings. Thus, the definition of toxics used in this conference applies to all pollutants monitored within buildings. This lack of standards forces us to apply occupational or ambient air quality standards to measurement results as reference guidelines. However, this is, at best, a questionable practice and in some cases is clearly inappropriate.

Background

Indoor air quality began to be an issue in the mid-seventies when research scientists began to investigate movement of outdoor pollutants indoors using equipment that had been developed for outdoor monitoring. It quickly became apparent that some pollutants found indoors could not be associated with outdoor sources; since those observations many important indoor pollutant sources have been found.

At approximately the same time energy conservation in buildings began to receive national attention. Since an inexpensive weatherization measure that had large potential energy savings was the reduction of outdoor ventilation air, concern began to be expressed about this measure's impact on indoor air quality. If the pollutant source strength remains constant, a decrease in ventilation rate (the most important pollutant removal process) should increase pollutant concentrations. As a result of these concerns the research program of our group, and others studying indoor air quality, was directed toward two major questions:

(1) What effects do weatherization and/or new building practices have on indoor air quality?

(2) What minimum ventilation rates are required to assure adequate indoor air quality in buildings?

Our work and the work of other research groups have shown that neither question has a unique answer. As a result, the emphasis of our group has shifted to the characterization of the physics and chemistry of pollutants found within buildings. This includes monitoring the concentrations found within buildings--often using instrumentation developed for studies in our laboratory experiments and using procedures developed for our laboratory studies. It does not include health studies, although our laboratory and field monitoring work informs those who study health effects about the concentrations that will be present within buildings. It does support the development of techniques that are specific to a single pollutant.

A major emphasis of our work continues to be the building. The building shell defines the volume and the environmental conditions in which pollutants are released. Changes in building operation, materials used in

building construction, and appliance use all affect pollutant concentrations and transformations.

LBL Research Project Areas

Combustion Products.

In this project we have studied the emission rates of gas stoves, unvented gas and kerosene space heaters, and wood stoves. Using these measured emission rates we have compared concentrations that would be predicted by modeling with concentrations obtained in field measurements made in research houses. These results show that concentrations of oxides of nitrogen or carbon can reach high levels indoors (levels in excess of NAAQS or OSHA standards).

Major questions remain in this area concerning the transport and transformation of pollutants--and ultimately the control of combustion pollutant concentrations.

Radon and Radon Progeny.

In the United States the emission rates of radon from common building materials are too small to explain the radon concentrations seen within buildings. This observation and other direct evidence of radon entry point to the soil as the major source of radon in buildings. Comparisons of radon concentrations and ventilation rates show that the large variability in concentrations seen in buildings is primarily due to source variations rather than to variations in ventilation rates in buildings. These observations define a series of important questions that must be investigated before the problem of radon and radon progeny can be resolved. These include the mechanisms of radon entry into buildings, the behavior of radon and radon progeny in the air after entry, and ultimately the control of these pollutant species.

Formaldehyde and Other Organics.

Work in this area has demonstrated a clear dependence of indoor concentrations on building materials and furnishings found within the space. Many questions remain to be addressed and answered in the study of airborne organics. Included are the detailed identification of source emission rates, the dependence of emission rates on environmental factors, the development of reliable and low-cost methods of sampling the concentrations of organics in the indoor air, and development of an improved understanding of the health effects associated with any single contaminant or combination of these contaminants.

Instrumentation Development.

This is an ongoing part of any research effort and has been an important part of our program. Instruments developed range from the sophistication of an automated device that samples radon progeny remotely under microprocessor control to the simplicity of passive samplers that combine low cost and ease of operation with measurement precision.

Efforts to develop and test passive samplers have been a particular interest for our project. Passive samplers will allow a large-scale survey of indoor air quality in buildings to be conducted. The samplers that are presently available include those that measure radon, nitrogen dioxide, formaldehyde, carbon monoxide, and water vapor. We are currently beginning studies to develop a carbon dioxide sampler.

Major issues yet to be resolved include developing a better understanding of the limitations of passive samplers through field experience and testing. Even more important is the issue of the utility of passive sampler results, i.e., long-term average concentrations. If health risks depend more on short-term peak concentrations than on long-term exposure to some average pollutant concentration, then passive samplers are inappropriate for monitoring purposes. However, their low cost and simplicity make them very attractive for possible future use, particularly for screening large numbers of buildings for potential air quality problems.

Indoor Air Quality Control Techniques.

Our major effort in this area in the past has been the study of ventilation using mechanical systems employing air-to-air heat exchangers that minimize energy use. Construction trends (in colder climates) are moving to tight buildings where ventilation is supplied mechanically. Energy use in these buildings is minimized if the mechanical systems employ heat recovery from the exhaust air. Our group has measured the thermal effectiveness of these systems, their ability to remove pollutants from the air, and ventilation effectiveness (the ratio of air delivered to a space to the amount predicted by the manufacturer). Cost effectiveness studies show that heat exchangers are not always the best solution; their utility depends on the cost of energy, building tightness, and the climate of an area.

The controls project is moving away from studies of ventilation systems to studies that investigate pollutant-specific control techniques. Studies in progress or planning include air washing to remove formaldehyde, ultraviolet photodecomposition to remove formaldehyde, and reactivity studies to control nitrogen dioxide.

Summary

Our group, and others, have shown the importance of indoor pollutant sources in determining indoor pollutant concentrations. While ventilation continues to be an important indoor air quality control technique, one must consider both sources and removal mechanisms to adequately describe air quality within buildings.

INDOOR EXPOSURES TO TOXIC AIR POLLUTANTS

BY

Ken Sexton, Sc.D.
Indoor Air Quality Program
California Department of Health Services
Air & Industrial Hygiene Laboratory
2151 Berkeley Way
Berkeley, California 94704

It is now recognized that air pollution is not exclusively an outdoor phenomenon. Many toxic and hazardous air pollutants are present at elevated concentrations in indoor nonindustrial environments. The implications of this finding for policy decisions about air toxics regulation have not been explored fully. Nevertheless, evidence continues to mount that for a large segment of the U. S. population, exposure to many air pollutants is the result of indoor, not outdoor sources.

IMPORTANCE OF INDOOR EXPOSURES

Concerns about potential public health problems due to indoor air pollution are based on several factors. Most urban residents spend more than 90% of their time indoors (60-70% at home), while some groups, such as infants, the elderly, and the infirm, are indoors virtually all the time. Therefore, even if indoor concentrations are low, they make a significant contribution to time-weighted, integrated exposures.

But indoor concentrations are often elevated in private and public buildings, and may exceed outdoor values by an order of magnitude. The critical role of indoor environments is emphasized by the fact that personal exposure to most air pollutants is not characterized adequately by outdoor measurements. The situation is likely to become more acute due to recent trends toward reduced ventilation in buildings, increased use of synthetic building materials, and increased reliance on unvented space heaters.

A broad spectrum of toxic contaminants arise from indoor sources. Among the pollutants which are typically elevated indoors are aeropathogens, aeroallergens, combustion by-products (e.g., polycyclic aromatic hydrocarbons, NO_2, CO), fiber glass, formaldehyde, PCB's, radon and radon decay products, respirable particles, "sidestream" tobacco smoke, and many volatile organic compounds. Health effects known to be associated with this diverse group of contaminants range from mild irritation of nasal and mucous membranes to irreversible toxic and carcinogenic effects.

Volatile organic compounds are one class of chemicals for which control of outdoor sources has been touted as a means of reducing population exposures. Yet a plethora of potential indoor sources exists, including structural materials (e.g., particle board, plywood), furnishings (e.g., carpet,

furniture), combustion (e.g., unvented space heaters, gas-fired appliances, fireplaces), sidestream tobacco smoke, pesticides, consumer products (e.g., personal care and cleaning products), and human activities (e.g., cooking, hobbies). Among the organic air pollutants which have been measured indoors are aliphatic, halogenated, and aromatic hydrocarbons, alcohols, ketones, esters, monomers, plasticizers, acetaldehyde, acrolein, chlordane, malathion, and dichlorvos.

Because society has been slow to recognize the importance of indoor air quality, there are insufficient data to evaluate health consequences. In many cases it is not feasible to delineate the relative contribution of indoor and outdoor sources to toxic air pollutant exposures. However, information on hand indicates that evaluation of indoor as well as outdoor exposures is essential for realistic health effects assessment. The importance of safeguarding indoor air quality is underscored by the high toxicity of many identified indoor pollutants, evidence of elevated concentrations indoors, and the large number of people potentially at risk.

PUBLIC POLICY ISSUES

For a given dose of a specific chemical, the toxic effects are the same whether exposure occurs indoors or outdoors, all other factors being equal. However, there are critical differences between indoor and outdoor air pollution which have ramifications for policy choices about appropriate public responses.

The rationale for government regulation to control outdoor sources focuses on the issue that those who suffer the effects are not compensated, nor is their interest in cleaner air readily effective in influencing polluters. In economic terms, outdoor air is a "public good" since members of a community breathe basically the same ambient air. Public intervention has been deemed appropriate in the case of ambient air pollution, because 1) no rational individual will attempt to cleanup dirty air over cities since his or her share of the benefits are much smaller than the costs, 2) efforts at voluntary cooperation to reduce pollution are doomed, since those who refuse to contribute can not be excluded from the benefits, and 3) no pollution source will spend enough on abatement in the absence of regulations or legal liability due to the difficulties of collecting from beneficiaries.

Although indoor air quality is often spoken of in a generic sense, there are in fact a wide range of indoor environments. Among important distinctions are 1) occupational, both industrial and nonindustrial, 2) nonoccupational, including residential, commercial, institutional, and public, and 3) transportation microenvironments, including automobiles, airplanes, and subways. It is therefore clear that there are both private and public indoor settings; a fact which may influence decisions about public intervention.

Indoor air in private residences does not have the characteristics of a public good, since the costs and benefits of abatement are internalized within the household. If occupants foul the air in their own home, they are forced to breathe it. If they attempt to improve its quality by increasing ventilation or installing air-cleaning devices, they bear the costs and enjoy the benefits. Prescription of indoor air quality standards and regulations must

confront the fact that households are already making decisions about their own air quality.

However, not all buildings are residences and not all residences are owner-occupied. Air quality in large public buildings shares some characteristics with outdoor air. The case for indoor air quality regulations is much stronger in hospitals and convention halls than in private dwellings. It is common practice to regulate construction and operation of public buildings to ensure that adequate provisions are made for health and safety. In addressing the issue of indoor air quality, decision-makers must remember that the role of government may depend on the degree of "publicness" of a particular building.

CALIFORNIA'S INDOOR AIR QUALITY PROGRAM

Assembly Bill No. 3200 directs the Department of Health Services to coordinate efforts to assess, protect, and enhance indoor environmental quality. Specifically, the State Legislature declared "...that the public interest shall be safeguarded by a coordinated, coherent State effort to protect and enhance the indoor environmental quality in residences, public buildings, and offices in the state." In accordance with the directives outlined in Assembly Bill No. 3200, the Indoor Air Quality Program was established within the Department of Health Services, Air and Industrial Hygiene Laboratory.

The California Indoor Air Quality Program is a multidisciplinary unit responsible for promoting and conducting research aimed at understanding the determinants of healthful indoor environments. The ultimate goal is to assess the nature and magnitude of potential hazards within the State so that health risks can be evaluated rationally. This information is an essential component of policy decisions about the need for public intervention.

SUMMARY

Most of the current discussion concerning control strategies for toxic air pollutants has focused on outdoor sources. It is becoming increasingly apparent, however, that development of an effective program to reduce population exposure must take indoor environments into account. In order to assess health risks, establish suitable standards, and implement appropriate control strategies, information is required about the number of people exposed, severity and pattern of exposures, and dose-response relationships. Evaluation of indoor exposures to toxic chemicals is an integral part of this process.

A more indepth discussion of the issues raised here may be obtained from the following references.

Ken Sexton and Robert Repetto, Indoor Air Pollution and Public Policy, Environment International 8:5-10, 1982.

John Spengler and Ken Sexton, Indoor Air Pollution: A Public Health Perspective, Science 221:9-17, 1983.

A STUDY OF THE RELATIONSHIP BETWEEN CANCER INCIDENCE AND AIR POLLUTION IN CONTRA COSTA COUNTY, CALIFORNIA

Dr. Donald Austin
Chief, Resource for Cancer Epidemiology and California Tumor Registry
California Department of Health Services
Emeryville, CA

ABSTRACT

A study of the relationship of lung cancer incidence in Contra Costa County to ambient levels of air pollution has been concluded. The study was generated as the result of public officals and private citizens groups concerned about reports of elevated lung cancer incidence in the county. It had been suspected by some that the presence of industrial plants in the county, mainly petrochemical refineries, could be a contributing factor. The study, initiated with a grant from the EPA, consisted of five parts.

First, an incidence analysis established that when the county was divided into two parts, the Industrial portion of the county had an excess of lung cancer as compared to the remaining Non-industrial portion. The incidence of lung cancer for the county as a whole was unremarkable as compared to four other local counties.

More detailed information on the patterns of air pollution in the county were obtained in the second phase of the study. Five permanent air monitoring stations and ten temporary stations monitored the levels of 12 air pollutants for a period of one year. These data were incorporated into later phases of the study.

In the third portion of the study, through a correlation analysis of 1970-79 lung cancer rates and various air pollution constituents, a relationship between ambient air SO_4 and lung cancer in males, but not in females, was found to be statistically significant. However, the percent of the working population categorized as blue collar was also associated with lung cancer in males and the previous association between lung cancer in males and ambient air SO_4 levels was eliminated when this third factor was taken into consideration.

Part four of the study was to have consisted of a linkage of occupational group cohorts to registry cancer incidence files but was not conducted for lack of easy availability of occupational group records.

Part five of the study was an analysis of case-control interview data on a final sample of 622 individuals. Demographic, work history, residential history, dietary, and smoking history questions comprised the bulk of the data collected. Analysis of the data indicated that the major contribution to lung cancer in Contra Costa County was due to cigarette smoking.

Further, there was no identified effect on lung cancer risk contributed from any measured constituent of air pollution. Of five broad occupational categories (indicating possible hazardous exposures) none had any significant relationship to lung cancer. Detailed evaluation of the effect of specific occupational groups awaits final analysis.

