CHAPTER 10

Accusations
Social Construction of Environmental Disease

Some of them gets lead-pisoned soon, and some of them gets lead-pisoned later, and some but not many, niver; and 'tis all according to the constitooshun, Sur, and some constitooshuns is strong and some is weak"

Charles Dickens*: The Uncommerical Traveler*

.....many environmental managers feel that Shakespeare's advice to 'kill all the lawyers' should also apply to analytical chemists

Frank B. Friedman: Practical Guide to Environmental Management, 8th Ed.

Lead and the Americans: Occupational Disease: Feminized Science

In this chapter our goal is to trace the evolution of our understanding of lead as an environmental disease. As we will see, through time, lead poisoning shifted from being a lamentable but inevitable part of a workers life, to being a disease which stalks children of all walks of life.

The Eight Hour Work Day

The recognition of lead poisoning as a disease, rather than an inevitable fact of life, was a political process. The process began somewhat earlier in Europe, and became full fledged in the United States from 1910-1925. It is inextricably related to the growth of unionization.The control of the medical evidence and the definition of disability play a great role. Disability definitions moved from clear loss of limb, to inability to labor within the specific trade, to inability to labor at optimum wages or in the future. Owners were either for against workmen's health insurance for lead depending upon the cost of insurance vs the cost of lawsuits (Markowitz and Rosen, 1991).

It is suggested that about 9,000 lead/silver miners in the west of the USA were poisoned by lead, a high amount due to the cerrusite vs galena form of many of the western ores (McCord, 1954). Another disease of concern to miners was pulmonosis, related to intake of activated silica (ground or sand blasted).

The legal standard for the time (Leadville, Co.) was that workers recognized the hazards of the job and took on the risks by taking the job. They were free to leave at any time. All medical care costs were to be assumed by the miner out of the $3/day wage for a 10/12 hours of work (1). This should be put in context of the Leadville mine in which over the course of 20 years $250,000,000 of Au and Ag were extracted.

Leadville was initially founded as a gold prospecting town (1861, Pike's Peak Gold Rush which led to the formation of the Colorado Territory). In 1874 a particularly rich vein of lead carbonate bearing 20-40 oz of Ag/ton (discovered 1874), requiring industrialization (train services, hydraulics for water washing) and transforming the mine from placer (nugget) sluicing. The lead could be sold at 6 cents/lb and the silver for $1.16/oz making it particularly profitable. In 1877 another vein was discovered containing 50% lead and >200 oz. Ag.

In certain locations lead poisoning was known as Mine Sickness, described in the *St. Louis Medical and Surgical Journal* in 1848 by Dr. S. Skeel (Voynick, 1984):

Figure 10.1. White lead worker circa 1890. From Lynn Willes: *Lead and Lead Mining: A Shire Album.*

There is another class of individuals, who are continually receiving [lead] into the system by every possible means, but, probably, in a less active form, but it is no less sure to lay the foundation of this disease. It is the miner, the digger and cleaner of mineral; he is handling it, he is inhaling it, and the flying particles mingling with the saliva, he is swallowing a considerable portion of if; consequently, he is more obnoxious to this complaint, in all its varied forms, than any other class of person; and it is with this latter class, that the symptoms are more variable, the cause, notwithstanding he is a miner, more obscure, and less likely, in many instances, to be suspected by the practitioner.....Sept. 19, 1838. Was called in the night to visit E. B. Harris, an overseer, in Valle's mine, aged 45 years, sanguineous temperament, plethoric, been three days sick, pulse slow and full tongue white, pain, (to use his own words) in the stomach, belly, back and collar-bones; no vomiting and but little sick stomach, obstinate constipation, great thirst, but drinks but little, and this warm sage tea. It is mine sickness, (has) had the disease before, two years ago. Upon enquiring what he had done for himself, said, I have taken a half ounce of calomel, and a pint and a half of castor oil; have given myself about a hundred injections (enemas), and gone into the warm bath, which is the only thing that gives me any ease, but I cannot bear it more than a half minute, without fainting. ...

While the primary disease afflicting miners, particularly after industrialization was pneumonia and tuberculosis derived from silicosis, lead poisoning was also very high. The conditions in Park City in the Coeur d'Alenes was the worst in the industry. More than seven thousand Park City miners were leaded badly enough to seek treatment (Derickson, 1988, p. 53).

The basis of union activity in the Idaho/Montana region was the establishment of medical care under the control of the workers. Previously workers had wages deducted for death benefits and for mine controlled physicians. In 1891, the Western Miners Union were able to establish collection of dues of $1/month for the Miners' Union Hospital. The Hospital documented occupational health hazards. The first quarterly report indicated that conditions treated were rheumatism with lead poisoning (Derickson, 1988, p. 98):

A shovel full of lead-silver ore will weigh three and four times as much as a shovel full of white quartz, and to inhale the lead dust is the very essence of poison. Of course none of our mine owners have ever been "leaded" except politically, but the hospital records will show that sickness from that cause is quite prevalent. There is perhaps no harder task allotted to man than to shovel lead-silver ore for ten hours.

The length of the working day was linked in union demands to other health issues. The Western Federation campaigned for an 8 hour work day in the 1890s, based on health issues in order to combat the framing of the issue as a right to work on the part of the owners. The unions cited the unique risks of their employment for state mandated 8 hour work days. The law passed by Utah in 1895 was challenged in the Supreme Court in 1898 in Holden v Hardy, but upheld as constitutional. By 1913, the last state to succumb to

Figure 10.2 Miners in the Joplin lead mines circa 1900. Postcard (A. Fitch)

the 8 hour day was Colorado (Derickson, 1988, p. 150).

Another mining example is the case of the Broken Hill Associated smelters (BHAS) in Port Pirie, South Australia (Gillespie, 1990). After the South Australia Workmen's Compensation Act of 1912 was passed providing for lead poisoning a dramatic increase of lead poisoning compensation claims were made (1 in 1917 to 265 in 1925). A Royal Commission established in 1925 to study the increase suggested that only a portion of these cases were real. It also suggested that lead poisoning occurred primarily among southern European immigrants who were either particularly susceptible or were malingering. Gillespie wrote:

The workers are portrayed as careless, for they apparently refused to use respirators, did not wash their hands prior to eating and few took showers at the end of the shift. In the most dramatic version, mining magnate and financier W. S. Robinson recalled in his memoirs that the increase was due to a fraudulent scheme organized by Italian workers, in which they deliberately consumed lead in order to receive lump sum compensation.

This account of worker culpability was noted earlier in Huxham's account of the Devonshire Colic and in Ben Franklin's memories of working as a lead typesetter.

The commission in Australia heard testimony from Henry George Chapman, professor of physiology at Sydney University and his colleague Stewart Arthur Smith. They distinguished between lead poisoning and lead absorption (Gillespie, p. 318):

Chapman stated: "I do not regard any change induced in the body by lead as necessarily being lead poisoning." Symptoms such as a blue line in the gums,

lead in the urine or stippling of the red blood cells were only signs of lead absorption, to be found in most workers exposed to significant amounts of lead. They argued that lead poisoning should not be diagnosed in cases where the worker presented with symptoms of lassitude, weakness, constipation and pains in the extremities, unless these were accompanied by clear evidence of colic, the painful stomach cramps long considered a classic sign of lead poisoning, or of undoubted nervous lesions in the affected extremities..... Moulden, a former medical officer to the Broken Hill mining companies and now a consultant to BHAS argued that such symptoms as loss of weight, pallor and constipation were only signs of lead absorption, and were to be expected among new workers until they developed a "tolerance" to lead.

Gillespie argues that industrial physicians were trained to treat the individual worker. Their focus was on individual susceptibility to lead poisoning rather than on aggregate occupational health hazards.

Hamilton and Feminized Science

In 1906, the American Association for Labor Legislation (AALL) was founded. It chose as its first issues industrial diseases, establishing a national commission on industrial hygiene, and helping the American Medical Association hold its first conference on industrial diseases (Sicherman, 1984). In 1908, the commission approached Alice Hamilton for help in this area.

Alice Hamilton was born in 1869 (Figure 10.3). She was one of the earliest American women allowed into medicine. In 1890 she entered the Fort Wayne College of Medicine, and then transferred to the University of Michigan as a special student. At the time the University of Michigan was engaged in an exciting reformation of curriculum, which involved moving from pure lecture, to science classes and clinic work. After graduating she worked for a while in Boston and took up a position at the Woman's Medical School of Northwestern University, a backwater segregated from the men's medical college, where she was a professor of pathology and director of the histalogical and pathological laboratories, The most critical part of her life was her residential move to one of social reformer Jane Addams' (1860-1935) settlement houses, an experiment in urban sociology. The Hull House, the third of the settlements, was located in an immigrant community on the near west side of Chicago. It was in this milieu that Hamilton became exposed to industrial diseases of the immigrant population.

It was my experience at Hull-House that aroused my interest in industrial diseases. Living in a working-class quarter, coming in contact with laborers and their wives, I could not fail to hear tales of the dangers that workingmen faced, of cases of carbon-monoxide gassing in the great steel mills, of painters disabled by lead palsy, of pneumonia and rheumatism among the men in the stockyards. Illinois then had not legislation providing compensation for accident or disease caused by occupation.....

His article (a scathing account by William Hard of an industrial accident) and a copy of Sir Thomas Oliver's Dangerous Trades, which came into my hands just then, sent me to the Crerar Library to read everything I could find on the dangers to industrial workers, and what could be done to protect them. But it was all German, or British, Austrian, Dutch, Swiss, even Italian or Spanish - everything but American. In those countries industrial medicine was a recognized branch of the medical sciences; in my own country it did not exist. When I talked to my medical friends about the strange silence on this subject in American medical magazines and textbooks, I gained the impression that here was a subject tainted with Socialism or with feminine sentimentality for the poor (Hamilton, 1985, p. 115).

Everyone with whom I talked assured me that the foreign writings could not apply to American conditions, for our workmen were so much better paid, their standard of living was so much higher, and the factories they worked in so much finer in every way than the European, that they did not suffer from the evils to which the poor foreigner was subject...

Professor Charles Henderson of the University of Chicago, influenced by the sociological movement in Germany, had studied the German sickness insurance for the working class (the Krankenkassen) and in 1910 persuaded the reform governor Charles Deenen of Illinois to appoint an Occupational Disease Commission, to which Hamilton and 6 other (male) members were appointed. They were charged to provide a survey of occupational health of the Illinois within one year. Of the diseases to be cataloged were carbon monoxide, arsenic, brass, cyanides, turpentine, and lead.

Hamilton drew lead. She and her assistants visited 304 establishments and discovered more than 70 processes that exposed workers to lead poisoning

Figure 10.3 Alice Hamilton, considered one of the founders of American Occupational Medicine. Image source: UIC Alice Hamilton conference 2002.

(making car and can seals, polishing cut glass, wrapping cigars in "tinfoil" and laying electric cables). She appears to have been most proud of her discovery of bathtub and toilet bowl enamelers lead exposure, since these were not trades described in the European literature. She found this by checking the medical records of the Alexian Brothers' Hospital and finding a case of colic and double wrist drop of a Pole. She found that the enameling process within the furnace blast involved sprinkling a finely ground enamel of lead oxide over a red-hot tub where it melts and flows over the surface. "*A specimen of it which I secured from a workman, who said he often took some home to his wife for scouring pans and knives, proved to contain as much as 20% soluble lead*".

She saw many cases of acute lead poisoning, all among the immigrant population. Her notes included:

A Bohemian, an enameler of bathtubs, had worked eighteen months at his trade, without apparently becoming poisoned, though his health had suffered. One day, while at the furnace, he fainted away and for four days he lay in coma, then passed into delirium during which it was found that both forearms and both ankles were palsied. He made a partial recovery during the following six months but when he left for his home in Bohemia he was still partly paralyzed.

A Hungarian, thirty-six years old, worked for seven years grinding lead paint. During this time he had three attacks of colic, with vomiting and headache. I saw him in the hospital, a skeleton of a man, looking almost twice his age, his limbs soft and flabby, his muscles wasted. He was extremely emaciated, his color was a dirty grayish yellow, his eyes dull and expressionless. He lay in a an apathetic condition, rousing when spoken to and answering rationally but slowly ,with an often appreciable delay....

...They were almost always foreigners, Bulgarians, Serbs, Poles, Italians, Hungarians, who had come to this country in the search for a better life for themselves and their children. Sometimes they thought they had found it, then when sickness struck down the father things grew very black and there were not old friends and neighbors and cousins to fall back on as there had been in the old country. Often it was an agent of a steamship company who had coaxed them over with promises of a land flowing with jobs and high wages. Six hundred Bulgarians had been induced to leave their villages by these super-salesman, and to come to Chicago. Of course they took the first job they could find and if it proved to be one that weakened and crippled them...well, that was their bad luck! (Hamilton, 1985, p. 126).

Table10.1

Occupational Distribution of Deaths from Chronic Lead Poisoning, U.S., 1910-1924

(Poisoned Painters: Organized painters' Response to Lead Poisoning in Early 20th-Century America. Christopher A. Eldridge, Bulletin of Science, Technology, and Society, 1998, 18,4, 266-280)

Occupation	Number	%
Lead Industry	62	3.89
Metal Miners	35	2.2
Painters	841	52.83
Paint mixers	15	0.94
Plumbers	25	1.57
Potters and tile workers	13	0.82
Rubber workers	3	0.19
Storage battery workers	5	0.31
Printers	67	4.21
Tin and coppersmiths	9	0.57
Metal workers	23	1.44
Carpenters and joiners	18	1.13
Laborers	93	5.84
Farmers	61	3.83
Professional	29	1.82
Miscellaneous	114	7.16
Unknown or retired	103	3.02
Women	11	0.69
Boys younger than 18	11	0.69
Girls younger than 18	8	0.5
Total	1,592`	100

Because enameling tubs was notoriously hard, hot, and dangerous, most American men shunned it and I found foreign-born workmen..I remember a foreman saying to me, as we watched the enamelers at work, "They don't last long at it. Four years at the most, I should say, then they quit and go home to the Old Country." "To die?" I asked. " Well, I suppose that is about the size of it," he answered.

Hamilton had difficulty with many of the employers who felt that the cause of lead poisoning was the cleanliness of the workers:

...in 1910 and for many years after, the firm (and comforting) belief of foremen and employers was that if a man was poisoned by lead it was because he did not wash his hands and scrub his nails.... (Hamilton, 1985, p. 122) and again....*I interviewed a doctor, a well-trained man, who was in charge of the employees of a great Utah smelter. He said to me, " I always tell the men that if they are careful to scrub their nails they need not fear lead poisoning." (P. 154).*

She found approximately 1,769 workers out of 7,400 workers in 19 major smelters and refineries were lead intoxicated.

In 1919, she was appointed to the faculty of the newly formed division of occupational health at Harvard and edited the *Journal of Industrial Hygiene*. While there she participated in a clinical/laboratory study lead by J. C. Aub which proved conclusively that it was the airborne lead that was of the greatest danger to the workers (breathable lead), a fact crucial to formulating changes in the industrial process.

In 1911 she was appointed the United States Bureau of Labor (founded 1903), and began a study of the Old Dutch paint process. In 1913 a Full-fledged Department of Labor was created and she became part of the Bureau of Labor Statistics. Her report *Lead Poisoning in Potteries, Tile Works, and Porcelain Enameled Sanitary Ware Factories* appeared in 1912. It was attacked by Dr. H. T. Sutton, member of the Ohio State Board of Health who was also employed by the American Encaustic Tile Works

...This woman...(report is) a striking example of exaggeration, either a false and apparently a malicious and slanderous report, or an erroneous one.

The AALL pushed standard occupational disease bills, one of which was passed by the Pennsylvania legislature in July 1913. The bill had many objectors, including Theodore Ahrens, president of the Standard Sanitary Manufacturing Company in Pittsburgh. Despite such opposition during the next ten years, occupational standards, and insurance became

Figure 10.4. Painters clothing is contaminated with paint. When working with lead paints this will contaminate the home of the worker. Image from Dutch Boy Painter Magazine, 29, 2, 1936.

part of the American scene and Alice Hamilton became the leading expert in lead poisoning. By 1925 the situation had changed so much that the United States was considered the leader in legislation and research at the International Congress on Occupational Accidents and Diseases.

Painters' Sickness vs Lead Sickness

As part of the increasing awareness of lead poisoning as an occupational health issue, and, as part of the growing strength of the labor movement, regulation of white lead paint was considered. The change in perspective between the early 1900s and the 1921 International Labor Conference (an offshoot of the League of Nations) is illustrative.

Until the early 1900s lead poisoning was considered inevitable and for the most part a problem of personal cleanliness on the part of the painter (or miner) (Eldridge, 1998). Dr. Louis A. Dreyfus (1906) writing for the trade journal *Painter and Decorator* "more often the lead is taken into the system by absorption, meticulous cleanliness is the answer" (Dreyfus, 1907). J. A. McMartin, a painter who had suffered from lead poisoning also wrote in 1906 that painters needed to keep themselves clean, that the breathing of lead fumes was dangerous but no more than contact of particles with the skin (McMartin, 1906). A 15 point list of actions that could be taken to prevent lead poisoning in 1905 listed 14 as related to the responsibility of the painter for personal hygiene. By 1920 the many members of the magazine were encouraging each other to switch to ZnO. But others argued that this paint had less covering power and durability. In particular contractors argued against a switch to ZnO as requiring more coats (more labor, more paint, and more cost).

This increased attention had results according to an analysis by Christopher Eldridge. In 1909 National Lead was advertising that skilled workmen prefer white-lead. Following articles in 1913 in the *Painter and Decorator* about bills to prohibit use of white lead in building, repairing, restoration, or interior work in Wisconsin, and articles in 1913 asking workers to push for cessation of white lead advertising changed. A 1922 ad in *Painter and Decorator* read:

There is no satisfactory substitute for white-lead, it is linseed oil's most natural companion, it covers well and spreads far, it lasts long and it brushes out easily and smoothly. White-Lead is the painter's paint. It is the material of the professional workman. The property owner has confidence in the painter who mixes his paint from white-lead and linseed oil. The painter has confidence in his ability to do satisfactory work when he knows his white-lead is right. Dutch Boy White-lead is right.

By 1914 some of the state laws guaranteeing compensation to workers and families of workers injured on the job moved to include disability from white lead paint. Workers maintained that they needed 2 full days per week to rest from the lead dust and fumes that accompanied their work.

The tide toward regulation of the occupational health hazard of lead was swinging so strongly against white lead that the 1921 International Labor Conference was heavily lobbied by the industry. The book length document entitled "*The Case Against Prohibition*" put together by British interests put forth four main points excerpted below.

The fact is that, long ago, white lead got a bad name and it is a case of 'give a dog a bad name and hang him'. People are unconsciously affected by the memory of the old days when the conscience of the community was stirred by the many cases of poisoning in the Potteries and in white lead works. They forget, or do not know, that this is all past history and that Regulations have almost completely removed the danger in these industries.

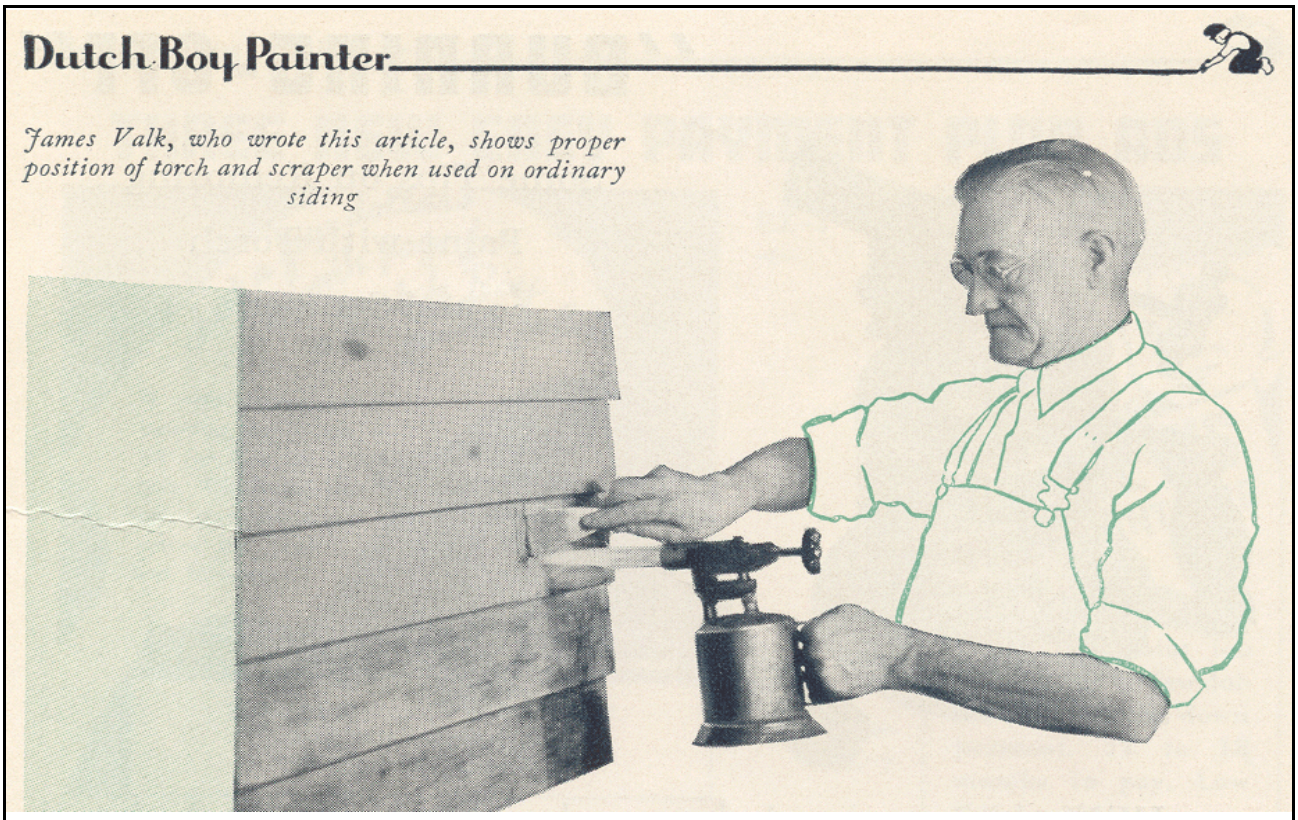


Figure 10.5. An example of acceptable paint removal from the Dutch Boy Painter Magazine, 29, 2, 1936, p. 42.

The reasons why Prohibition is the wrong way of dealing with the problem are shortly that:

1. Painters' sickness is not lead sickness but is paint sickness. It comes from the use, for paint, of all compounds of heavy metals, whether white lead or zinc white or any other, and from the use of the volatile thinners, such as turpentine, which are common to all paints.

2. Prohibition would cause unemployment in the British white lead industry and among British and Imperial lead workers. Further, it would make it necessary to use an inferior paint produced chiefly by the foreigners.

3. Prohibition would be difficult to put into force, and, in any case, would only deal with part of the causes of sickness among painters.

4. Painters' sickness, which is not more than an ordinary trade risk, can be far better dealt with by simple Regulation to avoid the dangers of dust and the vapor of turpentine, etc., and to ensure cleanliness.

These points were elaborated within the document. Figures on industrial death rates were produced showing painters die at a rate of 12.31/1000 compared to the larger industrial death rate of 13.85/1000. (Notice that the emphasis here is on death not health). Further, only 1 per 4347 death certificates of painters actually listed lead poisoning and these could as easily be attributed to poor diagnosis. It was difficult to distinguish between lead poisoning and chronic rheumatism. (Notice that the definition of the disease depends upon an overt, or macroscopic, observable, ailment.)

The authors of the document proceeded to indicate that there was no data available for sickness alone (apart from death). With respect to sickness they attributed the ***Painters' Sickness*** to the combined effect of the three components of paint: pigment, linseed oil, and thinner (turpentine). (Emphasis here added to indicate the importance of controlling the spin on the subject.) The specific effect of the pigment was attributed to dust which could be controlled by wet sanding, washing of overalls, and avoiding hand to mouth contact. Much of the illness was attributed to turpentine, as proven by the fact that miners have different sets of illnesses. They then suggested that the main component of Painters' Sickness was from

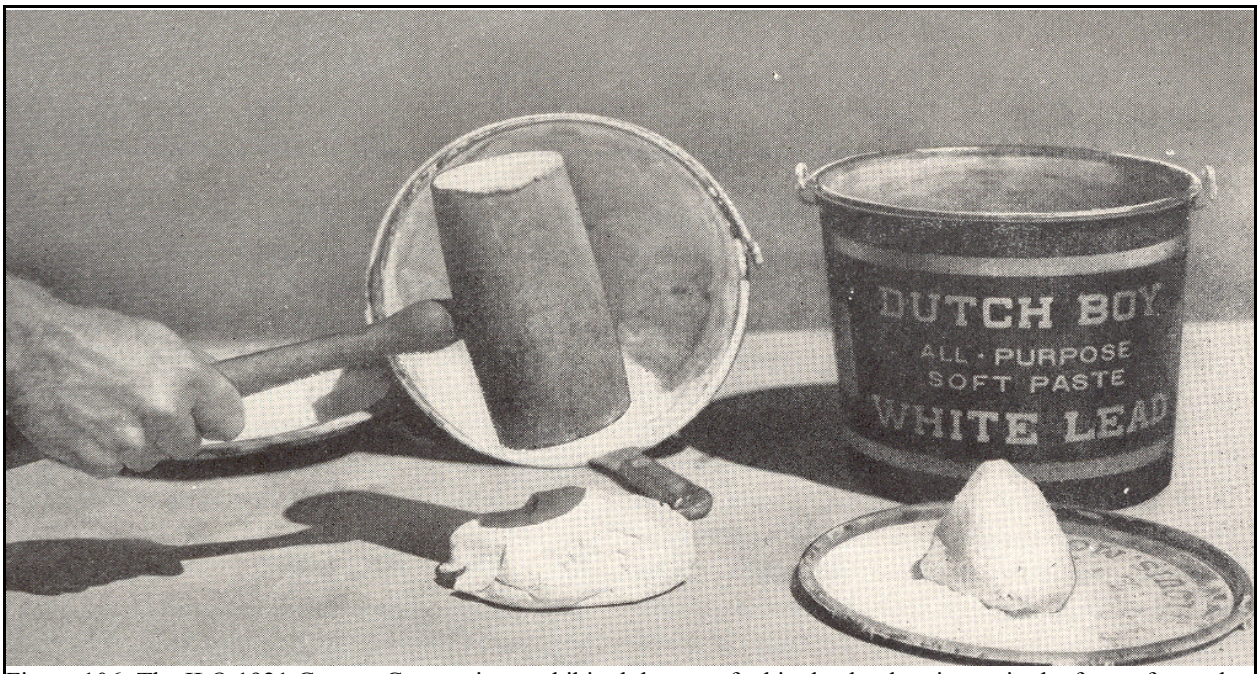


Figure 106: The ILO 1921 Geneva Convention prohibited the use of white lead unless it was in the form of a ready made paint. The use of the paint paste shown here, could give rise to dust which exacerbated lead exposure. Image from *Dutch boy Painter Magazine*, Vol. 31, 6, 1938. P. 128..

turpentine. They made the comment: *many painters are addicted to turpentine* as an alcohol. This tactic both diverted attention from lead and impugned the workers.

The next set of arguments were based on the success of regulation and the probable failure of prohibition. First, prohibition would idle factories which could not be adapted to other uses and many thousands of workers would be unemployed. An example given was the case of the lead mining industry which would surely fail with the cessation of white lead production since 20% of the lead mining industry produce was directed toward paint. No corresponding increase in employment could be envisioned from the competitive technologies (ZnO) as zinc was produced in Belgium, Germany, and the U.S. and not in the English or Imperial (Australia and Burmese) mines. Finally, prohibition would give the workers a false sense of safety from the real agent, turpentine.

Regulation, on the other hand, had shown a demonstrable decline in industrial disease. Lead poisoning decreased from 399 in 1899 to 10 in 1919 and among Potteries from 200 in 1900 to 21 in 1919.

The authors then quote the report from the 1907 Geneva Comission on *La Ceruse*:

The danger is not sufficient to necessitate the prohibition of a material, the careful use of which does not constitute a social peril. There is an industrial risk and we do not suppose that it can ever be abolished in painting any more than in any other occupation. At the forge, the slate-quarry, the loom, as a fileworker, or a dyer, the worker is exposed to risks often more serious. We do not desire, by yielding to the quite natural sentiments of those who are concerned for the workers and demand prohibition, unnecessarily to do the business of certain financial and commercial monopolies and of certain manufacturers of zinc, who are and who will be the masters of the price of it.

The *Case Against Prohibition* concludes by advocating three regulations which had been variously adopted by many European countries: 1. Prohibit dry sanding; 2. Medically track the workers better; and 3. Require employers to wash the clothing of the painters once a week and to provide written advice with respect to Painters' Sickness (Figures 9.30 and 9.31).

The White Lead (Painting) Convention of 1921, No. 13, adopted several of the proposals and issued a further set. The first article of the convention states:

1. Each Member of the International Labor Organization ratifying the present Convention undertakes to prohibit, with the exceptions provided for in Article 2, the use of white lead and sulphate of lead and of all products containing these pigments, in the internal painting of buildings, except where the use of white lead or sulphate of lead or products containing these pigments is considered necessary for railway stations or industrial establishments by the competent authority after consultation with the employers' and workers' organizations concerned.
2. It shall nevertheless be permissible to use white pigments containing a maximum of 2 per cent lof lead expressed in terms of metallic lead.

The second article exempted artistic painting. The third article prohibited males under 18, unless apprenticed, and women from industrial painting with white lead. Article 5 specified that painters should not work with lead based powders (should use ready made paints, Figure 10.6), should not work with lead based paint sprays, and should avoid dry rubbing and scraping of lead based paints (Figure 10.5). Facilities for washing should be provided, changing rooms to remove overall before leaving work, and washing of overalls by the employer (Figure 10.4) Cases of lead poisoning or suspected lead poisoning should be tracked by appropriate medical authorities.

Conditions within the white lead paint industry remained difficult for workers as indicated from a description of labor difficulties besetting a would be manager in an engineering dissertation of 1929 (*Managerial Problems of the White Lead Industry*). A chapter was devoted to the view point of the worker.

Respirators have proved an unsatisfactory means of protection because they either make breathing difficult or fail to collect all the dust. They usually fit so poorly that considerable dust enters between the edges of the mask and cheeks. Consequently, they merely give a sense of security without providing adequate protection.

On numerous occasions the older employees in the industry have attempted to have the working day reduced to 8/hrs a day. They argue that they could accomplish just as much as they do now and yet would be confronted with the possibility of lead poisoning to a much lesser degree than at present. Under this arrangement they feel that the original wage should be raised to 50 cents/hour which would give them the same aggregate wage as now received.

The dissertation writers agreed because they noted that there was a high labor turn over because of the difficulty of the work (Purdy and Hessley, 1929).

The Kehoe "Show Me" Rule

The debate, however, continued, as new uses of lead were found. An example is the public health debate that occurred over the introduction of lead to gasoline. The history of this episode, as analyzed by Rosner and Markowitz (Rosner and Markowitz, 1985), is presented below.

In 1922 Thomas Midgley and co-workers at the General Motors Research laboratory in Dayton, Ohio found that addition of tetraethyl lead to gasoline allowed one to raise the compression in the cylinder, which translated, to more power, and more speed. Leaded gasoline was placed on sale in selected markets on February 1, 1923. William Mansfield Clark, professor of Chemistry, wrote to A. M. Stimson, Assistant Surgeon General at the Public Health Service in Oct. 1922 warning of a serious menace to the public health of tetraethyllead which had several documented cases of laboratory toxicity and the probability that lead oxides would remain along the streets. Stimson recommended that data be obtained from the test marketed product. Pierre S. DuPont said that no in-house tests were conducted, and recommended that the Bureau of Mines do testing, but that:

the Bureau refrain from giving out the usual press and progress reports during the course of the work, as [GM] feels that the newspapers are apt to give scare headlines and false impressions before we definitely know what the influence of the material will be.

Because of the "scare" factor the product, tetraethyl-leaded gasoline, was named ethyl gasoline instead. (Just as Nuclear Magnetic Resonance in a hospital setting is called Magnetic Resonance Imaging.)

In 1924 a tetraethyllead plant employing 49 workers had 5 workers die and 35 others experiencing severe palsies, tremors, hallucinations. The vice-president of General Motors, Thomas Midgley responded:

the men, regardless of warnings and provision for their protection, had failed to appreciate the dangers of constant absorption of the fluid by their hands and arms.

Figure 10.7. An ad for Ethyl gasoline fromapproximately 1950 in the magazine *Country Gentleman*.

At that same time the Bureau of Mines report gave a clean bill of health to the product saying that the final product had none of the toxicity associated with its manufacture. The newspapers, however, ferreted out a series of deaths and illnesses at other production plants. The *Times* found over 300 cases of lead poisoning in two years at Deepwater, New Jersey. The plant was called the House of Butterflies because of hallucinations of insects that worker had:

The Victim pauses, perhaps while at work or in a rational conversation, gazes intently at space and snatches at something not there.

The Federal government convened a conference in 1925. The industrial representatives presented three main arguments. 1. Production of leaded gasoline was essential to industrial progress in America, by conserving fuel, with leaded gasoline a "gift of God". 2. Any progress has risks (no pain, no gain!). Parmalee said: "its casualties were negligible compared to human sacrifice in the development of many other industrial enterprises." Hayhurst, an expert on public health arguing on the side of the industry, wrote privately:

I am afraid human progress cannot go on under such restrictions and that where things can be handled safely by proper supervision and regulation they must be allowed to proceed if we are to survive among the nations. Dr. Thompson's arguments might also be applied to gasoline and to the thousand and one other poisons and hazards which characterize our modern civilization.

A third reason put forth by industry was that the major cause of illness was worker carelessness. Midgley (VP of General Motors) said

the essential thing necessary to safely handle [tetraethyl lead] was careful discipline of our men....becomes dangerous due to carelessness of the men in handling it..... The minute a man shows signs of exhilaration he is laid off. If he spills the stuff on himself he is fired. Because he doesn't want to lose his job, he doesn't spill it.

This message was accompanied by a disclaimer that even so lead exposure wasn't that dangerous. To prove his point Midgley, in the presence of a reporter, washed his hands in pure tetraethyllead:

I am not taking any chance whatever, nor would I take any chance doing that every day.

He neglected to tell the reporter that he had just returned from a holiday taken to recover from the effects of lead poisoning.

The opponents focused not only on the issue of worker health but on large dispersion of lead through the environment. These views prevailed. At the end of the conference sales were halted pending a further test of the product. However, the further test was of 257 individuals of the general public, and was conducted over a 7 month period, which found no hazard to the general population. Leaded gasoline production was resumed. No further long term experiment was performed. Rosner and Markowitz conclude:

Ultimately it was impossible [for "objective" scientists] to separate their "science" from the demands of an economy and society that was being built around the automobile. How else, then, do we explain public health scientists' willingness to conduct a short term study that could not resolve the long-term health issues. By agreeing to provide quick answers they guaranteed that this vital industry would not be disrupted. The symptoms of lead accumulation due to exhaust emissions would be unlike anything they had previously encountered in industrial populations. In the long run, those most affected would not be adults, but children, slowly accumulating lead. Their suffering speaks more to the interlocking relationships between science and society than to the absence of a link between lead and disease.

Once tetraethyllead was introduced as a consumer product of wide dispersion it became difficult to stop production based on scientific or health arguments.

