SILENT SCOURGE

Children, Pollution, and Why Scientists Disagree

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CHAPTER 1

Lead and the Roots of Environmental Controversies

How exposure to lead affects children's development has been studied extensively in the last 20 years. There is evidence that exposure to lead lowers children's IQ test scores and raises the likelihood of restless and inattentive behavior. But the history of lead in the United States brings us face to face with this question: what decision standard do we use to regulate a new technology that has uncertain risks and benefits? Lead also provides a striking example of how scientists clash over environmental issues, with experts on opposite sides drawing different conclusions. A major part of the scientific conflict is rooted in the decision standards adopted and in the assumptions on which the research is based. Those decision standards and assumptions involve implicit value judgments that affect the conclusions. The conclusions scientists draw ultimately affect the pollution prevention and cleanup decisions of our governments.

Lead also tells a tale of dramatic social injustice: African American children in low-income families in the United States have the highest exposure to lead of any segment of the American population. But the scientists argue about whether lead exposure is a cause of lower IQ test scores and behavior problems or whether it just appears that lead exposure has negative effects on children because lead exposure is higher in socially disadvantaged circumstances. The conclusions a researcher draws about whether lead causes problems in children depend critically on the scientist's assumptions about IQ test scores and what influences them. So,

en route to learning about how lead affects children's development, this chapter also involves decision standards in science and shows how unquestioned, status quo viewpoints within science influence scientists' interpretations of their research results in very important ways.

A BRIEF HISTORY OF LEAD EXPOSURE AND REGULATION IN THE UNITED STATES

Alice Hamilton, M.D., "Social Reformer"

Lead has been known to be poisonous since the Roman period, when Pliny the Elder wrote about it. But factory workers after the industrial revolution were exposed to higher levels of lead in greater numbers than before. Alice Hamilton, M.D., was an early worker in the battles for worker safety. Figure 1.1 shows the postage stamp issued in her honor. Starting in 1910 she systematically studied lead exposure and its effects in industrial workers (Lippmann, 1990).

Dr. Hamilton and her team asked to enter the production facilities of companies where they suspected lead exposure in the workers. In her autobiography, *Exploring the Dangerous Trades*, she described the reaction of Edward Cornish, who later became president of the National Lead Company. Mr. Cornish denied that his men were "leaded," and he issued this challenge to Dr. Hamilton: if she could prove to him that the work environment was damaging the health of his workers, he would do whatever she said to prevent that lead poisoning, including hiring doctors.

After surveying local hospitals for cases of "plumbism" (lead poisoning), Dr. Hamilton presented Mr. Cornish with records of 22 verified cases of his own workers who had been lead poisoned (see table 1.1 for symptoms of lead poison-

Figure 1.1. Postage stamp honoring Dr. Alice Hamilton

Table 1.1 Effects of Acute Lead Poisoning

Fatigue
Discolored (blue-gray) teeth and gums
Jaundice
Colic
Numbness
Trembling and lack of motor control (palsy)
Muscle cramps and contractions
Pain in the extremities
Paralysis and loss of sensation
Hallucinations
Kidney failure
Seizures and coma
Death

ing). Under Cornish's leadership, the Sangamon Street works of the National Lead Company took steps that were unprecedented at that time to prevent workers' exposure to lead dust and fumes. The company had its engineers invent methods for confining dust and fumes and then used the same methods at other plants in the Chicago area. Dr. Hamilton wrote, "I have met many admirable men in industry throughout these thirty-two years, but my warmest gratitude and admiration goes to Edward Cornish" (quoted from Lippmann, 1990, p. 3).

Mr. Cornish, acting on the advice of Alice Hamilton, was clearly a pioneer in worker safety. But the advent of tetraethyl lead in gasoline posed other challenges to industry leaders as well as public health physicians like Dr. Hamilton.

1926: Tetraethyl Lead in Gasoline Gets OK from Surgeon General

The battle over lead in gasoline began prior to any specific federal legal authority to regulate pollution. In some ways this struggle established precedents for how pollution is regulated. There were two clear sides: some argued that exposure to lead in gasoline was not enough above background to be harmful, whereas other scientists argued that lead from gasoline was a major pollutant and a public health hazard (Rosner & Markowitz, 1985).

In February 1925 Dr. Hamilton wrote to the Surgeon General suggesting an impartial investigation to consider whether tetraethyl lead in gasoline was a health hazard. Other doctors also wrote letters at this time. The public health doctors were extremely alarmed about the number of lead poisonings and deaths that were happening in the plants that had just begun producing the lead additive about a year before. In October 1924 at the Standard Oil Company's tetraethyl lead labs in New Jersey, 35 of 49 workers became severely lead poisoned, with palsy (muscle tremors) and hallucinations. Five workers died. Other workers handling tetraethyl lead had died in the plant in Dayton, Ohio, and at the DuPont plant in New Jersey. In May 1925 the *New York Times* reported that 300 workers at the DuPont plant had been poisoned and that 8 had died. The *Times* said the

workers called the tetraethyl lead facility the "House of Butterflies" because those who worked there, even just repairing the equipment, were often poisoned to the point of hallucinations (Rosner & Markowitz, 1985).

As a result of the outcry from public health doctors like Alice Hamilton, the Surgeon General held a conference in May 1925. At the conference at least three people spoke strongly against adding lead to gasoline: Dr. Hamilton, Professor Yandell Henderson of Yale University, and Dr. David Edsall, dean of the Harvard Medical School. Hamilton said, "You may control conditions within a factory, but how are you going to control the whole country?" Henderson expressed his belief that lead was a public hazard on the same scale as a serious infectious disease. He thought that lead combustion in the engines of automobiles would cause lead to fall from the air into every major city in the country: "Conditions would grow worse so gradually and development of lead poisoning will come on . . . insidiously . . . before the public and the government awaken to the situation" (Rosner & Markowitz, 1985). The argument in favor of the use of tetraethyl lead was that it increased engine efficiency and would extend scarce gasoline supplies. Its supporters called its invention a "gift from God" (Rosner & Markowitz, 1985).

The majority of those who attended the conference concluded that tetraethyl lead should not be banned unless there was *proof* that it was a public health hazard. Research was obviously needed, so a blue ribbon committee of public health experts and industry representatives was appointed by the Surgeon General to carry out some research and produce a report. The Surgeon General's blue ribbon committee issued its report seven months after the conference. The report concluded that "there are at present no good grounds for prohibiting use of ethyl gasoline . . . provided that its distribution and use are controlled by proper regulation" (Rosner & Markowitz, 1985, p. 350). But the committee also issued a warning that the hazard from lead could grow over time and that as the number of automobiles in the country increased, it would be very important to study the possible effects of lead on a continuing basis. The committee suggested that funding from Congress be requested to conduct such studies (Rosner & Markowitz, 1985). Unfortunately, such follow-up assessments were not conducted.

A One-Sided Decision Standard: Benefits Supersede Risks?

A more important outcome of the Surgeon General's conference than the blue ribbon committee was what some call the "Kehoe Paradigm" for environmental decision making (Nriagu, 1998). Dr. Robert Kehoe was a physician on the faculty at the University of Cincinnati who worked as a consultant with the lead industry and became director of the Kettering Laboratory. The Kettering Laboratory was funded by the lead industry and conducted research on lead and its effects.

At the Surgeon General's conference Kehoe proposed that any decision about tetraethyl lead be based on facts already known: "If it is shown . . . that an actual hazard exists in the handling of ethyl gasoline, that an actual hazard exists from exhaust gases from motors, that an actual danger to the public is had as a result

of treatment of gasoline with lead, the distribution of gasoline with lead in it will be discontinued" (Nriagu, 1998, p. 73). In contrast, the medical director of a New York City hospital, Dr. Touart, stated part of what is now called the "Precautionary Principle": "It seems to me that perhaps the attitude should be taken that this ethyl gasoline is under suspicion and therefore should be withheld from public consumption until it is conclusively shown that it is not poisonous" (quoted in Nriagu, 1998, p. 73). Notice that the Surgeon General's special committee implicitly adopted the Kehoe Paradigm: they recommended that *until* there was evidence of harm, tetraethyl lead should be allowed to go forward.

Let's restate the two positions and analyze them a bit.

Position 1 (an early version of the Precautionary Principle): Tetraethyl lead should be banned, until it is shown to be safe, on the basis of the known toxicity of lead to humans, the likely wide exposure of human populations to lead as a result of burning it in gasoline, and the likely harm that would result from widespread environmental contamination with a known toxic element.

Position 2 (the Kehoe Paradigm): Tetraethyl lead should be allowed unless and until it is shown to be a health hazard in the general population because there are benefits of its use, and there is uncertainty about both the amount of exposure that would occur from burning it in gasoline and the effects of that exposure.

Position 1 has three elements: the known toxicity of the substance in high doses, the likely exposure of large populations to uncertain doses, and the potential damage to large populations. Position 2 also has three elements: the known benefits of the new technology, the unknown amount of exposure of the general population, and the unknown effects of exposure on the general population.

Notice that these two viewpoints implicitly weigh the unknowns of the future in dramatically different ways. Both positions contain strong *implicit value judgments* of the benefits of technological innovation in comparison with the uncertainties of negative health impacts. Position 1 says that uncertain harm should be prevented. Position 2 says that a potential benefit should be grasped. Position 2 gives no importance to uncertain future harm. An important point to realize is that benefits of new technologies are often assumed to be a certainty, as stated in Position 2. Future benefits are not usually subjected to the same scientific scrutiny that possibilities of future harm are. A final important point is that the two positions differ not so much in their *scientific* evaluations as in the *personal moral values* they express. However, the difference in personal values is a bit veiled. Kehoe once objected that those opposed to the use of tetraethyl lead wanted *proof* that it was safe but that the opponents did not say what would constitute proof. On the other hand, the Kehoe Paradigm (Position 2) also requires proof of harm without providing a standard. These are starkly different decision standards.

We see these two contrasting viewpoints echoed in many current controversies over new technologies, from cell phones to genetically engineered foods. And we see the arguments deflected from the basic value differences and instead placed on the science of estimating the amount of harm at different exposures. Each

side in an environmental controversy will find an expert to provide an interpretation of the scientific evidence that is favorable to its own side. I hope to show you why scientists can disagree about the scientific evidence without being dishonest: they are usually applying different decision standards. But as you will see, the scientific controversies surrounding the effects of low-level exposure to lead are particularly fierce, and they continue right up to the present time.

1965: Clair Patterson Challenges the Scientific Status Quo

Prior to approximately 1965, the lead industry funded most of the research on lead and lead poisoning through the Kettering Laboratory. Dr. Kehoe described the Kettering Laboratory: "This laboratory is the only source of new information on the subject, and its conclusions have wide influence in this country and abroad in shaping the point of view and activities with respect to this question" (quoted in Nriagu, 1998).