PROJECT SUMMARY

EPIDEMIOLOGICAL STUDY OF THE INCIDENCE OF CANCER AS RELATED TO INDUSTRIAL EMISSIONS IN CONTRA COSTA COUNTY, CALIFORNIA

Donald F. Austin, M.D., Verne Nelson, Bix Swain,
Linda Johnson, Susan Lum, Peter Flessel

The purpose of this study was to examine the relationship of lung cancer incidence in Contra Costa County to ambient levels of air pollution. It was suspected that the presence of heavy industry in the county, mainly petrochemical plants and oil refineries, could be a contributing factor.

Initially, an incidence analysis established that the Industrial portion of the county had an excess of lung cancer as compared to the remaining Non-industrial portion.

Air pollution patterns were subsequently determined by five permanent air monitoring stations and ten temporary stations which monitored the levels of 12 air pollutants for a period of one year.

By correlating the 1970-79 lung cancer rates for each census tract and tract levels of air pollution constituents, a statistically significant relationship between ambient air SO_4 and lung cancer in males, but not in females, was found. However, when adjusted for the percent of the working population categorized as blue collar, the association was eliminated.

An interview study of 249 cases and 373 controls was then conducted. Demographic, work history, residential history, dietary, and smoking history questions comprised the bulk of the data collected. Analysis indicated that the major contribution to lung cancer in the county was due to cigarette smoking. No significant association between lung cancer risk and measured constituents of air pollution was found. Of five broad occupational categories (indicating possible hazardous exposures) none had any significant relationship to lung cancer.

INTRODUCTION

Contra Costa County, located in the northeastern part of the San Francisco Bay Area, is one of 39 US counties found to have a high mortality rate for specific cancer sites. The fact that the county also has five major petroleum refineries and numerous petrochemical plants, and that 68% of the total stationary air pollution in the Bay Area originates from the county, prompted an epidemiological study of the incidence of cancer in Contra Costa County. The major objective was to determine whether industrial emissions have a measurable effect on cancer occurrence. The study consisted of four parts:

1. A comparison of cancer incidence in heavily industrialized sections of the county to nonindustrialized sections.

2. Ambient air monitoring, consisting of sampling and chemical analysis of components of particulate pollution.

3. Correlation analysis of lung cancer incidence rates with air pollution constituents and census tract characteristics.

4. A case-control study to identify specific environmental factors associated with lung cancer incidence in the county.

METHODS

Cancer Incidence

Cases included for analysis were malignant, invasive, resident incidence cases with primary sites of lung, bronchus or trachea for the period of 1969-1978. Age adjusted incidence rates were generated for the Industrial and Non-Industrial areas.

Air Pollution Monitoring

A total of 15 hi-volume particulate samplers were strategically sited at 13 locations in Contra Costa County and two locations in adjacent counties.

Air particulate material was collected every sixth day at each of the 15 sampling sites from November, 1978 to October, 1979. Particulate matter was analyzed for total suspended particulates (TSP), benzene soluble organics (BSO), sulfate (SO_4), nitrate (NO_3), lead (Pb), selected polycyclic aromatic hydrocarbons (PAH), and mutagenic activity. Standard chemical techniques were used to analyze TSP, BSO, SO_4, NO_3, and Pb. Specific PAH were separated by high preformance liquid chromatography and analyzed using ultraviolet absorption and fluorescence. Mutagenicity was measured using the Ames test.

Correlation of cancer incidence data to air pollution measurements required interpolation of the station data to 115 census tract population centroids using a contour mapping program called SYMAP.

Correlation analysis

Pearson correlation coefficients for census tract data between each air pollutant constituent and the 5- and 10-year average annual age-adjusted lung cancer incidence rates were computed for white males and females (two atypical tracts were removed from the analysis). Partial correlation coefficients for the same data were compared using socio-economic variables as controls.

Case-control Study

A case-control questionnaire study was conducted. All cases of cancer of the trachea, bronchus or lung among black or white residents of Contra Costa County, diagnosed between May 8, 1980 and July 31, 1981, and who were at least 35 years of age and less than 75 years of age at diagnosis, comprised a group of 332 eligible cases. Proxies were interviewed where cases were too ill or were deceased.

Controls were matched to cases of the same race and sex, and 5-year age group in each of 32 age, race, and sex strata. Controls were selected from the general population of Contra Costa County by random digit dialing.

At the end of the matching and data editing processes 19 cases and 37 controls were deleted leaving 249 cases and 373 controls for analysis.

The measure of the respondent's exposure to air pollution was expressed as an estimated cumulative dose for each pollutant, based on the residential history in the county.

The respondents' smoking experience was characterized by several parameters; total smoking duration, total pack years and average packs smoked per day.

The occupational exposure analysis was based on the coding of each work experience using occupation and industry titles in the Census' 1980

Alphabetical Index of Industries and Occupations. Each blue collar job experience was assigned to one of four broad industry categories: construction, petrochemical, metal, and other industries.

The duration of time worked in an industrial category was calculated and accumulated for each respondent.

An asbestos exposure variable was created from various occupational categories. All shipyard occupations plus all other jobs for which asbestos exposures were reported were combined to form a total duration of asbestos exposure per respondent.

Each respondent was assigned a water source based on the water source for each census tract of residency at the time of interview or, for cases, diagnosis.

Certain census tracts in Contra Costa County contain known dumps of toxic or chemical waste. Each respondent was coded to indicate whether or not their census tract of residence contained a dump site.

To evaluate possible response variation among controls, the number of controls expected from each census tract was computed and compared to the number actually obtained. One area of the county was overrepresented and a separate small area of the county was underrepresented so that these responses were appropriately weighted in the analysis.

The amount of alcohol consumed per week was determined by history and formed an estimate of alcohol consumption. A dietary questionnaire provided estimates of weekly consumption of certain dietary items.

Within a particular race, sex and 5-year age group, controls were matched to cases by age using a variable matching ratio. Thus a case may have one or more matched controls.

Analysis of the data was carried out using multiple logistics regression procedures.

CONCLUSIONS

Incidence Analysis

The incidence analysis established that when the county was divided into two parts, the Industrial portion of the county had a 40% excess of lung cancer as compared to the Non-industrial portion in the 1975-79 time period.

Air Pollution Monitoring

The Pb map was consistent with the fact that the largest source of Pb in the area is the automobile and the map conformed approximately to the paths of freeways. Comparison of the BSO and Pb maps suggests the contribution of the automobile to the BSO levels may be significant. The SO_4 distribution differs from the Pb by conforming to the industrial

belt. This is consistent with the fact that SO_2, the precursor of SO_4, is emitted by stationary sources, primarily chemical industries, refineries and power plants, all located along the industrial belt. The patterns of the five PAH are similiar to one another and to lead.

The correlation coefficients between pollutants for the 15 monitoring stations show very similar relationships to those based on the 113 census tracts which provided validation for their use in subsequent correlation analyses.

Correlation Analysis

A correlation analysis of 1970-79 lung cancer rates by census tract and various air pollution constituents showed only one statistically significant relationship. That relationship was between ambient air SO_4 and lung cancer in males, but not in females. However, when controlled for the percent of the population categorized as blue collar workers the relationship was eliminated.

Case-control Study

Using multiple logistics regression analysis, all air pollution constituents were individually reviewed for their relationship with lung cancer. None of the measured air pollutants showed a statistically significant relationship. However, because SO_4 had shown a relationship in correlation analysis, it was included in the study as discussed below.

Because of the relevance of smoking to lung cancer, two statistically significant smoking variables for males ($p<.01$), average packs smoked per day and total smoking duration, were analyzed in conjunction with any other single variable. In this series of analyses only one additional variable emerged as a statistically significant factor in reducing the risk of lung cancer, but only for males. This was an indirect measure of dietary intake of vitamin A: the consumption of green vegetables ($p<0.01$). A similar but not statistically significant effect was found for females ($p<0.16$).

Although no other variables suggested a significant effect on the risk of lung cancer, further analyses were done adding more variables in different combinations, to identify possibly significant relationships obscured in simpler models. In more complex analytical models the effect of SO_4 dose, TSP dose, and other pollutant doses were analyzed separately controlling for the effects of smoking, drinking, diet, occupation and asbestos exposure. Again, no variables for males, other than green vegetables and the smoking variables emerged as statistically significant. For females, one smoking variable, average packs smoked per day, was significant.

The most complex analysis contained all variables which, in simpler models, had shown a statistically significant relationship to lung cancer, or was a known causal factor, or was of particular interest because of previous analyses. This analysis contained a total of 13 variables and

represents a "saturated" model. The model included variables related to smoking, diet, alcohol, asbestos, SO_4 dose, occupation, and water source.

No additional statistically significant relationships with lung cancer risk appeared. Other than smoking, and the one dietary factor for males, no other relationships approached statistical significance.

DISCUSSION

This analysis of case-control data suggests that the major contributor to lung cancer in Contra Costa County is smoking. Further, smoking accounts for most of the previously identified difference in lung cancer incidence between the Industrial and Non-Industrial areas.

There was no identified effect on lung cancer risk contributed from any measured constituent of air pollution. The one air pollutant (SO_4) significantly correlated with male lung cancer incidence in the indirect correlational analysis, had a positive but not statistically significant relationship with lung cancer risk in the case-control analysis only when SO_4 level at the current address was used as the measurement. When a measure of total lifetime dose of SO_4 from Contra Costa County was used, no elevated risk was apparent.

One dietary factor had a significant ($p<0.01$) protective effect for males and a similar but not statistically significant ($p<0.16$) effect for females. This factor, weekly servings of green vegetables, is a crude measure for several dietary constitituents believed to reduce the risk of cancer of several types. Both vitamin A and cruciferous vegetables would be included in this dietary measure. The dietary measure, weekly servings of yellow vegetables, did not discriminate between cases and controls.

None of the occupational categories had any significant relationship to lung cancer risk in males. The occupational categories are very broad and undoubtedly contain specific occupations that are of higher and lower risk. The occupational analysis therefore likely explains less lung cancer than potentially it could. This supposition is supported by the fact that a higher proportion of lung cancer among females is explained in the analytical models than among males. Males would be expected to have a higher proportion of their numbers in occupations with carcinogenic hazards. A more detailed analysis of the effect of various occupations on lung cancer risk is planned.

The effect of asbestos exposure, as measured, did not bear a statistically significant relationship to lung cancer in this analysis. In any subsequent analysis a more quantitative measure of asbestos exposure would be desirable.

There was no apparent effect of source of drinking water or proximity to known toxic waste dumps on the risk of lung cancer.

These data confirm the known causal relationship between smoking and lung cancer. They provide some reassurence that constituents of particulate air pollution do not contribute measurably to the risk of lung cancer. This is consistent with the findings of several other studies. These data provide supportive evidence for the protective effect of dietary factors on cancer risk, a finding consistent with other epidemiologic and laboratory studies. The need for a more detailed analysis of occupation and lung cancer risk is apparent.

AIR EMISSIONS FROM A FORMER DISPOSAL SITE

Kathleen Shimmin
Chief, Field Operations Branch
U.S. Environmental Protection Agency
San Francisco, CA

Air Emissions From a Former Disposal Site and the Process of Solving a Hazardous Waste Problem

The McColl site is located in Fullerton, Orange County, California, adjacent to a residential area which grew up around what was originally vacant land. In the 1940's, acid refinery sludge, from the wartime production of high octane aviation fuel, was deposited in sumps in the ground. Later, from 1951 to 1964, drilling mud was placed over the sludge to cover the sumps. The public perception of problems at the site intensified as housing development increased in the vicinity. Table One, Chronology of McColl Site, chronicles the history of the site development and subsequent measures to remedy problems.

Because of the complex and inter-related nature of the issues which led to identification of McColl as a significant hazardous waste site in California, a group was formed representing all pertinent governmental agencies, land owners, and potentially responsible industries. This group formalized its existence through memoranda of understanding and identified the McColl solution in three phases: Phase I, Characterization of the Site; Phase II, Selection of Remedial Alternatives; Phase III, Site Cleanup. Voluntary funding for Phase I, in excess of $1 million was provided by industry and the State of California. Phase II was funded by the State. Funding for Phase III is being negotiated. At each step, the coalition of entities tried to anticipate problems and make orderly plans for timely solutions. Public review of progress was a key element to the process.

As a result of the site characterization studies, which focused on air quality, odors, water quality, soil and waste description, and identification of health symptoms, it was determined that odor, sulfur dioxide, and benzene were the emissions of concern (Table Two: Air/Odor Results, Undisturbed Site). These three factors had to be controlled in any site remedy. The remedial alternative selected by the California Department of Health Services - excavation of the waste - was first pilot tested to determine that these factors could be controlled to a level satisfactory for the protection of public health (Table Three: Findings during Pilot Excavation).

As the final remedy is approached at this site, there are inescapable conclusions which may be drawn from the process: 1) technical problems in a vacuum usually may be solved without incident: it is rare, however, that such a situation exists with a significant hazardous waste site - the public has expectations of success, wants to scrutinize the process and may have an inherent distrust of motivations for government and industry; 2) the key is to lay out an orderly process of problem assessment and to make practical estimates of schedules and costs. All of this must be done with public involvement at each stage and education of everyone so that options are understood and reasonable choices undertaken.

