GLOBAL CHEMICAL POLLUTION p814-819

32353 PDF PE0015F

Curtis C. Travis
Sheri T. Hester
Oak Ridge National Laboratory
Oak Ridge, TN 37831-6109

Over the past decade, public and governmental awareness of environmental problems has grown steadily, with an accompanying increase in the regulation of point sources of pollution. As a result, great strides have been made in cleaning polluted rivers and decreasing air pollution near factories. However, traditional regulatory approaches to environmental pollution have focused primarily on protecting the maximally exposed individual located in the immediate vicinity of the pollution source. Little attention has been given to the global implications of human production and use of synthetic chemicals. If chemicals are produced, either intentionally or as by-products of industrial activities, and not destroyed naturally or by humans, they eventually reach the environment. Once in the environment they are transported globally, partition into biological media, and result in essentially the entire world population being exposed to trace levels of chemical contamination.

Until recently, trace levels of environmental contamination were thought to be relatively benign. However, a consensus is emerging that even trace levels of environmental contamination can have potentially devastating environmental consequences. Tall stacks were once hailed as the solution to local sulfate problems from coal-fired power plants; now acid rain is recognized as an environmental problem. Carbon dioxide emissions are leading to global climate warming; chlorofluorocarbon releases are resulting in depletion of the Earth's protective ozone layer. With alarming regularity, we find reports of chemical contamination in parts of the world previously thought to be pristine. Mercury is found in fish in remote lakes; lead is found in Greenland snow; and polychlorinated biphenyls (PCBs), dioxin, and pesticides are found in the Arctic. Even though PCBs and DDT have been banned in the United States, these chemicals are exported to other parts of the world where, following use, they volatilize and are globally translocated in the atmosphere. Aerial fluxes of these pollutants contribute a major portion of pollutant loadings to the Great Lakes (*1*), the Chesapeake Bay (*2*), and other lakes. Although the environmental impacts of global pollutants (e.g., climate warming, ozone depletion) have received widespread attention, little heed has been given to their possible impacts on health. We maintain that ambient levels of pollution have risen to the point where human health is being affected on a global scale.

Mercury is found in fish in remote lakes; lead is found in Greenland snow; and PCBs, dioxin, and pesticides are found in the Arctic.

Environmental partitioning

Chemicals, once they are released into the environment, seek out the environmental media (air, water, soil, or biota) in which they are most soluble. Trichloroethylene (TCE) and benzene (two volatile organics) are most soluble in air; thus they tend to be found in air, and inhalation is the principal means of human exposure. DDT and PCBs are most soluble in organic matter; thus they tend to bioconcentrate in biota and are transferred to humans through the food chain. The dynamics of environmental partitioning of chemicals are fairly well understood, and multimedia environmental transport models can provide a coherent account of concentrations of environmental chemicals in all media (*3, 4*). These models have been successful at accounting for the equilibrium partitioning and environmental behavior of such organic chemicals as trichloroethylene, benzene, pentachlorophenol, dioxin, and benzo(*a*)pyrene.

Atmospheric transport is recognized as the primary mode of global distribution and entry into the food chain for organic chemicals. Although most lipophilic compounds reside mainly in the soil, small amounts volatilize to the atmosphere and are transported globally. During transport, organics attach to particles in the atmosphere and are eventually transferred back to the Earth through processes of wet and dry deposition (*5*). After deposition from the atmosphere, lipophilic compounds bioaccumulate in vegetation, beef, milk, and fish; thus the food chain becomes the primary pathway of human exposure for most global pollutants.

Atmospheric pollutants enter the food chain through three routes: direct atmospheric deposition onto leaf surfaces, vapor phase air-to-leaf transfer, and root uptake. For many years vegetative contamination was thought to result primarily from root uptake. Now, however, direct air-to-leaf transfer of vapor phase organics is recognized as the primary pathway of vegetative contamination for most organics (*6–8*). Herbivores take up atmospheric pollutants by feeding on the contaminated vegetation. The pollutants are then taken up in the predators that feed on herbivores. Larsson et al. (*9*) show a direct correlation between atmospheric deposition rates and body burdens in predators.

