The Impact of Toxic Substances On the Poor in Developing Countries

Prepared by
Lynn Goldman
and
Nga Tran
for the
World Bank

I V E S U M M A R Y

COVER:
Informal factories and workshops cluster in and around Dhaka city, Bangladesh, near the Buriganga River. One such industry specializes in the recycling of dry cell batteries gathered from around the area. Hundreds of women and children are employed in this industry. All day long the children break apart used batteries to remove tiny pieces of metal that are sent for recycling. In the process of breaking used batteries, children inhale millions of fine carbon dust particles from the batteries throughout the day. Depending on how much work they do, each of them get between 5-15 Taka per day (US$ 1.00 = Taka 60).

PREVENTABLE TRAGEDIES:

The Impact of Toxic Substances On the Poor in Developing Countries

Prepared by
Lynn Goldman
and
Nga Tran
for the
World Bank

E X E C U T I V E A R Y

LETTER FROM THE DIRECTOR

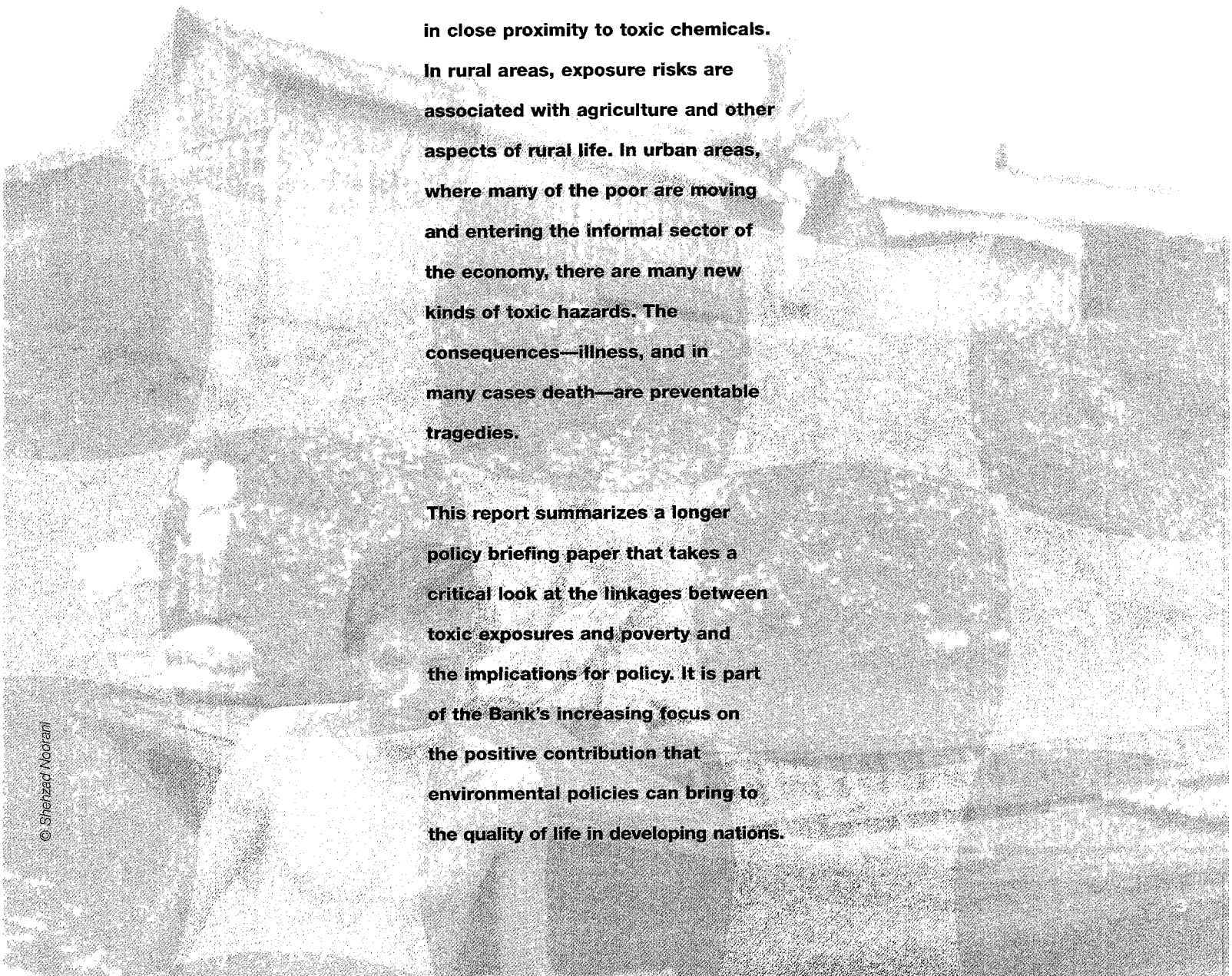
People living in poverty in developing countries must struggle every day to gain the most basic necessities of life. Environmental problems—degraded land, poor air and water quality—make the struggle more difficult. In extreme cases, such as exposure to toxic substances, these problems are often life-threatening.

Exposure to toxics can lead to a range of consequences, from a weakened ability to combat disease to death. The exposure is particularly dire for children, whose small bodies and early development make the effects all the more severe. Economic conditions that force children to work to help their families often increase opportunities for exposure.

Although the adverse impacts of toxics on poor people seem all too apparent, it is essential to take a critical look at the issue in order to understand how to mitigate the effects and improve the conditions in which people live. With this report, Preventable Tragedies: The Impact of Toxic Substances On the Poor in Developing Countries, *we hope to illuminate the links between people living in poverty and their exposure to toxic substances and draw some policy implications.*

In its recent Environment Strategy, the World Bank outlined three main objectives: improving the quality of life, improving the quality of growth, and protecting the quality of the regional and global commons. This report focuses on all three objectives, but primarily on the quality of life—environmental issues affecting people's health, livelihoods, and vulnerability. We hope this report contributes to a better understanding of the dangers toxics pose to the health and well-being of the poor in developing countries as part of a broader global chemicals agenda and the ongoing efforts toward achieving sustainable development.

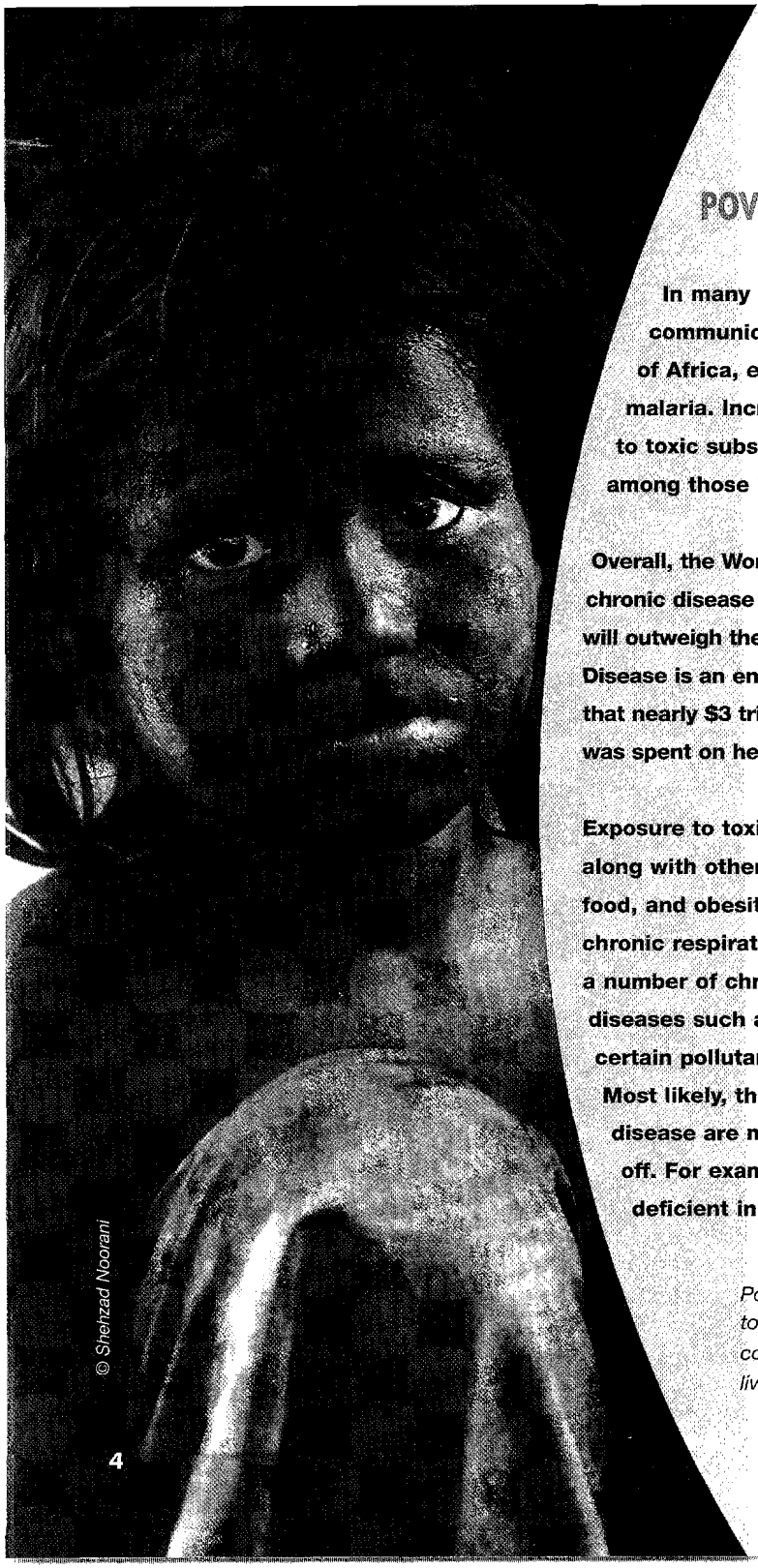
Kristalina Georgieva
Director
Environment Department



