CRITICAL CONDITION

Human Health and the Environment

a report by Physicians for Social Responsibility

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Food Contamination due to Environmental Pollution

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Human life is sustained by an environment that provides adequate food derived from plants, minerals, and animals. Whereas the spoilage of food and its contamination by infectious agents and their toxins have long been of concern in the field of public health, contamination by environmental pollution has been less well recognized. We will concentrate on the latter, and we will not discuss hazards related to food processing (such as food irradiation, food additives, cooking, and preservation techniques) or to natural food toxins.

Environmental contamination of food can occur through multiple pathways on land, by air, and in fresh and salt water. The polluting agents of most significance include pesticides, radionuclides, halogenated cyclic compounds, and heavy metals.

A great deal of overlap exists between contamination of food and contamination of drinking water (see the preceding chapter) with respect to the toxins involved and the sources of pollution. In particular, aquatic animals serve as important contributors to the nutritional protein, lipid, and vitamin requirements of humans, and serve to cycle water-borne anthropogenic toxic chemicals back to human consumers in the form of food.[1]

In general, one the main differences between water contamination and food contamination is the tendency of plants and animals in the food chain to concentrate certain toxins, thereby increasing the exposure of unwary consumers. For instance, radioactive strontium concen-

:rates in milk, and mercury (as the organic compound methyl mercury) :oncentrates in the tissues of fish.

The toxicity of contaminants in food can be compounded by malnutrition. For example, children who are deficient in iron, calcium, phosphorus, protein, or zinc absorb more lead than do well-nourished children with identical environmental lead exposures.[2] And malnutrition weakens the immune system, thus making an affected person more vulnerable to infectious pathogens and possibly to chemical agents.

Exposure to environmental food contamination may not be borne equally. In the United States, approximately three-fourths of the toxic waste disposal sites that failed to comply with the regulations of the U.S. Environmental Protection Agency were located in impoverished communities of people of color,[3] placing them at greater risk of food and water contamination. These are also the individuals who are at greatest risk for malnutrition and occupational exposures to pesticides, toxic metals, and other hazardous substances.

Responsibility for monitoring and control of contaminants in food is shared by a number of agencies. In the United States, the Food and Drug Administration (FDA) monitors dietary intake of selected contaminants; the Food Safety Inspection Service of the Department of Agriculture monitors residues in meat and poultry; and the Environmental Protection Agency's National Human Monitoring Program estimates total body exposure to toxic substances, including pesticides. Elsewhere, a growing number of countries are participating in the Global Environment Monitoring System, a program of food monitoring supported by the World Health Organization and the United Nations. By 1988, 35 countries participated, representing countries in every continent.[63]

Pesticides

Pesticides are used in agriculture in all parts of the world. While most cases of acute, high-exposure pesticide poisoning are related to occupational exposure to the applicators themselves (there are more than 200,000 deaths worldwide each year, mainly in this population, from acute pesticide poisoning[4]), significant exposure can occur through ingestion of treated food. At least 37 epidemics directly due to pesticide contamination of food have been reported.[5]

The term *pesticides* includes insecticides, herbicides, rodenticides, food preservatives, and plant growth regulators. We will concentrate on chemical insecticides. Chemical insecticides include synthetic organic insecticides and inorganic chemicals (mostly metals, such as arsenic). Other insecticides, such as those from biological sources—nicotine, pyrethrin, phermones, and insect-specific bacteria and viruses—will not be considered in this chapter. Synthetic organic insecticides can be further broken down into the chlorobenzene derivatives (e.g., dichlorodiphenyltrichloroethane (DDT)), cyclodienes (chlordane, aldrin, dieldrin), benzenehexachlorides (lindane), carbamates, and organophosphates (malathion).

While the mechanism of action differs among different classes of agents, most chemical pesticides are designed to be acutely toxic to their target organism. At high levels of exposure, they are also acutely toxic to humans, usually causing general symptoms of poisoning (nausea, vomiting, malaise, headache) as well as neurological symptoms (excitability, tremors, convulsions). Pesticide applicators are most at risk for high levels of exposure.