We offer the following examples of global chemical pollutants that result in human exposure of significant proportions.

Examples of global pollutants

PCBs. Polychlorinated biphenyls are widely versatile, synthetic chlorinated compounds whose manufacture was banned in 1977. In the late 1960s, PCBs were used as dye solvents, plasticizers, dielectric fluids, and hydraulic fluids (*10*). PCBs are highly lipophilic, with the consequence that more than 99% of PCB mass is found in soil. Thus one might not expect PCBs to be a multimedia problem. However, volatilization of PCBs from spills, landfills, road oils, and other sources results in measurable atmospheric emissions (*11, 12*). It is estimated that 9×10^8 g per year of PCBs cycle through the U.S. atmosphere (*13*). This is less than 1% of the total PCBs in the environment. Nevertheless, atmospheric transport is now recognized as the primary mode of global distribution of PCBs. Eisenreich et al. (*1*) estimated that the atmospheric pathway contributes 60–90% of PCB input into the Great Lakes, and Lake Superior receives more than 78% of its PCB burden from the atmosphere (*14*).

Human exposure to PCBs occurs primarily via low-level food contamination. Market basket studies show a background daily intake of 1.4×10^{-5} mg/kg per day (*15*). Twenty-three percent of humans tested have a measurable body burden of PCBs (*16*), and the average level of PCBs in breast milk in the United States is 89 µg/kg (*17*). If we use a cancer potency estimate of 7.7 (mg/kg per day)$^{-1}$, the lifetime cancer risk of background PCB intake is 1.1×10^{-4}.

Primarily as a result of consumption of contaminated fish and aquatic mammals, Arctic inhabitants are experiencing near-toxic levels of PCB exposure. The cold climate increases the bioaccumulation of lipophilic substances in the food chain, causing the PCB levels in the breast milk of the Inuit women of Arctic Quebec to be among the highest ever recorded (*18*). Breast-fed Inuit infants have PCB blood levels that are believed to be causing immune response impairment and increased infection rates (*18*).

Dioxins

2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD) is the most potent chemical carcinogen evaluated by EPA. There are many different forms of polychlorinated dibenzo-*p*-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs), but TCDD is commonly referred to as dioxin. There is a general perception that TCDD contamination is a localized problem and that control of a few specific sources (such as municipal solid waste incinerators) will reduce human exposure to tolerable levels. The reality is that PCDDs and PCDFs are everywhere and virtually every man, woman, and child in the world is exposed to these compounds daily (*19*). PCDDs and PCDFs, including the toxic TCDD, have been measured in practically *all* media—air, soil, meat, milk, fish, vegetation, and human biological samples.

Because dioxin is highly lipophilic, one would expect to find it almost exclusively in the soil. However, a small fraction volatilizes and partitions into the atmosphere and is transported globally. About 20% of atmospheric TCDD exists in the vapor phase (*5, 20*). This vapor phase, after uptake by vegetation, is primarily responsible for human exposure to TCDD. Air-to-vegetation transfer of atmospheric TCDD accounts for about 66% of TCDD in forage that beef and dairy cattle consume (*21*). Consequently, the food chain, especially meat and dairy products, accounts for 99% of human exposure to TCDD (*21*).

Market basket studies of randomly collected food samples of the human diet (*22–25*) show that the mean background dietary intake of PCDDs and PCDFs in industrialized countries is about 93 pg toxic equivalents per day. If we use a cancer potency estimate of 1.56×10^5 (mg/kg per day)$^{-1}$, the lifetime cancer risk from ingestion of food contaminated with background levels of dioxins and furans is 2.1×10^{-4}.