As discussed in articles by two important researchers in the field of environmental lead, Nriagu and Needleman (Needleman, 1998; Nriagu, 1998), the standard of proof was set by Robert A. Kehoe, occupational physician, and Medical Director of Ethyl Corporation. He was hired by Kettering in 1924 at the age of 31 to look into the occupational effects of lead and became the most influential researcher on occupational health effects of lead in America. In the 1925 Public Health hearings his "show me" argument was articulated:

I am convinced from the association I have had with

the company that has had charge of the distribution of this commodity and their attitude is one with complete regard to facts. They have expressed themselves repeatedly not so much as being interested in opinions as being interested in facts, and if it can be shown - if it is shown as a result of this discussion - 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 the treatment of the gasoline with lead, the distribution of gasoline with lead in it will be discontinued from that moment. Of that there is no question.... When a material is found to be of this importance for the conservation of fuel and for increasing the efficiency of the automobile it is not a thing which may be thrown into the discard on the basis of opinion. It is a thing which should be treated solely on the basis of facts. That has been our attitude from the beginning and that will continue to be our attitude.

This statement was to frame the debate for the next 60 years, as discussed by Alan Loeb (Loeb, 1994). The public was to have to prove the harm of the product, which would come only after it had been dispersed, and where the threads of scientific proof would be complicated by the complexity of the environmental system.

Lead and the Americans: Analytical Chemistry and "Background"

Julian Chisholm Introduces Analytical Chemistry

...many environmental managers feel that Shakespeare's advice to 'kill all the lawyers' should also apply to analytical chemists

Frank B. Friedman: Practical Guide to Environmental Management, 8th Ed.**, 1998**

The difficulty of proving that lead dispersal was dangerous depended on proof of several different points. The first point required the ability to measure lead poisoning in cases which did not extend to classical, acute, clinical poisoning manifested by death, weight loss, anemia, and wrist drop. In essence, tools were required to measure both physiologic effects of lead (reduced nerve conduction velocities, for example) and the actual content of lead within the body.

The second point required development of tools to measure environmental lead that were more sensitive than the gravimetric lead sulphide or chromate

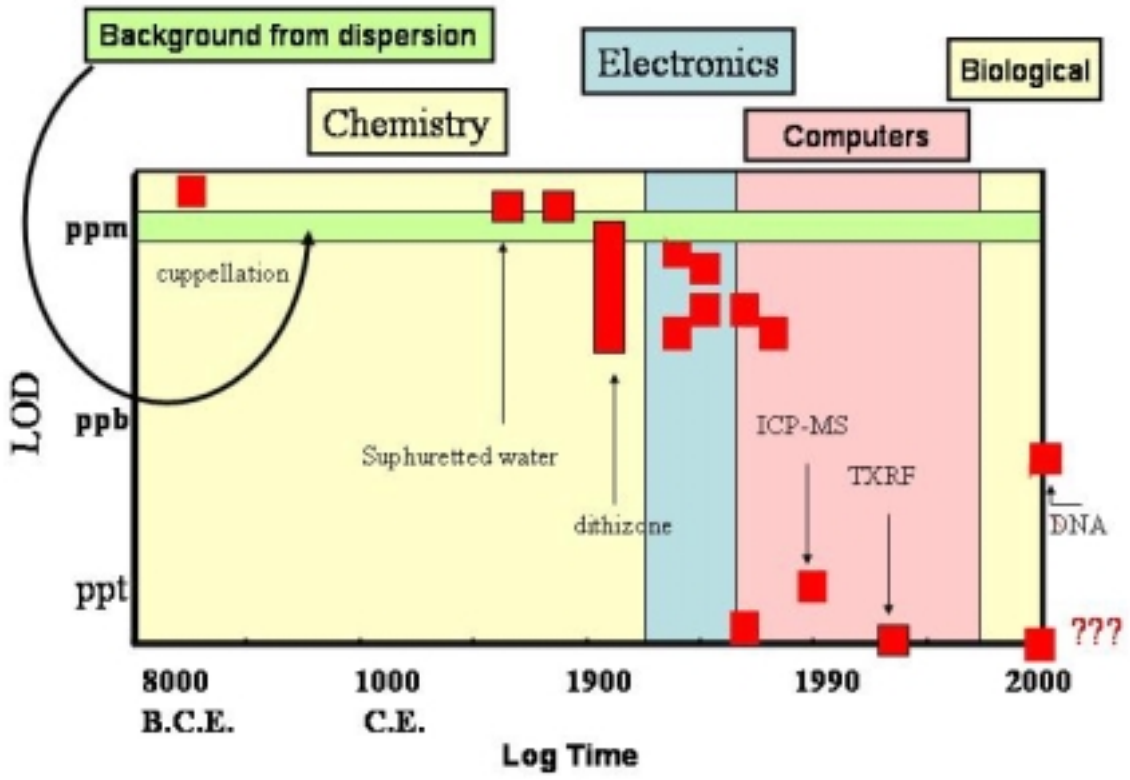


Figure 10.8 The limits of detection have dropped by 1,000,000 (6 orders of magnitude) from 1900 to 2000. The first methods depended upon weight measurements. Early in the 20th century the chemical method of dithizone was developed. It was exacting so although analog instruments developed after WWII had similar limits of detection it did not get used for widespread environmental sampling. Computerized instrumentation drove the limits of detection down another three orders of magnitude. The background level of lead contamination is approximately shown by the green horizontal box.

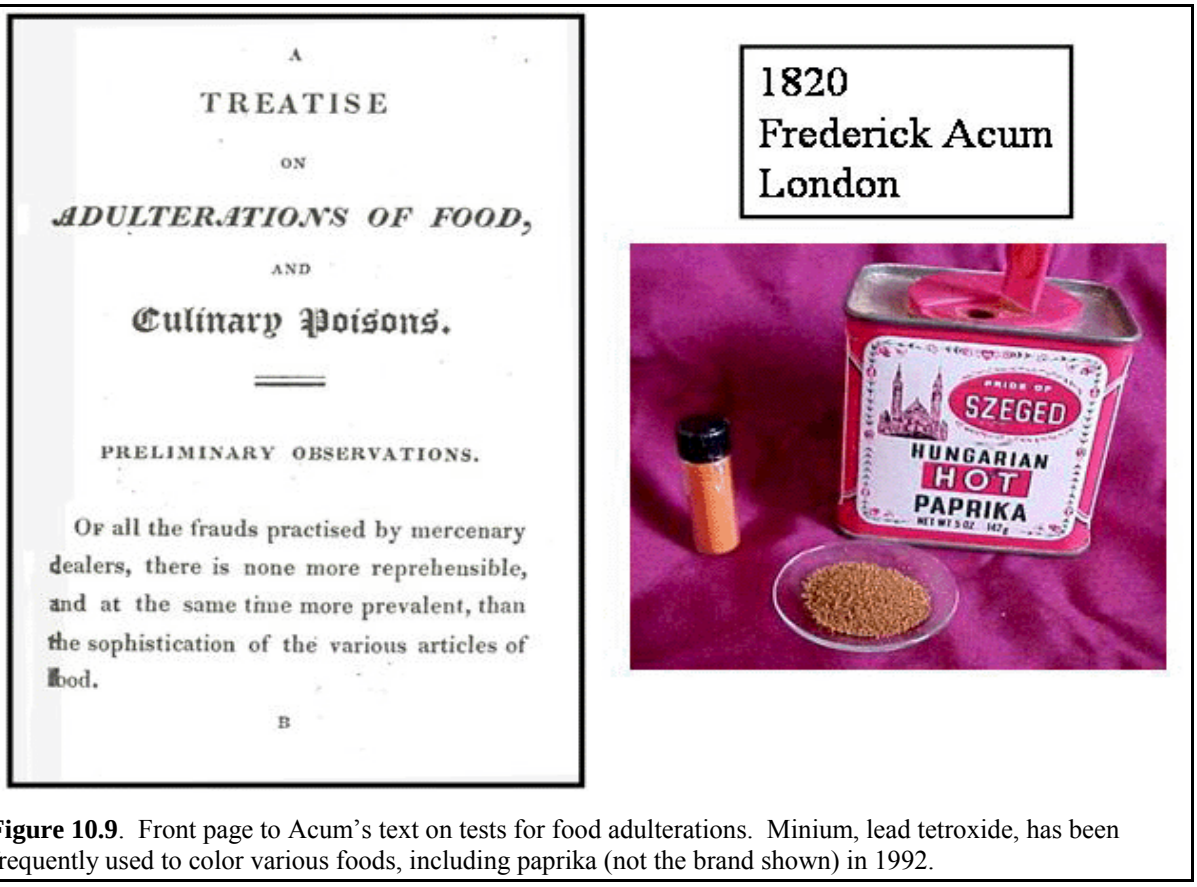


Figure 10.9. Front page to Acum's text on tests for food adulterations. Minium, lead tetroxide, has been frequently used to color various foods, including paprika (not the brand shown) in 1992.

tests used in 1820s and 1920s. The third requirement was the ability to find sites or materials to measure that had not been subject to the dispersal of lead produced by the Greeks, Romans, and leaded gasoline. The fourth requirement was the resolution of what was meant by lead absorption vs lead poisoning.

As seen in the cases above, it was widely assumed that 1) occupational health diseases were an inevitable effect of progress, 2) that lead was everywhere and natural, 3) that biology was adaptable to lead to a threshold level, such that lead poisoning could be distinguished from lead adsorption. As Charles Dickens wrote in "The Uncommercial Traveler":

Some of them gets lead-pisoned soon, and some of them gets lead-pisoned later, and some but not many, niver; and 'tis all according to the constitooshun, Sur, and some constitooshuns is strong and some is weak"

To understand the time lag between the introduction of tetraethyl lead and its banning, or the time lag between the 1921 ILO convention's recommendation for banning of indoor white lead paint and its subsequent banning in the U.S., we need to follow the history of the field of analytical chemistry. As shown in Figure 10.8 during most of that time frame the tools capable of measuring "background" values of lead had very high limits of detection. The lowest measurable value of lead associated with the analytical techniques available were higher than the amount of lead dispersed into the environment.

The first analytical measurements of lead were associated with cupellation and measured the purity of lead within a few tenths of a percent, or in parts per hundred parts. With the advent of minerals acids (discussed in Chapter 6) greater precision was available but the limits of detection still remained high. Despite the high limits of detection chemists were finding toxic effects of lead as did Acum in 1820 (Figure 10.9).

Little changed in the intervening 100 years between Acum's work and the occupational health debate associated with the ILO conference and the public health debate associated with the introduction of tetraethyllead in 1924.

A quirk in the fate of history could, perhaps,

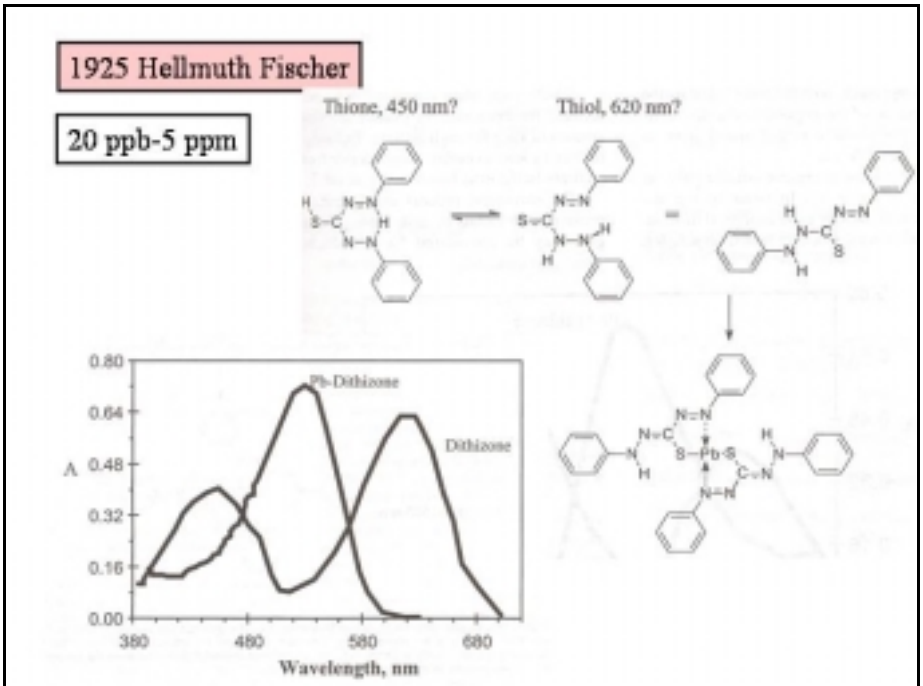


Figure 10.10. The dithizone method causes a change in absorption based on perturbation of conjugated bonds. The color changes from a brilliant green to a deep red.

have altered the debate. In 1925 Hellmuth Fischer of Germany introduced a much more sensitive technique for lead analysis, that based on dithizone (Figure 10.10). Siemens munitions work needed higher control of the metal content than was available with previous methodologies.

The dithizone method introduced became the workhorse of lead analysis for the next nearly 40 years. It is highly difficult method requiring skilled manipulation of the chemical conditions, and, also, fraught with the possibility of false positive measurements. Despite these drawbacks, it allowed the level of lead analysis to drop to the range of 20 parts per billion to 5 parts per million (Figure 10.10 and 10.8). Achieving the 20 ppb detection limits requires significant control of the background reactions.

Julian Chisolm, Jr., a prominent researcher in the area of lead and public health at the Kennedy Krieger Institute in Baltimore Ma., wrote an article in the late 1990s discussing the effect of the introduction of dithizone on the measurement of childhood lead poisoning in America (Chisholm Jr., 2001). Chisholm noted that diagnosis and follow up of cases of childhood lead poisoning was *no doubt due to the total dependence on physicians on identifying the symptoms and signs of childhood lead poisoning and the absence of any feasible laboratory procedure such as the dithizone procedure, with which lead oculd be measured in blood, urine, and tissues to document the clinical diagnoses.*

He noted that Levinson and Harris in 1936 suggested that children should be followed for neurobehavioral disturbances but that the first such tracking occurred in 1943 where 19 out of 20 cases of treated acute childhood lead poisoning resulted in exclusion from 1st and 2nd grade.

The first location in the U.S. to introduce the use of dithizone for blood lead testing was Baltimore. In 1931 Baltimore had experienced an epidemic of childhood lead poisoning caused by the burning of lead acid battery casings which had been recycled and donated to the poor for fuel. To track the cases of poisoning the head of Pediatrics at Johns Hopkins, Dr. Park, facilitated the acquisition of the dithizone technology by sending Dr. Emanuel Kaplan, newly minted chemist, to DuPont to learn the dithizone technique. Much of the early work in the U.S. on the epidemiology of lead (seasonal distribution, subclincial cases, etc., differential effect on children as opposed to adults) came out of Baltimore.

Work from Baltimore found that between 1931-1940 200 deaths of children under 15 from lead poisoning were reported in the U.S. with 24.3% of the cases reports originating from Baltimore. The Lead Industry Association (LIA) found these results astonishing and donated money for a follow up study. In 1955 when the studies were confirmed the paint industry adopted a voluntary standard to remove lead from indoor paint.

Other screening tests were required for paint content and urinary screening. Early paint tests depended upon the fairly unreliable chromate gravimetric tests used from the prior century. More

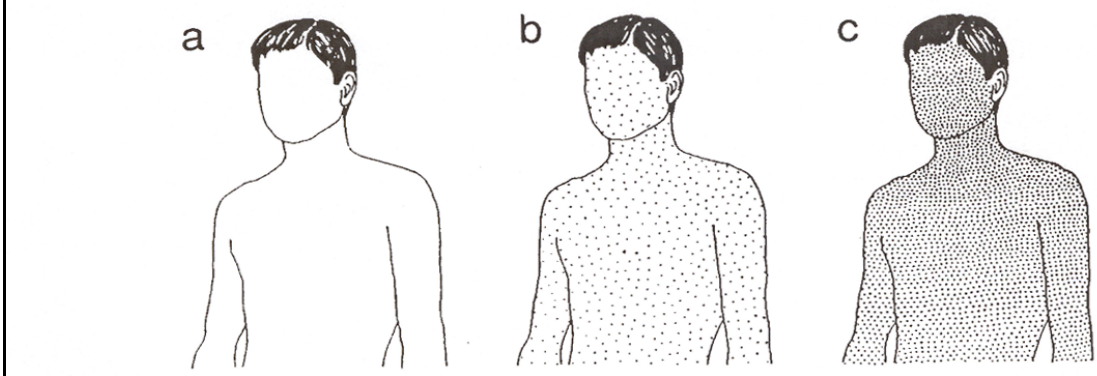


Figure 10.11 Patterson's historic measles image which was part of the National Academy of Science 1980 report on lead. (Source: Needleman, Env. Res. A., 1998, 78, 79-85.)

routine, less sensitive, lead exposure screening tests, were introduced in 1951 by Schwartz, Zieve, and Watson based on elevated urinary coproporphyrin (UCP) (Schwartz et al., 1951). ALA measurements were introduced in 1968. In 1970 atomic absorption spectrophotometric (AAS) methods and anodic stripping voltammetric methods (ASV) were developing to the point that dithizone methods would begin to be replaced as the laboratory workhorse. The fluorescence based rapid screening protoporphyrin test (little sample preparation required) was introduced in 1975.

Chisholm's report gives several examples of the difficulties involved in tracking all sources of contamination from measurement tools that would affect the final measured amounts of lead in the various biological samples. For example, homogenizing feces with commercial equipment resulted in lead contamination from the bearings, presumable Babbitt metals and lead containing lubricants (see Chapter 7).

Patterson and Background Contamination

The introduction of very sensitive, more easily used methods, did not change the regulatory climate or satisfy Kehoe's "show me" argument. A sensitive method can only measure values as low as exist in the laboratory and reagents used in the analysis, as indicated by Chisholm's example of contamination of feces samples by the grinding method in the lab.

The absolute necessity of truly lead free testing was demonstrated by the work of geochemist Clair C. Patterson (Figure 10.12). Patterson began his career in the field of geochronology studying the problem of assigning an accurate date to the age of the earth. To measure the age of the earth he analyzed the decay of uranium to lead in meteors. This necessitated the accurate measurement of very small amounts of both uranium and lead. Patterson wrote (Patterson and Settle, 1974)

Nuclear geochemists studying the geochronology of U-Pb systems and the isotope geochemistry of lead by means of isotope dilution mass spectrometric techniques have developed, during the past 25 years, a considerable knowledge of methods of obtaining low level lead blanks and of clean laboratory methods. These investigators wished to study ubiquitous mineral systems found in small crystals of common rocks, so they were restricted to microgram sized U-Pb systems. This forced them to solve contamination problems at extremely low levels. A critical constraint was imposed upon their solutions to lead contamination problems. Existing knowledge of chemistry, physics, and geology was interlocked with their work, and errors in evaluating lead blanks were immediately exposed because these errors would yield data which tended to violate known laws of nuclear physics or known benchmarks in the geochronology of the earth.

Patterson's major role in the social history of lead poisoning was to directly confront Kehoe over the "show me" issue (Flegal, 1998). Development of sensitive analytical methods was, alone, insufficient to alter the public debate. As each method was employed in analysis of the natural environment one conclusion that could have emerged was that the "natural" level of

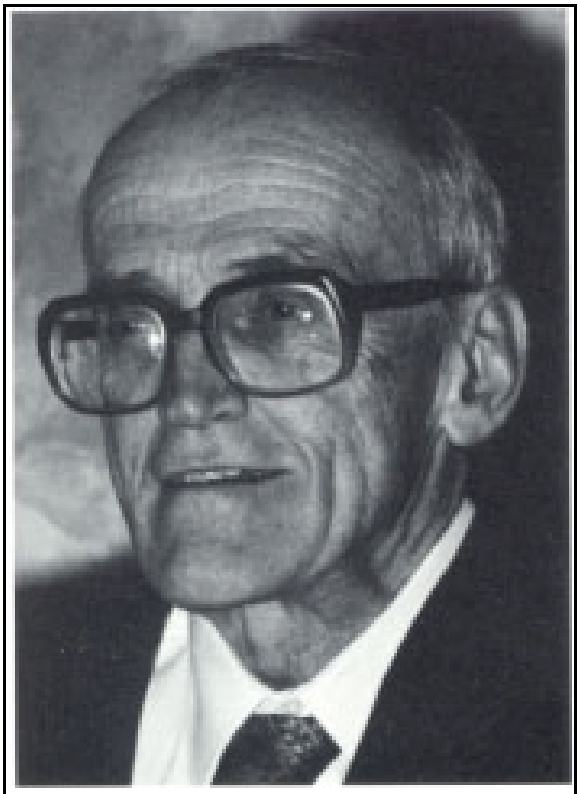


Figure 10.12 Clair C. "Pat" Patterson. Geochemist at Caltech who established "natural" levels of lead in the environment.

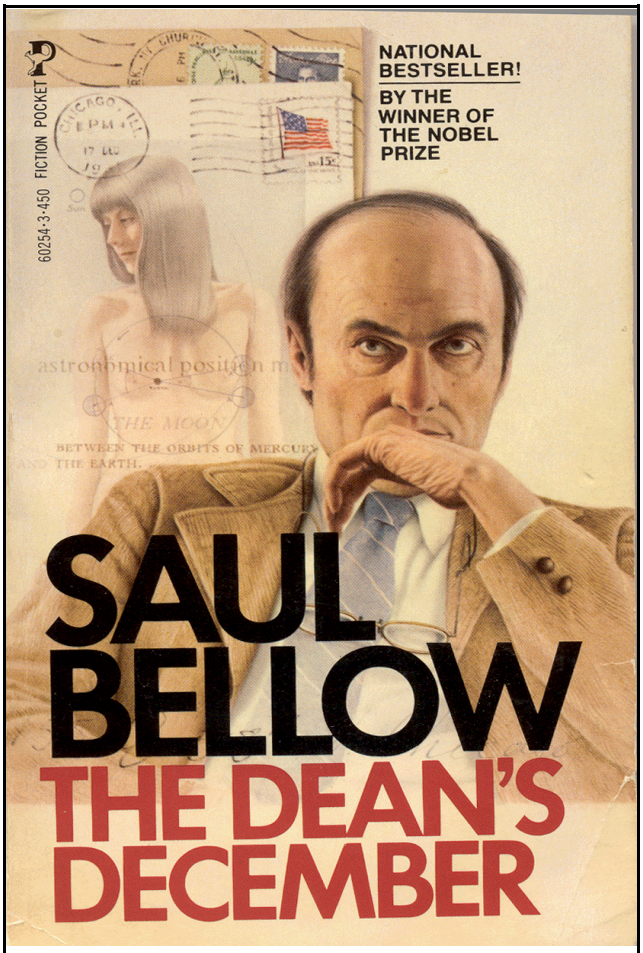


Figure 10.13 Saul Bellow's *The Dean's December* is based on the work of Patterson. Note the background image with alchemical symbolism

lead was quite high and that the biological organism was adapted to the high level of lead. This result would arise from the widespread dispersion of lead into the environment by the Greeks, Romans, and by the use of tetraethyl lead and white paint (Figure 1.3).

Patterson applied his geochronological results to a variety of environmental matrices. Three of the most important experiments follow. In order to establish a "natural" as opposed to "average" lead background Patterson needed to find environmental conditions that escaped widespread industrial pollution. One case was that of the arctic ice cores. He showed that the arctic ice cores contained lead that could be accurately tracked from the time of the Romans to current tetraethyl lead uses (Figures 1.3, 2.52, and 2.53) (Murozumi et al., 1969). The data indicated a 350 times elevation over "natural" values of lead. In order to find biological tissue that could give a "natural" as opposed to "average" blood lead content he studied marine species that inhabited depths of the ocean least likely to be contaminated (Patterson and Settle, 1977).

The third decisive case was that of archaeological bone samples from Peruvians. As discussed in Chapters 1 and 2 metallurgy in the New World did not develop to the same extent as in the Old World. In the first case, the population density was lower, a result of fewer large animal species available for domestication and the result of a land mass that required information exchange of non-compatible ecological zones, as compared to agricultural spread over the Eurasian land mass. A lower population density in the presence of a larger amount of surface ore bodies led to less depletion of placer and oxidized ore deposits. The result was that the technique of cupellation was not required for the needs of the population and large scale lead contamination as exercised by silver refining of the Greeks and Romans did not occur. By the time that the Europeans arrived in the New World alternative techniques had been developed, specifically the Patio process, which

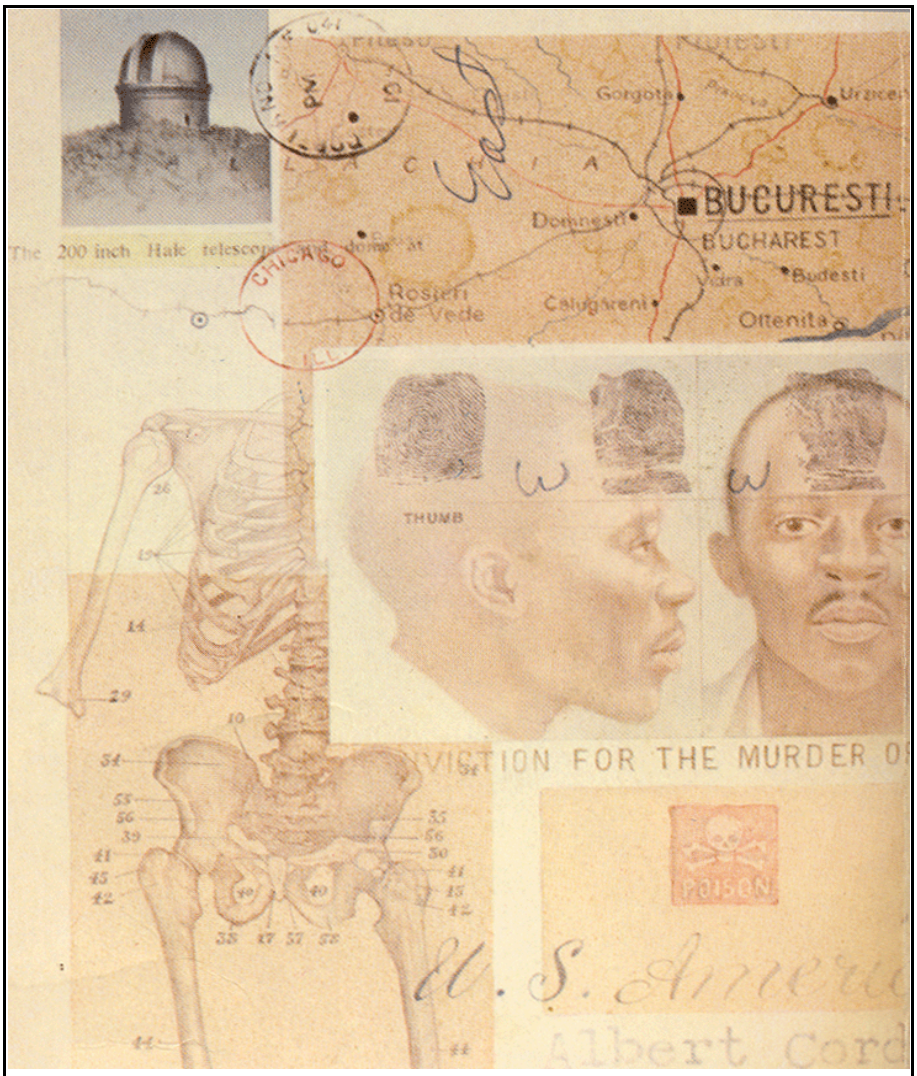


Figure 10.14 The back cover to Saul Bellow's *The Dean's December*. The images relate to the geochronological work of Beech (the Patterson character); the bone lead measurements, and to Patterson's suggestion that the inner city ills of 1960 and 1970 of the U.S. related to universal and extensive blood lead poisoning.

produced large amounts of mercury pollution, but not of lead pollution. As a result the "natural" exposure of humanity to lead could be better gaged from pre-Columbian bone samples (Ericson et al., 1979; Patterson et al., 1991). These results indicated that baseline levels in humans were 500-1000 times larger than "natural" (Table 9.4). From these results Patterson was able to address the issue of lead absorption vs lead poisoning. In Figure 10.11 the prevailing view was that the natural lead level of humanity was reflected by torso "B". Patterson showed that the "natural" state of humanity is better reflected by torso "A".

Patterson's research on lead continued for the next twenty years. He early on suggested that much of the "inner city" social problems were a manifestation of this 350 to 1000 times elevation in background lead. This viewpoint is articulated by Noble Laureate Saul Bellow in his book: *The Dean's December* (Figures 10.13 and 10.14). In the Dean's December, the dean, Albert Corde is courted by a geochemist, Beech, to publicize the effects of lead poisoning on the inner city dynamics of violence and education (Bellow, 1982). The character Beech is based on Patterson.

Beech: "You, the author of those special article might - you just might - be able to blow the whistle. I want to stop everybody in their tracks and force them to follow. And you can be gripping. As with the blacks you described in public housing and in the jails...."

"I didn't please everybody."

"I would assume not. That's exactly it. And when I read your description of the inner city, I said, 'Here's a man who will want the real explanation of what goes on in those slums."

"And the explanation? What is the real explanation?"

"Millions of tons of intractable lead residues poisoning the children of the poor. They're the most exposed. The concentration is measurably heaviest in those old slum neighborhoods, piled up there for decades. It's the growing children who assimilate the lead fastest. The calcium takes it up. And if you watch the behavior of those kids with a clinical eye, you see the classic symptoms of chronic lead insult. I've asked Vlada Voynich to include Needleman's neuropsychiatric findings from the New England

Journal of Medicine with the other papers. Crime and social disorganization in inner city populations can all be traced to the effects of lead. It comes down to the nerves, to brain damage."

....[requires nuclear science to set the background levels]

The conclusion: Chronic lead insult now affects all mankind. Biological dysfunctions, especially observable in the most advanced populations, must be considered among the causes of wars and revolutions. Mental disturbances resulting from lead poison are reflected in terrorism, barbarism, crime, cultural degradation. Visible everywhere are the irritability, emotional instability, general restlessness, reduced acuity of the reasoning powers, the difficulty of focusing, et cetera, which the practiced clinician can readily identify.

....

Here was an apocalypse - yet another apocalypse to set before the public. It wouldn't be easy. The public was used to doom warnings; seasoned, hell - it was marinated in them.

.....

"Lead as a mineral may or may not be the threat that Beech warns us against, but being 'leaden' certainly is a characteristic. Sometimes I say 'earthen' - we often experience this earthenness. Sometimes I say 'sclerotic', or 'blind', 'eyes that see not, ears that hear not' - and this leads up to the general end of everything' heralded by sclerotic, blind and earthen. 'Lead' is more sinister, maybe because of its color, hue or weight. Lead communicates something special to us about matter, our existence in matter.....There's poetry in that, isn't there? Man's great technical works, looming over him, have coated him with deadly metal. We can't carry the weight. The blood is sobbing in us. Our brains grow feebler. This disaster also overtook the Roman Empire. It wasn't the barbarians, it wasn't the Christians, it wasn't moral corruption: his theory is that the real cause was the use of lead to prevent the souring of wine. Lead was the true source of the madness of the Caesars. Leaded wine brought the empire to ruin.

And that was only Rome. Now it's the whole world. And it isn't the Grand Inquisitor's universal anthill that we have to worry about after all, but something worse, more Titanic - universal stupefaction, a Saturnian wild, gloomy murderousness, the raging of irritated nerves, and intelligence reduced by metal poison, so that the main ideas of mankind die out, including of course the idea of freedom."

The quoted passages contains all the elements of the current debate, biological mechanism, background levels, use of radio-tracing to set background levels, the issue of scare science. It also contains historical elements associated with lead, such as alchemical phraseology associated with lead.

The most important role Patterson played according lead researchers Needleman and Nriagu was in directly confronting Kehoe. Patterson's 1965 article on *Contaminated and Natural Lead Environments of Man* was reviewed by Kehoe who argued that it be accepted for publication (Needleman, 1998):

I should let the man, with his obvious faults, speak in such a way as to display these faults.... The inferences as to the natural human body burden of lead, are I think, remarkably naive.... It is an example of how wrong one can be in his biological postulated and conclusions, when he steps into this field, of which he is so woefully ignorant and so lacking in any concept of the depth of his ignorance, that he is not even cautious in drawing sweeping conclusions. This bespeaks the brash young man, or perhaps the not so young passionate supporter of a cause. In either case hardly the mark of the critical investigator.

We hav e been working with the physiological aspects of this problem carefully and step by step for more than thirty years... It is disappointing that our work has not been viewed in this manner by Dr. Patterson, but the issue which he has raised, in this article and by word of mouth elsewhere, cannot be 'swept under the rug'.

The virtue of the paper is its examination of the manner in which man has altered 'the face of the earth' in a variety of ways, and has disturbed the composition of the human milieu in so doing. It is strange that Dr. Patterson does not realize that this has happened to the large proportion of the mineral components of the earth, and that this is one of the outstanding physiological problems of our time. Can we adapt to these changes, individually and collectively? Are our physiological mechanisms flexible enough to cope with them? It appears, in the case of lead, that they are...

Patterson and Kehoe were both questioned at the 1966 Senate hearings on the Clean Air Act. Kehoe reiterated his "show me" stand and contended that no one had yet shown that lead had increased in the environment or that it was toxic. Again, Needleman quotes Kehoe:

There is no evidence that (TEL) has introduced a

*danger in the field of public health.... I may say the work of the Kettering Laboratory (*his laboratory*) in this field, that lead is an inevitable element in the surface of the earth, in its vegetation, in its animal life, and that there is not way in which man has ever been able to escape the absorption of lead while living in this planet.*

Although Patterson was making his case for widespread background contamination in the mid 1960s he was not appointed to the National Academy of Science's committee to deliberate on air pollution from leaded gasoline in 1970. The committee did appoint Kehoe, Gordon Stopps, Kamran Habibi and John Perrard from DuPont and Gary Ter from Ethyl Corporation. The 1972 report of the committee concluded that there was no unambiguous evidence to suggest that there was any toxicity to lead at low levels The debate over two main occupational lead poisoning issues continued into the mid to late 1970s. These two issues centered around what constituted lead poisoning and what was a better measure for control of occupational lead poisoning.

The First Lead Paint Laws

As a result of the scientific measurements made in the 1950s and 1960s, and as part of the general greening of the American consciousness, in 1971 President Nixon signed the Lead-Based Paint Poisoning Preention Act, LPPPA, which prohibited lead use in housing owned or subsidized by the federal government. The LPPPA prohibited the future use of paint containing 0.5% lead by dry weight. See Appendix for a time line of ever changing lead regulations J.17.

This act was amended in 1973 with an authorization for the Department of Housing and Urban Development (HUD) to implement procedures to eliminate, as far as parcticable, the hazards of lead-based paint poisoning in all public housing. HUD released amended regulations for this act in 1976.

Lead Absorption vs Lead Poisoning

As we saw above, occupational lead poisoning was initially defined by the acute clinical symptoms which accompanied blood lead levels above 80 µg/dL. Diagnosing lead poisoning below that required that workers' symptoms of fatigue and headache be accurate reflections of lead poisoning. In the view of the industrial experts of the 1920s many of those reporting those symptoms were faking. Unbiased measurements were sought, but required the development of accurate chemical measures.

1974 Blood Lead Values, NIOSH symposium	
Normal:	10-40 µg/dL
Abnormal but Safe	50-70 µg/dL
Lead Poisoned* (generally accepted value)	80 µg/dL

The blood lead levels used for diagnosing lead poisoning in 1974 were 10-40 µg/dL, normal; 50-70 µg/dL, abnormal but safe; and 91 µg/dL potentially dangerous. Generally 80 µg/dL was considered a threshold value for poisoning.

A NIOSH symposium was held in 1974 to review the standards associated with occupational exposure to lead. Many of the participants of the symposium suggested that there were many cases of acute symptoms that occurred at blood lead values below 80. Dr. Charles Hine of American Smelting and Refining Co. countered the arguments with the statement:

I have reviewed data representative of 5000 man-year, about 20,000 analyses, carried out by a laboratory of excellent and highest quality. I have never seen clinical symptoms in a man whose lead value was less than 80. It has never been reported in this group. At exposures leading to blood lead values about 80 and up to 120 µg/dL, we have seen probably an average of one man a year who had symptoms of lead intoxication. To explain reports in the literature of clinical damage or clinical symptoms occurring below this level I refer back to your original remarks, doctor, that the variabilities in the efficiency of lead analysis is considerable amoung most laboratories.