Kehoe apparently thought lead in gasoline was not dangerous. He measured lead in workers who did not directly handle tetraethyl lead, and he found that they had rather high lead concentrations, though they were not visibly poisoned. He assumed that lead was normally present in humans, without realizing that these workers had been exposed by inhaling fumes. Later he studied people in a remote area of Mexico and found that they too had a relatively high level of lead in their blood. But these people too had already been exposed to lead because they used tableware made with lead glazes. Kehoe reasoned that because symptoms of lead poisoning were not apparent in either the workers or the Mexican villagers, humans must have adapted to having a certain level of lead in their bodies: he concluded that lead exposure was *natural*. He thought there was a kind of equilibrium level for lead absorption and that when higher lead exposure occurred, more lead was eliminated from the body. Poisoning would therefore be rare. In short, he argued that there was a clear threshold for lead poisoning and that the human body had a natural defense against reaching that threshold (Nriagu, 1998).

It was not until 1965 that Kehoe's evidence and arguments were seriously challenged. Dr. Clair ("Pat") Patterson, a geochemist at the California Institute of Technology, was the scientist who did it. He was studying the age of the earth and began studying lead pollution because it was important to eliminate it from his laboratory measurements. Working in the 1950s in southern California, where smog was a health threat, Patterson found that lead in the air, especially from leaded gasoline exhaust, was the root of lead contamination in his lab. Careful attention to lead pollution in the air allowed Patterson to make a scientific breakthrough in 1955: the earth was more than a billion years older than scientists had thought. Previous researchers had conducted their measurements in lead-polluted air (Nriagu, 1998; Patterson, 1965).

In 1965 Patterson published a paper in the *Archives of Environmental Health* in which he distinguished between "natural" and "contaminated" sources of lead in

the environment and in people. Because his conclusion conflicted with the widely accepted viewpoint of Kehoe, and because Patterson was a geochemist and not a medical doctor, Patterson's article was met with ridicule, anger, and skepticism. Patterson had challenged a central point in Kehoe's case for the safety of lead: that a high background level of lead exposure is "natural." The ensuing controversy resulted in loss of Patterson's research funding, and some members of the Board of Trustees of the California Institute of Technology requested that he be fired. But Patterson continued to pursue his research. He analyzed deep ice from the polar regions and also estimated lead contamination in preindustrial humans by using bones from museums. He showed that lead levels in Americans were approximately 500 to 1,000 times the lead concentrations found in ancient humans (presently Americans have approximately 100–200 times ancient exposure). Patterson had shown that Kehoe was flat wrong about what "normal" was (Nriagu, 1998).

In 1966 both Kehoe and Patterson testified in U.S. Senate hearings on the first Clean Air Act. Patterson bluntly pointed out that some of Kehoe's data on lead in the air in major U.S. cities were erroneous. More important, Patterson also pointed out the need to separate the scientific research itself from the *use* of scientific research to advocate a particular position: "It is clear, from the history of development of the lead pollution problem in the United States that responsible and regulatory persons and organizations concerned in this matter have failed to distinguish between *scientific activity* and the *utilization* of observations for a material purpose. [Such utilization] is not science. . . . It is the defense and promotion of industrial activity. This utilization is not done objectively. It is done subjectively" (Patterson, quoted in Nriagu, 1998, p. 76, emphasis added; see also Nriagu, 1998; Flegal, 1998; Needleman, 1998a, 2000).

The conflict between Patterson and the status quo position of Kehoe shows the importance of distinguishing between scientific results themselves and the uses of scientific results to advocate certain public policies. In the expert testimony of a scientist, one ought to be able to find the line between the findings themselves and inferences and conclusions relevant to policy that are drawn from the findings. Regulatory decisions are political and value-laden by their very nature. I will leave to the reader's judgment the question of whether the regulatory process follows the legacy of Kehoe's paradigm of insistence on a "smoking gun" of evidence of harm prior to regulation, especially for new technologies. In the last chapter of this book I'll pull together some issues on how we manage societal risks in the United States, and the implications of scientific disagreements for what has become known as the Precautionary Principle (see chapter 7).

Lead Exposure, Government Regulation, and Child Development

Lead in the environment comes not just from leaded gas but also from paint and other sources. Let's take a brief walk through time and consider some other key events with lead. A time line of key industrial, government, and medical events relevant to lead in paint and gasoline is given in table 1.2.

Table 1.2 Time line of events in the modern history of lead

1904–1927	Numerous medical articles published documenting lead poisoning in both children and workers
1909	France, Belgium, and Austria restrict use of lead paint indoors
1910	Dr. Alice Hamilton convinces Edward Cornish of the National Lead Company to protect workers' health by controlling dust and fumes in the factory
1922–1931	Great Britain, Sweden, Poland, Czechoslovakia, Spain, Yugoslavia, Tunisia, and Greece restrict use of lead paint indoors
1922	Third International Labor Conference of the League of Nations recommends banning lead in indoor paint
1920–1929	Dutch Boy paint promotes the use of lead paint, including around children, and issues "paint booklets" for dealers to give away to children
1925	Surgeon General's conference on leaded gasoline
1928	Lead Industries Association (LIA) formed
1933	Dr. Robert Kehoe endorses eliminating lead from children's environments
1938	LIA launches campaign promoting lead paint inside low-cost housing, schools, and hospitals
1939	National Paint, Varnish and Lacquer Association suggests that members voluntarily label toxic materials
1943	*Time* magazine covers research of Byers & Lord linking lead poisoning to later poor school performance and behavior problems
1945	California enacts lead paint labeling regulations
1949	Maryland enacts lead paint labeling regulations, shortly repealed
1952	LIA publishes book, "Lead in Modern Industry," stating that lead paint has "practically no undesirable qualities"
1954–55	Research shows lasting aftereffects of childhood lead poisoning
1954	AMA recommends that lead paint be labeled "poisonous" and not for interior use or around children
1954	New York City enacts regulation limiting lead to 1% in paint
1955	ANSI adopts voluntary limit of 1% lead in interior paint
1955	*New York Daily News* reports that 10 Brooklyn children died of lead poisoning during the preceding year
1955	Dr. Clair "Pat" Patterson publishes research revising the estimated age of the earth; results required eliminating lead pollution from his lab
1956	*Parade* magazine and CBS TV cover childhood lead poisoning
1957	City of Baltimore screens homes for lead paint
1959	Ethyl Corporation seeks permission to raise lead to 4 cc/gallon in gasoline
1959	Surgeon General convenes a committee to evaluate the health effects of atmospheric lead
1961	City of Baltimore Health Department suggests removal of lead paint from housing in areas of the city with high rates of lead poisoning
1965	Dr. Clair Patterson publishes paper questioning "natural" level of lead in the environment and people
1966	Senate hearings on Clean Air Act include testimony from Robert Kehoe and Clair Patterson

Table 1.2 (*continued*)

1970	Congress passes Lead Paint Poisoning Prevention Act. Use of lead paint inside federally funded housing is prohibited; federal funds are allocated to study health impacts of lead on children. Lead in paint to be phased out.
1970	Congress passes the Clean Air Act. EPA fails to write rules regulating lead in air.
1970	Surgeon General calls for early identification of children with lead exposure > 40 μg/dl
1970	To meet air pollution regulations for hydrocarbons, General Motors announces it will begin installing catalytic converters that require lead-free gas
1971	New York City Health Department tests 76 kinds of paint and finds 8 with lead between 2% and 10%
1972	EPA issues rules requiring each gas station to have one lead-free pump
1973	Natural Resources Defense Council sues EPA for failing to issue air pollution regulations on lead. Appeal Court upholds suit.
1973	EPA issues first air pollution regulations for lead in gasoline. Ethyl Corp. and DuPont sue to prevent enforcement. EPA wins in Supreme Court appeal.
1976	California Air Resources Board sets standard for lead in air at maximum of 1.5 μg per cubic meter
1976	Natural Resources Defense Council wins another suit against EPA over failure to set an air pollution standard for lead
1977	EPA endorses 1.5 μg / cubic meter standard for lead air pollution
1978	Lead in paint prohibited
1979	OSHA limits lead dust in workplaces to 40 μg per cubic meter, and workers' blood lead to 50 μg/dl
1980	LIA petitions the EPA to rescind lead air pollution regulations
1982	EPA Administrator Ann Gorsuch promises privately not to enforce lead regulations in New Mexico. Bad publicity ensues.
1982	EPA committee appointed to review health effects relevant to air pollution regulations. Claire Ernhart and Herbert Needleman clash as members of the committee.
1986	EPA issues draft of health effects of air pollution
1986	*New York Times* reports that Ethyl Corporation representative said the EPA lead rules were influenced by "rabid environmentalists"
1991	CDC sets 10 μg/dl blood lead as "level of concern" for children and issues strategic plan to end childhood lead poisoning
1992	Title X of the Housing and Community Development Act directs HUD to make recommendations on lead paint hazard reduction and financing

Sources: Compiled from Berney, 1993; Flegal, 1998; Markowitz & Rosen, 2000; Needleman, 1998a, 1998b, 2000; Nriagu, 1998; Rosner & Markowitz, 1985

In 1909, before World War I, three European countries restricted the indoor use of paint containing white-lead pigment. This restriction was based on dangers that lead paint posed to workers. Restrictions were imposed by other countries between 1922 and 1931. In 1922, the League of Nations Third International Labor Conference recommended banning the use of white-lead paint indoors. Meanwhile, in the United States, Dutch Boy white-lead paint was heavily advertised by its manufacturer, the National Lead Company, for use indoors, including around children. The Lead Industry Association was formed in 1928 to promote the use of lead in a variety of products and industry. But by the mid-1920s many medical studies had been published documenting lead poisoning in both factory workers and children who chewed paint at home. In 1933, partly as a result of these studies, Robert Kehoe, still working for the Lead Industry Association, endorsed efforts to remove lead from children's environments. But in 1938, the Lead Industry Association seemingly ignored his advice and launched a campaign to promote the use of white-lead paint inside low-cost housing and institutions such as schools and hospitals (Markowitz & Rosner, 2000).

Lead poisoning in the 1930s was thought to be an acute disease from which children would make a full recovery if the source of lead exposure were eliminated (Byers & Lord, 1943). This idea fit Kehoe's theory of lead's toxic effects: the idea that there is a threshold of exposure in order for illness to occur. According to the threshold idea, once a child's lead was brought back below that level, the child would recover. "Normal" lead levels were considered to be anything below about 70 $\mu g/dl$[1] unless accompanied by symptoms. Lead poisoning was diagnosed in small children by the presence of a known source of lead exposure (either from chewing paint off toys, cribs, and windowsills or from breathing lead paint fumes) and symptoms such as abnormal red blood cells, excretion of lead in abnormal quantities in the urine and stools, anemia or noticeable paleness, loss of appetite and vomiting, weakness and uncoordinated muscle movements, partial paralysis, colic (intestinal pain) or serious constipation, hypertension (high blood pressure), headache, seizures, and encephalopathy with swelling of the brain (see Byers & Lord, 1943, for case reports of childhood lead poisoning victims).