Benzene. Benzene is a widely produced chemical that has been shown to cause leukemia in humans (*26*). Benzene is used commercially as an intermediate in the production of many chemicals and is a by-product of various combustion processes, such as forest fires and the burning of wood, garbage, organic wastes, and cigarettes (*27–29*). The fact that benzene has been measured in air, water, and human biological samples (*30–33*) suggests that environmental contamination from benzene is widespread.

Benzene partitions mainly into the air (99%), with less than 1% partitioning into water, soil, sediment, suspended sediment, and biota. Only a negligible fraction of benzene is expected to sorb to particulates in the air (*5*). Because benzene is not very lipophilic, it does not accumulate greatly in the food chain. Inhalation is the primary human exposure pathway for benzene. Biomonitoring studies show that human exposure to benzene is widespread, with a mean concentration of benzene in personal air of 13.7 µg/m^3, even in rural areas removed from industrial sources of benzene (*32*). Assuming an adult inhalation rate of 20 m^3 per day an
cancer potency estimate for be
zene of 2.6×10^{-2} (mg/kg per day
the corresponding lifetime can
risk from background exposure
benzene is 1×10^{-4}.

Mercury. Mercury is a highly v
atile toxic metal that can exist in
por form in the atmosphere.
primary anthropogenic sources
mercury are the burning of fossil
els; mining and extraction of mer
ry from cinnabar; the chloralkali
dustries; and, to a lesser deg
volatilization from paper pu
paints, fungicides, electrical equi
ment, and municipal waste incin
ation (*34*). With increasing regula
ty, reports are surfacing of larg
scale mercury contamination of fi
from remote lakes with no near
sources of anthropogenic mercur
In remote Michigan lakes, 15%
fish contain mercury in excess
the state health advisory level of
ppm (*35*). Approximately 30%
Wisconsin lakes and 50% of Flori
lakes analyzed (about 100 majo
systems) contain fish with mercu
levels exceeding state health sta
dards (*36*; Ware, F., Florida Gam
and Fresh Water Fish Commissio
personal communication, Ju
1990). This phenomenon suppo
the hypothesis that mercury poll
tion is becoming a global probl
and is not limited to the area su
rounding point sources.

Anthropogenic emissions of me
cury partition mainly between a
(about 66%) and water and so
(about 34%) (*34*) with only a neg
gible fraction found on atmospher
particulates. A high volatility an
long atmospheric residence tim
(about a year) result in its global a
mospheric transport (*34*), atmo
spheric deposition being the maj
source of mercury input into remo
seepage lakes. After deposition in
lakes, mercury is methylated an
bioaccumulates in fish as methy
mercury, the form most toxic to h
mans. Fish consumption is the p
mary pathway of human exposu
to methylmercury.

Anthropogenic mercury releas
do not originate solely from indu
trialized countries in the Northe
Hemisphere. Mercury emissio
from the Amazon region of Sou
America, where mercury is used
the recovery of gold and silver,
count for about 2% (70 tons p
year) of total global atmospher
mercury emissions. Measured me
cury concentrations in fish ne
mining operations in the Amazo
reach 2.7 ppm, five times high

than the Brazilian standard (*38*).

Lead. Lead is a persistent and ubiquitous metal found in paint, gasoline, and many other consumer items. In the United States, about 39,000 tons of lead are emitted to the atmosphere each year, including 35,000 tons as gasoline additives (*39*). Although emissions from leaded gasoline combustion have declined with the decrease in gasoline lead content (*40, 41*), leaded gasoline combustion in vehicles accounted for 90% of the total anthropogenic input into the atmosphere in the United States in 1984. The remaining 10% was from stationary sources: 5% from the metallurgical industry, 4% from waste combustion and energy production, and 1% from miscellaneous sources (*39*). Lead deposited on soil enters the surface layer, where it is subsequently taken up by grazing animals and thus enters the food chain or is consumed directly by children engaging in normal hand-to-mouth behavior (*42*). The major routes of human exposure to lead, therefore, are ingestion and inhalation (*43*). The average baseline lead exposure (µg/day) for a 2-year-old child is 0.05 in inhaled air, 25.1 in food and beverages, and 21.0 in dust, for a total of 46.6 (*39*).