In shifting from a gross clinical symptom of lead poisoning to a chemical measurement two choices were possible. One could monitor the worker and shift the worker from the site if their blood lead level rose too much or one could monitor the environment and require environmental controls. Industry favored the prior approach. In 1973, as recounted by Dr. Paul E. Caplan of NIOSH (Caplan, 1976) wrote:

Last year, in an OSHA Review Board decision, on an appeal by a major lead producer, the original judgement was upheld that administrative control is not an acceptable alternative to reducing environmental

exposures to a safe level. That is, moving people in and out of a hazardous area, on the basis of medical and biological testing, is not an acceptable alternative to reducing atmospheric exposures to the TLV (threshold limit value) for lead. Nor is the use of respirators acceptable as an alternative control method, except in unusual, or temporary circumstances. As we have often seen, respirators are not accepted by workers nor do they keep exposure down.

The Lead Industry Association, on the other hand, said in 1972 that:

It is the position of the Lead Industries Association that a biological standard, based on blood lead determinations, provides the best means of protecting the worker and determining compliance under the OSH Act of 1970. Biochemical indices provide a much more accurate assessment of possible hazard to lead exposure than do air concentrations. It is recommended that air sampling be used only to indicate the necessity to institute biological monitoring and to evaluate engineering controls.

At the same occupational health symposium of 1974, Dr. Jerome F. Cole, Lead Industries Association, Inc., submitted an abstract which read:

While TLV's (threshold limit value) have been successfully used by occupational health practitioners as guidelines, for some substances, most notably lead, the worker serves as a better environmental sampler than mechanical air sampling devices. The use of the lead content of the blood as an index of the 'effective total exposure' for workers has been shown to provide a useful index of lead exposure. There are many inadequacies in air sample measurements, currently preferred by NIOSH and OSHA to determine compliance with the Occupational Safety and Health Act. Air samples do not represent the amount of absorbed lead because they do not reflect the differences in particle size and solubilities or measure ingestion exposure. The Biological Limit Value (BLV) of 80 µg/dL of blood is in the best interest of all concerned, especially exposed workers.

Dr. Cole was correct that, at the time, air sampling devices were cumbersome. Air sampling devices still (2002) are optimally designed to either capture total particulate load or to size fractionate.

Other workers at the conference, including representatives of the workers, disagreed with using the workers as a "better environmental sampler". Dr. Morris M Joselow wrote:

On quite legitimate moral grounds, a worker may object to being sampled periodically to serve, much like a guinea pig, as a monitor of the safety of the work environment provided for him.

We will summarize here some of the arguments made with respect to sampling, absorption, individual susceptibility, and quality of analysis because the exact same arguments arise in the next phase of the debate: childhood lead poisoning.

Testing the environment is an expensive proposition because of the quantity of samples and duration of the sampling required and because of the access required for the sampling. Once environmental testing is in place then environmental sampling may serve to set standards implemented which are intended to protect the worker or person.

However, if the environmental hazard exists naturally, and if people can absorb the hazard, that is, adapt to it, then the focus of environmental sampling has been shifted away from protecting a minority of affected people to a much more difficult to manage and more expensive proposition of controlling the general environment. Consequently, from an efficiency point of view, sampling the worker (or the child who is at risk) makes more sense than sampling every site and location in the U.S.

Once sampling is expanded the oversight required for multiple laboratory comparisons, for sampling procedure, is thought to become a nightmare.

This debate is still in full force as can be deduced from the arguments put forward by Peter Huber in his 1999 book *Hard Green, Saving the Environment from the Environmentalists: A Conservative Manifesto*

p. 131 Micro-toxic alarms are sounded far faster than reliable science can respond, or than a finite economy can realistically hope to address. When science eventually catches up with one or another, the pursuit turns out - much more often than not - to have been a complete waste of time.

Property rights don't help here. No system can fix green problems that science itself is unable to

define. It is impossible seriously to imagine a system of tradable permits for the trace toxins that so preoccupy the Softs [typical environmentalist on the political left], *the ones addressed in most of the far-future and hyper-dispersion models, the statistical ones, the ones discerned so much more clearly in computers than in the world at large.*

A robust body of mainstream science now confirms that the benefits of micro-environmental regulation are far more modest than was believed two decades ago, when Soft environmentalism embraced this new regulatory mission. In the visible, countable, measurable world, the numbers are clear. People first, life expectancies increase, birth defects and cancer rates hold steady or decline; taken altogether the things that assail people seem to be losing ground, not gaining. On the environmental front, there is no reason to suppose that the effects have been much different. Many environmental declines have occurred, but most are readily attributable not to microscopic assaults but to macroscopic ones, like bulldozer, asphalt, and fires used to clear forest for farmland. The micro-assault theories are a dreadful distraction here. By and large, we should welcome the industrial changes that trade off macroscopic assaults like those for the comparatively innocent micro-pollutants on which we waste so much money and concern.

Huber points out the levels measured of toxics have dropped and correlates those drops with economic wealth, not with the regulation adopted in the 1970s. He stresses that catastrophic loss of biodiversity facing the world in 2000 is related to habitat loss. He asserts that the primary goal of environmentalists should be habitat conservation.

The Birth of Risk Assessment

The ability of analytical chemistry to measure ever lower amounts of lead and the ability of medical and sociological sciences to correlate adverse effects to the lower amounts of blood lead led to a major management problem.

The early management of lead poisoning was based on two inherent assumptions about lead. The first was that there were three different dose/response ranges for lead (Figure 10.15). There was some low amount of lead exposure which had no adverse effect (similar in medicine to a non-efficacious amount of a drug); an exposure amount which caused a physiological effect (similar to an efficacious drug dose); and an exposure to lead which was toxic (similar to drug overdose). Management then was tailored to the individual susceptibilities of patients (as in setting the appropriate drug dose). Patients who had been exposed to toxic amounts could be helped by a rapid detoxification with chelation and by removal from the leaded environment. The corollary to this assumption was that effects were reversible. A worker who had blood lead levels above 80 could be relieved from work until such time has his/her blood lead dropped.

As lead poisoning no longer was defined by the most acute lead poisoning symptoms, but by a sliding scale of effects, management had no benchmarks with which to evaluate an exposed individual. Further, as analytical chemistry moved from microgram/g detection limits to 1 million to 1 billion times lower detection limits, it was no longer possible intellectually to conceive of finding a truly "lead free" environment. For every 99% reduction in lead, a finite amount of lead, often measurable, remains. Philosophically this means that the concept of "nothing", no longer truly exists.

The "sliding scale" of effects coupled with the widespread dispersion of lead from silver mining, from leaded gasoline, and painted homes required managers and the public to ask "What risk?" in multiple environments with multiple individuals. The field of *Risk Assessment* was born and with it the hope that "hard" science could give us objective benchmarks for setting regulatory standards. Risk assessors modeled trajectories of toxic materials from various environmental storehouses into the human body.

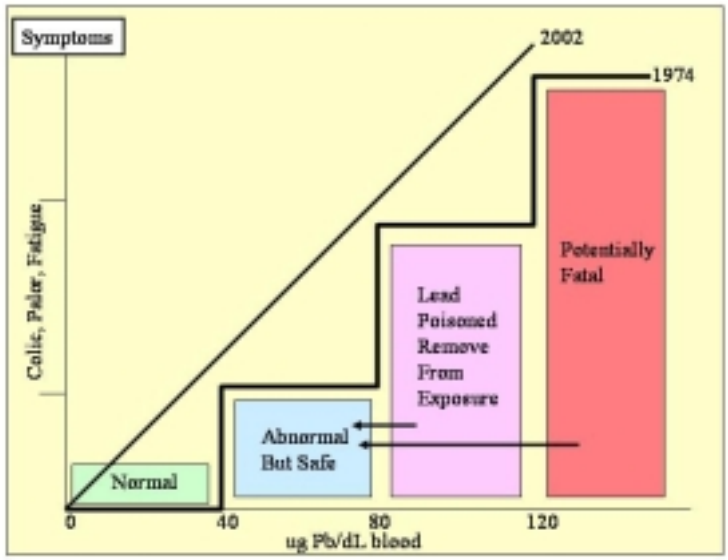


Figure 10.15 In 1974 lead poisoning management had clear stages of tolerable lead exposure. A worker could be managed by removing the worker from exposure (arrows). In 2002 lead poisoning has no lower boundary. Any exposure can be documented to affect children.

One of the difficulties in modeling uptake of lead by children and relating the uptake to blood lead of children was the multiplicity of lead sources and the multiplicity of the chemical forms of lead. Figure 10.16 details some of the various components that are associated with modeling blood lead in children as a function of environmental exposure. The model is described in greater detail in Part II of this chapter. The model attempts to determine the major route of lead ingestion from environment to child.

A major scientific/policy battle erupted over whether or not soil lead (primarily lead carbonate) or lead dust inside the house was a greater culprit. This battle was politically potent, because liability to the real estate landlord, or mining company depends very much on whether the primary source of lead uptake by a child is through dust from within the home or external to the home. If it is external (even if it was originally exterior paint) the moving finger could be pointed

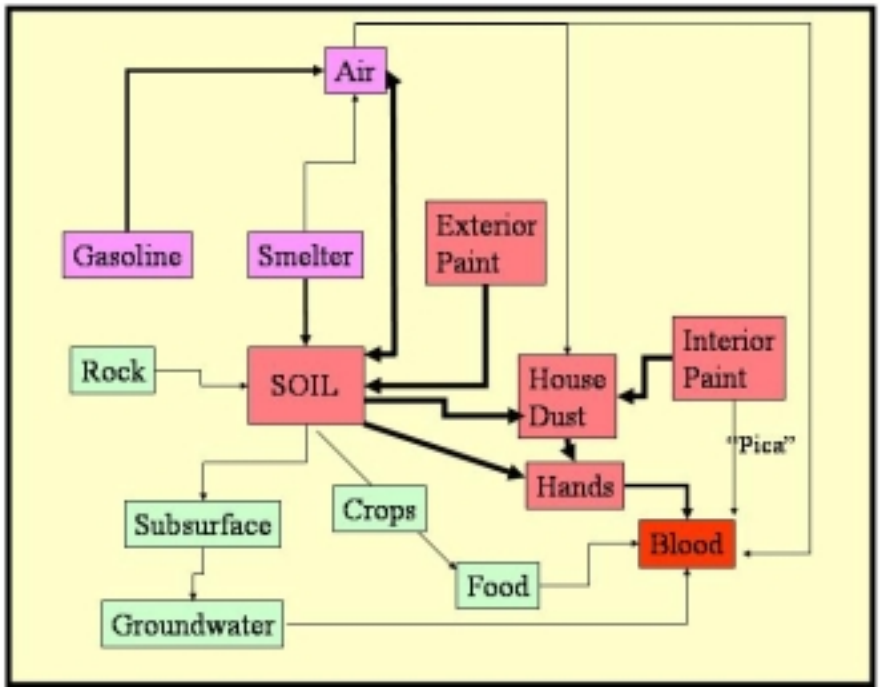


Figure 10.16 Risk assessment models attempt to quantitate the amount of lead a child will take up from various sources. Those in green are least likely to affect a child. Those in red are most likely. The model shown here does not distinguish between chemical forms of lead in the soil and indicates that both exterior and interior paint contribute to house dust via soil tracked into homes.

to a different responsible party. This prompted a slew of studies of soil chemistry and uptake models which began appearing in the late 1980s.

The biokinetic model assumes that the soil concentration, C_s is related to the blood lead concentration, PbB, by an age factor, F_{age}, the amount of lead ingested per day, and the amount of lead adsorbed compared to that ingested. This amount is modulated by a variety of factors, f, which empirically account for the chemical form and physical size of the lead (bioavailability), the soil cover, the nutrition of the child, the social behavior of the child and other factors. (Wixson and Davies, 1993). The model requires the user to know or have an accurate estimate of the amount of soil lead that the child will ingest, the soil lead concentration, and some knowledge of the type of lead ingested.

As the field of risk assessment grew so did the doubts. In particular it was found that the goal of creating a "scientific" assessment foundered not only on the particular weaknesses outlined above, but upon setting up a value system for the risk (Fischer, 2000; Slovic, 2000). The stakes in this battle were high because management of lead poisoning depends upon its definition, which becomes enshrined in legal regulations as to the amount of lead allowed in a variety of environmental matrices. Changing the definition of lead poisoning changes the scale of the problem by billions of dollars.

As opposition to assessed risk grew from various segments of society, researchers began to look into the "why" of the doubts. If one begins with the assumption that the risk level posited by risk professionals was indeed "scientific" and "valid". From this starting pont one may make several postulates. If the public does not agree with the experts on risk this could be due to the lower education level and access to information of the general public. The soltuion the discrepancy is to increase education and access to information. If the public, indeed, has access to information and is adequately educated on the topic, then disagreement about the level of risks by depend on where the costs of protecting against a small risk lie. If the public does not pay out of their own pocket (as opposed to federal tax dollars which are one step removed from ones own pocket) then the public's assessment of risk may be skewed to assuming a higher risk. The solution in this case, again, is to adequate educate the public on the hidden transactional costs of regulation.

A different strand of research found that not only do lay and experts disagree but that experts with equivalent training projected variable risk depending upon the gender of the risk assessor, and upon the employment of the risk assessor (industrial vs academic or governmental). The value-free or objective scientist was found to be non-objective. These findings correlate with the historical analysis of the controversy associated with occupational hazards of lead (the Australian case

discussed above).

From the finding of subjectivity in risk assessment two strands of thought about risk assessment arose through the late 1980s and into the 1990s. One set of research focused on the process by which the human animal makes calculations of risk. One interesting outcome of this research is the observation that risk calculation is very difficult for most individuals when risk encompasses long periods of time or involves non-ordinary (daily) contexts (Slovic, 2000, and references therein). Furthermore, when risks are calculated the problem is usually reduced to a minimal number of parameters instead of the multiplicity present in the environment. This suggests something specific about the nature of human cognition. The second strand of thought embraced the idea that risk assessment is inherently socially driven and non-scientific and began to study community derived knowledge and research. This second field of research focused on geographic specificity of environmental knowledge (Fischer, 2000, and references therein).

The Social Definition of Environmental Disease
Community Based Sampling & Democratized Science

Needleman as Provacateur

By the mid 1970s Patterson's prognostications about the effect of 1000 times elevated lead background on behavior was being tested. Some of the earliest work was carried out by Landrigan (Landrigan et al., 1975) and by Ernhard (Perino and Ernhart, 1974). Early debate in this field centered on statistical parameters necessary to establish behavioral disorder associated with subclinical lead exposure. An example is the work associated with Needleman's seminal 1979 study on IQ and lead. Needleman (Figure 10.20) and co-workers made one of the first correlations using complex statistical packages to account for socioeconomic class. He and his coworkers concluded that a measurable shift downward in IQ resulted from lead exposure (Figure 10.17) Following these reports, the public health, as opposed to occupational health, debate over lead was re-engaged in the early to mid 1980s (Marshall, 1984).

The debate on Needleman's work began with the question "Are the results real?" The average IQ of the population is a bell curve centered at about 100 with a standard deviation of about 10 to 15 IQ points (Figure 10.18). This bell curve could be the result of random variation in innate IQ. (One has an equal chance of being born with a high as with a low IQ.) Alternatively this curve could be the result of several groups of people who have been impacted differently or in a discriminatory manner. If so, the total IQ of the population could shifted by moving removing the factors which impact negatively (Figure 10.19).

How does one distinguish the difference between a random distribution and a clustering? The total variability of the population from the average IQ must be compared to the variability between clusters. If the clusters of populations show a small range of internal variability, then one is confident in assigning the variability between clusters to some external factor such as lead exposure. In order to determine how much of the variability can be assigned to randomness and how much to lead exposure one needs a fairly large population of individuals in both sets of clusters. One estimate suggests that to be 90% confident that lead has a negative impact one must have a minimum population size of 200 (Pitcher, 1989).

The first set of public health debates centered on defining who should be in a cluster. Do we exclude lead exposed children whose parents come from a wealthy background? How do we deal with the families income, parental IQ, and parental involvement? Suppose that the real reason the population varies in IQ is not due to lead exposure, but due to economic class. Inner city children have both a high lead exposure and belong to a low economic class. Which is the cause of a lower IQ score?

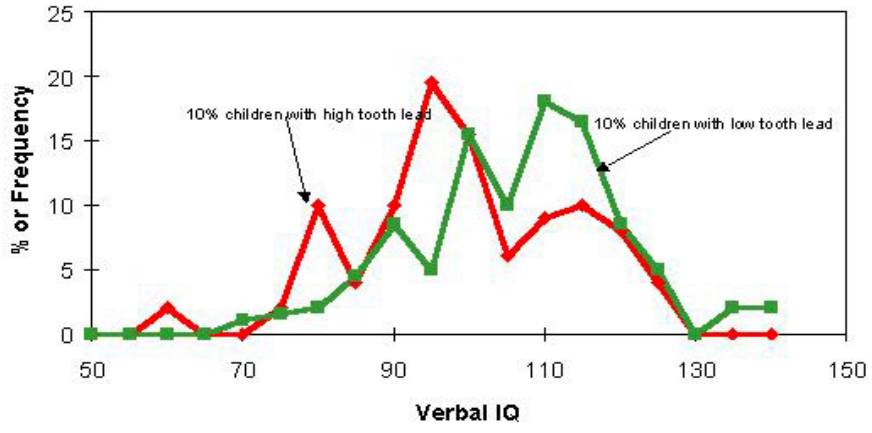


Figure 10.17eedleman's data in 1979 was plotted as cumulative percents. The data has been transcribed and replotted here as "bell" curves associated with twopopulations.

While Needleman's early work played a major role in framing the issues, the work was vigorously attacked. His statistics, his population size (Needleman's final group was <100), his control groups have been pointed out as variable, too small, and not well defined, respectively. This debate itself was fully as acrimonious as that debate which took place in the 1920s. Needleman's work was brought before NIH as

falsified in 1992. He

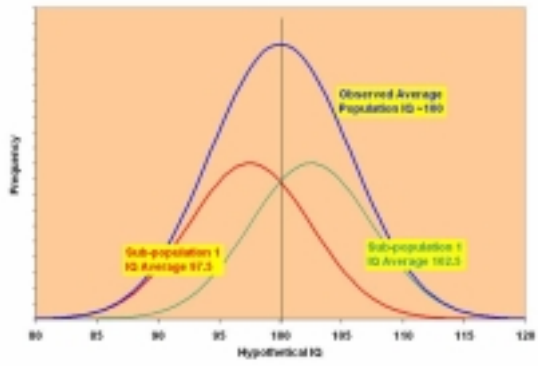


Figure 10.18wo hypothetical populations can combine to give an apparent single population with an IQ centered around 100.

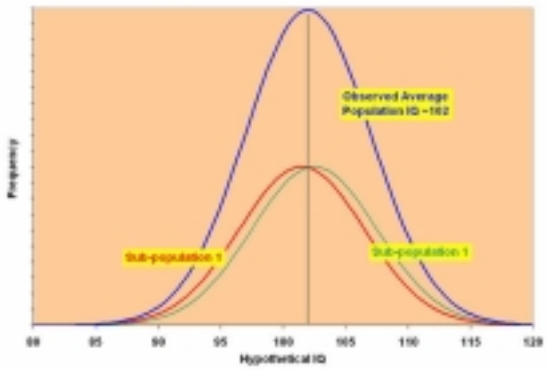


Figure 10.19f the cause of the low IQ of sub-population 1 is removed, the average IQ should shift up and the general population average will also shift.

claimed that the attackers are encouraged by the lead industry.

Scarr and Ernhard were retained by defendants of the Couer d'Alene mine in a Superfund case (Sharon Steel). They requested Needleman's raw data since it appeared that portions of the population of children had been removed, after data analysis, in such a way as to enhance the separation of IQ averages of the two groups. Needleman refused access to the data except by inspection on site of the original hardcopy (no xeroxes). Needleman sought to have those handwritten notes destroyed but was stayed in a 1991 court order.

Ernhard and Scarr alleged from the examination of Needleman's data that age was not properly accounted for, that there was exclusion of data from 40 to 50% of the children, with exclusion of lead exposed children with high IQ values. Pittsburg University held an internal hearing in which Needleman acted as a belligerent prosecutorial lawyer, although Scarr and Ernhard were not allowed legal representation (Ernhart et al., 1993). Needleman pointed out that Scarr had received $375,000 in grant funds from the Lead and Zinc Industry umbrella organization ((Needleman, 1991).

Needleman, in responding to Scarr and Ernhard, sidesteped the accusation of belligerence and belittlement. He quoted from a speech of Scarr and Ernhardt:

So we left with our notes, trembling, trembling. We went to the airport, set ourselves up with a martini at the United Airlines Red Carpet Club and wrote an outline for our report. So the report results from the two of us having done notes on all of his analyses which is what I did, and note on how he got rid of subjects that were contrary to his hypothesis.

Figure 10.20 Herbert Needleman

and appended the comment:

It is not surprising that the fevered state of mind incubated a troubled report.

The matter went before an National Institute of Health review broad where it was concluded that while Needleman was not guilty of legal definition of scientific misconduct there were questionable practices.

The subsequent 20 years of work has, however, roundly supported the original conclusions drawn from Needleman's work. Needleman has continued to press the issue. A *Journal of the American Medical Associate* publication (1996) relates bone lead content (a measure of long term lead exposure due to the long half life of lead in bone) to delinquency rates and finds a positive correlation (Needleman et al., 1996). In general, it has been demonstrated the every 10 µg/dL of lead decreases the mean or average IQ in children by 4 to 7 points. The IQ link has been demonstrated even for children with blood lead levels less than the benchmark 10 µg/dL (Landrigan and Garg, 2002; Landrigan et al., 2003). Children scored 11.1 lower points on the Standford-Binet IQ test than children with 1 µg/dL or less (Canfield et al., 2003).

Work in the late 1990s has begun to implicate lead with a number of other social ills. For example 90% of children awaiting foster care placement had blood lead levels above 10 µg/dL while children currently in foster care lower blood lead levels (Chung, 2001). Koss found the delinquent behavior is linked to prenatal lead exposure (Koss and Dietrich, 2002). Some researchers have postulated that the sharp drop in crime experienced in the U.S. in the 1990s was due not to the aging of a large youth population size (Baby Boomlet) but to the phase out of tetraethyleaded gasoline which dropped the blood lead levels of the nation in general as observed from the NHANES surveys (Eskenazi and Landrigan, 2002). A 2001 study found that the only indicator accounting for homicide rates in counties across the U.S. was air lead (Steresky and Lynch, 2001). A link has been postulated been Attention-Deficit Hyperactivity Disorder (ADHD) and lead (Rice, 2000; Tuthill, 1996). Even autism has been postulated to be linked to metal exposure. 99% of autism patients exhibited evidence of a metal metabolic disorder that impaired development of the brain and could result in hypersensitivity to toxic environmental substances. Very low levels of blood lead (3 ug/dL) have been shown to be correlated with delayed onset of puberty of girls (Selevan et al., 2003).

At first glance, the fact that lower and lower lead exposure levels do not seem to result with an "end to lead poisoning", seems counter intuitive. The "social science" studies linking ever lower blood levelvs with significant long term IQ and behavioral problems are consistent with the emerging understanding of the role of lead in disrupting caclium metabolism. As was shown in Chapter 8 calcium and GABA triggers for neural development change from the infant to the adult brain. Also shown was the fact that low lead levels cause calcium sensitive biochemical triggers to turn on when they should be off, while high lead levels cause the triggers to turn off when they should be on (Rogan and Ware, 2003).

As a consequence of this body of work, in 2002, the CDC began weighing lowering acceptable blood lead level for children from 10 µg/dL to 5 µg/dL (Lambrecht, 2002).

Predicting the Effect of Regulation

The results discussed above led to calls for more stringent regulation throughout the 1980s and early 1990s. Those calls were met with predictions about the benefits and effects of regulation.

The cost of not regulating was predicted from loss of income from an IQ reduction of the general population. This was assessed by evaluating the cost it would take to educate people more intensively and by a decreased job capacity. The educational costs in 1988 were assessed at $374 million, with the total societal cost of a decreased IQ at $6,731 million. Assessments in 2002 suggest that future earnings of children who were 2 years old in 2000 will increase between $110 billion and $318 billion, compared with their counterparts in the mid-1970s, as a result of their reduced exposure to lead in the environment. The 2002 cost of exposure was estimated to be $48.8 and $643.8 billion according to Philip Landrigan (Landrigan, 2002).

The cost of testing was estimated. In Dec. 1993, the *American Family Physician* published pediatrician Dr. Schoen's argument against blood lead screening based on the following five points. 1. Science does not support neuro-behavioral problems as the low lead levels suggested as a cut off value. 2. Blood lead levels have been significantly reduced already by elimination of the production of leaded gasolines and paints. 3. Government estimates of paint removal and lead exposure costs are low. Blood tests alone cost $33/test which translates into a billion/year price tag. 4. Pressure to treatment children will automatically follow a diagnosis of low lead poisoning. Current methods of treatment have not been shown to be efficacious and present toxic side effects as the chelators are not specific for lead but will remove essential metals from the body. 5. Where does a program of $600 billion fit into the general health care

reform scene?

The cost of preventing lead poisoning was discussed. A 1993 estimate by the insurance industry suggested that half of the US housing stock contained leaded paint. The entire removal of the paint will cost $30 to $50 billion dollars (King, 1993). An alternative estimate is $7.6 to $9.9 billion/yr for 10 years. (The insurance industry is an affected party because the abatement companies will require licensing and insurance for worker exposure and claims against faulty work). Who will bear the brunt of this cost? One concern raised was that rental property owners would abandon the property if the cost of abatement was perceived to exceed the market value on the property (Ford and Gilligan, 1988). Since the estimated cost of remediation was $15,000 per housing unit (1988, Baltimore), the property value from rent proceeds must exceed this value. Another concern might be the use of lead as a leverage tool in gentrification battles. If a owner/occupant can not afford clean up and if the law requires such clean up for rental of the second unit, then the owner/occupant may be forced to sell or tear down. (The Lindheimer Observatory at Northwestern University was torn down in 1995 because of an estimated cost of $1,000,000 to remediate the flaking lead-based paint as well as asbestos.) Can the backup industries (insurers) of rental properties keep pace with the laws? Will insurers refuse coverage, thus removing housing stock from society? If property is covered, to what extent is it covered? Who is liable, the original owner who applied the lead paint, the paint industry, or the owner who unsuccessfully remediated the lead? If it is the paint industry, do we have adequate analytical tools to determine which paint derived from which company? What is the individual price tag of a child who has been lead exposed? Do we assume that any lead exposure leads to an IQ deficit of 10 points? Is the cost of the 10 points worth $1,000,000 in damages? (One settlement put a $2.4 million price tag on a possible 5 point IQ deficit (Christ, 1992; Kerry, 1992)).

Materials began to appear to prepare the legal profession and the real estate profession for the upcoming litigation. Materials were produced by firms previously associated with asbestos removal (environmental engineering firms) which gave a concise blueprint for the management including two chapters on legal liability (Colucco, 1995).

One book, *Lead-Based Paint Hazards, Assessment and Management,* had a chapter focused on possible claims against lead abatement contractors over issues of General liability (business ownership), professional liability (errors in tests and opinions), and statutory liability (federal, state, and local administrative law). Avoiding being sued (risk management) involves not doing the work, controlling possibilities which give rise to lawsuits, and management of insurance (spread the liability out). Controlling the possibilities involve training of all personnel, keeping track of new regulations, having standard operating procedures, close supervision, and good housekeeping. Insurance is more difficult because the insurance industry is reluctant to insure because: a) the debate over lead levels has not been clarified; b) the methodology of remediation has not been established (so one may clean up by sand blasting create a larger mess and be more liable), c) increased efforts to clean up have raised public awareness which will lead to more lawsuits, and d) the kinds of decisions rendered (involving children) is likely to be a political hot potato (Anderson Jr., 1995).

A second chapter dealt with the liability of the landlord (Placitella and Sugarman, 1995). Causes for lawsuits are negligence, strict liability, breach of contract and violations of landlord-tenant law. Negligence arises from a breach of duty that a reasonable man might have to provide a safe and sanitary condition. Negligence can be proved that collecting rents in person should allow a reasonable man to see that deteriorated paint exists regardless of whether it is known that the paint is leaded or not. The law also requires inspection of homes in which children reside who have been found to have high blood lead levels. This is a statutory violation and can be used to provide evidence against a "reasonable man" Claims have also been made as to negligent abatement. The authors of this chapter argue that the sued landlord should immediately counter sue for negligent parent status, which will pose such a financial burden that the original lawsuit will be withdrawn. The chapter also notes that the another ploy frequently used is to portray the landlord as a slumlord, a label which will stick more likely than not.

Estimated Lbs White Lead/Housing Unit

1914-23	110
1920-29	87
1930-39	42
1940-49	22
1950-59	7
1960-69	3
1970-79	1

Controlling the Face of the Debate

To counteract the dollars and cents arguments made above, activists have worked to control the face of the debate. Because lead does not smell and does not produce a visible gas, and because subclinical lead poisoning does not manifest itself "spectacularly" it is difficult to keep the cause in the public sphere and, from there, in the legislative docket.

Community activists worked to educate both their own public and the wider public on the issue of childhood lead poisoning. Literally hundreds of press stories over the past 20 years have been devoted to putting a "face" on the issue. Excerpts from one such case follows from the Providence Journal, May 16, 2001:

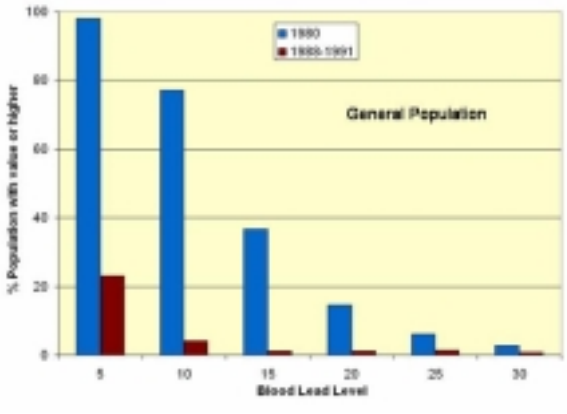


Figure 10.21 HANES data for the general population. Data represents % of population with blood lead levels equal to or larger than value given.

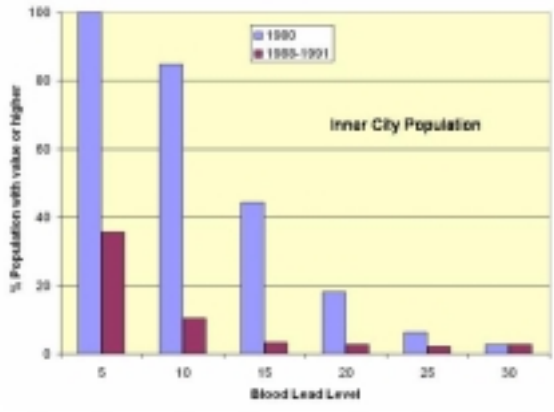


Figure 10.22 NHANES data for inner city population. Values for blood lead are higher at every level.

Struggling for the right words
Peter B. Lord

"Jet," says Ashley Matthews.

She tries to write the word, but instead of a "J" she writes a "C" with a dot on top.

When her teacher points out that the letter isn't quite right, Ashley giggles, scratches her head, and reaches for one of two big erasers.

She uses the erasers, baby talk, and her sweet disposition to apologize for and correct a string of mistakes as she struggles through the second-grade phonics workbooks with her teacher, Loire Levesque.

They are working in a closet-size room on the second floor of the Providence Street School Annex. Ashley, wearing a gray T-shirt adorned with little monkeys, faces Levesque. Both sit on the tiny chairs you see only in elementary schools and patiently work on Ashley's words.

Ashley is almost 8 years old, but she has trouble saying certain sounds, particularly "th" sounds. She struggles with sentences and with grammar. And it's difficult for her to "retrieve" words, Levesque explains. Like an older person losing her memory, Ashley looks at things and knows what they are, but just can't think of the words for them.

Because of her learning difficulties Ashley gets more than five hours of special-education help every week outside her regular classroom. Some sessions are one-on-one with Levesque, who uses much the same strategies she learned working primarily with elderly people who have suffered brain damage form strokes. Ashley also spends hours as just one of the three students with another teacher, Marie Strumolo.

Her mother, Brenda Matthews, likes Ashley's teachers but says Ashley is learning very slowly and has a long way to go. "She's always taking her books around. She looks at the pictures and makes up her own stories, because she can't read."

Ashley is lead poisoned.

No one can say how much Ashley will learn, or how far she'll go in school. But it's clear she faces huge obstacles.

*One recent morning at her home, Ashley played on a rug as Matthews talked about what she was like before the move [*that caused the lead poisoning].

On that day, Ashley stared at a book, and appeared to be daydreaming. She didn't say a word.

"She was learning faster than the other two," Matthews said. "She could count to five. She knew most of her primary colors and part of the alphabet."

Mathews told a psychologist who evaluated Ashley that she was walking at 12 months and speaking early.

But she stopped reciting the alphabet, "or doing her colors. I figured it was the move," Matthews said.

In early 1998, Ashley had a routine physical exam. Then came the bad news.

"I got a call from Midland Medical saying I had to rush her to the hospital. They were in a panic. They scared the hell out of me."

Matthews took Ashley to Hasbro Children's Hospital. Her lead level was found to be 61, about three times the level considered significantly poisoned.

Information and strategies to place such articles are shared through the Alliance to End Childhood Lead Poisoning, founded in 1990. Many of the cases come from parents who have been turned into activists by their own experiences.

Many of the cases of lead poisoning in the U.S. occur in the inner eastern U. S. city. The general cause is that the housing stock in eastern cities contains a large number of older and poorly maintained homes. Residential sections of various cities have older housing stock which correlates with higher incidence of lead poisoning. The urban/suburban divide can be seen by looking at data from NHANES which documents blood lead values of the nation in approximate 10 year increments (Figures 10.21 and 10.22).

Some reports put a "Yuppie" spin on the lead poisoning accounts in order to balance the perception that lead poisoning is a "class" disease that affects only children of the poor [which can be a code word for "black"]. If the disease is only that of the poor it can be rationalized as a product of poor hygiene. An example of a "Yuppie" spun article occurred in the Post-Standard of Syracuse, New York, July 23, 2001:

Family learns lead ignores the wallet
By Luis Perez

Lisa and Friz Blok learned a painful lesson: Lead poisoning is not limited to poor people and run-down city neighborhoods.

Their son, Zachary, a blond-haired toddler who loves playing with his Max Steel action figures, was poisoned in a house in a stable Buffalo neighborhood. The family moves to East Syracuse, expecting to get away rom lead. But Zachary found it there, too.

"People think it's only something in the inner city," Fritz Blok said. "And it's not. It's everywhere."

Zachary, now 4, still has unsafe lead levels - despite more than two years of treatments and precautions. He's doing well in school, said the Bloks, who have bought a home in East Syracuse. But they wonder if the speech therapy he needs is the result of lead poisoning.

Lisa Blok was not concerned about lead when she moved into her Buffalo home in 1997. On Frontenac Avenue, houses sell for $50,000 to $100,000. She described the house as immaculate, with no cracking or chipping paint.

But the hazard to Zachary and his sister, Lauren, now 7, was the lead-laden dust on windowsills and the front porch, and lead in the soil around the garage in Buffalo. Inevitably, that lead dust settles on the floors and toys.

Zachary crawled on those floors. He played with those toys.

In December 1998, the family learned Zachary had almost four times the acceptable level of lead in his blood - a condition the Bloks knew nothing about.

Zachary looked healthy, Lisa Blok said. He showed no symptoms. Yet the toxic element flowed through his blood.

Lisa Blok has channeled her experience into founding the Syracuse chapter of United Parents Against Lead, a national parents' advocacy network fighting lead. She has taking training in lead poisoning in hopes of education school staffs, public health officials and other parents.

"I want to be able to look at him (Zachary) when he's 15 and say: 'Mommy made a difference,' not only in his life, maybe in one other child's, too," Lisa Blok said."