Pesticide contamination of food is mostly of concern because, while exposures are at lower levels, they involve much larger segments of the population (all consumers). In addition, many pesticides concentrate in the food chain and can accumulate in human tissue, where their slow metabolism and solubility in adipose (fat) tissue can lead to lifelong storage. Organochlorine pesticides have been found throughout the food chain, even in zooplankton and fish in the Arctic Ocean.[6] One recent study in Asia found these same pesticides at particularly high levels in preserved fruits, eggs, and fish.[7] Another study in Africa found the presence of chlorinated pesticides in over 80% of samples of eggs, poultry liver, and bovine liver and kidney; 7.5% of samples had levels higher than international tolerance levels.[8] In the United States, the commercial milk supply in Hawaii was contaminated by heptachlor epoxide during 1981 and 1982.[9] Isomers of dioxin have been found in crustaceans and finfish off the east coast, probably as the result of a combination of municipal and industrial combustion processes.[10]

Lindane has been detected in the blood and adipose tissue of the general public in a number of countries, probably because of food contamination. In autopsy surveys conducted in the 1970s, lindane and other benzenehexachlorides were present in more than 90% of human adipose tissue samples at a level of around 300 ppb.[11,12] Benzenehexachlorides were also found to be present in 82% of human breast milk samples at a mean concentration of 81 ppb with a range of 0–480 ppb.[13]

With respect to low-level exposure to humans, the toxic outcomes of greatest concern are cancer, immunotoxicity, and reproductive effects. This is an area of great concern, given the potential for population-wide exposures to a wide variety of pesticide residues, and great dispute, given the lack of toxicity and epidemiological data on most of these substances.

Most of the data on pesticides derive from in vitro assays that test the potential of a chemical to alter the genetic material of bacteria, and from studies on rodents. In the United States, these tests are now being coordinated by the National Toxicology Program. *In vitro* assays, however, cannot reliably be related to humans. Animal studies typically test high doses of a chemical. Statistical methods for extrapolating the risk of low-dose exposure from high-dose tests vary widely, give widely differing results, and are another subject of debate.

Nevertheless, the Delaney Clause of the Federal Food, Drug and Cosmetics Act has mandated both the EPA and the FDA to specially target food additives, including pesticides, that have been found to induce cancer when ingested by either animals or humans. This complicated legislation sets out specific criteria for the designation of contaminants in foods as potential carcinogens; once designated, potential carcinogens are then banned from foods sold in interstate commerce.[64] Lending support to the Delaney clause is a new study which has shown a strong association between breast cancer in women and elevated levels of serum DDE, the main metabolite of DDT.[65]

The current status of several major pesticides and the complicated interplay between evidence of carcinogenicity, scientific standards of proof, and regulatory standards can be seen in table 1.

The approach developed by regulatory agencies in the United States has not been without controversy. In the debate on the setting of

Table 1 Evidence for pesticide carcinogenicity. Adapted, with permission, from Council on Scientific Affairs. "Cancer Risk of Pesticides in Agricultural Workers," *Journal of the American Medical Association* 260 (1988), no. 7: 959–966.