The idea that childhood lead poisoning was only an acute illness changed in 1943. Randolph Byers, M.D., and Elizabeth Lord, Ph.D., are credited with being the first researchers to follow lead poisoned children for several years to see how they were doing in school and in their overall adjustment. They reported a study in which they tracked 20 lead poisoned children who had been discharged from a Boston hospital as "cured." They gave the children IQ tests and also looked at their school progress and any reports of difficult behavior problems. Byers and Lord found that only one of the children could really be considered to be developing normally and succeeding in school. Five of them were borderline mentally retarded (IQ scores below 85), and some of the other children's IQ test performance declined over the years. Three children had been expelled from schools for serious misbehavior, and two others showed marked restlessness and inattention.

Byers and Lord concluded that lead poisoning had disrupted the normal processes of development. They ended their article with this conclusion: "Lead poisoning is a serious disease developing from entirely man-made hazards, which should be controlled by appropriate legislation" (p. 484).

Time magazine reported Byers and Lord's work, intensifying publicity of the toxic potentials of lead in children (Markowitz & Rosner, 2000). Lead poisoning was no longer only an acute illness; it was now recognized to have deleterious longer-term effects. Other follow-up studies also showed that many lead poisoned children continued to have problems later (Mellins & Jenkins, 1955; see Berney, 1993, for a synopsis).

In 1945, based on the mounting evidence of hazards from lead paint, California enacted lead paint labeling regulations, as did Maryland in 1949 (though Maryland's were repealed). In 1954 New York City enacted a regulation limiting lead in interior paint to 1%. That same year, the American Medical Association recommended labels on lead paint that would read: "WARNING: This paint contains an amount of lead which may be poisonous and should not be used to paint children's toys or furniture or interior surfaces in dwelling units which might be chewed by children." This warning was not adopted verbatim anywhere in the country (Needleman, 1998a). Instead, paint companies adopted warnings that did not use the term "poisonous." In the midst of these events, in 1952 the Lead Industries Association published a booklet on the value of lead, stating that white-lead paint has "practically no undesirable qualities" (quoted from Markowitz & Rosner, 2000, p. 43).

Negative publicity about childhood lead poisoning grew. In 1956, both *Parade* magazine (the Sunday newspaper insert) and CBS TV produced feature stories on lead poisoning in children. In 1955 the American National Standards Association (an industry standards organization) had adopted a *voluntary* limit of 1% lead in paint. But in 1971, when the New York City Health Department tested paints off the shelf, 10% of the brands contained anywhere from 2 to 10% lead, showing that voluntary limits had failed.

Finally, in 1970, during the Nixon administration, Congress passed the Lead Poisoning Prevention Act. It provided money to screen children for lead exposure. Also, the Consumer Products Safety Commission ruled that use of all lead paint must be phased out by the end of February 1978. The phaseout of leaded gasoline followed later under the Clean Air Act. It was a long road from the Surgeon General's meeting in 1925 to the effective regulation of lead in the United States in 1978 (Markowitz & Rosner, 2000).

The Behavioral Scientists Get Involved

In the 1970s research began in earnest on the intellectual and behavioral effects of childhood lead exposure. These studies were spurred partly by the funds Congress allocated for children's lead screening. Studies in the 1970s and early 1980s provided evidence that lead had negative effects on children at lower concentra-

tions than previously thought: values ranging from about 10 to 60 μg/dl, levels that were formerly considered to be normal, or below the poisoning threshold. In 1979, Herbert Needleman, M.D., published a study of approximately 150 children, almost all middle-class white children from suburbs around Boston. He found differences in performance on IQ tests and teacher ratings of attention as a function of lead. The children's blood lead ranged from about 12 to 54 μg/dl (Needleman et al., 1979). These lead levels are high by today's standards but were below the threshold for poisoning at the time. In 1974 Claire Ernhart, Ph.D., and a collaborator published a study based on 80 African American children from Queens, New York City, also showing that high blood lead levels (but below 60 μg/dl) were associated with lower IQ test performance in preschoolers (Perino & Ernhart, 1974). Other researchers in the 1970s produced similar findings.

CONFLICTS ERUPT AGAIN The controversy over lead that started in 1925 with the Surgeon General's conference and continued with the contentious Senate Hearing testimony of Patterson and Kehoe erupted again in the early 1980s. But now behavioral scientists were involved. In 1982–1983 Needleman and Ernhart both served on an EPA panel dealing with the dangers of lead in air. The two researchers disagreed violently about whether subpoisoning levels of lead exposure were harmful to children. Why the conflict?—hadn't both of them found negative effects of lead exposure in the 1970s? Yes, but Ernhart had followed up 63 of the children in her earlier study and had concluded that lead exposure was no longer related to the children's IQ scores when they were 8–9 years old (Ernhart et al., 1981). Needleman was also in the midst of conducting long-term follow-up studies. The two scientists disagreed so strongly about the effects of lead exposure on children that an independent committee was appointed to examine the details of the research that both of them had done (Needleman, 1992, 2000; Scarr & Ernhart, 1996).

Allegations of biased science seemed to linger in the background of the disagreement. *Science* magazine reported that Ernhart's work was partially supported by the lead industry and also pointed out that Needleman's research was probably being gone over with a fine-tooth comb because of the lead industry's opposition to the EPA's cleanup proposals (Marshall, 1983). Ernhart (1993a) later pointed out that Needleman had potential to earn rather large fees as an expert witness and that her grant funding from the lead industry did not begin until 1983, so it could not have biased her 1981 results. Needleman (1998b) later said that his own point of view on lead had been partly shaped by meeting some of the lead-exposed workers from the "House of Butterflies" when he had a summer job at DuPont.

The special committee appointed by the EPA decided that neither Ernhart's nor Needleman's study provided conclusive evidence about the effects of subpoisoning lead exposure on children's IQ scores. The EPA based its decisions to phase lead out of gasoline on other health effects, not those having to do with children's scholastic abilities and behavior (Marshall, 1983).

After the EPA meetings, Ernhart and Needleman published a series of com-

mentaries critical of each other's work (Ernhart, 1986, 1987; Needleman, 1986, 1987), as well as reanalyses of their own earlier data. In 1985 Ernhart published a reanalysis of both her 1974 and 1981 data that changed her 1974 conclusion (Ernhart et al., 1985). In 1974 Perino and Ernhart had written, "While the effects of subclinical lead intoxication may not be noted in the individual cases seen in a pediatric clinic, analysis of group data indicate quite clearly that performance on an intelligence test is impaired" (p. 30). The 1974 publication had been based on Perino's Ph.D. dissertation conducted under Ernhart's supervision (Perino, 1973). But in 1985 Ernhart and her colleagues concluded, "The reanalyses provide no reasonable support for an interpretation of lead effects in these data" (p. 478). Perino was not a coauthor on either the 1985 reanalysis or the 1981 article.

What changed between 1974 and 1985 to make Ernhart change her mind about the effects of lead on children? The main change was that after correction of some minor errors and the elimination of the data of one participant, the statistics no longer met the standard decision cutoff for interpretation as "significant"—5 chances or less out of 100 that the results would occur as a fluke. Instead, the likelihood of getting the results purely by chance was between 7 and 9 chances in 100. Otherwise, the results reported in the 1985 reanalysis were virtually identical to those of 1974. This brings us back to the issue of what decision standards should be applied: Kehoe's or the Precautionary Principle advocated by the other public health doctors.

FALSE POSITIVE AND FALSE NEGATIVE ERRORS IN SCIENTIFIC DECISION MAKING

Those readers unfamiliar with research practices are undoubtedly amazed that the difference between 5 and 7 chances in 100 would make a researcher draw a totally different conclusion. If you are deciding whether to buy a lottery ticket, the difference between 5 and 7 in 100 probably wouldn't make you change your mind. Many other researchers would not have changed their minds in the way Ernhart did. However, if the original results that Perino and Ernhart published in 1974 had not met the cutoff of 5 chances in 100 or less of occurring by chance, they probably would not have been published in a scientific journal. When research is not published, it does not affect policy or health practices.

The principles in this section on decision standards apply to most scientific research, not just behavioral science.[2] First, it is important for the public to realize that it is exceedingly rare for science to yield a totally definitive answer, or proof. If every child who came in contact with the teensiest amount of lead were killed, we wouldn't need scientific research to tell us that. But we live in a world in which events happen with uncertainty, so researchers rely on statistics to help them draw their conclusions.

When any researcher draws a conclusion such as "Substance X *does* 'significantly' harm children's development" or "Taking cholesterol lowering medication gives a 'significantly' lower chance of having a heart attack," statistical analyses are behind it. Of course, some people taking cholesterol medication do have heart attacks, and some children are unharmed by substance X. The code word "sig-

nificant" normally means that the likelihood is only 5 chances in 100 or less that the conclusion "Substance X is harmful to children's development" is a *false positive error*. In environmental research a false positive error is concluding something is harmful when it is not. In the research on cholesterol medication, a false positive error would be concluding the medication helps when it does not. The "5 in 100 or lower" cutoff is a social convention among scientists that is essentially arbitrary. So when Ernhart changed her conclusion based on her reanalyses, she was following the social conventions of science, though more strictly than many others would have. The statistics said the chance of a false positive error was too large to fit the scientific social convention (it was more than 5 in 100) that would allow her to draw the conclusion that lead is harmful to children. So she concluded that "there was no reasonable support for lead effects in these data."[3]

The other kind of decision error is a *false negative error*—in environmental research, a conclusion that "there is no evidence that substance X is harmful to children's development," when it is harmful. In medicine, an example of a false negative error is "The test provided no evidence that you have cancer," when you actually do. False positive and false negative errors have different consequences. The chance of making a false negative error is *not* preset by scientific convention: science emphasizes having a *low preset chance of a false positive error*. This is so that when a scientist does draw a positive conclusion, we know there is only a small chance of it being wrong. That is what science is for: drawing conclusions we can rely on.

The standards in science are more lax for a conclusion that there is "no evidence." In most research, the chance of a false negative error is much greater than 5 in 100. But studies that draw the "no evidence" conclusion are not published very often in the scientific journals. The researcher goes back to the drawing board and tries to figure out a way to do the research better the next time.

In environmental topics, research that finds no evidence of risk obviously does not necessarily mean that there is no risk; the conclusion could be a false negative error. If you are thinking that in environmental topics it would be good to make sure that the likelihood of a false negative error is as low as the chance of a false positive error, some scientists think so too (Cairns, 1999). A research study that is not very sensitive will usually result in the "no evidence" conclusion. A study could be insensitive because the sample size might be too small or the measures used are not well refined, or it might just be sloppy. A study with low sensitivity has a high chance of a false negative error. An insensitive study will usually give us a "no evidence" conclusion, but it will not really tell us anything because the chance of a false negative error is so high.