The opposing side, concerned with cost and regulation, put a different face on the issue:

March 19, 2001
Getting the Lead Hysteria Out, Steven Milloy on Fox News website

The Centers for Disease Control and Prevention reported last December that levels of lead in children continue to decline. That's a scary thought to

the lead-heads, who this week launched an effort to "head off" any further good news about lead.

To spotlight the upcoming publication of a new study by activist-researcher Dr. Bruce Lanphear, of the Cincinnati Children's Hospital Medical Center, the Alliance to End Childhood Lead Poisoning sponsored a press conference.

Though the study will not be published/available for review by the public for weeks, Dr. Lanphear announced he had linked learning problems in children to extremely low levels of lead exposure, on the order of 5micrograms of lead per deciliter of blood (ug/dL) and below

Dr. Lanphear shrilled, "Lead is a major cause of many diseases of industrialization....There is no safe level of exposure to lead...Each of us has been adversely affected by lead and will be adversely affected until the day we die." Dr. Lanphear called for a total ban on the commercial use of led.

But Lanphear's alarmism comes across as a lead balloon.

There is no question that "lead poisoning" can be harmful. But what is "lead poisoning"? Physicians and the lead-heads disagree.

Medical treatment for "lead poisoning" is recommended for blood lead levels above 45 ug/dL, according to the American Academy of Pediatrics. Environmental intervention -such as cleaning and repairing a home with deteriorating lead paint - is recommended for blood lead levels over 25 ug/dL.

But such high blood lead levels are rare and are becoming more so.

The CDC reported last December a 28 percent drop (from 10.5 percent to 7.6 percent) in the number of children with blood lead levels above 10 ug/dL. The CDC reported a 20 percent drop (from1.5 percent to 1.2 percent) in the number of children with blood lead levels above 20 ug/dL.

Up until 10 years ago, such numbers would be cause for celebration at CDC. But in 1991, the lead-heads convinced CDC to reduce the level of concern for blood lead levels from 25 ug/dL to 10 ug/dL - that's where the lead controversy enters the junk science world.

Lead-heads claim low-level lead exposure is associated with lower IQ scores and behavior among children. - a claim launched by a controversial 1979 New England Journal of Medicine study by University of Pittsburgh research Dr. Herbert Needleman.

Not everyone was impressed with Needleman's work, though. Critics uncovered many problems. Needleman didn't control for the confounding factor of child's age. Factoring in age yielded few significant results. Needleman also excluded from his analysis children who were "lead poisoned" with out impaired intelligence. He also omitted other results that didn't support his conclusion.

Needleman was subsequently accused, but not convicted, of scientific misconduct. His work, however, was never vindicated.

As pointed out in the New England Journal of Medicine, " the investigative bodies found Needleman's studies scientifically flawed...involving a "pattern of errors, omissions, contractions and incomplete information." The University of Pittsburgh...stated that had Needleman accurately described his methodology and subject selection, he "would have risked rejection" of his article by the New England Journal of Medicine. In addition, the [federal] Office of Research Integrity cited misplotted graph points, which were found 'difficult to explain as honest error'..."

Subsequent studies, generally conducted by activist-researchers such as Lanphear, purport to support Needleman's original claims. But the studies suffer from the same basic flaw: Their weak statistical associations between blood lead levels and learning and behavior problems could easily be explained by socio-economic factors not adequately considered by the researchers.

After all, who's surprised that poor, inner-city kids underperform on cognitive tests?

Exposure to lead is to a problem for the vast majority of Americans and their children. More than $100 million is spent every year to monitor and reduce lead exposure among the populations most at risk for lead poisoning. The CDC data shows that progress is being made.

This progress is especially notable since commercial lead use has never been greater. Though lead is no longer used in U.S. gasoline and paints, lead has many other uses. Eighty percent of lead use is in automotive-type batteries. Leaded glass makes it possible to watch television and use computers safely. Sadly, though, the lead-heads only see progress on lead exposure as a threats to their viability. They admitted as much during the press conference stating the purpose of Lanphear's study was to prevent the CDC report from being interpreted "incorrectly."

Ironically the lower child blood lead levels go, the more shrill the lead-heads get. Lanphear noted that technology now enables the detection of lead in blood to the nano gram level - a billionth of a gram or 1,000 times lower than the microgram level.

The lead-heads apparently plan to be around

for a while.

Steven Milloy is a biostatistician, lawyer and adjunct scholar at the Cato Institute, a conservative U. S. think tank.

Management

Public health management of childhood lead poisoning has taken several main trajectories. The first, and least desirable, is the goal to test all children at an early age for blood lead, in essence, using children as lead detectors. The second addresses testing of housing stock and the third addresses updating of housing stock.

Strategically, the easiest blood lead sampling program is to require blood lead tests for all incoming children into the school system at kindergarten. Blood lead testing can be piggy backed onto requirements for various immunizations. The testing can be enforced by school admission requirements. Unfortunately, testing at the age of 6 or 7 is too late.

An alternative strategy is mandatory universal testing at an earlier age, but this runs up against either a major shift in doctor training and practice and/or some sort of regulatory stick. Parents are not the only uninformed population. Many physicians feel that with the near cessation of acute and/or fatal lead poisoning cases, lead poisoning has been solved. Further, two other issues may arise. Even if the physician is aware of the asymptomatic lead poisoning they may be reluctant to test for and then report such cases because elevated blood lead usually involves a requirement to move and some sort of legal action in housing court. Secondly, blood lead testing is not automatically covered by all forms of insurance and the issue of reimbursement for costs may arise.

A less universal strategy may be to piggy back blood lead testing into programs that serve the high risk populations, such as federal nutrition and food assistance programs like WIC.

Several studies have been published which attempt to take a more targeted approach to blood lead sampling. In 2000, the CDC recommended universal screening when the prevalence of elevated blood lead (>10 µg/dL) is above 12% of the area. For prevalences less than that, targeted screening is permissible. An example is a 1998 report "*Cost-effectiveness Analysis of Lead Poisoning Screening Strategies Following the 1997 Guidelines of the Centers for Disease Control and Prevention*" (CDC). This study lists the costs of various lead poisoning screening strategies shown in Table 10.2. The article concluded that a cost per child for screening via venous testing would run $47.19, $69.50, and $215.97 per child from low to medium to high prevalence areas (Kemper et al., 1998).

Table 10.2 Estimated Cost of Lead Poisoning Management
Source: Kenys et al. 1998

Test	Baseline Estimate $	Range,$
Venipuncture	6.53	3.00-15.00
Capillary Blood sampling	3.27	1.00-10.00
Lead assay	17.42	5.00-75.00
Risk Assessment Questionnaire	2	0.25-3.00
Nurse-only visit	32.00	15.00-50.00
Physician visit	80.00	30.00-120.00
Environmental Investigation	335.00	200.00-1000.00

Even once the data is collected managing the data is difficult. A case in point is the D.C. Department of Health's blood lead testing program. Their pre 1999 data was recorded manually and are deemed unreliable (Chan, 2000). Although the area put into place a 1983 law requiring testing of all children entering early-childhood programs, little of the mandated screening took place. Similarly, a report from the New Jersey Department of Health and Senior Services on Dec. 18, 2001 found that not all targeted children were tested, although the testing levels had risen. 34% (75,147) of all 1 and 2 year olds were tested (Ben-Ali, 2001).

State laws enacted near the millennium include the July 13, 2001 Missouri law for targeted testing of children 6 months to 6 years old in high risk areas (older houses under renovation and children who have lead poisoned siblings) (Parish, 2001a). A Maine law was enacted May 17, 2002 which required the Department of Human Services to create a list of screening questions, based on a similar list proposed by the Centers for Disease Control. Based on the questionnaire doctors can take a blood lead test (Taylor, 2002). Maine's law was designed to address the fact that physicians are unconvinced of the severity of the problem. Use of a questionnaire will allow physicians to "ease into higher screening rates" (Haskell, 2002). Missouri passed a law May 21, 2001 which targeted children considered at high risk including those who live in housing built before 1950 and those who have sibling or playmate who were poisoned (Parish, 2001b).

Figure 10.27 Children can be trained to accurately sample dust within their homes. Photo: Loyola University Instrumental analysis class with Orozco elementary school children, 2000.

Significant lobbying effort has been put into targeting perceived "deep-pockets" as the source of funding for the blood lead tests. An example is a national law requiring State Medicaid programs to pay for blood lead testing. A trial balloon was floated by the second Bush administration in April, 2002 which would have ended the federal requirement for Medicaid payments for blood lead testing (Parker, 2002). Various lead poisoning prevention activist groups mobilized for letter writing campaigns within the day. A month later (May 15, 2002) the HHS backed off the plan.

Medicaid serves approximately 6.3 million children under 6 of whom approximately 20% are screened. (The cost of this screening using the values shown in Table 10.2 above amounts to $8.5 million dollars). The ability to enforce screening is difficult. Ruben King-Shaw, deputy administrator for the federal Centers for Medicare and Medicaid Services told the Senate Banking, Housing and Urban Affairs subcommittee that "We have very few powers to enforce this kind of provision." He said that virtually the only enforcement mechanism available to the agency for health care providers who do not perform the screening is a suspension of Medicaid payments. "I don't know that people want to see that as an enforcement measure," he said (Robinson, 2002).

The G. W. Bush administration has sought to reduce the regulatory burden associated with lead poisoning management strategies. Once such policy change enacted by the Bush administration is related to who gets to advise and control the science used for setting policy echoing the earlier politics over the composition of the National Academy of Sciences committee composition which excluded Clair C. Patterson in the early 1970s.. In 2002, 3 science oversight committees (patients' rights, genetic testing industry oversight, and environmental chemical effects) have had the science advisory board changed over the recommendations of the agencies who rely upon their science (Stolberg, 2002; Weiss, 2002). The CDC staff nominations for the CDC Advisory Committee on Childhood Lead Poisoning Prevention (ACCLPP) were overruled by Secretary of Health and Human Services, Tommy Thompson. Rejected nominations included Dr. Michael Weitzman, Pediatrician in Chief at Rochester General Hospital; Dr. Bruce Lanphear; Sloan Professor of Children's Environmental Health at the University of Cincinnati; and Dr. Susan Klitzman, Associate Professor of Urban Public Health at Hunter College. Secretary Thompson's appointees include Dr. William Banner, a witness for the lead industry in legal trials; Dr. Joyce Tsuji a consultant to ASARCO and DuPont; and Dr. Kimberly Thompson of the Harvard Center for Risk Analysis. Editorials in *Science* have excoriated Bush's appointments. Even the *Wall Street Journal* published an op ed piece in opposition to the appointments. The strategy harkens back to the discredited NAS committee of 1970 headed by tetraethyllead industry researcher Kehoe. That committee dismissed fears of envrionmental contamination from leaded gasoline.

The Bush administration was heavily lobbied to reduce requirements to report on lead. As part of the revamping of regulations, the EPA in 1999 moved to make small amounts of lead use part of the reporting requirements of the Toxics Release Inventory (TRI). 67 industry trade associations wrote Christine Todd Whitman (head of EPA) urging her to delay or suspend new rules about lead pollution. EPA rules issued in Jan. 2001 dropping the threshold amount from 10,000

pounds per year use to 100 pounds per year use of lead for reporting. Despite lobbying from industry, which felt that such reporting would be onerous and of little public health benefit, the requirements were upheld (Allen, 2001). However mining groups won one battle in which subsurface reduced ores dumped at the surface and subject to oxidation did not have to be classified as toxic materials used in an industrial production process.

Community Based Sampling: Democratized Science

While activists have worked hard to get various forms of universal blood lead screening it has always been a "second best" strategy because it makes the children biological environmental samplers. Getting an accurate environmental survey of homes, housing stock, and soils, however, is fraught with legal implications and problems in accessing sites. A landmark bill in 1992 addressed this issue.

This bill requires that any real-estate transaction involving pre-1978 property be accompanied by paperwork signed at the point of transfer which states that the property has not been tested for lead. If it has been tested the seller is required to disclose the amount of lead in the property.

This law, like that of universal blood lead testing, has not been practiced all of the time.

In order to comply with the law it was envisioned that a significant increase in testing and remediation for lead would follow and the law made provisions for certified samplers and certified remediation workmen. The apparent result of the law was to create a financial barrier circumventing testing. In September 2000 HUD revamped its regulations with respect to lead. The new strategy hopes to increase by 20 times the number of lead samplers with subsidized training for the samplers (Ryan and Scott, 2000). Community based sampling is envisioned as requiring less sampling training and less cost than certified samplers.

The law also required the EPA to set national standards (Section 403) of Title X. The standards were released nine years later in Jan 2001. Dust-lead hazards were set to 40 $\mu g/ft^2$ on floors and 250 $\mu g/ft^2$ on window sills. The floor standards were lowered from a 1994 standard of 100 $\mu g/ft^2$ and from a proposed 50 $\mu g/ft^2$ to be compatible with HUD regulations. Soil-lead hazard was reduced to 400 ppms in play areas. Other soils may remain at a bare level of 1,200 ppm.

This ruling was appealed in March 2002 by the National Multi Housing council, the National Apartment Association, and the National Leased Housing Association with the Battery Council International as an intervenor. The landlord groups argued lead paint hazards (Section 403) were limited to lead in dust and soil that originated in lead paint. The EPA argued that the standards apply to lead regardless of source. Any interior dust or bare soil lead above the set limits are in violation of the regulation. The court ruled in favor of the EPA June 2002.

In 2002 the EPA also dropped the restriction that only a certified risk assessor performing a full risk assessment can identify a lead hazard. Shortly thereafter (September 2002) Milwaukee authorized warrants for carpenters to enter and inspect and perform remediation to specified homes with the bill attached to the property tax of the homeowner. The response outraged apartment complex owners:

Looking at the property rights and constitutional rights, having to serve a warrant to conduct maintenance on a property does fall within the scope of city government

said Tom O'Brien, president of the Apartment Association of Southeast Wisconsin (Held, 2002).

Remediation

As indicated by the Milwaukee case lead poisoning prevention management ultimate goal is removal of lead, not just "living with" lead. Since the enactment of Title X various amounts of money have been earmarked toward full remediation. A few examples follow.

2000: HUD announcement #00-227 details $105 million for lead poisoning prevention programs of which $84 million was to conduct lead testing in federally funded low income housing.

2001: FY Administrative budget called for $164.5 million for lead hazard control grants and enforcement. June, 2001: HUD release #01-057 indicated that HUD expanded courses to train thousands of workers in lead safe work practices. The goal was 30,000 new workers. Oct. 2001 HUD release # 01-108: HUD awarded over $67 million to protect children from dangerous lead and other environmental hazards. $59 million was earmarked for lead removal from 7,000 privately owned homes in 16 states.

2002: Feb. HUD announced $10,000 for 60,000 lead dust clearance tests that follow remediation. April: the CDC announced awards of $1.7 million for development of alternative surveillance assessments or to conduct evaluations on the effectiveness of lead

screening.

Enforcement of Notification

One way to use the disclosure law of 1992 is to leverage lower fines in return for abatement which results in permanent lead removal. HUD and EPA moved into this proactive position following the setting of the section 403 standards.

The Chicago EPA Region 5 seems to be particular fond of this strategy. An Oct. 2, 2001 settlement was announced against Wolvin Levin, Inc., East Lake Management and Development Corporation, and Oak Park Real Estate. The three agreed to test for and clean up any lead-based paint. Wolin Levin in addition agreed to pay $100,000 to the Chicago city Health Department as part of a child health improvement project. East Lake agreed to give $77,000 to a community based health center for free blood lead testing.

An April 17, 2002 complaint was filed against Belair Edison, Inc., which owned and operated five pre-1950s housing units by Maryland Department of the Environment. A penalty of $41,350 was issued.

In Chicago a Hyde Park Realty was assessed a $20,000 fine based on its ability to pay. The company managed more than 1,000 residential units in 40 buildings. The company was ordered to partner with the Chicago Department of Public Health to hold workshops for its tenants (PRNewswire, 2001).

In Denver, in Jan 2002 the EPA and HUD announced a landmark lead disclosure settlement involving more than 130,000 families in 47 states against the Denver-based Apartment Investment and Management Co. (AIMCO) (PRNewswire, 2002). AIMCO allegedly failed to warn tenants that their homes may contain lead-based paint hazards in violation of the Residential Lead-Based Pain Hazard Reduction Act of 1992. Similar leveraging occurred in Los Angeles in 2003 (PRNewswire, 2003).

Title X simply requires notification of knowledge upon a real estate transaction. The law, in the minds of many, simply encouraged landlords and owners to avoid knowledge. If the landlord was "technically" ignorant then they had no requirement to constructively act. Several laws addressed this "loophole". In May 2002 San Diego, California indicated that it would begin to prosecute landlords who did not actively remediate their property (LaRue, 2002).

One important legal battle has been carried out in New York. In 1959, shortly after the Baltimore studies of the early 1950s were confirmed, the city banned the use of lead paint in residential dwellings. In 1970 it passed the Lead Paint Poisoning Prevention Program. This law was amended in in 1982 to hold landlords of pre-1960s housing stock liable for lead poisoning of children unless the owner had removed or covered the lead paint (Pedro, 2000). The law is termed a "constructive notice". A 1999 law, Local Law 38, reduced the landlord obligation to a once a year inspection for peeling paint (Bosch, 2002). The law was overturned by a judge in 2000 on the grounds that no environmental impact study was taken in setting the law (Lambert, 2000).

Outside of the city the reverse trend took place. New York State passed the Control of Lead Poisoning Act (CLPA) in 1970 which directed the State Commissioner of Public Health to develop a comprehensive statewide plan to reduce and prevent lead poisoning. It mandated blood lead testing of children, and inspection for the presence of lead in residential buildings. In many communities the law held that the landlord had to have "actual" proof of lead within the house. The general knowledge of lead hazards in pre-1978 housing and the knowledge of lead's danger to children do not constitute actual knowledge. In Chapman v. Silber 128, 2001, a landlord was sued for negligence and was guilty. A lower court dismissed the case. The N.Y. court of appeals overturned the lower court hearing, reversing a trend of prior legal determinations. The court further said that in the absence of state law stating when landlords were to be held liable it would provide guidelines (Romano, 2001; Stashenko, 2001).

The Predicted Lawsuits Begin

In the early 1990s it was predicted that the only sure outcome of regulation was legal action, in which lead was turned to gold by legal alchemy. Sandler, called lead "legal alchemy", a way in which lead is turned to profit the legal profession (Sandler, 1993). This view point was based on the past history associated with asbestos removal.

As predicted, the setting of standards and the implementation of childhood lead prevention programs, spawned a spate of lawsuits. There were generally four targets for the lawsuits. The first is the landlord, the second public housing, the third the lead prevention agencies, and the fourth and largest target, the lead industry. The lawsuits targeting the lead industry will be discussed in Chapter 10.

Landlords. Many of the headline grabbing cases involved large landlords. In Dec. 20, 2001, a

property manager and his company in New Hampshire plead guilty to lead poisoning charges. The manager was sentenced to 15 months in jail and a fine of $40,000. The company faced fines of up to $3.2 million for a case involving the death of a 2 year old child (AP, 2001). A landlord in D.C. was sentenced to a fine of $50,000 and 2 years in prison for failing to disclose lead hazards in homes. Six children living in those homes were subsequently found to have elevated blood lead levels (Casteneda, 2002).

One case involved a suit against a landlord who required pre-testing of blood lead before rental. The landlord would only rent if a child's blood lead was lower than 9µg/dL. A potential renter's child was found to have 17 µg/dL and was denied the right to rent. The tenant sued for discrimination and won (Biemer, 2000).

Laws to protect landlords are beginning to surface. An example is that which went into effect Jul 2000 in Virginia. The law makes it more difficult to prove that property is improperly managed (Cohn and Crockett, 2000).

Public Housing Agencies. In many locations public housing agencies have been targeted for poor management leading to deteriorating paint and subsequent childhood lead poisoning. Chicago is, apparently, not one. An urban myth suggests that the Chicago public housing's apparent lack of lead contamination is traced to the 1950s boss of the public works who billed for the expensive lead paints but delivered cheaper alternatives.

Other locales were not so lucky. Montgomery, Alabama lost a lead poisoning lawsuit Oct. 4, 2002 for $860,000. The jury found the Montgomery Housing Authority had failed to warn the family and repair and maintain the apartment (McGrew, 2002). In January of 2002 a Maryland mid-level appeals court ruled that renters poisoned by lead paint have a right to file negligence lawsuits against public housing agencies. The agency's lawyers had argued that the it should have blanket governmental immunity from lawsuits and that it should not be forced to pay any judgements because its insurance coverage for lead cases had been canceled by the insurance carrier (Roig-Franzia, 2002).

Childhood Lead Prevention Agencies. Two high profile cases involving agencies involve San Francisco, Ca. and Albany, N.Y. The Arbor Hill Concerned Citizens Neighborhood Association (AHCCNA) and the Natural Resources Defense Council (NRDC) joined to sue Mayor Gerald Jennings of Albany May 22, 2002 for gross mismanagement of a program designed to reduce children's exposure to the dangers of lead paint. The City had received $13 million for removal or containment of lead based paint in older and low income homes. The community groups and NRDC allege that the city violated lead safe work practices during remediation. New York state was also in the spotlight over the level of blood lead screening. The state set requirements for screening of children and pregnant women in 1992, with the Health department targeting all children at age 1 and 2 and of at risk children at ages 3 through 5. The New York Public Research Group (NYPIRG) believes that screening only increased slightly after the enactment of the law (Caher, 2001).

The State Department of Health Services of California was similarly sued by Public Advocates Inc., Bay Area Legal Air and the National Center for Youth Law. The suit alleged that the July 1993 law to set up and screen children was not enforced (Finz, 2000). In response regulations went into effect in October but were ruled inadequate by a Superior Court Judge. The regulations did not include penalties for doctors who fail to comply, for instance, and left it up to doctors to decide what to do if lead is found in a child's blood (Hill, 2000).

A Case Study: Local control, risk, chemistry, lawsuits, Superfund, and Title X

Case Example on Defining Lead Risk

A case example is that of Coeur d'Alene, Idaho (Steele, 2002). This highly productive mining area began operation in the late 1800s as a silver/lead mine concern. The town of Kellogg was built around mines and the Bunker Hill smelter. Slag heaps were built up within the town. Soil lead within the town measured in the 1970s ranged from 626 ppm to 18,600 ppm. Until 1968 debris was also dumped directly to the river. 70 million tons of debris moved to the Coeur d'Alene lake and on to the Spokane River. Development of a flotation process to extract more metals in 1912 and dumping of the "fines" into the river sparked lawsuit down river from farmers for death of animals and crops. In 1968 the Bunker Hill smelter was taken over by Gulf Resources. A fire in 1973 destroyed the filters in the smelter but Gulf proceeded to operate the smelter for a year. An estimated 25-30 tons of PbO was deposited per square mile. Children in the vicinity of the smelter got dizzy and developed stomach cramps. (An internal document at the Houston headquarters for Gulf calculated that at $5 to $10,000 per lead poisoned child the cost would be

$7 million compared to the expected revenues of $259 million.) Those children made ill in the exposure as adults in 2000-2002 suffer from memory loss, and other long term neurologic problems associated with severe lead poisoning.

In the 1970s and early 1980s local residents pressured the EPA to force compliance with clean and water standards. Simultaneously, foreign lead began entering the country in mass leading to a decline in lead prices. Miners refused to accept wage rollbacks (Schneider, 2002). In 1981 Gulf Resources and Chemical Co. declared bankruptcy, terminating 2,100 jobs. Assets were sent offshore. The date is referred to as "Black August". The governor appointed a committee to sell the smelter which was bought by local businessmen in 1982. In 1983 the EPA declared the largest ever superfund site in and around Kellogg. The 21 square miles so designated was known as "The Box". The EPA set the action limit for soil lead values within The Box at 1000 ppm and planned to remove soil from the 2,100 home properties within The Box.

In 1986 Idaho settled with the mining companies for $4.5 million as part of the cost of removing soil from yards. The soil was deposited on the slag heaps for capping. 185,000 cubic yards of dirt, some with 74,700 ppm lead, were removed by 1993. A consent decree was achieved for work within The Box in 1994.

This initial phase of work was tolerated by the local community, despite the disruption of their property and homes. Hostility erupted, however, when the 1,800 member Coeur d'Alene Tribe sued over contamination of the Coeur d'Alene lake and the associated waterways. The tribe sued Hecla, ASARCO, and the Coeur d'Alene Mines in 1991.

By 1996 the EPA felt that it would be dragged into the civil lawsuit by the Coeur d'Alene tribe and decided to join as a plaintiff. In 1998 the EPA announced a basin wide study of the entire watershed. ASARCO and Hecla stocks dropped in value because of worries about increased liability. ASARCO became the target of an unfriendly takeover by Grupo Mexico. Hecla was forced to sell assets. In 1999 the Sunshine Mining and Refining Co. settled.

The trial commenced in January 2001 with much behind the scenes manuevering with Gov. Dirk Kempthorne working as a broker (Russell, 2001b).

At the same time, settlements in the EPA Superfund case were proceeding. In March the Coeur d'Alene Mines settled for $3,871,924 and a % of future profits (net smelter returns on silver if silver broke $650/oz..). In August the Hecla Mining Co. settled for $138 million with more to be paid if the company began to turn a profit again. All of the settlements were thrown out in October 2001 when U.S. District Judge Edward Lodge voided the 1994 EPA consent decree because the EPA had "broken faith" by moving its activities outside of The Box. A year later (August 14, 2002) federal, state, and tribal groups signed a plan to put local and state authorities in charge of the cleanup of the watershed leaving EPA in charge solely of The Box. One week later Hecla backed out of its $138 million Superfund settlement because the deal was "no longer favorable to the company." They felt that the costs of cleanup would drop dramatically with the EPA out of the picture (Russell, 2001a). By Mar., 2003, a plan floated with the ultimate goal of rescinding Superfund status was criticized by nearly every stakeholder as unlikely to lead to de-listing of the community (Steele, 2003).

Local citizens were sharply divided over the Superfund designation and the activity of the EPA. Citizens for EPA Accountability Now was organized to fight expansion of the Superfund area. Bret Bowers of the organization said:

Superfund was intended to clean up 20-acre industrial sites. Superfund is not intended for a 1,5000-square-mile region. The EPA would like people to believe we have this major health concern, when the fact is we should be celebrating the vast gains we have made [inside The Box]. We have community leaders up and down the basin saying we know the Superfund process is a mess and we don't want to go through it.

Robert C. Huntley, a former member of the Idaho Supreme Court, who worked for mandating blood lead testing said,

It's really a Chamber of Commerce-type thing, where we don't want to admit we have a problem because it would have an adverse effect on tourism.

There's a long list of things that are far greater risk [to children] than heavy metals. There is older housing in the Silver Valley where lead paint is an issue. You can't just automatically conclude that it's all from meandering sediment
said, state Sen. Jack Riggs, a physician (Murphy, 2000).

Robin Stanley, superintendent of Mullan Schools, near Kellogg, is more concerned about poverty in the area.

What's scary about this is, I see kids coming to school and open up a lunch bucket with one slice of bread in there. That bothers me, I don't see kids with lead poisoning. I see them coming with clothes that have holes in them and shoes that aren't protecting their feet from the cold because their parents can't get a job.

He believes that the lead sulfides found in the valley aren't bioavailable (Picard, 2000). His sentiments are echoed by Dale Lavigne:

I was born and raised in this valley. I've been in the pharmacy business since 1953. I've never seen a kid in this valley who is what you really call leaded. These people who say there's mental retardation and all that? That is bullshit.

One local newspaper writer suggested that property owners should be *reaffirmed their right to shoot trespassers on sight if they are agents or employees of the EPA*.

The town of Couer d'Alene along the lake is a "quaint" village with antique stores and art galleries. It lies next to the Coeur d'Alen Resort. Members of that community were most enraged about increasing the Superfund designation to the watershed.

Superfund designation puts the entire soical and economic framework at risk, Bowers said.

Similarly Connie Fudge says

We live here. We have bright kids. We feel safe here. They say there is a health problem here,and we're saying there is no health problem. Show us the health problem here.

Fudge and others like her feel the EPA scientists are condescending and that their scientific model of how much soil lead will contribute to blood lead is flawed by the fact that it does not discriminate between lead carbonates and lead sulfides (McClure, 2001).

The presence of Superfund designation has scared away home buyers. The Paddock family moved into the state in search of a lower paced rural lifestyle. They purchased property in the area, but then sued under Title X lead disclosure when they found out that they had moved into a lead contaminated area (Murphy, 2000). The case clarified rules with respect to lead disclosure mandated by Title X.

We're trying to diversify, we're trying to revamp an economy. But you go out and try to attract a company, and if they look at the Silver Valley and it's a Superfund site, well then you're automatically dropped in the tank. With tourists, it's exactly the same thing. Nobody wants to come to a place they feel is unsafe.

Said Bill Dire a city councilman in Wallace (Verhovek, 2002).

The case highlights local science control vs federal science control, risk assessment based on who is most affected by designation of risk, the chemistry of bioavailability (lead sulfides vs lead carbonates vs lead oxides); industrial malfeasance with the use of the smelter without filters, and a debate about the intent of Title X requirements to divulge knowledge of lead risks during a real estate sale.

The model shown in Figure 10.16 does NOT distinguish the chemical form of lead. We saw much earlier that lead ores based on lead carbonate were much more toxic than those based on lead sulfide, a result of the variable solubility of the two compounds. From a regulatory/measurement perspective setting limits based on type of lead would compound the complexity of the issue because monitoring and assessment is already unwieldy given the vast dispersion of lead. From an environmental monitoring perspective routinely, rapidly, and cheaply identifying the chemical form of lead is extremely difficult if not impossible.

A Sad Ending for Dr. Chisholm

In this chapter we noted the prominent role that Dr. Julian Chisholm played in the fight against childhood lead poisoning. He developed many of the tools used to test for and document childhood lead poisoning. Perhaps more importantly, his towering contribution was in the development of chelation therapy for lead poisoning. One of Dr. Chisholm's last studies, just prior to his death (2001) was in conjunction with Dr. Mark Farel. The goal of this study was to determine if efforts spent on remediation (removal of lead) from homes from a variety of methods actually resulted in the reduction of blood lead poisoning. Because researchers allowed children to live in homes with incomplete removal of old lead paint, the courts compared the experiment to the infamous and blatantly racist Tuskegee study of untreated syphillus in which government doctors withheld treatment from ailing black men so they could study the disease's progression. Farfel said "Society was already doing a Tuskegee

experiment. Very little if anything was happening to remove lead while children were being poisoned." The researchers, associated with the Kennedy Krieger Institute, an affiliate of Johns Hopkins University, were sued by parents of children whose blood level while living in homes partially remediated by Kennedy Krieger The Kennedy Krieger brief indicated that 95% of homes in poor Baltimore neighborhoods where the study subjects lived have lead paint in them. The cleanup performed by the researchers reduced the risk by 80%. However, living with the remaining 20% could cause a rise in blood lead (Pelton, 2001; Roig-Franzia, 2002).

Other studies indicte that deleading by scraping and sanding of a home causes a transient increase in blood lead followed by long term blood lead decreases (Amitai et al., 1991). Such sanding and scraping should be very cautiously used as cases of lead poisoning of children have been shown to be the result of home renovations (Marino et al., 1990).

Environmental Racisim

The incidence of lead poisoning dropped dramatically in the United States between the 2nd and third National Health and Nutrition Examination Surveys (NHANES) (1976 to 1991) (Figures 10.25 and 10.26). The drop was remarkable, but was disparate between white and non-white children. Black children have a much higher probability of lead poisoning due to a confluence of residential segregation to older housing stock, and poor incomes, leading to lower nutrition. A decrease in calcium and iron uptake increases lead uptake (Lanphear et al., 1996; Weintraub, 1997).

The *correlation* of lead poisoning with race and poverty in the United States has been termed environmental racism. The term environmental racism was coined by Rev. Benjamin Chavis in a report from the United Church of Christ 1987 which overlay information about toxic sites (both operational and abandoned) and race. This report was, in it's turn, in response to what has been called the Afton riots. In 1982 North Carolina decided to dump 32,000 cubic yards of polychlorinated biphenyls (PCBs) contaminated soil removed from a predominately white area of the state in a landfill in Afton, Warren county, North Carolina, a predominately Black area. 500 demonstrators were arrested (Blais, 1996).

In response, the U.S. General Accounting Office (GAO) produced a study of the relationship between four existing hazardous waste landfills and racial composition in the surrounding communities which tended to support the allegation of a *correlation* with race (Wright, 1993). Figure 10.25 shows the distribution of "superfund" in the United States. A "superfund" site is that fitting the contamination criteria of the Comprehensive Environmental Response, Compensation and Liability Act (CERCLA) of 1980 and the Superfund Amendments Reauthorization Acta (SARA) of 1986 and consists of an "abandoned" site.

Sites listed by the Environmental Protection Agency as superfund are not typically searched for but are referred. The maximum potential sites in the U.S. are suggested to be 439,000. In addition to superfund (CERCLA) sites there are active sites that are regulated by the Resource Conservation and Recovery Act (RCRA). These are the sites that incinerate lead containing metals (among others) or may be involved in lead acid battery recycling, for example. Of the 142 superfund (CERCLA) sites in Chicago, several contain lead as the primary toxic compound. Sites include Benjamin Moore Paints (1897-1949), Devoe and Raynolds (Paints) (1897-1941), Diamond Red Paint Co. (1923-1954), Illinois Smelting Refining (late 1888-early 1900s) Magmus Co. Inc (1926-1974) manufacturer of lead bearings for railroad cars; Sinclair and Valentine makers of printing inks (1948-1991) (Coursey, 1994).

The GAO study was quickly followed by a study from the United Church of Christ corroborating the correlation of CERCLA and RCRA sites with communities of color. In this study Rev. Chavez coined the phrase *environmental racism*. He later defined it more precisely as (Fisher, 1995):

Environmental racism is defined as racial discrimination in environmental policy making and the unequal enforcement of environmental laws and regulations. It is the deliberate targeting of people of color communities for toxic waste facilities and the official sanctioning of life-threatening presence of poisons and pollutants in people of color communities.

Once environmental racism had been "discovered" it was followed with a number of similar *correlative* studies finding that blacks have a higher burden of exposure from toxic sites than do whites. Based on these studies President Clinton in 1994 issued an executive order No. 12,898, 3 C.F.R. 859 *reprinted in* U.S.C.A. 4321 1994., which required all federal agencies to consider the possibility of disproportionate siting of environmentally sensitive land uses in every decision in which the issue might be relevant.

The correlation had been noted by others. It some cases the correlation falls under the category of

Minorities still more likely to live near toxic sites

Despite increased public awareness and a recent executive order, commercial hazardous waste facilities are now more likely to be located in minority communities than they were in 1980, exposing affected populations to disproportionate health and environmental risks, a new study finds.

According to "Toxic Wastes & Race Revisited," in 1993, ethnic minorities were 47% more likely than whites to live near a Resource Conservation & Recovery Act (RCRA)-permitted toxic waste facility. In fact, between 1980 and 1993, the concentration of minorities living in communities with such facilities increased from 25% to nearly 31%. If the increased growth in minority population in the U.S. is factored in, the real increase in racial disparity in siting toxic waste facilities is 9%.

The study analyzed 530 commercial hazardous facilities in operation as of 1992. Only RCRA-permitted facilities were considered. Superfund sites, facilities closed before 1990, municipal solid waste facilities, and on-site commercial storage facilities were excluded. Population demographics came from the 1990 U.S. Census, updated with 1993 data.

The changes during the 13-year period can be attributed to several factors: migration, birth, or death of individuals, and relocation, startup, or closure of such facilities. Study analysis, however, does not determine which factor is most responsible for the increase. Benjamin A. Goldman, director of Boston-based Jobs & Environment Campaign and a study author, tells C&EN, "The degree to which only one of the six is primarily responsible would take more detailed analysis.