	Evidence				
Compound	Animal	Human	*In vitro*	IARC*	EPA†
Aldrin	Limited	Inadequate	Inadequate	3	C
Amitrole	Sufficient	Inadequate	Inadequate	2B	B2
	Sufficient	Inadequate	Inadequate	3	C
α-Naphthyithiourea	Inadequate	Inadequate	. . .	3	C
Aramite	Sufficient	. . .	. . .	. . .	. . .
Arsenicals	Inadequate	Sufficient	Limited	1	A
Benzal chloride	Limited	Inadequate	Limited	3	C
Benzotrichloride	Sufficient	Inadequate	Limited	2B	B2
Benzoyl chloride	Inadequate	Inadequate	Inadequate	3	C
Benzyl chloride	Limited	Inadequate	Sufficient	3	C
Captan	Limited	Insufficient	. . .	. . .	. . .
Carbon tetrachloride	Sufficient	Inadequate	Inadequate	2B	B2
Chlordane	Limited	Inadequate	Inadequate	3	C
Chlordimeform (metabolite)	No data	Insufficient	. . .	. . .	. . .
	Sufficient	. . .	. . .	. . .	. . .
Chlorobenzilate	Limited	Insufficient	. . .	. . .	. . .
Chlorophenols	. . .	Limited	. . .	2B	B2
Chlorothalonil	Limited	Insufficient	. . .	. . .	. . .
Diallate	Limited	Insufficient	. . .	. . .	. . .
1,3-Dibromochloropropane	Sufficient	. . .	. . .	. . .	. . .
p-Dichlorobenzene	Sufficient	No data	. . .	2B	B2
2-Dichloroethane	Sufficient	. . .	. . .	. . .	. . .
2,4-Dichlorophenoxyacetic acid esters	Inadequate	Inadequate	Inadequate	3	C
p,p'-Dichlorodiphenyltrichloroethane	Sufficient	Inadequate	Inadequate	2B	B2
Dicofol (Kelthane)	Limited	Insufficient	. . .	. . .	. . .
Dieldrin	Limited	Inadequate	Inadequate	3	C
Ethylene dibromide	Sufficient	Inadequate	Sufficient	2B	B2

Table 1 (continued)

Compound	Evidence: Animal	Human	*In vitro*	IARC*	EPA†
Ethylene oxide	Limited Sufficient	Inadequate Inadequate	Sufficient ...	2B ...	B2 ...
Ethylene thiourea	Sufficient	Inadequate	Limited	2B	B2
Fluometuron	Inadequate	No evaluation	...	...	...
Formaldehyde	Sufficient	Inadequate	Sufficient	2B	B2
Heptachlor	Limited	Inadequate	Inadequate	3	C
Hexachlorobenzene	Sufficient	...	...	...	...
Kepone (chlordecone)	Sufficient	...	...	...	...
Lindane (γ-hexachlorocyclohexane)	Limited	Inadequate	Inadequate	3	C
Malathion	No evidence	No data	...	...	...
(4-chloro-2-methylphenoxy) acetic acid	Inadequate	Inadequate	...	3	C
Methyl parathion	No evidence	No evidence	...	3	C
Mirex	Sufficient	...	...	...	...
Nitrofen	Sufficient	No data	...	...	...
Parathion	Inadequate	Insufficient	...	...	...
Pentachlorophenol	Inadequate	Inadequate	Inadequate	3	C
Phenoxy acids	...	Limited	...	2B	B2
o-Phenylphenol	Limited	Insufficient	...	...	...
Piperonyl butoxide	No evidence	No evidence	...	...	...
Sulfallate	Sufficient	No data	...	...	...
2,3,7,8-Tetrachlorodibenzo-*p*-dioxin	Sufficient	Inadequate	Inadequate	2B	B2
Tetrachlorovinphos	Limited	Insufficient	...	...	...
Thiourea	Sufficient	...	...	...	...
Toxaphene	Sufficient	...	...	...	...
Trichlorfon	Inadequate	Insufficient	...	...	...

Table 1 (continued)

Compound	Evidence: Animal	Human	*In vitro*	IARC*	EPA†
2,4,5-Trichlorophenol	Inadequate	Inadequate	No data	3	C
2,4,6-Trichlorophenol	Sufficient	Inadequate	No data	2B	B2
2,4,5-trichlorophenoxyacetic acid	Inadequate	Inadequate	Inadequate	3	C
Vinyl chloride	Sufficient	Sufficient	Sufficient	1	A

*IARC indicates International Agency for Research on Cancer. Evidence is divided into the following categories: 1, evidence is sufficient to establish a causal relationship between the agent and human cancer; 2, agent, or process, is probably carcinogenic to humans; 2A, limited, almost sufficient evidence for carcinogenicity in humans; 2B, combination of sufficient evidence in animals and inadequate human data; and 3, cannot be classified according to carcinogenicity in humans.

