UNITED STATES ENVIRONMENTAL PROTECTION AGENCY

JECT: Serious Investigation of 15 Chemicals

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TO:
Assistant Administrators
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The Office of Toxic Substances has prepared a draft status assessment of available information concerning the 15 chemicals for which Administrator Costle has publicly announced a serious investigation during the next year. Regulatory actions on many of the substances are likely to result from the investigation.

I would appreciate your review of this attached draft material. If you possess information which can be used to make the draft more complete, please include the information as it is received. Dr. I.E. Wallen, of the Office of Toxic Substances, has been designated to coordinate this information development effort. Your comments may be sent directly to him. We plan to share all information during the next few months as gathered internally, from other agencies and in international cooperation, in order that full chemical management action may be considered.

For purposes of information sharing you may wish to designate a person or persons in your office as a contact point for Dr. Wallen. We will duplicate and distribute major papers and other documents.

ACRYLONITRILE

Statement of Concern

In January 1977, the Manufacturing Chemists Association released the midpoint results of a two-year study showing that high levels of acrylonitrile incorporated into the drinking water of rats used in the experiment caused lowered body weight, changes in the gastric epithelium, tissue masses in the ear duct, and central nervous system lesions. In May 1977, DuPont reported preliminary findings of increased cancer incidence and mortality at a textile fibers plant where workers were exposed to acrylonitrile. Both studies are scheduled for completion in early 1978.

Health and Ecological Effects, Environmental Behavior

Preliminary results of a survey of workers at a DuPont plant which used acrylonitrile indicate that cancers were 2.5 to 3 times the expected rate among the employees who had worked in that part of the plant beginning 20 or more years before. Cancers of the lung and large intestine predominated. Further efforts to follow up on employees no longer with the company and the gathering of other etiological data, such as on smoking habits, will be needed before the full meaning of the data can be assessed.

The MCA is sponsoring three studies of the oral and inhaled toxicity of acrylonitrile. Dow is the researcher in all three tests. In pregnant rats, 25 mg/kg/day of acrylonitrile administered by gavage increased teratogenic effects, particularly skeletal and circulatory anomalies. The dams themselves demonstrated a variety of toxic effects at 65 mg/kg, such as reduced weight gain, increased liver/body weight ratio, and gastric thickening. In the two-year drinking water study, 12-month results indicate increased occurrence of subcutaneous mammary masses in females, masses in the ear canal, hyperplasia and polyps of the stomach, and lesions in the central nervous system, particularly in the high-dose groups (300 ppm). The full significance of these findings will be determined when the study is completed in early 1978. The two-year inhalation study is about half completed, and data are also due to be available in early 1978. Preliminary findings show similar effects by inhalation as from ingestion in water.

Acrylonitrile is highly toxic and can be absorbed through intact skin, by inhalation, and by ingestion. It possesses many of the characteristics of poisoning by cyanide, but studies have shown it to be the compound itself, and not the liberated cyanide, which causes the toxicity. The toxicity by other routes of administration has been studied. Orally, the LD_{50} in the mouse is on the order of 35 mg/kg; in the rat, about 78 mg/kg; and in the guinea pig, about 90 mg/kg. Acrylonitrile is known to be a severe skin and eye irritant.

Aquatic studies have shown a LD_{100} - 24 hours at 100 mg/l for fish. In aerobic treatment systems, studies have shown that it exhibits negligible oxygen utilization and the dehydrogenase response indicates that it had an inhibitory effect initially on the microbial population. The standard cyanide analytical test methods for water measure are liberated cyanide (cyanide ion) and not the organic cyanides. At the present time, there are no standard analytical tests for acrylonitrile or for the other organic cyanides.

Sources, Environmental Levels, and Population at Risk

Acrylonitrile is used as an intermediate in the production of a wide variety of acrylic fiber plastics and elastomers.

The largest use of acrylic fibers is in wearing apparel and carpeting and it is used in blankets, draperies, upholstery, wigs, man-made furs, sand bags, and industrial cloth. ABS (acrylonitrile-butadiene-styrene) resins are used in automobiles, applicances, and piping. SAN (styrene-acrylonitrile) resins are used in automobile panels, battery cases, tumblers and filaments. Nitrile elastomers (copolymerizing with butadiene) are used in seals, gaskets, hoses, belts, adhesives, can closures, footwear, and brake linings.

It is used as an intermediate in the manufacture of adiponitrile, and acrylamide monomers, and polymers. Adiponitrile is one of the materials used in Nylon 66. Acrylamide polymers are primarily used in manufacturing chemicals for water and waste treatment processes. Other polyacrylamide uses are for papermaking strengtheners and retention aids, drilling mud additives, textile treatment and surface coatings. Nitrile barrier resins are being test-marketed for making beverage and food containers. Other uses for acrylonitrile are as a grain fumigant, an anti-stall additive for gasoline (Dow Ambifal 200), and for other industrial chemicals.

In 1973, it was the 50th largest chemical produced, 1.353 billion pounds, which was up by 20 percent over 1972. 1974 projected production was 1.55 billion pounds.

At the present time, there are four companies producing at six locations. Exports have been decreasing each year as foreign capacity comes on stream.

The amount of by-products produced from acrylonitrile production is estimated to be 150 million pounds each for acetonitrile and hydrocyanic acid in 1974. Both of these materials are highly toxic by all routes to man, and are toxic to all species of aquatic life.

Although monitoring data are not available, there is predicted wide-spread low level exposure to acrylonitrile.

All polymers contain some unreacted monomer and oligomers (incompletely polymerized material), the amount depending on the conditions used in polymerization and subsequent processing. These unreacted materials are slowly released during use and disposal of the final products. Since many fabrics contain acrylonitrile, and since acrylonitrile can be absorbed through intact skin, there is a potential hazard of direct exposure based on unreacted monomers. In addition, unreacted monomer is probably leached during laundering, thus creating a water pollution problem. The presence of residual acrylonitrile in polyacrylamide used for water treatment is an additional potential source of water pollution. Further testing to determine unreacted monomer levels is needed.

Additional hazards result from the disposal of acetonitrile and HCN, by products from the manufacture of acrylonitrile, and the production of HCN resulting from improper incineration of acrylonitrile products.

Technical and Economic Considerations

The economic impacts and technical feasibility of controls cannot be estimated because information to determine regulatory approaches is not yet available. These data should be available later in the year.

Regulatory Action in Progress

OSHA has established the threshold limit-value of 45 mg/m^3 as a workplace exposure standard. A downward revision of the standard is under consideration. NIOSH is preparing documentation for use in this effort.

The Food and Drug Administration has regulated the unreacted monomer content of nitrile rubber that comes into contact with food at 11 ppm, and has proposed a ban on plastic soft drink bottles made of acrylonitrile.

Acrylonitrile used as a grain fumigant is being considered for rebuttable presumption proceedings under FIFRA. The substance is also registered under the gasoline additive provisions of the Clean Air Act.

ARSENIC

Statement of Concern

In 1975, OSHA proposed a strict standard for workplace air exposure limits to inorganic arsenic. Earlier EPA sampling had found that atmospheric concentrations near two copper smelters exceeded the proposed limit (Anaconda, Montana; and Tacoma, Washington) and closely approached it at three other smelter sites. Results of an EPA-sponsored pilot epidemiology study near an arsenical pesticide plant in Baltimore reveal lung cancer rates several times the national average. A number of arsenical compounds are being considered for rebuttable presumption proceedings under FIFRA/FEPCA. The Cancer Assessment Group of EPA has confirmed the carcinogenicity of inhaled arsenic.

Health and Ecological Effects, Environmental Behavior

Liver, skin, lung, and lymphatic cancers, and adverse effects on the thyroid gland have been reported in epidemiological studies of occupationally exposed individuals. The main threat of arsenic as a carcinogen is inhalation of the inorganic forms. A preliminary mortality study of the population surrounding Allied Chemical Company's arsenical pesticide plant in Baltimore revealed a lung cancer rate sixteen times the national average. A previous study has shown that retired workers from this plant suffer from lung cancer at a rate seventeen times the national average. A Dow Chemical Company study indicated an excess of lung and lymphatic cancers among their workers who had been exposed to arsenical compounds. Arsenic occurs in two forms: trivalent and pentavalent. Inorganic trivalent arsenical compounds are much more toxic than pentavalent, both acutely and chronically. Pentavalent arsenic is often found in metallo-arsenicals, and is of concern because it can degrade into the trivalent form. A study of workers at the Tacoma plant of ASARCO has shown that neural conductivity in workers is altered as a result of exposure to arsenic.