Prepared by: Kathleen G. Shimmin, Chief, Field Operations Branch
Toxics & Waste Management Division, EPA Region 9
October, 1983

1942-46	Refinery Acid Sludge Disposal
1951-64	Drilling mud cover placed
1960	Golf course built
1968	Residential development to east of site
1970's	Ownership changes
1980	Residential development east, south, north
1980	Government conducts abbreviated study of site
Nov. 1980	Public hearing
Dec. 1980	EPA sends information request to potentially responsible parties
1981	Negotiations --> MOA (Phase I)
	Site characterization
	Technical Study plans developed
	Process defined and initiated
Jan.-Feb. 1982	$1 million committed and contractor selected
Feb. 1982	Public review of plans
Mar. 1982	CA Hazardous Site List published. McColl is #1
Apr.-Aug. 1982	Technical Studies conducted
	(Site characterization)
Sept. 1982	Phase II MOA. Evaluation of remedial alternatives
Apr. 1983	Alternative selected
	Pilot excavation conducted
Summer 1983	Early preparations commence
	Environmental assessment report completed
Fall 1983	Cleanup to commence (Phase III)

Table One: Chronology of McColl Site

Findings: Odor

Components include: aromatics, tetrachloroethane, phenols, ethylbenzene, alkenes, cyclic compounds, tetrahydrathiophene, carbon disulfide

Identifiable pattern for "McColl Odor"

Findings: Air

Sulfur Dioxide (100 Samples)

Background, ambient: 0.012 ± 0.022 ppm (v)
Site concentrations: 96% of samples less than two times background. Average concentration is 0.011 ppm (v)

Total Hydrocarbon (100 Samples)

Background: 2.0 ± 0.7 ppm (v)
Site: 100% of samples less than twice background

Table Two: Air/Odor Results. Undisturbed Site. McColl

Findings, Pilot Excavation

- Odor can be controlled
  Baffles, shields, foams, covering used

- Backup system for contingency
  Tent enclosure

- Estimated Magnitude
  Maximum: 200,000 cubic yards material
  (approximately 8000 truckloads)

Table Three: Findings, during Pilot Excavation

EMISSIONS FROM THE ASARCO COPPER SMELTER IN TACOMA, WASHINGTON

Michael Johnston
Chief, Air Operations Section
U.S. Environmental Protection Agency
Seattle, WA

Dana Davoli
Environmental Scientist
U.S. Environmental Protection Agency
Seattle, WA

Arsenic Emissions from the ASARCO Smelter Tacoma, Washington

Presented by
Michael M. Johnston
Dana Davoli

US Environmental Protection Agency
Region 9
Air Toxics Conference

September 13-14, 1983

San Francisco, California

Arsenic Emissions from the ASARCO Smelter in Tacoma, Washington

On July 11, 1983, the US Environmental Protection Agency (US EPA) proposed an arsenic NESHAP (National Emission Standard for Hazardous Air Pollutants) under Section 112 of the Clean Air Act. This NESHAP was proposed for three industrial categories, copper smelters processing high-arsenic feed ore, copper smelters processing low-arsenic feed ore, and glass manufacturing plants. EPA's approach in developing this NESHAP was to require at a minimum best available technology (BAT) for control of arsenic emissions at source categories estimated to cause a significant public health risk. More stringent controls could be required if necessary to prevent unreasonable health risks remaining after BAT (taking costs and technical feasibility into account).

The only facility in the first industrial category (high-arsenic copper smelters) is the ASARCO smelter in Tacoma, Washington. It was built in 1890 and operated as a lead smelter until ASARCO bought it in 1905 and converted it to a copper smelter. This copper smelter processes copper ores (many of which are from foreign sources) with an average arsenic content of 4% compared to the other copper smelters in the US which use feed ores with less than 0.6% arsenic.

EPA has estimated that current emissions of arsenic from this facility are about 311 tons per year - 165 tons per year from stack emissions and the rest from fugitive sources.* Proposed BAT, which is hooding over one of the processes at the plant to capture fugitive emissions, is projected to reduce arsenic emissions from 311 to 189 tons per year.

Several epidemiological studies done on workers, including those at the ASARCO facility, indicate that exposure to airborne arsenic causes respiratory cancer. Because it is a carcinogen, EPA has presumed that arsenic is a no-threshold pollutant and that effects may occur at any level of exposure. The risk assessment for residents living near the smelter was extrapolated from the cancer risks seen in the workplace at higher levels of arsenic exposure, using a linear model. Estimates of exposure in the population around the smelter were developed from population data and the projected ambient air concentrations calculated from dispersion modeling. It was estimated that in a population of 368,000 people living near the smelter the excess lung cancer cases expected to result from ASARCO emissions ranged from 1.1. to 17.4 per year before BAT is installed to 0.21 to 3.4 following BAT. Lifetime risks for the highest calculated level of exposure (30 ug/m^3) was 9/100 before controls and 2/100 after BAT. Because of the assumptions made (e.g., linear extrapolation model), these estimates are thought to be conservative and indicative of upper bound life-time cancer risks. There is also much uncertainty in these numbers because of the difficulty in obtaining fugitive source emission data and because many assumptions must be made in developing the risk assessments.

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* These estimates will likely be lowered based upon additional testing done at the facility during September and October.

Much attention has been focused upon the arsenic NESHAP for the Tacoma facility because the estimated residual health risks remaining after BAT are high relative to those estimated for other sources regulated by NESHAPs. Additionally, William Ruckelshaus, the Administrator of EPA, has decided to involve the public more in the risk management decisions made by EPA. The arsenic NESHAP is the first such regulation targeted for enhanced public involvement, with EPA's efforts directed thus far on the ASARCO facility. To involve the public, EPA has put much effort into press releases and other published material, attempting to explain technical information and the decision-making process in terms the public can understand. Three public workshops were conducted to present the data and answer the public's questions and numerous presentations were made before interested groups. Although it has been reported in the press that EPA has asked the citizens living around the smelter to "vote on the issue of health versus jobs", this is not the case. This decision will be made by the Administrator of EPA alone. The purpose of the workshops and other public programs is to provide as much information to the public as possible so that their comments will be made with the full knowledge of the technical and other issues upon which the regulatory decision will be made.

The proposed arsenic NESHAP deals with controls of current emissions of arsenic from the ASARCO smelter. However, potential problems also exist in the community surrounding the smelter as a result of historic emissions of arsenic. Deposition of arsenic has resulted in contamination of soils, household dusts, and vegetables, with the highest levels occurring closest to the smelter. For example, recent sampling of surface soil close to the facility has shown soil arsenic levels of several thousand parts per million (ppm) while garden soil (which is tilled and often mixed with non-soil nutrients) is at levels of several hundred ppm. The most recent analyses have shown that the average arsenic content of vegetables from these gardens is about 24 ppm, while maximum values of over 400 ppm have been found. Household dust has been shown to contain levels of arsenic as high as 4641 ppm. Analyses of urine samples from children living near the smelter also show that arsenic levels are significantly above normal. Average urinary arsenic levels have ranged from 20-300 ug/l (micrograms per liter) with maximum levels up to 890 ug/l. (Background levels for unexposed populations are usually less than 25 ug/l).

The flow diagram shown in Figure 1 illustrates the pathways and routes of exposure that may be responsible for the increased arsenic body burden in these children. In addition to inhalation of recently emitted arsenic, inhalation of resuspended soils and dusts are also possible. Contaminated soils and dusts are of particular concern for young children because they ingest small amounts in normal hand-to-mouth activity. It has been estimated that children with pica, an abnormal craving for dirt, can ingest several grams of soil a day. Studies around lead smelters have confirmed that this can be a significant exposure route for children. Finally, ingestion of contaminated vegetables and water are potential sources of arsenic, although studies thus far do not show problems with drinking water supplies in the smelter area.

While current arsenic emissions are to be controlled through the arsenic NESHAPs, the potential problems resulting from past emissions are being dealt with through EPA's Superfund program. The Washington Department of Ecology (WDOE) under a cooperative agreement with EPA, is the lead agency in these Superfund efforts and is working with EPA and the state and local health agencies to design the investigations discussed below.

The high urinary arsenic levels of children living near the smelter show that they are exposed to arsenic, but the pathways leading to this exposure are not clear. These pathways must be determined before decisions can be made about implementing remedial actions which will result in lowered urinary arsenic levels. For example if current emissions are a significant source, then controls on these emissions at the plant are appropriate. If resuspended soil is an important exposure source, it may be necessary to remove soil or cover it with sod or paving. The Superfund investigations are being designed to provide data on sources of exposure which will in turn be used to plan remedial measures.

Although several samplings of soils, vegetables and urine have been made in the past, much of this data may not reflect the current situation and was not collected in a way that answers the questions on exposure pathways. Therefore several types of additional studies have been proposed and are in the design stage, including an Exposure Assessment Study. (See Table 1 for examples).

The Exposure Assessment Study will be patterned after those already conducted around several lead sources in the US. It involves taking measurements of contaminants in various media (e.g., soil, dust) concurrently with measurements of body burden (urinary arsenic in this case). Multivarient statistical techniques will then be used to attempt to correlate excess urinary arsenic levels with contamination levels in the different media. Studies such as these can include measurements of contaminants in soils, house dusts, and vegetation. To obtain an estimate of the amount of contamination on children's hands, hand loading studies are done. This involves dipping children's hands into a dilute acidic solution (about the pH of vinegar) and analyzing the solution. The Center for Disease Control is now working with WDOE, EPA and the local health agencies to design and implement this Exposure Assessment for the ASARCO area.

The State health department is also assessing the need for further epidemiological studies. Two lung cancer mortality studies done in the Tacoma area did not demonstrate an increased risk for persons living near the smelter, however this effect is probably too small to study epidemiologically. Blood analyses and hearing tests on children attending the school near the smelter also appear normal.

As can be seen from this discussion, emissions of arsenic from the ASARCO smelter have resulted in potential problems due to air pollution as well as to contamination of other environmental media. This situation stresses the need for pollution control agencies to take a more integrated approach in dealing with toxicants.

Figure 1

Conceptual Framework for Arsenic Exposure

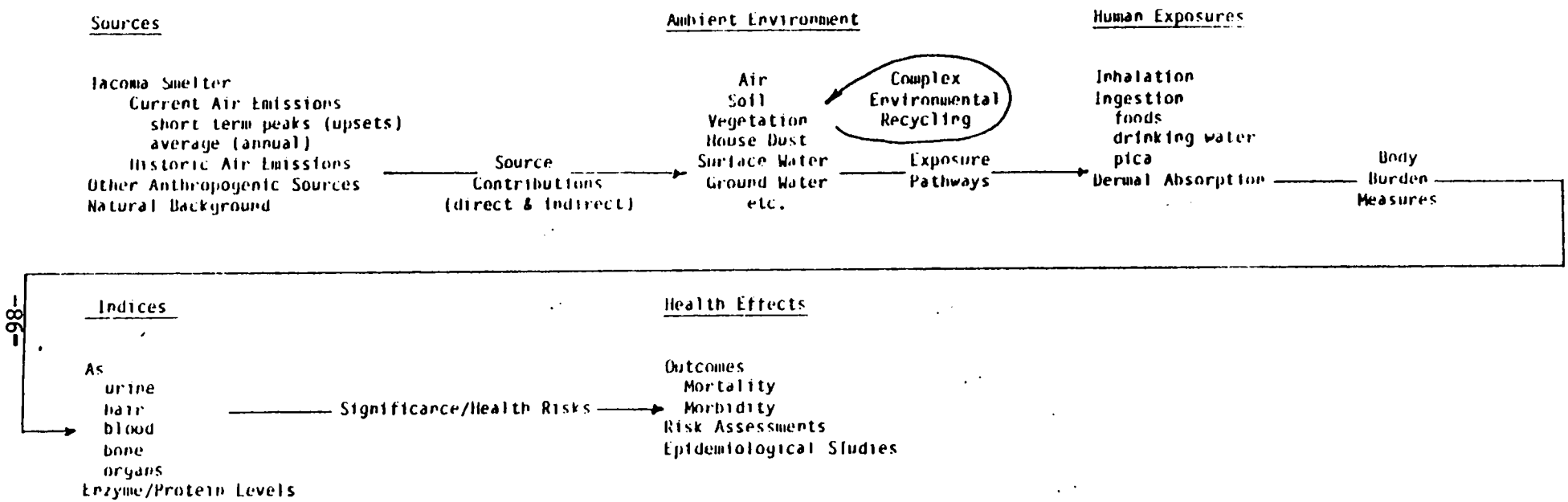


Table 1

Examples of Planned Studies

Exposure Assessment Study - May include measurements of contaminants in soils, vegetables, household dusts and air concurrent with measurements of body burden levels (such as urinary and hand loadings). This is done for a sampling of residents in the affected areas. Multivariate statistical analysis is performed on the resulting data to delineate and quantify sources of exposure and to predict the effectiveness of different remedial actions (Already completed or in progress at smelters in Texas, Idaho and Montana).

Deposition Monitoring - Use of acid precipitation equipment to determine the level of current deposition of arsenic occurring near the smelter. Related to past vs. present smelter operation issues as well as potential remedial actions involving soil clean up.

Soils - Isopleth Study - Soil sampling of areas near the smelter to determine the geographic extent of the most contaminated areas and the areas that may require remedial action.

Drinking Water Survey - Sampling and analyses of well water cisterns and other drinking water supplies in areas of high arsenic in soil or air.

Soil Leaching Tests - Leaching tests on soils with high contamination levels to determine potential for environmental movement, especially to groundwater.

Source Apportionment Model - Uses chemical mass balance approaches to determine the contribution of specific emission points within a facility to ambient pollutant levels as well as the contribution of resuspended contaminated soils and dusts. Distinguishes between air contamination due to current vs. historical deposition.

TOXIC AIR POLLUTION IN HENDERSON, NEVADA

Michael Naylor
Director, Air Quality
Clark County Health District
Las Vegas, NV

TOXIC AIR POLLUTION IN HENDERSON, NEVADA

The first part of the presentation consisted of aerial photographs of the Southeast Valley cloud, as observed by a helicopter traversing from downtown Las Vegas to Henderson. The slides displayed the low vertical depth of the cloud and its proximity to the Industrial Complex.

I discussed some of the examples of complaints we have received from citizens, which include: odor, eye burning, visibility and breathing difficulty, and frustration with continuing air pollution.

The toxic air pollutants in the Southeast Valley consist of:

chlorine
hydrogen chloride
ammonia
Southeast Valley cloud
peroxyacetylnitrate (PAN)
peroxybenzoylnitrate (PB_zN)
formaldehyde

The criteria air pollutants of concern are:

ozone, and
total suspended particulate

Chlorine is important because: it is a precursor for ozone, eye irritants hydrochloric acid and nitric acid, and it contributes to odor occurrences.

Ozone levels have exceeded the standard, and we have determined that chlorine emissions cause the unusual winter-time morning ozone excursions. The key component in the ozone generation is the photolysis of chlorine molecules (emitted from the Industrial Complex) into radical chlorine atoms. The radical chlorine atoms attack hydrocarbon molecules and remove hydrogen atoms, yielding a reactive hydrocarbon radical and hydrogen chloride. These reactive hydrocarbons then initiate the conventional ozone generation cycle which involves the oxidation of NO to NO_2. Similar reactions lead to the formation of eye irritants PAN and PB_zN.