†EPA indicates Environmental Protection Agency. Evidence is divided into the following groups: A, carcinogenic to humans (epidemiologic evidence supports a causal relationship); B, probably carcinogenic to humans (B1, epidemiologic evidence is limited or the weight of evidence from animal studies is sufficient or B2, evidence is sufficient from animal studies but epidemiologic studies provide inadequate evidence or no data); and C, possibly carcinogenic to humans (limited evidence from animal studies and no human data).

standards and allowable risks, some have argued that the risks posed by man-made pesticides are dwarfed by those posed by "natural" carcinogens, such as aflatoxin, ochratoxin A, and other mycotoxins.[14,15] Clearly, the relative impact of naturally occurring carcinogens needs better assessment, and attempts are underway in several countries to regulate the levels of these substances in food.[16] Nevertheless, the consensus among most public health authorities has been that it is critically important to continue to tighten the regulation of man-made pesticides, even while scientific research continues.[17]

But pesticides that are banned in the United States specifically because of their toxicity are still being exported. According to a 1987 United Nations report,[18] the U.S. EPA identified a significant risk for cancer for 18 of the 72 most commonly used pesticides. Of these 72 pesticides, 20 have been banned, withdrawn, or severely restricted in at least one country. Many are still produced and exported by U.S. companies, however.

Aggressive marketing by producers of pesticides is at least partially responsible for their indiscriminate use in the developing world. Dangers inherent in the use of pesticides are often minimized. Alternatives are not encouraged. Conditions in the developing world, including illiteracy, lack of personal protective equipment, and inadequate sanitation, increase the risk of both occupational and food-borne pesticide exposure to workers, their children, and entire communities.[14]

Only recently has there been a movement away from indiscriminate use of pesticides and toward a philosophy of "integrated pest management." Numerous studies have demonstrated clear health advantages to discontinuing current methods of intensive pesticide use in favor of "sustainable farming" (natural pest control, crop rotation, organic fertilizers, etc.).[19]

Radioactive Fallout

Decades of testing, operations, and accidents involving nuclear weapons production and nuclear energy power plants have resulted in contamination of plants and animals important to the food chain. Exposure to food is of concern because of the potential for internal irradiation and the permanent incorporation of radioactive material in the molecules of cells. Many radionuclides are involved. Of most significance with regard to levels of exposure, half-life, and toxicity are strontium 90, cesium 137, zirconium 95, carbon 14, plutonium 239, and iodine 131. Milk and other livestock products are particularly at risk.

Radionuclides accumulate in plants and animals according to their biological significance or their chemical similarity to biologically active elements in the same chemical groups.[20] Kinetic models of radioactivity transfer suggest that the dose from fallout is most sensitive to levels of deposition and direct transfer from contaminated feed and pastures to milk or meat.[21] The consumption of contaminated marine and freshwater fish is also of concern.[22]

Radioactive fallout that has already occurred, principally from atmospheric testing of nuclear weapons, is expected to cause at least 430,000 cancer deaths by the end of the 20th century. Because of the long half-lives of many fallout radionuclides, the total toll in premature human deaths has been estimated at more than 2 million.[23]

Polyhalogenated Aromatic Hydrocarbons

Much publicity has been generated on the subject of environmental contamination by polychlorinated biphenyls (PCBs). In the 1970s these synthetic compounds, once widely used in electrical transformers, lubricants, sealants, television sets, and other household products, were found to be potent animal carcinogens and to concentrate in the food chain (particularly in food fish). They were phased out and banned in the United States.