© Shehzad Noorani

It is a fact of life in many developing countries that poor people are living and working in close proximity to toxic chemicals. In rural areas, exposure risks are associated with agriculture and other aspects of rural life. In urban areas, where many of the poor are moving and entering the informal sector of the economy, there are many new kinds of toxic hazards. The consequences—illness, and in many cases death—are preventable tragedies.

This report summarizes a longer policy briefing paper that takes a critical look at the linkages between toxic exposures and poverty and the implications for policy. It is part of the Bank's increasing focus on the positive contribution that environmental policies can bring to the quality of life in developing nations.



© Shehzad Noorani

POVERTY AND THE BURDEN OF CHRONIC DISEASE

In many parts of the world, public health institutions focus on communicable diseases of epidemic proportions, like HIV infection in parts of Africa, enteric diseases from contaminated drinking water and food, and malaria. Increasingly, however, chronic diseases—caused in part by exposure to toxic substances—are emerging as problems in developing countries and among those in poverty.

Overall, the World Health Organization (WHO) projects that the burden of chronic disease in developing countries is becoming relatively more important, and will outweigh the burden from infectious disease by 2020 (Murray and Lopez, 1997). Disease is an enormous burden for the global economy. In 2000, WHO estimated that nearly $3 trillion—almost 8 percent of world gross domestic product (GDP)—was spent on health care (WHO, 2000).

Exposure to toxic substances is an important contributor to chronic disease, along with other factors like smoking, alcohol consumption, excess intake of food, and obesity. Pollution is a factor in the development of cancer and chronic respiratory diseases. It probably also plays a role in birth defects and a number of chronic diseases in adults, including neurodegenerative diseases such as Parkinson's (Lockwood, 2000). Recent data also indicate that certain pollutants may play a role in diabetes (Longnecker and others, 2001). Most likely, those who are more poorly nourished and who have concurrent disease are more susceptible to toxic chemicals than those who are better off. For example, lead is known to be more toxic to children whose diets are deficient in calories, iron, and calcium (Mahaffey, 1995).

Poor children employed in the informal labor sector...are exposed to a multitude of toxic chemicals. Working children must also commonly contend with low wages, long working hours, appalling living conditions, and food scarcity.

Children as a Vulnerable Population

Many documented cases of exposure to toxic metals, pesticides, and industrial chemicals involve children. In all these cases, children were the most vulnerable and suffered the most harm. Children's participation in certain kinds of labor further magnifies their exposure to toxic chemicals. Of the roughly 250 million working children[1] between the ages of 5 and 14 in the poor developing regions of the world, the International Labour Organisation (ILO) estimates that more than 60 percent[2] have been exposed to hazardous conditions.[3] Approximately two-thirds of the exposures occurred in rural areas. With the rapid urbanization of most developing countries and an increasing number of youngsters migrating to towns and cities, the total volume of child labor at urban centers is also expected to grow steadily (ILO, 1992). In urban areas, child labor is found mainly in trade, services (especially domestic work), and the manufacturing sectors. Poor children employed in the informal labor sector or engaged in scavenging activities are exposed to a multitude of toxic chemicals (Laraqui and others, 2000). In addition to toxic exposures, working children must also commonly contend with low wages, long working hours, appalling living conditions, and food scarcity.

Linkage between Poverty and Toxic Exposure

Poverty and adverse environmental conditions go hand-in-hand. Large populations of extremely poor people are threatened by sanitary conditions that result in contamination of drinking water and food with pathogens. By burning wood for cooking and heating in poorly ventilated homes, these same populations also are more likely to suffer from exposure to high levels of household air pollution from hazardous combustion products. Exposure to toxic substances also plays an important role in the health of poor people.

Data compiled by the World Bank indicate that poor countries face higher levels of pollution. Emissions of sulfur dioxide (SO_2) from burning fossil fuels, especially coal, and from certain industrial sources have been decreasing worldwide since 1980, yet per-capita levels of SO_2 emissions in developing countries remain above those of high-income Organisation for Economic Cooperation and Development (OECD) countries. Studies by the World Bank also show that developing countries have higher emission levels of organic water pollutants than high-income OECD countries (World Bank, 2000).

Exposure to a toxic chemical may occur when a person has contact with the chemical. Toxics can enter the body in a variety of ways. They can be inhaled, ingested, or absorbed through the skin. A pregnant woman can transfer toxics to her fetus, and mothers can transfer toxics to children through breastfeeding. All chemical substances can cause injury or disease in humans at sufficient doses.

Some chemicals may cause injury to one or more organs of the body, such as the lungs, liver, or kidneys. Others may adversely affect the functioning of the nervous system, the immune system, the reproductive system, or the endocrine system. Some can cause specific diseases, such as cancer or various types of birth defects. Some may irritate the skin or eyes; in some cases, the irritation may be serious enough to cause permanent damage, while in other cases the injury may be completely reversible. Depending on the size of the exposure and its duration, a single chemical may produce several different types of toxicity.

There are some case reports on toxic exposures in the developing world. Nevertheless, the data are sparse and have not been systematically collected. This paper summarizes the available evidence in the peer-reviewed literature that shows the connection between poverty and toxic exposure.

---

[1] In this document the terms "working child" and "child labor" have been interpreted to mean "economically active child", "child workforce", or "child labor force". All these terms have been used interchangeably.
[2] Sixty percent (2.21 million) of the 3.67 million economically active children included in the ILO survey.
[3] Hazardous conditions included biological, chemical, and physical hazards.

POVERTY AND TOXIC EXPOSURE

Metals

Lead, mercury, arsenic, cadmium, manganese, and chromium pose serious threats to human health. Exposure can occur through contamination of food or drinking water. Metals can also be emitted during the combustion of fuels and wastes, posing the risk of exposure through inhalation.

The risks to human health of most toxic metals have never been studied in developing countries. Nevertheless, there is ample evidence of significant environmental and/or human exposures in poor and developing countries; examples include industrial discharges in Rwanda (Gasana and others, 1997), wastewater spreading in Morocco (Lekouch and others, 1999), and mining activities in Mexico (Villanueva and Botello, 1998). With industrialization, more exposures to toxic metals are likely, mostly in the context of workplace environments, poorly contained wastes, and communities that are near mining, smelting, and certain industrial activities.

Of all toxics, the ones that affect people living in poverty the most are lead and mercury.