Here is the bottom line: good science tells us not only the conclusion but also the chance that the conclusion is wrong. For a positive conclusion, the chance of being wrong is preset (less than 5 chances in 100) and is also reported in the published paper. For a negative conclusion, the chance of being wrong depends on a lot of details pertaining to exactly how the study was conducted.

One upshot of the decision standards that scientists normally use is that

when an expert at a public hearing on an environmental controversy says, "There is no evidence that substance X is harmful to people," someone should ask, "What's the chance that conclusion is wrong?" If the scientific expert cannot give at least an approximate answer, or claims to be absolutely certain the conclusion is correct, then he or she is not a very credible expert. A scientist's job is to provide the public and policy makers not just with a conclusion but also with information about the *uncertainty* of that conclusion.

Here is one more complexity about decisions in science: the preset cutoff for a false positive error is indirectly linked to the chance of a false negative error (see appendix for more detail). If a scientist uses a very strict false positive cutoff (say, only 1 chance in 1,000), then the chances of making a false negative error are going to be pretty high. Setting such a strict cutoff means that the researcher would need a very sensitive piece of research in order to reach a positive conclusion. But if he or she did reach a positive conclusion, we could pretty well bank on it because it only has 1 chance in 1,000 of being wrong.

KEHOE'S PARADIGM The Kehoe Paradigm, that leaded gas needs to be proven harmful before it is withdrawn from the market, needs to be considered in the light of the way scientists normally make decisions. The normal standard of "proof of harm" is that the conclusion "Substance X harms the public" should have 5 chances or less in 100 of being a false positive error. The problem is that where there are many studies conducted on a topic (as there are with lead and its effects on children) there will always be some studies that yield the conclusion "The data provide no convincing evidence that substance X is harmful." One very important reason this happens is that not every study is sensitive enough. The proponents of a new technology that pollutes can point to the inconsistencies among research studies as lack of proof of harm. But remember that in most research the chance that a "no evidence of harm" conclusion is wrong is often much higher than 5 in 100. Only if a study is exceedingly well carried out with a very large sample will the chance of a false negative conclusion be smaller than the chance of a false positive conclusion.

Ernhart and Needleman's conflict was based partly on what decision criterion should be used. Had Ernhart decided that it was not too much to take a 7 in 100 chance of concluding that lead is harmful when it may not be, then she and Needleman would have both concluded that subpoisoning lead exposure is harmful to children. But that is not what happened, and the conflict between them did not end.

CONFLICT ERUPTS ONCE MORE After exchanging critical commentaries in the scientific journals, Ernhart and Needleman clashed again in 1990 as expert witnesses on opposite sides in a Superfund[4] case involving lead pollution. The judge ordered Needleman to allow Ernhart and another scientist (Professor Sandra Scarr of the University of Virginia, who was also a consultant to a polluter in the Superfund case) access to his original data in his laboratory. Ernhart and Scarr

apparently found unreported statistical analyses which they thought could change the interpretation of Needleman's results. They also questioned the criteria that Needleman used to include or exclude the data of particular children: were the criteria decided before seeing the results, or afterward? They found a published graph that was slightly in error, and Needleman eventually published a correction (Needleman, 1994). Ernhart and Scarr reported their suspicions of scientific misconduct to the proper authorities, the National Institutes of Health. Any scientist who suspects another of "fudging" results has a responsibility to report it. In a public lecture after bringing the accusations, Scarr said, "We feel there are significant deviations from normal scientific practice here and we feel that the data has been massaged, to put it mildly" (quoted in Needleman, 1992, p. 979).

Needleman was investigated by the University of Pittsburgh and the National Institutes of Health's Office of Scientific Integrity. He challenged the makeup of the panel selected by the University of Pittsburgh to investigate him because he felt it was biased: two members of it were close associates of Scarr and Ernhart. On May 27, 1992, the *Wall Street Journal* reported that Needleman's data were "cleared by the panel" (cited in Needleman, 1992). Needleman was never accused of making up data, only of omitting certain cases and misrepresenting procedures for selecting cases.

The editor of the journal *Pediatrics* wrote, "I am confused. Dr. Needleman believes he has been found not guilty. The government (Environmental Protection Agency) and other scientists also believe this, but others may not (see . . . the preliminary report of the Inquiry Panel). How long must this go on? Has Dr. Needleman been victimized over a difference of opinion about the quality of his science? . . . Conflicting opinions are common and very important in science. . . . Many studies are needed before one side convinces the other that they are right" (*Pediatrics*, 1992).

HOW LONG MUST IT GO ON? Ernhart has also written commentaries criticizing not just the work of Herbert Needleman but of others who conclude that low amounts of lead exposure have an effect on children's intellectual development (Ernhart, 1993a, 1994, 1995a, 1995b, 1996, 1998). For his part, Needleman (1998a, 1998b, 2000) continues to write commentaries emphasizing the importance of reducing children's lead exposure and the benefits that have been realized from reductions in children's lead exposure.

WHAT THE RESEARCH SHOWS

In the meantime, a lot of research on how lead affects children's development has accumulated. By the 1980s there were many long-term studies under way in different parts of the United States and other countries. There are several reviews available (see Bellinger, 2000; Berney, 1993; Schwartz, 1994; Pocock et al., 1994; Smith, 1985). The reviews cover cognitive functioning quite thoroughly (especially

intelligence test scores) but do not cover behavior problems very well. The most recent reviews conclude that there is at least a small effect of low-level lead exposure on children's IQ scores.

My own assessment of the research on lead exposure at low levels is that it does show *at least* small effects of lead exposure on *both* cognitive functioning and children's behavior problems such as inattention, restlessness, and aggression. And in a later section, I will explain why I think status quo thinking has led the researchers to underestimate the effects of lead on children's development.

Before I explain my conclusion, I give an overview of two studies, both from "down under"—New Zealand and Australia. I chose these examples because they meet the highest scientific standards in many ways: they used good sample sizes (approximately 800 and 500 children) and measured other important variables that are presumed to influence children's behavior and scholastic performance. I could have chosen other well-conducted studies as examples, but these studies have not been controversial, and they were conducted away from the heat of the regulatory controversy over lead that has occurred in America.

Christchurch Child Development Study, New Zealand

All New Zealanders in the Christchurch urban area who were born in 1977, more than 1,000 children, were enrolled in a longitudinal study. To measure lead exposure, the children's parents were asked to save one of their child's baby teeth for the researchers. Because lead is stored in our bones and teeth, tooth lead provides a measure of lead exposure over time. The children were tested at 8, 9, 13, and 18 years of age with standardized tests of reading and IQ. Standardized questionnaires were also given to the teachers and to the mothers to measure the children's behavior. The research team also gathered extensive data on other variables that can influence children's school and IQ test performance, such as parent education, family socioeconomic status, ethnicity, and social quality of the home environment. Such family background variables are called either "confounders" or "covariates" because they vary along with the target variable the investigator is interested in: either lead exposure itself or the children's IQ scores, school performance, and behavior. The sample had relatively low tooth lead. Thus, the Christchurch study provides a more conservative test of the effects of low-level lead exposure than studies with higher lead levels, such as Needleman's and Ernhart's studies (Fergusson et al., 1988a, 1997).

COGNITIVE AND SCHOOL PERFORMANCE In the Christchurch study the results were similar for the cognitive and school performance measures. At ages 8 and 9 years, IQ scores were not related to lead exposure, but reading scores and teacher ratings of school performance were. Lead exposure accounted for between about 1% and 0.6% of the differences in the children in the sample in these variables. At age 12, lead exposure accounted for about 0.5% of standardized tests of academic performance and for about 2% of teacher ratings of academic performance.

The researchers in Christchurch also assessed educational outcomes at age 18: number of School Certificate passes, leaving school with or without "formal qualifications," number of years of secondary education completed, standardized reading test scores, and whether the person scored below the 12-year-old level on reading. The results showed that tooth lead at age 7 was associated with all five educational outcome measures at age 18. These results hold even after taking into account all the relevant family background variables.

RESTLESSNESS AND INATTENTION The Christchurch study also found that at age 8 and 9 years, tooth lead was related to both teacher and mother ratings of inattention and restlessness. Again, this relationship held even when background variables were included. Lead exposure was estimated to account for between 3.5% and 0.6% of the differences among children in restlessness/inattention. The lower estimate, 0.6%, is exceedingly conservative because it includes pica (attempting to eat dirt and other inedible things, a source of lead exposure) as a background variable. The results also held at age 12 to 13 years: even after including a multitude of background variables, tooth lead accounts for about 1.5% of the differences among children in attention and restlessness.

CHICKEN OR EGG? Lead exposure and children's behavior is a classic "chicken or egg" problem. Which came first, lead exposure or inattentive and restless behavior? One idea is that children who are inattentive and restless are likely to do things that unintentionally expose them to more lead than children who are less restless. The Christchurch research team tested this statistically. The results showed that between 1.5% and 0.6% of restless behavior is attributable to lead after taking into account the idea that behavior may have come first, and lead exposure may result from behavior. Hence, these estimates of the effects of lead are exceedingly conservative. They are exceedingly conservative in another way: the Christchurch study included not just a history of pica but also "residence in old weatherboard housing" as a background variable. "Residence in old weatherboard housing" is also a source of lead exposure, due to deteriorating lead paint in such buildings. But actual measured lead in the children's baby teeth was associated with behavior at ages 12–13 over and above the kind of housing the child lived in.

RESEARCHERS' CONCLUSIONS In 1988 the Christchurch team drew this conclusion: "We do not believe that this evidence provides a justification for the view that the reduction of environmental lead levels will make a major contribution to the well-being of children. . . . The findings, at best, provide some limited support for the eminently sensible and cautious view that it is wisest to make all reasonable attempts to reduce all sources of environmental lead to a minimum" (Fergusson et al., 1988, p. 823). In 1997 they concluded, "The significance of the present study is that it shows that the harmful effects of early lead exposure are

not short-lived but extend into young adulthood, having impacts on later educational and life opportunities" (Fergusson et al., 1997, p. 477).

The Port Pirie Study, Australia

The Port Pirie study enrolled 90% of children born in the area between 1979 and 1982. There is a lead smelter there, so lead exposure is a concern to the local residents. Children's blood lead was measured at birth in the umbilical cord, and periodically until ages 11–13 years. The average was about 14 μg/dl, just barely below Australia's current action level of 15 μg/dL.