A 1972 outbreak of arsenic poisoning in Getchell, Nevada, is attributed to stack effluent from a gold smelter. Studies made abroad have suggested that arsenic may be a skin carcinogen when ingested in drinking water at levels as low as 0.3 mg/l. The debate over the carcinogenicity of arsenic is largely due to the fact that the animal studies conducted to date have not shown a relationship between ingested arsenic and cancer. Recent studies have shown that organic arsenical compounds produce additional toxicological problems. For example, Carbarsone has been reported to produce liver cancer in trout through ingestion (480 mg/100 g diet).

Trivalent arsenic oxide is particularly toxic to legumes and other crop plants. Depending on the soil type, 6 ppm arsenic can cause a 50 percent growth reduction. Phytotoxic levels of arsenic have been found as far as two miles from the Tacoma smelter. Once combined in soil, arsenic is extremely persistent.

Sources, Environmental Levels, and Population at Risk

Inorganic arsenic is emitted to the air from several sources, including copper, lead, and zinc smelters, glass production plants, coal-burning facilities, cotton gins, arsenical-compound (including pesticides) production plants, and pesticide application. Organic arsenic discharges are associated with the manufacture and use of pesticides. Trivalent arsenic occurs naturally, is a common contaminant of ores, and is the major component of arsenic emissions from smelters. Based on EPA estimates, the 15 copper smelters contribute most heavily to air emissions of inorganic arsenic. The Anaconda copper smelter in Montana, and the ASARCO copper smelter and arsenic plant in Tacoma, Washington, have been identified as having the highest arsenic emissions. Other industrial sources generally emit less arsenic than copper smelters. Air levels in most urban areas for 1973 and 1974 were at or below the level of detection (0.001 ug/m^3). Levels in areas near smelters ranged from 0.003 to 4.86 ug/m^3.

The land disposal of arsenical wastes can become a long-range public health hazard. A good example is Perham, Minnesota, where eleven people were poisoned by contaminated well water in 1972.

A 1975 survey of drinking water supplies showed that about one percent exceeded the interim drinking water standard of 0.05 mg/l. Trivalent arsenic is found at high levels in some groundwater. Underground injection of arsenical pesticide wastes in Philadelphia has contaminated a nearby stream which is being considered for use as a drinking water supply.

Three new technologies for energy production have important arsenic implications. Early data on coal gasification indicate that two-thirds of the arsenic present is volatilized. Oil shale exploitation and geothermal energy development may also release large quantities of arsenic.

Technologic and Economic Imolications

In general, particulate control measures (multicyclones, balloon flues, and electrostatic precipitators) are used to reduce arsenic emissions. Baghouses offer the greatest potential for control, but have not been widely adopted by the smelting industry because of high capital and maintenance costs. Costs and feasibility of emission controls will vary from plant to plant. Significant control efforts are being planned at the ASARCO smelter in Tacoma, and are underway at Anaconda. Conventional water treatment technology has been shown to be effective in meeting the arsenic drinking water standard. Arsenic concentrations of 0.1 and 1.6 mg/l in wastewater can inhibit waste treatment by activated sludge and anaerobic digestion respectively. Thus, concentrations exceeding these levels can present an additional hazard in wastewaters subjected to these treatment methods. Air and water pollution control efforts normally result in a solid waste or sludge. At present these materials are being stored, pending development of acceptable disposal technologies.

Regulatory Actions in Progress

EPA is locating and monitoring arsenical discharges, and is conducting several studies to determine the toxicity of various arsenical compounds. Limited epidemiological studies are planned to help determine effect levels. Studies have been initiated to determine control technologies and costs for arsenic reduction, and an Air Pollution Assessment Report on Arsenic has been prepared. EPA is considering the development of regulations under the Clean Air Act based on the findings of the Cancer Assessment Group. A review of the use of arsenical pesticides has recently been completed, and research into disposal techniques for arsenical wastes is planned. A scientific and Technical Assessment Report is being prepared in EPA and will incorporate the National Academy of Sciences study on health effects.

In November 1975, OSHA proposed a workplace exposure limit for inorganic arsenic at 4 ug/m^3 (8 hour). After review of comments and hearing transcripts, that Agency expects to promulgate a standard in early fall of 1977.

ASBESTOS

Statement of Concern

OSHA has proposed lowering its workplace standard by a factor of ten on the basis of recent epidemiological data suggesting wider spread health effects than previously suspected. A number of major commercial sources of airborne asbestos are limited by EPA regulations. The Agency is investigating taconite and other hard-rock mining operations, where asbestos is a major ore contaminant. EPA's nationwide sampling program is showing levels of asbestos fibers in water supplies, natural runoff, and discharges from manufacturing and mining sites. Serpentinite rock crushed and used in playgrounds and roadways has been found to be a major contribution to asbestos levels reported in the Washington, D. C. area.

Health and Ecological Effects, Environmental Behavior

Airborne asbestos fibers have been known to cause asbestosis, lung cancer, and pleural and peritoneal mesothelioma. OSHA cites a number of studies showing gastrointestinal (GI) cancer in workers exposed to asbestos. In one study of insulation workers in the United States, seven percent of deaths could be attributed to asbestosis, which on the average appeared about 20 years after first exposure -- the same latency period as for most cancers. Available epidemiological data show that lung cancer is responsible for as much as 20 percent of all deaths among certain types of asbestos workers; mesothelioma, 11 percent; and GI cancer, 8 percent.

There are few if any data on the dose-response relationships of asbestos fibers in either air or water. Effects of airborne asbestos are far better documented than those of waterborne. OSHA cited workers who had developed mesothelioma at exposure levels below the previous standard of 5 fibers per cubic centimeter in its recent proposal to reduce the level by a factor of ten. There is some evidence that asbestos diseases, including mesothelioma, occur in families of workers exposed to asbestos at levels presumed to be much lower than direct occupational exposure.

Asbestos fibers are extremely resistant to degradation in the environment. Thus far, it has been impossible to demonstrate adverse effects on plants. Some adverse effects on animals have recently been reported.

Sources, Environmental Levels, and Population at Risk

The United States utilized approximately 800,000 tons of asbestos fiber in 1974. Asbestos products are widely used in the construction industry (asbestos-cement pipe, building and other construction products, and floor tile). Other products include friction materials (such as brake linings), felt and paper, packings and gaskets, and fireproof textiles.

It has been estimated that 85 percent of the asbestos is tightly bound in products and is therefore not as available to the environment as are airborne and waterborne asbestos fibers generated in the mining and milling of asbestos ore, manufacture and fabrication of asbestos products, and disposal of solid wastes from these processes. Asbestos was used in spray insulation in buildings between 1950 and 1972. This may become a major source of environmental discharge as buildings constructed during this period are demolished.

Asbestos minerals are found throughout the United States. Significant quantities of asbestos fibers appear in rivers and streams draining from areas where asbestos bearing outcroppings of serpentine rock are found. Some of these outcroppings are being mined. Asbestos fibers have been found in a number of drinking water supplies, but the health implications of ingesting asbestos are not fully documented. Emissions of asbestos fibers into water and air are known to result from mining and processing of some minerals. Asbestiform-fibers in the drinking water of Duluth and nearby communities at levels of 12 million fibers per liter have been attributed to the discharge of 67,000 tons of taconite tailings per day into Lake Michigan by Reserve Mining.

Exposure to asbestos fibers may occur throughout urban environments. A recent study of street dust in Washington, D. C. showed approximately 50,000 fibers per gram, much of which appeared to come from brake linings. Autopsies of New York City residents with no known occupational exposure showed 24 of 28 lung samples to contain asbestos fibers, perhaps resulting from asbestos from brake linings and the flaking of sprayed asbestos insulation material.

Technologic and Economic Implications

Coagulation treatment and filtration are necessary to remove contaminant asbestos from water. Filtration technologies for air, while meeting the no-visible-emission standard, permit large quantities of asbestos fibers to escape. Fibrous glass has frequently been substituted for applications requiring insulative properties, but there is some debate over its safety. For some other applications, such as brake linings, economically feasible substitutes may not be available.

There is no inexpensive, standardized analytical method for measuring asbestos, and monitoring costs are very high.

Regulatory Actions in Progress

An air standard has been promulgated for a number of major commercial sources of asbestos fibers. Hard-rock mining and taconite beneficiation, where asbestos is an ore contaminant, are being investigated. Effluent guidelines have been promulgated under the Federal Water Pollution Control Act which, together with the NPDES permit program, should reduce asbestos discharges.