LEAD. Lead is a toxic metal that has adverse effects on the brain, kidneys, blood pressure, and blood cells. Lead exposure is particularly dangerous for children: it can affect the growth of the fetus and child–and can have particularly adverse effects on IQ and the behavior of developing children. Poverty and lead exposure have gone hand-in-hand everywhere in the world, even in developed nations like the United States, where average lead levels among poor children are four times higher than levels among children who are not in poverty (Brody and others, 1994).

Lead exposure has been extensively studied in Mexico and in China. Studies in the early 1990s documented very high lead levels among children in Mexico. Numerous investigations have concluded that leaded gasoline and industrial activities have created pervasive problems of lead exposure to children in China. The situations in Mexico and China are replicated everywhere in the developing world, suggesting that other countries with the same patterns of development have similar problems. Many studies have found higher blood lead levels associated with certain activities, including:

- Lead pottery usage in Arab countries (Manor and Freundlich, 1983)
- Children employed in the ceramic tile industry in Ecuador (Harari and Cullen, 1995)
- Children in Manila whose parents repaired and recycled batteries at home (Suplido and Ong, 2000)
- Adults recycling batteries in Taiwan (Wang and others, 1998), Jamaica (Matte and others, 1989) and the Dominican Republic (Kaul and Mukerjee, 1999)
- People living near battery–manufacturing facilities in Nicaragua (Morales Bonilla and Mauss, 1998)
- People living near a former lead smelter in Jamaica (Lalor and others, 2001)
- Uncontrolled lead waste disposal in Chile (Sepulveda, Vega and Delgado, 2000).

Poverty and proximity to roadways are often important factors in lead exposure. In Bangladesh, air-lead levels and low parental education (an index of poverty) both significantly increased the odds of having high blood-lead levels (Kaiser and others, 2001). Similar findings were reported from Saudi Arabia (Al-Saleh and others, 1999); Indonesia (Heinze and others, 1998); China (Shen and others, 1997); and South Africa (Nriagu and others, 1997; von Schirnding and others, 1991).

MERCURY. Exposure to hazardous mercury levels can cause permanent

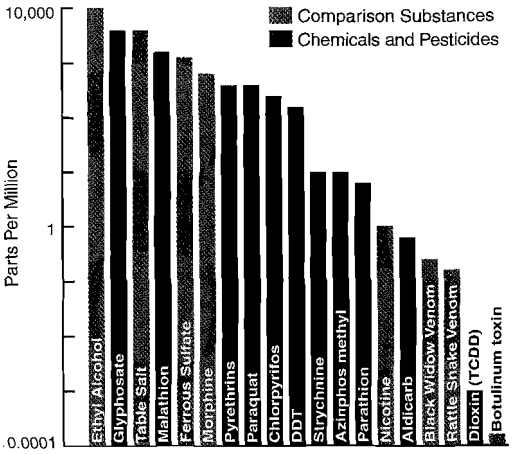
neurologic and kidney impairment. With rapid industrialization, mercury processing is being carried out in developing countries with poor workplace control; a predictable consequence is mercury intoxication of workers. This has been documented in many cases, including Zulu workers in a mercury processing plant in South Africa (Powell, 2000) and people eating fish in communities downstream from that plant (Oosthuizen and Ehrlich, 2001). In another case, poorly controlled mercury-containing waste caused hazardous exposures to mercury among workers in Cambodia (Hess and Frumkin, 2000). Small-scale ("artisanal") gold mining is another important source of mercury in developing countries. Studies in Brazil were the first to document that gold miners were using mercury to extract gold from river sediments, which caused high levels of the toxic organic compound methylmercury in fish (Malm, 1998). Researchers have documented elevated levels of mercury, and neurotoxic effects, among people who consumed those fish (Lebel and others, 1998). This has also been documented in Suriname (de Kom, von der Voet, and de Wolff, 1998), Tanzania (van Straaten, 2000), and the Philippines (Drasch and others, 2001; Appleton and others, 1999).

Pesticides

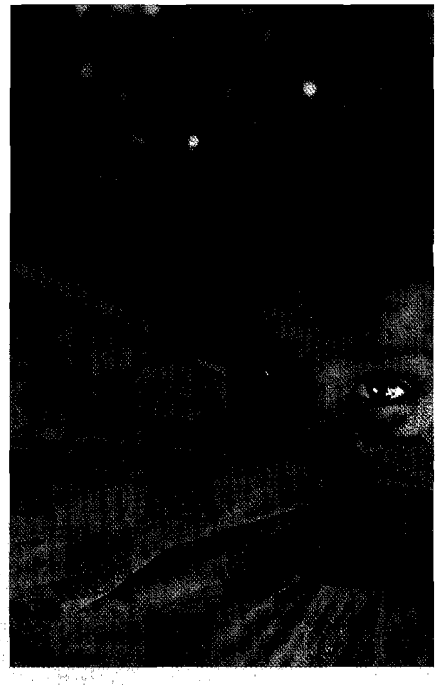
Pesticides have great variations in their potential for adverse effects. Three classes of pesticides—chlorinated hydrocarbons, organophosphates, and dithiocarbamate pesticides—have a high index of toxicity. Chlorinated hydrocarbons also are highly persistent in the environment. An analysis of data collected from 1990 to 1998 by the Food and Agriculture Organization of the United Nations (FAO) indicates that developing countries other than the least-developed nations[4] consumed the bulk of reported organochlorines, organophosphates, and dithiocarbamates. This finding is not a surprise, since pesticides are expensive inputs to agriculture. These pesticides are older pesticides that are off-patent, and therefore are much less expensive on the world market than some of the newer, and often safer, pesticides.

Exposure to pesticides can cause a range of health effects, including acute poisoning; chronic long-term effects on the nervous or immune systems; and cancer. The figure on this page shows the great variability in acute toxicity of pesticides compared with other substances. Aldicarb is among the most toxic; a dose of only 0.9 mg/kg is lethal to half the animal population ingesting it (the "LD50"). Nonetheless, it is less toxic than botulinum toxin and dioxin (lethal at 0.0001 and 0.001 mg/kg, respectively.) The herbicide glyphosate is less acutely lethal than common table salt. Chronic effects of pesticides in people are more difficult to assess; most information is based on animal studies.

LD50 FOR PESTICIDES AND COMPARISON SUBSTANCES (FOR RATS)



[4] *Using the available UNDP income and human poverty indicators, the Bank's data on international poverty lines (i.e., $1 and $2 a day per person), and the UN's "least developed" country classification, developing countries were dichotomized. As a group, developing countries below the line have a 53 percent rate of overall poverty and a 30 percent rate of extreme poverty. These rates are two times higher than other developing countries and six times those of high-income OECD countries.*



© Shehzad Noorani

PESTICIDE POISONING IN INDIA

Too often, for the poor, there is little separation between work and home environments and work related toxic substances expose children. Exposures to toxic chemicals can occur through contaminated food and water, skin absorption, inhalation, or tranmission from mother to child across the placenta, and in breast milk.

Pesticide poisoning is a major public health problem in developing countries (Ecobichon, 2001; Koh and others, 1996). The extent of the problem is unknown; the World Health Organization (WHO, 1990) has estimated an annual worldwide incidence of 3 million cases of acute, severe poisoning (including suicides), matched possibly by a much greater number of unreported cases of mild-to-moderate intoxication, with some 220,000 deaths (Koh and Jeyaratnam, 1996; Ecobichon, 2001). Approximately 99 percent of these deaths occurred in developing countries, with suicides making up about two-thirds of the total (McConnell and Hruska, 1993; Koh and Jeyaratnam, 1996). Poisoning incidents in these countries often involved organophosphates, carbamates, paraquat, and diquats (Ecobichon, 2001; Ramasamy and Tajol Akos, 1988, cited in Castro-Gutierrez and others, 1997). These numbers mostly reflect very severe incidents that involve hospitalizations and thus are an underestimate and also are likely to over-represent suicides. The WHO is currently attempting to improve its capability to detect incidents that are treated outside hospitals.