"The bottom line," says Goldman, "is not only has racial disparity not declined, but it has increased. This highlights the urgency of the problem and suggests the need for greater action than simply public awareness."

He believes "the U.S. needs to start turning words into action to reverse this trend, including [enactment of] public policies that result in a significant decrease in the generation of hazardous wastes." Such policies also need "to be targeted, especially on communities currently suffering most of the brunt of the problem."

Reducing toxic waste at the source is one of five points in a policy proposed for state legislatures by Washington, D.C.-based Center for Policy Alternatives, a cosponsor of the study. The other study sponsors are the National Association for the Advancement of Colored People and the United Church of Christ Commission for Racial Justice (UCC-CRJ).

The center's proposed policy also focuses on empowerment—involving affected communities from the beginning in the siting and monitoring of toxic waste facilities. Another policy suggestion is that communities with these facilities receive additional resources earmarked for such things as investments in infrastructure, education and training, and small-business development. And finally, the policy framework says state legislators should make certain that the health of minorities in affected communities is protected and that clustering of such facilities is avoided.

Richard Gold, a special assistant to Environmental Protection Agency Administrator Carol M. Browner, says the federal government "is working very hard to push the envelope of environmental justice. But frankly, the federal government has little authority to change the numbers, because land use is a state and local government decision."

"Toxic Wastes & Race Revisited" is an update of UCC-CRJ's 1987 study "Toxic Wastes & Race in the U.S.," which was the first to quantify the racial disparity in siting of toxic waste facilities. The 1987 study sparked national debate on environmental justice.

Lois Ember

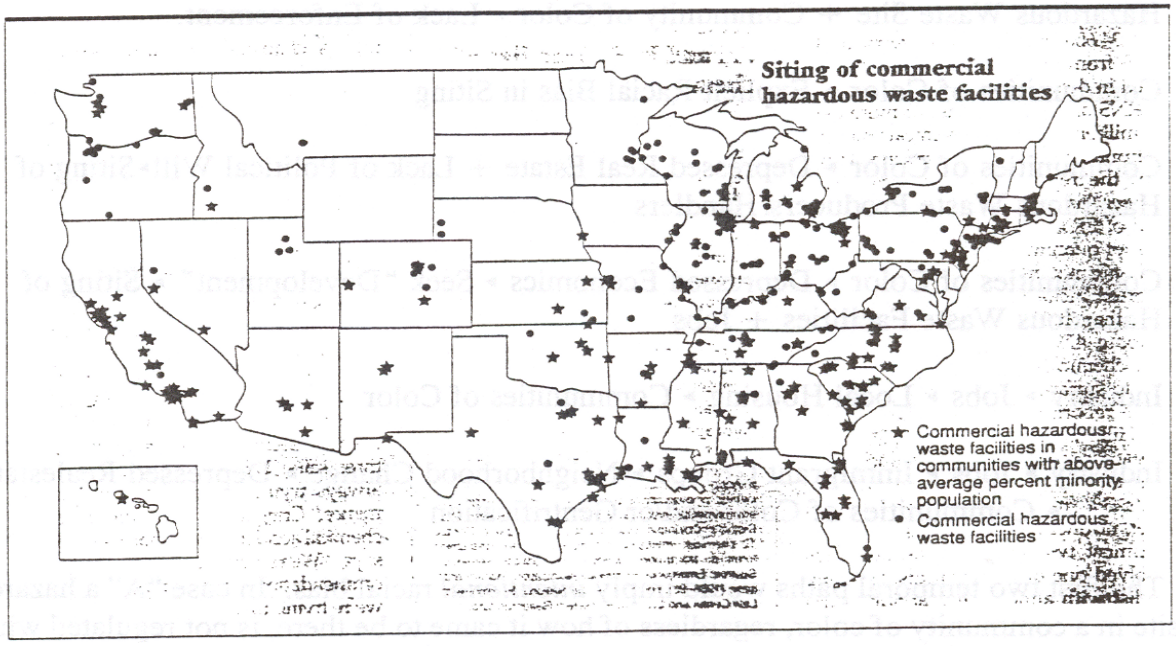


Figure 10.25 Environmental racism was presented on the pages on the chemistry newsmagazine, *Chemical and Engineering News.*

"blame the victim" as was noted above in Benjamin Franklin's letter:

some low-income and racial minority communities appear to have greater than average observed and potential exposure to certain pollutants because of historical patterns affecting where they live and work and what they eat [emphasis added here]*... they may have greater than average potential for exposure to some pollutants because they ...may be more likely to live near a waste site.*

Buried in this statement is a statement of fact which can be interpreted to mean that the poor make free choices to live in contaminated environments and to feed with foods that enhance the effect of contamination (Paustenbach, 1989).

In addition, it was quickly noted that *correlation* does imply causation. That is, unless the full temporal history of the community is known, already sited facilities in communities of color may occur by several trajectories (Coursey, 1994; Pulido, 1996; Pulido et al., 1996).

A. Hazardous Waste Site + Community of Color ▸ Lack of Enforcement.

B Communities of Color ▸ Explicit Racial Bias in Siting

C Communities of Color ▸ Depressed Real Estate + Lack of Political Power▸Siting of Hazardous Waste Producers/Handlers

D. Communities of Color ▸ Depressed Economics ▸ Seek "Development" ▸ Siting of Hazardous Waste Facilities + Jobs

E. Industry ▸ Jobs ▸ Local Housing ▸ Communities of Color

F. Industry ▸ Jobs ▸ Immigrant Groups ▸ Neighborhood Change ▸ Depressed Realestate ▸ Communities of Color and/or Gentrification

The first two temporal paths would imply *intentional* racial bias. In case "A" a hazardous waste site in a community of color, regardless of how it came to be there, is not regulated with the same efficacy as a site in a white community. It was found in a 1992 study published in the *National Law Journal*, that federal environmental enforcement is fuller in white communities than in communities of color (Lavelle and Coyle, 1992). A later study found that there was no racial correlation with the EPA decisions on the speed of cleanup of contaminated sites (Gupta et al., 1995).

In case "B" an intentional racial bias places a waste site within a pre-existing community of color. As of 1997 no case had been successfully proved in court of *intentional* racial bias (Blais, 1996).

Despite the inability to prove *intentional racial* bias it has been suggested that temporal path C can be considered to be a form of *institutional* racism. In this scenario siting of a hazardous waste facility, after all "scientific" considerations are weighed (wind direction, exposure, soil type), is based on the cost of the site. Since the real estate prices in communities of color are typically depressed, it is an economic rather than a racial decision to site the facility within the community. It also helps that members of these communities generally have a lower participation in political decision making.

The opposing argument is that shown in temporal path D. In this case members of the community weigh the cost/benefit ratio of living next to a hazardous waste facility and decide that the benefit of jobs outweighs the cost of exposure and aesthetics and depressed land prices. The difference between C and D lies in the *informed* consent on the part of the community. In fact, some have suggested that to interpret all events as falling under path C is at best a form of paternalism and at worst a form of racism which implies that members of the community are ill equipped to make rational decisions.

These two temporal pathways play out in other places besides racial politics in the United States. Equivalent arguments are made with respect to the siting of industrial or hazardous waste facilities in developing nations. The term *Global Environmental Racism* or *Environmental Justice* is most often applied to these situations (Marbury, 1995; Torres, 1992). The same financial factors play out both in the siting of hazardous waste sites on Native American lands in the United States as in Africa and Latin America. This case is particularly true of the siting of 650 solid waste disposal facilities on Native American lands. 108 were in existence prior to RCRA standards for such landfills, and only two of those were in compliance with RCRA requirements by 1993 (Chase, 1993). As long as the environmental laws and restrictions are less stringent than in the country of origin and the cost of living low, then it is economically (to the producer) to export the waste than to comply with internal restrictions on the waste.

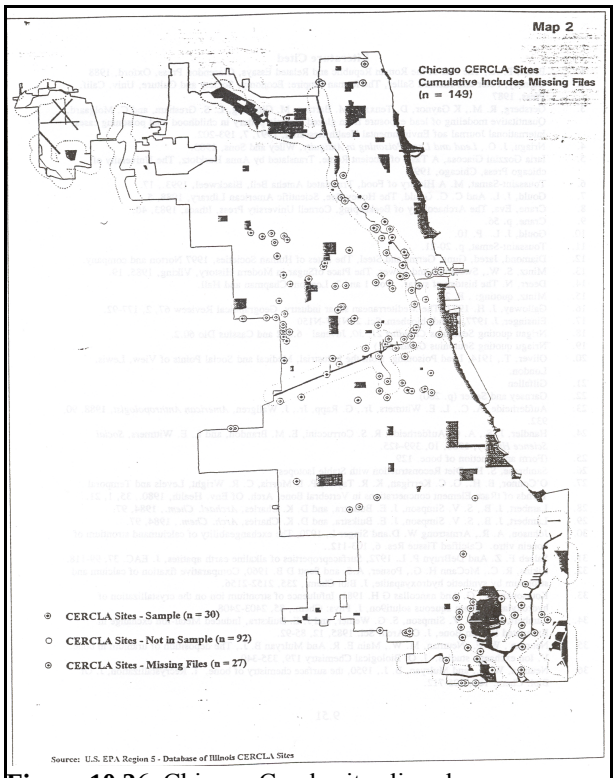


Figure 10.26 Chicago Cercla sites lies along waterways. Those on the south side are now predominately lower income neighborhoods.

A similar economic scenario plays in temporal path E. Here a pre-existing site **attracts** job seekers wanting to live close to the facility. A variant on this is the company town motif, where housing is provided for workers and is provided in a racially discriminatory manner, as was shown for mid century Los Angeles (Pulido et al., 1996). While this variant can be seen as intentionally racist the general path E, is not.

A very similar temporal path is described in case F. Here the process of job attraction starts much earlier in history, with European immigrants from the 1880s-1910 arriving and living next to the industrial toxic producer which was also the source of jobs. As they prospered the cost benefit ratio associated with living near the facility increased and they moved on, leaving a depressed real estate accessible to minority groups. Case F can be seen as institutionalized racism. If a particular minority community does not have free mobility in making housing choices (choices are constrained by intentional discrimination in the housing market) then the influx of people of color into an area with a toxic burden would also constitute a form of environmental racism. This point is most forcefully argued by the sociologist Bullock who defines environmental racism as:

Any policy, practice, or directive that, intentionally or unintentionally, differentially impacts or disadvantages individuals, groups, or communities based on race or color, [as well as the] exclusionary and restrictive practices that limit participation by people of color in decision making boards, commissions, and staffs (Bullard, 1993).

In these formulations environmental racism has an expanded context, one which intertwines poverty with race. The success of a Title VI (civil rights) challenge will be much more difficult, but not impossible, as it has been recognized in prior lawsuits that race and poverty are so strongly intertwined that a civil rights lawsuit is not required demonstrate the poverty is not the cause (and race is) (NAACP v. Wilminton)(1981):

In the United States, both now and at the time Title VI was passed, poverty and minority status were deemed so closely correlated as to be almost indistinguishable. Thus actions adversely impacting the poor will almost invariably adversely impact upon minority groups. The fact that the disparate impact is caused by socio-economic differences between the races rather than genetic ones is irrelevant.

In the case of the City of Chicago CERCLA (superfund) sites tend to derive from the industrial period of 1880-1950 and are located predominately along waterways where effluent was directly discharged (see Figure 10.26). (Coursey, 1994). The racial composition of the areas changed with loss of heavy industry. In some areas these sites became low income enclaves and still remain so, in others, the waterway (now clean) have made the sites high value and subject to "gentrification".

CORRELATIONS BETWEEN SOIL/DUST LEAD AND BLOOD LEAD

Once a child has ingested leaded dust, the lead is digested by stomach acids. The EPA has developed an uptake/biokinetic model which estimates the amount of lead in the blood (PbB) based on the concentration of lead in a source, $[Pb_i]$, the amount of the source ingested, C_i (g Pb/day) , the amount of the ingested material which is actually absorbed, A (g soil adsorbed/g soil ingested), and a conversion factor, T_i, to blood lead, PbB:

[10.1] $PbB = PbB = T \sum_{i=1}^{n} A_i C_i [Pb]_i$

Alternatively, equation 10.1 can be written:

$$PbB = \left\{ F_{age} \frac{mg_{ingested}}{day} \frac{mg_{adsorbed}}{mg_{ingested}} \right\} \{ f_{bioavailability} f_{soil\,cover} f_{nutrition} f_{social} f_{other} \} C_s$$

Values used in the model are a compendium of a variety of research efforts (Hoffnagle, 1988). Values are shown in Table G.6. Using this model one can calculate the amount of reduction in the blood lead by changes in the source of exposure. A reduction of 1 ug/m^3 in air from 1.5 to 0.5 resulted in reduction of 0.34 ug/dL in children's blood. In this model house dust with 2,200 ppm can lead to 20 ug/dL.

For ingestion soil only the equation becomes:

[10.2] $PbB = 0.012[Pb_{\text{soil concentration in ppm}}]$

For example, a soil with 1000 ppm Pb would result, in this model, in a blood lead level of 12 µg Pb/dL which is sufficiently large, over the long term, to cause neurologic problems. The soil action level of 500 ppm would result in 6 µg Pb/dL.

This model has been applied in four cases associated with soils near primary smelters, Herculaneum, MO, Kellogg, ID, East Helena, MT, and near a secondary smelter, the Niagara Neighborhood in Toronto, Ontario. Agreement between the model and measured blood lead levels were excellent (Table G.7). It should be noted that the indoor dust in these studies was apparently dependent upon the smelter and not paint given the parallel values. It should also be noted that this data also measured lead from other sources such as inhaled, dietary, and drinking water. Of these sources the soil lead was found to be a significant fraction of the lead uptake, particularly in locations near the smelter.

This data would suggest that a model near smelters in which blood lead is related to soil content. A debate centers around whether lower soil levels of lead have any impact. The Center for Disease Control (CDC) concludes soils can contribute to blood lead when the soil exceeds 500 ppm (Bornschein et al., 1988). A correlation study concluded that soil lead could be related to lead in dust and from there to hand and thence to blood even at soil levels less than 500 ppm. Linear relationship between soil and blood lead occur at blood lead levels below 25 µg/dL with a slope of 2µg/dL/1000µg/gsoil. The relationship is nonlinear at higher blood lead levels. The non-linear nature of uptake above 25 µg/dL is attributed to a "buffering" or adsorbing capacity of the plasma (Marcus and Cohen, 1988).

A more recent study makes a reasonable prediction of the "background" value of blood lead level (BLL) assuming background values of lead in the U.S. as 5.0 ug/ft^2 dust, soil lead of 72 ppm, interior paint concentrations of 1.6 mg/cm^2, and water lead of 1 ppb. The uptake model predicts 4 ug/dL geometric mean, very consistent with a metal analysis of multinational origin (Lanphear et al., 1998). An analysis of a number of collected studies shows that dust is the major source of lead n blood (Lanphear et al., 1998).

Chemical mass balance of lead in a home (measured by analysis by XRF of size fractioned particles) indicated that soil yard and street dusts accounted for 2/3 of the lead present in house dusts while leaded paint accounted for 1/3 of the lead present in the house dust (Adgate et al., 1998). This is consistent with the observation that 4 out of 5 samples of dust did not compare well with lead within the home as measured by lead isotope ratios (Jaeger et al., 1998). Other models have been proposed (Succop et al., 1998).

Bioavailability of leaded dust and soil

The above studies are based on total lead in the soil, although discrepancies in the modeling studies

have been attributed to large particle sizes (Bornschein et al., 1988). The larger particle sizes adhere less well to hands and consequently do not move indoors and also are not moved from hand to mouth as easily. Particle sizes of less than 50-100 μm adhere while particle sizes larger do not. The concentration in Pb increases in these particles as lead increases as particle size decreases (Duggan and Inskip, 1985; Duggan et al., 1985). Lead in larger particle size materials may be imbedded in more inert matrices and are less easy to digest.

Lead in soils tends to be in a non-crystalline form and consists of $PbSO_4$, Pb^o, Pb_3O_4, $PbO\text{-}PbSO_4$, and $2PbCO_3\text{-}Pb(OH)_2$, and some crystalline $PbCO_3$. (Harrison et al., 1981). Equilibrium calcuations for lead in soils would suggest that Pb^{4+} cannot exist (would be reduced), nor can Pb^o, as it would be oxidized, $PbSO_4$ will be too soluble and $PbCO_3$ will be too soluble than $Pb_5(PO_4)_4Cl$, a pyromorphite which should form if both phosphate and chlorides are present (Garcia-Miragaya, 1984; Lindsay, 1979; Nriagu, 1974). The bioavailability of the dust or soil depends upon the mineral type (Chaney et al., 1989; Davis et al., 1992; Ruby et al., 1992). Lead sulfide materials are generally less soluble than lead sulfate or carbonate, as we might recall from our discussion on the mechanisms involved in the formation of the originally ore bodies (Freeman et al., 1994; Freeman et al., 1992).Acid dissolution studies suggest that dust from lead mine tailings consisting of predominately lead phosphates, lead-manganese oxides, and sulfates were generally indigestible by stomach acids, while mine tailings with predominately lead oxides had more digestible amounts of lead (Ruby et al., 1993). In general, however, the lead in soil is not in equilibrium with discrete lead minerals, but as a cation adsorbed to either humic materials or to iron oxides (Harter, 1983; Hassett, 1974; Kinniburgh et al., 1976; McKenzie, 1978; Wolf et al., 1977; Zimdahl and Skogerboe, 1977) or via the cation exchange capacity of the soil imparted by the negative charge from indigenous clays.

Bioavailability will depend on the rate of dissolution which depends upon the soil form of the lead and the acid environment in which dissolution occurs. Interestingly, what is more acutely toxic for cattle differs from that for humans since cattle have an anaerobic digestive system, high in pH and with a high concentration of sulfides (Chaney et al., 1989). Gastric fluids in cattle is usually pH 3.5 to 4.5 while in humans it usually 1. Rats make a better control. Rats fed equal concentrations lead from house dust, road dust and lead acetate were more acutely affected by lead acetate. Soil Pb was 70% as toxic as lead acetate. Chaney argues that there is a non-linear response to soil lead because at low soil lead levels, lead mobilized by the acid stomach environment can be adsorbed by other soil minerals, consequently at higher soil lead levels one expects a saturation of sites, and less buffering of the free lead to be adsorbed.

Because of the variable nature of the availability of the ingested lead as a function of source some researchers propose that the one-level-only guidelines for soil lead are meaningless and should be adjusted site by site to account for the specific availability of lead in the particular soil (Wixson and Davies, 1994). Ultimately, however, the enactment of Title X required the federal government to set a single consistent guideline for soil lead regardless of source.

Costs of Remediation

Where does lead go once we decide to divorce ourselves from it? The main materials to be removed are painted walls and contaminated soils. There are a host of smaller sources of lead which are removed (road dust to sewers, discarded materials) and industrial water streams.

Dealing with Contaminated Soil

Remediation and/or Removal of Contaminated Soils

Contaminated soils make up a large segment of the Superfund sites in the United States (Forstner, 1995). Of a total of 952 Superfund Sites 41% made the list due to metal contamination, with another 26% having problems with both metals and organics. Of the metals listed at the contaminated sites lead was the most predominate. Metal containing sites were listed as landfills/chemical waste dumps, metal finishing/plating sites, mining and ore processing, DOD and DOE sites, and battery recycling sites. Treatment of these sites and of soils near homes contaminated with leaded paints follow four possibilities: removal, capping, cleaning, and stabilizing.

Removal.

Removal of soil is costly and presents a disposal problem. It may not be effective. One public health study remediated contaminated soils at a cost of $14,000/house by removing the upper 20 cm, laying down a polymer barrier, and replacing with uncontaminated soil. No benefit was found from the procedure (Weitzman et al., 1993). Disposal of the large amount of soil in the study was also pinpointed as precluding a larger scale experiment. "Capping" of soils has been suggested but has not yet been adequately studied. Capping could consist of sodding the surface.

Superfund sites of lead contamination have been treated variously by removal and containment (limiting access of humans) as occurred at the Brown's Battery breaking site Pennsylvania. The site soil was consolidated and removed on site to a capping location. Consolidation was recommended by thermal treatment. Contaminated ground water was to be pumped, pH adjusted, precipitated, ion exchanged with on site discharge. Sludge generated during the treatment was to be removed to a toxic waste site. Total cost of remediation was estimated to be $12,316,000.

Barriers

A second battery recycling site in Virginia was to be treated at a 1000 ug/g action level of lead. The lead contaminated soil was to be "moated" by injection of cement stabilizers followed by covering the site with 6 inches of top soil and revegetation to obtain a 120 ppm background soil level. The Granite City, Il. NL Industries and Taracorp site remediation involved excavating a total of 94,820 cubic yards of lead contaminated soil. The soil was to be consolidated and covered with a RCRA multimedia cap, and the expansion of the pile to be under set with a clay liner. The final site was to be left with an upgraded security fence around the expanded pile. Three down gradient deep wells and one up gradient deep well was to be installed to monitor transport of pollutants to groundwater. The NL Industries, Taracorp site in Mn. was capped and wells drilled for monitoring of the groundwater (EPA report). The cleanup goals for lead were to drop the total lead on the site to a range of 200-500 ppm (in comparison to background soils of 2-200). Specific Remedial Technologies suggested by EPA include containment utilizing caps, vertical barriers, and horizontal barriers. Caps can range from simple soil to RCRA Subtitle C, a composite cover. Vertical barriers minimize the movement of contaminated groundwater off-site or limit the flow of uncontaminated groundwater onsite. The barriers include slurry walls in excavated trenches, grout curtains injected into soil borings, cement-bentonite grout-filled boring, driven steel sheet-pile walls. The cement-bentonite barriers are not good when exposed to high concentrations of creosote, water soluble salts or fire retardant salts. The salt concentration should be low to prevent expansion of the bentonite. The steel walls can also corrode in dilute acid. Cost estimates of these horizontal barriers are not well know as few have been constructed.

Soil Washing

Most soil cleaning methods depend on mixing large quantities of soil ex situ (removed) with an extractant in a rotating drum. The extractant may be a chelate (EDTA) or an acid or a sequence of acid and extractants. An EDTA extractant has been reported to reduce lead in contaminated soils to 22% of its original concentration (Tuin and Tels, 1990). Several firms have been created to meet Superfund needs based on this technology (Biotrol; C.F. Systems; Harmon Env. Services, Inc.; IT Corporation; Resources Cons. Co.; Waste-Tech Services, Inc.). The amount of soil handled can be as much as 600 tons/day (Dennis et al.,

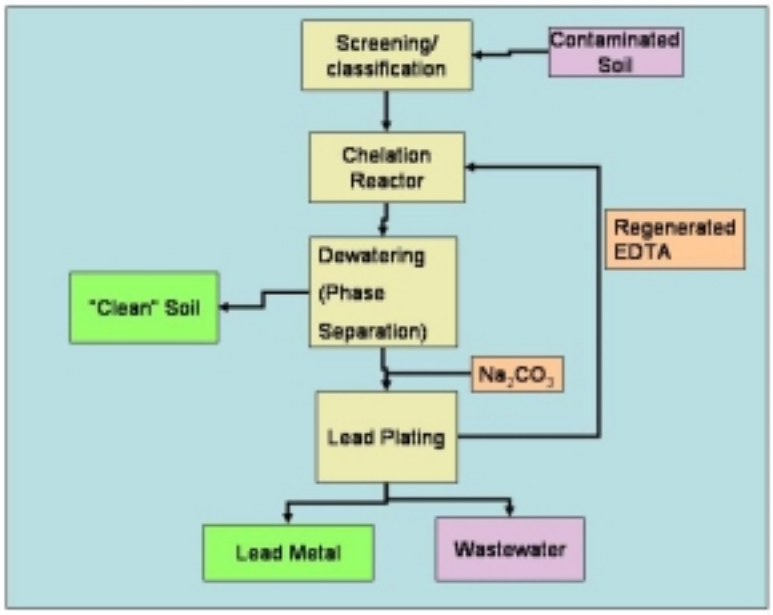


Figure 10.27 Soil washing involves removal of soil followed by water based extraction of lead.

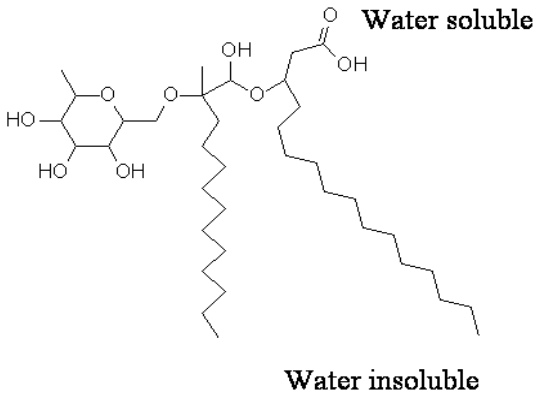


Figure 10.28 Soil bioremediation involves using bacterial produced materials to trap lead. The compound must be other water soluble and water insoluble to facilitiate transport.

1992).

One of the more mundane methods of soil washing depends upon some chemistry that we have already explored. The object is to recover lead from highly contaminated soils such as might occur near a lead smelting plant, a battery reclamation plant, or a lead-based paint manufacturing plant. Some of these soils may contain as much as 5% lead (0.05 g/g = 50,000 ppm). As will be noted by the reader (Figure 10.27), the number of steps involved in the cleaning process will render this method virtually inapplicable to more wide spread and less intensively lead contaminated sites. The soil is first screened, separated, and crushed. The goal is to reduce the particle size which increases the reactive surface area. An EDTA solution is added to the soil and reacted at a pH of 11-12 (Peters and Shem, 1995). The high pH enhances lead hydroxide precipitation but this process is mitigated by the higher activity of the chelating agent at this pH. The soil sludge is next de-watered and the water solution containing lead EDTA is sent to an electrolysis cell where lead is removed from the solution by reduction. Alternatively the lead can be precipitated from the solution (see below) or gathered up by filtration (also see below).

Soil Flushing

This involves leaving the soil in place and passing the washing solution through to be collected by wells with the reagent to be recovered. One method involves the use of a water soluble chelator developed by Los Alamos National Laboratory. Lead is bound to the water soluble species at pH values above 5.5 and is removed with 90% efficiency when the pH is dropped to 2.5. Lead is then released to be further treated to recover the expensive component (the chelator) (Tuin and Tels, 1990). This material, Metaset-Z does not appear to be affected to the same degree as EDTA by ionic strength, pH and the presence of other metal ions. It has a MW which can range up to 75,000 so once the solution is acidified to drop the collected lead, it can be filtered for reuse, while the lead containing solution can be subjected to electrolytic scavenging or to precipitation methods (Rampley and Ogden, 1998).

Bioremediation

Bioremediation of soils can occur either by harvesting crops that bioconcentration lead, or by addition of bacteria which produces substances that can facilitate lead removal. The ligand complex generated by the bacteria must be small enough to avoid entrapment in the finer pores of the soil network during soil washing. A biosurfactant, monorhamnolipid

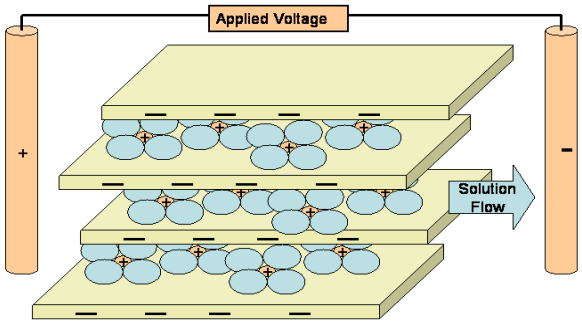


Figure 10.29 Soils can be cleaned by applying a voltage across a distance. Cations which are accumulated at clay surfaces hold water. As cations move toward the electrode they pull with them water.

(Figure 10.28) produced by Pseudomonas aeruginosa ATCC 9027 had a binding constant for lead of $10^{6.6}$ (Herman et al., 1995). The idea is that the colloid will adsorb lead and act as a mobility enhancer. One potential difficulty of this method is that the facilitator itself, monorhamnolipid, strongly adsorbs to the soil constituents.

Phytoremediation of lead-contaminated soils involves the use of a few rare plant species that accumulate lead, known as metal hyper accumulating plants. One such plant is the Thlaspi rotundifolium which can tolerate shoot lead concentrations of 8500 µg/g dry weight (Reeves and R., 1983). One of the difficulties in using lead hyper accumulating plants is that the lead is, for the most part, unavailable to the plant, requiring that the plant harvest be accompanied by cultivation with aids to solubilize the lead. An example of this is the Indian mustard plant, Brassica Juncea (Blaylock et al., 1997). Application of EDTA to the soil was found to allow accumulation of lead in the plant tissue. A similar study showed that EDTA added to the soil allowed corn and pea to accumulate lead (Huang et al., 1997; Wu et al., 1999). Estimated removal by B. juncea has been calculated. Assuming 6 t/ha biomass, 6 weeks of cultivation, and three crops per season B. juncea can remove 180 kg of Pb/ha.

Colloidal Precipitation

Several commercially available colloidal materials are being investigated for their efficacy in promoting chelation/extraction in soil washing procedures (Roy and Dzombak, 1997). Transport of lead through soil will be moderated by adsorption of the ion onto colloidal fractions which prevent localization to the stationary phase through competition and which move fairly rapidly due to size exclusion effects. One such facilitator of transport are the goethite (α-FeOOH), or iron oxides. These species have a fairly strong affinity for lead (Coughlin et al., 1995) which can be modulated by the presence of competing organic soluble ligands.

Electroremediation

One method of soil cleaning depends imaginatively upon electrochemistry (Hicks and Tondorf, 1994; Shapiro and Probstein, 1993). The process depends upon the fact that a large portion of soil particles are composed of clay sheets with dimensions of approximately 9 Å x 1 µ x 5 µ. The clay sheets are often negatively charged due to substitution of Fe^{2+} and Mg^{2+} for Al^{3+} in the Al-O lattice and Fe^{3+} for Si^{4+} in the Si-O lattice. One can imagine that the soil is an idealized brick lattice with negative charged flat surfaces defining small capillary channels. Clustering close the negatively charged channel surface will be a dense layer of cations surrounded by their water of hydration (Figure 10.29).

If a voltage is applied across the soil, the cation cloud with its associated water, migrates toward the negative electrode while anions in the system move toward the positive electrode. Because of the dense nature of the cation cloud, fluid flow develops at the clay surface and the flow extends into the center of the channel due to viscous forces. There is a net movement of solution toward the negative electrode. In order for the method to work lead must be present in a soluble form (Luckner and Schestakow, 1991). This is accomplished by introduction of a soluble chelate (EDTA or citrate) into the soil during electrokinesis.

One difficulty with this method is a lack of understanding of the fundamental mechanisms of electrophoretic transport in complex soil matrices which has hampered modeling of transport. It has been found that electroosmotic flow initiation is affected not only by the pH caused by reactions near the electrode, but also because the NaOH and HCL solutions formed have different conductivities than the bulk soil solution. When reduction occurs and NaOH is formed near the cathode the soil reactions remove charged species from solution:

[10.3] $\text{soil-H} + Na^+ + OH^- = \text{soil-}Na^+ + \text{water}$

This changes somewhat the effect of the acid/base front (Dzenitis, 1997). One solution to minimize the effect of the pH front and precipitation of lead hydroxide is to

create a membrane barrier between the electrode and the soil in which water is present. Precipitation reactions occur in this water. A cation selective membrane prevents movement of the high pH front with the hydroxyl (Li et al., 1998).

In-situ Stabilization

Other methods seek to stabilize the lead in situ and make it less bioavailable. Bioavailability will depend upon how much of the lead in the soil is soluble under stomach transit conditions. Table D.3 shows lead solubility as a function of native mineral type (Ruby et al., 1994; Ruby et al., 1992; Ruby et al., 1993).

If lead can be converted to a pyromorphite it is likely to pass through the stomach (2 hour residence time (2)), pH 1.5-2 fasting; 4-6 fed (Miller and Schricker, 1981)) undissolved. Modeling suggests that the soluble ppm of lead from the chloropyromorphite should be below allowable intake values (Table D.3).

Conversion of lead in soil can be accomplished by addition of a phosphate source, just as polyphosphate was added to municipal water supplies to decrease lead corrosion (Chapter 3). Hydroxyapatite (calcium phosphates as in bone material) can be added as a source of dissolvable phosphate (Ruby et al., 1994):

$$Ca_{10}(PO_4)_6(OH)_2 + 14H^+ \xrightarrow{slow} 10Ca^{2+} + 6H_2PO_4^{2-} + 2H_2O$$

$$10Pb^{2+} + 6H_2PO_4^{2-} + 2H_2O \xrightarrow{fast} Pb_{10}(PO_4)_6(OH)_2 + 14H^+$$

The rate of formation of the insoluble lead species is fast. Kinetic studies of lead immobilization by hydroxyapatite in aqueous solution indicate that a solution containing about 100 mg Pb/L is reduced to < 0.5 µg Pb/L in under 10 minutes by addition of finely divided hydroxyapatite. The rate of conversion depends on the dissolution of the calcium phosphate via a H^+ promoted nucleation (hole formation) mechanism. This will be facilitated under acidic conditions (Arends et al., 1987). Preliminary studies suggest that the other metal contaminates do not interfere with the immobilization process (Ma et al., 1995; Ma et al., 1994).

An alternative mechanism for lead immobilization on hydroxyapatite is based on ion exchange of Ca from the intact mineral for lead. You might wonder if it matters which mechanism is in effect. The answer is yes. The mechanism shown above depends strongly on the pH of the soil and the solubility of the phosphate amendment where the ion exchange mechanism would depend only on the diffusion of the lead through the soil to the phosphate mineral (Ma et al., 1993).

An estimate of the K_{sp} for tribasic calcium phosphate is given:

[10.6] $Ca_3(PO_4)_2 \leftrightarrows Ca^{2+} + 2PO_4^{3-}$ $\quad K = 10^{-28.7}$

with the K values for PO_4^{3-}

[10.7] $H_2PO_4^- \leftrightarrows HPO_4^{2-} + H^+$ $\quad K_{a2} = 10^{-7.21}$

[10.8] $HPO_4^{2-} \leftrightarrows PO_4^{3-} + H^+$ $\quad K_{a3} = 10^{-12.32}$

we can get an approximate value of K for the overall equation of the reaction:

[10.9] $Ca_3(PO_4)_2 \leftrightarrows Ca^{2+} + 2PO_4^{3-}$ $\quad K_{sp}$
[10.10] $2(PO_4^{3-} + H^+ \leftrightarrows HPO_4^{2-})$ $\quad (1/K_{a3})^2$
[10.11] $2(HPO_4^{2-} + H^+ \leftrightarrows H_2PO_4^-)$ $\quad (1/K_{a2})^2$
[10.12] $Ca_3(PO_4)_2 + 4H^+ \leftrightarrows 3Ca^{2+} + H_2PO_4^-$ $\quad K_{sp}/(K_{a3}K_{a2})^2 = K_{rx}$

Using the value above the overall reaction to solubilize the calcium phosphate is favorable. The K_{rx}, is $10^{10.36}$. The apparent solubility depends upon proton concentration and would go up significantly with a low pH. The corresponding reaction of lead with phosphate can be calculated from the Ksp value for $Pb_3(PO_4)_2$ ($10^{-42.1}$) to be unfavorable (does not want to dissolve), K_{rx}, for dissolution being $10^{-30.40}$. The overall reaction of calcium phosphate to lead phosphate is highly favorable: $10^{40.70}$. The numbers will change somewhat when apatite ($M_{10}(PO_4)_6(OH)_2$) is used.

The source of phosphate or iron (for hydrous iron oxide formation) affects the final formation of inactivated lead compounds. The soil type itself (acidity) is important and points to the need to design each treatment for each soil in place (Berti and Cunningham, 1997).

LEADED DUST

Soil can contribute to house dust as can internally applied leaded paints. The dust so formed is the primary route by which children are exposed to lead.

