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A PERSPECTIVE ON THE ENVIRONMENTAL LEAD ISSUE

Attached is an article which puts into perspective the issues and politics surrounding the identification and regulation of environmental sources of lead that I think you'll find interesting. A brief account highlighting Du Pont's position is given on page 33.

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Environmental lead: insidious health problem

Several federal programs aim to reduce human exposure to lead, but which source is most dangerous is subject of growing controversy

Lois R. Ember
C&EN, Washington

She was three years old, and lived in decaying inner-city housing. When she was admitted to the hospital she was stuporous, extremely pale, and her brain was swollen—a condition known as encephalopathy, the result of severe lead poisoning. Her doctor treated her with drugs called chelating agents. And she recovered.

This child was the turning point for Herbert Needleman, now at Children's Hospital Medical Center in Boston. At first, he recalls, "I felt triumphant; I had made a good diagnosis; I had done the right treatment; and I saw the child getting better. Then when I told the mother that her daughter couldn't go back into that home, she asked me: 'Where am I going to move? Every house on the block is the same.' Confronted by the mother's realities, I began to realize that it is not enough just to give a drug. The disease is out there in the world, not just in the child."

Over the past decade, six federal agencies acting under eight separate laws have developed control-strategy regulations, or are carrying on screening programs designed to protect the public from the hazards of lead.

Despite these efforts and a multi-million-dollar research program, a new National Academy of Sciences report finds that "Every member of the general population of the U.S. is exposed to elevated levels of lead in air, drinking water, and foods." Especially at risk are urban children who eat leaded paint chips or lead-laden dust and soil, adults exposed to lead in the workplace, and pregnant women.

Lead is not a natural constituent of the human body, and has no known

role in normal human physiology. Yet, because it has been mined, smelted, and manufactured into useful products for thousands of years, members of industrialized societies are grossly contaminated by this highly toxic metal. Its hazards have been known since antiquity.

Instructions for separating lead from silver ore can be found in the Old Testament. The first clear description of lead colic symptoms comes from the second century BC Greek poet/physician Nicander. Pliny warned of the hazards of breathing lead fumes, but touted the virtues of leaden wine kegs. The Romans mined silver ore for coinage, and used the by-product lead for wine vessels and their intricate plumbing system. In fact, some historians link the fall of Rome to the lower birth rates and madness of the ruling class, the probable result of increased lead intake from wine and water.

Today, U.S. industry consumes 1.3 million tons of lead annually to make such products as batteries, pigments, solders, pottery, and the antiknock agent in leaded gasoline. Smelting and fabricating lead for these products, and burning leaded gasoline to drive our mobile societies can expose workers to high lead levels, and send more than 600,000 tons of lead into the atmosphere to be inhaled or—after deposition on food crops, in fresh water, and on soils and streets—to be ingested by the general population.

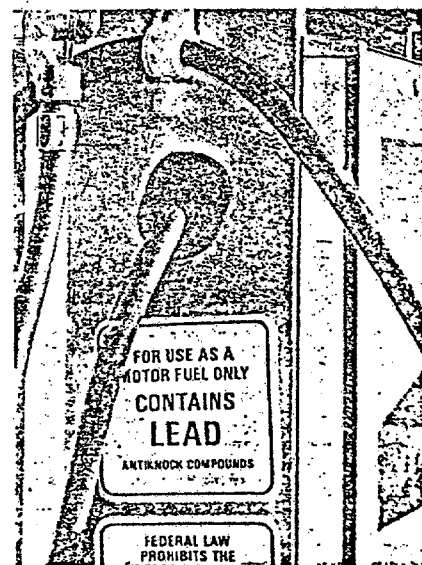
Food also can be contaminated by lead from the solder in tin cans, pesticide sprays, and cooking utensils. In older homes where the plumbing consists of lead pipes, and the water is acidic and low in mineral content, lead may leach into the water supplies. Weathering of lead-laden paint and putty in older homes contaminates dust with lead, which can be inhaled or ingested; chipping, peeling, and flaking paint in these homes may offer a child a tempting but dangerous morsel.

That tempting morsel, once ingested, can result in a very high body burden of lead that can severely damage the developing nervous system. Controlled animal studies have duplicated the neural effects observed in lead-poisoned children.