Some epidemiological studies of populations occupationally exposed to PCBs have reported excesses of liver and biliary tree cancers[24] and hematologic neoplasms[25]; however, other studies have been negative. A review of epidemiological studies of humans exposed to PCB-contaminated fish from the Great Lakes suggested a consistent relationship between PCB exposure and indicators of impaired neonatal and early infant health.[26]

Despite the banning of PCBs, and despite a few positive epidemiological studies, concern remains. The U.S. National Human Adipose Tissue Survey found that levels of PCBs above 3 ppm in the general population have been decreasing; however, the prevalence of any detectable level has been increasing.[27] Many bodies of water, such as the Great Lakes, remain heavily contaminated by PCBs.

Other polyhalogenated aromatic hydrocarbons of concern include the polybrominated biphenyls (PBBs) and the chlorinated benzenes (the most common chlorobenzene compound is hexachlorobenzene), both of which become concentrated in the adipose tissues of animals and humans. However, except in Michigan (where PBBs were accidentally mixed into livestock feed), widespread contamination by PBBs has not been a problem. Very little is known about the toxicity of PBBs, although anecdotal cases linking them to leukemia and other hematologic disorders have been reported.[28]

Heavy Metals

Lead

The contamination of food with lead is of major concern because of the high levels of exposure experienced around the world and because

of recent studies linking neurobehavioral toxicity to relatively minute quantities of lead in human tissues.

Lead is a frequent contaminant of water, which may be used to irrigate crops or to process food in factories or homes. Lead in glazed ceramic ware or in crystal glassware can leach out, particularly if the food or drink contained is acidic. (Glazed ceramic ware has been a particularly high contributor to population-wide lead exposure in Mexico.[29]) The combustion of leaded gasoline (which went on for decades) has led to fallout of lead oxide in dust, which can contaminate the soil used to grow crops and the feed of livestock and which also can contaminate foods directly. Home gardens can be contaminated by peeling chips or by rain-washed runoff from lead paint, a common interior and exterior coating for dwellings built before 1955. Up to 50% of U.S. housing has lead paint on exposed surfaces.[30] Green leafy vegetables, in particular, concentrate lead. The U.S. Agency for Toxic Substances and Disease Registry (ATSDR) estimates that a million U.S. children are exposed to enough lead in food to cause lead poisoning.[37]

In 1980, half of the food and beverage cans produced in the United States were lead-soldered. Now few domestic products are, but food and drink cans manufactured outside the United States typically continue to contain lead solder, which can leach into food, and many such products are imported and consumed. Lead compounds are also used deliberately in substantial quantities in a variety of traditional "folk" medicines and cosmetics. Such toxic exposures have been reported among residents of China, India, the Middle East, and South America, and among immigrants from those regions.[19]

Lead exposure through food also occurs occupationally. The National Institute for Occupational Safety and Health (NIOSH) estimates that more than a million U.S. workers in more than 100 different occupations are exposed to lead on the job. At the workplace, airborne lead dust settles on hands, food, water, clothing, and other objects, and can be inhaled, ingested, or carried home.[31]

Ingestion of leaded paint chips and lead-contaminated dust is the major route of exposure for U.S. children. While everyone inhales or ingests small amounts of dust, smoke, or soil (on the order of 10 milligrams per day), young children may ingest as much as 200 mg per day,[32] including lead paint dust and lead-contaminated soil, particularly in cities or near highways. While the United States has removed lead

from most gasoline, in many countries leaded gas and its airborne and soil contaminants are the dominant source of lead exposure, especially for children.