Lead exposure is particularly toxic for children and the young of other species (*44*). The major cause of child lead poisoning in the United States is lead in paint (ingestion), with lead in dust and soil (inhalation and ingestion) dispersed over the nation a close second (*44*). Three to four million children in the United States are estimated to be at risk of adverse health effects and impaired cognitive development from lead, using a blood criterion of 15 µg of lead per deciliter of blood. Maternal exposure to lead is estimated to place 400,000 fetuses per year at risk of excess absorption of lead (*44*).

Global lead pollution is a result of our mismanagement and heavy use of this toxic material. Global anthropogenic lead emissions are about 200 times higher today than in the past (*45*); anthropogenic sources contribute more to lead pollution than do natural sources by one to two orders of magnitude (*41*). There is a current trend to reduce the consumption of lead by removing it from products such as gasoline, paint, food containers, and other items. This reduction will significantly decrease human intake of lead from present sources (*41*). However, more effort is needed in this direction.

Health risk

Humans are exposed to hundreds of synthetic organic chemicals daily. Some of these exposures have been documented through market basket studies to determine dietary intake (*15*) and through adipose tissue surveys. However, the true extent of human exposure to environmental pollution has never been quantified. For example, the human adipose tissue survey has identified only a very small fraction of the chemical mass found in human adipose tissue. The total exposure assessment methodology study demonstrated that human exposure to volatile organics is widespread and that exposures in the indoor environment are generally higher than outside exposures (*32*). Because of widespread use of chemicals in our everyday environment (e.g., paints, gasoline, dry-cleaning agents, cleansers, pesticides), exposures in rural areas are as large as those in urban areas.

We have examined background cancer risks of 11 global pollutants (*46*). We focused our study on pollutants for which data were readily at hand, rather than attempt a comprehensive survey. Using actual measured exposure concentrations and intake levels (except for ethylenebisdithiocarbomatics) and EPA risk methodologies, we calculated the total background risk for these 11 carcinogens to be 1.4×10^{-3} (Table 1). Background cancer risk based on measured exposure can account for 1–2% of actual annual cancer deaths, a number consistent with other estimates of the contribution of chemical pollution to background cancer rates. This risk is far in excess of the generally accepted target range of 10^{-7} to 10^{-4} lifetime cancer risk.

Regulatory approaches

Current regulatory approaches for environmental pollution do not incorporate ways of dealing with global pollution. Instead the major focus has been on protecting the maximally exposed individual. To accomplish this, locally high concentrations of pollutants are decreased, not by destroying the chemicals or decreasing production, but rather by moving them to another environmental medium. For example, stack emissions are controlled by installing stack scrubbers, which use water to clean stack emissions of gases and particulates. However, scrubber water is often flushed into existing municipal sewer systems and carried to the city municipal waste treatment plants. During water aeration at these plants, up to 99% of volatiles are released into the air. In a study of human exposure to toxic substances through air and drinking water for the Philadelphia metropolitan area, EPA found that volatilization of organic compounds from the sewage treatment plant accounted for almost half the air cancer risks to the population (*47*).

The nation is currently engaged in a massive $100 billion effort to clean up hazardous waste sites. However, the usual practice is an exercise in transferring pollution from one environmental medium to another. At 68% of hazardous waste sites with contaminated groundwa-

TABLE 1
Background cancer risk from 11 [illegible]

Chemical and exposure medium	[illegible]
Indoor air	
Benzene	[illegible]
Trichloroethylene	[illegible]
Tetrachloroethylene	[illegible]
Carbon tetrachloride	[illegible]
Formaldehyde	[illegible]
Xylenes	[illegible]
Water	
Chloroform	[illegible]
Food chain	
Dioxins and furans	[illegible]
Dieldrin	[illegible]
EBDCs	[illegible]
PCBs	[illegible]
Total lifetime cancer risk	[illegible]

[a] Risk estimates are based on current EPA [illegible] uncertainties in these methodologies, the above risk estimates may not represent actual [illegible] individuals.