Some of the principal aerosol components in the Southeast Valley cloud include: ammonium, nitrate, chloride, organic carbon, and elemental carbon. We have observed that ammonium and Bscat (light scattering measured by a nephelometer) are correlated with an r of approximately 0.9.

Visual ranges asociated with the cloud occurrence vary from two to twenty-four miles. We believe the primary components responsible for visibility impairment, in order of importance, are: ammonium nitrate, ammonium chloride, elemental carbon, and ammonium sulfate. The ammonium nitrate results from the combination of ammonia and nitric acid. The nitric acid results from oxidation of nitrous oxide initiated by chlorine photolysis. One of the major research problems remaining for the cloud is to develop a model which relates precursor emissions to cloud intensity.

Chlorine emissions from the Industrial Complex have been dropping over the last eight years, from roughly 400 lbs/hr to approximately 25 lbs/hr. Ammonia emissions from documented sources have declined from about 15 lbs/hr to about 4 lbs/hr. However, there appears to be an unknown source of ammonia emissions (near the Industrial Complex) which seems to emit approximately 30 lbs/hr. As a result of these emission reductions, we have seen air quality improvements since 1980 for ozone, eye irritation, and odor complaints. However, there has been no improvement for the cloud occurrence and intensity.

Our work plan for 1983 and 1984 is to concentrate on several actions which could result in eventual further abatement of the air pollution problems, with particular emphasis on improvements in the cloud. These will include: smog chamber experiments; short-term low production runs at the remaining source of chlorine emissions; continuous in-plant monitoring for fugitive ammonia emissions; adopting a regulation requiring LAER for chlorine-emitting sources; and continuing ambient monitoring for aerosol compounds and gaseous pollutants.

EMISSIONS FROM THE SEMICONDUCTOR INDUSTRY

Milton Feldstein
Air Pollution Control Officer
Bay Area Air Quality Management District
San Francisco, CA

BAY AREA AIR QUALITY MANAGEMENT DISTRICT

Emissions From The Semiconductor Industry

Milton Feldstein
Air Pollution Control Officer

The semiconductor industry emits precursor organic emissions which contribute to ozone standard exceedances. Because of chemicals used by this industry, exotic gases and chlorinated solvents which are potentially of concern as air toxics are also emitted. Chlorinated solvents are used extensively in cleaning processes during semiconductor manufacture. The exotic gases such as phosphine, arsine, silane and diborane are used as "dopants" in the semiconductor manufacturing process. The District adopted Regulation 8, Rule 30 to control the precursor organic emissions from the semiconductor industry. This rule affects approximately 200 companies in the Bay Area and will accomplish a 3.5 ton/day reduction in precursor organic emissions by requiring 90% control from photoresist processes and controls for solvent sinks. The rule includes a provision to allow for an alternative compliance plan (bubble). This reduction of 3.5 tons/day ispart of the 1982 Plan's overall reduction of 85 tons/day. All large sources of precurors have already been controlled. We are now controlling the smaller sources and these controls are generally more expensive in terms of cost per ton of emission reduced.

With respect to toxic emissions, a determination needs to be made as to what substances need to be controlled. This identification needs to define threshold levels where possible. Federal and state agencies have the resources to do this. Once a list of air toxic contaminants is defined, then local air pollution control districts can proceed with the development of rules to control emissions of these substances. The technology to control emissions, especially the organic emissions, is available in the form of incineration or carbon absorption. This is a new area for air pollution regulatory agencies. The quantities of toxic air contaminants emitted is very small when compared with emission of criteria pollutants.

The Bay Area Air Quality Management District is currently conducting source tests to speciate the organic emissions from semiconductor sources and to quantify the emissions of arsine, phosgene, and HCl. The second phase of this program is to extend this source testing to other source categories as well as other compounds. This data will serve as the beginning of an emission inventory data base for air toxic contaminants.

Control of sources of these emissions will occur after the regulation development process is completed. This process for air toxic contaminants begins with the identification of the toxic contaminant at the state/federal level (ARB, DOHS, EPA). Once this occurs, then local districts can begin the development of rules to control the emission of the contaminant. This involves a participatory process which includes the affected sources as well as the public.

This is a logical way to proceed. We need to begin on a compound by compound basis to control these contaminants.

SEMICONDUCTOR MANUFACTURING

Chlorinated solvents are primarily used in semiconductor manufacture as surface cleaners and strippers; they are also used as diluents for surface coatings.

Hydrocarbon solvents (both chlorinated and un-) are used to clean and prepare wafer surfaces before virtually every step in the fabrication process. Strict quality control requirements restrict the reuse of cleaning solvents, resulting in high volumes of solvent usage. Used solvents are reclaimed and sold to other industries; most semiconductor firms will use nothing but virgin solvents.

Cellosolves are non-chlorinated hazardous solvents used as diluents for positive photoresist. Low volatility and low precursor organic emission rates make positive photoresist preferred over negative photoresist, at least from an ozone production standpoint.

Various chlorinated solvents are used as diluents for negative photoresist.

An average company uses about 30,000 gallons/yr of chlorinated (non-precursor) solvents; it emits about 3 tons/yr of atmospheric emissions.

Process Description

The manufacture of electronic devices from raw materials involves many steps (Figure 1). Toxic hydrocarbon solvents are used during some of these operations to act as a carrier or diluent for coatings and to strip material from the surface of the circuits. Most of the solvents used are collected and reclaimed or disposed of. A fraction evaporates and is exhausted to the atmosphere.

Wafer Production

CRYSTAL GROWTH: Molten silicon is grown into cylindrical ingots; a tiny crystal is used as a "seed" to allign the crystal lattice, making the entire ingot one single crystal.

WAFER MANUFACTURE: The ingot is sliced with a diamond saw into round, ultr-thin wafers and polished to a perfect mirror finish.

Integrated Circuit Fabrication

OXIDATION: The wafer is exposed to pure oxygen at an elevated (1200 C) temperature. A layer of silicon dioxide (SiO_2) grows on all sides of the wafer.

PHOTORESIST APPLICATION (Negative Photoresist): The wafer is coated with photoresist, an emulsion that hardens when exposed to ultraviolet light. This process frequently employes one or more cleaning steps, using a hydrocarbon solvent to remove contaminants and prepare the surface. The photoresist emulsion may contain solvents as well.

PHOTORESIST EXPOSURE: A glass mask is alligned with the wafer and ultraviolet light is projected through the mask. The shaded areas on the mask prevent light from reaching portions of the wafer; photoresist in these areas stay soft. Exposed photoresist hardens.

PHOTORESIST DEVELOPING: The wafer is washed in a solvent that removes the soft photoresist but leaves the hardened resist on the wafer. The oxide layer on the wafer surface is exposed wherever soft resist is removed.

ETCHING: Exposed oxide surface is removed using either an acid bath or a plasma etcher, revealing the original silicon surface; the oxide now forms a stencil of the mask pattern. The remaining photoresist is removed.

DIFFUSION: The wafer is placed in a diffusion furnace and exposed to dopant gases (phosphorous, arsenic, antimony, etc.) at high temperatures. The dopant atoms enter the exposed silicon, but are blocked by the oxide stencil.

EPITAXIAL GROWTH: The wafer is exposed to silane gas at high temperatures; a layer of silicon is grown over the entire wafer surface.

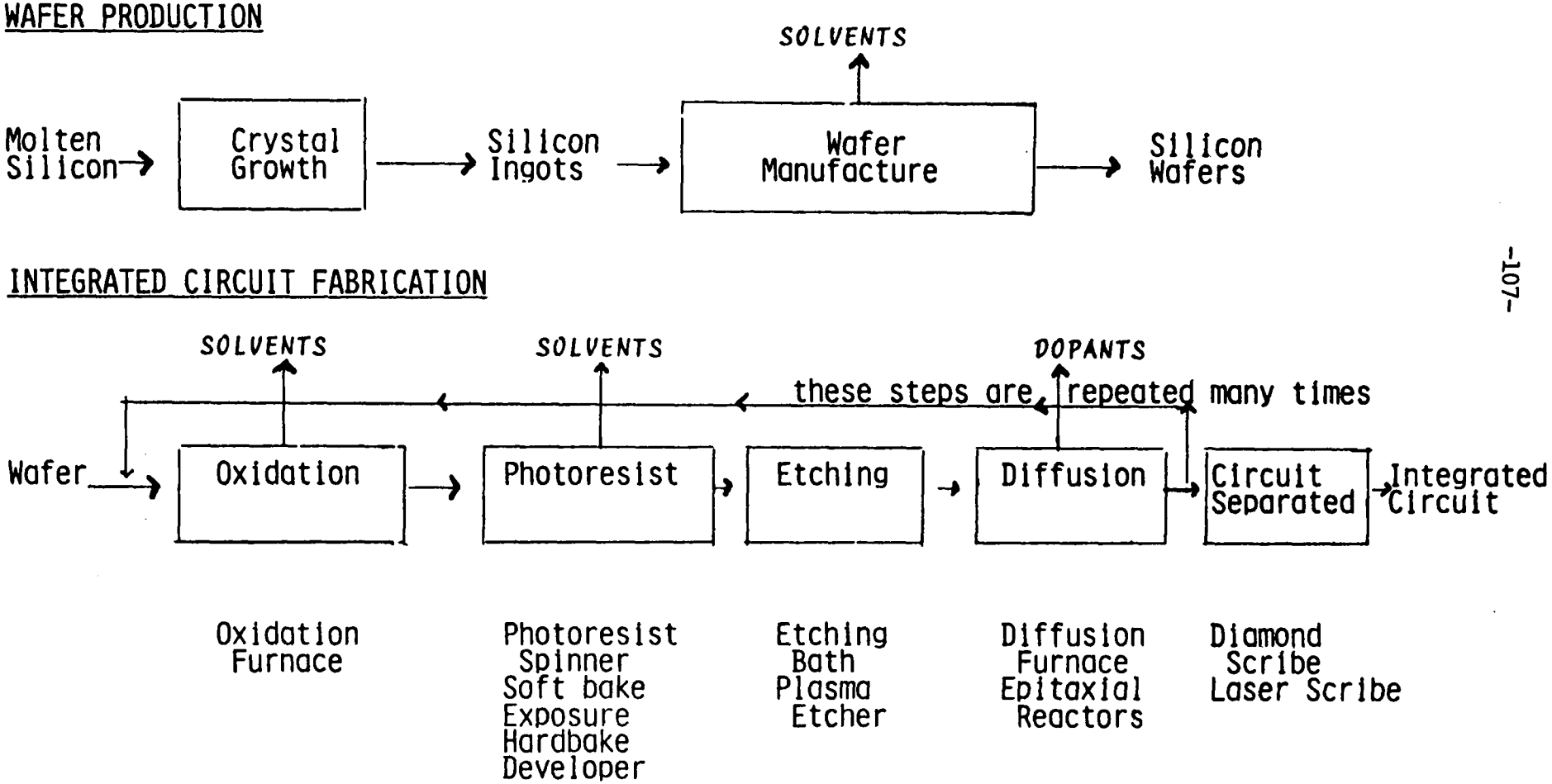
The steps will be repeated many times during integrated circuit fabrications; toxic organic solvents are used as surface cleaners, photoresist strippers and photoresist carriers throughout the operation.

BAAQMD 9/14/83

MF:gp

FIGURE 1

SEMICONDUCTOR MANUFACTURING



BAAQMD 9/14/83

VOLATILE ORGANIC EMISSIONS FROM LANDFILLS

Ed Camarena
Director, Enforcement Division
South Coast Air Qaulity Management District
El Monte, CA

South Coast
AIR QUALITY MANAGEMENT DISTRICT
9150 E. FLAIR DRIVE, EL MONTE, CA 91731

CONTROL OF VOLATILE ORGANIC EMISSIONS FROM LANDFILLS

PRESENTED SEPTEMBER 14, 1983

AT THE

AIR TOXICS CONFERENCE

SPONSORED BY

EPA, REGION IX

IN

SAN FRANCISCO, CALIFORNIA

BY

EDWARD CAMARENA

DIRECTOR OF ENFORCEMENT

SOUTH COAST AIR QUALITY MANAGEMENT DISTRICT

CONTROL OF VOLATILE ORGANIC EMISSIONS FROM LANDFILLS

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SOUTH COAST AIR QUALITY MANAGEMENT DISTRICT

INTRODUCTION

Traditionally air emissions have been regulated from the perspective of their contribution to the overall air pollution problem. The control efforts have been primarily focused on at those air contaminants for which either state or federal ambient air quality standards have been adopted. In recent years however, attention has been increasingly focused on toxic or hazardous air contaminants which may not be a region wide problem but which have a potential for creating a localized problem.

Included in the mission of the South Coast Air Quality Management District is the abatement of emissions of toxic and hazardous air contaminants in order to protect public health and welfare. This is done through:

A. Enforcement of applicable sections of the California Health and Safety Code and District rules and regulations;

B. Enforcement of National Emission Standards for Hazardous Air Pollutants (NESHAPS). These have been incorporated into District rules,

C. Enforcement of the state and District air pollution emergency plan, and

D. Assistance rendered to other agencies in the event of a spill of toxic or hazardous materials which may become airborne.

This paper will review some of the South Coast Air Quality Management District's efforts to control toxic and hazardous air contaminates from landfills.

TOXIC DUMPS

The toxic/hazardous nature of certain industrial wastes and their impact on the environment is only in recent years being recognized. Wastes from refineries, chemical plants, etc., have been deposited in convenient "rural" sites in the past without significant controls or consideration for their ultimate fate. As a result of our growing population, these formerly rural sites are now, in many instances, immediately adjacent to residential areas and have caused a myriad of problems.

Wastes at these sites may pose water and air pollution problems depending on the circumstances. Although there are a number of potential mitigation measures the most practical and effective seem to be;

A. Encapsulation, or

B. Excavation followed by proper disposal at an approved site.

Recent South Coast Air Quality Management District experience indicates that excavation is the method preferred by most regulatory agencies and the affected public. This method however, has been shown to cause some very significant though temporary air pollution problems including;

A. Temporary illness (nausea, headache) of hundreds of people and

B. Odor complaints as far as five miles downwind.

Refinery wastes have been most often identified as the problem for sites in the South Coast Air Quality Management District and the contaminants posing the greatest short term hazard from these sites have been identified as sulfur dioxide and tetrahydrothiophenes.