The primary source of transit is through direct ingestion of dust to hands to mouth (Table G.8) (Amitai et al., 1991; Bornschein et al., 1988; Charney

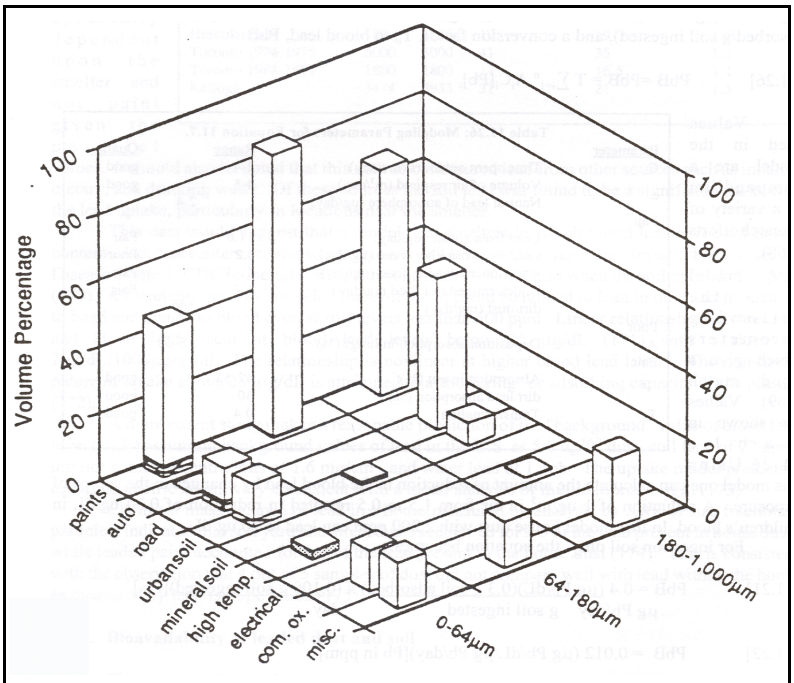


Figure 10.30 Leaded dust comes from a variety of source and with a variety of particles sizes. Hunt, Johnson, Watt, and Thornton, Env. Sci. Tech, 1992, 26, 8, 1513.

et al., 1983; Chisholm Jr. et al., 1985; Marino et al., 1990). In large part this is due to the child's handling of leaded materials and lack of washing. Children's hands can carry a large amount of lead as seen in Table G.11 (Fergusson, 1990).

Children come into contact with leaded dust in both the playground and in the home. Park dust comes primarily from traffic. Traffic dust is related to the idling of cars and the lead content of gasoline. Dust in the home comes from traffic dust and from paint dust generated by the fine grinding of paints along window sills. In urban areas a double whammy is felt as dust from traffic mixes with dust from paints (Fergusson, 1990).

The largest source of dust is from leaded paints. Large statistical data bases are allowing fingerprinting of the source of dust materials. For example, using combined imaging of size and topology with X-ray fluorescence of 19 elements a signature library was created. House dust samples compared to the source library suggest that the three largest sources of leaded dust derive from paints, road and urban soil (Figure 10.30) (Hunt et al., 1993) where the lead in urban soil is itself originally from external paint. Additional evidence for leaded paint as the source of lead in dust is the matching of isotope ratios (see Chapter 1 for isotope signatures) (Xu et al., 1992). Even dust in homes located near lead smelters the lead has been linked to poorly maintained paint on residences (Kimbrough et al., 1996). In one study isotopic ratios from the intact paint did not match the isotopic lead ratios in the dust sample within a home (Jaeger et al., 1998).

Table G.13 gives the incidence of U.S. homes with lead based paint as a percent of all 77,177,000 housing units built before 1980 (Clickner et al., 1995). The measurements are broken up as mg/cm^2. Current models seem to suggest that house dust derives from soil lead which in turn derives from external leaded paint (Brown et al., 1995; Burgoon et al., 1995; Yaffe et al., 1983).

Removal from the Home

Removal of housing materials is costly and one is left with the problem of disposal of the removed materials. Despite the costs, (estimated, 1993, $10,000 to $25,000 per housing unit (King, 1993)) such removal is the highest recommended form of remediation by the Chicago Department of Public Health.

Covering or capping the material, thus far, presents the most viable form of remediation. Encapsulation of lead painted surface with new wall materials is the second most highly recommended (Chicago Health Department, 1997) remediation method at an estimated cost of $2,000 to $8,000 per unit (King, 1993). This method is infinitely preferred to sanding (cleaning) which causes high dust levels during the remediation process. (Choe et al., 2000).

Cleaning of walls (extracting lead?) has not been raised as an option. Cleaning on a daily basis (wet wipe removal of dust) is strongly recommended to households affected by lead. Loyola University Chicago students (1996) in collaboration with an after school elementary science club demonstrated that the amount of lead in dust collected on baby wipes from homes could be reduced by 99.99%. Cleaning can be moderately effect if wet wiping is used (Ettinger et al., 2002).

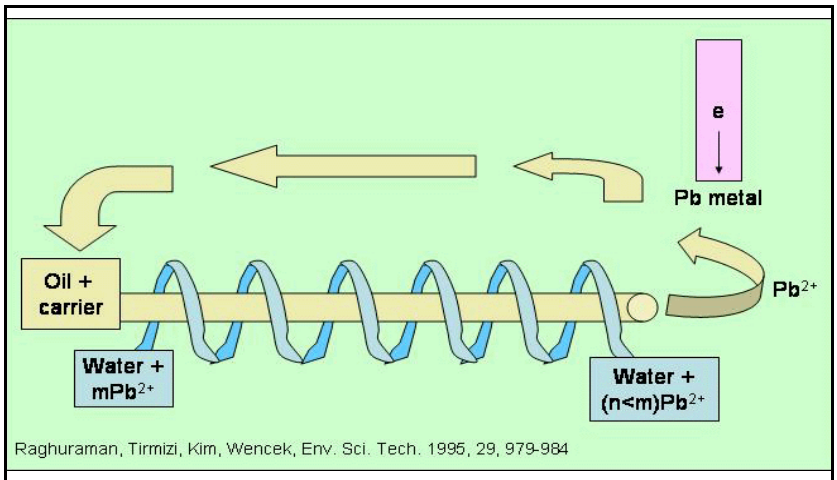


Figure 10.31Removal of lead from wtaer makes use of a phase separation and electrochemistry.

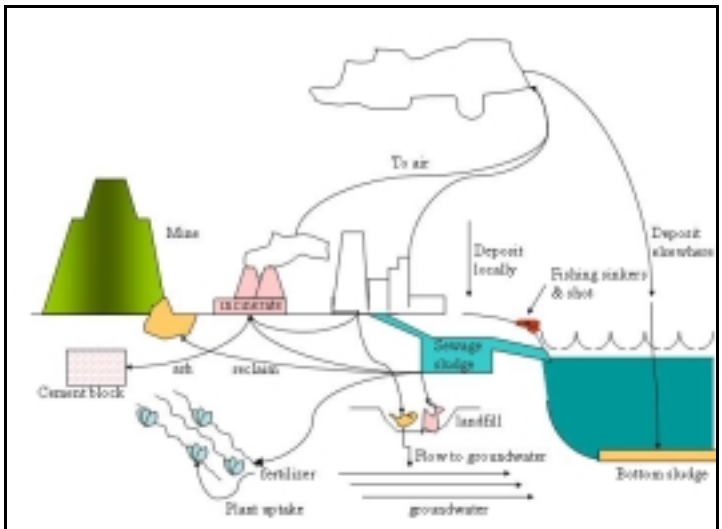


Figure 10.32 Lead global budget means that lead moves into urban waste streams and must be accounted for.

Removal to Water

Water sources can be contaminated by the washing of house dust into the sewer system or by the collection of airborne dusts into the sewer system.

Some of the water streams coming from industrial sources can be purified before hitting the sludge. The lead can be removed by method similar to those used to purify drinking water (phosphate feeds). The contaminated water can be purified from an industrial plant before entering a municipal waste stream by early purification. Such purification can consist of immobilization with phosphate rocks (Ma et al., 1995), or precipitated as lead hydroxide by raising the pH (Baltpurvins et al., 1996). The latter precipitation is affected by the presence of other salts. For example, the presence of chloride tends to increase solubility of lead due to the formation of soluble lead chlorides, the presence of sulfate tends to reduce lead across the entire pH regime as does the presence of carbonate. The precipitates identified included blixite ($Pb_4(OH)_6Cl_2$) and laurionite (Pb(OH)Cl) as boundary phase material dependent upon the total amount of chloride. Anglesite and cerrusite were found in both the low and high sulfate and carbonate systems.

The water can be passed over sorbent beds consisting of clays altered with organics to enhance lead adsorption such as the presence of carboxyldecyltriethylammonium cation which increases the carboxylic acid chelating functionality of the clay (Sheng et al., 1999). Other strategies include grafting sulfur containing moeities to clays such as (3-mercaptopropyl)trimethoxysilane clay-$OSi(OH)_2(CH_2)_3SH$ which increases the affinity of the clay for lead via the sulfur group. This also was shown to be effective for Pb and Hg sorption [Mercier, 1998 #878; Mercier, 1995 #877].

An alternative method is to agitate the solution with an emulsion (oil in water stabilized by surfactants) containing an extracting agent. The method works as shown in Figure 10.32. Lead is carried from the liquid waste stream to the emulsion phase interface where it reacts with a complexing reagent which moves the lead into the organic pahse. After agitation the dispersion is allowed to settle and the less dense aqueous phase drawn off. The emulsion in the upper phase is demulsified by changing applying a voltage field. The metals are recovered by electroplating or crystallization (Raghuraman et al., 1995).

Sewage

One factor not studied above was the washout of the deposited lead into the municipal sewer streams. Chicago grades its municipal waste into three

categories based on a range of pollutants including metals as well as DDT and PB discharges. The lead content varies as shown in Table H.1 (Lue-Hing et al., 1991). The sludge is sold as potting soil, used on agricultural soils as fertilizer, or used to reclaim coal strip mined sites. The metropolitan Water Reclamation District owns 15,6000 acres of line which it has revegetated 5,600 acres by application of 600,000 tons of dry waste.

Solid Waste Stream

Lead can enter various domestic waste streams as shown in Table H.3 and Figure 10.32. These waste streams are large as shown in Table H.2 (Pershing et al., 1993).

In what form does lead enter these various waste streams? Industrial waste streams (1,850,000 tons/yr) consist 47% organic fibers and non-cellulosic materials, 19% industrial organic chemicals, 17.5% other, 7.5% medicinals and botanicals, 4.5% plastics and resins, and 4.5% general chemical manufacturing. The list of industries which produce industrial wastes for incinerators is a somewhat surprising one. Apart from general chemical manufacturing, pesticide products, plastics and resins, electrical, gas, sanitation, pulp mills, explosives, and cement industries produce significant industrial wastes. Lead in these waste streams comes primarily (81%) from metal processing wastes, with 10% from plastic and resin wastes (Behmanesch et al., 1992).

The second major waste stream is that generated by the general public is municipal solid waste. What are the main constituents of municipal wastes and where does lead enter into this waste stream? Table H.3 (Bache et al., 1991a; Law and Gordon, 1979; Sullivan and Makar, 1976) shows the types of materials that we, the public, dispose of. The largest component is paper and yard wastes (thus we find mandatory recycling yard waste and paper goods being implemented nationwide).

Lead enters this domestic waste stream primarily as plumbing, paints (housing renovations, materials, discarded paints), and batteries (Williams, 1994). Other sources of metal in domestic waste comes from inks, clay fillers, whiteners, and photosensitizers (films) (Sullivan and Makar, 1976). Lead from ink printing is estimated to contribute 128 tons from packaging material, 1,900 tons from pigments, and 3,655 tons from plastics to be 5,683 tons from packaging total. The lead from the inks alone contributes 2.66% of the total lead in waste (Rustenholz and Smith, 1989). Lead is thought to consist of 110-1500 g/ton of waste material in the U.S. and from 750-2500 g/ton of waste material in Europe. Some of these materials can be recovered directly from the waste stream as shown by the data by Sullivan and Makar in Table H.3. It is important to note, as we will see below, that the lead that is burned in the municipal solid waste is often coupled with a high plastics content (approximately 5%, see Table H.3).

There are three main routes of disposal for these waste streams. The most common one is landfilling. The second route of disposal is that of incineration, or burning. The third route of disposal which can be coupled to incineration and/or wastefilling is solidification.

Landfilling

Any material presently landfilled must pass a leachability test. The idea is to prevent disposal of material into landfills where it may eventually end up in groundwater. Landfills are graded by the type of material to be disposed. Hazardous materials (containing leachable lead) have to be certified and disposed of separately than common municipal solid waste.

The leachability and transport of leached material from a landfill depends upon the pH (control of ionization of negatively charged surfaces capable of retaining the metal cation), the presence of precipitable anions (sulfides and hydroxides as compared to sulfates and chlorides), the presence of organics capable of mobilizing the metal, the oxidation state of sulfur (S^{2-} vs. SO_4^{2}) and solid phase material present. Lead can be carried adsorb onto solid surfaces via an exchange reaction:

[10.13] $\text{Surface-OH} + M^{z+} \leftrightarrows \text{Surface-O-}M^{(z-1)+} + H^{+}$

If the colloidal material is small enough the colloid can function as a carrier of lead (Liang and McCarthy, 1995).

The oxidizing vs reducing conditions of the landfill controls the chemical state of the sulfur which in turn will control the precipitation of lead. The expectation is that under high methane conditions sulfates would be low and lead would precipitate as galena, PbS. Table H.4 indicates that these expectations are met in a simulated landfill column filled with shredded municipal solid waste, doped with metals. Under an acid leach condition sulfide was not detectable and lead was measurable, but under reducing conditions the sulfate leachate concentration dropped,

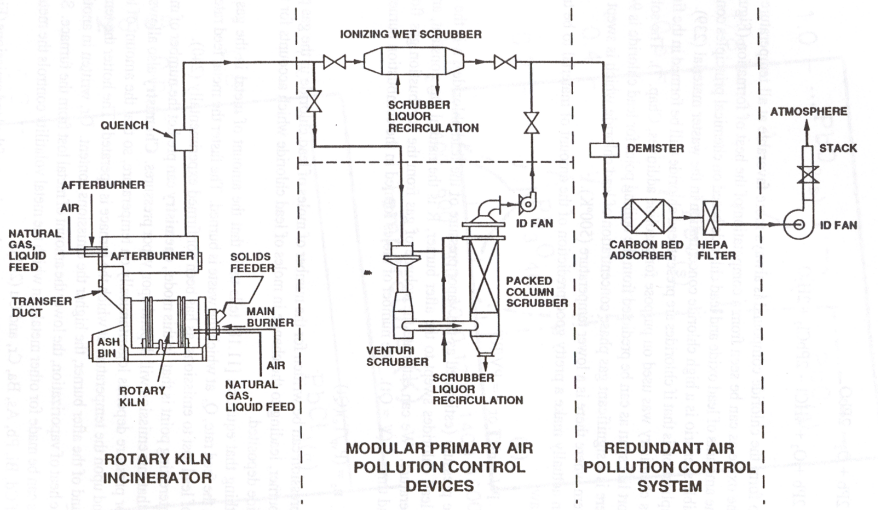


Figure 10.3 Basic components of an incineration system. Pershing et al. Combustion Sci. Tech. 1993, 93, 245-276.

sulfide became detectable and lead leachate dropped below the detection limit.

Ashes to Ashes: Metals and Waste Incineration

In this section we want to examine the fate of lead upon incineration. Incineration was used in the late 1980 is for two main reasons: cost and volume reduction. The cost is reduced by reducing transport problems, by the fact that landfill costs are increasing with the more sophisticated monitoring systems required, and by the fact that some of the energy can be recovered during incineration. For example, landfill costs in England are 5-30 English pounds/ton while cost of incineration is 15-30 tons (Petts, 1994). Table H.5 shows the relative amount of energy to be obtained from various waste streams as compared to coal and what percent of selected countries energy needs are met by waste incineration. Note that 10% of energy needs could be met by incineration. In 1988 14.5% of municipal solid waste (MSW) was incinerated in the US and it was predicted that this number would rise to 22% in 1995 (Wu et al., 1994). This estimate has proved wrong as a better understanding of the amount of lead emitted from incinerators has prompted re-evaluation of incineration as a method of waste disposal.

In Table H.5 we see that the three main types of incinerated wastes can produce energy that is comparable to that of coal. Note the low energy producing value of MSW which is associated with a high volume of foods and their associated water content. This water reduces the energy value by increasing weight and the need to use some of the energy produced in the burning to vaporize water. On the other hand, municipal sewage sludge very nearly approaches the energy content of coal. Sewage sludge wastes have been proposed as fertilizer materials for use as a source of phosphates. A comparison of the elements from a triple-superphosphate fertilizer to a sewage sludge ash is given in Table H.6 (Bierman and Rosen, 2994). In this study it was concluded that ash increased phosphorus as compared to the soil, but less so than the fertilizer. The ash resulted in higher amounds of Cu, Zn, and Cd in the tissue of the corn. The emissions associated with the burning of the sewage sludge also results in measureable amounts of metals which was not surprising given the metal content of the sewage sludges (an average of 40 cities found the median concentration of lead to be 265.7 mg/kg dry sewage sludge) (Hentz Jr. et al., 1992).

Metal Emissions

In general, the percent lead emitted is small (<2-5% (Morselli et al., 1993).) How small is small and what impact (what chemical form) does it have on emission, are the questions asked by near neighbors of the incinerators. Despite the small percent of emitted lead some researchers suggest that the primary hazard associated with incinerators is emission of toxic heavy metals (Waterland et al., 1991; Wu et al., 1994), (Candreva et al., 1986; Kimbrough et al., 1996; Sullivan and Makar, 1976), (Bache et al., 1991b).

The source of lead in the municipal solid waste derives from batteries, solder, lead tubing, electric wire, type metal, pipes, crystal glass, cathode-ray tubes, fluorescence lamps, PVC plastics, anti-corrosion paints and ceramic ware, crayons, and coloring agents in plastics (Nakamura et al., 1996). Lead is distributed throughout the combustion process as shown in Table H.7.

In the burning process gas and particulate matter are produced. The larger size diameter particles settle (bottom ash), while smaller size particles (fly

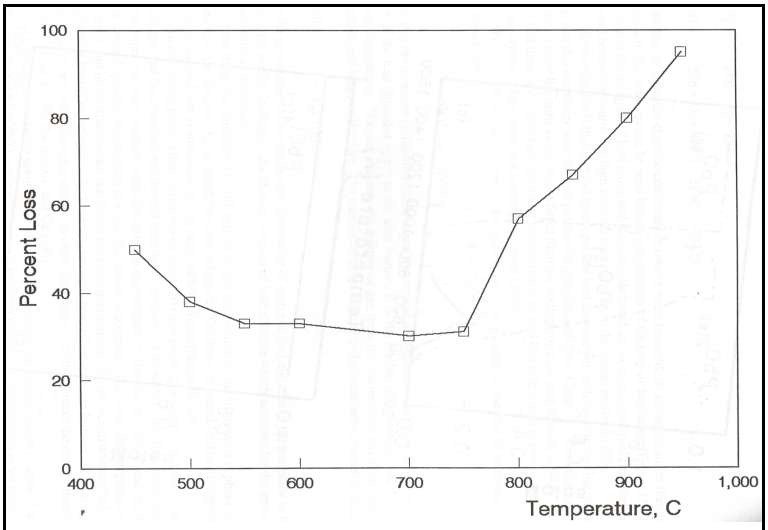


Figure 10.34 Lead emissions from incinerated waste increase between 700 and 800 C corresponding to the volatilization of PbO and $PbCl_2$ **.** Barton et. Al., 1991, 69, 29-36.

ash) can be captured in a scrubber or emitted (see Table H.8) (Brunner and Monch, 1986; Davison et al., 1974; Law and Gordon, 1979; Reimann, 1989). There would be no particular cause for concern if all the heavy metals were retained in the bottom ash or the scrubbed fraction of the fly ash as this material can be captured and disposed by solidification (see dust to dust). The problem arises, then, in the production of small size metal containing particles. Chemistry determines this process. Chemistry controls the total distribution of material into various sizes and the distribution of lead into those size particles. The chemistry is controlled by the type of material fed into the incinerator and the temperatures reached within the incinerator.

Figure 10.33 shows a typical incinerator set up. The garbage is fed in via a screw type feed to a primary combustion unit which heats and ignites the sample (Pershing et al., 1993). This part is generally operated at 1000-1200K. Gases and particulate matter from this burn unit are swept to an after burner operated at 600K. The total mass flow through the unit involves conversion of the waste to fallen ash in the initial burner, ash collected in the second burner, ash collected in the scrubber tower, and ash and gases emitted from the incinerator stack. The particle size of these ashes generally diminishes in transport through the incinerator so that the smallest particles are those that are finally emitted from the incinerator. Table H.8 shows some of the distribution of lead in various ash materials collected from incinerators.

The type of lead compound in the gas stream depends on the chemistry of the various compounds. Nitrates decompose to nitrogen containing gases at low temperature to leave behind stable oxides which do not decompose until very high temperatures are reached (Bass and Holcombe, 1988).

[10.14] $Pb(NO_3)_2 \leftrightarrows PbO + 2NO_{2(g)}\ 1/2O_2$

[10.15] $PbO\text{-}CO_{(s)} \leftrightarrows Pb_{(g)} + CO_2, CO_{(g)}$

The chlorides, on the other hand, dry then sublime after drying. The temperature of sublimation is lower than the temperature necessary for the decomposition of the oxides.

The temperature at which we produce PbO gas and $PbCl_2$ gas can be calculated from the following reactions

[10.16] $2Pb + O_2 \rightarrow 2PbO$

[10.17] $2Pb + O_2 + 4HCl \rightarrow 2PbCl_2 + 2H_2O$

The reaction to form the chloride compound is always more favorable at all temperatures than the formation of the oxide as can be seen from a computation of the heat of formation (Example 10.1). Modeling of the amounts of lead oxide and lead chloride based on chemical principles confirms that the worst possible scenario is a high chloride concentration in the waster material (Wu and Biswas, 1993), (Wu et al., 1994)

The implication is that if chlorides are present lead chloride will be formed in the first burner. Recall that this chemistry was used on purpose for gasoline additives, Chap. 7). The solid formed will have a short life span as can be predicted from the boiling point for lead chloride is 945°C. We expect that there is a significant gas phase concentration of lead chloride which is swept to the gas treatment system where there is a lower temperature (500°K).

We can actually make a pretty good prediction of the amount of material to be transported from the gas law:

[10.18] $PV = nRT$ or $n = PV/RT$

where P is the pressure (estimated as the vapor pressure of the lead chloride), n is the number of moles of the lead chlorides swept to the after burner, R is the natural gas constant, and T is the furnace temperature. We can estimate the volume of gas from the combustion gas

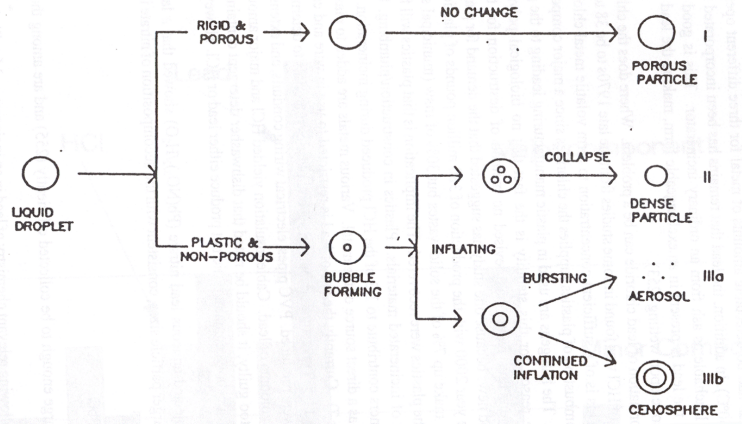


Figure 10.35 Particles are formed by complex processes during incineration. Barton et al., Combustion Sci. Tech., 1990, 74, 327-342.

flow rate, Q, (vol/time), and time, t, (V = Qt). The number of moles formed in the combustion burner is thus:

[10.19] $n_c = (P_{vp}/T_c)(Qt)$

A similar expression can be written for the number of moles of lead chloride in the gas phase in the cooler after burner, leading to the change in moles of lead chloride which accounts for the amount of lead chloride deposited.

One thing that equation [10.19] tells us is that the amount of metal in the gas phase will depend upon the feed rate, Q, at which the waste is burned. The faster the metal feed rate the greater the amount of lead lost to emissions, as has been confirmed experimentally (Ho et al., 1994).

An interesting point is that, in this model, chemistry can predict the number of moles of lead emitted, and that this emission will depend upon vapor pressures. Chemistry also allows us to assert that the vapor pressure depends logarithmically on temperature, so that the amount of lead emitted should depend upon the temperature at which the furnace is operated. The hotter the temperature of the furnace and of the after burner, the higher the emissions content. Or, written in another fashion, the higher the heat of vaporization, the lower the amount of metal lost from the furnace. Similar types of arguments can be made for other metals, in which the metal volatility controls the metal collection efficiency of Cd, Bi, Pb, As, Ba, Cr, and Cu (Waterland et al., 1991).

It is particularly easy to see this temperature dependence of the lead emissions (Figure 10.34). The jump in emissions at 800°C can easily be predicted from volatility considerations as we have just discussed (Barton et al., 1991). Clearly, one way of reducing emissions would be to lower the furnace temperature. The Kodak incinerator in Rochester was able to control emissions rather well using temperature control (Cowley et al., 1994). As is usual for every good idea there is usually a reason why the idea is not uniformly implemented. Lowering the furnace temperature will result in incomplete combustion of other materials such as volatile (and **toxic**) organics within the kiln (Pershing et al., 1993).

One way to lower the temperature of the burning process while maintaining nearly complete combustion of all materials is to change the kiln design. A kiln design designated as a fluidized bed allows for uniform heating of the sample by passing hot air (<750°C) through the bed (Ho et al., 1992). In this hot air, when the partial pressure of the lead chloride exceeds the stable value, solid matter will begin to form, in a process called nucleation (Figure 10.37). Homogeneous nucleation (lead chloride depositing onto itself) can proceed during rapid cooling which leads to very fine particle sizes, which could escape the scrubber. The rate of nucleation can be predicted (Barton et al., 1990).

$$I = \frac{2nV_m^{2/3}}{kT}\left[\frac{P\sigma}{2\pi}\right]^{1/2} \exp\left[\frac{-16\pi\sigma^3 V_m^2}{3(kT)^3(\ln S)^2}\right]$$

or

[10.21] $I \alpha nM_m V_m^{2/3}$

where I is the nucleation rate ($s^{-1}m^{-3}$), P is the partial pressure of the species of interest (atm, M_m is the molecular mass (kg), k is Boltzman's constant (j/K), T is temperature (K), V_m is the molecular volume (m^3), σ is the surface tension of the species as a condensed liquid at T (N/m), n is the concentration of species of interest, and S is the saturation ratio. The nucleation rate is proportional to the amount of material in the vapor phase and its volume.

Particulate matter formed by homogeneous nucleation is fine enough to escape the gas treatment flue (Fernandez et al., 1992) and so is to be avoided.

Nucleation could alternatively proceed by deposition onto some other material (heterogeneous

nucleation). If heterogeneous nucleation occurs, larger size particles are the product and these can be captured. Heterogeneous nucleation depends primarily on the size of the material onto which it condenses. The larger the diameter of the particle the more deposition will occur on it.

In order to promote heterogeneous nucleation stable solid matter (sand, limestone, and/or aluminum oxide) is added to the incinerated material to capture the volatilized lead. When the vapor pressure for $PbCl_2$ is too high $PbCl_2$ is redeposited onto the external surfaces of other particulate matter. The concentration on these particles is high since deposition occurs on the surface and the smaller the particle size the larger the surface area. If clay is used as an adsorbent the lead formed may become sintered, that is, fused into a glassy state. Lead can be held within the clay by electrostatic interactions, as well as by irreversible adsorption (Farrah and Pickering, 1977; McBride, 1976; McBride et al., 1975). Immobilization on the added support material can occur if lead is present as a salt of an unstable oxy-acid (for example, $PbNO_3$) then the lead can be converted to a lead oxide which is thermally stable past the temperature of clay scintering (about 805°C (Eddings et al., 1994) thus lead is likely to be retained within the clay matrix. If lead is present as a chloride, the salt remains intact up to the temperature of scintering and vaporized as the gaseous form of the salt.

How effective is the fluidized bed system in metal emission reduction? It is particularly good at reducing mercury emissions, because of the very low boiling point of mercury. Any reduction in the kiln operating temperature will lower the amount of mercury boiled off (Lee et al., 1993). Similarly, lead emissions are reduced. Flue gas had no detectable amounts of metal for three different operating temperatures (750, 800, and 850°C). In addition, the lead that remains has been incorporated into a glassy matrix which is not as leachable as ash from an ordinary incinerator. This is good news because in many ash materials the lead is present in an exchangeable form, making the lead more leachable and more likely to move upon wetting (Kuntz et al., 1990).

As we have noted the formation of lead chloride can be a problem. Where does the chloride come from? Gaseous emission of HCl was found in some studies from the late 1970s to be 38 to 113 mL HCl to 10^6 mL gas flow. This is of a sufficient concentration to form volatile metal chlorides (Greenberg et al., 1978). It is suggested that the combustion of plastics supplies the chlorides since a major component of plastics is polyvinylchloride. The halogens are used in plastic manufacturing leading to the great stability of the material. One reason for this stability is the fact that no biological polymers containing chlorides are common, thus nature has devised no method of destruction for these materials. This is really not good news because one study has suggested that the demand for plastics will hit 76 billion pounds by the year 2000 with the production of 380 million pounds of plastics in municipal solid waste. Plastics make up 7% of the solid wastes but 30% of msw (municipal solid waste). As of 1989 only 1% of the plastics were recovered. The implication is that plastics will form an every increasing component of incinerated materials. Plastics in construction/plumbing, food wrap, household product containers contribute to 1/3 of the HCl produced during incineration.

Plastics are also culprits as a direct source of the metal. Various metals are added to plastics to act as stabilizers (see Chapter 7). Currently the use of lead is restricted to use in wire and cable insulations. In one study investigators burned several plastics materials to determine if HCl and/or lead and cadmium were produced. PVC pipes, electrical wiring conduits, and extension cords all yielded HCl and minor amounts of lead. Cable insulation yielded HCl and major amounts of lead. So that we needn't feel too guilty, it should be noted that dishwasher detergent containers, antifreeze containers, and liquid toilet bowl containers did not produce either lead or HCl. However, all produced Cd.

A study of the fate of injected aqueous lead nitrate ($Pb(NO_3)_2/H_2O$) showed that a large fraction of lead is retained in the larger particle sizes, consistent with the decomposition of nitrate into oxides to form lead oxide particles

[10.23] $Pb(NO_3)_2 \rightarrow N_2 + PbO$

These larger sized particles are large enough to be collected efficiently (Mulholland and Sorofim, 1991) and are among those sampled in the fly ash (Pluss and Ferrel, 1991).

In summary we note the following relevant chemistry of lead in an incinerator. Metallic lead, having a large number of electrons is easily melted. Once melted it has a relatively low vapor pressure. Incinerator compounds of lead will most likely result in the production of lead chlorides or lead oxides. If lead chlorides are formed, they are volatile (easily brought into the gas phase) and can be transported to cooler parts of the furnace, where they are distributed into different size fractions, all of which have highly

soluble lead at the surface.

Reducing Incinerator Emissions

Several end process methods to reduce metal emissions have been suggested. Here the chemistry involves gas phase reactions with the exiting gas. One method involves spraying a slurry of $Ca(OH)_2$ into the hot flue gas. The following chemical reactions are thought to take place (Deuster et al., 1994):

[10.23]

$$SO_2 + Ca(OH)_2 + H_2O \rightarrow CaSO_3 \cdot ½H_2O + 3/2O_2$$

[10.24]

$$CaSO_3 \cdot ½H_2O + ½O_2 + 3/2H_2O \rightarrow CaSO_4 \cdot 2H_2O$$

The process of forming the solid calcium sulfate creates nucleation sites for metals, which can be captured simultaneously. The removal of lead was found to be 99.5% complete.

An alternative is to inject the scavenger sorbent particle (not form it via chemistry as in the example above) (Scotto et al., 1992). The particle size formed can be shifted from <0.1 micron to 0.2 to 0.8 micron size particles which are more amenable to capture. Types of sorbents might be kaolinite and alumina. These clays have the additional advantage of trapping lead, leading to sintering (see above).

One method that is common among incinerators is the electrostatic precipitator (ESP). In this system gas moves into the ESP and is sent downflow through a tube with protruding electrodes. Collision of the gas on the electrodes leads to a redox reaction which produces charged particles. The charged particles find each other in the gas phase, and condense to larger size particles, which fall by gravity into a particle collector (Figure 10.36). In one ESP lead was emitted with 98.6% control efficiency in the absence of the ESP device but at 99.9% efficiency with an ESP on line (Hentz Jr. et al., 1992). One advantage of the ESP method is that small size particulates (<5μ are deposited in lungs) can be captured. Particles in the size range 0.2 to 2 μ can be captures which helps esthetically since these are efficient light scatterers and contribute to the visibility of the smog (Henry, 1995).

The principle for the electrostatic precipitator was first observed by M. Hohlfeld in 1820 who noted that a spark discharged in a smoky bottle clears the bottle. Sir Oliver Lodge in 1883 suggested in the publication Nature that the principle could be used for air pollution devices. Walker and Hutchings built the first ESP in 1885 at a lead smelting works in North Wales. The device failed because the lead oxide fumes in the smelter had high electrical resistivity (hard to charge) and the voltage source was not large enough. The method was revived in 1907 by F. G. Cottrell with the advent of better voltage sources and used for sulfuric acid emissions and in 1910 for lead smelters. The patent right royalties enabled him to establish the Cottrell foundation which funds small grants for improvements in the teaching of chemistry.

How effective are various means of reducing emissions? Or, what are the health effects associated with the operation of the incinerator? The health effects of the emitted metals will depend upon the dilution factor of the emitted plume as it moves, and the size and chemistry of the emited particle. The size of the emitted particle, as we have seen, depends upon the chemistry of nucleation events within the stack. As we have also seen the size is important in determining whether or not the bronchial/tracheal system will filter effectively? The large the particle size the more likely it is to be expelled from the body. Particle sizes less than 0.05 μ can result in up to 60% deposition of the material in the lung. The data in Figure 10.37 shows that a significant fraction of incinerated $Pb(NO_3)_2$ ends up in the low particle size diameter with the fraction increasing with temperature (Mulholland and Sorofim, 1991; Scotto et al., 1992) but that adding a sorbent results in a change in the particle distribution (Figure 10.38).

A fair amount of lead is handled within the incinerator. One question that might arise is how safe the incinerator is to the workers at the site. One study surveyed 86 men out of 105 active employees at a Philadelphia furnace processing 375 tons of trash/day. The workers (bridge crane operators, mechanics, and firemen) were sampled for a variety of species including lead. Some areas were found to have lead content higher (0.49 mg/m^3) than that required by OSHA (0.15 mg/m^3). However, no difference in urinary or blood lead levels was found for workers classified as high and low lead exposure (Bresnitz et al., 1992).

II.F.3 Incinerator Risk Assessment of Emissions

We have yet to finish our cradle to grave assessment of metals in an incinerator. Once we have incinerated our waste, the remaining lead is either dispersed into the atmosphere or captured and still in

need of a final disposal. Where does the airborne metal end up and can it impact on the environment? The spread of the metal can be modeled by assuming a "bell curve" model to the shape and movement of the incinerator plume (Figure 2.21) (Seinfeld, 1986, p. 561-578). In this model the concentration of the metal at ground level at some distance downwind from the incinerator at a mean wind speed is a function of the range of wind directions vertically across the plume and the range of wind directions horizontally across the plume. The two main controllable factors in this model are the smoke stack height and the amount of emitted metal.