Source: Reference 47.

ter, pumping and treating for aquifer restoration is the selected remedy (*48*). However, treatment most often consists of air-stripping the volatile organics from groundwater, thereby releasing the organics into the air. A recent evaluation of the Superfund program concluded that only 19% of source remedies effectively and permanently cleaned up hazardous waste sites (*49*). Most remedies provide for containment only or partial treatment, or they utilize a technology of questionable effectiveness and permanence. These remedies are designed to minimize short-term exposure, rather than to treat the waste and thereby permanently eliminate the hazards. Thus current remedial practices at hazardous waste sites do not focus on effective reduction of pollutant mass in the global environment.

The only way to diminish global cycling of contaminants is to decrease production of pollutants or to destroy pollutants before they are released into the environment. The production of some especially hazardous pollutants such as DDT and PCBs has been banned. At present, the most commonly used method to destroy pollutants is incineration. However, emissions from incinerator stacks tend to release pollutants directly into the atmosphere. Choices designed to protect the environment are not easily made, and a multimedia perspective is imperative. As knowledge about the pathways, extent, and consequences of pollution increases, the implementation of more innovative ways of controlling the problem will become more urgent.

Reducing risk

The high background cancer risk documented in Table 1 raises the question of how effective EPA can be in reducing the overall cancer risk from environmental chemicals. EPA's regulatory focus is on controlling local exposure to large point sources of pollutants. These sources typically have maximum individual risks in the 10^{-3} to 10^{-4} range, but because risk drops off rapidly with distance from the source, risk to the average individual is typically in the 10^{-5} to 10^{-7} range. Thus EPA regulations, although protective of the maximum exposed individual, do little to reduce the overall U.S. rate of cancer resulting from exposure to chemical pollutants. This observation is consistent with that of Gough (*50*), who performed a detailed analysis of EPA regulations and found that the entire $100 billion spent each year on environmental protection results in at most a 1.3% reduction in cancer death rates.

The difficulty with regulating background risk is that it results from widespread global pollution from a multitude of widely dispersed sources. This pollution cannot be reduced significantly by controlling emissions associated with production and use. When these chemicals are produced and not destroyed naturally or by humans, they will eventually reach the environment. Once that happens, even relatively nonvolatile compounds like PCBs and dioxin are atmospherically transported on a global scale, resulting in global chemical pollution. If we do not want to change our standard of living, the only way to reduce global chemical pollution is to make our production and consumption processes more efficient and to lower the levels of production of these toxic chemicals. Thus the only reasonable solution to global pollution is not increased regulation of isolated point sources, but rather an increased emphasis on waste reduction and materials recycling. Until we focus on these issues, we will continue to experience background cancer risk in the 10^{-3} range.

Acknowledgment

The Oak Ridge National Laboratory is operated by Martin Marietta Energy Systems, Inc., under Contract No. DE-AC05-84OR21400 with the U.S. Department of Energy.

References

(1) Eisenreich, S. J.; Looney, B. B.; Thornton, J. D. *Environ. Sci. Technol.* **1981,** *15,* 30–38.

(2) Velinsky, D. J.; Wade, T. L.; Wong, G.T.F. *Atmos. Environ.* **1986,** *20,* 941–47.

(3) MacKay, D.; Patterson, S.; Cheung, B. *Chemosphere* **1985,** *14,* 859–63.

(4) MacKay, D. et al. *Chemosphere* **1985,** *14,* 335–74.

(5) Bidleman, T. F. *Environ. Sci. Technol.* **1988,** *22,* 361–67.