The South Coast Air Quality Management District has recently adopted Rule 1150, Excavation of Landfills which requires that prior to any landfill excavation, that an air pollution control plan be submitted and approved by the District. This provides the mechanism to require proper site content characterization and development and implementation of air emission mitigation measures prior to the start of a project. Depending on the type of materials to be excavated the plan may provide:

A. Emission mitigation measures such as workface size restrictions, truck covering requirements, use of foams to blanket workface, etc.,
B. Offsite monitoring,
C. Work stoppage criteria based on monitoring data and odor level,
D. Public notification and complaint hotlines,
E. Evacuation contingency plans.

The planning process for excavation of a site is a multi-entity activity usually involving the South Coast Air Quality Management District, state and county departments of health services, the Regional Water Quality Control Board, the State Solid Waste Management Board, other city/county agencies as well as the property owners, other responsible parties and the affected public. In an extreme case as the McColl site in Fullerton, the site characterization and mitigation planning process may take two or more years

and involve over a million dollars in expenditures before excavation, encapsulation or other mitigation work actually begins.

During an excavation, South Coast Air Quality Management District enforcement personnel are present on-site at all times and are responsible for assuring all excavation plan requirements are carried out and may require work stoppage when predetermined odor levels or air contaminant concentrations are reached. In addition, SCAQMD technicians, chemists and meteorologists provide a variety of services such as quality assurance checks on monitoring (usually done by contractor), odor dispersion estimates and stability and wind pattern predictions (to assist in daily activity planning). Depending on the nature and size of the site, a properly controlled excavation may take from a few days to several months of actual work.

ACTIVE LANDFILLS

Active landfills, whether they be Class I (can accept toxic/hazardous wastes) or Class II (cannot accept toxic/hazardous wastes), can be significant air pollution problems if not carefully managed. Below are two case histories describing the problems encountered at active landfills and the actions taken by the SCAQMD.

BKK LANDFILL (odors and vinyl chloride)

In October 1980, odor complaints from residents in the vicinity of the BKK Landfill (Class I) in West Covina prompted the District to issue violation notices alleging violations of Rule 402 (nuisance) and Section 41700 (nuisance) of the California Health and Safety Code. Odors emanating from the landfill were determined to originate almost entirely from the anaerobic

decomposition of wastes within the fill rather than directly from the daily disposal activities. The District required expansion of an existing gas collection and incineration system composed of a series of gas wells venting to a flare. Testing of the composition of the collected landfill gases revealed concentrations of vinyl chloride ranging up to 2,000 ppm. Although the collected gases containing high concentrations of vinyl chloride were efficiently destroyed by the flare, it was known that not all of the landfill gas was being collected and that emissions from the landfill surfaces was still a problem.

The South Coast Air Quality Management District immediately began monitoring ambient air in the residential areas to determine whether the remaining uncontrolled emissions could result in an exceedance of the ambient air quality standard for vinyl chloride. It was found that the vinyl chloride standard was being exceeded off-site about fourteen days a month and the maximum 24-hour average concentration was 0.05 ppm (five times the air quality standard).

The District immediately notified the state and local departments of health services and requested an evaluation of the data. A senior official of the Epidemiological Studies Section of the California Department of Health Services stated in a July 1981 letter to the District commenting on the vinyl chloride measurements taken in May and June 1981 near the BKK landfill: "We believe that these levels of vinyl chloride pose no imminent health hazard to the surrounding population, but they are of sufficient concern to require that effective mitigation measures be taken at the earliest possible time."

An interagency task force was created to develop a program to update emission control efforts in the West Covina landfill area. The task force, which included the District, the city of West Covina, Los Angeles County Health Department, State Solid Waste Management Board, State Department of Health Services and the Regional Water Quality Control Board began meeting regularly in May 1981 to develop ways to further reduce odors and emissions of volatile organic compounds, including vinyl chloride, at the landfill site. BKK Corporation also was prohibited by the State Department of Health Services from accepting industrial wastes containing vinyl chloride.

The task force implemented a program of additional gas well installation and waste gas incineration designed to reduce the concentration of odorous gas emitted to the atmosphere. Such incineration also controls vinyl chloride emissions. Since the implementation of this program in 1981, there have been fewer odor complaints and vinyl chloride concentrations have been reduced significantly. Whereas the standard was exceeded about fourteen days per month in 1981, it is now exceeded about two days per month and the maximum concentration detected in recent months is one fifth that found in 1981.

During July-October 1982, the District, in cooperation with the State Department of Health Services and the California Air Resources Board, conducted an extensive monitoring project to determine the concentration and health impact of other potentially toxic emissions from the BKK landfill. It was concluded that certain compounds were present at levels higher than at the selected control site (Pico Rivera). The State Department of Health Services concluded that:

A. Even at the highest observed concentrations, that the substances measured were present at concentrations well below their threshhold for toxic, non-carcinogenic action,

B. At the low level of exposure that it is not possible to calculate excess cancer risks. However, worst case estimates of the individual cancer risks suggests that those living immediately adjacent to the landfill may have accumulated excess risks to date of 5/100,000,

C. Based on the estimated exposed population within a one-mile radius, no additional cases of cancer are expected from exposure to date and exposure levels are declining, and

D. The individual cancer risks are at a relatively low level and do not constitute a public health emergency.

E. Since the stations around the site are not known to be near other sources of emissions of these compounds (dry cleaning establishments, plastics products manufacturers, metal finishing industries, etc.), the data suggest that the BKK site is a source of these compounds. The elevated levels of the chlorinated compounds in particular indicate a need for mitigation measures such as expansion of the landfill gas gathering system, upgraded maintenance programs, and changes in handling of wastes containing these compounds.

F. Further action is also mandated by the continued, periodic exceedences of the State's Ambient Air Quality Standard for vinyl chloride of 0.01 ppm (10 ppb).

The South Coast Air Quality Management District has recently filed petition for an Order for Abatement which would:

A. Require a plan to expand existing gas colllection and incineration system to assure no further exceedances of vinyl chloride ambient air quality standard,

B. Require implementation of the plan as soon as possible after approval by Executive Officer and according to a schedule specified by Hearing Board,

C. Require BKK to contract for monitoring for vinyl chloride, and

D. Require BKK to install monitors within fill to check effectiveness of gas collection system.

OPERATING INDUSTRIES, INC., LANDFILL

On April 5, 1983, the District Hearing Board issued an Order for Abatement against Operating Industries, Inc. (OII), the operator of a class II landfill located in the City of Monterey Park. This abatement order was stipulated to by OII and was in response to many problems at the landfill site, and its vicinity, including odors, migrating gases, and exposed leachate. The stipulation for the Order for Abatement was developed with the tooperation and assistance of other agencies to ensure that there would be no conflict with their requirements.

The abatement order provides a comprehensive program and strict compliance schedule for odor control, including an extensive gas collection and incineration system, leachate controls, gas monitoring, cover requirements, and final closure by December 31, 1984.

Subsequent to the issuance of the abatement order, District tests detected an increase in the concentration of vinyl chloride in the landfill gases above those trace amounts normally found at such sites. The cause of

the recent increase is not known at this time. While monitoring had not yet shown an exceedance of the state ambient air quality standard for vinyl chloride, calculations by District staff indicated that the emission rates were expected to result in exceedances under more stable weather conditions, unless prompt action was taken to minimize concentrations of this toxic contaminant.

In addition, District inspectors uncovered deficiencies in OII's system for screening and prohibiting the illegal disposal of toxic/hazardous wastes at the site. Such disposal may result in air emissions of toxic/ hazardous materials.

In order to prevent exceedances of the ambient air quality standard for vinyl chloride and to assure that no further illegal disposal of toxic/hazardous occurs, the District filed a petition for modification of the abatement order which, if approved by the Hearing Board, would:

A. Immediately stop disposal at OII of all liquid and solid wastes until;
   1. OII can screen out and prohibit the disposal of illegal hazardous loads; and
   2. OII completes and places the gas collection system into full operation to minimize odors and emissions, including vinyl chloride;
B. Require OII to pay the costs of continuously monitoring vinyl chloride in the residential area; and
C. Require OII to post a bond to assure completion and continuous future operation of the gas collection system after final closure.

Exceedances of the vinyl chloride standard were subsequently detected in the residential community adjacent to the landfill. The abatement order hearing is pending at this writing.

Immediately adjacent to the OII landfill is the Getty Synthetic Fuels, Inc. gas recovery facility. This facility draws landfill gas through a series of wells at OII, removes carbon dioxide (which is vented to the atmosphere) and sells the cleaned methane to the Southern California Gas Company. Vinyl chloride is vented along with the carbon dioxide. To prevent exceedances of the vinyl chloride air quality standard in the vicinity of the Getty facility, the District required the immediate relocation of the vent as far away from any residences as possible as a stop-gap measure. Destruction of the vinyl chloride through flaring (burning) is being required as the ultimate control. In addition, Getty's pending permit to operate application was denied due to the present inability to control vinyl chloride emissions.

The District will continue to inspect the OII and Getty sites at least three times per week to assure compliance with the abatement order and will continue ambient monitoring for vinyl chloride and landfill gas emissions testing and take appropriate enforcement actions until the problem is resolved.

CLOSING COMMENTS

Toxic Enforcement is a Resource Intensive Activity

In the absence of NESHAPs rules or ambient air quality standards for all but a few toxic/hazardous materials, the South Coast Air Quality Management District's approach has been to monitor air quality in the vicinity of

suspected sources and to present the data to the county or state departments of health services for a risk assessment analysis. This procedure is extremely resource intensive and time consuming and must be done on a case by case basis.

This enforcement process could be shortened considerably by the adoption of ambient air quality standards followed by rule adoption where emission reductions are needed. Such a process would permit the priortization of efforts based on which standards have been adopted. At present, in the absence of standards local districts are at a loss as to which contaminants and sources should receive attention first.

Cooperation with Other Agencies

More than with any other area of air pollution enforcement, local districts must work closely with other state and local agencies where toxic and hazardous air emissions are concerned. There are some areas of regulatory overlap. Solutions to one environmental problem, if not carefully coordinated, may result in creation of another. Through this close work, local districts and other agencies can find areas of mutual benefit, such as the sharing of data. Also, when there are air emissions problems involving toxic/hazardous materials, there are usually violations of other environmental regulations involved. When these are found, coordinated multi-agency enforcement action against a source can be very effective in bringing about prompt compliance and can avoid problems brought about by the source attempting to play one agency's requirements against another.

ASBESTOS DECONTAMINATION IN GLOBE, ARIZONA

David Chelgren
Manager, Compliance Section
Arizona Bureau of Air Quality Control
Phoenix, AZ

ASBESTOS DECONTAMINATION IN GLOBE, ARIZONA

A case study in air toxics response

David O. Chelgren, Manager for Compliance
in the Arizona Bureau of Air Quality Control

BACKGROUND

Chrysotile asbestos has been an important natural resource of Arizona since the early 1900's. During the first few decades of asbestos mining, the raw fiber was transported as far as 50 miles to the mills by pack animal. Impurities such as serpentine and limestone were first removed by hand cobbing in the mine before transport to the mills, which were located near Globe. There are over eighty registered asbestos claims in Arizona including two in the Grand Canyon. However, about seventy five of the minesites are located in Gila County. As many as fourteen asbestos mills were operating in and around Globe, Arizona at one time. The General Service's Administration established a depot in Globe in 1952 for the purchase and storage of strategic grades of asbestos fibers. In addition to the locally mined material some additional ore was brought into Arizona from sources outside of the state including overseas, for milling.

In a number of cases the milling of hand cobbed ore was accomplished at the minesite but starting in about 1939 several mills were built and operated in Globe. These included the Arizona Asbestos Company (Town) Mill, the Jaquays Asbestos Company Mill, and the Metate Asbestos Corporation Mill located at the junction of U.S. 70 and Arizona 77 east of Globe. These mills were operating when the National Emission Standard for Hazardous Air Pollutants (NESHAPs) relating to asbestos was promulgated in 1973, and were subject to regulation by the Pinal-Gila Counties Air Pollution Control District (PGCAPCD).

The Town Mill was shut down in 1973 because of violations of the applicable standards. The Metate Mill was denied an air pollution control operating permit in 1972 but did obtain a conditional permit for limited operations from the Gila County Air Pollution Control Hearing Board. An order to cease operation in violation of its Conditional Permit was issued to Metate by the local air agency in December, 1973. At this point Metate had already started subdividing the property around the mill as a mobile home park. The Globe City Council approved the subdivision plan in spite of a written recommendation by the Director of the PGCAPCD to the Mayor that residences should not be permitted in such close proximity to asbestos mills. A temporary injunction against Metate was obtained in early 1974 when it was determined that it was operating at night after a number of residents had moved into the park. A permanent injunction was issued on April 30, 1974. The Arizona Real Estate Department (ARED) which had also approved the subdivision negotiated an agreement with Metate in 1976 to resolve numerous complaints by the residents including a provision that the mill would be removed after forty two lots were sold.

ARIZONA STATE INVOLVEMENT

The Arizona Department of Health Services (ADHS) became aware of the asbestos contamination during an inspection of the park's waste water treatment plant by a representative of the Bureau of Water Quality Control in early October, 1979. Exposed tailings and contaminated equipment were observed at numerous locations in the subdivision and on the adjacent railroad right-of-way (ROW). Other former asbestos facilities exhibiting exposed asbestos containing materials were located and inspected by representatives of the Bureau of Waste Control including the Town, inactive Jaquays, and Kyle millsites.

Bulk soil samples exhibited asbestos contamination ranging from five to sixty percent in most lots of the subdivision, on the ROW, and at the other millsites. Water samples from Globe, the Salt River and in the Phoenix Water System were determined to contain from 200,000 to 2,000,000 chrysotile fibers per liter along with much lower levels of amphiboles. Once the results of the soil sample analyses started coming in it was

apparent that a potential for high level exposure to airborne asbestos was present and a meeting was held with representatives of the City of Globe, the Gila County Health Department, the PGCAPCD and the State Real Estate Commissioner to advise them of the problem. Additional lot sales were prevented by the withdrawal of the subdivision approval by ARED. A letter was also sent to each of the residents in the subdivision advising them of the soil sample results and recommending precautions to minimize the potential for exposure to and inhalation of asbestos fibers. Personal monitor air samples taken during soil sampling, vacuuming inside a mobile home and outside the trailers were analyzed by the Arizona Industrial Commission using the Occupational Safety and Health Administration (OSHA) approved optical microscopy method and were later reported to contain between 0.003 and 0.350 fibers per cubic centimeter.