EPA is sponsoring an extensive national asbestos monitoring program. Preliminary findings indicate that asbestos is a widespread contaminant of drinking water. NAS is reviewing the implications of these preliminary findings. Reserve Mining has agreed to halt the discharge of taconite tailings into Lake Superior. Standard analytical methods are being developed for both research and monitoring purposes. A number of epidemiology studies to further clarify the health risks of asbestos are being sponsored by EPA.

In 1972, OSHA established a workplace exposure standard. In October 1975, OSHA proposed a further reduction in the level. The National Institute of Environmental Health Sciences (NIEHS) is conducting ingestion experiments to clarify health hazards of this route of exposure; EPA is partially sponsoring these studies. The results will be available in 1979.

BENZENE

Statement of Concern

Benzene, a component in gasoline and an important feedstock for the chemical industry, has been the subject of numerous published reports linking leukemia with worker exposure. Large quantities of benzene are discharged into the environment from automobiles, and probably from stationary sources. NIOSH has promulgated an emergency workplace standard which has been stayed by court order. EPA has listed benzene as a hazardous pollutant under Section No. 112 of the Clean Air Act.

Health and Ecological Effects, Environmental Behavior

Numerous fatalities from occupational benzene poisoning have been reported since the early 1900's. After inhalation or ingestion, benzene is absorbed rapidly by the blood. At non-lethal concentrations, a variety of human central nervous system disorders are observed, depending upon the extent of exposure. These include euphoria followed by giddiness, headache, nausea and staggering gait, as well as fatigue, insomnia, dizziness, and unconsciousness. Observed damage to the human blood-forming system includes anemia, reduction in platelet numbers, and depression of the white blood cell count.

Chronic benzene exposure also has resulted in chromosome aberrations in human lymphocytes. As early as the 1930's, benzene was suspected in cases of leukemia. Available epidemiological data indicate that the compound does induce leukemia although the data cannot be considered to constitute unequivocal evidence that benzene acting alone is leukemogenic. Attempts by the National Cancer Institue and others to induce leukemia in animals with benzene have not been successful. However, the results of inhalation experiments with mice, the species most susceptible to leukemia, are not yet available.

Based on its vapor pressure and chemical stability, benzene is expected to be quite mobile and persistent. Adverse effects on ecological resources have not been reported.

Sources, Environmental Levels, and Population at Risk

In 1973, over 10 billion pounds of benzene were produced from petroleum and coal in the United States. This volatile, colorless, flammable liquid is used mostly for synthesis of organic chemicals. It has been estimated that at least 80 million pounds of benzene may be lost to the environment during benzene production, storage, and transport, while as much as 650 million pounds may be released during its use to produce other organic chemicals. The latter figure was calculated from the difference between 100% yield and the reported yield in these reactions. Therefore, this is only a crude measure of the worst-case benzene emissions during usage. Emissions are concentrated in the Texas Gulf area and the Northeast.

It has been calculated that approximately one billion pounds of benzene were released with hydrocarbon emissions from motor vehicles in 1971 in a geographical pattern similar to population distribution. Another 22-24 million pounds of benzene may be released into the environment each year with spilled oil. Hydrocarbon emissions from non-transportation sources, such as coke ovens and power plants, may also contain considerable amount of benzene. Additionally, benzene is an active ingredient in a number of insecticides and miticides, although the amount of release to the environment from this source has not been calculated.

In an EPA study of organic compounds in the drinking water of ten cities, benzene was detected in water from four cities at concentrations ranging from 0.1-0.3 ug/l. Previous studies reported levels up to 10 ug/l. Average levels of benzene detected in air in a limited number of studies are in the low ppb range with one high reading of 23 ppm reported in the vicinity of a solvent reclamation plant. No data have been found on levels of benzene in soil, wildlife, and fish. Benzene is widely enough distributed that most people are probably exposed to very low levels; the health implications of this type of exposure are not known.

Technologic and Economic Implications

Reduction in organic compound emissions to achieve the National Ambient Air Quality Standard for oxidants should result in some reduction in benzene emissions. As a result of lead removal from gasoline, the average content of aromatics, including benzene, in gasoline is likely to increase slowly. However, hydrocarbon emission controls on motor vehicles should result in a net reduction in benzene emissions.

Regulatory Actions in Progress

In 1974 NIOSH published a criteria document for occupational exposure to benzene which recommended adherence to the existing Federal standard of 10 ppm as a time-weighted average with a ceiling of 25 ppm. OSHA has proposed a reduction of the standard to a 1 ppm 8-hour time weighted average. with a ceiling of 5 ppm over a 15-minute period. An OSHA Emergency Temporary Standard has been stayed, pending a hearing on a request for injunction. NIOSH is conducting retrospective studies of benzene mortality and airborne benzene levels in service stations.

EPA has initiated an air monitoring program which will determine benzene levels in selected areas. Qualitative results obtained to date indicate widespread low-level benzene contamination. Studies are in progress to document the extent of hazard and the best regulatory approach under the Clean Air Act. EPA has conducted a limited survey of drinking water supplies in which benzene was identified in some samples, and has begun a more

extensive survey which will seek out benzene as well as a number of other pollutants. Benzene has been designated a hazardous substance under FWPCA, and ocean dumping is already strictly regulated. The Consumer Products Safety Commission is awaiting the results of the National Academy of Sciences study on the health effects of benzene, and will determine if action is appropriate when the results have been received.

BENZIDINE

Statement of Concern

Benzidine, a known human carcinogen, is used as an intermediate in the manufacture of a number of azo dyes for textile, leather, and paper products. In addition to the EPA concern over liquid effluent discharges containing benzidine, recent research results suggest that some of the benzidine-derived azo dyes may reconvert to benzidine in man or in certain environments. A major labor union (AFL/CIO) has expressed strong interest in any action taken on benzidine.

Health and Ecological Effects, Environmental Behavior

For a number of years, the manufacture and use of benzidine have been associated with a high risk of bladder cancer among exposed workers. Many scientists believe that tumors can result from ingestion, inhalation, or skin absorption of benzidine. A number of animal studies have demonstrated the carcinogenic potential of benzidine. Mice, rats, and hamsters develop liver tumors, and dogs may develop bladder cancer as a result of exposure. Such studies have many deficiencies for estimating the risk to humans which is associated with the levels of exposure to carcinogens likely to be encountered in the environment.

Free benzidine has been detected in the urine of monkeys fed benzidine-derived azo dyes, establishing a potential for reconversion of azo dyes to benzidine. Metabolism of benzidine-derived azo dyes may be similar in humans. Japanese silk painters reportedly have a high incidence of bladder cancer, possibly resulting from licking brushes and spatulas coated with benzidine-derived azo dyes. However, the carcinogenicity of such dyes has not been specifically determined.

Industrial data indicate that benzidine entering a waterway dissipates and may be degraded by naturally occurring processes. Confirmatory investigations have not been conducted. Other aspects of environmental behavior have not been addressed. It has been hypothesized that azo dyes can reconvert to benzidine under certain undefined environmental conditions.

Sources, Environmental Levels, and Population at Risk

The three identified manufacturers (Allied, GAF, and Fabricolor) estimate that they produce 45 million pounds of azo dyes annually from benzidine. The dyes are used by about 300 major manufacturers of textile, paper, and leather products. The largest manufacturer (Allied) recently announced its intention to phase out benzidine production.

The principal environmental concern at benzidine production facilities has been the amount of benzidine in the waste effluents discharged to publicly owned waste water treatment works (POTW's). However, the only discharge measurements to date have been made by industry, which has contended that discharges at any facility usually do not exceed one pound per day. Benzidine

is believed to be present in the sludge removed from industrial pretreatment plants. The environmental adequacy of land disposal of these sludges is unknown. According to industry data, discharges from the POTW are usually below the limit of detection. However, there have been significant accidental releases to POTW's. Levels of benzidine exceeding 5 mg/l can inhibit anaerobic digestion wastewater treatment processes. Thus, concentrations above this level at the POTW present a problem to POTW's using this process, and a possible hazard to the receiving waters.

Free benzidine is present in the benzidine-derived azo dyes. According to industry, quality control specifications require that the level not exceed 20 ppm and in practice the level is usually below 10 ppm. Industry has estimated a total environmental discharge at the 300 user facility sites of 450 pounds per year or about 1.5 pounds per year per facility, assuming all of the free benzidine is discharged in the liquid effluent.

No measurements have been reported for benzidine in ambient air, surface water, or drinking water. Further, no measurements for free benzidine in finished products containing azo dyes have been reported.