At the other end of the spectrum, Needleman, in a widely discussed study of over 2000 "normal" first- and second-grade children, was able to relate lead levels in deciduous teeth to behavioral and intellectual problems in the children. He found no threshold, but a continuum of effects. As dentine lead levels increased, performance on standard intelligence, auditory, and language-skill tests decreased—whereas negative behavioral patterns, as noted by teachers, increased.

The laboratory correlate to Needleman's study is Ellen Silbergeld's rat model. At the National Institutes of Health, Silbergeld has developed an animal model of childhood lead poisoning. Her rats receive low levels of lead—levels that would be asymptomatic or subclinical in children—in their drinking water for the first 30 days of life (the equivalent of a child's first five years). On this regimen of lead exposure, the rats display distinct neurobehavioral and learning disorders.

Significantly, Silbergeld has been able to show that in these rats, lead gets inside the nerve cells where it tenaciously remains, first affecting the energy metabolism of the cells, but more important altering the concentration and flow of crucial ions, thereby disrupting the release of



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neurotransmitters—the chemical communication links between nerve cells.

Dovetailed, Silbergeld and Needleman's studies add to the mounting evidence that lead at low levels is a very serious health problem, and one that requires a "strategy of prevention," Silbergeld says.

The social implications of this research are vast. Silbergeld points to the loss of self-esteem in a child behind his age group in school, and the effect that might have on him in later years. Clair Patterson, a California Institute of Technology geochemist, in his minority report to the 488-page NAS study on "Lead in the Human Environment," takes a still wider, more alarmist view. Sometime in the future, he says, the average adult in the U.S. will have been found to "experience a variety of significant physiological and intellectual dysfunctions caused by long-term chronic lead insult to their bodies and minds which results from excess exposures to industrial lead that are 500-fold above natural levels of lead exposure, and that such dysfunctions on this massive scale may have significantly influenced the course of American history." There are no unexposed control groups, he argues.

All is not gloom, however. The federal effort has resulted in a dramatic decrease in the number of cases of severe lead poisoning like that experienced by Needleman's three-year-old patient. But asymptomatic children with blood-lead levels greater than 30 micrograms per 100 ml and elevated red-blood-cell protoporphyrin levels—the marker for

"undue lead absorption"—are prevalent. According to the academy's report, a growing body of evidence "suggests that commonplace exposure to lead in urban environments may be associated with detrimental effects on the intellectual development and behavior of children."

The NAS report attributes the limited results from the federal effort to nonintegration and noncoordination among current laws: No single federal agency is empowered "to deal with total exposure in an integrated fashion." And, as a result, policies are "fragmented and sometimes inconsistent."

To remedy this fragmentation, the 12-member panel of lead experts recommends the token academy solution: Chuck out all the old laws and write a new, comprehensive statute. The logical sequel to this politically naive solution, some suggest, is the formation of a Department of Lead to execute the law.

Short of such sweeping action, the panel suggests the creation of a formal coordinative mechanism such as the Interagency Regulatory Liaison Group. Why this recommendation when IRLG already has a task force on lead, the agencies of IRLG have a common research agenda on lead, and these agencies coordinate regulatory activities? One reason is that the Department of Housing & Urban Development and the Department of Health & Human Services' Center for Disease Control (CDC)—agencies that have responsibility for portions of the lead problem under the 1971 Lead-based Paint Poisoning Prevention Act—are not now, but according to the panel should be, included in IRLG activities concerned with lead issues.

Still another possible solution to the fragmentation between parts of HUD and CDC is a Congressional amendment to the 1971 act that would consolidate authority for decisions about the control of lead paint hazards under one health-oriented regulatory agency. This would remove HUD from the picture, an outcome some in CDC would welcome because they consider HUD's Environmental Hazards Research Program as an obstacle to the protection of children's health.

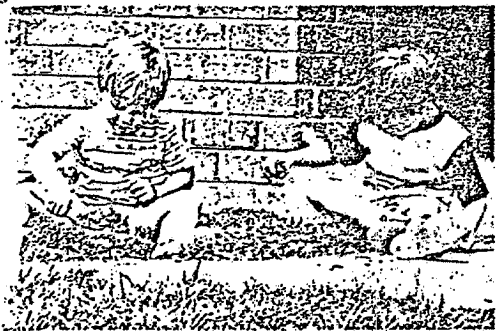
The turf fighting that the lead panel perceived among agencies responsible for solving various parts of the lead problem is a story not found in the academy's report. Lead, it turns out, is not only a metal that is molded into useful products, but it is a mold of activists—a catalyst that transforms scientists and medical researchers into political advocates.