A study of childhood poisoning—using data from eight regional hospitals in India—found that accidental poisoning was common, and that 50-90 percent of the cases involved children below 5 years of age. Pesticide poisoning was more prevalent in Punjab and West Bengal. Various regions in the country showed some variation in types and frequency of childhood poisoning, which could be attributed to different geographical and socioeconomic backgrounds (Dutta and others, 1998). Pulmonary studies on 489 pesticide workers engaged in spraying operations on mango plantations revealed that occupational exposure to pesticides had a direct bearing on respiratory impairment. These workers were exposed to a variety of organochlorines and organophosphorus pesticides (Rastogi and others, 1989).

Outbreaks of fatal food poisoning are common. For example, on July 6, 1997, 60 men aged 20-30 years attended a communal lunch cooked in the community kitchen. They ate chapatti, cooked vegetables, pulses, and halva. They all developed nausea, vomiting, and abdominal pain over the next three hours. All were successfully treated, except for one who died from the exposure. Malathion was found in leftover food samples from the shared lunch (Chaudhry and others, 1998).

A number of pesticide poisoning cases in impoverished countries of Africa have been reported. Examples of overexposure include worker exposure due to poor handling and storage practices in the Accra Plains, Ghana (Clarke and others, 1997); organophosphate (parathion) poisoning in Nairobi, Kenya (Obel, 1984); and high rates of illness among agricultural workers on farms producing cotton, tobacco, flowers, and horticultural crops (Ohayo-Mitoko and others, 1999). In Africa, it is estimated that 11 million cases of pesticide overexposure occur annually (Choudhury, 1989, cited in Koh and, Jeyaratnam, 1996). Although not all the exposures would result in acute illness, such exposures may increase the risk for chronic health impacts.

In Asia, India has had a large number of poisoning cases (see page 8). An estimated 5 percent of Sri Lankan workers and 13 percent of Malaysian workers have been poisoned by pesticides, mainly paraquat, organophosphates, and organochlorines, according to a study conducted during the 1980s (Jeyaratnam, Lun, and Phoon, 1987). In Thailand, a total of 4,046 cases of pesticide illness resulting in 289 deaths was recorded in 1985 (Kritalugsana, 1988, cited in Koh and Jeyaratnam, 1996). Local studies in Indonesia indicate that 30,000 cases of pesticide poisoning occur annually in the nation as a whole (Jeyaratnam, 1990, cited in Koh and Jeyaratnam, 1996).

Stories of exposure to toxic pesticides among the poor are also common throughout Latin American. In Costa Rica, pesticides—the only widespread chemical pollutants in rural areas—are the main sources of agricultural workers' exposures and illnesses (Wesseling, Castillo, and Elinder, 1993). In El Salvador, studies have found that subsistence farmers are widely exposed to organophosphates (Azaroff, 1999). In Chiapas, Mexico, peasants from the poorest communities face the greatest risk of pesticide exposure (Tinoco-Ojanguren and Halperin, 1998). On Nicaragua's cotton farms, pesticide poisoning has also long been a major public health problem; studies have shown that children in nearby households have been poisoned by organophosphates (McConnell and Hruska, 1992); (McConnell and others, 1999). The 1999 tragedy of Peruvian children dying after eating a government-donated breakfast contaminated with parathion is further evidence that poor children in poor nations are very vulnerable to toxic exposures.

Characteristics associated with poverty—lack of education, poor housing, high-risk jobs, and child labor—are intertwined with toxic exposures. Many health concerns are closely associated with particular occupations. For example:

- Farmers in developing countries are unaware of the short- and long-term hazards associated with exposure to many pesticide products.
- In many developing countries, pesticides are used neither efficiently nor safely. Advanced technologies such as chemical pesticide application require knowledge that goes beyond traditional agricultural practices.
- Poor knowledge and understanding of safe pesticide use, deficiencies in safety training (often leading to careless handling), excessive use of pesticides, eating and drinking while working, lack of water and facilities for personal hygiene, lax storage practices, and careless disposal of empty pesticide containers all lead to toxic exposures.
- Poor maintenance facilities for spray equipment can lead to hazardous contamination and use of pesticide mixtures. Occupational poisoning occurs largely during spraying, mixing, and diluting of pesticides.

Other concerns are not directly associated with occupations. For example :

- Bystander poisonings can be attributed to spray drift and residues in homes of populations living near sprayed fields (Ames, Howd and Doherty, 1993; Richter and others, 1992; Scarborough and others, 1989, cited in Azaroff, 1999).
- Especially high levels of community pesticide exposure can be expected in poor countries. Conditions contributing to such exposure include excessive use of very toxic compounds; uncontrolled patterns of spraying; lack of washing facilities; improper storage of pesticides in homes; improper use of empty containers for the storage or transfer of water, vegetable oils, or food; short time intervals between pesticide applications and workers doing farm work; not observing intervals between the last pesticide application and the harvesting and eating of produce; and hand washing of clothes worn by farmers during pesticide applications.
- Occupational health legislation and regulations are extremely weak in the developing countries. Most developing countries still do not require that imported pesticides be registered in the country of origin. Pesticides banned or restricted in the country of origin are used widely.

Persistent Organic Pollutants

Twelve of the persistent organic pollutants (POPs)—all chlorinated compounds—have become the focus of international action through the convention on POPs signed in Stockholm in May 2001: polychlorinated biphenyls (PCBs), dioxins, furans, aldrin, dieldrin, DDT, endrin, chlordane, hexachlorobenzene (HCB), mirex, toxaphene, and heptachlor. PCBs are industrial chemicals; dioxin and furans are unwanted by-products of various technological processes, but were never produced commercially and have no intended use. Aldrin, dieldrin, DDT, endrin, chlordane, HCB, mirex, toxaphene, and heptachlor were initially developed as pesticides.

Because POPs persist in the environment and accumulate in body fat, the major pathway of human exposure to POPs is via the food chain. Contamination of food may occur through environmental pollution of the air, water, and soil. Short-and long-term exposure to POPs can be associated with a wide range of adverse health effects, from acute toxicity to intergenerational endocrine-disrupting effects. A growing body of evidence suggests that there may be a relationship between exposure to some POPs and reproductive dysfunction and cancers (Ritter and others, 1995).

The risk of exposure to POPs in developing countries is high. Their use in agriculture in the past resulted in a large number of deaths and injuries. In 1990 in the Philippines, for example, endosulfan was the number one cause of poisoning among subsistence rice farmers and mango sprayers (Ritter and others, 1995). With an annual average consumption of 16,354 metric tons during the 1990-98 period, India is the largest consumer of POPs (FAO, 1999). While Costa Rica consumes much less than India, its consumption rate in metric tons per square kilometer of land area is among the highest (8.83 Mt/km^2). POP insecticides left over from past use now form a significant proportion of obsolete pesticide stockpiles and pose significant ongoing risk of exposure in the developing world. DDT used for public health purposes—notably, control of mosquito vectors of malaria, yellow fever, dengue fever, and lymphatic fiariasis—is probably the largest current use.

Once POPs are in the bodies of mothers, they are readily transferred to the fetus via the placenta and to infants via breast milk. Breast milk is the best food for newborn infants, so the contamination of food and breast milk by POPs is a particular concern. Available data demonstrate that contamination of breast milk and food

by POPs is a worldwide phenomenon. Nevertheless, there has been little monitoring of POPs body burdens and environmental levels in the developing countries. A limited number of peer-reviewed journal articles on environmental contamination, population exposure to POPs, and possible health effects in poor and developing countries is available.

The most extensive published, peer-reviewed studies were found for Kazakhstan and VietNam. The POPs story in Kazakhstan, part of the former Soviet Union, involves historical agricultural use of pesticides in cotton fields. Human consumption of fish contaminated with industrial pollution in the Aral Sea Region is another source of exposure. Reportedly, many children in Kazakhstan are in poor health and suffering from myriad neurological defects due to toxic exposures.

In VietNam, the U.S. military's use of the defoliant Agent Orange during the VietNam war resulted in a widely documented and persistent dioxin contamination and human exposure (Schecter and others, 2001). Studies have also uncovered environmental contamination involving other POPs—including hexachlorocyclohexane (HCH), DDT, and HCB—as the result of ongoing use of pesticides in this region.

© Mark Davis, Pesticide Action Network U.K.