(6) Bacci, E. et al. *Environ. Sci. Technol.* **1990,** *24,* 885–89.

(7) Hermanson, M.; Hites, R. *Environ. Sci. Technol.* **1990,** *24,* 666–71.

(8) Reiderer, M. *Environ. Sci. Technol.* **1990,** *24,* 829–37.

(9) Larsson, P.; Okla, L.; Woin, P. *Environ. Sci. Technol.* **1990,** *24,* 1599–1601.

(10) Charles, M. J.; Hites, R. A. In *Sources and Fates of Aquatic Pollutants;* Hites, R. A.; Eisenreich, S. J., Eds.; Advances in Chemistry 216; American Chemical Society: Washington, DC, 1987, pp. 365–89.

(11) Weaver, G. *Environ. Sci. Technol.* **1984,** *18,* 22A–27A.

(12) Lewis, R. L.; Martin, B. E. *Envir Sci. Technol.* **1985,** *19,* 986–91.

(13) Murphy, T. J. et al. *Environ. Sci. Te nol.* **1985,** *19,* 942–46.

(14) Eisenreich, S. J. In *Sources and F of Aquatic Pollutants;* Hites, R. A Eisenreich, S. J., Eds.; Advances Chemistry 216; American Chemi Society: Washington, DC, 1987, p 393–469.

(15) Gartrell, M. J. et al. *J. Assoc. Off. An Chem.* **1986,** *69*(1), 146–59.

(16) Murphy, R. F.; Kutz, R. W.; Stra man, S. C. *Environ. Health Pers* **1983,** *48,* 81–86.

(17) Attachment D, final report of Septe ber 1977 on PCBs in human milk, di tributed at a conference on PCB sponsored by the DHEW Committ to Coordinate Toxicology and Relat Programs; Bethesda, MD; Jan. 1978

(18) Dewailly, E. et al. *Bull. Environ. Co tam. Toxicol.* **1989,** *43,* 641–46.

(19) Travis, C. C.; Hattemer-Frey, H. A Silbergeld, E. *Environ. Sci. Techn* **1989,** *23,* 1061–63.

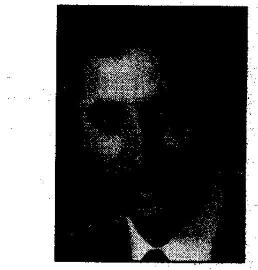
(20) Hunt, G. T.; Maisel, B. E. *Chemo sphere* **1990,** *20,* 1455–62.

(21) Travis, C.; Hattemer-Frey, H. *Chemo sphere* **1987,** *16,* 2331–42.

(22) Beck, H. et al. *Chemosphere* **1989, 18** 587–92.

(23) Ono, M. et al. *Chemosphere* **1987, 16** 1823–28.

(24) Ontario Ministry of the Environment "Polychlorinated Dibenzo-*p*-dioxins and Polychlorinated Dibenzofurans



***Curtis C. Travis** is the director of the Office of Risk Analysis at the Oak Ridge National Laboratory in Oak Ridge, TN. He has a Ph.D. in mathematics from the University of California–Davis.*

***Sheri T. Hester** is an information specialist at Integrated Laboratory Systems in Oak Ridge, TN. She holds a bachelor's degree from the University of Tennessee. Her research topics include hazardous waste cleanup and risk analysis.*

Environ.
-91.
Sci. Tech-

and Fates
es, R. A.;
vances in
Chemical
1987, pp.

Off. Anal.

N.; Strass-
lth Persp.

of Septem-
ı milk, dis-
on PCBs
Committee
nd Related
an. 1978.
viron. Con-
1–46.
rey, H. A.;
i. Technol.