From this activism has come labile alliances and shifting priorities. Abrasive personalities, fragmented mandates, seemingly unsolvable problems, and staggering economics have combined to pit research scientist against medical researcher, agency against agency, industry against government.

This activism has encouraged buck passing, even though the academy legitimized conventional wisdom when it said that all important environmental sources contribute to elevated lead body burdens. For example, the lead industries blame lead in paint as the source of the problem; the food industry cites paint and atmospheric emissions; and the director of HUD's lead research group indicts leaded gasoline, thereby passing the problem on to the Environmental Protection Agency.

The tale began in 1971 when Congress passed the Lead-based Paint Poisoning Prevention Act, and split responsibility for portions of the law between the then-Department of Health, Education & Welfare—which was to search for lead-poisoned children, treat them, and remove the source of lead—and HUD, which was to seek ways to abate and correct the hazards of lead-based paint. HEW/CDC began an outreach screening program in major urban cities. HUD sponsored research on abatement techniques and, with mainly HEW-generated data, began to conduct studies on blood-lead levels and the environmental sources of lead.

The HEW-gathered data came from the screening programs, and were never meant to be used as research data and, according to CDC's director of environmental health services Vernon Houk, "cannot be used for that purpose." Nevertheless,



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Dark areas are lead deposits in mitochondria of neonatal rats

these were frequently the only blood-lead data available to HUD, and they are, in the main, the data that Irwin Billick, then director of HUD's lead-based paint poisoning research and now director of HUD's environmental hazards research, used in his quest for an association between a specific action and a decrease in blood-lead levels.

In his studies, Billick finds correlations between air-lead levels and blood-lead levels, and between gasoline-lead levels and blood-lead levels, but not between lead in paint and lead in blood. In fact, he says, in manipulating the available medical data on children, he has found "inconsistencies with the generally accepted wisdom that lead in paint is the major cause of lead in blood."

For example, he cites unpublished



Iron and lead are contained in this auto exhaust particle (magnified 1200X)

HEW data that show that if lead paint is taken off the walls, children's blood-lead levels will go down in 75% of the cases and up in 25% of the cases, but the same percentages hold if the paint is not taken off the walls. He further cites preliminary data from a HUD-sponsored New York City study indicating that blood-lead values appear to be unaffected by the different intervention techniques tested, including total lead paint removal.

More to the point, Billick admits to C&EN that he does not feel that lead-based paint is a health hazard to children, but he emphasizes that he is speaking for himself and not for his agency. Furthermore, he believes that the correct government action would be one that lowered blood-lead values for the least cost.

His studies have convinced him that taking leaded paint off the walls of old housing stock may not be the most efficacious expenditure of taxpayers' dollars. From his studies on leaded gasoline and blood-lead levels, and his dedicated promotion of these studies, some have come to believe that Billick believes most children would benefit from removing lead from gasoline, whereas only a few children would benefit from solving the lead-paint problem; therefore, the "biggest bang for the bucks" would be achieved by pursuing the former strategy. Billick, however, is very careful not to state explicitly this implication, sidestepping the issue by saying that it is a "political decision" outside his bailiwick.

Billick came to NAS in June 1978 asking for a quick assessment of HUD's seven-year, multimillion-dollar research program, which he hoped to use in his fiscal budget exercise in the fall. According to Ted Schad, NAS deputy executive director of the Commission on Natural Resources, who was present at that first meeting, Billick expressed his belief that lead in paint is not a health problem, and that he hoped the academy's eventual report would support his position. For political reasons, such a finding by the academy would support Billick's efforts to reallocate funds from lead-based paint research to other efforts without raising cries that HUD was abandoning the inner city.

However, Billick was told that even if the academy undertook a study, it could never be completed by the fall of 1978; that any study undertaken would have to address all environmental pathways of lead that affect health; and that there was no guarantee the panel assembled by the academy's Environmental Studies Board would reach the conclusion

that lead-based paint was not a health risk to children.

Billick accepted these conditions, and in late June 1978, when HUD defined the scope of work and supplied \$254,000 to support the effort, NAS began the study. In December 1979, HUD received the completed report, and was sorely dissatisfied with it. So much so, that Billick and his colleague V. Eugene Gray have resorted to attempts at discrediting the report before it receives wide circulation.