Obsolete Pesticide Stocks

The FAO defines obsolete pesticide stocks to include all pesticide stocks not in current use because they have been banned, are deteriorated or damaged, have passed their expiration date, or are not wanted by the current owner. More than 20 percent of obsolete stockpiles consist of POP pesticides. Organophosphates are the other important group of pesticides often found among obsolete stocks. Carbamates and synthetic pyrethroid insecticides, various fungicide and herbicide groups, and even some botanical and microbial products are also included in obsolete stockpiles.

Quantification of obsolete stocks is difficult because of the wide distribution of pesticides and the remote location of many of the storage points. The FAO's recent inventories, completed for 53 countries in Africa and the Near East, recorded a total of more than 47,000 tons of obsolete pesticides (FAO, 2001; FAO, 2001b). Two countries, Botswana and Mali, had the highest quantities of obsolete pesticide stocks in their inventories—over 10,000 tons.

In countries where they exist, obsolete stocks are distributed widely. In Ethiopia, hazardous pesticide waste is stored at nearly 1,000 sites around the country. Although pesticide stores were initially constructed away from residential areas, populations have grown and urban areas now surround many pesticide storage sites. It is common to find pesticide stores with obsolete stocks in densely populated areas, a situation that obviously presents the threat of human exposure.

Open or damaged containers present a particularly serious public health concern. Spills and leaks from containers can find their way into surface waters from runoff or into groundwater from leaching through soil. Where pesticides are stored in the open, people who work, live, travel, or play in the vicinity are likely to be exposed and may suffer acute or chronic health effects. For example, in the village of Arjo, Ethiopia, family huts where women prepare food and

children play are located a few meters away from a pesticide dump site and an unsecured dilapidated barn that stores 5.5 tons of obsolete pesticides— including DDT and organophosphates (malathion, pirimiphos-methyl, and fenitrothion)— in drums, boxes, and bags.

There are many barriers to solving the problem of obsolete pesticide stockpiles. The FAO has identified several of these barriers. For example:

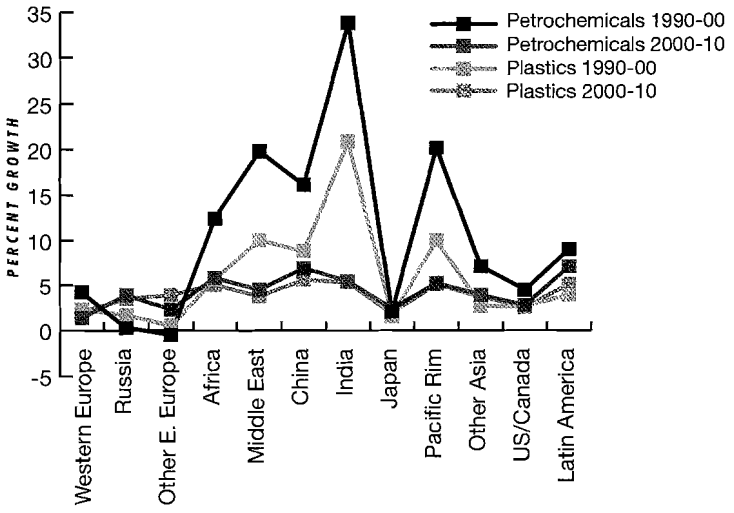
- Developing countries lack the extensive training and sophisticated safety and handling equipment required to deal with hazardous wastes such as obsolete pesticides.
- The removal and destruction of obsolete stocks is expensive, and funding from donors has been slow.
- There are few alternatives to chemical pesticides.
- The problem is compounded by the lack of infrastructure, the wide dispersal of obsolete stocks, and their deteriorated condition.
- Pesticide distribution in developing countries is slow and inefficient. As a result, obsolete stocks continue to accumulate in developing countries.

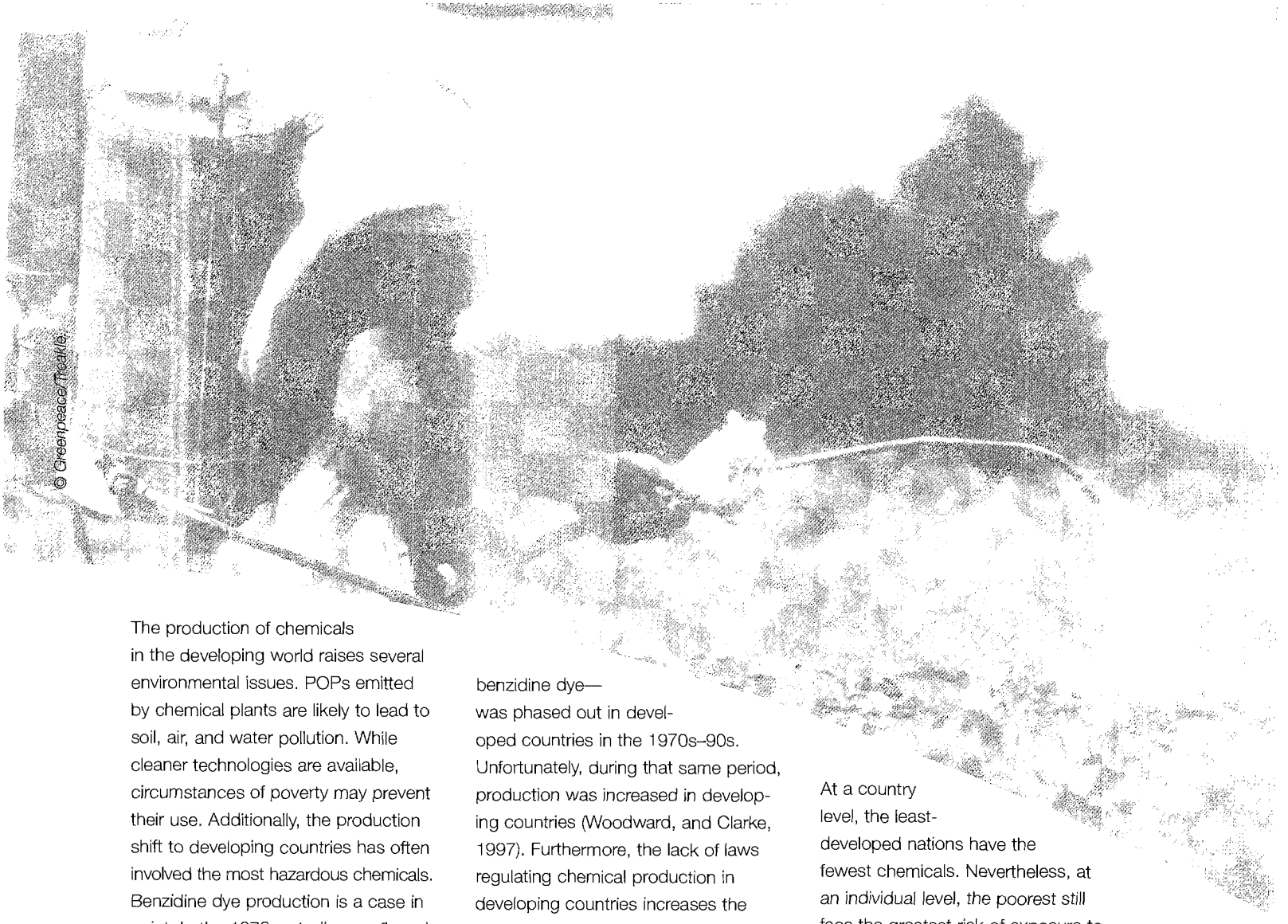
Industrial Chemicals

In relative terms, there has been a steady shift in chemical production from OECD countries to non-OECD countries over the past 40 years (OECD, 2001). With the globalization of trade, this shift is likely to continue. Generally, the shift in production has involved large-scale production chemicals like petrochemicals and plastics. The most dramatic increases have occurred in India, where there was a 33.8 percent increase in petrochemical production and a 20.8 percent increase in plastics production between 1990 and 2000.

Likewise, the Pacific Rim, the Middle East, China, and Africa had large increases. There was little or no growth in Eastern Europe, Russia, and Japan. The outlook for 2000-10 is for slower growth on a global basis, with a rate of between 4 and–6 percent in China, the Middle East, Africa, India, the Pacific Rim, and Latin America (European Chemical News, 1999). (See figure below.)