Technologic and Economic Implications

The principal liquid effluent control technology currently being used is the reaction of benzidine with nitrous acid. While effective in destroying benzidine, hazardous decomposition by-products may be formed. Industry thus far has rejected carbon adsorption as uneconomical. The costs of treatment at the benzidine manufacturing plants are of far less concern than at the user plants. Thus, there is a continuing industrial emphasis on reducing the levels of free benzidine in dyes, which result from more complete reactions and release less benzidine into the environment.

If limitations were imposed on benzidine production or use, the vacuum would probably be filled by imported benzidine-derived dyes and substitute dyes. However, some of the possible substitutes, such as ortho-toluidine, are also of environmental and occupational health concern. Industry estimates that adequate substitutes would be three to five times more expensive. In some highly specialized uses, particularly for the halogenated benzidine dyes, a technically adequate substitute may not be available.

Regulatory Actions in Progress

The stringent work place standards required by OSHA because of the carcinogenic nature of benzidine reduce environmental discharges resulting from inadequate house-keeping procedures at benzidine manufacturing sites.

EPA proposed a toxic pollutant effluent standard in December 1973, and is planning to repropose such a standard and a pretreatment requirement during the next few months. The results of current animal experiments at the National Center for Toxicological Research, addressing chronic toxicity, carcinogenicity, and metabolic behavior, should be available during the next year and a half. Benzidine is also being examined in the expanded EPA drinking water survey.

CADMIUM (Cd)

Statement of Concern

As evidence emerges that cadmium levels in the environment may be increasing, concern mounts over the accumulation of this substance's in the body at low levels of exposure. Cadmium, which is used in a variety of commercial and consumer products, is believed to reach man through a number of routes, particularly as a contaminant of fish and other foods. There is recent concern over the presence of cadmium in sludge which might reach the food chain as a result of leaching from disposal sites or use as a soil conditioner. A proposed amendment to the Clean Air Act calls for explicit EPA attention by 1977 to a possible air standard for cadmium.

Health and Ecological Effects, Environmental Behavior

Cadmium accumulates in the kidney cortex, where it can cause damage to the renal tubules at levels on the order of 200 ppm. The results of autopsy studies show current levels of 15-50 ppm in the kidneys of people over the age of 50 who were not occupationally exposed; the higher levels generally reflect those found in individuals who had been smokers. Autopsy data on the occupationally exposed are inconclusive because samples have been too small.

At high levels of cadmium exposure, other effects, such as bone brittling, have been observed, mainly in Japan, where widespread occurrence of Itai-Itai disease caused nearly 100 deaths. These effects resulted from an estimated intake of 600 ug/day. The average American diet contains 50-75 ug/day. Heavy fish eaters receive a higher dose, but well below the levels observed in Japan. About five percent of ingested cadmium is retained in the body, and its biological half-life in humans is estimated to be at least 15 years.

Prolonged exposure to cadmium dust can cause emphysema. Recent epidemiological studies indicate abnormally high rates of several forms of cancer due to occupational exposure. Hypertension has been developed in laboratory animals after prolonged exposure to low levels. The presence of cadmium in human fetal tissues during prenatal life shown that the metal traverses the placenta. Experimental studies in laboratory animals have confirmed this observation and have also shown that cadmium is a potent teratogen.

Cadmium particulate in air falls out into water and soils. Plants take it up from the soil, and people and animals ingest cadmium from these sources. Uptake from contaminated water has not been so well documented; it is suspected that this is the significant route of exposure to fish.

Sources, Environmental Levels, and Population at Risk

Cadmium is produced in conjunction with zinc refining. In 1974, the total U.S. consumption of cadmium was about 6300 metric tons, at a cost of about $8500 per metric ton. About one-third was imported. By 1985, demand is expected to reach 9600 metric tons. Of the total use in 1975, about 55 percent went to electroplating, 21 percent to plastic stabilization, 12 percent to pigments, 5 percent to batteries, and 7 percent to a variety of other uses. Major growth is expected in the nickel-cadmium battery industry.

An EPA-sponsored study estimated that a total of 1800 metric tons of cadmium were released to the environment in 1974. Of this, about 20 percent was from zinc mining and smelting, via air, water, and tailings; 50 percent was from such indirect sources as fossil fuel combustion, fertilizer use, and disposal of sewage sludge; and 30 percent was from industrial uses, such as resmelting of cadmium-plated scrap, incineration of plastics containing cadmium, and electroplating.

The major sources of human exposure are food and tobacco contamination, while direct water and air intake appear to be very minor contributors. Groundwater contamination as a result of waste disposal, however, is common. The Food and Drug Administration marketbasket survey has been identifying low levels of cadmium in most composite class samples. Thus, virtually everyone is exposed to trace levels of cadmium. Recent studies indicate that, in Sweden, cadmium concentrations in wheat may be increasing at a rate roughly proportional to levels of industrial use. Increasing soil levels may result from airborne fallout, fertilizer use, and cadmium in irrigation waters. Cadmium has been identified in soils at several locations at levels of 0.55 to 2.45 ppm.

Cadmium levels of 1 to 10 ug/l have been found in 42 percent of available ambient water samples, with more than 10 ug/l in four percent. Fifty-four percent of the samples did not contain measurable amounts. The annual release of cadmium to the air at one copper smelter was estimated to be 250 tons per year. Ambient air levels averaged .021 ug/m3. Soil levels of cadmium were about 1.6 ppm, between one and five miles from the smelter, and were reflected by average findings of 4.7 ppm in leafty vegetables.

Technologic and Economic Implications

Substitutes are or will soon be available for most but not all electroplating uses and for plastic stabilizer use at comparable cost and efficacy.

A cadmium level of 0.02 mg/l, has been shown to inhibit wastewater treatment by anaerobic digestion. Should cadmium concentrations exceeding that level reach a wastewater treatment plant using anaerobes, a hazard may be presented to the receiving water. Trace contamination of air and water by cadmium is common. Removal of such components is usually extremly costly.

Regulatory Actions in Progress

NIOSH is expected to submit a criteria document to OSHA this year at which time the existing workspace standard will be reviewed. The Food and Drug Administration has banned certain uses of cadmium pigments and cadmium-containing materials.

Epidemiological studies are being conducted by the World Health Organization to determine whether cadmium may be a factor in hypertension and cardiac disease in humans. The National Cancer Institute is sponsoring studies to investigate the carcinogenic potential of cadmium metal, cadmium oxide, and cadmium sulfide.

EPA has prohibited the ocean dumping of cadmium, except as trace contamination. The effluent guidelines for the electroplating industry address cadmium released from this segment of the economy, and hazardous spill regulations include some cadmium compounds among the substances for which spill penalties have been established. An Interim Primary Drinking Water Standard has been issued and pesticides containing cadmium are being reviewed for possible Rebuttable Presumption Against Registration proceedings. A Scientific and Technical Assessment Report on cadmium has documented health and technological concerns for EPA.

HEXACHLOROBENZENE

Statement of Concern

Despite the steps taken by several States and several companies to reduce environmental discharges of hexachlorobenzene, environmental contamination persists. Recent reports of the occurrence of hexachlorobenzene in human adipose tissues (95 percent of those sampled), the food supply, effluents, drinking water, and pesticides (in addition to registered pesticidal use) add to earlier concerns of U. S. regulatory agencies. In 1973, EPA made a public commitment in response to a petition from the Department of Agriculture to set a food tolerance for hexachlorobenzene. Hexachlorobenzene has recently been shown to be a carcinogen in hamsters.

Health and Ecological Effects, Environmental Behavior

The death of breast-fed infants and an epidemic of skin sores and skin discoloration were associated with accidental consumption of hexachlorobenzene-contaminated seed grain in Turkey in the mid-1950's. Doses were estimated at 50 to 200 mg/day for several months to two years. Clinical manifestations included weight loss, enlargement of the thyroid and lymph nodes, skin photosensitization, and abnormal growth of body hair. Hexachlorobenzene levels of up to 23 ppb in blood are believed to have contributed to enzyme disruptions in the population of a small community in southern Louisiana in 1973.

Long-term (up to 3 years) animal ingestion studies show a detectable increase in deaths at 32 ppm, cellular alteration at 1 ppm, biochemical effects at .5 ppm, and behavioral alteration between .5 and 5 ppm. Apparently, the effective dosage to offspring is increased by exposure to the parent. A 12 percent reduction in offspring survival results when exposure to very low levels had been continuous for three generations. Teratogenic effects appear minimal.