REMEDIAL RESPONSE

By mid-December, 1979 the Director of ADHS asserted jurisdiction of the inactive millsites which was later modified to include all asbestos facilities subject to NESHAPs in Gila County. Letters were sent to each of the mill operators ordering them to submit plans for decontamination to achieve compliance with NESHAPs within thirty days. The responsibility for the planning and verification of the decontamination activities was then assigned to the Bureau of Air Quality Control (BAQC) because NESHAPs were the only regulations that could be enforced to achieve the resolution of the problem.

The site was also inspected by representatives of the Centers for Disease Control and the National Institute for Occupational Safety and Health who then recommended immediate evacuation of the residents as well as restricting public access to the site. This recommendation was supported in a letter from an Assistant U.S. Surgeon General as well as by knowledgeable members of the Department staff. These recommendations were passed on to Governor Babbitt who declared the State of Emergency on January 16, 1980 in order to free up funds for temporary relocation of residents and contracts for cleanup of the ROW and the lots and mobile homes in the park by the Governor's Division of Emergency Services. An agreement was reached with Metate relative to its demolition and decontamination of the millsite. Written instructions were provided to the residents for the evacuation and they were advised of the plans for evacuation in a meeting on February 1, 1980. Air monitoring stations were placed in and around the park which were activated at that time. Residents desiring temporary relocation were evacuated and the initial decontamination of the affected areas then proceeded under the continuing surveillance of BAQC in the following steps, starting on February 6, 1980:

1. Metate Mill demolished and buried on site with a minimum cover of two feet of clean compacted cover.

2. Exposed tailings and stored fibers and ore buried under two feet of clean compacted cover at Town Mill.

3. Rip rap installed on wash bank and exposed asbestos covered with clean fill at inactive Jaquays millsite.

4. No action taken at Kyle millsite because the property was in estate probate and the estate had no funds for cleanup, and low potential for release of airborne fibers at its location.

5. Contaminated equipment on ROW cleaned and removed, tailings buried and minimum of two feet of clean compacted fill placed.

6. Lots analysed as positive for soil asbestos were provided with six inches of topsoil and grass seed where requested by residents. Bulk soil samples after the cover was placed contained less than one percent asbestos.

7. Interiors of mobile homes cleaned up by commercial cleaning company and residents allowed to return.

8. Metate slab scrubbed, pump well debris removed, and cracks sealed with asphalt sealant.

9. Wash gunited.

10. Residents were offered delivery of additional topsoil and grass seed.

11. State of Emergency was ended on June 30, 1980. Cost of cleanup to State was approximately $260,000.

12. Analysis of random soil samples at Metate millsite reported as less than one percent by U.S. Bureau of Mines.

13. Restriction on lot sales lifted on condition that Metate maintain millsite to prevent exposure or releases of asbestos fibers from millsite.

FINAL GLOBE MILLSITE DECONTAMINATION

The initial decontamination of this inactive Jaquays millsite was ineffective because of persistent erosion of the cover over the asbestos tailings. However, we negotiated a voluntary cleanup of this property by Junction Partners, Inc. which bought the property for commercial and residential development. Over 9,000 yards of contaminated material was excavated and buried on site under a plastic barrier with about nine feet of clean compacted cover. This work was completed under Bureau supervision and in full compliance with all applicable air and OSHA regulations.

KYLE MILLSITE STATUS

Negotiations for decontamination and disposal of the limited asbestos containing material were initiated in September, 1982. However, the Administrator of the Estate has refused to perform a voluntary cleanup. An Order of Abatement requiring decontamination was issued in June, 1983. The Order specifying compliance with NESHAPs Regulations has thus far been ignored. We are now preparing to file for injunctive relief to require compliance with the Order.

AIR QUALITY MONITORING

Ambient air monitoring for asbestos has continued to determine the effectiveness of the initial decontamination of the subdivision as well as to monitor the potential exposure to emissions from the adjacent Jaquays Mill. The mill has not operated since the end of 1981 but still represents a potential source of asbestos emissions from its exposed tailings piles. The data was analyzed as it became available. Once the data base from our original sampling station was available, statistical analyses showed a clear trend toward increasing levels of exposure over time. This potential was also supported by continuing on site inspections which indicated that the vegetative cover had not been established or maintained in the topsoil cover provided and that the resulting erosion had exposed asbestos contaminated materials.

When the probability of increasing levels of exposure to airborne asbestos fibers became apparent the site was nominated and qualified for Superfund cleanup, which is now in progress.

Statistical analyses of the air samples were undertaken in an attempt to identify the source of ambient exposure. No conclusive correlations were established within an

acceptable confidence level. In fact measured asbestos concentrations were generally higher at low wind speeds than at high average wind speeds. Thus, it might be concluded that the higher levels of exposure were associated with activities in the near vicinity of the monitoring site. A slight positive correlation with wind speed did occur in the sector containing the Jaquays millsite but the limited number of valid data points prevents any conclusions within an acceptable level of confidence.

The usable data base for the wind analyses was very limited due to poor data recovery by the wind instruments. The monitoring station is now equipped with more reliable data recorders and data averaging to expedite data reduction.

Simultaneous high volume and cassette samples were collected for a large number of the early measurements using millipore filters of 5.0 and 0.8 pore size respectively. Significantly higher fiber counts were detected on the cassette filters. Although this might be attributed to the difference in filter pore size, it may also have resulted from differences in the method of preparing Transmission Electron Microscopy sample grids by different laboratories.

Laboratory quality assurance checks at the two commercial laboratories was limited to the counting of blanks and prototype NBS specimen counting grids which were satisfactorily reported. Prepared TEM grids were also interchanged between laboratories. Interlab correlation was virtually non-existent. However, this lack of correlation may have resulted from non-uniform distribution of fibers on the grids. We intend to specify the use of "Finder Grids" in future TEM work so that specific grid openings can be identified for interlab verifications.

SITE MAINTENANCE

Independent of the specific method of decontamination and on-site disposal there is a need to provide for long term maintenance to assure that asbestos contaminated material is not disturbed or exposed. Simple compliance with the current NESHAPs provisions which formed the basis for the initial decontamination was obviously inadequate in this respect. However, we believe that on-site disposal, similar to that utilized at the Globe millsite and contemplated at the Kyle millsite, will minimize long term maintenance requirements.

PANEL DISCUSSION

Dr. Herschel Griffin (Panel Moderator)
Associate Dean, College of Human Services
San Diego State University
San Diego, CA

Milton Feldstein
Air Pollution Control Officer
Bay Area Air Quality Management District
San Francisco, CA

Jeanne Harvey
State Water and Air Quality Director
League of Women Voters of California
Ojai, CA

Maureen Lennon
Advisor, Regulatory Affairs Group
Atlantic Richfield Company
Los Angeles, CA

David Patrick
Chief, Pollutant Assessment Branch
U.S. Environmental Protection Agency
Research Triangle Park, NC

Michael Scheible
Chief, Office of Program Planning, Evaluation, and Coordination
California Air Resources Board
Sacramento, CA

PANEL QUESTION

In a speech entitled "Science, Risk, and Public Policy" delivered to the National Academy of Sciences on June 22, Mr. Ruckelshaus stated that:

> ...we must now deal with a class of pollutants for which a safe level is difficult, if not impossible, to establish... we must assume that life now takes place in a minefield of risks from hundreds, perhaps thousands, of substances.

At the same time, Section 112 of the Clean Air Act charges EPA with establishing standards for air toxics such that "an ample margin of safety is provided to protect the public health."

Given the difficulty of establishing safe levels for air toxics, what directions should EPA take regarding policy and/or research in an effort to apply the Clean Air Act's mandate to the regulation of air toxics?

BAY AREA AIR QUALITY MANAGEMENT DISTRICT

Response To Panel Question

Milton Feldstein
Air Pollution Control Officer

Standards can be set for toxic air contaminants based on thresholds or acceptable risk. EPA does have or should have the reources necessary to develop these tandards. Methods to analyze for these contaminants need to be developed. EPA needs to increase their resources devoted to research and methodologies for data base (emission inventory and ambient monitoring) compilation for these contaminants. There will always be disagreement as to what is an appropriate threshold level or risk level. More resources need to be devoted to the study of the effects these contaminants are currently having on people. This should be done through increased epidemiological studies, with adequate resources devoted to the studies.

Control of organic toxic emissions is not a mystery. Control can be accomplished through incineration or carbon absorption. There are trade-offs in emissions since incineration will result in increased NO_x emissions. The benefit of reduced toxic emissions and the effects of increased NO_x emissions need to be balanced.

The public needs to be actively involved in policy development and in the development of the criteria used to assess risk. The "public" which especially needs to be actively involved are those people who are exposed. In order for this "public" to effectively participate, they have to be informed of the risks involved. Better public information on the risks involved needs to be prepared and made available. This information needs to be understandable and readily available (e.g. made available through the media rather than through the Federal Register).

Local air pollution control districts have the expertise to control the emissions through the development, adoption, and enforcement of regulations. EPA needs to define the contaminants of concern , appropriate thresholds and risk analysis.

EPA should also accelerate reserach for analytical methods and new methods of control.

SEPTEMBER 14, 1983

STATEMENT BY THE LEAGUE OF WOMEN VOTERS TO BE PRESENTED

AT THE E.P.A. CONFERENCE ON TOXIC AIR

I represent the League of Women Voters of California, a grass roots citizens organization with well over 12,000 members. All with a long standing interest in human and environmental affairs.

The League is committed to the principle that democratic government depends upon the informed and active participation of its citizens in all areas of public policy. We are pleased to think that this conference represents a renewed trend toward a broad based citizen involvement in EPA policy decision making.

Our historical position with respect to the CLEAN AIR ACT has been one of strong support. Without belaboring the past, I should say that in early 1980, when the Act came up for review and reauthorization and was the target of attack from industrial interests as well as a showcase for regulatory relief, the League made it a priority to oppose any retreat from the public health and environmental safeguards contained in the law. And one of the key provisions that we have sought to strengthen has been the acceleration in control of toxic air pollutants.

As for the mandate "to establish standards to provide for the protection of public health" the wording seems clear. It reflects the will of the people as perceived by Congress.

I shall speak of the ambiguities in the CLEAN AIR ACT and the problems of its implementation as they seem to be viewed by the decision makers. I shall also take this opportunity to present some of the needed changes as perceived by the League.

That the setting of standards should be predicated upon what scientific and medical information exists seems evident. The paralysis occurs when a decision is required to take that scientific information, imperfect and incomplete as it may be, and act upon it. Yet this is the administrative burden of the EPA. If indeed, Section 112 is not enforceable, then legislation may be the only answer. It is our contention that such is the case and that an amendment of the Act must be sought. EPA would be strengthened by an amendment to the CAA that would set standards by imposing a known scientific criterion. Standards that would be at least as strict as a more clearly defined Best Available Technology. The EPA continues to rely heavily on the economic aspects of BAT, much more so than the League thinks is justified.

Which brings us to some economic considerations. We do not recognize cost as a valid criterion for setting acceptable levels of risk. While costs are not to be denied, they can be taken into account when strategies and technologies to achieve reduction goals are chosen. At that time alternate processing methods and compounds can be studied. For example, we suggest a combination of population density and cancer incidence as being appropriate considerations in risk managment plans. As an adjunct to this thinking, industry must recognize that developing effective controls are a cost of doing business.

Realistically, political considerations have been high on the list of uncertainties in the administration of all public agencies. Repeated and recent polls point to an enormous political force favoring implementation of the CAA, even at the risk of some loss of jobs. The public does not demand absolute scientific proof of how toxins endanger their health, but the past few months have given us ample evidence that they are quick to place blame when protection has not been afforded them. The disovery of dioxin that necessitated the abandonment and reparations for a whole town in Missouri as well as the identification in California of toxins in the Stringfellow Pits and the ground water contamination in San Jose are good examples. It seems better to err on the side of strict regulation than to run these risks. On-going scientific findings can be easily folded into an existing program, particularly if it involves reduction of control and cost.

Another EPA concern exists for the rights of states, localities and industry. However, the suggestion that they undertake to voluntarily set their own control standards is, unfortunately, highly impractical. The proposal for the Agency to carry on a continuous monitoring program of multiple entities is inefficient and fraught with opportunity for inaction. The costs of scientific expertise, in itself, would be prohibitive. In addition, states need the authority of the Federal government in order to expedite reforms. Without such support, competition for development is bound to impair individual states ability to set and enforce standards in the public interest.

As I mentioned at the beginning, any evidence of opportunity for public review is welcomed by the League. By this I do not mean that

the public should be burdened with making repeated decisions about what they want. That decision has been made. And a further cautionary note, all public information and opportunities for participation are useless if a course of action has been predetermined. Clearly defined channels for participation and provision for dessiminating objective information must be provided in order that citizens can fill the important role of helping to develop policy decisions, such as how to make the best use of the limited dollars.

To summarize - the League would like to see some changes in the law that would give EPA the necessary authority and incentive to set a timetable to identify air toxins and implement control measures - and that proper channels for public input continue to be given a high priority.

PRESENTED BY JEANNE G. HARVEY
Air Quality Director
LEAGUE OF WOMEN VOTERS OF CALIFORNIA
4275 Grand Av.
Ojai, Ca. 93023

Summary of Remarks Made by Michael Scheible
at Panel Discussion at Region IX Toxics Conference
September 14, 1983

The question that we were asked to address raises the issue of whether the Clean Air Act has placed EPA in an impossible situation of eliminating all exposures to substances for which no safe level can be established. First, it is my personal view that the goal established by Congress to protect public health with an ample margin of safety and the statement by Administrator Ruckelshaus are both correct, and from a policy perspective these two positions do not present irreconcilable conflicts. Although legal interpretations may be applied to the Clean Air Act that would make it difficult to totally resolve this issue, I believe that progress can be made. There are clearly other technical-legal inconsistencies in the Act of comparable magnitude. Perhaps the best example is the requirement that the ozone standard be attained in the South Coast Air Basin by 1987. While these inconsistencies have caused problems, they have not prevented state and local actions that will result in very significant progress in cleaning the air.