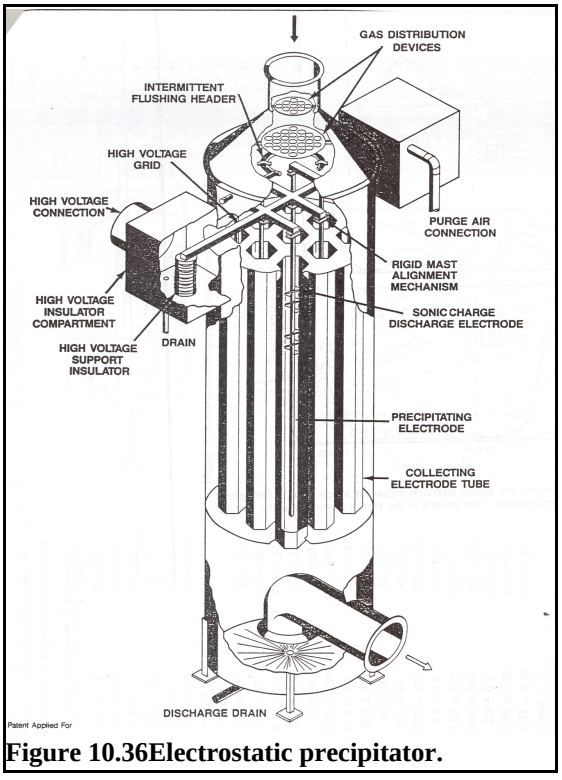


Figure 10.36Electrostatic precipitator.

[10.25]

$$\chi_x = \frac{Q}{2\pi\sigma_y\sigma_z u} e^{\left[\frac{-y^2}{2\sigma_y^2}\right]} \left\{ e^{\frac{(H-2)^2}{2\sigma_z^2}} + e^{\frac{-(H+2)^2}{2\sigma_z^2}} \right\}$$

where χ_x is the concentration of in the plume in the x distance, Q is the concentration as the point of exit, u is the wind velocity, H is the stack height, and $\sigma_{,y,z}$ are the standard deviation of the size of the plume in the x, y, and z direction. The standard deviation of the plume in the z direction varies with distance x (Turner, 1995).

The higher the stack the greater the dilution or dispersion of the metal and the smaller its concentration at any one given site. In general, it has been found that deposition varies as a logarithmic function of distance from the stack (Bache et al., 1991a; Candreva et al., 1986; Kimbrough and Suffet, 1995). For example in one stack 65 m high in England lead was emitted at 14 kg/day with a concentration of 100-1000 ng Pb/m^3. Deposition was 275 µg/m^2-day at 150 m, falling to 110 at 900 m, to 110 at 1500m to 50 at 2400 m from the stack.

Given that metal deposition has been found associated with an incinerator, what is the risk associated with such deposition? The risk to the surrounding community comes in two forms, risk of inhalation, and risk of soil contamination (Williams et al., 1987) and subsequent ingestion. Of these two, the risk of inhalation is thought to be greater (see Chapter 8). It is proposed that the most exposed individual (mei) with a 70 year exposure to the source should intake no more than 0.09 µg Pb/m^3 background concentration of air, which corresponds to an initial concentration of lead in rotary kiln burn of 32g/hr (Linak and Wendt, 1993). In general risk assessment studies suggest that risk is minimal, although it has been noted that when an individual has no choice in the matter, public tolerance is for zero (not minimal) risk.

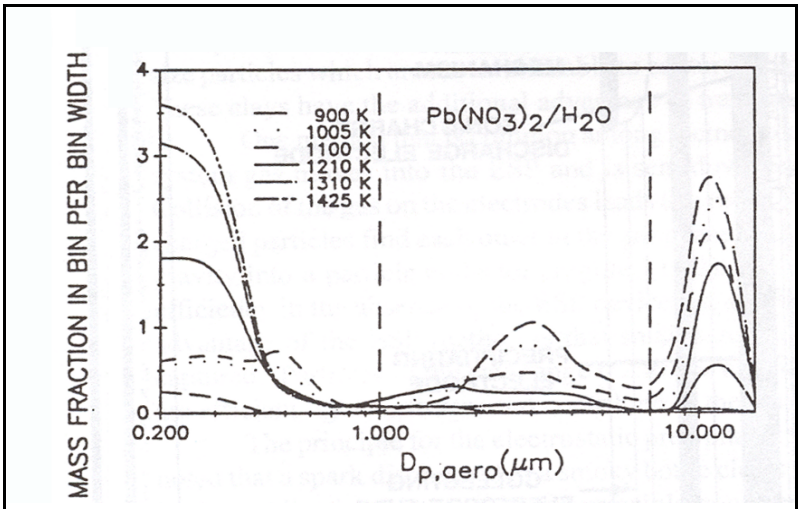


Figure 10.37 Particle size of lead nitrate formed during incineration. Mulholland, Env. Sci. Tech., 1992, 25, 268-274.

With decreasing amounts of lead

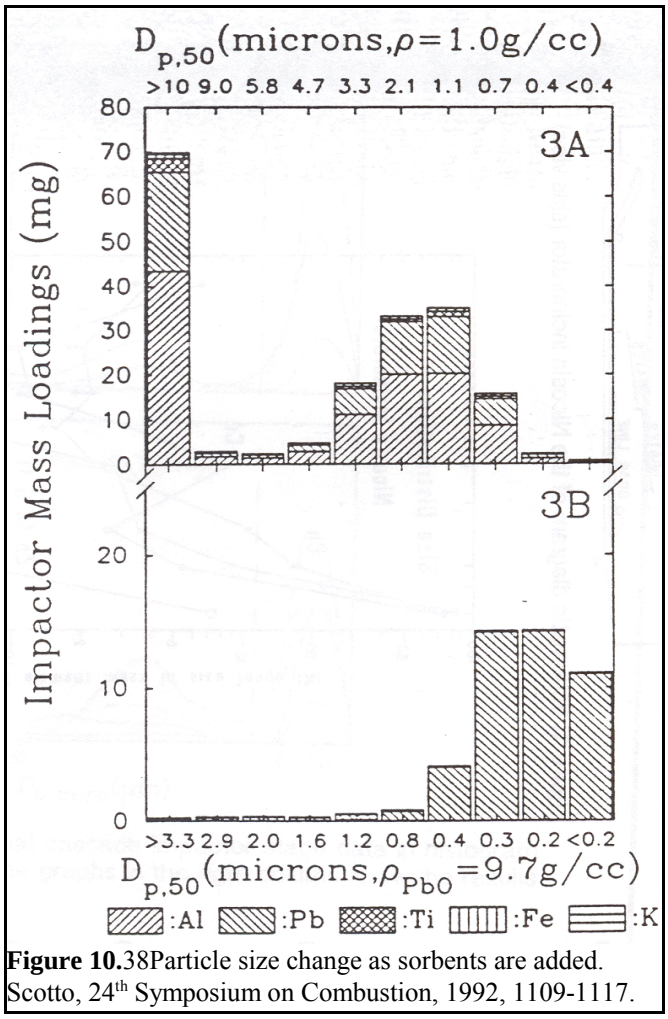


Figure 10.38Particle size change as sorbents are added. Scotto, 24th Symposium on Combustion, 1992, 1109-1117.

added to the air from leaded gasoline incinerator emissions are considered to be the major source of airborne lead. For N.Y. city it has been suggested that incinerator and not gasoline was the dominate source of lead in the last century (Jost, 1999).

Incinerators: Treatment of Fly Ash

We now take up the topic of the lead recovered in the ashed material. The ashes still need to be disposed of in a benign fashion. Most of the ash material is fairly leachable. This could be predicted from our study of the method by which lead is trapped in ash material. For the larger particles lead will be deposited as lead chloride on the surface matrix. These ashes can not be merely tossed to the wind, but must be disposed of with care.

These ashes, can, conceivably be buried like other waste, but their leachability must be first ascertained to see if the ashes will be stable under land fill conditions. One study looked at the water extractable lead from the ashes (Belevi et al., 1992). The concentration of lead fell with time, Table H.9. Using this data, the total rainfall of the system, the depth of the landfill, the concentration of the landfill effluent was calculated to be less than that concentration allowed. The solubility (leachability) of the ash was affected by the temperature at which the ash was formed, suggesting that chemical processes in incineration form different types of ash material. Less lead is leached from the ashes when the ashes were formed at higher temperatures. This does not mean that the ashes had a more stable form of lead, an alternative explanation is that a higher processing temperature for the waste resulted in greater thermal or vapor loss of the lead, thus reducing the lead content in the ash (Nublein et al., 1994).

The type of metal within the ash has been suggested to fall into five categories. The categories are those readily soluble (10 minute reaction at pH 7); those bound with carbonate (5 hours in pH 5), those bound to Fe/Mn oxides, those bound to organic material and sulphides, and the remainder non-extractable. The amount of lead present was also found to depend upon the source of the ash (Vela et al., 1993).

Fly ash composition has also been analyzed for speciation and physical size by X-ray element mapping and image analysis. X-ray diffraction showed lead is present in pure compounds (1-10μm in diameter); pure compounds within the glassy structure, and mixed within and on the surface of the silicate glass. The chemical composition of the lead compound was found to be primarily oxides as opposed to chlorides, sulfides, or carbonates (Landsberger et al., 1993).

Other workers hope to increase the stability of the ashes so that they be used in construction (cement blocks) or in road fill (Gong and Kirk, 1994). More work on the pH of various extracting solutions (pH of road run off?) will be needed to confirm that the bottom ash is suitable as roadfill material (Castagnoli et al., 1992).

One final alternative is to remove all Pb from the ash material. Thermal treatment at 670°C to 1300°C has been suggested since at this temperature lead should volatilize. At 1100°C (low enough to not melt the ash) 98-100% of lead was transferred to the gas phase within 1 hour [Jakkob, 1995 #1021].

Dust to Dust: Solidification and Stabilization in

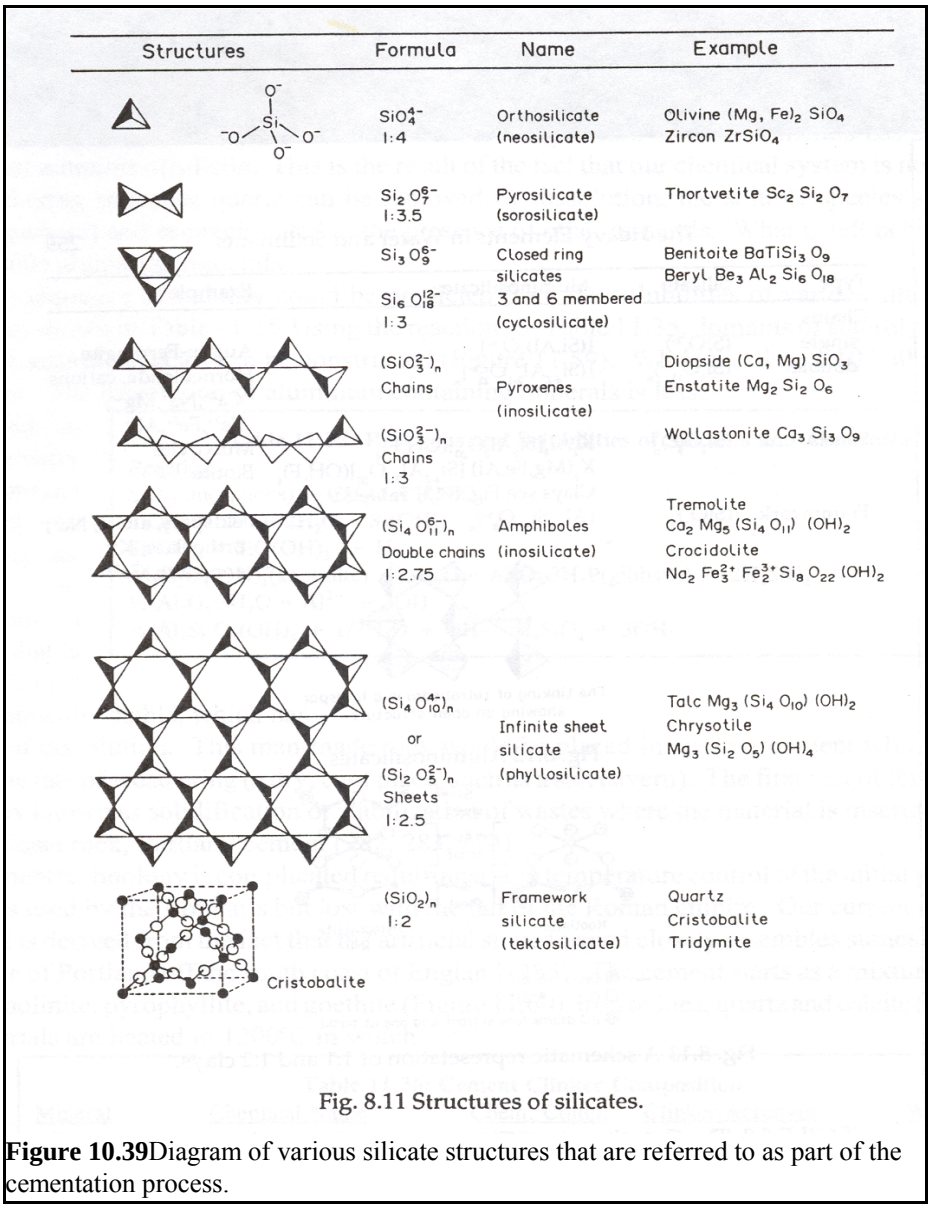


Fig. 8.11 Structures of silicates.

Figure 10.39 Diagram of various silicate structures that are referred to as part of the cementation process.

Cement

The end of the cycle for lead is its re-insertion into rock. The rock form in which we re-insert the lead should be one that is thermodynamically stable under earth crustal weathering conditions. Cementation technologies can be used for fly ash and for sewage sludges (Montgomery et al., 1988a).

Among the most common elements on earth (see Figure 1.13) are carbon, oxygen, silica, aluminum, so we expect to find these as common rocks. In particular, we find that most minerals contain Si, Al, and O. As chemists we might predict the thermodynamic stability of various configurations of Si, Al, and O based on the energies of formation involved in bonding. Consider the silicates, which we have already examined in some detail in a discussion of glasses in Chapter 4. If you recall silicate can form SiO_4^{2-} tetrahedra, with different degrees of crosslinking through oxygen to another cation (Si or other) (Figure 10.39). Tetrahedra are linked via oxygen. When two oxygen are shared a chain forms. When three oxygen are shared a sheet forms. When four oxygen are shared a three-dimensional network is formed (Bergna, 1994). Table H.10 shows some terminology associated with Si chemistry and mineralogy. The terminology in both cases is associated with the number and type of linkages between tetrahedron. The heats of formation can be calculated for neso-, soro-, ino- phyllo-, and tecto-silicates and are found to increase by a factor of two in the sequence. This suggests that, thermodynamically, weathering should favor dissolution of olivine to form diopside, which in turn dissolves to form hornblende, and finally feldspar.

Actual weathering sequences have been determined by soil scientists and geologists (Garner et al., 1993) shown in Table B.7, H.11. Notice that sequence 1-6 of Table H.11 is accurately predicted by the heats of formation in Table D.7. The final seven minerals do not appear to follow the heat of formation predictions, but notice that the composition of these minerals has shifted to include lesser amounts of silicon. This is the result of the fact that our chemical system is not closed. In the weathering sequence quartz can be removed by dissolution, the soluble

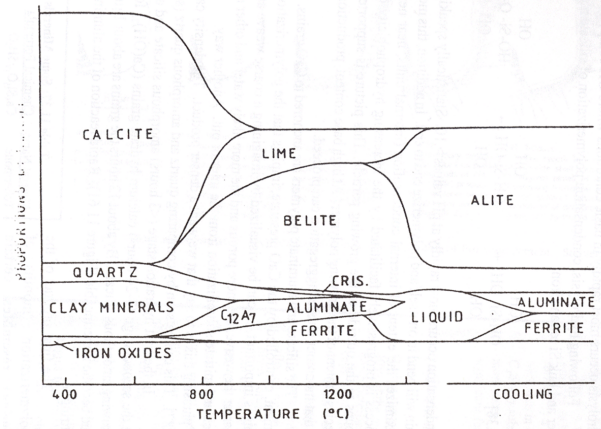


Figure 10.40 Clinker composition as a function of temperature.

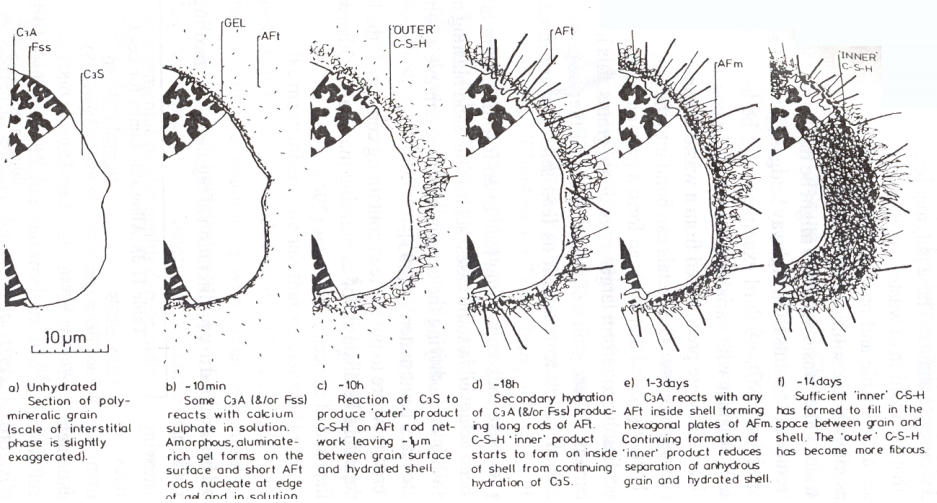


Figure 10.41 Growth of cement grains.

species separated (carried elsewhere) and re-precipitated in the presence of other minerals. What is left behind is the more insoluble aluminum minerals.

This sequence of stability could be predicted from the solubilities of various silicates and aluminates as shown in Table H.11 Using the reactions in Table H.11, domains of stability for solid SiO_2 and its solution species can be constructed. When $Si(OH)_4 < 10^{-3}$ all minerals will dissolve. The dissolution of aluminum containing minerals is less.

Based on this geochemistry we might predict that the best way of stabilizing our lead waste is to insert it into a rock phase that is "relatively" thermodynamically stable, which has a low **rate** of dissolution. This man-made rock would be placed in an environment which further decreases the rate of weathering (a dry, cool place, such as a salt cavern). The first part of this process is commonly known as solidification or stabilization of wastes where the material is inserted into an alumino-silicate rock, Portland cement (Daniali, 1990; Montgomery et al., 1988a; Montgomery et al., 1988b; Sebastian and 1992, 1992).

Cement technology is complicated requiring a high temperature control of the initial products. Cement was used by the Romans but the technology was lost with the fall of the Roman empire. Our current name for the material is derived from the fact that the artificial stone formed closely resembles stones quarried from the Isle of Portland off the south coast of England (Young, 1955). The cement starts as a mixture of clay minerals (kaolinite, pyrophyllite, and goethite (Figure 10.40), iron oxides, quartz and calcite ($CaCO_3$). These materials are heated to 1200°C in which endothermic reactions occur to produce an intermediate, CaO(lime), which reacts with other starting materials to give the final cement "clinker" composition of alite, β-belite, aluminate, and ferrite (Table H.13) (Taylor, 1990).

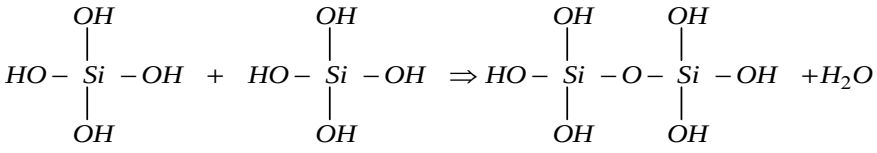
When added to water the "clinker" begins to hydrate to form Q^1 species which begin polymerization. The growth of silica chains can be followed by ^{29}Si NMR because the Si-O bond becomes strained and because the growth of the chain is related to the number of Si-OH bonds present. The formation of silica solid is thought to proceed via hydrolysis of an existing silicate:

$$\equiv Si - O - X + H_2O \underset{\leftarrow}{\overrightarrow{}} \equiv Si - OH + X - OH$$

Hydrolysis occurs most rapidly in basic conditions where OH competes with X more easily.

Following hydrolysis, condensation/polymerization of silicate monomers occurs to remove water and link Si tetrahedron:

Condensation occurs most rapidly at pH values > 10. Statistically speaking, it is more likely that the chain will bend in on itself and polymerize end to end. In addition, this mechanism of polymerization maximizes



the extent of internal condensation (internal units, near neighbor crosslinking). This process is progressively facilitated by the increasing hydrophobicity of the system pushing OH^- groups to the outside of the growing particle. This picture is supported by a high Q_2, silanediol, species content of the growing polymer. At high base content, production of monomers will be high, so that monomer cluster aggregation can proceed.

The silicate and aluminate polymers are anchored to CaO grains. The basicity of the hydrated cement, controlled by the CaO presence, implies that the polymerization chains are short in length. This is important, as can be visualized by imagining a coarse weave and a fine weave textile. The coarser the weave, the more porous and the more likely water and other materials are to penetrate the rock and initiate solubilization from the interior out. Another way of phrasing this is to say that we want a denser product. The density can be related to the solubility (Iler, 1955). This can be seen by comparing quartz and amorphous quartz (see Chapter 4).

In the first hydration stage (<3 hours) amorphous silicate and aluminate chains are formed. In the second stage (3-24 hours) calcium hydrate grains ($Ca(OH)_2$) form and are engulfed by the growing amorphous silicate. By about 12 hours the grains are about 1 μm thick and begin to coalesce and become impermeable (Figure 10.41). Rapid reaction of the aluminate phase takes place to form ettringite.

The final product of the polymerization (months of curing) resembles certain naturally occurring minerals: tobermorite, jennite, and ettringite (Table H.13). In nature jennite is associated with tobomorite and both are occasionally found with calcite. It is a late forming (end of the chemical sequence) mineral which fills open spaces in calcite (Carpenter et al., 1966).

The final stability of the cement is controlled by the density, which, in turn, is controlled by the polymer length in the silicate and aluminate chains. These polymer lengths are controlled by the basicity of the mix. It is at this point that the addition of wastes can impact on the form of the cement obtained. The types of wastes added are variable and have, consequently, variable effect on the cement. Some example waste streams might be the solutions removed during soil washing. The lead in this solution is precipitated as a nitrate. Other waste materials might be the 150,000-300,000 tons of blasting materials per year used to remove old paints from highway bridges (Braband and Loehr, 1993). These materials are already high in sand or silicate fraction (Benson Jr. et al., 1985). A third example might be lead contaminated sewage sludges (Montgomery et al., 1988a) which are high in organics with variable pH controlling constituents.

Those materials that contain lead that were precipitated from a waste stream may have lead somewhat soluble during the cement curing process. The metals can bond into the silicate phase by reaction with oxygen groups. It has been suggested that lead can form a gelatinous coating around the clinker grain. Since the grain must dissolve to reform larger more dense grains, this lead coating could inhibit the final curing of the cement. These grains of lead salts can further be re-solubilized and moved to the exterior of the cement block to redeposit as lead salts that are more highly leachable.

As implied above, the ability to make rock containing lead in an insoluble form is still an imperfect art. Success, however has been achieved in some cases, such as the sand blasting material (Barna et al., 1994). Research currently focuses on ways of modifying the cement matrix to achieve a better lock on the lead. Modification may include latex paint materials (Daniali, 1990).

The final question to be answered is where to put the lead containing cement. If the lead is as stable as hoped for, the cement blocks could be disposed of as building materials. Otherwise the blocks will need to be landfilled or disposed of in ocean floor sites (Lechich and Roethal, 1988).

Summary

A systems understanding of lead has resulted in a proliferation of regulations regardling limits of lead allowed in various media. These limits are generally moving downward in response to advances in understanding the biological mechanism for lead poisoning and increasingly sophisticated methods for detecting trace amounts. Public tolerance for lead poisoning is generally zero when the source of lead is

from "somewhere else" and higher when the lead is the result of individual choices and responsibility (own home/yard).

Environmental chemistry in the 1980s and 1990s has been focused on mapping the mass balance and flow within the environment of substances of interest. An example is the map shown in Figure 10.35. Types of values associated with Figure 10.35 for the City of Chicago can be given gross estimates. The last city owned operating municipal waste incinerator was cited for violations that allowed 7 pounds of lead/hr (124 lbs/day) to be emitted (the incinerator was shut down in 1995). The incinerator handled 1,200 tons of garbage a day in 1992. Estimates of lead in garbage from the early 1990s run from 110 to 1,500 g of lead/ton, leading to an estimate of total lead input to the incinerator of 290-3964 lbs Pb/day. Using the larger value the amount of lead emitted represented approximately 3.2% of the total lead input, consistent with other estimates of older (non-electrostatic precipitator retro-fitted stacks).

The fly ash therefore consists of approximately 160 to 3840 pounds of lead/day, leading to a very toxic bottom material. The current removal of lead from Chicago now moves through the municipal solid waste stream (no incineration) and through the Metropolitan Solid Waste District. The city of Chicago produces 1,115,000 tons of solid waste per year (1997) of which 157,112 tons are recycled. Of the recycled amount 154.54 tons consists of lead acid batteries. From these numbers it can be estimated that Chicago produces 2624 tons of garbage/day. Again applying the estimaged weight of lead to dry weight of garbage we calculate that the lead in the waste stream runs from 655 to 8669 lbs/day.

The sewage stream consists of approximately 450 dry tons of aged (5 year) sludge/day. The average lead content of that sludge is 166 ppm (dry weight). From this we can calculate an exit of 159.4 lbs Pb/day through the sewer system. This value of lead is sufficiently low that the sludge can be sold and used for surface application without restriction (Granato, 1996). Other researchers have shown that leafy crops (lettuce) grown on sludge amended soils have larger amounts of surface lead ((Sterrett et al., 1996).

An estimate (1998) of lead deposition from the air in the Chicago area show highest deposition over the city, less over Lake Michigan, and still less on the receiving end of Lake Michigan (Paode et al., 1998). This data shows 0.07 mg/m^2-day in the city, with 0.003 mg/m^2-day deposited to the southern portion of the Lake. The city of Chicago is 228.5 mi^2 from which we can estimate a dry deposition of 89.8 lbs/day of lead. AIRS data from the EPA for Chicago in 1999 shows an average air concentration of 0.0287 ug/m^3. If one assumes a well mixed air for tropospheric heights (12 km) and the surface area of Chicago, this implies that there is a total 469 lbs Pb in the total air directly over the city, which suggests that 19% of the atmospheric lead is deposited into the city and the remainder is lost. This estimate is low compared to more sophisticated estimates for the Los Angeles city basin (Lankey et al., 1998).

Chapter 10: Problems

1. In nearly all the cases of occupational health with respect to lead, how are workers portrayed?
2. Who was Midgely?
3. What does the term House of Butterflies refer to?
4. When was lead added to gasoline? Was it questioned?
5. When was lead banned from gasoline in the U.S.
6. How does B. Franklin get into our lead story?
7. What is the most significant phrase in B. Franklin's letter, in your opinion?
8. What is the difference between public and occupational health?
9. Where does the public health debate sit today?
10. What is the average estimated cost of renovating a housing unit (de-leading it?).
11. What might be some unintended consequences of laws intended to remove lead from housing units?
12. Who wrote the first important articles linking childhood disease to home paints?
13. How many years elapsed between that publication and banning of leaded paints in the U.S.?
14. Who brought attention to the link between childhood learning the leaded paints?

Chapter 10: Literature Cited

1981. NAACP v Wilminton Medical Ctr., INc. 49` F. Supp. 290, 307 n. 158 (D. Del. 1980), aff'd 657 F.2d 1322 (3d Cir. 1981).

Adgate, J.L., R.D. Willis, T.J. Buckley, J.C. Chow, J.G. Watson, G.G. Rhoads, and P.J. Lioy. 1998. Chemical Mass Balance Source Apportionment of Lead in House Dust. Environmental Science and Technology 32:108-114.

Allen, M. 2001. Bush to adopt Clinton's lead disclosure rules Washington Post, Washington, D. C.

Amitai, Y., M.J. Brown, J.W. Graef, and E. Cosgrove. 1991. Residential deleading: effects on the blood lead levels of lead-poisoned children. Pediatrics 88:893-7.

Anderson Jr., J.L. 1995. p. 209, *In* V. C. Coluccio, ed. Lead-Based Paint Hazards, Assessment and Management. Van Nostrand Reinhold, N.Y.

AP. 2001. New Hamshire Man pleads guilty in Lead paint poison case.

Arends, J., J. Christoffersen, M.R. Christoffersen, H. Eckert, B.O. Fowler, J.C. Heughebaert, G.H. Nancollas, J.P. Yesinowski, and S.F. Zawacki. 1987. A calcium hydroxyapatite precipitated from an aqueous solution; an international multimethod anlaysis. Journal of Crystal Growth 84:515-532.

Bache, C.A., W.H. Gutenmann, M. Rutzke, G. Chu, D.C. Elfving, and D.J. Lisk. 1991a. Concentrations of metals in grasses in the vicinity of a municipal refuse incinerator. Archives of Environmental Contamination and Toxicology 20:538-42.

Bache, C.A., W.H. Gutenmann, M. Rutzke, G. Chu, D.C. Elfving, and D.J. Lisk. 1991b. Concentrations of metals in grasses in the vicinity of a municipal refuse incinerator. Archives of Environmental Contamination & Toxicology 20:538-542.

Baltpurvins, K.A., R.C. Burns, G.A. Lawanace, and A.D. Stuart. 1996. Use of the solubility domain approach for the modeling of the hydroxide precipitation of heavy metals from wastewater. Environmental Science and Technology 30:1493-1499.

Barna, B., P. Moszkowica, J. Veron, and M. Tirnovearnu. 1994. Solubility model for the pore solution of leached concrete containing solidified waste. Journal of Hazardous Materials 37:33-38.

Barton, R.G., W.D. Clark, and W.R. Seeker. 1990. Fate of Metals in wste combustion systems. Combustion Science and Technology 74:327-342.

Barton, R.G., W.R. Seeker, and H.W. Bostian. 1991. The Behavior of Metals in Municiipal sludge incinerators. Process Safety and Enviornmental Protection: Transactions of teh Instruction of Chemical Engineers 69:29-36.

Bass, D.A., and J.A. Holcombe. 1988. Mechanisms of lead vaporization from an oxygenated graphite surface using mass spectrometry and atomic absorption. Analytical Chemistry 60:578-82.

Behmanesch, N., D.T. Aleen, and J.L. Warren. 1992. Flow rates and compositions of incinerated waste streams in the United States. J. Air Waste Manage. Assoc. 42:437-442.

Belevi, H., D.M. Stampfli, and P. Baccini. 1992. Chemical Behavior of Municipal Solid Waste Incinerator Bottom Ash in Monofills. Waste Management and Research 10:153-167.

Bellow, S. 1982. The Dean's December Pocket Books, N.Y.

Ben-Ali, R. 2001. Instances of Lead Poisoning Decrease Star-Ledger.

Benson Jr., R.E., H.W. chandler, and K.A. Chavez. 1985. Hazardous Waste Disposal As Concrete Admixture. Journal of Environmental Engineering 111:441-447.

Bergna, H.E. 1994. The colloid Chemistry of Silica American Chemical Society, Washington D. C.

Berti, W.R., and S.D. Cunningham. 1997. In-place inactivation of Pb in Pb-contaminated soils. Environmental Science and Technology 31:1359-1364.

Biemer, J. 2000. Landlord settles lawsuit for refusing to rent to lead-poisoned families Associated Press.

Bierman, P.M., and C.J. Rosen. 2994. Phosphate and Trace Metal Availability from Sewage-sludge incinerator Ash. Journal of Environmental Quality 23:822.

Blais, L.E. 1996. Environmental Racism Reconsidered. North Carolina Law Review 75:76-151.

Blaylock, M.J., D.E. Salt, S. Dushenkov, O. Zakharova, C. Gussman, Y. Kapulnik, B.D. Ensley, and I. Rasken. 1997. Enhanced accumulation of Pb in Indian Mustard by soil-applied chelating agents. Environmental Science and

Technology 1997:860-865.
Bornschein, R.L., c.S. Clark, J. Grote, B. Peace, S. Roda, and P. Succop. 1988. Soil lead-blood lead relationship in a former lead mining town, p. 149-160, *In* B. E. Davies and B. G. Wixson, eds. Lead in Soil: Issues and Guidelines. Science Reviews Limited, Northwood.
Bosch, C. 2002. Councilmember Dilan no help on "lead belt" relief Village Voice, N.Y.
Braband, D.J., and R.C. Loehr. 1993. Solidification/stabilization of spent abrasives and use as nonstructural concrete. Waste Management 13:333-339.
Bresnitz, E.A., J. Roseman, D. Becker, and E. Gracely. 1992. Morbidity among municipal waste incinerator workers. American Journal of Industrial Medicine 22:363-378.
Brown, S.F., B.D. Schultz, R.P. Clickner, and S. Weitz. 1995. Data analysis of lead in soil (HUD Survey Data), p. 3-12, *In* J. Breen, J. and C. R. Stroup, eds. Lead Poisoning exposure, abatement, regulation. CRC Presss, Boca Raton, Fla.
Brunner, P.H., and H. Monch. 1986. The flux of metals through municipal solid waste incinerators. Waste Management and Research 4:105-119.
Bullard, R.D. 1993. Environmental Equity: Examining the Evidence of Environmental Racism. Land Use Forum Winter:6.
Burgoon, D.A., S.W. Rust, and K.A. Hogan. 1995. Relationships among lead levels in blood, dust, and soil, p. 255-264, *In* J. J. Breen and C. R. Stroup, eds. Lead Poisoning exposure, abatement, regulation. CRC Press, Boca Raton, Fla.
Caher, J. 2001. NYPIRG seeks computer files on lead paint testing. New York Law Journal April 5.
Candreva, F., G. Vermeir, M. Nagels, and R. Dams. 1986. Heavy metals: atmospheric deposition around a municipal incinerator plant in Belgium. Studies in Environmental Science 29:197-205.
Canfield, R.L., C.R. Henderson, D.A. Cory-Slechta, C. Cox, T.A. Justo, and B.P. Lanphear. 2003. 348. 169 1517-1526.
Caplan, P.E. 1976. Monitoring of Workplaces for lead and problems involted Health Effects of Occupational Lead and Arsenic Exposure, a Symposium, Vol. HEW publicaton 76-134. U.S. Department of Health, Education, and Welfare (NIOSH), Washington, D. C.
Carpenter, A.B., R.A. Chalmers, J.A. Gard, K. Speakman, and H.F.W. Taylor. 1966. Jennite, a new mineral. American Mineralogist 51:56.
Castagnoli, O., E. Zavattiero, L. Musmeci, A. Alimonti, and S. Caroli. 1992. Environmental problems with the use of urban solid waste residues produced by thermodestruction. Spectroscopy Europe 10:10-16.
Casteneda, R. 2002. Landord gets 2 years in probe of lead paint Washington Post, Washington, D.C.
Chan, S. 2000. Lead's Toll on D. C. Children Washington Post, Nov. 29, 2000 ed, Washington, D. C.
Chaney, R.L., H.W. Mielke, and S.B. Sterrett. 1989. Environmental Geochemistry and Health 11:105.
Charney, E., B. Kessler, M. Farfel, and D. Jackson. 1983. Childhood lead poisoning. A controlled trial of the effect of dust-control measures on blood lead levels. New England Journal of Medicine 309:1089-93.
Chase, A.R. 1993. Assessing and Addressing Problems Posed by Environmental Racism. Rutgers Law Review 45:285-369.
Chisholm Jr., J.J. 2001. Evolution of the Management and Prevention of Childhood Lead Poisoning: Dependence of Advances in Public Health on Technological Advances in the Determination of Lead and RElated Biochemical Indicators of Its Toxicity. Environmental Research Section A. 86:111-121.
Chisholm Jr., J.J., E.D. Mellits, and S.A. Quaskey. 1985. The relationship between the level of lead absorption in children and the age, type, and coniditon of housing. Environmental Research 38:31-45.
Choe, K.T., M. Trunov, S.A. Grunshpun, K. Willeke, J. Harney, S. Trakumas, G. Mainelis, R.L. Bornschein, S. Clark, and W. Friedman. 2000. Particle settling after lead-basedpaint abatement work and clearnace waiting period. AIHAJ 61:798-807.
Christ, D. 1992. National Underwriters June 26.
Chung, E.K. 2001. Pediatrics:107.
Clickner, R.P., V.A. Albright, and S. Weitz. 1995. The Prevalence of Lead-Based Paint in Housing: Findings from the National Survey, *In* J. J. Breen and C. R. Stroup, eds. Lead Poisoning: Exposure, Abatement, Regulation. CRC Lewis.
Cohn, M., and S. Crockett. 2000. New law aims to protect landlords from lead-paint lawsuits Virginian-Pilot.
Colucco, V.M., (ed.) 1995. Lead-Based Paint Hazards Assessment and Management. Van Nostrand Reinhold, N.Y.