A recent feeding study in hamsters showed that hexachlorobenzene causes hepatomas and hemangioendotheliomas of the liver. Doses of 50, 100, and 200 ppm of hexachlorobenzene mixed with feed were used. Hepatomas were observed at all dose levels, at rates and latencies appearing to be dose related, but not in the controls. Hemangioendotheliomas appeared only in the high dose group (9% in females, 34% in males), and three of these tumors in males gave metastases.

While hexachlorobenzene appears to have little effect on aquatic organisms, a bioaccumulation factor of 15,000 has been demonstrated in catfish. The chemical is toxic to some birds. Eighty ppm caused death, and 5 ppm caused liver enlargement and other effects in quail. The half-life in cattle and sheep is almost 90 days. Hexachlorobenzene is very stable. It readily vaporizes from soil into the air; emissions to air in turn contaminate the soil.

Sources, Environmental Levels, and Population at Risk

About 90 percent of the estimated 8 million pounds of hexachlorobenzene produced annually in the United States is as a by-product at 10 perchloroethylene, 5 trichloroethylene, and 11 carbon tetrachloride manufacturing plants. Hexachlorobenzene is commonly detected in solid wastes and liquid effluents. Most of the remaining production is as a by-product at more than 70 other sites producing chlorine and certain pesticides. About 45,000 pounds per year are released into the environment during pesticide use. Hexachlorobenzene has also been found in the waste tars from vinyl chloride and other chlorine-product plants.

In 1975, forty-six percent of the soil samples collected at 26 locations along a 150-mile transect in Louisiana were contaminated with HCB at levels from 20 to 440 ppb. Although water samples were generally below 3 ppb, one sample below an industrial discharge contained 90-ppb. Air immediately adjacent to production facilities has shown concentrations from 1.0 to 23.6 ug/m^3. Most of the hexachlorobenzene appeared to be associated with particulates, but low levels were found in the gaseous phase as well, which might result from volatilization from solid wastes. Samples collected from pastureland near a known production site revealed concentrations in the vegetation from 0.01-630 ppm and in the soil from 0.01-300 ppb. ≤ 2,100 × Biomagnification

Hexachlorobenzene residues have been found in soil, wildlife, fish and food samples collected from all over the world. In the United States, residues have been reported in birds and bird eggs collected from Maine to Florida, duck tissues collected from across the country, and fish and fish eggs from the East Coast and Oregon. Animal foods, including chicken feed, fish food, and general laboratory feeds, have been found to contain residues. The frequency of detection of residues in domestic meats has been steadily increasing since 1972, in part because of closer scrutiny. The chemical has been detected in trace amounts in only two drinking water supplies.

EPA's monitoring of human adipose tissues collected from across the United States reveals that about 95 percent of the population has trace residues.

Technologic and Economic Implications

If a food tolerance is established by EPA at about .5 ppm (the interim tolerance), there is no reason to believe that substantial numbers of animals or crops will be held off the market. However, a level of .3 ppm or lower would probably prevent the marketing of some products. The feasibility and costs of air emission and water effluent controls, particularly the effectiveness of particulate reduction and of better housekeeping practices, have not been estimated. Effective incineration of wastes has been demonstrated. Proper landfill practice may serve this purpose; however, studies indicate that soil and other covers only delay volatilization.

Regulatory Actions in Progress

In the wake of widespread hexachlorobenzene contamination of cattle in Louisiana in 1973, and concern over possible contamination of sheep in California, EPA established an interim tolerance of .5 ppm. Concurrently, the State of Louisiana and several companies took immediate steps to tighten up solid waste disposal practices from manufacturing. Also, supplies of Dacthal containing 10 percent HCB as an inert ingredient were voluntarily withdrawn from the California market.

As soon as the needed toxicological data are available, a food tolerance will be established. Also, all pesticidal uses will be reviewed, including pesticides which contain hexachlorobenzene as a contaminant. Studies of land and other disposal methods have been completed. Ocean dumping of hexachlorobenzene -laden tars is prohibited. Although not directly addressed by the EPA permit program, provisions relating to suspended solids, and oil and grease may provide some degree of control if hexachlorobenzene enters the effluent stream.

LEAD AND ITS COMPOUNDS

Statement of Concern

The carcinogenicity of several lead salts has been demonstrated in laboratory animals; there is an absence of adequate human data. The potential for lead poisoning in sensitive populations is a major concern, particularly in children, pregnant mothers, and occupationally-exposed workers in urban environments where elevated levels of lead in air, dustfall, and soil represent significant additional sources of lead intake. A single episode of lead poisoning in a child may result in brain damage ranging from subtle learning disabilities to extensive mental handicaps.

Health and Ecological Effects, Environmental Behavior

Clinical effects of lead poisoning may include acute and chronic central nervous system damage, peripheral nerve paralysis, kidney damage, and damage to blood formation processes which may lead to anemia. The risk of clear-cut clinical effects is greatest when blood lead levels increase above 80 ug/100g whole blood, and at lower blood levels when anemia prevails. Mild poisoning may be reversible if exposure to lead is discontinued. Maternal over-exposure may have greater toxic effects on the fetus than on the mother.

Common sources of livestock lead poisoning include lead-based paints, storage batteries, used motor oil and airborne lead contamination of hay and pasture vegetation from smelting and other lead industries. A daily lead intake of 6-7 mg/kg body weight has been suggested as the minimum level which may result in poisoning in cattle. As much as two to three percent of the U.S. waterfowl population may die annually from lead poisoning, primarily due to injestion of spent lead shot.

Sources, Environmental Levels, and Population at Risk

U.S. consumption of lead was 1.6 million short tons in 1974. Major uses included storage batteries (52 percent), gasoline additives (16 percent) pigments (seven percent), ammunitions (five percent), solder (four percent), and cable covering (three percent). The elevated atmospheric levels of lead in larger cities was attributed primarily to motor vehicle combustion of leaded fuel additives. About 180,000 tons of lead are emitted to air annually from motor vehicle combustion of fuel additives. Other sources of lead pollution include coal and fuel oil combustion, lead smelting, incineration of solid wastes, and lead paints.

Lead levels in ambient air averaged 1-3 ug/m3 in urban areas, 0.1-0.5 ug/m3 in suburan areas, and less than 0.05 ug/m3 in rural areas, except in the vicinity of point sources and heavy traffic. The levels of lead in natural waters and finished drinking water rarely exceed 0.05 mg/l except in areas of lead ore depcsits and point source discharges.

The average daily oral intake of lead is estimated at 300 ug for adults, and 130 ug for children. Childhood and infant pica has resulted in numerous cases of lead poisoning from the ingestion of peeling lead paint and other lead-containing substances, and the inhalation of street dust. Illicitly distilled whiskey and consistent consumption of game animals previously wounded by lead bullets or shot are potentially hazardous dietary sources.

Occupational health supervision has limited the magnitude of industrial exposures. However, incidents of lead poisoning still occur where medical surveillance and exposure controls are inadequate; as recently seen in an auto battery plant in Visalia, California, and a pigments plant in Beltsville, Maryland.

Technologic and Economic Implications

Substitutes are readily available for lead chromate pigments in highway paints and for lead shot. The economic impact of banning lead chromate paint for highway markings would fall primarily on the producers of lead chromate. The economic impact of actions to promote recycle of lead batteries should be minimal, if any, since the rate of recycle is currently high, and the industry is willing to receive all used lead batteries it can get. EPA has specified standard methods for analysis of total lead in water, and is currently considering methods for air and other media.

Regulatory Actions in Progress

The Food and Drug Administration has restricted the use of lead in paints to less than 0.5 percent by weight for use on residential surfaces assessible to children, has proposed a tolerance level (0.3 ppm) for lead in evaporated milk and an action level (7ug/ml) for leachable lead in pottery and enamelware, and is considering limitations below 0.5 ppm for lead in other canned foods. OSHA has proposed an action level of 50 ug/m3 and a permissible exposure limit of 100 ug/m3 during an average eight hour work period for lead and its compounds. The Consumer Products Safety Commission is currently undertaking a review of the "safe" level of lead in paints.

EPA has established effluent limitation guidelines on lead discharges from several industrial point source categories. Fifteen lead compounds have been designated as hazardous substances. Special care is required in ocean dumping or other disposal of lead and its compounds. Interim Primary Drinking Water Standards set a maximum contaminant level for lead of 0.05 mg/l.

Pursuant to Section 111 of the Clean Air Act, EPA has established standards of performance for primary and secondary lead smelters. Lead has been added to the list of air pollutants under Section 108(a) of the Clean Air Act, and the issuance of a national ambient air quality standard for lead is planned for the near future.