Although the Port Pirie study has shown consistent relationships between lead exposure and cognitive functioning from age 2 to ages 11–13 years, here I will concentrate on the results for children's behavior problems. Children's behavior problems were measured by having the mothers fill out a standardized questionnaire called the Child Behavior Checklist (Burns et al., 1999). The Child Behavior Checklist assesses a wide range of children's everyday functioning, including disobedience, destructiveness, sleeping habits, moodiness, toilet habits, temper tantrums, stealing, anxiety, bad dreams, immaturity, and so on (Achenbach, 1991).

Total behavior problems reported by the mother were related to lifetime blood lead even after including family background variables such as father's and mother's education and employment, mother's IQ test score, and mother's psychological adjustment. The results are shown in figure 1.2. The effects of lead on boys and girls differed in the ways you would probably expect. For boys, lead was associated with aggressive and delinquent behaviors, and for girls it was associated not just with aggression but also with attention, anxiety/depression, social withdrawal, and thought problems. (These "internal" problems such as anxiety, depression, and social withdrawal are usually more characteristic of girls than of boys.)

The Port Pirie research team drew this conclusion: "Overall, the results indicate that any deleterious effect of environmental lead is not likely to be large and that only a small fraction of the overall variation in childhood emotional and behavioral problems can be attributed to past exposure. Nevertheless, the social consequences of such an effect are not negligible. . . . Until such time as compelling evidence to the contrary is available, policy makers should treat low level lead exposure as a potential source of harm to children" (Burns et al., 1999).

Other research supports the conclusion that childhood lead exposure is related to behavior problems. In a predominantly African American sample of 15- to 17-year-olds from the Cincinnati area, delinquency reported by the youths on a questionnaire was associated with lead exposure measured in umbilical cord blood at birth and with lead exposure measured at 6 years of age. Prenatal lead exposures accounted for approximately 5% of the differences among the youths in delinquency (Dietrich et al., 2001).

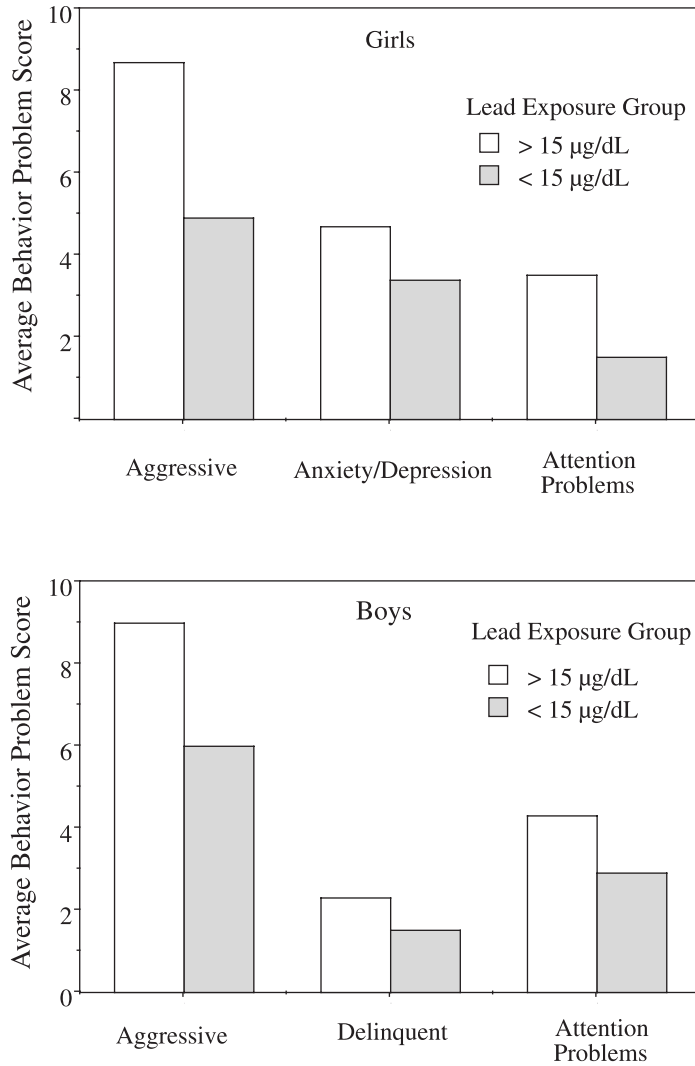

Figure 1.2. Mean behavior problem scores for boys and girls in the Port Pirie, Australia, lead exposure study. Source: Data from Burns et al., 1999.

Are the Effects of Lead Exposure Reversible?

Both the Port Pirie and Christchurch studies showed long-term effects of lead exposure. Does this mean the effects are irreversible? The research does not really tell us. But children with high lead at one point tend to have high lead later in life.

What happens if children's lead exposure is lowered? Although there are controversies, some research on children who have been treated to remove lead from their bloodstream (a procedure called chelation) showed that their attention and restlessness problems were better after treatment (David et al., 1983). Another study with a sample of mostly disadvantaged preschoolers (95% African American and Hispanic low-income children in the Bronx, New York City) showed that their IQ scores went up as their lead levels went down (Ruff et al., 1993). These studies are good news, though there is still some controversy about these treatments. Nevertheless, the results imply that you should do whatever you can to reduce children's lead exposure. The results also bolster the conclusion that enforcement of lead paint removal and abatement regulations is important.

How Much Are Children Exposed to Lead Now, and How Does Exposure Occur?

Another piece of good news is that since lead was phased out of gasoline in the early 1980s, and from paint in 1978, the average amount of lead in people's blood in the United States has plummeted from approximately 16 μg/dl in 1976 to less than 3 μg/dl by 1990 (Pirkle et al., 1994). What a dramatic drop! But lead poisoning still occurs. And the bulk of the research shows that there is not a specific threshold below which lead is known to be safe. Most children's lead exposure is highest (average of 4.1 μg/dl) when they are about 2 to 3 years old, the age when they are walking and starting to talk and are putting almost everything in their mouths if you let them (Brody et al., 1994). So it is important to know the routes by which children are exposed to lead. Sources of lead are: house dust, old lead-based paint in the home, drinking water, lead finishes on older dishes and utensils, dirt outside (especially near the foundations of the houses built before 1978), use of lead in hobbies, and tracking home of lead by family members who work in lead industries or construction trades.

To see how lead gets into children, researchers at the University of Rochester tested children's blood lead and measured lead on the children's hands, lead dust in the home, lead in the soil near the home, amount of lead-based paint in the home, lead in home water, personal housekeeping habits of the family, and whether the child tried to eat dirt. The children were 1 to 3 years old and from Rochester families; about half of those families were single-parent households with income below about $16,000 (in 1991), almost two-thirds were renters, and about 40% of the sample was African American.

The personal characteristic contributing most to blood lead was being an African American; ethnic group accounted for 19% of the differences in children's blood lead (Lanphear & Roghmann, 1997). In their sample the African Americans had lower average income than the whites and were more likely to be renters living in homes with lead paint. Low income and renting one's home were also associated with higher blood lead. Other important variables were the presence of lead paint (which contributes to lead in house dust and lead on children's

hands), concentration of lead in dust, and income. Children who play outside and eat dirt (or try to eat dirt) have higher lead levels, and lead in drinking water in the home also adds a bit to children's blood lead (Lanphear & Roghmann, 1997; Lanphear et al., 1998).

Other researchers have found that maternal involvement with the child is associated with lower lead exposure (Bornschein, 1985). This is probably because parents who are able to be more involved with a child can prevent the child from eating dirt and mouthing dirty objects that may have lead dust on them. And perhaps more attentive parents wash their children's hands more often. But blaming children's lead exposure on parenting is not fair. Today's parents are not responsible for the presence of lead in their homes: if the home did not have lead in it, and in the soil around it, then the children would not be exposed. Public policy that allowed the unregulated use of lead in paint and gasoline for approximately 60 years is what put the lead in the environment of today's children. (See concluding section, "Protect Your Family, Protect Our Planet," for ways to help "get the lead out.")

THE SOCIAL INJUSTICE OF LEAD EXPOSURE IN CHILDREN

The latest national survey of blood lead shows dramatic income and racial/ethnic differences. Figure 1.3 shows the percentages of African American, white, and Mexican American children with different income levels who have blood lead exceeding 10 μg/dl (the current cutoff for "undue lead exposure"). These racial, ethnic, and income differences were also true in an earlier national survey in 1976. This is shocking and a shame to America. The graph shows that children living in poverty have much higher lead exposure than those who are more well-to-do and that the most likely recipients of high lead exposure are poor African American children (Brody et al., 1994). I want to make it perfectly clear that excessive lead exposure does occur in white children from well-to-do families, but it is less likely. And, of course, excessive lead exposure does not occur universally among African American children living on incomes below the poverty level, but it is almost *7 times* more likely than for well-to-do white children.

What are the sources of the racial/ethnic and income disparities in lead exposure? Remember that the Rochester researchers (Lanphear & Roghmann, 1997) found that African Americans in their sample were more likely to have lower incomes and to be living in rental housing with lead paint than nonblacks in their sample. Renters with low income have little control over the condition of the paint in their homes. And I will pose another question: Is there still housing discrimination in parts of America such that African Americans do not have as much choice in housing as people from a European heritage? Two other influential factors are poor nutrition and health care.

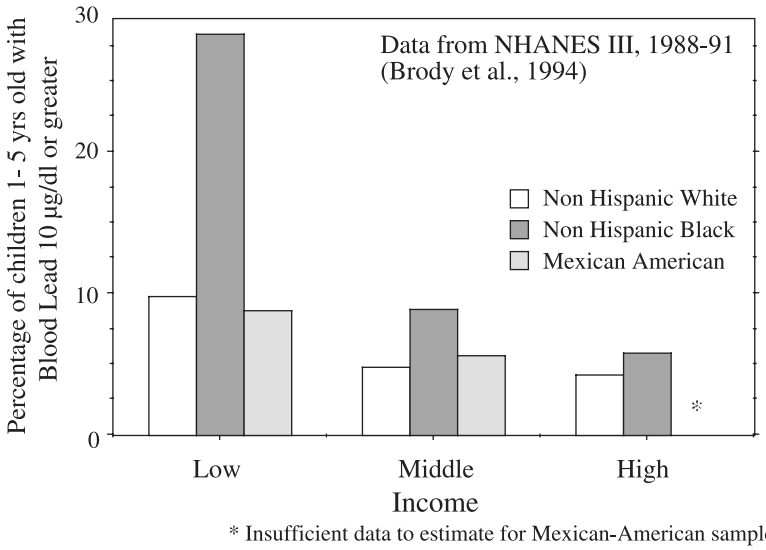

Figure 1.3. Racial/ethnic and income disparity in lead exposure of children 1–5 years old in the United States. Source: Data from Brody et al., 1994.