GROWTH RATES FOR CHEMICAL PRODUCTION BY REGION





© Greenpeace/Tearle

The production of chemicals in the developing world raises several environmental issues. POPs emitted by chemical plants are likely to lead to soil, air, and water pollution. While cleaner technologies are available, circumstances of poverty may prevent their use. Additionally, the production shift to developing countries has often involved the most hazardous chemicals. Benzidine dye production is a case in point. In the 1970s, studies confirmed that benzidine dye caused bladder cancer in dye workers. It was classified by the WHO International Agency for Research on Cancer as a known human carcinogen. As a result, production—of benzidine and benzidine dye—was phased out in developed countries in the 1970s–90s. Unfortunately, during that same period, production was increased in developing countries (Woodward, and Clarke, 1997). Furthermore, the lack of laws regulating chemical production in developing countries increases the opportunities for environmental pollution and subsequent human exposures. No one knows exactly how many or which chemicals are in use in these nations.

At a country level, the least-developed nations have the fewest chemicals. Nevertheless, at *an individual level, the poorest still* face the greatest risk of exposure to chemicals. The most exposed are likely to be people who work directly with chemicals, such as Mexican service station workers being exposed to the carcinogen benzene (Meneses and others, 1999; Romieu and others, 1999); workers in paint manufacture in

A man in Burkina Faso mining gold. Artisanal gold mining processes can result in environmental contamination by mercury and other toxic substances.

© Curt Carnemark

© Curt Carnemark

Two boys working at a sugarloaves field in Naivasha Horticulture, Kenya. Worldwide, an estimated two-thirds of hazardous workplace exposures to working children occur in rural areas. Working children are at particular risk from exposure to hazardous pesticides in farm work because of their smaller size (and greater potential contact with contaminated vegetation) and because they are actively growing and developing.

South Africa (Nell and others, 1993); and petrochemical workers in Brazil (Saretto and others, 1997). There are also ample opportunities for poor children employed in the informal labor sector to be exposed to a multitude of toxic chemicals (Laraqui and others, 2000).

Worldwide, communities involved with scavenging of wastes have high exposures to toxic chemicals. An investigation in Bangkok of a waste dump—where approximately 400 people of all ages work on a daily basis as scavengers—found high levels of a number of chemicals in the air, including toluene, benzene, ethylbenzene, xylene, methylene chloride, and methyl chloroform (Kungskulniti and others, 1991). These chemicals have a variety of toxic effects, including cancer and toxicity to the nervous system.

Industrial chemicals have also contaminated foods, pharmaceuticals, and consumer products, causing harmful exposures. There are many stories of toxic exposure to diethylene glycol throughout the developing countries. In 1998 in India, 36 children were hospitalized with kidney failure due to cough syrup contaminated with diethylene glycol; 33 of them died (Singh and others, 2001). A similar episode occurred in Haiti in 1998, when acetaminophen was contaminated with diethylene glycol; 109 children fell ill and 85 died (O'Brien and others, 1998). Likewise, in Bangladesh in 1990, 339 children were exposed to diethylene glycol–contaminated paracetamide (Hanif and others, 1995), and 47 children died after exposure to contaminated paracetamide in Nigeria that same year (Okuonghae and others, 1992). What all these cases had in common was that children were the most vulnerable and suffered the most harm; in all cases, the use of poorly manufactured glycerin in the manufacture of a pharmaceutical caused exposure to diethylene glycol. Good manufacturing practices can completely prevent this problem, but many of the least-developed countries have not established legal systems to ensure that such practices will be put in place.

CONCLUSIONS AND POLICY IMPLICATIONS

Poverty, development, and potential exposure to toxic substances are closely related. While development can lead to overall economic benefits, exposure to toxic substances can also occur at each stage of development, though with each new stage the kinds of hazards are likely to change. Unfortunately, most of the negative impacts are also likely to be borne disproportionately by the poorest communities. Short-term health impacts from heavy metals and highly toxic pesticides have been particularly devastating among those in poverty. The combination of rapid industrialization and persis-tent poverty create the riskiest situations. Working children in poverty are especially at risk of exposure to toxics. They often work around highly toxic substances, they are less able to protect themselves, and they have long work hours. In addition, they may have other serious risk factors like malnourishment and poor living conditions.

In both the short and long term, the environmental and public health threats associated with POPs are real for children living in the developing world. These children are exposed to high levels of POPs *in utero*, via breast milk; through consumption of contaminated foods; and by playing or working in areas with soil or sediment contamination. Living in substandard conditions, often undernourished and typically with poor health status, they are also more vulnerable to environmental chemical insults. Simply put, children living in these impoverished countries are most vulnerable to high levels of exposure and harmful effects of POPs.

Long-term Objectives.

There is a need for long-term efforts to develop legislation, regulations, and infrastructure for enforcing such legislation in poor countries; adequate financial support to maintain such infrastructure; and scientific expertise to conduct environmental and human health monitoring and surveillance. This is a large undertaking. Strong regulatory institutions will not emerge in developing countries overnight. There are many more pressing problems, including urgent socioeconomic needs, disease control, and other public health problems. Health problems posed by chemicals cannot be dealt with in isolation separate from other dire socioeconomic needs. Moreover, many exposures related to poverty may be best addressed via general improvements in living and working conditions, increased educational opportunities, and the elimination of inappropriate forms of child labor.

Short-term Objectives.

Several important policy reforms can be implemented in the short term, including:

- ***Prior Informed Consent:*** The Rotterdam Convention on Prior Informed Consent (PIC) will provide countries with the means to exclude from import any chemicals that have been determined by an international body to be very hazardous. This is an important short-term area in capacity development.

- ***Persistent Organic Pollutants:*** The global POPs convention is the first international agreement to ban or restrict chemicals based on human health concerns. To be workable, developing countries must be able to inventory and manage the use and production of POPs within their own borders as well as across borders.

- ***Globally Harmonized System:*** The ILO, the WHO, UNEP, and the OECD are collaborating on the development of a globally harmonized system for classifying and labeling industrial chemicals and pesticides. To be workable, developing countries will need to build the capacity to understand the new labels and to take protective actions in response to their warnings.

- ***Assistance Efforts:*** Given the worldwide scarcity of scientific and technical resources to address chemical risks, it is incumbent on developed countries to provide technical information, assistance, and resources to assist developing countries in putting in place chemical–risk–management efforts. In addition, all countries need to engage in efforts to develop international systems for managing chemicals.

- ***Integrated Pest Management:*** Methods of integrated pest management (IPM) that seek to minimize unnecessary pesticide use can be taught. Sustainability is ensured through community preparation, building on available local resources and commitments, weaving IPM into local community development planning processes, and situating program concerns within the local government system. Women and children may be important targets of these efforts, since they handle pesticides.

- ***Integrated Waste Management:*** In many developing countries, national and local authorities have only limited resources for managing toxic waste. It is imperative that pesticide suppliers and their distribution networks be involved in the development and implementation of the safe management and disposal of pesticide-related wastes. NGOs and farmers' organizations are invaluable channels for information and advice in rural communities. The chemical industry "Responsible Care" initiative, which has been adopted internationally, can play a role in bringing private sector resources to bear.

- ***Responsible Care and Product Stewardship:*** Chemical and pesticide suppliers should be encouraged to implement product stewardship systems that cover all stages of chemical and pesticide production, distribution, and use, as well as management of wastes. Incentives need to be in place to encourage "leap-frogging," using environmentally benign chemicals and production processes in the first instance rather than building facilities that would be unacceptable in the developed world. Pesticide and chemical companies need to engage in responsible promotion, packaging, and advertising of their products in the developing world to prevent untoward exposures. Manufacturers should be given incentives to include chemical and pesticide disposal facilities that allow their customers to dispose of empty containers and related waste materials safely. They can also invest in the capacity of communities to prepare for and respond to chemical emergencies and spills, which often require special equipment for cleanup.

- ***Obsolete Pesticide Stockpiles:*** International coordinated action is needed to address the current problems of obsolete pesticide stockpiles in the developing world, including international financial aid in disposing of existing hazardous chemicals as well as for capacity building and training to help prevent future accumulations of obsolete stocks.