E. Chemo-

H. Chemo-

re 1989, 18,

re 1987, 16,

nvironment
o-p-dioxins
benzofurans

tor of the O
e Oak Ridg
k Ridge, TN
itics from th
ivis.

ormation sp
atory Syste
olds a bach
versity of T
s include h
l risk analy

and Other Organochlorine Contaminants in Food"; Ministry of Agriculture and Food and Environment: Toronto, 1988.

(25) Furst, P.; Furst, C.; Groebel, W. *Chemosphere* **1990,** *20,* 787–92.

(26) *Fed. Regist.* **1984,** *49,* No. 30.

(27) Fishbein, L. *Sci. Total Environ.* **1984,** *40,* 189–218.

(28) International Agency for Research on Cancer. "Monograph on the Evaluation of the Carcinogenic Risk of Chemicals to Humans, Vol. 29, Some Industrial Chemicals and Dyestuffs"; Lyon, France, 1982, pp. 93–148.

(29) Webster, R. C. et al. *J. Toxicol. Environ. Health* **1986,** *18,* 567–73.

(30) Antoine, S. R.; DeLeon, I. R.; O'Dell-Smith, R. M. *Bull. Environ. Contam. Toxicol.* **1986,** *36,* 364–71.

(31) Wakeham, S. G.; Canuel, E. A.; Doering, P. H. *Geochim. Acta* **1986,** *50,* 1163–72.

(32) Wallace, L. *Toxicol. Environ. Chem.* **1986,** *12,* 215–36.

(33) Wallace, L. et al. *Arch. Environ. Health* **1987,** *42,* 272–79.

(34) Mitra, S. *Mercury in the Ecosystem: Its Dispersion and Pollution Today*; Technomic: Lancaster, PA, 1986.

(35) Porcella, D. *EPRI Journal* **1990,** April/May, 46–49.

(36) "Health Advisory for People Who Eat Sport Fish from Wisconsin Water"; Wisconsin Department of Natural Resources/Wisconsin Division of Health: Madison, WI, 1987.

(37) Holdren, J. P. *Energy* **1987,** *12*(10/11), 975–92.

(38) Pfeiffer, W. C. et al. *Sci Total Environ.* **1989,** *87/88,* 233–40.

(39) "Air Quality Criteria for Lead"; U.S. Environmental Protection Agency. U.S. Government Printing Office: Washington, DC, 1986; EPA-600/8-83/028df.

(40) Annest, J. L. et al. *N. Engl. J. Med.* **1983,** *308,* 1373–77.

(41) Jaworski, J. F. et al. In *Lead, Mercury, Cadmium and Arsenic in the Environment*; Hutchinson, T. C.; Meema, K. M., Eds.; Wiley: London, 1987, pp. 3–16.

(42) Jaworski, J. F. et al. In *Changing Metal Cycles and Human Health*; Nriagu, J. O., Ed.; Dahlem Konferenzen No. 28; Springer-Verlag: New York, 1984, pp. 375–88.

(43) Hutton, M. In *Lead, Mercury, Cadmium and Arsenic in the Environment*; Hutchinson, T. C.; Meema, K. M., Eds.; Wiley: London, 1987, pp. 53–68.

(44) "The Nature and Extent of Lead Poisoning in Children in the United States: A Report to Congress"; U.S. Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry: Atlanta, GA, 1988.

(45) Settle, M. D.; Patterson, C. C. *Science* **1980,** *207,* 1167–76.

(46) Travis, C. C.; Hester, S. T. *Risk Anal.* **1990,** *10*(4), 463–65.

(47) Haemisegger, E. R.; Jones, A. D.; Reinhardt, F. L. *J. Air Pollut. Control Assoc.* **1985,** *35,* 809–15.

(48) Doty, C. B.; Travis, C. C. *J. Air Pollut. Control Assoc.* **1989,** *39*(12), 1535–43.

(49) Travis, C. C.; Doty, C. B. *Environ. Sci. Technol.* **1989,** *23,* 1333–34.

(50) Gough, M. *Risk Anal.* **1990,** *10*(1), 1–6.