Several weeks ago, a scathing, impolitic internal memorandum written by Gray to Billick just "happened" to fall into the hands of the Associated Press. According to Billick, the AP story—which contained such Gray rhetoric as, "I am at a loss for words to describe my nausea" and "This report is a good example of the intellectual masturbatory activities engaged in by NAS at federal agency expense"—was printed in 100 newspapers.

This leak was timed to coincide with the academy's woes over the widely criticized, controversial Food & Nutrition Board's "Toward Healthful Diets."

Earlier, C&EN was given Gray's memo to read. His final paragraph clearly reveals the environmental hazards research division's assessment of lead-based paint research. Since researchers are not gleaming anything new from their \$18 million, three-year effort, and "there are very few cases of lead poisoning anymore, let's transfer that \$18 million from lead research to other research on more important problems," Gray writes.

Not all of HUD agrees with Gray. The policy arm, for instance, headed by deputy assistant secretary for regulatory functions Patricia Worthy, will "carry out its statutory and Congressional mandate to deal with lead-based paint hazards." Worthy's group is beginning to develop close working relationships with Health & Human Services disease control centers throughout the country. Under this arrangement, Worthy says, HHS will alert HUD to lead-poisoned children living in HUD-associated housing. HUD then will move in to remove the source of the lead from the children's environment.

This cooperation between HHS and Worthy's group at HUD does not, however, extend to Billick's research group. CDC's Vernon Houk and Billick have been at odds ever since Billick "decided to prove that lead-based paint is no problem, when in fact it is a major problem," Houk says. Houk sharply objected to Billick and Gray's report, "Lead Based Paint Poisoning Research: Review and Evaluation

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1971-1977," which assessed HUD's \$8.8 million research effort, an effort that has now climbed to about \$10 million.

Houk's objections to that report are in accord with Needleman, who in a letter to HUD Secretary Patricia R. Harris wrote that the Billick and Gray document "will serve as a justification for further delay in the removal of lead from housing stock."

Billick and Houk are in agreement that lead is a multisource problem. But they part ways on the significance of lead-based paint. For a discrete segment of children in this country—those living in the so-called poor, inner-city lead belts—the most hazardous high-dose source of lead is paint, followed by soil and dust contaminated from "previous land use, lead-based paint, and fallout from industrial and automotive emissions," Houk declares. However, Billick can find no association between lead in paint and blood-lead levels in the young populations he studies.

According to the former principal staff officer of NAS's lead panel, Edward Groth III, who worked with both men, Houk is less concerned about whether Billick is correct scientifically than he is by the possibility that if Billick is taken as correct politically, children will continue to suffer from lead poisoning, and appropriations to eliminate this hazard will dry up because of the presumption that the problem has been solved.

Although Houk and Billick have disagreed publicly and vehemently, Billick has been able to maintain symbiotic relationships with scientists at the Environmental Protection Agency and staff members on the President's Council on Environmental Quality. His cooperation was so successful with CEQ that he got the



Billick: lead-based paint not a hazard

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staff to report only his data on the relationship of pediatric blood-lead values to lead in gasoline in the council's 10th annual report published in December 1979. Because no other data were reported in the five-page section on lead, the reader is left with the impression that lead in gasoline—rather than lead in paint or lead from both sources—is principally responsible for elevated blood-lead levels in children.

After reading this report, Needleman wrote a letter to CEQ chairman Gus Speth expressing his "consternation." He called the lead section "unscientific and tendentious," and a disservice "to the children living in leaded housing." And he charged Billick's research group with attempting "to diminish the role of lead paint as a source of hazardous exposure for children," because it has "been unequal to its appointed task—improving housing stock. Rather than expend the energy and funds to tackle this formidable job, it has made it disappear by the magic of inferior biostatistics."

Du Pont, one of four U.S. producers of lead additives for gasoline, agrees with Needleman's assessment of the statistics. So does Donald Lynam of the Lead Industries Association, who says "HUD has made

more out of these data than is scientifically valid." Although Lynam concedes that lead is a multimedia problem, he claims that lead in paint "is by far the most significant source."

Even though the inclusion of only Billick's data in the CEQ annual report made for a less than comprehensive discussion of the lead issue—a condition that CEQ member Robert Harris tells C&EN will be rectified in the next annual report—the same data were used by EPA to ward off industry and the Energy Department's attacks on EPA's efforts to phase out lead in gasoline. Because of the presumed gasoline shortage in 1979, EPA relaxed but did not rescind its lead phase-down program. By October 1980, the final lead-in-gasoline standard of 0.5 gram per gal is to go into effect.