- ***Empowering Civil Society via Participation and Right to Know:*** Even in the absence of strong regulatory systems, community mobilization and awareness–raising on issues such as child labor and toxic exposures, appropriate use of pesticides, and toxic wastes can be a powerful force for change in developing countries. Information and disclosure are tools that are non–regulatory yet can prompt action by companies to change their practices

REFERENCES

Al-Saleh I, Nester M, DeVol E, Shinwari N, Al-Shahria S. 1999. Determinants of blood lead levels in Saudi Arabian schoolgirls. *Int J Occup Environ Health* 5(2): 107-114.

Ames RG, Howd RA, Doherty L. 1993. Community exposure to a paraquat drift. *Arch Environ Health* 48(1): 47-52.

Appleton JD, Williams TM, Breward N, Apostol A, Miguel J, Miranda C. 1999. Mercury contamination associated with artisanal gold mining on the island of Mindanao, the Philippines. *Sci Total Environ* 228(2-3): 95-109.

Azaroff LS. 1999. Biomarkers of exposure to organophosphorous insecticides among farmers' families in rural El Salvador: factors associated with exposure. *Environ Res* 80(2 Pt 1): 138-147.

Brody DJ, Pirkle JL, Kramer RA, Flegal KM, Matte TD, Gunter EW, Paschal DC. 1994. Blood lead levels in the US population. Phase 1 of the Third National Health and Nutrition Examination Survey (NHANES III, 1988 to 1991). *Jama* 272(4): 277-283.

Castro-Gutierrez N, McConnell R, Andersson K, Pacheco-Anton F, Hogstedt C. 1997. Respiratory symptoms, spirometry and chronic occupational paraquat exposure. *Scand J Work Environ Health* 23(6): 421-427.

Chaudhry R, Lall SB, Mishra B, Dhawan B. 1998. A foodborne outbreak of organophosphate poisoning. *Bmj* 317(7153): 268-269.

Choudhury A. 1989. Health Hazards of Pesticide Use in Africa. In S Lehtinen, K Kurppak, E Korhonew, L Saarinen (Eds.), *The East Africa Regional Symposium on Chemical Accidents and Occupational Health:* pp. 70-74: Helsinki, Institute of Occupational Health.

Clarke EE, Levy LS, Spurgeon A, Calvert IA. 1997. The problems associated with pesticide use by irrigation workers in Ghana. *Occup Med* (Lond) 47(5): 301-308.

de Kom JF, van der Voet GB, de Wolff FA. 1998. Mercury exposure of maroon workers in the small scale gold mining in Suriname. *Environ Res* 77(2): 91-97.

Drasch G, Bose-O'Reilly S, Beinhoff C, Roider G, Maydl S. 2001. The Mt. Diwata study on the Philippines 1999--assessing mercury intoxication of the population by small scale gold mining. *Sci Total Environ* 267(1-3): 151-168.

Dutta AK, Seth A, Goyal PK, Aggarwal V, Mittal SK, Sharma R, Bahl L, Thakur JS, Verma M, Chhatwal J, Chacko B, Saini V, Singhal A, Sharma P, Sharma U, Chaturvedi P, Kumar S, Prajapati NC, Vaidya J, Garg N, Basu SN, Lahiri M, Das CK, Pal DK, Lall SB, and others 1998. Poisoning in children: Indian scenario. *Indian J Pediatr* 65(3): 365-370.

Ecobichon DJ. 2001. Pesticide use in developing countries. *Toxicology* 160(1-3): 27-33.

European Chemical News. 1999. Building a secure future from 2000. European Chemical News.

Food and Agriculture Organization. 1999. Codex Alimentarius: Pesticide Residues in Food. FAOSTAT. FAO: Rome.

Food and Agriculture Organization. 2001a. Obsolete pesticides threaten communities in Ethiopia. FAO: Rome.

Food and Agriculture Organization. 2001b. Prevention and Disposal of Obsolete Pesticides. FAO: Rome.

Food and Agriculture Organization. 2001. Baseline Study on the Problem of Obsolete Pesticide Stocks. FAO: Rome.

Gasana J, Twagilimana L, Hallenbeck W, Brenniman G. 1997. Industrial discharges of metals in Kigali, Rwanda, and the impact on drinking water quality. *Bull Environ Contam Toxicol* 58(4): 523-526.

Hanif M, Mobarak MR, Ronan A, Rahman D, Donovan JJ, Jr., Bennish ML. 1995. Fatal renal failure caused by diethylene glycol in paracetamol elixir: the Bangladesh epidemic. *Bmj* 311(6997): 88-91.

Harari R, Cullen MR. 1995. Childhood lead intoxication associated with manufacture of roof tiles and ceramics in the Ecuadorian Andes. *Arch Environ Health* 50(5): 393.

Heinze I, Gross R, Stehle P, Dillon D. 1998. Assessment of lead exposure in schoolchildren from Jakarta. *Environ Health Perspect* 106(8): 499-501.

Hess J, Frumkin H. 2000. The international trade in toxic waste: the case of Sihanoukville, Cambodia. *Int J Occup Environ Health* 6(4): 331-344.

International Labour Organisation. 1992. World Labour Report. ILO: Geneva.

Jeyaratnam J. 1990. Acute pesticide poisoning: a major global health problem. *World Health Stat Q* 43(3): 139-144.

Jeyaratnam J, Lun KC, Phoon WO. 1987. Survey of acute pesticide poisoning among agricultural workers in four Asian countries. *Bull World Health Organ* 65(4): 521-527.

Kaiser R, Henderson AK, Daley WR, Naughton M, Khan MH, Rahman M, Kieszak S, Rubin CH. 2001. Blood lead levels of primary school children in Dhaka, Bangladesh. *Environ Health Perspect* 109(6): 563-566.

Kaul B, Mukerjee H. 1999. Elevated blood lead and erythrocyte protoporphyrin levels of children near a battery-recycling plant in Haina, Dominican Republic. *Int J Occup Environ Health* 5(4): 307-312.

Koh D, Jeyaratnam J. 1996. Pesticides hazards in developing countries. *Sci Total Environ* 188 Suppl 1: S78-85.

Kritalugsana S. 1988. *Pesticide Poisoning Studies and Data Collection in Thailand.* Manila Island Publishers: Manila.

Kungskulniti N, Pulket C, Miller FD, Smith KR. 1991. Solid waste scavenger community: an investigation in Bangkok, Thailand. *Asia Pac J Public Health* 5(1): 54-65.

Lalor G, Rattray R, Vutchkov M, Campbell B, Lewis-Bell K. 2001. Blood lead levels in Jamaican school children. *Sci Total Environ* 269(1-3): 171-181.

Laraqui CH, Caubet A, Laraqui O, Belamallem I, Harourate K, Curtes JP, Verger C. 2000. [Child labor in the artisan sector of Morocco: determinants and health effects]. *Sante Publique* 12(1): 31-43.

Lebel J, Mergler D, Branches F, Lucotte M, Amorim M, Larribe F, Dolbec J. 1998. Neurotoxic effects of low-level methylmercury contamination in the Amazonian Basin. *Environ Res* 79(1): 20-32.

Lekouch N, Sedki A, Bouhouch S, Nejmeddine A, Pineau A, Pihan JC. 1999. Trace elements in children's hair, as related exposure in wastewater spreading field of Marrakesh (Morocco). *Sci Total Environ* 243-244: 323-328.

Lockwood AH. 2000. Pesticides and parkinsonism: is there an etiological link? *Curr Opin Neurol* 13(6): 687-690.

Longnecker MP, Daniels JL. 2001. Environmental contaminants as etiologic factors for diabetes. *Environ Health Perspect* 109 Suppl 6: 871-876.

Mahaffey KR. 1995. Nutrition and Lead: Strategies for Public Health. *Environmental Health Perspect* 103(Supplement 6): 191-196.

Malm O. 1998. Gold mining as a source of mercury exposure in the Brazilian Amazon. *Environ Res* 77(2): 73-78.

Manor E, Freundlich E. 1983. Lead poisoning from ceramics in the Arab population. *Arch Toxicol Suppl* 6: 275-278.