Final regulations promulgated on December 6, 1973, control the amount of lead additives in gasoline. Lead levels in gasoline must meet the 1.4 g/gal limitation beginning October 1, 1976.

MERCURY

Statement of Concern

Despite recent action by EPA to limit mercury discharges during sludge incineration and through pesticidal use, and earlier Agency efforts to control air emissions and liquid effluent discharges, mercury continues to enter the environment. While more stringent enforcement of existing regulations should be helpful, discharges of mercury from fossil fuel plants, especially those that have shifted to coal from other less contaminated fuels, leaching of mercury from land-disposal sites, particularly into ground water, and urban runoff, are among the currently uncontrolled problems.

Health and Ecological Effects, Environmental Behavior

Mercury in many forms is highly toxic to man and other living things. In terms of toxicity, mercury and its compounds can be divided into three categories: 1) alkyl mercury compounds; 2) elemental mercury; 3) inorganic mercury salts and phenyl and methoxy ethyl compounds. Alkyl compounds, particularly methyl mercury, are the most toxic. Over 90 percent of ingested methyl mercury is absorbed in the gastrointestinal tract, and its whole-body biological half life is 70-90 days. Methyl mercury is transported in blood cells to, and concentrates in, brain and other central nervous system tissues where it can cause irreversible damage. In addition, it can cross the placental barrier and cause abnormalities in fetal tissues and irreversible damage to the fetus at levels that appear to cause no symptoms in the mother. Elemental mercury, phenyl and methoxy ethyl compounds, and inorganic mercury salts are far less dangerous than methyl mercury, because less are ingested and the rates of excretion are higher.

The Food and Drug Administration action level of 0.5 ppm of mercury for fish and shellfish, both raw and processed, is based on a 30 ug/day maximum intake of methyl mercury. This is one tenth of the 300 ug/day average intake resulting in a blood level of 0.2 ppb in adults, the lowest level at which neurological symptoms have been observed.

Mercury is readily transported to water by leaching from soil and fallout from air; most forms of mercury in soil and water can be biologically or chemically transformed to methyl mercury.

Sources, Environmental Levels, and Population at Risk

In 1973, United States use of mercury was slightly less than 1900 metric tons at an estimated cost of $3800 per metric ton. The chief uses were for battery manufacture (29.9 percent) and chlor-alkali production (24.1 percent). Use in 1965 had been approximately 2700 metric tons; the reduction of use resulted largely from a recognition of the hazards in use of the substance. In the period 1965 - 1973, several uses (particularly as preservatives and in gold recovery) were eliminated. Mercury is still used to make paints and industrial instruments.

NIOSH estimates that 150,000 workers are exposed to mercury. Because the vapor is colorless and odorless, overexposures can easily go unnoticed until symptoms appear. Of a total of 1900 metric tons used, it has been estimated that as much as 80 percent is discharged into the environment. Distribution of mercury discharges from man-related sources to the environment is about 31 percent to the air, 6 percent to water, and 36 percent to land. Concentrations in the various media are measured in terms of total mercury rather than the more hazardous methyl forms; thus, the data collected do not represent the true hazard.

Mercury is also a contaminant of coal, and may be a runoff problem from slag piles. In addition, landfills are a source of leaching mercury; this problem may be particularly severe in areas where drinking water supplies are drawn from ground water. Exposure to mercury is widespread, but inadequate documentation of levels of methyl mercury makes estimates of risk difficult.

Technologic and Economic Implications

Because most mercury losses occur during the use and disposal of products, recycling may provide the best method for reducing environmental discharges from batteries and instruments. Mercury emissions from the chlor-alkali industry would still be significant, even if state-of-the-art controls are applied to the production stream. Diaphram-cell technology could eliminate mercury emissions, but might add to problems associated with asbestos and lead. New developments in this technology are reducing use of lead and eliminating asbestos; thus increased future reliance on new without the added environmental burden. Diaphragm cells may offer the desired reduction in mercury emissions.

Regulatory Actions in Progress

FDA has proposed an action level for mercury in fish and shellfish. As a result of NIOSH recommendations, OSHA is considering revised workplace standards for inorganic and alkyl mercury.

EPA has set a hazardous air pollutant standard for mercury under Section 112 of the Clean Air Act, and is considering New Source Performance Standards to require zero emissions of mercury from new chlor-alkali plants.

EPA has addressed the problem through effluent guidelines for a few industrial categories and may expand this coverage in the future. The National Interim Primary Drinking Water Standard for mercury is 2 ppb. Ocean dumping is tightly controlled.

The EPA recently ordered an end to the registration of most pesticides containing mercury and particularly those used in paints, although the decision has been stayed pending completion of judicial review.

PHOSPHATES

Statement of Concern

Phosphates, a nutrient necessary to plant life, in excess quantities accelerate eutrophication of freshwater aquatic systems, particularly lakes-- both natural and artificial. Such an excessive nutrient loading creates problems ranging from discoloration and obnoxious odors to destruction of economically important freshwater fishes. Although effluent guidelines have been established for certain phosphate-producing activities, they have not been adequate to address eutrophication problems which may result from the non-essential use of phosphates, as example, as detergent builders or in fertilizers.

Health and Ecological Effects, Environmental Behavior.

Health effects on humans are limited to occasional outbreaks of gastroenteritis, when certain blue-green algae whose growth is promoted by phosphate loading are inadvertently ingested. The chief human impacts are on recreational and aesthetic values of lakes, value of lakeshore property, and costs of water treatment. Continued depression of the commercial fishing industry, particularly in Lake Erie, is another economic impact.

Eutrophication is a natural process of aging in lakes, normally taking place over millenia. Oligotrophic lakes receive little phosphorus nutrients, and thus do not support many forms of life. Over time, nutrients reach the lake from runoff, photosynthesis, and other sources in sufficient quantity to promote growth of a variety of plant and animal species. Over many centuries, the phosphate levels will increase because of continued accumulation from land runoff and other factors, and the living species present will change. Certain algal organisms may take over, form dense mats, die, and decompose, depleting oxygen supplies. Other plant and animal life also dies, decomposes, and further depletes the oxygen, causing further destruction of life. The lake may become murky, malodorous, and stagnant. It generally becomes swampy and ultimately is filled in.

The addition of phosphates from manmade sources at abnormally high levels (cultural eutrophication) accelerates the eutrophication process so that it can occur within only a few years. Artificial lakes, such as water supply reservoirs, appear to be susceptible to accelerated eutrophication.

Phosphates are water-soluble, adhere to particulate matter, and thus are transported in runoff. They are incorporated into plant tissues, and, as the plants die, phosphates tend to accumulate in the sediments. There is little evidence of soil transport of phosphates, except in sandy or gravelly soils.

Sources, Environmental Levels, and Populations Exposed.

Excess phosphates may reach lakes from land runoff, industrial or municipal sewage, and agricultural activities. These substances are used in fertilizers and as detergent builders, and are found in soft drinks, beer, animal feeds, and a variety of industrial wastes.

Levels of phosphate phosphorus in the Great Lakes range from 5 ug/l in the oligotrophic portions of Lake Superior to 61 ug/l in Lake Erie. Extensive sampling of lakes has been conducted within the EPA National Eutrophication Survey. Levels of phosphate phosphorus are of less concern, per se than the combination of factors which accelerate aging in the lake.

Because of the limited health effects which would be expected to result from the intake of lake water, it is more germane to discuss economic injuries as a result of eutrophication. Commercial fishermen, businesses or supply equipment for those activities are most severly affected by the effects of phosphate loading. Excess nutrient enrichment is a problem of varying intensity in most parts of the country and may continue to increase with population pressures.

Technologic and Economic Considerations.

Substitutes are available for detergent-builder applications; however, the relative cost and safety of those chemicals is not well documented. Some years ago, many of the detergent makers planned to substitute nitrilotriacetic acid for sodium tripolyphosphate (the most commonly used builder at the time,) and then learned of evidence suggesting that nitrilotriacetic acid is a carcinogen. More recently, the National Cancer Institute, the International Joint Commission, and Canadian agencies have undertaken health and environmental studies of the effects of release of phosphates. Results are expected within the next few months.

Tertiary treatment technology can remove phosphate phosphorus from municipal and industrial waste streams. These systems are expensive, and create potential disposal problems with spent activated carbon columns and other filters. Some secondary treatment methods commonly in use may provide adequate removal of phosphorus from certain waste streams.

Treatment of drinking water taken from highly eutrophic lakes is a difficult and expensive undertaking, although the technology does exist. Such treatment reduces taste, odor, and color problems, but does not resolve the excess phosphate issue.