As figure 1 illustrates, lead interferes with many functions and structures in humans. Of most concern has been epidemiologic evidence demonstrating a link between lead exposure and adverse affects on the indices of intelligence and neurobehavioral development in children.[34,35] Even modest amounts seem to exert effects. Recently, the U.S. Centers for Disease Control (CDC) set the maximal level recommended in a child's blood at 10 µg per deciliter. This level was arrived

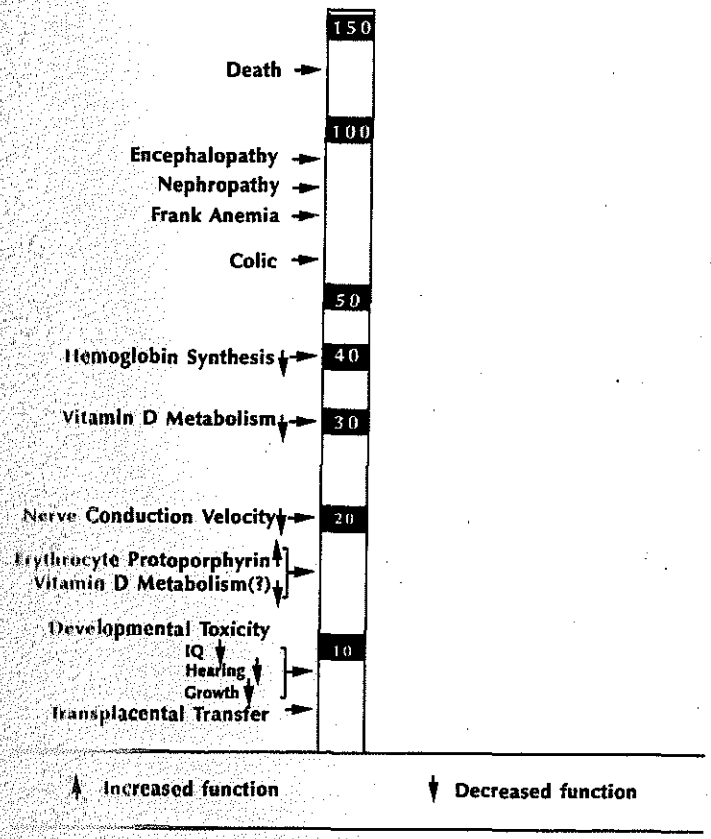

Figure 1. Lowest observed effect of levels of inorganic lead in children. (The levels are expressed in µg/dl of blood lead. They do not necessarily indicate the lowest levels at which lead exerts an effect; rather, these are the levels at which studies have adequately demonstrated an effect.) Source: reference 33.

at because of compelling evidence that there is no threshold to lead's harmful effect on intelligence. By this criterion, about one-sixth of the children in the United States have been estimated to have an excessive lead body burden that impairs health and interferes with the ability to learn.[36, 66] Fortunately, this cognitive and neurobehavioral damage may be partly reversible if blood lead levels are reduced over time.[67]

In addition, in the United States, an estimated 4 million women of child-bearing age are exposed to excessive environmental lead. Maternal and umbilical-cord blood lead levels of 10 μg/dl are associated with low birth weight and prematurity, conditions that expose an infant to a host of health and developmental risks. Major societal investments of resources in the near future will be necessary to protect those women's fetuses from *in utero* lead poisoning. They may be expected to bear some 10 million children at risk of lead poisoning at birth.[37]

Problems of similar or even greater magnitude confront many other nations, including those in Eastern Europe and the former Soviet Union. Responsible public-health decision makers are continually revising "safe" standards as more refined data become available. The same process will no doubt be true of many other toxins in our food, water, air, and homes about which we have far less knowledge than we do about lead.

Other Heavy Metals

Lead is not the only metal to contaminate food. Several highly toxic metals that are often used in agricultural and industrial applications may enter the food supply intentionally or inadvertently. Common domestic sources of exposure to arsenic, cadmium, copper, and mercury are pottery, metal pans, teapots, cooking utensils, and packaging materials. Arsenic, copper, and mercury are also used in herbicides, fungicides, and insecticides. Any and all of these routes of exposure can cause food contamination and can produce acute or chronic illnesses.