Lead Paint Abatement Controversy

Research shows that strict enforcement of regulations for lead paint in housing protects residents from lead exposure. Researchers at the Massachusetts Department of Public Health and Harvard University studied two adjoining states where the enforcement policies differed. In the area with strict enforcement, when a child was lead poisoned, the authorities automatically did three things: (1) notified the state lead poisoning prevention program, (2) notified the property owner that there were unsafe levels of lead in the building and that abatement was required or that penalties would be imposed, and (3) notified all tenants in the building that a child had been lead poisoned and informed them of the process for obtaining a lead inspection of their own units. In the area with limited enforcement, the only inspection was of the particular living unit in which the poisoned child lived, no penalties were assessed against property owners, and other tenants were not notified of the presence of lead hazards.

Over a 5-year period, the researchers studied the lead exposure of children 6 years or younger who were living in the same addresses in which a child had previously been identified as lead poisoned (blood lead greater than 25 µg/dL). The results showed that in the limited-enforcement state, addresses with a previously lead poisoned child were 4.6 times more likely to house a child with high lead exposure (> 10 µg/dL) and 6.6 times more likely to have a child with blood lead 25 µg/dL or greater compared to the strict enforcement state (Brown et al., 2001).

The authors concluded, "Public policy is the result of a complex interplay of laws, regulations, and custom. Although policies are implemented across communities, they are designed to influence the lives of individuals. Thus residents are 'exposed' to the public policies in force in their communities. For lead poisoning, these policies include abatement of lead hazards in individual units, property owner liability, notification and referral for services of affected parties, screening, and public education. . . . This study . . . suggested that strict enforcement of lead poisoning prevention statutes is an effective primary prevention strategy" (Brown et al., 2001, pp. 623–624).

As of 2001, an article in a Centers for Disease Control (CDC) publication stated that "deteriorated leaded paint and elevated levels of lead-contaminated house dust . . . are found in an estimated 24 million U.S. dwellings, 4.4 million of which are home to one or more children aged <6 years (U.S. Department of Housing and Urban Development, unpublished data, 2001). Lead hazards are especially common in homes built before 1960 (58%)" (CDC, 2001).

But controversy surrounds how to deal with the lead-based paint hazard in housing. In a discussion paper, Herbert Needleman (1998a) raised the importance of primary prevention of lead poisoning in children. He recounted the reasons he saw for the abandonment of a comprehensive lead poisoning elimination strategy proposed by the U.S. Public Health Service in 1991. The proposed strategy to end lead poisoning had four elements: (1) establishment of child lead poisoning prevention programs in states across the country, (2) abatement of lead paint and dust in older housing considered to be high risk, (3) reduction of lead exposure from sources other than paint, and (4) national surveillance and reporting system for elevated blood lead in children. As part of the plan, the CDC called for blood lead screening of all children 1 to 5 years of age. One reason for widespread screening is that the symptoms of lead poisoning in children even at 50–60 μg/dL—lethargy, sporadic vomiting, and constipation—are nonspecific and easy to mistake for other conditions. Blood lead measurements are the only sure way to diagnose lead poisoning (CDC, 2001).

Needleman's article seems to blame racism (stereotyping lead poisoning as exclusively a problem of poor African Americans), as well as political and industry forces, both within and outside of government agencies, for the failure of the 1991 strategy to go forward. In addition, he chided the Alliance to End Childhood Lead Poisoning (of which he was the founding chairman) for having "adopted a diluted position toward the abatement of leaded properties. Like HUD [Department of Housing and Urban Development], the alliance recoiled from the cost of true abatement. It began to seek avenues of rapprochement with realtors and insurance agencies" (Needleman, 1998a, p. 1875). Needleman also pointed out that the National Center for Lead-Safe Housing (NCLSH) was created by a grant to real estate businesses involved in low-cost housing, along with the Alliance to End Childhood Lead Poisoning, to offer "a real alternative to 'all-or-nothing' solutions [to the problem of lead paint poisoning] that usually mean nothing

gets done to help the millions of children at risk" (quoted in Needleman, 1998a, p. 1875). Needleman comments that the NCLSH focused on the *costs* of abatement and ignored the societal monies that would be *saved* through abatement.

Needleman's 1998 paper was controversial. The journal's editors received an "unusual number" of letters to the editor in response to the article. The journal published six of them, along with Needleman's reply, and the editors' own comments on the nature of controversies in the history of medicine (see Brown & Fee, 1999; Jacobs, 1999; Mushak, 1999; Needleman, 1999; Piomelli & Schoenbrod, 1999; Rosen, 1999; Ryan, 1999; Vernon, 1999). Some applauded Needleman's article, while others were exceedingly critical. Piomelli and Schoenbrod worried that universal screening would cause anxiety in parents. Jacobs commented that "lead hazard control actually costs more than the CDC estimated in 1991" but that "an updated analysis of the benefits and costs of modern lead hazard control still shows a large net benefit of investments to make homes lead safe" (p. 1127). In reply, Needleman (1999) asserted that "if the costs of abatement have grown since the CDC's 1991 estimate, the costs of special education and health care have increased even more sharply" (p. 1130).

The journal's editors summed up their own view of the controversy by saying that "some writers presume that there is one straightforward and objective account of—in this case—the history of lead poisoning prevention policy, on which all right-thinking people would agree. We question this presumption. . . . By and large, professional historians do not seek to present a misleadingly 'objective' review of events because they are quite aware that other equally well-trained historians will soon challenge their construction of events and their claims to objectivity. . . . We believe that such debate can be useful not only for advancing historical discourse but also for clarifying options in policy discussions within public health" (Brown & Fee, 1999, p. 1131).

NAACP Calls for Action on Lead Poisoning

On July 10, 2001, the National Association for the Advancement of Colored People (NAACP) issued a call for action to reduce the "silent epidemic" of lead paint poisoning of children. Kweisi Mfume, the president and CEO of the NAACP, said, "This is an entirely preventable disease. We call on this president and this Congress to take federal action by withholding monies to states that are not complying and following through on basic requirements under the Medicaid Bill and the Medicaid funding that they get. Our actions are not just against the industry but also against states that are violating and have violated federal policy and cities that are violating state policy." Mfume called poisoning due to lead paint "a civil rights issue that affects children of all races, ethnic backgrounds and income levels" (NAACP, 2001).

UNDERESTIMATING THE LEAD PROBLEM WITH STATUS QUO SCIENCE

Scientists still disagree about low-level lead exposure, in spite of all the evidence that it harms children's development. Scientists do agree that lead in high doses is toxic, but they disagree about low doses. Ernhart said in 1986, "Perhaps we will find reliable and consistent effects of low level lead exposure. If so, they will probably be small and of relatively little consequence in the overall complex of conditions that affect children, particularly the disadvantaged children who tend to evidence somewhat higher levels of exposure" (p. 323). This quote by Ernhart succinctly expresses the widely held belief that family background factors like race and ethnicity, income, parental IQ and education, and social quality of the home environment are the most important influences on children's IQ test scores. Because these variables cannot be completely separated from lead exposure, some researchers feel that conclusions that low-level lead causes problems in children are unwarranted. Researchers regularly argue about whether a specific study included proper statistical control of such family background factors so that conclusions about the effects of lead exposure can be valid.

But entrenched ideas about IQ scores are involved too. Published critical commentaries on lead research center mostly on IQ scores, not the behavior problems of inattention and restlessness. Behavioral scientists seem hesitant to draw conclusions pointing to lead exposure as a key influence on IQ.

Here is the standard way researchers think. First, they "know" that a major part of IQ is inherited from the parents (see Neisser et al., 1995, for a readable review of issues surrounding intelligence). Second, they "know" that the social quality of the home also predicts part of the differences in children's IQ scores (Bradley & Caldwell, 1977). Third, they know that socioeconomic status affects children's IQ scores (White, 1982). And other features of life in poverty are related to children's IQ scores, such as changing residence frequently and growing up in a single-parent family or families that have a change of parent figures (McLoyd, 1998). And, fourth, they "know" that on average African Americans score lower than Euro-Americans (Jensen, 1985). Because they know all this, researchers would judge the effect of lead to be important only if it *added* some information about children's IQ scores above and beyond those family background factors.

But wait: are these researchers putting the cart before the horse? The income and racial/ethnic inequities in lead exposure in the United States are huge. So when the researchers consider the effects of family background factors *before* looking for the effect of lead, haven't they really removed most of the sources of lead exposure itself? Lead exposure itself is related to the major family background variables, including socioeconomic status, parent IQ, and social quality of the home.

Here is some evidence that lead exposure itself is related to these family background variables. In one of Ernhart's studies (Ernhart et al., 1989) with a

sample of families in Cleveland (35% African American, and half of them selected for having a history of alcohol problems), she found that up to 13% of the differences in children's lead exposure itself were related to maternal IQ scores, 18% to the social quality of the home environment (including maternal involvement, home organization, and play materials), and 8% to parental education. These results are startling, given that Ernhart and her colleagues found that maternal IQ scores only accounted for about 12% of the differences in the children's IQ scores (a more typical finding is about 25%). In the Christchurch study in New Zealand, the researchers found that maternal IQ scores, social quality of the home, and socioeconomic status were all significantly related to lead exposure (Burns et al., 1999). In Cincinnati, researchers found that up to 21% of differences in the children's blood lead levels were related to their measure of the social quality of the home environment, and 14% of the differences in lead were related to socioeconomic status (Bornschein, 1985).

I want to turn the standard reasoning around and put the horse in front of the cart. Suppose that a lot of what psychologists think they know about children's IQ scores is really analogous to what geologists thought they knew about the age of the earth before Clair Patterson figured out that lead pollution had been spoiling everyone's results. After all, almost all of what behavioral scientists know about the influences on children's IQ scores was found in a lead-polluted environment—between 1925 and now. Remember that when Patterson got the lead out of the air in his lab, his results revised scientists' estimates of the age of Earth enormously.

Here is a revisionist interpretation. First, from the research on lead, we "know" that income and race/ethnicity are related to lead exposure. Look at figure 1.3 again, which shows that poor African American children have 7 times the chance of having high lead compared to whites who are well-to-do. This inequity has been true for as long as researchers have been measuring lead exposure in America, and similar effects occur in other countries for minority groups. What accounts for the disparity in lead exposure? The primary source is poor-quality housing containing deteriorated lead paint. Lower-income children are also more likely to have poor nutrition, which will cause them to absorb more of the lead to which they are exposed, and African Americans are more likely than Americans of European descent to be lactose intolerant. Lactose intolerance means that cow's milk cannot be the major source of calcium, and lower calcium intake is tied to higher lead absorption. Lower-income children are also more likely to be unsupervised or placed in lower-quality day care, or perhaps day care in a building that contains lead paint. Many low-income parents work at two or three low-wage jobs in order to make ends meet, and working so many hours implies an inability to be home to supervise children closely. It is also difficult to breast-feed an infant when working extended hours.