Phosphate phosphorus is an essential ingredient in fertilizer, no known substitute will achieve the desired purpose.

Regulatory Actions in Progress

Effluent guidelines for runoff from feedlots, fertilizer manufacturing, and phosphate manufacturing have been issued incorporating water quality criteria. These guidelines govern permits issued to persons engaged in these businesses, but the criteria are not designed to address accelerated eutrophication.

EPA's Regional office and the International Joint Commission have recommended that States abutting the Great Lakes adopt a discharge level of 1 mg/l of phosphate phosphorus in effluents which enter the lake.

Four States have banned the use of phosphate detergents (Michigan, Minnesota, New York, and Indiana). Indiana has reported some success in reversing eutrophication damages in its lakes. EPA has urged the Great Lakes States to adopt similar bans. Three Great Lakes cities also have adopted bans on phosphate detergents.

EPA is studying phosphates to determine if regulations including a ban on certain uses are needed either nationally or on a regional/conditional basis. Source and impact analyses are in progress.

POLYBROMINATED BIPHENYLS

Statement of Concern

In 1973, one to two tons of polybrominated biphenyls, a highly toxic flame retardant, were accidentally mixed into an animal feed supplement and fed to cattle in Michigan. Contamination also resulted from traces of polybrominated biphenyls being discharged into the environment at the manufacturing site and at other facilities involved in handling polybrominated biphenyls. Dairy and cattle farms were quarantined, tens of thousands of swine and cattle and more than one million chickens have been destroyed, and litigation involving hundreds of millions of dollars has been instituted. Before the nature of the contamination was recognized, many of the contaminated animals had been slaughtered, marketed, and eaten, and eggs and milk of the contaminated animals were consumed. Thus, large numbers of people were exposed to polybrominated biphenyls. Commercial manufacture for polybrominated biphenyls distribution in the U. S. has been discontinued, but manufacture for export continues.

Health and Ecological Effects, Environmental Behavior

Among the 10,000 people who have been identified as having consumed polybrominated biphenyls, contaminated meat, milk products, poultry, and eggs, no overt symptoms have been reported to date. Health effects can only be extrapolated from animal data. Based on experimental data, polybrominated biphenyls may be much more toxic than polychlorinated biphenyls.

Short-term rat, mice, and cattle studies have shown that polybrominated biphenyls may interfere with reproduction and liver functions, promote nervous disorders, and react as a teratogenic agent in tissues. Polybrominated biphenyls have produced pathological changes in the livers of rats, mice, guinea pigs, cows, and rabbits. In an experiment with guinea pigs, the chemical was demonstrated to be an immuno-suppressant agent. About 400 cows given contaminated feed for about 16 days exhibited anorexia, decreased milk production, increased frequency of urination, some lameness, abnormal hoof growth, and shrinking of the udder. Later signs of toxic effects included bloody blebs, malformed or dead fetuses, abscesses, weight loss, and high susceptibility to stress. Non-lactating cows died within six months while the lactating animals survived and gradually improved. Massive liver abscesses were found in dead animals.

Studies at the University of Michigan medical school have shown profound adverse effects on the kidney, liver, and thyroid in rats and mice. Neoplastic liver nodules appeared in rats.

Fish taken from streams known to have been contaminated by polybrominated biphenyls have demonstrated that the chemical can bioaccumulate to 20,000 to 30,000 times the ambient levels. Polybrominated biphenyls are believed to be quite persistent in the environment. Polybrominated biphenyls readily vaporize.

Sources, Environmental Levels, and Population at Risk

Polybrominated biphenyls have been used commercially as flame retardant additives in synthetic fibers and molded thermoplastic parts. Polybrominated biphenyls have been incorporated into the plastic housings of many commercial products, such as typewriters, calculators, and microfilm readers, and consumer products, such as radio and television parts, thermostats, shavers, and hand tools.

Michigan Chemical Corporation produced approximately 11 million pounds of polybrominated biphenyls from 1970 to 1974. The White Chemical Corporation produced approximately 100,000 pounds of the closely related compounds, octabromobiphenyl and decabromobiphenyl, from 1970 through 1973. In addition, nine companies have been suppliers of laboratory quantities of polybrominated biphenyls, each producing about five pounds per year. There is no indication of importation of the material. Two plants in New Jersey produced about one million pounds for export in 1976.

Monitoring in the Pine River near the facility where polybrominated biphenyls were produced indicated that levels diminished from 3.2 ppb in the ambient stream near the effluent discharge to .01 ppb eight miles downstream. Fish obtained in this eight-mile stretch had levels of .09 to 1.33 ppm. In the New Jersey samples a level of 100 ppm was found in a sediment sample, 10 to 60 ppb in receiving water, and 1 to 2.7 ppm in the soils near the two producing plants. Land-based plant samples showed 0.3 to 10 ppm and aquatic specimens had 0.3 to 1 ppm. Three of nine human hair samples contained .03, 1, and 2 ppm respectively.

Detailed data are available on levels found in cattle and hogs, with the highest level detected being 2.27 ppm. Data are not yet available on the levels found in the 10,000 or more exposed persons.

Technologic and Economic Implications

Michigan Chemical Corporation reportedly had paid $20 million in settling a $270 million suit, with claims of $500 million still outstanding. However, the financial dimensions of the incident are still not known.

Among the substitutes for polybrominated biphenyls are the more expensive decabromobiphenyl oxide and several halogenated aliphatic compounds. However, the environmental acceptability of these compounds has not been assessed.

Monitoring methods have been developed and are being standardized.

Regulatory Actions in Progress

The State of Michigan has been the focal point for responding to the contamination incident. In addition, the Department of Agriculture, the Food and Drug Administration, the National Cancer Institute, the Communicable Disease Center,

EPA, the Michigan State University, and the University of Michigan have conducted a wide array of epidemiological, toxicological, analytical, and related projects to clarify the effects of the chemical on humans and animals and to assess the extent of contamination. The Toxicology Coordinating Committee of the Department of Health, Education and Welfare is preparing a synthesis of available health effects information. EPA provides assistance in environmental monitoring.

The Food and Drug Administration has set temporary action levels for polybrominated biphenyls in contaminated foods and in animal feed. The State of Michigan has issued warnings to sport fishermen along the Pine River.

EPA's studies to document the full scope of the hazard and determine the appropriateness of regulations under TSCA and other statutes are continuing.

POLYNUCLEAR AROMATIC HYDROCARBONS

Statement of Concern

Increased exposure to polynuclear aromatic hydrocarbons and other air pollutants has been implicated by some researchers in increased rates of cancer, especially of the lung. Over 30 polynuclear aromatic hydrocarbons have been identified as urban air pollutants, including several carcinogens. These chemicals are emitted during fossil fuel combustion, in natural combustion processes, and as a result of a variety of human activities. They have been found at low levels in liquid effluents, some drinking water supplies, and food.

Health and Ecological Effects, Environmental Behavior

Certain polynuclear aromatic hydrocarbons which have been demonstrated as carcinogenic in test animals at relatively high exposure levels are being found in urban air at very low levels. Various environmental fate tests suggest that they are photo-oxidized, and react with oxidants and oxides of sulfur. Because the chemicals are adsorbed on particulate matter, chemical half-lives may vary greatly, from a matter of a few hours to several days. One researcher reports that photo-oxidized fractions of air extracts also appear to be carcinogenic. Environmental behavior/fate data have not been developed for the class as a whole.

It has been observed that polynuclear aromatic hydrocarbons are highly soluble in adipose tissue and lipids. Most of the compounds taken in by mammals are oxidized and the metabolites excreted. Effects of that portion remaining in the body at low levels have not been documented.

Benzo(a)pyrene, one of the most commonly found and hazardous of the polynuclear aromatic hydrocarbons has been the subject of a variety of toxicological tests, which have been summarized by the International Agency for Research on Cancer. As little as 50-100 ppm administered in the diet for 122-197 days produced stomach tumors in 70 percent of the mice studied. A dose level of 250 ppm produced tumors in the forestomach of 100 percent of the mice after 30 days. A single oral administration of 100 mg to nine rats produced mammary tumors in eight of them. Skin cancers have been induced in a variety of animals at very low levels, and using a variety of solvents (length of application was not specified). Lung cancer developed in 2 of 21 rats exposed to 10 mg/m^3 benzo(a) pyrene and 3.5 ppm sulfur dioxide for 1 hour per/day, five days a week, for more than one year. Five of 21 rats receiving 10 ppm sulfur dioxide for 6 hr/day, in addition to the foregoing dosage, developed similar carcinomas. No carcinomas were noted in rats receiving only sulfur dioxide. No animals were exposed only to benzo(a) pyrene. Transplacental migration of benzo(a) pyrene has been demonstrated in mice. Most other polynuclear aromatic hydrocarbons compounds have not been subjected to testing.