Second, I believe that the current process used by EPA relies too heavily on federal actions and that the Act requires EPA to devise controls in an unrealistically short timeframe once a substance has been identified as a hazardous air pollutant. In many cases, airborne exposures to hazardous substances result from the emissions from thousands, or in the case of pollutants emitted from automobiles, millions of sources. It will take time to design effective strategies in such cases. To deal with the complexity of the problem, EPA needs to rely more on the capabilities of local agencies, perhaps in a process similar to that used to develop attainment plans for criteria pollutants, to identify and implement actions to reduce emissions of hazardous pollutants to acceptable levels.

Third, EPA needs to develop an overall policy and context for making decisions on how sources of hazardous pollutants will be controlled. I think it is unfortunate that much of this overall policy may be made based on experiences with the Tacoma Copper Smelter, a situation largely seen by the public as a conflict between health and jobs. I believe that such situations will be the exception and that trying to produce reasonable policies out of such an emotional setting will be very difficult.

Fourth, EPA needs to expedite its process to make decisions on the identification of additional hazardous pollutants. The review pipeline has been full for some time, and it is doubtful that significant additional information that would aid the resolution of existing scientific controversies will become available in the near future. Because state and local agencies must rely very heavily on the federal government for health assessments it is imperative that EPA break the current logjam and make decisions on the substances now under review.

Finally, I would like to echo support for more public involvement. Ultimately, it is the public who both pays for and benefits from the control of hazardous air pollutants. However, as is true in most cases, the cost and benefits are not equally distributed. Rarely is the public that must bear the increased health risk the same as the public that derives benefits from the economic activities that result in emissions of hazardous materials. EPA, states and local agencies must improve our past efforts to inform and involve the public in decisions that ultimately involve the public acceptance of some level of risk. We must make explicit the scientific and economic issues involved and better educate the public.

LIST OF PARTICIPANTS

EPA REGION 9 AIR TOXICS CONFERENCE

September 13 and 14, 1983
Sheraton Palace Hotel
San Francisco, CA

Mr. Ben Almario
U.S. Department of the Navy
P.O. Box 727
San Bruno, CA 94066

Mr. Gerald Anderson
Systems Applications, Inc.
101 Lucas Valley Road
San Rafael, CA 94903

Mr. Donald Ames
California Air Resources Board
1102 Q Street
Sacramento, CA 95812

Mr. Donald F. Austin, M.D.
Department of Health Services
5850 Shellmound, #200
Emeryville, CA 94608

Mr. Lynn Baker
1615 Phantom Avenue
San Jose, CA 95125

Mr. Richard Baldwin
County of Ventura APCD
800 South Victoria Avenue
Ventura, CA 93009

Mr. Bob Barham
California Air Resources Board
1102 Q Street
Sacramento, CA 95812

Mr. William Barker
University of California
Los Angeles Graduate School
1427 - 25th Street, #4
Santa Monica, CA 90404

Mr. Cliff Bast
Hewlett Packard
300 Hanover Street, 20DJ
Palo Alto, CA 94304

Mr. Michael Belliveau
Citizens for a Better Environment
88 First Street
San Francisco, CA

Mr. Ravi Bhatia
Envirosphere Company
444 Castro Street
Mountain View, CA 94041

Mr. Robert Bishop
E.P.A., Region 9
215 Fremont Street
San Francisco, CA 94105

Mr. Phil Bobel
E.P.A., Region 9
215 Fremont Street
San Francisco, CA 94105

J.M. Bodie, M.D.
San Mateo County Department of
Health Services
225 - 37th Avenue
San Mateo, CA 94403

Mr. Steve Body
E.P.A., Region 9
215 Fremont Street
San Francisco, CA 94105

Meredith Boli Associates
8857 West Olympic Blvd.
Suite 200
Beverly Hills, CA 90211

Mr. Frank Bonamassa
California Air Resources Board
9528 Telestar Avenue
El Monte, CA 91731

Mr. Michael L. Borden
Safety Specialists, Inc.
P.O. Box 4420
Santa Clara, CA 95054

Mr. David Bauer
IT Corporation
336 West Anaheim Street
Wilmington, CA 90744

Ms. Rebecca L. Beemer
Thermal Power Company
601 California Street
San Francisco, CA 94108

Ms. Kandice Bellamy
E.P.A., Region 9
215 Fremont Street
San Francisco, CA 94105

Mr. Richard Bradley
California Air Resources Board
P.O. Box 2815
Sacramento, CA 95812

Mr. Tom Brady
Los Angeles Mayor's Office
City Hall, Room M-1
Los Angeles, CA 90012

Mr. Joseph J. Brecher
506 - 15th Street
Oakland, CA 94612

Mr. James Breitlow
E.P.A., Region 9
215 Fremont Street
San Francisco, CA 94105

Mr. T. Brennan
Bay Area AQMD
939 Ellis Street
San Francisco, CA 94109

Ms. Debbie Bright
Bright and Associates
1200 North Jefferson, Suite B
Anaheim, CA 92807

Ms. Kim Brobeck
University of San Francisco
400 Willamette Drive
Vacaville, CA 95688

Mr. Dave Brooks
Hewlett Packard
300 Hanover Street, 20 DJ
Palo Alto, CA 94304

Mr. Timothy Bruce
City of West Covina
1444 West Garvey
West Covina, CA 91790

Mr. Larry Bowen
South Coast AQMD
9150 Flair Drive
El Monte, CA 91731

Mr. Larry Bowerman
E.P.A., Region 9
215 Fremont Street
San Francisco, CA 94105

Ms. Jan Bush
CAPCOA
254 Day Road
Ventura, CA 93003

Mr. Michael Cardin
Union Oil Company of California
461 South Boylston Street
Los Angeles, CA 90017

Mr. Robert W. Carr
San Luis Obispo County APCD
2146 Sierra Way, Suite B
Arroyo Grande, CA 93420

Mr. Willard Chin
E.P.A., Region 9
215 Fremont Street
San Francisco, CA 94105

Mr. Robert C. Cofer, P.E.
Sacramento County APCD
3701 Branch Center Road
Sacramento, CA 95827

Mr. Joel Cohen
J. M. Cohen, Inc.
111 Winding Way
San Carlos, CA 94070

Ms. Carol B. Coleman
Atlantic Richfield Company
515 South Flower Street, Room 4084
Los Angeles, CA 90017

Mr. Garry D. Criscione
Tulare County APCD
Health Department
County Civic Center
Visalia, CA 93291

Mr. Randy Crissmon
E.P.A.
401 M Street, SW
Washington, D.C. 20460

Mr. Don Crowe
California Air Resources Board
1102 Q Street
Sacramento, CA 95814

Mr. David Calkins
E.P.A., Region 9
215 Fremont Street
San Francisco, CA 94105

Vinh Cam
U.S. EPA
Air Programs Branch
Room 1005
26 Federal Plaza
New York, NY 10278

Mr. Ed Camarena
South Coast AQMD
9150 Flair Drive
El Monte, CA 91731

Mr. Ken Davis
Monsanto Company
1121 L Street, Suite #906
Sacramento, CA 95814

Ms. Dana Davoli
E.P.A., Region 10
1200 - 6th Avenue, M/S 532
Seattle, WA 98101

Mr. Bill DeBoisblanc
Bay Area AQMD
939 Ellis Street
San Francisco, CA 94109

Ms. E. DeFalco
League of Women Voters, Bay Area
117 Natalie Drive
Moraga, CA 94556

Mr. Jeffrey H. Desautels
Anaconda Minerals Company
555 - 17th Street
Denver, CO 80202

Mr. David DiJulio
Southern California Association
of Governments
600 South Commonwealth Ave., Suite 1000
Los Angeles, CA 90005

Ms. Paulette Durand
University of San Diego
Alcala Park
San Diego, CA 92110

Mr. Dennis Dykstra
Chevron Research
576 Standard Avenue
Richmond, CA 94802

Ms. Janise Ehman
Engineering-Science
600 Bancroft Way
Berkeley, CA 94710

Dr. Larry T. Cupitt
GKPB, MD-84
U.S. Environmental Protection Agency
Research Triangle Park, NC 27711

Ilene R. Danse, M.D.
Chevron Environ. Health Center, Inc.
P.O. Box 4054
Richmond, CA 94804

Mr. Allen Danzig
San Diego, APCD
9150 Chesapeake Drive
San Diego, CA 92123

Ms. Anna Fan
Department of Health Services
2151 Berkeley Way
Berkeley, CA 94704

Mr. Don Fast
IBM Corporation
5600 Cottle Road
San Jose, CA 95193

Mr. Milton Feldstein
Bay Area AQMD
939 Ellis Street
San Francisco, CA 94109

Mr. Bryant Fischback
The Dow Chemical Company
P.O. Box 1398
Pittsburg, CA 94565

B. N. Fleischer
Allied Chemical
Nichols Road
Pittsburg, CA 94565

Mr. Chuck Flippo
E.P.A., Region 9
215 Fremont Street
San Francisco, CA 94105

Mr. James Forrest
E.P.A., Region 9
215 Fremont Street
San Francisco, CA 94105

Mr. Michael S. Foster
Cheveron U.S.A.
575 Market Street Street, Room 1566
San Francisco, CA 94105

Mr. C. Lee Fox
Pima County Health Department
151 West Congress Street
Tucson, AZ 87501

Ms. Victoria A. Evans
Gaia Associates
Golden Gate Energy Center
Fort Cronkhite, Building 1055
Sausalito, CA 94965

Mr. Robert W. Evans
Maricopa County APCD
P.O. Box 2111
Phoenix, AZ 85001

Mr. Gerry Fait
Pillsbury, Madison & Sutro
225 Bush Street
San Francisco, CA 94104

Mr. Patrick Frost
SMUD
3580 Buoy Way
Sacramento, CA 95871

Mr. George Fujimoto
Hawaii Department of Health
645 Halekauwila, 3rd Floor
Honolulu, HI 96813

Mr. Barry Garelick
Woodward-Clyde Consultants
1 Walnut Creek Center
100 Pringle Avenue
Walnut Creek, CA 94596

Mr. Morris G. Gary
IBM Corporation
Dept. 843/124
5600 Cottle Road
San Jose, CA 95193

Mr. Bob Gaynor
Bay Area AQMD
939 Ellis Street
San Francisco, CA 94109

Mr. Ralph George
BKK Corporation
2550 - 237th Street
Torrance, CA 90505

Mr. D.J. Gladen
SoCalGas
810 South Flower
Los Angeles, CA 90017

Ms. Caren Glassel
E.P.A., Region 9
215 Fremont Street
San Francisco, CA 94105

Mr. Robert S. Frederick
State Board of Fabric Care
1900 San ysidro
Beverly Hills, CA 90210

Ms. Terry Freeman
SRI International
333 Ravenswood Avenue
Menlo Park, CA 94025

Mr. Jim Frolich
Ch_2m Hill
2200 Powell Street, 8th Floor
Emeryville, CA 94608

Mr. Roger D. Griffin
KVB, Inc.
18006 Skypark
Irvine, CA 92714

Mr. Herschel Griffin
San Diego State University
College of Human Services
San Diego, CA 92182

Mr. David Grimsrud
Lawrence Berkeley Laboratory
Building 90 - 3058
Berkeley, CA 94720

Mr. Jack Grisanti, President
Cal-Alga Resources
15509 Mountain View
Kingsburg, CA 93631

Mr. Colin K. Guptill
Pinal-Gila APCD
Box 426
Kearny, AZ 85237

Mr. Don Harvey
E.P.A., Region 9
215 Fremont Street
San Francisco, CA 94105

Ms. Jeanne Harvey
League of Women Voters
4275 Grand Avenue
Ojai, CA 93023

Mr. G. C. Hass
California Air Resources Board
1102 Q Street
Sacramento, CA 95814

Mr. Dan Goalwin
Bay Area AQMD
939 Ellis Street
San Francisco, CA 94105

Mr. Kevin Golden
E.P.A., Region 9
215 Fremont Street
San Francisco, CA 94105

Mr. Mary Gothberg
Ford Aerospace
3939 Fabian Way
Palo Alto, CA 94303

Ms. Lois Green
E.P.A., Region 9
215 Fremont Street, MS: A-3-3
San Francisco, CA 94105

Mr. J. T. Holcombe
Pacific Gas and Electric Company
77 Beale Street, Room 1373
San Francisco, CA 1373
San Francisco, CA 94106

Dr. Kim Hooper
Department of Health Services
2151 Berkeley Way
Berkeley, CA 94704

Barry R. Horn, M.D.
Bay Area AQMD
2063 Oakland Avenue
Piedmond, CA 94611

Mr. David Howekamp
E.P.A., Region 9
215 Fremont Street
San Francisco, CA 94105

Ms. Mary Humboldt
Campaign for Economic Democracy
1337 Santa Monica Mall, #310
Santa Monica, CA 90401

Ms. Sharon Huse
WESTNAVFACECOM
P.O. Box 727
San Bruno, CA 94066

Ms. Betty Ichikawa
California Air Resources Board
P.O. Box 2815
Sacramento, CA 95812

Mr. Miles R. Imada
Department of Health Services
2151 Berkeley Way
Berkeley, CA 94704

Ms. Stana Hearne
League of Women Voters, Bay Area
5931 Rincon Drive
Oakland, CA 94611

Ms. Evelyn F. Heidelberg
California Council for Environmental
and Economic Balance
215 Market Street, Suite 1311
San Francisco, CA 94105

Mr. Steven Hill
Bay Area AQMD
939 Ellis Street
San Francisco, CA 94109

Ms. Kay Jensen
California Air Resources Board
1102 Q Street
Sacramento, CA 95814

Ms. Jeri Johnson
E.P.A., Region 9
215 Fremont Street
San Francisco, CA 94105

Mr. Joseph R. Johnson
BKK Corp.
2550 - 237th Street
Torrance, CA 90505

Mr. Neal Johnson
Waste Management Board
1020 - 9th Street, Suite 200
Sacramento, CA 95814

Ms. Kathleen M. Kahler
American Lung Association
833 Market Street
San Francisco, CA 94103

Mr. Charles B. Kay
Texaco Inc.
3350 Wilshire Boulevard
Los Angeles, CA 90010

Mr. Larry Kerrigan
Los Angeles Dept. of Water & Power
111 North Hope Street, Room 632
Los Angeles, CA 90051