Coughlin, B.R., A.T. Stone, and G.W.C. Whiting. 1995. Nonreversible adsorption of divalent metal ions (MnII, CoII, NiII, CuII, and PbII) onto Goethite: Effects of acidification, fell addition, and picolinic acid addition. Environmental Science and Technology 29:2445-55.
Coursey, D. 1994. Environmental Racism in the City of Chicgo: The History of EPA Hazardous Wste Sites in African American NEighborhoods. University of Chicago.
Cowley, R.J., B.P. Gallagher, and B.M. Nee Jr. 1994. Development and Execution of a Metals Pretest Program for a Hazardous Waste Incinerator. Hazardous Waste and Hazardous Materials 11:31-51.
Daniali, S. 1990. Solidifcation/stabilization of heavy metals in latex modified Portland cement matrices. Journal of Hazardous Materials 24:225-230.
Davis, A., M. Ruby, V., and P.D. Bergstrom. 1992. Bioavailability of arsenic and lead in soils from the Butte, Montana, mining district. Environmental Science and Technology 26:461.
Davison, R.L., D.F.S. Natusch, J.R. Wallace, and C.A. Evans Jr. 1974. Trace Elements in Flay Ash. Dependence of concentration on particle size. Environmental Science and Technology 8:1107-1113.
Dennis, R.M., D. Dworkin, W.L. Lowe, and A.F. Zupko. 1992. Evaluation of commercially available soil-washing processes for site remediation. Hazardous and Industrial Wastes 24th:515-25.
Derickson, A. 1988. Workers' Health, Workers' Democracy Cornell University Press, Ithaca.
Deuster, E.V., A. Mensing, M.X. Jiang, and H. Majdeski. 1994. Eng. Prog. 113:149.
Dreyfus, L.A. 1907. The Health of the Painter. Painter and Decorator 20:277-278.
Duggan, M.J., and M.J. Inskip. 1985. Childhood exposure to lead in surface dust and soil - a community health problem. Public Health Review 13:1-54.
Duggan, M.J., M.J. Inskip, S.A. Rundle, and J.S. Moorcraft. 1985. Lead inplayground dust and on the hands of school children. Science of the Total Environment 44:65-79.
Dzenitis, J.M. 1997. Soil chemistry effects and flow prediction in electroremediation of soil. Environmental Science and Technology 31:1191-1197.
Eddings, E.G., J.S. Lighty, and J.A. Kozinski. 1994. Determination of Metal behavior during the incineration of a contaminated montmorillonite clay. Environmental Science and Technology 28:1791-1800.
Eldridge, C.A. 1998. Poisoned Painters: Organized Painters' Responses to Lead Poisoning in Earlth 20th Century America. Bulletin of Science, Technology and Society 18:266-280.
Ericson, J.B., H. Shirahata, and C.C. Patterson. 1979. Skeletal concentrations of lead in ancient Peruvians. N. Eng. J. of Med. 300:946-951.
Ernhart, C.B., S. Scarr, and D.F. Geneson. 1993. On Being a Whistle Blower, The Needleman Case. Ethics and Behavior 34:73.
Eskenazi, B., and P.J. Landrigan. 2002. Environmental Health perspectives andchildren's environmental health. Environmental Health Perspectives 110:A559-60.
Ettinger, A.S., R.L. Bornschein, M. Farfel, C. Campbell, N.B. Ragan, G.G. Rhoads, M. Brophy, S. Wilkens, and D.W. Dockery. 2002. Assessment of cleaning to control lead dust in homes of children with moderatelead poisoning: treatment of lead-exposed children trial. Environmental Health Perspectives 110:A773-A779.
Farrah, H., and W.F. Pickering. 1977. The sorption of lead and cadmium species by clay minerals. Australian Journal of Chemistry 30:1417-22.
Fergusson, J.E. 1990. The Heavy Elements: Chemistry, Environmental Impact, and Health Effects Pergamon Press.
Fernandez, M.A., L. Martinez, M. Segaria, J.C. Garcia, and F. Espiell. 1992. Behavior of Heavy Metals in the Combustion of gases of urban waste incinerators. Environmental Science and Technology 26:1040-1047.
Finz, S. 2000. Health dept. sued over lead tested: no screening of kids required, suit says The San Francisco Chronicle, San Francisco.
Fischer, F. 2000. Citizens, Experts, and the Environment Duke University Press, London.
Fisher, M. 1995. Environmental Racism Claims Brought Under Title VI of the civil rights Act. Environmental Law 25.
Flegal, A.R. 1998. Clair Patterson's influence on environmental research. Environmental Research Section A 78:65-70.
Ford, D.A., and M. Gilligan. 1988. The Effect of Lead Paint Abatement Laws on Rental Property Values. Am. J. Real Estate and Urban Economic Association 16:84.
Forstner, U. 1995. Land contamination by metals -global scope and magnitude of problem, p. 1-24, *In* H. Allen, E., et al., eds. Metal Speciation and Contamination of Soil. CRC Press, Boca Raton, Fla.
Freeman, G.B., J.D. Johnson, S.C. Liao, P.I. Feder, A. Davis, M. Ruby, V., R.A. Schoof, R.L. Chaney, and P.D.

Bergstrom. 1994. Absolute bioavailability of lead acetate and mining waste lead in rats. Toxicology 91:151-63.
Freeman, G.B., J.D. Johnson, J.M. Killinger, S.C. Liao, P.I. Feder, A. Davis, M. Ruby, V., R.L. Chaney, S.C. Lovre, and P.D. Bergstrom. 1992. Relative bioavailability of lead from mining waste soil in rats. Fundamental and Applied Toxicology 19:388-98.
Garcia-Miragaya, J. 1984. Levels, chemical fractination, and solubility of lead in roadside soils of Caracas, Venezuela. Soil Science 138:147-152.
Garner, A.G., R.L. Carrasquillo, and D.W. Fowler, (eds.) 1993, Vol. 37. The American Ceramic Society, Westerville, Ohio.
Gillespie, R. 1990. Accounting for lead poisoning: the medical politics of occupational health. Social History 15:303-331.
Gong, Y.D., and D.W. Kirk. 1994. Behavior of municipal solid waste incinerator flyash I: general leaching study. J. of Hazardous Materials 36:249-264.
Granato, T. 1996. *In* A. Fitch, (ed.), Chicago.
Greenberg, R.R., W.H. Zoller, and G.E. Gordan. 1978. Composition and size distributions of particles released in refuse incinerator. Environmental Science and Technology 12:566.
Gupta, S., G. van Houtven, and M. Cropper. 1995. Do benefits and costs matter in environmental regulation? An analysis of EPA decisions under Superfund, *In* R. L. Revesz and R. B. Steward, eds. Analyzing Superfund: Economics, Science and Law. Resources for the Future, Washington, D.C.
Hamilton, A. 1985. Exploring the Dangerous Trades Northeastern University Press, Boston.
Harrison, R.M., D.P.H. Laxen, and S.J. Wilson. 1981. Lead orthophosphates. IV. Formation and stability in the environment. Environmental Science and Technology 15:1378-1383.
Harter, R.D. 1983. Effect of Soil pH on Adsorption of Lead, Copper, Zinc, and Nickel. Soil Science Society of America Journal 47:47-51.
Haskell, M. 2002. Defusing a ticking "time bomb" is lead bill aim Maine Times.
Hassett, J.J. 1974. Capacity of selected Illinois soils to remove lead from aqueous solutions. Soil Science and Plant Analysis 5:499-505.
Held, T. 2002. City Using contractors to remove leadpaint in homes Journal Sentinel, Milwaukee.
Henry, N.E. 1995. Electrostatic Precipitators, *In* J. C. Mycock, et al., eds. Handbook of Air Polution Control Engineering and Technology. CRC Press, Boca Raton, Fla.
Hentz Jr., L.H., F.B. Johnson, and A. Baturay. 1992. Air emission studies of sewage sludge incinerators at the Western Branch wastewater treatment plant. Water Environment Research 64:111-119.
Herman, D.C., J.F. Artiola, and R.M. Miller. 1995. Removal of Cadmium, lead, and zinc from Soil by a rhamnolipid biosurfactant. Environmental Science and Technology 29:2280-5.
Hicks, R.E., and S. Tondorf. 1994. Electrorestoration of Metal contaminated soils. Environmental Science and Technology 28:2203-10.
Hill, J. 2000. Lead-test rules for kids inadequate, judge rules Bee Capitol Bureau.
Ho, T.C., C. Chen, J.R. Hopper, and D.A. Oberacker. 1992. Metal Capture during fluidized bed incineration of wastes contaminated with lead chloride. Combustion Science and Technology 85:101-116.
Ho, T.C., L. Hom-Ti, T.H. Kuo, D. Chen, and W.D. Bostick. 1994. Analysis of Incinerator Performance and Metal Emissions fro REcent Trial and Test Burns. Hazardous Waste and Hazardous Materials 11:53-70.
Hoffnagle, G.F. 1988. Real World Modeling of Blood-lead from Environmental Sources, p. p. 73, *In* B. E. Davies and B. G. Wixson, eds. Lead in Soil: Issues and Guidelines, Vol. 9. Science Reviews Limited, Northwood.
Huang, J.W., J. Chen, and S.D. Cunningham. 1997. Phytoextraction of lead from contaminated soils, p. 283-298 Phytoremediation of Soil and Water contaminants, Vol. 664. ACS, Washington D.C.
Hunt, A., D.L. Johnson, I. Thornton, and J.M. Watt. 1993. Apportioning the sources of lead in house dusts in the London borough of richmond, England. Science of the Total Environment 138:183-206.
Iler, R.K. 1955. The colloidal chemistry of silica and silicates Cornell University Press, Ithaca, N. Y.
Jaeger, R.J., A.L. Weiss, and W.I. Manton. 1998. Isotopic Ratio Analysis in Residential Lead-Based Paint and Associated Surficial Dust. Clinical Toxicology 36:691-703.
Jost, W. 1999. Researchers point to incinerator emissions as a major source of atmospheric lead concentrations. Environmental Science & Technology March 1:106A.
Kemper, A.R., W. Clayton Bordley, and S. Downs. 1998. Cost-effectiveness Analysis of Lead Poisoning Screening

Strategies Following the 1997 Guidelines of the Centers for Disease Control and Prevention. Archives of Pediatric Adolescent Medicine 152:1202.
Kerry, C.F. 1992. Lead Poisoning Claims, Awards are Multiplying. National Underwriters:32.
Kimbrough, D.E., and I.H. Suffet. 1995. Off-Site Forensic Determination of Airborne Elemental Emissions by Mult9-Media Analysis: A Case Study at Two SEcondary Lead Smelters. Environmental Science and Technology 29:2217-2221.
Kimbrough, R., M. LeVois, and D. Webb. 1996. Survey of lead exposure around a closed lead smelter. Pediatrics 97:603-4.
King, J.S. 1993. Will lead become a golden opportunity? Best's Review 94:24-7.
Kinniburgh, D.G., M.L. Jackson, and J.K. Syers. 1976. Adsorption of alkaline earth, transition, and heavy metal cations by hydrous oxide gels of iron and aluminum. Soil Science Society of America Journal 40:796-9.
Koss, E., and K.N. Dietrich. 2002. Neurotoxicology and Teratology.
Kuntz, H.T., C.A. Bache, and D.J. Lisk. 1990. Arch. Env. Contam. Toxicol. 19:399-404.
Lambert, B. 2000. Judge overturns lead-paint control law, citing perfunctory review by city council New York Times, New York.
Lambrecht, W. 2002. U.S. weighs lowering acceptable lead level in children Post-dispatch, St Louis.
Landrigan, P. 2002. Poor environment creates wealth of problems. Environmental Health Perspectives 110.
Landrigan, P.J., and A. Garg. 2002. Chronic effects of toxic environmental exposures on children's health. Journal of Toxicology. Clinical Toxicology 40:449-56.
Landrigan, P.J., C.B. Schechter, J.M. Lipton, M.C. Fahs, and J. Schwartz. 2003. Environmental pollutants and disease in American children: estimates of morbidity, mortality, and costs for lead poisoning, asthma, cancer, and developmental disabilities. Environmental Health Perspectives 110:721-8.
Landrigan, P.J., R.H. Whitworth, R.W. Baloh, N.W. Staehling, W.F. Barthel, and B.F. Rosenblum. 1975. Neurophysiologic dysfunction in children with chronic low-level lead absorption. Lancet:708-712.
Landsberger, S., B.A. Buchholz, M. Kaminski, and M. Plewa. 1993. Trace Elements in municipal solid waste incinerator fly ash. J. of Radioana. and Nuclear Chem. 167:331-340.
Lankey, R.L., C.I. Davidson, and F.C. McMichael. 1998. Mass Balance for Lead in the California South Coast Air Basin: an Update. Environmental Research 78:86093.
Lanphear, B.P., M. Weitzman, and S. Eberly. 1996. Racial differences in urbanchildren's environmental exposures to lead. American Journal of Public Health 86:1460-1463.
Lanphear, B.P., T.D. Matte, J. Rogers, R.P. Clickner, B. Dietz, R.L. Bornschein, P. Succop, K.R. Mahaffey, S. Dixon, W. Galke, M. Rabinowitz, M. Farfel, C. Rohde, J. Schwartz, P. Ashely, and D.E. Jacobs. 1998. The Contribution of Lead-Contaminated House Dust and Residential Soil to Children's Blood Lead Levels. Environmental Research Section A 79:51-68.
LaRue, S. 2002. Landlords who allow lead hazards may soon face prosecution Union-Tribune, South Dakota.
Lavelle, M., and M. Coyle. 1992. Unequal protection: the racial divide in environmental law: a special investigaton. National Law Journal:S2.
Law, S.L., and G.E. Gordon. 1979. Source of Metals in Municipal Incinerator Emissions. Environmental Science & Technology 13:431.
Lechich, A.F., and F.J. Roethal. 1988. Marine Disposal of stabilized metal processing waste. J. Water Poll. Cont. Fed. 60:93-99.
Lee, J.-K., K.-h. Lee, and H.-S. Chun. 1993. Behavior of Trace Metals in Fluidized-Bed Incineration of Sewage Sludge. J. of Chem. Eng. of Japan 26:179-182.
Li, Z., J.-W. Yu, and I. Neretnieks. 1998. Electroremediation, removal of heavy metals from soils by using cation selective membrane. Environmental Science and Technology 32:394-397.
Liang, L., and J.F. McCarthy. 1995. Colloidal transport of metal contaminants in groundwater, p. 87-112, *In* H. Allen, E., et al., eds. Metal Speciation and Contamination of Soil. Lewis Publishers, Boca Raton.
Linak, W.P., and J.O.L. Wendt. 1993. Toxic Metal Emissions From incineration: Mechanisms and Control. Prog. Energy Combust. Sci. 19:145-185.
Lindsay, W.L. 1979. Chemical Equilibrium in Soils Wiley, New York.
Loeb, A.P. 1994. The hazards of gasoline lead additives The Car, Its Fuel, Air Pollution, and Regulation.
Luckner, L., and W.M. Schestakow, (eds.) 1991. Migration Processes in the soil and groundwater zone. Lewis, Chelsea,

Michigan.
Lue-Hing, C., D.R. Zenz, T. Granato, and g.R. Richards. 1991. Impact of proposed USEPA 40 CFR Part 502 regulations on sewage sludge management by themetropolitan water reclamation district of greater Chicago. Metropolitan Water Reclamation District of grater Chicago, Chicago.
Ma, Q.Y., S.J. Traina, and T.J. Logan. 1993. In Situ Lead Immobilization by Apatite. Environmental Science and Technology 27:1803-1810.
Ma, Q.Y., T.J. Logan, and S.J. Traina. 1995. Lead immobilization from aqueous solutions and contaminated soils using phosphate rocks. Environmental Science and Technology 29:1118-1126.
Ma, Q.Y., S.J. Traina, T.J. Logan, and J.A. Ryan. 1994. Effects of Aqueous Al, Cd, Cu, Fe(II), Ni, and Zn on Pb immobilization by hydroxyapatite. Environmental Science and Technology 28:1219-1228.
Marbury, H.J. 1995. Hazardous Waste Exportation: The Global Manifestation of Environmental Racism. Vanderbilt Journal of Transnational Law 28:251-294.
Marcus, A.M., and J. Cohen. 1988. Modelling the blood lead- soil lead relationship, p. 161-171, *In* B. E. Davies and B. G. Wixson, eds. Lead in Soil. Science Review Limited, Northwood.
Marino, P.E., P.J. Landrigan, J.W. Graef, A. Nussbaum, G. Bayan, K. Boch, and S. Boch. 1990. A case report of lead paint poisoning during renovation of a Victorian farmhouse. American Journal of Public Health 80:1183-5.
Markowitz, G., and G. Rosen. 1991. The Illusion of Medical Certainty: Silicosis and the Politics of Industrial Disability, 1930-1960, *In* C. E. Rosenberg and J. Golden, eds. Framing Disease, Studies in Cultural History. Rutgers University Press, New Brunswick.
Marshall, E. 1984. Senate considers Lead gasoline ban. Science 225:34.
McBride, M.B. 1976. Nitroxide spin probeson smectite surfaces. Temperature and solvation effects on the mobility of exchange cation. Journal of Physical Chemistry 80:196-203.
McBride, M.B., T.J. Pinnavaia, and M.M. Mortland. 1975. Electron spin resonance studies of cation orientation in restricted water layers on phyllosilicate (smectite) surface. Journal of Physical Chemistry 79:2430-2435.
McClure, R. 2001. Town split over direction of cleanup Seattle Post-Intelligencer, Seattle.
McCord, C.P. 1954. Lead and Lead Poisoning in Early America: Clinical Lead Poisoning in the Colonies. Industrial Medicine and Surgery 23:120-125.
McGrew, J. 2002. Woman awarded $860,000 Montgomery Advertiser.
McKenzie, R.M. 1978. The effect of two manganese dioxides on the uptake of lead, cobalt, nickel, copper and zinc by subterranean clover. Australian Journal of Soil Research 16:208-214.
McMartin, A.M. 1906. Correspondence. Painter and Decorator 20:84.
Miller, D.D., and B.R. Schricker. 1981. In Vitro Estimation of Food Iron Bioavailability, *In* C. Kies, ed. Nutritional Bioavailability of Iron, Vol. 203. American Chemical Society, Washington.
Montgomery, D.M., C.I. Sollars, and R. Perry. 1988a. Cement-based solidification for the safe disposal of heavy metal contaminated sewage sludge. Waste Management and Research 6:217-226.
Montgomery, D.M., C.J. Sollars, and R. Perry. 1988b. Cement-based solidification for the safe disposal of heavy metal contaminated sewage sludge. Environmental Technology 6:217-26.
Morselli, L., S. Zappoli, and S. Militerno. 1993. The presence and distribution of heavy metals in municipal solid waste incinerators. Toxicological and Environmental Chemistry 37:137-145.
Mulholland, J.A., and A.F. Sorofim. 1991. Mechanisms of inorganic particle formation during suspension heating of simulated aqueous wastes. Environmental Science and Technology 25:268-274.
Murozumi, M., T.J. Chow, and C.C. Patterson. 1969. Chemical Concentrations of pollutant lead aerosols, terrestrial dusts and sea salts in Greenland and Antarctic snow strata. Geochimica et Cosmochimica Acta 33:1247-1294.
Murphy, K. 2000. A deep and wide mining scar in Idaho L. A. Times, Los Angelos.
Nakamura, K., S. Kinoshita, and H. Takatsuki. 1996. The Origin and behavior of lead, cadmium and antimony in MSW incinerator. Waste Management 16:509-517.
Needleman, H.L. 1991. Reply to Ernhard, Scarr, and Geneson. Ethics and Behavior 3:95-101.
Needleman, H.L. 1998. Clair Patterson and Robert Kehoe: "Two Views of Lead Toxicity". Environmental Research 78:79-85.
Needleman, H.L., J.A. Riess, M.J. Tobin, G.E. Biesecker, and J.B. Greenhouse. 1996. Bone lead levels and delinquent behavior. Journal of the American Medical Association 275:363-370.
Nriagu, J.O. 1974. Lead Orthophosphates IV. Formation and stability in the enviornment. Geochimica Acta 38:887-898.

Nriagu, J.O. 1998. Clair Patterson and Robert Kehoe's Paradigm of "Show Me the Data" On Environmental Lead Poisoning. Environmental Research Section A 78:71-78.
Nublein, F., P. Wunsch, F. Rampp, and A. Kettrup. 1994. Influence of Combustion Bed Temperature on Concentration and Leachability of Metals in Slags from an incinerating Plant. Chemosphere 28:349.
Paode, R.D., S.C. Sofuoglu, J. Sivadechathep, K.W. Woll, and T.M. Holsen. 1998. Dry deposition fluxes and mass size distribution of Pb, Cu, and Zn Measured in Southern Lake Michigan during AEOLOS. Environmental Science and Technology 32:1629.
Parish, N. 2001a. Law Will Require Lead Testing of Some Children St. Louis Post-Dispatch, St. Louis.
Parish, N. 2001b. Missouri legislators ok plan for annual testing of children for lead poisoning St. Louis Post-Dispatch, St. Louis.
Parker, L. 2002. Lead testing policy may change Bush administration considers letting states decide on screening of children USA Today.
Patterson, C.C., and D.M. Settle. 1974. The Reduction of Orders of Magnitude Errors in Lead Analyses of Biological Materials and Natural Waters by Evaluating and Controlling the Extent and Sources of Industrial Lead Contamination Introduced during sample collecting, handling, and analysis. Accuracy in Trace Analysis, Vol. Special Publication 422. National Bureau of Standards.
Patterson, C.C., and D.M. Settle. 1977. Comparative distributions of alkalies, alkaline earths and lead among major tissues of the tuna, Thunnas alalunga. Marine Biology 39:289-295.
Patterson, C.C., J.B. Ericson, M. Manea-Krichten, and H. Shirahata. 1991. Natural skeletal levels of lead in Homo sapiens spaiens uncontaminated by technological lead. Science of the Total Environment 107:205-263.
Paustenbach, D.J. 1989. A survey of health risk assessment. The risk assessment of environmental and human health hazards 27:27.
Pedro, V.A. 2000. Still Haxy after all these years: New York city's local law 38 andthe legislative debate over landlord liability in lead paint poisonig cases. Seton Hall Legislative Journal 24:541.
Pelton, T. 2001. Lead paint rule could limit research in Maryland Sun.
Perino, J., and C.B. Ernhart. 1974. The relation of subclinical lead level to cognitive and sensorimotor impairment in black preschoolers. J. Learn. Disab. 7:26-30.
Pershing, D.W., J.S. Lighty, G.D. Silcox, M.P. Heap, and W.D. Owens. 1993. Solid Waste incineration in rotary kilns. Combustion Science and Technology 93:245-276.
Peters, R.W., and L. Shem. 1995. Treatment of soils contaminated with heavy metals, p. 255-274, *In* H. Allen, E., et al., eds. Metal
speciation and Contamination of Soil. CRC Press, Boca Raton, Fla.
Petts, J. 1994. Incineration as a waste management option, *In* R. E. Hester and R. M. Harrison, eds. Issues in Environmental Science and Technology, 2: Waste Incineration and the Environment. Royal Society of Chemistry, Cambridge.
Picard, K. 2000. Lead Astray Missoula Independent, Missoula, Montana.
Pitcher, H.M. 1989. IQ and lead exposure: analytic issues arising in the water lead and gasoline lead standards. American Journal of Agricultural Economics 71:475-479.
Placitella, C.M., and B.R. Sugarman. 1995. *In* V. M. Colucco, ed. Lead Based Paint Hazards Assessment and Management. Van Nostrand Reinhold, N.Y.
Pluss, A., and R.E. Ferrel. 1991. Characterization of Lead and Other Heavy Metals in Fly Ash From Municipal Waste Incinerators. Hazardous Waste and Hazarous Materials 8:275-293.
PRNewswire. 2001. EPA setles with Hyde Park realty on lead-based paint, Chicago.
PRNewswire. 2002. EPA and HUD announce landmark led disclosure settlement, Chicago.
PRNewswire. 2003. U. S. settles case against Los Angeles Landlords and More than 4,5000 Apartments to beome lead save.
Pulido, L. 1996. A Critical Review of The Methodology of Environmental Racism Research. Antipode 28:142-159.
Pulido, L., S. Sidawi, and R.O. Vos. 1996. An Archaeology of Enviornmental Racism in Los Angeles. Urban Geography 17:419-439.
Purdy, R.D., and C.B. Hessley. 1929. Managerial Problems of the White Lead Industry, Chicago.
Raghuraman, B.J., N.P. Tirmizi, K. B.-S., and J.M. Wiencek. 1995. Emulsion liquid membranes for wastewater treatment: equilibrium models for lead- and cadmium-di-ethylhexyl phosphoric acid systems. Environmental

Science and Technology 29:979-984.
Rampley, C.G., and K.L. Ogden. 1998. Preliminary studies for removal of lead from surrogate and real soils using a water soluble chelator: adsorption and batch extraction. Environmental Science and Technology 32:987-993.
Reeves, R.D., and B.R. R. 1983. Hyperaccumulation of lead and zinc by two metallophytes from mining areas of Centra Europe. Environmental Pollution, Series A: Ecological and Biological 31:277-285.
Reimann, D.O. 1989. Heavy Metals in domestic refuses and then their distribution in incinerator residues. Waste Management and Research 4:105-119.
Rice, D.C. 2000. Parallels between Attention Deficit Hyperactivity Disorder and Behavioural Deficits produced by Neurotoxin Exposure to Monkeys. Environmental Health Perspectives 108:405-408.
Robinson, M.B. 2002. Few Medicaid kids screened for lead. South Florida Sun-Sentinel.
Rogan, W.J., and J.H. Ware. 2003. Exposure to lead in Children -How low is low enough? New England Journal of Medicine 348:1515.
Roig-Franzia, M. 2002. Md. Court decides lead paint appeal renters can sue housing agencies. Washington Post Washington, D. C.
Romano, J. 2001. Lead Paint - A ruling for tenants N. Y. Times, New. York.
Rosner, D., and G. Markowitz. 1985. "A gift of God?": the public health controversy over leaded gasoline during the 1920s. American Journal of Public Health 75:344.
Roy, S.B., and D.A. Dzombak. 1997. Chemical factors influencing colloid-facilitated transport of contaminants in porous media. Environmental Science and Technology 31:656-664.
Ruby, M., V., A. Davis, and A. Nicholson. 1994. In situ formation of lead phosphate in soils as a method to immobilize lead. Environmental Science and Technology 28:646-654.
Ruby, M., V., A. Davis, J.H. Kempton, J.W. Drexler, and P.D. Bergstrom. 1992. Lead Bioavailability: Dissolution kinetics under simulated gastric conditions. Environmental Science and Technology 26:1242-1248.
Ruby, M., V., A. Davis, T.E. Link, R. Schoof, R.L. Chaney, G.B. Freeman, and P. Bergstrom. 1993. Development of an in Vitro Screening Test to Evaluate the in Vivo Bioaccessibility of Ingested Mine-Waste Lead. Environmental Scienceand& Technology 27:2870-2877.
Russell, B.Z. 2001a. Ruling stalls cleanup at Bunker Hill Spokesman Review, Oct. 4, 2001 ed.
Russell, B.Z. 2001b. Basin Cleanup trial opens in Boise The Spokesman Review.
Rustenholz, W., and H. Smith. 1989. American Ink Maker November:12-17.
Ryan, D., and R. Scott. 2000. The promise of environmental sampling and right-to-know laws for at-risk communities. Public Health Reports 115:511.
Sandler, H.M. 1993. Lead - legal alchemy. Occupational Hazards Aug.:49-50.
Schneider, A. 2002. EPA still draws ire in the idaho panhandle Post Dispatch.
Schwartz, S., L. Zieve, and C.F. Watson. 1951. An improved method for determination of urinary coproporphyrin and an evaluation of factors influencing the analysis. J. Lab. Clin. Med. 37:843-859.
Scotto, M.V., T.W. Peterson, and J.O.L. Wendt. 1992. Hazardous waste incineration: the In-situ capture of lead by sorbents in a laboratory down flow combustor. Symposium on Combustion 24:1109-1117.
Sebastian, M., and 1992. 1992. Applicaton of thermal analysis to solid industrial waste treatment. Journal of Thermal Analysis 38:973-9.
Seinfeld, J.H. 1986. Atmospheric Chemistry and Physics of Air Pollution Wiley, New York.
Selevan, S., G., D. Rice, C., K. Hogan, A., S. Euling, Y., A. Pfahles-Hutchens, and J. Bethel. 2003. Blood lead concentration and delayed puberty in girls. The New England Journal of Medicine 348:1526.
Shapiro, A.P., and r.F. Probstein. 1993. Removal of contaminants from saturated clay be electroosmosis. Environmental Science and Technology 27:283-291.
Sheng, G., S. Xu, and S.A. Boyd. 1999. A dual function organoclay sorbent for lead and chlorobenzene. Soil Science Society of America Journal 63:73-78.
Sicherman, B. 1984. Alice Hamilton, A Life in Letters Harvard University Press, Cambridge.
Slovic, P. 2000. The Perception of Risk Earthscan, London.
Stashenko, N. 2001. High court extends landlord liability in lead poisoning cases Associated Press.
Steele, K.D. 2002. Mining enriched region, left big mess The Spokesman-Review, Seattle.
Steele, K.D. 2003. Critics rip plan to safguard Lake Cour d'Alene The Spokesman-Review.
Steresky, P.B., and M.J. Lynch. 2001. Arch. Pediatr. Adolesc. Med. 155:579-582.

Sterrett, S.B., R.L. Chaney, C.H. Gifford, and H.W. Mielke. 1996. Influence of fertilizer and sewage sludge compost on yield and heavy metal accumulation by lettuce grown in urban soils. Environmental Geochemistry and Health 18:135-142.
Stolberg, S.G. 2002. Bush's science advisers drawing criticism New York Times, New York.
Succop, P., R.L. Bornschein, K. Brown, and C.-Y. Tseng. 1998. An empirical comparison of lead exposure pathway models. Env. Health Perspectives 106:1577-1583.
E. Aleshin (ed.) 1976. Mineral Waste Utilization Symposium, Chicago Il. IIT Research Institute.
Taylor, H.F.W. 1990. Cement Chemistry Academic Press, London.
Taylor, S. 2002. Lawmaker seeks better procedures to check for lead poisoning Lewiston Maine Sun Journal, Lewiston, Maine.
Torres, G. 1992. Race, Class, and Environmental REgulation. Colorado Law Review 63:839-848.
Tuin, B.J.W., and M. Tels. 1990. Distribution of Six Heavy Metals in contaminated Clay Soils before and after extractive cleaning. Environmental Technology 11:935-948.
Turner, B.D. 1995. Workbook of Atmospheric Dispersion Estimates. 2nd ed. CRC Press, Boca Raton, Fla.
Tuthill, R.W. 1996. Hair lead levels related to children's classroom attention deficit behavior. Archives of Environmental Health 51:214-220.
Vela, L.D., R.E. Jervis, and S.S. Krishnan. 1993. The Leachability of elements in solid wastes. J. of Radioanal. and Nuclear Chem. 169:39-45.
Verhovek, S. 2002. In twist, Idahoans fight toxic cleanup N. Y. Times, N. Y.
Voynick, S.M. 1984. Leadville: A Miner's Epic Mountain Press Publishing Co, Missoula, Montana.
Waterland, L.R., D.J. Fournier Jr., J.W. Lee, and G.J. Carroll. 1991. Trace Metal Fate in a rotary kiln incinerator with an ionizing scrubber. Waste Management 11:103.
Weintraub, M. 1997. Racism and Lead Poisoning. American Journal of Public Health 87:1871.
Weiss, R. 2002. HHS seeks science advice to match Bush views Washington Post, Washington D.C.
Weitzman, M., A. Asehengrau, D.C. Bellinger, and R. Jones. 1993. Lead-contaminated soil abatement and urban children's blood lead levels. Journal of the American Medical Association 269:1647.
Williams, F.L.R., O.L. Lloyd, and M.M. Lloyd. 1987. Animal and Human Disease Around a Site with Two Incinerators: A Review,. Asia-Pacific Symposium on Environmental and Occupational Toxicology:475.
Williams, P.T. 1994. Pollutants from incinerators: An overview, p. 27-52, *In* R. E. Hester and R. M. Harrison, eds. Issues in Environmental Science and Technology 2: Waste Incineratin and the Environment. Royal Society of Chemistry, Cambridge.
Wixson, B.G., and B.E. Davies. 1993. Lead in Soil, Recommended Guidelines Northwood.
Wixson, B.G., and B.E. Davies. 1994. Guidelines for lead in soil. Proposal of the Society for Environmental Geochemistry and Health. Environmental Science and Technology 28:26a-31a.
Wolf, A., K. Bunzl, F. Dietl, and W.F. Schmidt. 1977. Effect of calcium ions on the adsorption of lead(2+), copper(2+), cadmium(2+), and zinc(2+) by humic substances. Chemosphere 6:207-213.
Wright, A. 1993. Unequal Protection Under the Environmental Laws: Reviewing the Evidence on Enviornmental Racism and the Inequities of Environmental Legislation. The Wayne Law Review 39:1725-1752.
Wu, C.Y., and P. Biswas. 1993. An Equilibrium Analysis to Determine the Speciation of Metals in an Incinerator. Combustion and Flame 93:31-40.
Wu, C.Y., P. Biswas, and N.J. Fendinger. 1994. Model to assess heavy metal emission from municipal solid waste incineration. Hazardous Waste and hazardous Materials 11:71-92.
Wu, J., F.C. Hsu, and S.D. Cunningham. 1999. Chelate-assisted Pb Phytoextracton: Pb Availability, uptake, and translocation constraints. Environmental Science and Technology 33:1898-1904.
Xu, J.-S., X. Xu, Q.-J. Yan, Y.-S. Jin, and X.-C. fu. 1992. Oxidative coupling of methane over Sr-Pb-O and Sr-Pb-O/gama-Al2O3 Catalysts. Journal of Natural Gas Chemistry:187-199.
Yaffe, Y., C.P. Flessel, J.J. Esolowski, A. del Rosario, G.N. Guirguis, V. Matias, T.E. Degarmo, G.C. Coleman, J.W. Gramlich, and W.R. Kelly. 1983. Identification f Lead Sources in Californai Children Using the Stable Isotope Ratio Technique. Archives of Environmental Health 30:237-245.
Young, J.S. 1955. A Brief outline of the history of cement Lehigh Portland Cement Co., Allentown, Pa.
Zimdahl, R.L., and R.K. Skogerboe. 1977. Behavior of lead in soils. Environmental Science and Technology 11:1202-1207.

1. Voynick
2. Ruby, M. V., Davis, A., Kempton, J. H., Drexler, J. W., Bergstrom, P.D., **Env. Sci. Tech**, 1992, 26(6), 1242-1248.