Ethyl Corp., the major U.S. producer of the antiknock compound tetraethyllead, along with Du Pont is fighting this final phase-down effort. The addition of the additive to a gallon of gasoline does extend that gallon 6%, and Ethyl has taken this fact to the American public. In newspaper ads, Ethyl says that \$4.71 of its compound in gasoline can conserve a barrel of crude oil, thereby improving U.S. balance of payments, increasing

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gasoline availability, and adding to the petrochemical feedstocks.

Du Pont is taking a quieter tack. In a long critique of Billick's study on the association of blood-lead values and gasoline-lead levels, Du Pont tells EPA that:

- "HUD used the wrong data to represent gasoline lead use in New York City.

- The HUD blood-lead data are seriously flawed by sampling irregularities, procedure changes, and data handling inconsistencies—all of which affect blood-lead values.

- HUD's treatment of the blood-lead data suffers from serious deficiencies.

- HUD's blood-lead/gasoline-lead relationship is unreliable."

Also joining the fray is NAS, which finds that "empirical correlations between measurements of lead in the body and lead in the air, water, soil, or gasoline are valuable qualitative indicators of source contributions, but they rest on too few data and too many unproven assumptions to be accepted as being quantitatively reliable." At first glance the finding appears to support Du Pont's position. However, the lead panel's call for continued control of all sources of lead, including lead in gasoline, effectively neutralizes that support.

If anything, the academy's report is likely to support EPA's ambient lead and lead-in-gasoline programs. EPA now estimates that from 1970 to 1977, airborne-lead levels have dropped 26%; during this same period lead consumed for gasoline sales has declined 30%. Although it is not possible to break out the contribution of various sources to ambient lead levels, EPA estimates that vehicle emissions account for more than 88% of total lead emissions.

Diet is still another major source of body-burden lead, especially in adults. According to the Food & Drug Administration, most of the lead in food and water is there as a result of natural background levels and pollution—airborne lead from industry and vehicles redepositing on crops and soils and in drinking water supplies. A portion of the lead is present as a result of food processing.

FDA, the agency charged with ensuring the public a safe food supply, has never said what's a safe lead level in food. But that does not mean it has been totally silent on the issue. Since 1973 it has moved slowly to persuade industry to decrease voluntarily lead levels in milk and in baby foods and formulas—a program that in some instances has resulted in a 90% drop in lead levels.

Last year FDA issued an advance

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notice of proposed rule-making on lead in food as a first step in setting "action levels" to reduce lead intake 50% in five years from lead-soldered cans, the only source of lead FDA believes it can control through regulation. Replying to this notice, Consumers Union applauded the move, but argued that the agency is moving too slowly, and that its goal of reducing lead exposure of children to less than 100 micrograms per day is not adequate for health protection. A safe maximum exposure range, the consumer's group argues, is from 0.6 to 10 micrograms per day for infants and young children, and from 10 to 78 micrograms per day for pregnant women, the two most vulnerable groups.

In a March issue of *Science* magazine, Patterson published an article on lead in tuna that was widely discussed in the general press. In this paper, geochemist Patterson argues that industrial lead is so pervasive in the environment that it "masquerades as natural lead in foods before they are processed." This so-called "natural" level plus additional contamination from processing and canning has elevated the lead concentration in today's U.S. diet 100-fold over prehistoric diets, he says.

This, in turn, has contributed to the 1000-fold increase in the lead burden carried by people today over their prehistoric ancestors.

Using sophisticated analytic techniques in an ultraclean laboratory, one fresh tuna sample and a small number of canned tuna samples, Patterson finds much higher lead concentrations in the commercially processed tuna. He, therefore, calls for a ban on all lead-soldered cans.

FDA director of the Bureau of Foods Sanford A. Miller takes issue with Patterson's sample size, sampling technique, and the "involved analytical techniques," which he says have not been validated for food measurements and are not "practical to meet the needs of regulatory agencies such as FDA for monitoring the higher levels of lead encountered in the food supply."

Kathryn R. Mahaffey, FDA nutritional biochemist, expert on lead, and member along with Patterson of the NAS lead panel, recognizes Patterson's sincerity and intensity of concern, but she contends that this concern does not reflect the realities of food handling, processing, and marketing, or the broader aspects of public health.

Nutritionally deprived individuals

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