Mr. Peter King
E.P.A., Region 9
215 Fremont Street
San Francisco, CA 94105

Ms. Laura King
Natural Resources Defense
Council, Inc.
25 Kearny Street
San Francisco, CA 94108

Ms. Jean Kitchens
League of Women Voters
318 Randloph Street
Napa, CA 94558

Mr. Kent Kitchingman
E.P.A., Region 9
215 Fremont Street
San Francisco, CA 94105

Ms. Meredith Jane Klein
Pillsbury, Madison & Sutro
P.O. Box 7880
San Francisco, CA 94120

Mr. Irwin Koehler
Roy F. Weston, Inc.
153 Kearny Street, Suite 506
San Francisco, CA 94108

Ms. Ruth H. Koehler
League of Women Voters
64 Stuart Court
Los Altos, CA 94022

Mr. Carl Kohnert
E.P.A., Region 9
215 Fremont Street
San Francisco, CA 94105

Mr. Imants Krese
E.P.A., Region 9
215 Fremont Street
San Francisco, CA 94105

Mr. Bob Kuhlman
California Air Resources Board
1102 Q Street
Sacramento, CA 95814

Mr. Linda Larson
E.P.A., Region 9
215 Fremont Street
San Francisco, CA 94105

Mr. Sven A. Larsson
Formica Corp.
P.O. Box 519
Rocklin, CA 95677

Mr. Ron Leach
E.P.A., Region 9
215 Fremont Street
San Francisco, CA 94105

Mr. Olaf Leifson
Department of Food and Agriculture
1220 N Street
Sacramento, CA 95826

Ms. Maureen Lennon
Atlantic Richfield Co.
515 South Flower St., AP 410
Los Angeles, CA 90071

Mr. Richard Lewis
c/o Mike Miller
City of West Covina
P.O. Box 1440
West Covina, CA 91768

Mr. Alan C. Lloyd
Environmental Research
& Technology, Inc.
2625 Townsgate Road
Westlake Village, CA 91361

Mr. William Loscutoff
California Air Resources Board
1102 Q Street
Sacramento, CA 95814

Mr. Dick Lundquist
California Air Resources Board
1102 Q Street
San Francisco, CA 94105

Ms. Barbara Maco
E.P.A.
236 - 4th Avenue
San Francisco, CA 94118

Mr. Tirlochan S. Mangat
Bay Area AQMD
939 Ellis Street
San Francisco, CA 94109

Mr. Terry McGuire
California Air Resouces Board
1102 Q Street
San Francisco, CA 95814

Mr. J. Craig Mckenzie
Chemical Waste Management
P.O. Box 471
Kettlemen City, CA 93239

Mr. Ralph Mead
255 Sycamore Avenue
Mill Valley, CA 94941

Mr. Raymond Menebroker
California Air Resources Board
1102 Q Street
Sacramento, CA 95814

Mr. Herman Myer
Sacramento County APCD
37018BranchaCentertRoad
Sacramento, CA 95827

Mr. Edward Miller
Bay Area AQMD
939 Ellis Street
San Francisco, CA 94109

Mr. Michael L. Miller
City of West Covina
P.O. Box 1440
West Covina, CA 91793

Mr. Jerry Miller
Teknekron, Inc.
2118 Milva Street
Berkeley, CA 94704

Mr. David A. Monroe
Union Oil of California
461 South Boylston Street
Los Angeles, CA 90017

Mr. Walter R. Mook
San Bernardino County APCD
15579 - 8th Street
Victorville, CA 92392

Mr. David Morell
E.P.A.
215 Fremont Street
San Francisco, CA 94105

Mr. Wayne Morgan
Stanislaus County APCD
1716 Morgan Road
Modesto, CA 95351

Mr. Joel D. Mulder
E.P.A. Center for Disease Control
215 Fremont Street
San Francisco, CA 94105

Carol or Arthur Murray
13 Miramonte Drive
Morage, CA 94556

Mr. Michael Naylor
Clark County Health District
P.O. Box 4426
Las Vegas, NV 89127

Mr. Michael Neale
Dow Chemical U.S.A.
2800 Mitchell Drive
Walnut Creek, CA 94598

Mr. Fernando I. Nell
Safety Specialists, Inc.
P.O. Box 4420
Santa Clara, CA 95054

Ms. Emily Pearson Nelson
Graduate Research Assistant
715-1/2 North Marguerita
Alhambra, CA 91801

Mr. Lyler R. Nelson
Southern California Edison Co.
2244 Walnut Grove Avenue
Rosemead, CA 91770

Mr. James F. Norton
1885 Ridgeview Drive
Roseville, CA 95678

Mr. Leslie Norton
Engineering-Science
125 West Huntington Drive
Arcadia, CA 91006

Ms. Mitsi Okamoto
E.P.A., Region 9
215 Fremont Street
San Francisco, CA 94105

Mr. William Rogers Oliver
Systems Applications, Inc.
101 Lucas Valley Road
San Rafael, CA 94903

Mr. John Ong
E.P.A., Region 9
215 Fremont Street
San Francisco, CA 94105

Ms. Jean J. Ospital
Southern California Edison
P.O. Box 800
Rosemead, CA 91770

Ms. Patricia M. O'Toole
Union Oil Company of California
461 South Boylston Street
Los Angeles, CA 90017

Mr. Charles E. Owens
United Airlines M.O.C.
San Francisco Internaitonal Airport
San Francisco, CA 94128

Mr. Coe Owen
E.P.A., Region 9
215 Fremont Street
San Francisco, CA 94105

Mr. Gordon Palmer
Southern California Association
of Governments
600 Commonwealth Avenue
Suite 1000
Los Angeles, CA 90005

Mr. David Patrick
E.P.A., Air Quality Planning
& Standards
Research Traingle Park, NC 27711

Ms. Joan Patton
League of Women Voters, Bay Area
5845 Ocean View Drive
Oakland, CA 94618

Mr. Thomas A. Peters
Engineering-Science
125 West Huntington Drive
Arcadia, CA 91016

Mr. Daniel V. Phelan
155 Jackston, Suite 305
San Francisco, CA 94111

Mr. Ralph Propper
Coalition for Clean Air
309 Santa Monica Blvd., #312
Santa Monica, CA 90401

Mr. Doug Quetin
Monterely Bay Unified APCD
1064 Monroe Street, Suite No. 10
Salinas, CA 93901

Mrs. Doroty M. Rankin
Pinal-Gila Counties AQCD
P.O. Box 1076
Florence, AZ 85232

Mr. Tom Rarick
E.P.A., Region 9
215 Fremont Street
San Francisco, CA 94105

Mr. Michael Redemer
Beacon Oil Company
525 West 3rd Street
Hanford, CA 93230

Mr. Ralph M. Riggin
Battelle
505 King Avenue
Columbus, OH 43085

Ms. Joan E. Riley
Chemical Manufacturers Association
2501 M Street, N.W.
Washington, D.C. 20037

Mr. Donald Robbins
Asarco Inc.
3422 South 700 West
Salt Lake City, UT 84119

Mr. Richard Rollins
National Semiconductor
2900 Semiconductor Drive, M/S B540
Santa Clara, CA 95051

Mr. Scott Ross
UCLA-SPH
1427 - 25th Street
Santa Monica, CA 94025

Mr. Marc Rothschild
E.P.A., Region 9
215 Fremont Street
San Francisco, CA 94105

Ms. Sharon F. Rubalcava
McCutchen, Black, Verleger & Shea
600 Wilshire Boulevard
Los Angeles, CA 90017

Mr. Kelly Runyuon
S.F. Sold Waste Managment Program
City Hall, Room 271
San Francisco, CA (4102

Mr. Hanafi Russell
Department of Health Services
2151 Berkeley Way
Berkeley, CA 94704

Ms. Adelia Sabiston
League of Women Voters, Bay Area
3953 Campolinda Drive
Morago, CA 94556

Ms. Sue Sakaki
E.P.A., Region 9
215 Fremont Street
San Francisco, CA 94105

Mr. Mark Saperstein
Electric Power Research Insititue
3412 Hill iew Avenue
Palo Alto, CA 94303

Mr. Michael Seaman
California Air Resources Board
1102 Q Street
Sacramento, CA 95814

Mr. Ray Seid
E.P.A., Region 9
215 Fremont Street
San Franciso, CA 94105

Mr. Richard Seraydarian
WESTNAVFACENGCOM, CODE 1144
P.O. Box 727
San Bruno, CA 94066

Dr. Ken Sexton
Deparmtent of Health Services
2151 Berkeley Way
Berkeley, CA 94704

Mr. Michael Scheible
California Air Resources Board
1102 Q Street
Sacramento, CA 95814

Mr. Rolf D. Schmued
Rockwell International
Rocketdyne Division
6633 Canoga Avenue - d/543-FB66
Canoga Park, CA 91304

Mr. Herb Schuyten
Chevron U.S.A.
575 Market Street, Room 1564
San Francisco, CA 94105

Dr. J.C. Schwegmann
Kaiser Aluminum & Chemical Corp.
300 Lakeside Drive
Oakland, CA 94643

Ms. Celia Shen
California Air Resources Board
1309 T Street
Sacramento, CA 95814

Mr. J. Gareth Shepherd
Kaiser Refractories
Moss Landing, CA 95039

Mr. James M. Shikiya
California Air Resources Board
9528 Telestar Avenue
El Monte, CA 91731

Ms. Merceditas Shikiya
South Coast AQMD
9150 Flair Drive
El Monte, CA 91731

Ms. Kathleen Shimmin
E.P.A., Region 9
215 Fremont Street
San Francisco, CA 94105

Mr. Bart Simmons
Department of Health Services
2151 Berkeley Way
Berkelehy, CA 94704

Dr. Hanwant B. Singh
SRI International
333 Ravenswood
Menlo Park, CA 94025

Mr. Eric P. Skelton
Sacramento County APCD
3701 Branch Center Road
Sacramento, CA 95827

Kathryn Smick, M.D.
1115 Oakhill Road
Lafayette, CA 94549

Mr. Robert N. Smiley
Vulcan Chemicals
333 Hegenberger Road, #208
Oakland, CA 94621

Ms. Alexandra B. Smith
E.P.A., Region 10
1200 - 6th Avenue, M/S 529
Seattle, EA 98101

Mr. Ted Smith
Silicon Valley Toxics Coalition
1025 North 4th Street
San Jose, CA 95112

Mr. David Solomon
E.P.A., Region 9
215 Fremont Street
San Franciso, CA 94105

Mr. Todd I. Sostek
SoCalGas
Box 3249, Terminal Annex
Los Angeles, CA 90051

Mr. David R. Souten
System Application, Inc.
101 Lucas Valley Road
San Rafael, CA 94903

Ms. Pat Springer
E.P.A., Region 9
215 Fremont Street
San Francisco, CA (4105

Mr. Arnold Stein
Engineering Science
125 West Huntington Drive
Arcadia, CA 91006

Mr. Mike Stenburg
E.P.A., Region 9
215 Fremont Street
San Francisco, CA (4105

Mr. R. Stephens
Department of Health Services
215 Berkeley Way
Berkeley, CA 94704

Ms. Leslie Stewart
League of Women Voters
3557 Mt. Diablo Boulevard
Lafayette, CA 94549

Ms. Alexis Strauss
E.P.A., Region 9
215 Fremont Street
San Francisco, CA 94105

Mr. J.A. Stuart
South Coast AQMD
9150 Flair Drive
El Monte, CA 91731

Mr. Rick Sugarek
E.P.A., Region 9
215 Fremont Street
San Francisco, CA 94105

Ms. Andree Tamony
Dow Chemical
P.O. Box 1398
Pittsburg, CA 94509

Ms. Melinda Taplin
E.P.A., Region 9
215 Fremont Street
San Francisco, CA 94105

Ms. Ruby Tarleton
League of Women Voters
2243 Redwood Road
Napa, CA 94558

Mr. Russ Tate
California Air Resources Board
P.O. Box 2815
Sacramento, CA 95812

Ms. Vivian Thomson
E.P.A., Region 9
215 Fremont Street
San Francisco, CA 94105

Ms. Phyllis Tichinin
Toxic Assessment Group
2530 J Street
Sacramento, CA 95816

Mr. G. Tsou
California Air Resources Board
1102 Q Street
Sacramento, CA 95814

Ms. Lucille van Ommering
E.P.A., Region 9
215 Fremont Street
San Francisco, CA 94105

Mr. Peter Venturini
Callifornia Air Resources Board
1102 Q Street
Sacramento, CA 94105

Mr. Mark Volmert
Los Angeles County
856 Hall of Administration
500 West Temple
Los Angeles, CA 90012

Mr. Alan Waltner
E.P.A., Region 9
215 Fremont Street
San Francisco, CA 94105

Mr. Jerry Wesolowski
Department of Health Services
2151 Berkeley Way
Berkeley, CA 94704

Ms. Alice Westerinen
California Air Resources Board
1131 S Street
Sacramento, CA 95815

Ms. Deanna Wieman
E.P.A., Region 9
215 Fremont Street
San Francisco, CA (4105

Mr. Daniel Wilkowsky
National Semiconductor
2900 Semiconductor Drive, M/S d3940
Santa Clara, CA 95051

Ms. Stell Wilcox
San Diego APCD
9150 Chesapeake Drive
San Diego, CA 92123

Ms. Julie Williams
Signetics Corporation
811 East Arques Avenue, M/S 0458
Sunnyvale, CA 94088

Mr. John Wise
E.P.A., Region 9
215 Fremont Street
San Francisco, CA 94105

Mr. Harmon Wong-Woo
California Air Resources Board
1102 Q Street
San Francisco, CA 95814

Mr. Michael Work
E.P.A., Region 9
215 Fremont Street
San Francisco, CA 94105

Robert A. Wyman, Esq.
Lathman & Watkins
555 South Flower Street
LOs Angeles, CA 90071-2466

Dr. Terry Young
Environmental Defense Fund
2606 Dwight Way
Berkeley, CA 94704

Mr. Thomas Zosel
3M Company 33331
St. Paul, MN 55133

Mr. Mark Zuckerman
Signetics Corporation
811 Esta Arquest Avenue, MS-2539
Sunnyvale, CA 94088