Second, we also "know" that the social quality of the home is related to lead exposure (Bornschein, 1985, Ernhart et al. 1989). Why might this occur? Attentive parents are more likely to stop a child from chewing and mouthing painted

surfaces or dirty objects that contain lead dust. Measures of the social quality of the home also include whether there are appropriate toys and educational materials available. Availability of toys may also help deter children from handling and mouthing lead-contaminated objects and surfaces. Young children examine virtually everything in their environments out of curiosity. In a home with few toys but with lead-based paint, this means that children will likely mouth and manipulate lead-contaminated surfaces. Also, social quality of the home includes how organized the home appears to the researcher. Homes that appear more organized may also be cleaned more regularly, and regular cleaning reduces lead dust in the home (Milar et al., 1982).

Third, we "know" that parental IQ scores and education are related to children's lead exposure (Ernhart et al., 1989; Yamins, 1977; Perino & Ernhart, 1974). A likely cause of this is that smarter, better-educated parents simply know more about the hazards of lead exposure and how to prevent it. Maybe their doctors mentioned it; maybe they learned it from other parents or read about it in a parenting magazine or in a child development course in college. In addition, parent IQ is related to socioeconomic status, which in turn is related to residence in housing with lead paint.

It is as valid to think of family background variables that are related to lead exposure as *causes* of lead exposure itself as it is to think of the same variables as causes of IQ. In fact, when researchers examine the sources of lead exposure itself (rather than IQ scores), this is exactly how they do their research. Figure 1.4 shows a composite conceptual model of lead exposure based on the research conducted in both Rochester and Cincinnati (Lanphear & Roghmann, 1997; Bornschein, 1985). In this diagram, I have drawn all the arrows pointing toward blood lead because lead exposure is the variable being predicted. When researchers are trying to predict lead exposure, the family background variables of race, income, and social quality of the home are conceptualized as potential indicators of or causes of lead exposure.

Now compare figure 1.4 with figure 1.5. Figure 1.5 shows a conceptual model that represents the status quo science approach to finding out whether lead exposure has an effect on children's test scores. In figure 1.5 lead exposure can have an effect on test scores only if it contributes something *over and above* the effects of all the other family background factors. Housing quality is usually included as a family background factor, even though it is an obvious source of lead exposure.

My proposal is that lead exposure should be conceptualized as a cause of lowered IQ scores and altered restlessness, inattention, and aggression and that family background factors should be conceptualized as potential causes of lead exposure. Animal research supports the causal link between lead and altered development. Research in monkeys shows that lead causes learning problems and alterations in other behavior at levels down to about 10–15 μg/dl (comparable to the average levels of exposure in Americans two decades ago) (Rice, 1993). Variables such as poverty, racial discrimination, and social quality of the home are eliminated in animal research.

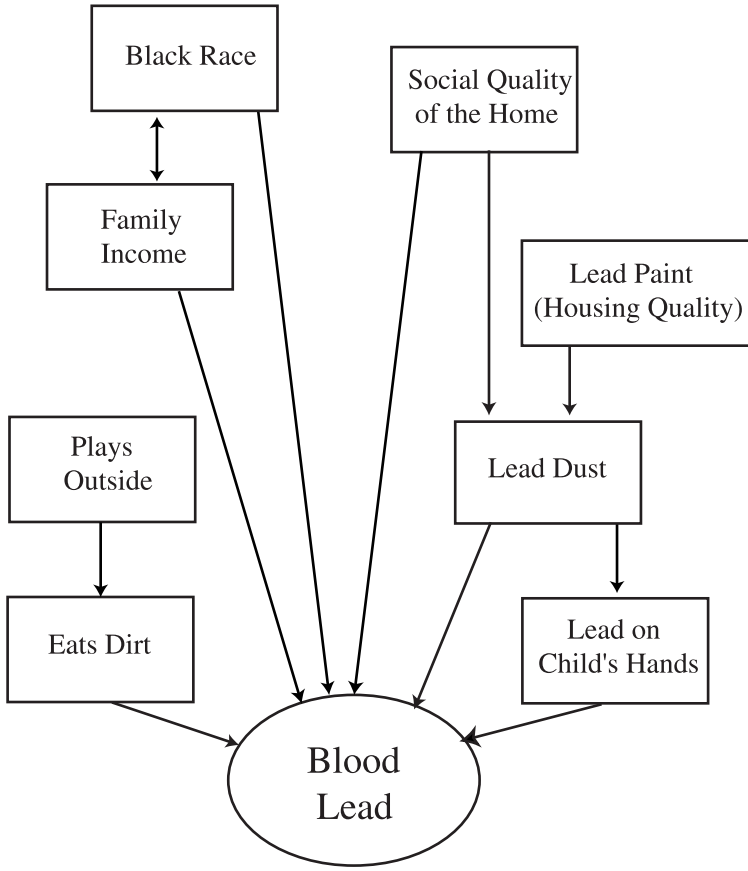

Figure 1.4. Composite conceptual model of lead exposure. *Sources*: Based in part on Lanphear & Roghmann, 1997, and Bornschein et al., 1985.

If examined in the way I suggest, virtually every study of children's lead exposure and IQ scores or behavior would show much stronger effects of lead. For example, in Perino and Ernhart's (1974) study with black preschoolers in New York, preschool lead exposure would account for almost 18% of the differences among the children's IQ scores at age 9, and in Ernhart's (1989) long-term study in Cleveland, lead measured at age 2 (when lead exposure for most children peaks) would account for about 14% of the children's IQ scores at age 4. Many other studies would yield results in the same range as these two examples. The estimates that lead is responsible for about 10–18% of differences among children in IQ scores are quite different from the conclusions drawn from the status quo approach, estimates of less than 1% to about 5% (see the appendix for a brief explanation of how overlapping variables alter correlations).

I do not expect my revisionist interpretation to be accepted by most social scientists: ideas about IQ scores and their heritability are much too entrenched

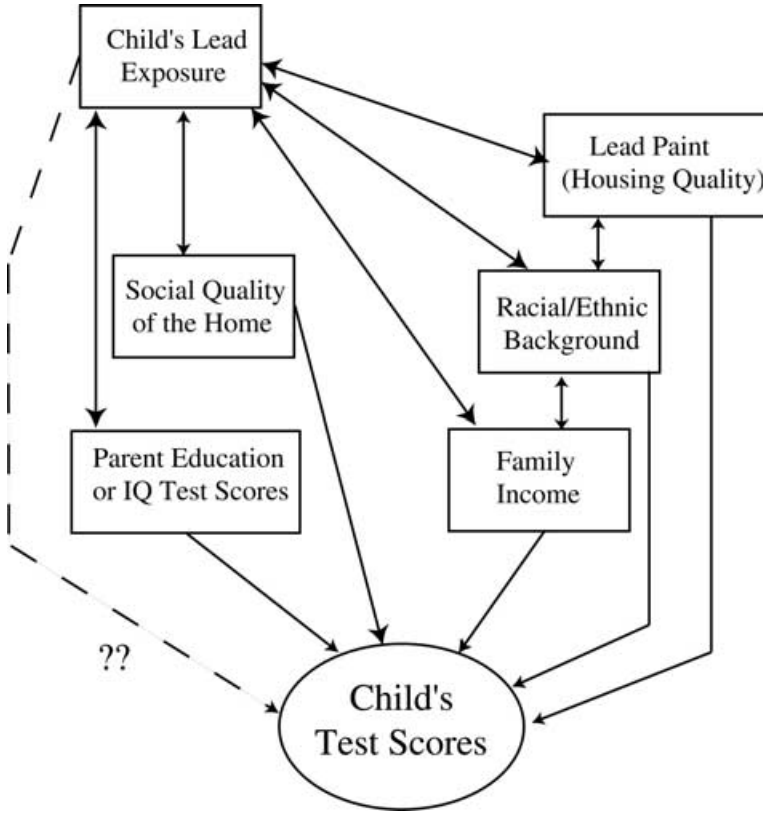

Figure 1.5. Conceptual diaphragm of the "status quo science" estimate of the effects of lead on children's test scores. Lead can influence test scores only after the association of lead with other family background variables is removed.

to be shaken. I am not saying that family background factors are unimportant in child development. Poverty and racial discrimination are unfair experiences that saddle children with many disadvantages. My main point is that we do not know how much of the disadvantage of poverty and race is linked to lead exposure, because lead exposure, poverty, and race are inextricably intertwined. Status quo science about IQ is a major element of the conclusion that lead has only a small effect (see Scarr, 1985, for an excellent discussion of how scientists' beliefs affect the type of research they do).

ARGUMENTS THAT LEAD EXPOSURE IS TRIVIAL, AND COUNTERARGUMENTS

Q1: If lead is so bad, how did people survive such high exposures in the past seemingly unharmed?

A: Many people *didn't* survive, literally. Death rates for lead poisoned children and lead workers were not trivial. In the United States, there had not been a child death attributed to lead poisoning since 1990. But in New Hampshire in March 2000 a 2-year-old girl died from lead poisoning due to peeling lead paint in the home (CDC, 2001).

Q2: But death from lead poisoning is less likely now, so isn't the problem pretty much solved? Isn't fussing over small changes like 1% in IQ scores and children's behavior silly? You cannot look at a child and tell if he or she has a little too much lead, so it must not be a problem.

A: Lead exposure is a "silent" malady, like high blood pressure and coronary artery disease (Bellinger & Mathews, 1998). And because human behavior is so complex and determined by multiple causes working together, doctors or psychologists will never be able to point to a specific behavior in a specific child and say, "See, that behavior shows that child got too much lead." You will never really know if your child's IQ score is just a little lower because of lead exposure than it would have been. Restlessness, inattention, aggression, lowered IQ scores, and poor school performance come from many sources combined. There is a difference between seeing diagnosable effects in an individual child and seeing small effects in a large population of children. Something that affects a large population is a public health problem. As Perino and Ernhart said in their 1974 paper, "While the effects of subclinical lead intoxication may not be noted in the individual cases seen in a pediatric clinic, analysis of group data indicate quite clearly that performance on an intelligence test is impaired" (p. 30).

Here is a way to think about lead as a public health problem. Take the best estimate of how much lead affects children's IQ scores, school performance, or problematic behavior such as aggression, inattention, and restlessness (even based on status quo thinking). Project the changes due to current levels of lead exposure onto the population of the United States. This is exactly what the Center for Disease Control did in 1991. For example, if lead lowers IQ scores even just a little bit, our country is going to end up with more children needing special education. The CDC's 1991 "Strategic Plan for the Elimination of Childhood Lead Poisoning" estimated that each 1 μg/dl reduction in lead exposure would save approximately $2,000 per child in avoided special education and medical costs (Needleman, 1998a). The CDC's cost-benefit estimates showed that removing lead paint from old housing would save more money for the country than it would cost (Needleman, 1998a). And these savings are *just dollars*—there would also be a lot of heartbreak saved for parents who avoid the agony of having a child who just cannot do well in school, has behavior problems, or has developed a seizure disorder after lead poisoning. Heartbreak does not have a dollar value and is not easily accommodated in cost-benefit analyses.