Cadmium

Cadmium can contaminate food by its presence in pesticides, pigments, paints, plastics, and cigarettes. In the United States, 500,000 individuals have occupational exposures to cadmium in mining, welding, galvanizing, battery production, and many other industries. Families who live near the sites of such industries or who are engaged in cottage industries involving cadmium-containing pigments or batteries may also develop cadmium toxicity through exposure to cadmium in food, air, soil, and water.[38] Substantial cadmium pollution can occur in areas where arsenic, zinc, copper, lead, and cadmium are mined from iron ore. In Japan, cadmium runoff from mines has polluted rivers that were used to irrigate rice paddies. Individuals who consumed cadmium-contaminated rice developed chronic cadmium poisoning and had shortened life spans.[39]

Cadmium accumulates throughout life. High exposure has been linked to osteomalacia, a softening of the bones.[40] Cadmium damages renal tubules, causing proteinuria, a condition in which serum proteins are excreted in excess in the urine. A dose-response relationship has been shown between the prevalence of proteinuria and the cadmium content of rice in contaminated regions.[41] Finally, substantial concern exists over the possibility, suggested by animal research and epidemiological studies, that chronic lower-dose cadmium exposure can cause cancer, particular of the lung and of the prostate.

Mercury

Mercury contamination of food has been well documented in locations as diverse as Michigan, Iraq, and Japan. A classic episode occurred in the 1950s in Minamata Bay, Japan. A chemical factory that made vinyl chloride dumped mercury into the bay. Individuals who ate contaminated fish developed mercury toxicity accompanied by neurological disorders, including progressive peripheral paresthesias with numbness and tingling sensations in the extremities, loss of muscle coordination with unsteadiness of gait and limbs, slurred speech, irritability, memory loss, insomnia, and depression. Forty deaths and at least 30 cases of cerebral palsy with permanent disability were reported.[42]

A much larger epidemic of similar neurological disorders occurred in Iraq when seed grain treated with mercury fungicide, instead of being planted, was mistakenly incorporated into wheat flour and baked into bread. More than 450 persons died, and more than 6,000 were hospitalized.[43]

In the United States, an estimated 68,000 workers are exposed to mercury in the workplace. The major agricultural and industrial sources of mercury are fungicides, pesticides, paints, pharmaceuticals, batteries, electrical equipment, thermometers, and the industrial production of chlorine and vinyl chloride.[44]

Ingestion of contaminated fish and fish products is a major source of environmental exposure to mercury. In the United States, mercury contamination of freshwater fish is prevalent in the Great Lakes region. Excessive levels of methylmercury have been reported in fish in scores of Michigan lakes. Public health authorities in 20 states have issued advisories that children, women of child-bearing age, and pregnant and lactating women should avoid eating certain fishes from contaminated lakes. However, an estimated 20% of the fish and shellfish consumed in the United States comes from subsistence fishing or recreational fishing and is not subject to adequate monitoring from an environmental health standpoint.[45]

Mercury compounds from agricultural and industrial sources are converted by bacteria into methylmercury, which is soluble, mobile, and rapidly incorporated into aquatic food chains. Mercury concentrates as it moves up the food chain, accumulating in carnivorous fishes (such as the northern pike) to levels 10,000–100,000 times the concentrations in the surrounding water.[46] Marine fishes, especially carnivorous ones such as the swordfish, have been found to contain high levels of mercury, exceeding 1 μg per gram.[47] Between 70% and 90% of the mercury detected in fish muscle is in the bioavailable form of methylmercury and hence is readily absorbed.[48]

Environmental agencies in New York, Wisconsin, and Minnesota have reported an association between lake acidification from acid rain and increasing levels of mercury in fish. Tropospheric ozone pollution and global warming may also lead to increased levels of mercury in freshwater fish, the former by increasing the rate of conversion of elemental mercury to methyl mercury[49] and the latter through increased atmospheric mercury deposition.[50] In-depth reviews of the subject of food-related mercury toxicity and safety can be found in references 51 and 52.