Matte TD, Figueroa JP, Burr G, Flesch JP, Keenlyside RA, Baker EL. 1989. Lead exposure among lead-acid battery workers in Jamaica. *Am J Ind Med* 16(2): 167-177.

McConnell R, Cordon M, Murray DL, Magnotti R. 1992. Hazards of closed pesticide mixing and loading systems: the paradox of protective technology in the Third World. *Br J Ind Med* 49(9): 615-619.

McConnell R, Hruska AJ. 1993. An epidemic of pesticide poisoning in Nicaragua: implications for prevention in developing countries. *Am J Public Health* 83(11): 1559-1562.

McConnell R, Pacheco F, Wahlberg K, Klein W, Malespin O, Magnotti R, Akerblom M, Murray D. 1999. Subclinical health effects of environmental pesticide contamination in a developing country: cholinesterase depression in children. *Environ Res* 81(2): 87-91.

Meneses F, Romieu I, Ramirez M, Colome S, Fung K, Ashley D, Hernandez-Avila M. 1999. A survey of personal exposures to benzene in Mexico City. *Arch Environ Health* 54(5): 359-363.

Morales Bonilla C, Mauss EA. 1998. A community-initiated study of blood lead levels of Nicaraguan children living near a battery factory. *Am J Public Health* 88(12): 1843-1845.

Murray CJ, Lopez AD. 1997. Alternative projections of mortality and disability by cause 1990-2020: Global Burden of Disease Study. *Lancet* 349(9064): 1498-1504.

Nell V, Myers J, Colvin M, Rees D. 1993. Neuropsychological assessment of organic solvent effects in South Africa: test selection, adaptation, scoring, and validation issues. *Environ Res* 63(2): 301-318.

Nriagu J, Jinabhai CC, Naidoo R, Coutsoudis A. 1997. Lead poisoning of children in Africa, II. Kwazulu/Natal, South Africa. *Sci Total Environ* 197 (1-3): 1-11.

Obel AO. 1984. Practical therapeutics: first aid and therapy of poisoning from agricultural insecticides and herbicides. *East Afr Med J* 61(4): 335-338.

O'Brien KL, Selanikio JD, Hecdivert C, Placide MF, Louis M, Barr DB, Barr JR, Hospedales CJ, Lewis MJ, Schwartz B, Philen RM, St Victor S, Espindola J, Needham LL, Denerville K. 1998. Epidemic of pediatric deaths from acute renal failure caused by diethylene glycol poisoning. Acute Renal Failure Investigation Team. *Jama* 279(15): 1175-1180.

Ohayo-Mitoko GJ, Kromhout H, Karumba PN, Boleij JS. 1999. Identification of determinants of pesticide exposure among Kenyan agricultural workers using empirical modelling. *Ann Occup Hyg* 43(8): 519-525.

Okuonghae HO, Ighogboja IS, Lawson JO, Nwana EJ. 1992. Diethylene glycol poisoning in Nigerian children. *Ann Trop Paediatr* 12(3): 235-238.

Oosthuizen J, Ehrlich R. 2001. The impact of pollution from a mercury processing plant in KwaZulu- Natal, South Africa, on the health of fish-eating communities in the area: an environmental health risk assessment. *Int J Environ Health Res* 11(1): 41-50.

Organisation for Economic Cooperation and Development. 2001. Environmental Outlook for the Chemicals Industry: 168 pages.

Powell TJ. 2000. Chronic neurobehavioural effects of mercury poisoning on a group of Zulu chemical workers. *Brain Inj* 14(9): 797-814.

Ramasamy S, Tajol Akos N. 1988. A survey of pesticide use and associated incidence of poisoning in Peninsular Malaysia. *J Pl Prot Tropics* 5: pp. 1-9.

Rastogi SK, Gupta BN, Husain T, Mathur N, Garg N. 1989. Study of respiratory impairment among pesticide sprayers in Mango plantations. *Am J Ind Med* 16(5): 529-538.

Richter ED, Chuwers P, Levy Y, Gordon M, Grauer F, Marzouk J, Levy S, Barron S, Gruener N. 1992. Health effects from exposure to organophosphate pesticides in workers and residents in Israel. *Isr J Med Sci* 28(8-9): 584-598.

Ritter L, Forget J, Solomon KR. 1995. Persistent Organic Pollutants: 41 pages. Canadian Network of Toxicology Centres: Ontario.

Romieu I, Ramirez M, Meneses F, Ashley D, Lemire S, Colome S, Fung K, Hernandez-Avila M. 1999. Environmental exposure to volatile organic compounds among workers in Mexico City as assessed by personal monitors and blood concentrations. *Environ Health Perspect* 107(7): 511-515.

Saretto G, Oliveira FC, Kato M, Reis EJ, Castro RC, Dorigatti F, Camerino D, Gilioli R. 1997. Exposure to organic solvents and its effects on the central nervous system in workers of the Camacari petrochemical complex in Brazil. *G Ital Med Lav Ergon* 19(3): 66-71.

Scarborough ME, Ames RG, Lipsett MJ, Jackson RJ. 1989. Acute health effects of community exposure to cotton defoliants. *Arch Environ Health* 44(6): 355-360.

Schecter A, Dai LC, Papke O, Prange J, Constable JD, Matsuda M, Thao VD, Piskac AL. 2001. Recent dioxin contamination from Agent Orange in residents of a southern Vietnam city. *J Occup Environ Med* 43(5): 435-443.

Sepulveda V, Vega J, Delgado I. 2000. [Severe exposure to environmental lead in a child population in Antofagasta, Chile]. *Rev Med Chil* 128(2): 221-232.

Shen XM, Yan CH, Guo D, Wu SM, Li RQ, Huang H, Ao LM, Zhou JD, Hong ZY, Xu JD, Jin XM, Tang JM. 1997. Umbilical cord blood lead levels in Shanghai, China. *Biomed Environ Sci* 10(1): 38-46.

Singh J, Dutta AK, Khare S, Dubey NK, Harit AK, Jain NK, Wadhwa TC, Gupta SR, Dhariwal AC, Jain DC, Bhatia R, Sokhey J. 2001. Diethylene glycol poisoning in Gurgaon, India, 1998. *Bull World Health Organ* 79(2): 88-95.

Suplido ML, Ong CN. 2000. Lead exposure among small-scale battery recyclers, automobile radiator mechanics, and their children in Manila, the Philippines. *Environ Res* 82(3): 231-238.

The World Bank. 2000. *Attacking Poverty.* Oxford University Press, Inc.: Washington D.C.

Tinoco-Ojanguren R, Halperin DC. 1998. Poverty, production, and health: inhibition of erythrocyte cholinesterase via occupational exposure to organophosphate insecticides in Chiapas, Mexico. *Arch Environ Health* 53(1): 29-35.

van Straaten P. 2000. Human exposure to mercury due to small scale gold mining in northern Tanzania. *Sci Total Environ* 259(1-3): 45-53.

Villanueva SF, Botello AV. 1998. Metal pollution in coastal areas of Mexico. *Rev Environ Contam Toxicol* 157: 53-94.

von Schirnding Y, Bradshaw D, Fuggle R, Stokol M. 1991. Blood lead levels in South African inner-city children. *Environ Health Perspect* 94: 125-130.

Wang JD, Soong WT, Chao KY, Hwang YH, Jang CS. 1998. Occupational and environmental lead poisoning: case study of a battery recycling smelter in Taiwan. *J Toxicol Sci* 23 Suppl 2: 241-245.

Wesseling C, Castillo L, Elinder CG. 1993. Pesticide poisonings in Costa Rica. *Scand J Work Environ Health* 19(4): 227-235.

Woodward R and Clarke E. 1997. Voluntary cessation of manufacture of benzidine dyes, in: Organization for Economic Co-Operation and Development, *Proceedings of the OECD Workshop on Non-regulatory Initiatives for Chemical Risk Management*. OECD: Paris, p. 25.

World Health Organization. 1990. *Public Health Impact of Pesticides used in Agriculture.* WHO: Geneva.

World Health Organization. 2000. *The World Health Report 2000*: Annex Table 3. WHO: Geneva.

Design: studio grafik, Herndon, Virginia

THE WORLD BANK

1818 H Street, NW

Washington, DC 20433