LEAD AND ADULTS

Other health effects of lead exposure are documented in adults. At 10–20 μg/dL lead interferes with the body's ability to synthesize heme, a component of red blood cells. The risk of a pregnant woman having a premature baby increases at 10–20 μg/dl. Nerve conduction slows when blood lead is 30–40 μg/dl, and kidney damage occurs at 40 μg/dl (McMichael, 1995). Higher systolic and diastolic blood pressure is also associated with lead exposure (for a review see Lippmann, 1990). The effects of lead on blood pressure seem to plateau—that is, increases from low lead levels have a larger effect on blood pressure than increases from high lead levels.

In former lead industry workers, the higher the bone lead, the worse the workers performed on tests of manual dexterity, eye-hand coordination, and IQ (Stewart et al., 1999). In that study, the workers had not been exposed to lead for more than 17 years. The effects of lead exposure later in life as people age have not been well studied. Finally, the children of *men* who work in lead industries have been found to be at risk for low birth weight (Min et al., 1996). Low birth weight is associated with a variety of negative developmental outcomes.

LEAD AND WILDLIFE

Lead can be toxic to wildlife, too. Yellowstone National Park banned the use of lead for fishing sinkers because of the effects on wildlife. In 1991, the U.S. Fish and Wildlife Service banned lead shot for hunting ducks and geese. Ducks and geese were being lead poisoned by accidentally eating lead shot off the bottom of wetlands. Researchers tested the lead levels in ducks in 1996 and 1997 and found that lead levels had declined 64% since the lead ban went into effect. The team of scientists estimated that the ban prevented more than 1 million ducks per year from being poisoned by lead. The lead shot ban also indirectly lowers lead exposure in other animals, birds, and people that prey on ducks (*National Wildlife Magazine*, 2001, p. 64). A recent study also suggests that lead shot in mourning doves can be a hazard to hunters who eat them (hunting mourning doves is not permitted in all states). A study of loons in New England found that loons are very susceptible to lead poisoning from eating lead weights attached to fishing tackle (Nadis, 2001).

DECISION CRITERIA AGAIN

Earlier I explained that false positive and false negative errors have different consequences. What if the "best estimate" of how much lead affects children's IQ scores and behavior is a false positive error? Then as a nation we would be

spending money preventing further lead pollution and cleaning up lead (and perhaps forcing some lead-related businesses into bankruptcy) and we would not get any benefits from the cleanup, financial or otherwise. We would spend health care money screening children's blood lead, when that health care money could be spent on something else.

Whenever an agency like the EPA decides to regulate a form of pollution or technology, it is taking the risk of a false positive error. But when regulation is based on sound research, we know the chance of that error, and usually it is much smaller than 5 in 100, often more like 1 in 1,000 or less. Good science tells us not only the conclusion but also the chance that it is wrong.

On the other side, if the currently allowable levels of lead exposure and abatement are a false negative error, the government may be allowing lead exposure at levels that are quite harmful. False negative errors have consequences too, usually for public health. When a government agency decides to allow a pollutant (either a new one or an old one) because it has not been proven hazardous, the risk is a false negative error. False negative errors subject people and the environment to an avoidable hazard while the businesses benefit financially from the product. For leaded gasoline, the companies profited, and the public drove faster, more fuel-efficient cars, but the public also received the involuntary high lead exposure, without sufficient information about the risks. History suggests that when the 1925 Surgeon General's committee decided there was not enough evidence to ban lead in gasoline, it took too high a risk of a false negative error. The only research was hastily done with small samples: it was insensitive research. In 1925, the Surgeon General's committee handled this by asking that follow-up studies be done. Such studies were not carried out. If the United States had signed an international agreement restricting lead paint from indoor use in 1925, many childhood deaths due to lead poisoning would have been avoided, and there would not be an ongoing debate over what to do about old lead paint inside homes built after that date.

Because businesses often bear a major part of the costs of cleanups, businesses want to avoid false positive errors. The public usually wants to avoid exposure to involuntarily imposed hazards (like lead in the air), and it is the government's job to protect the public. But the public seemingly also wants fast cars, cheap gas, paint that covers in one coat, cheap food, and new technologies like cell phones. In order to innovate in business, risks must be taken—both financial and environmental. The issues involved in regulation are inherently value-based. Scientists cannot be expected to tell us as citizens what regulatory decisions to make, because regulatory decisions involve personal ethics and values. In the last chapter of this book, I'll tackle some more complexities of societal risks and examine how values and ethics of both scientists and the public enter into that process. Ethics enter the process even when they are not discussed openly. It is important that the values and ethics be brought out into the open, where all of us can discuss them.

PROTECT YOUR FAMILY, PROTECT OUR PLANET

Lead is a useful industrial metal, but it should be kept out of the environment. Less lead exposure is better. The scientific consensus is that there is no threshold for negative effects of lead. The EPA estimates that almost 1 million American children have lead above 10 μg/dl, the CDC's current level for concern. Here are some tips for reducing your own family's lead exposure and preventing further dispersal of lead in the environment.

1. *Blood testing.* If you live in an older home and have children, or your children go to day care in an older building, *ask your doctor* about testing their lead. This is especially important if you have done remodeling while you lived in the building.

2. *Lead paint and dust* in the home. If you live in a home built before 1978, your home likely has at least some lead-based paint in it. You can test the paint in your home with a kit from the hardware store that costs about $6, but the EPA says those kits are not very reliable. If you have lead paint, dust in your home will contain lead. When you do remodeling projects, protect your family from the dust of the project. The EPA used to recommend mopping washable surfaces with dishwasher detergent because the lead particles stick to it better than to other kinds of soaps (dishwasher detergents have phosphates in them). Now the EPA pamphlet just says to mop with an all-purpose cleaner or water and rinse the mop very thoroughly. *Vacuum* frequently (rather than using a broom or carpet sweeper), and if possible get a HEPA (high-efficiency particle accumulating) vac. If you rent, ask your landlord about lead-based paint, or test it yourself and ask the landlord about doing lead abatement. If you are purchasing a home, ask your seller about lead-based paint, and if possible get the seller to carry out EPA-approved lead abatement.

If you are a do-it-yourselfer in a home with lead paint, during projects such as painting and remodeling in an older home that creates lead dust, make sure you *wash your work clothes separately* from other clothes, and wash your own hands and face with soap before eating. Clean and dust the house much more frequently during projects, *wash children's hands* and faces before snacks and meals, and don't let them play in the work area. Change your furnace filter more often.

3. *Lead in soil* near your home. Plant ground cover to prevent bare soil from being accessible to children and wildlife. If you are a vegetable gardener with an older home or in an older part of a city, have your soil tested for lead. The EPA limit on lead in soil is 400 ppm (parts per million). For housing built before 1978, when you or your landlord paints the exterior, make sure the chips are captured on plastic ground cloths, bagged, and sent to your community's toxic waste collection program. Keep windows closed when lead dust is being produced by exterior painting projects.

4. Lead in *drinking water*. If you live in an older house or older part of a

city, run the water for about one minute in the morning before drinking. Many cities used lead water mains, and many older homes have lead pipe connecting the house to the water main. Older solder on copper pipe contains lead. The good news is that the amount of lead (and other pollutants) permitted in municipal water supplies is regulated by the EPA. (The regulatory standards for bottled water are lax compared to those for city water.) Cities with lead water mains are now in the process of replacing them. Of course, running the water to clear lead has an environmental downside: water conservation is important, even in the water-rich upper Great Lakes area, where I live. Even if the city water supply uses water from wells, as the population and water use grows, wells draw down the water table, and natural springs decrease their flow and can dry up. This affects aquatic ecosystems negatively. If you live in an arid climate, consider a water filter that will remove lead, if your drinking water has too much lead.

5. *Hobbies*. *Fishing weights* are normally made of lead, and those of you who tie your own fishing flies may have lead in your kit. Lead is illegal in Yellowstone National Park. Use alternatives to lead (such as nonleaded wire inside your flies, and steel shot). If you do use lead when fishing, handle these items carefully, do not lick your fingers, and do not use your teeth as pliers to pinch a weight onto your line. The EPA is currently reviewing lead fishing weights.

Handle *gunshot* very carefully if you load your own. Steel shot works for waterfowl. Can you use it for other types of hunting as well? The U.S. military is currently in the process of developing nonleaded ammunition.

Solders for making *stained glass* usually have lead in them. If you make *pottery*, use lead-free glazes. If you *paint*, know your materials and handle them carefully.

6. *Good nutrition*. Maintain a good intake of calcium in your family's diet. Calcium and lead have chemical similarities. Less lead is absorbed if calcium intake is adequate.

7. If you *work* in an environment with lead, use your personal safety equipment properly to reduce your own exposure, wash your hands with soap before eating, and do not bring lead home on your work clothes.

8. *Dispose* of car and motorcycle *batteries*, computers, and electronic appliances in your community's toxic waste disposal and recycling program. Car batteries have lead in them, and the electronics often have lead shielding and lead solders in them.

9. Don't assume that *toys* are automatically safe for children to put in their mouths. Toys in the United States are supposed to be lead-free, but as recently as 1994 crayons made in China were recalled for containing lead (Spears, 1994). The EPA also has notices about vinyl toys and lead pigment on its Web site.

10. Do not use *utensils* made with lead (pewter) or with lead glazing. Check suspicious items with a test kit from the hardware store.

11. *Imported* traditional medicines and foods. The CDC has reported cases of lead poisoning in both children and adults from imported remedies (pills that contain lead) and foods that are brought in by relatives (CDC, 1998, 1999). Be

exceedingly careful about any substance not authorized for import into the United States. In one case, pills imported from Hong Kong were found to contain 1–3 ppm lead, and a woman suffered lead poisoning. In other cases, children who ate candy from Mexico that was in a lead-glazed jar were lead poisoned, and food coloring from Iraq lead poisoned nine members of one family (CDC, 1998).

12. *Advocate and educate.* Advocate for strong enforcement of lead paint abatement in rental housing in your city, county, and state. Advocate for rules against lead in fishing weights. Share information with others, especially those with children living in older homes or apartments. Resist efforts to relax current lead regulations. As crude oil supplies dwindle over the next 20 to 30 years, I personally would not be surprised to see gasoline makers propose putting tetraethyl lead in gasoline again. As Bellinger and Matthews (1998) said, if you wanted to poison people with a toxic substance, what better way than to coat the interiors of their dwellings and distribute the substance broadly in the air? That's what happened with lead.