Arsenic

Arsenic is used widely in insecticides, fungicides, and herbicides, and may contaminate food by all these routes. Diet represents the largest source of arsenic exposure for the general population, followed by groundwater contamination. In addition, an estimated 55,000 U.S. workers have had occupational exposures to arsenic.

Arsenic is found in 28% of U.S. "Superfund" hazardous-waste sites, and migration from those sites, with subsequent contamination of food and water, has been documented.[53] And young children living near pesticide factories or copper smelters may ingest arsenic-contaminated soil on playgrounds, adding to the possibility of their developing arsenic toxicity.

Symptoms of acute arsenic toxicity are nausea, vomiting, diarrhea, abdominal pain, and metallic taste. Severe toxicity may cause circulatory collapse, seizures, and kidney failure due to acute tubular necrosis. Chronic exposure to moderately high levels of arsenic is associated with fatigue, weakness, gastroenteritis, dermatitis, and peripheral neuropathies that begin with painful feet and progress to a loss of normal sensation in the hands and feet in a "stocking and glove" pattern.

In Taiwan, chronic exposure to moderately elevated levels of arsenic in food or drinking water have been linked to an increased risk of skin cancer (and also, perhaps, to "black foot disease").[54] A positive dose-response relationship was also observed for bladder, liver, and lung cancers.[55]

The potential for carcinogenicity remains a primary concern for exposure to arsenic at low levels. As with many topics related to food toxicology, little epidemiological research exists which can address this issue; extrapolation from high-exposure studies using conventional methods suggests that significant risks may exist.

Copper

Copper is used widely in many industries, including agriculture; it is used in plumbing and in cookware; and has been identified in 18% of U.S. hazardous-waste sites. Acidic drinking water mobilizes copper from plumbing. In many countries, including the United States, copper

sulfate is added directly to reservoirs to control algae. This sharply raises the level of copper in drinking water for several days.[52]

With very high levels of exposure, acute copper poisoning results in nausea, vomiting, diarrhea, and metallic taste. Chronic copper toxicity has been studied in the context of Wilson's disease, a rare inherited metabolic disease in which copper accumulation leading to central-nervous-system degeneration, liver disease, and anemia.[56] *In vitro* studies and mammalian *in vivo* studies suggest that copper may also be a human mutagen.[57] Relatively little is known about the potential toxicity of copper at the levels of exposure most commonly encountered. There is reason for concern, however, because of the very broad human exposure to copper compounds.

Miscellaneous Contamination

Food can be inadvertently contaminated by industrial chemicals mistakenly introduced during processing and distribution. For instance, the ingestion of refined aniline-adulterated rapeseed oil in Spain in 1981 was associated with the development of a toxic syndrome with autoimmunological features.[58]

The recent introduction of food irradiation has generated some concerns regarding the potential induction of harmful radioactivity, radiolytic products (such as superoxide radicals), and mutant strains of microorganisms.[59] Little hard evidence exists that supports these concerns.[60] Nevertheless, additional research seems prudent in view of the widespread potential application of this method of preserving food.

Conclusion

The integrity of food is threatened by a number of man-made pollutants that can be introduced at any step in the food chain and in the food-processing industry. There have been a number of instances in which high-level poisoning has occurred through human error and negligence. The potential toxicity of exposures to pesticides, metals, radionuclides, and other contaminants that have slowly accumulated in soils and the food chain is of growing concern.

Most of these toxins are invisible and are not easily detected by consumers. Moreover, the processing or cooking of food is generally not effective in neutralizing their impact. For instance, broiling fish contaminated with polychlorinated biphenyls and pesticides has not been found to significantly alter their levels.[61]

Painfully slow research has begun to clarify the risks associated with food contamination. New tools are being developed to better define accumulated exposure and early health effects in humans[62]; in the meantime, it would seem prudent to pursue primary prevention and to vigilantly guard against the contamination of the food supply by environmental pollutants.

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