Sources, Environmental Level, and Population at Risk

Several polynuclear aromatic hydrocarbons can be formed in any hydrocarbon combustion process and may be released from oil spills. The less efficient the combustion process, the higher the emission factor is likely to be. The major sources are stationary sources, such as heat and power generation, refuse burning, industrial activity, such as coke ovens, and coal refuse heaps. While polynuclear aromatic hydrocarbons can be formed naturally (lightning-ignited forest fires), impact of these sources appears to be minimal. It should be noted, however, that transportation sources account for only about one percent of production of these chemicals.

Diesel powered vehicles produce more particulate emissions than gasoline powered; the nature of the fuel is such that the emissions would be expected to contain greater amounts of polynuclear aromatic hydrocarbons, and limited studies have confirmed this. EPA has tested gasoline-powered passenger vehicles to determine the amount of the chemical in the exhaust. However, this characterization is of particulates; little is known of vaporous components.

Polynuclear aromatic hydrocarbons have been detected in urban water supplies at low levels. In water and soils they are adsorbed on minerals or organic particulate matter; algae and invertebrates contain concentrations as high as 200 times those of the surrounding waters. Levels detected in plants, on the other hand, are slightly lower than soil levels. Sludge samples taken near a steel refinery showed combined benzo(e)- and benzo(a)pyrene levels of 0.91-19.0 mg/kg (dried weight). Liquid effluents did not appear to contain these substances.

Although a variety of these compounds have been observed in particulates from urban air samples, these are not routinely monitored. Atmospheric concentrations are generally represented by measurements of benzo(a)pyrene concentrations. In heavily industrialized areas, benzo(a)pyrene levels have been as high as 20 nanograms (ng)/m3. Urban levels are generally 2-7 ng/m3; and rural, 0.3 ng/3. In 1971-73, nationwide annual emissions of benzo(a)pyrene were estimated at 900 tons. It has been estimated that benzo(a)pyrene represents 2-5 percent of the total polynuclear aromatic hydrocarbons emitted from automobiles; a similar and as yet undetermined relationship may exist for stationary source emissions.

Because of the large number of sources, most people are exposed to very low levels of the chemical. Benzo(a)pyrene has been detected in a variety of foods throughout the world. A possible source is mineral oils and petroleum waxes used in food containers and as release agents for food containers. Studies of the Food and Drug Administration have indicated that no health hazard exists from these sources.

Technologic and Economic Implications

Good particulate emission controls can substantially reduce polynuclear aromatic hydrocarbons emissions. However, the costs that would be incurred in further limiting polynuclear aromatic hydrocarbons emission from stationary and vehicular combustion sources are not known. The application of oxidation catalyst exhaust treatment has been effective in dramatically reducing emissions from automobiles when such systems are operating properly. Similar controls at stationary sources may have a similar effect.

Regulatory Actions in Progress

Limitation of carbon monoxide and hydrocarbon emissions from motor vehicles have simultaneously and dramatically reduced the emissions. A 1974 analysis of stationary source problems concluded that control regulations designed specifically for benzo(a)pyrene or polynuclear aromatic hydrocarbons were not warranted or practical, but noted that compliance with existing regulations for incinerators, open burning, coal combustion, and coking operations could significantly reduce emissions. Additional efforts to document stationary source emissions, atmospheric chemistry, and human exposure have been initiated on a limited scale. Consideration is being given to establishing revised standards for coke oven emissions under the Clean Air Act. EPA's Scientific and Technical Assessment Report document and a National Academy of Sciences report have detailed much of the hazard and technologic aspects of this class of compounds.

TRICHLOROETHYLENE

Statement of Concern

Trichloroethylene has been identified by the National Cancer Institute as a carcinogen in laboratory animals. It is widely used for degreasing of fabricated metals and, to a lesser extent, in cleaning fluids. In addition to extensive worker exposure, it has been detected in ambient air and water in industrial areas, in food, and in human tissues.

Health and Ecological Effects, Environmental Behavior

Trichloroethylene induces tumors in mice at high dose levels, predominantly liver cancer with some metastases (transfer) to the lungs. It is absorbed rapidly by the lungs; only a small amount is eliminated by exhalation, 58-70% being retained. This is slowly eliminated in the urine as trichloroacetic acid or trichloroethanol. The first major review of trichloroethylene poisoning studied 284 cases, including 26 fatalities, in European plants where vapors were inhaled. Results indicated that toxic action involves the central nervous system. A number of short-term studies indicate that exposure to a concentration of 100 ppm in air may interfere with psychophysiological efficiency. In one study, six students exposed to 110 ppm for two four-hour periods separated by 1-1/2 hours showed significantly lower levels of performance in perception, memory, and manual dexterity tests. A confirmatory test using six workers in a plant using trichloroethylene produced almost identical results. There is a reported case of a man operating a metal degreaser who lost his sense of taste after one month's exposure to trichloroethylene concentrations which occasionally escaped in visible quantities. Two months later the same man lost facial mobility and sensation, and developed electroencephalogram recorded changes which did not clear up during the following two years.

Several incidents of workers being overcome by fumes have been recorded. Also, people who sniffed trichloroethylene have been reported as dying when exerting themselves shortly thereafter because it acts as a cardiac sensitizer. As an example of the hazards of concern, in April 1977, a worker at at a General Motors plant was killed while attempting to rescue a coworker who had been overcome by methyl chloroform fumes while removing residues from a cleaning operation. Fifteen workers were hospitalized and have since recovered.

Trichloroethylene frequently has been detected in the environment; however, its behavior and transport have not been documented. Adverse ecological effects have not been reported.

Because of its low solubility, high vapor pressure, and high photodegradation rate at sea level (half-life in air is about eight hours), trichloroethylene is not expected to accumulate in the atmosphere. Its half-life in water is on the order of months.

Sources, Environmental Levels, and Population at Risk:
Domestic production in 1974 was about 215,000 tons by five producers. Over 90% is used for vapor degreasing of fabricated metals. Ambient concentrations in the atmosphere of industrialized areas have been estimated by industry to be 2-16 ppt. Water concentrations are about 0.1 ppt. The character of the water was not defined, but trace amounts have been identified by EPA in drinking water. There have been several reports of contamination of wells and groundwaters from careless disposal practices and accidents.

Over 20,000 workers are exposed to trichloroethylene. The general public may be exposed via inhalation of cleaning fluids and ingestion of foods, spices, and medicines from which undesirable components have been removed by trichloroethylene extraction. In foreign studies, residues ranging from 0.02 to 22 ppt have been detected in foods and concentrations of up to 32 ppt have been detected in human tissues. FDA is preparing to determine if the compound can be detected in their food monitoring programs.

Technologic and Economic Implications

Cold metal cleaning, vapor metal degreasing, and dry cleaning operations present different control problems. Both methyl chloroform and perchloroethylene are being considered as possible substitutes for trichloroethylene as degreasing agents. Both appear less damaging to air quality, however, methyl chloroform may adversely impact the ozone layer. All three are comparably priced and, since many users have already made this change, the economic impact should be minor. Closed loop systems could permit recovery; however, this may represent a higher cost factor than the use of substitutes. Preliminary studies of the National Cancer Institute indicate that perchloroethylene may also present health hazards. Further studies are needed to determine if the substitutes are acceptable.

Regulatory Actions in Progress

In October 1975, OSHA proposed a reduction in the workplace standard, and is reviewing the proposal, together with possible standards for methyl chloroform and perchloroethylene. NIOSH is preparing a hazard review to revise the 1973 criteria document.

Trichloroethylene producers have conducted epidemiological studies, long-term animal feeding studies, and long-term animal inhalation studies, and an in-depth literature survey has been completed for the Manufacturing Chemists Association.

Since trichloroethylene contributes to photochemical smog, State Implementation Plans provide a mechanism for limiting emissions. Detailed health, environmental, and economic analyses are being done as a basis for determining the necessity for revisions to these plans. Discharge permits limiting bacterial oxygen demand, chemical oxygen demand, and suspended solids also provide some control over effluent discharges.

The National Cancer Institute will begin testing trichloroethylene in an oral exposure study, using their revised protocol. Doses are based on levels resulting in 10 percent weight loss, rather than lethal levels.

EPA is conducting a detailed survey to determine if regulations are warranted. The report is designed to collect the data needed to justify regulations using TSCA and other authorities, if appropriate.