

Standing Up to the Lead Industry: An Interview with Herbert Needleman

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INTRODUCTION

Herbert Needleman, MD, is a pioneer in the history of medicine who has helped transform our understanding of the effect of lead on children's health. In the 1970s, he revolutionized the field by documenting the impact of low lead exposure on the intellectual development and behavior of children. In 1979, he published a highly influential study in the *New England Journal of Medicine*¹ that transformed the focus of lead research for the next generation and played a critical role in the elimination of lead in gasoline and the lowering of the CDC's blood lead standard for children. Building on a study by Byers and Lord in 1943 and those of Julian Chisolm and others in the 1950s and 1960s,

which had documented a variety of chronic damage affecting children who showed acute symptoms of lead poisoning, Needleman's innovative study analyzed the lead content of the teeth of schoolchildren, correlating it with the children's behavior, IQ, and school performance.

Not surprisingly, Needleman became the focus of the lead industry's ire. Beginning in the early 1980s, the industry's attacks on his research and use of public relations firms and scientific consultants to undermine his credibility became a classic example of how an industry seeks to shape science and call into question the credibility of those whose research threatens it. Industry consultants demanded that the Environmental Protection Agency and, later, the Office of Scientific Integrity at the National Institutes of Health, investigate Needleman's work. And then, in 1991, under pressure from industry consultants, the University of Pittsburgh formed a committee to evaluate the integrity of his lead studies.

Ultimately the federal government and the university found no basis for questioning Needleman's integrity or the results of his research. But the impact of the industry's actions affected both Needleman's academic life and the field of lead research. On the one hand, the industry explicitly showed the power it had to disrupt researchers' lives if they dared to question the safety of its products. On the other hand, Needleman's experience galvanized a generation of researchers who were profoundly influenced by the implications of his studies. In the quarter century since "Deficits in Psychologic and Classroom Performance of Children with Elevated Dentine Lead Levels"¹



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was published, Philip Landrigan, John Rosen, Bruce Lanphear, Kim Dietrich, and others have built on Needleman's work, confirming his findings as well as opening new areas of research that have shown that lead, at virtually any level, has negative, life-altering consequences for children. This interview, conducted on the eve of his 75th birthday, recounts a small part of Herb Needleman's experiences over the course of the last half century.

Editors' note: Dr. Needleman was interviewed in his office at the University of Pittsburgh on December 11, 2003, by Drs. Rosner and Markowitz.

PHR: Let's start with a little background about your family and your education.

I'm a Philadelphian by birth. I was born in 1927. My father was in the furniture business. I was the first person in my family to go to college. I went to Muhlenberg College in Allentown, Pennsylvania, and then to the University of Pennsylvania Medical School. I interned at Philadelphia General Hospital. I had initially intended to be an internist, but I discovered I was having much more fun in pediatrics. I did a fellowship in rheumatic fever research at Children's Hospital in Philadelphia. I then went into the Army and was an Army pediatrician. I hadn't been trained yet, but I worked under a board pediatrician. When he was discharged [from the Army], I became the chief of pediatrics. I had 12 inpatient beds, and a big, and very busy, outpatient clinic. We had a hundred deliveries a month at Ft. Meade, so that meant we had seven preemies [a month]. I learned about pediatrics in a hurry.

I had a consultant from Johns Hopkins who came out once a week—a very fine, distinguished pediatrician, Barton Childs, who helped me survive that period. Then I went back and finished my training at Children's Hospital, where I became the Chief Resident.

The experience that turned me toward lead is very clear in my mind. I was working on the infant ward at the Children's, and a child was brought up from the ER with severe acute lead toxicity. I did what I'd been trained to do. I gave her EDTA [chelation therapy]. She was stuporous and very ill. Slowly she got better. It was a gratifying experience, and I felt very smug. I told the mother that she had to move out of that house: "You can not go back to that house because if she has a second episode she's going to be retarded." This was what I'd been trained to do in medical school. She looked at me and said, "Where am I going to move to? All the houses I can afford are the same age." I suddenly realized that the issue was not just making diagnoses and treating them. The issue was in the life story of people. This was a very powerful learning experience.

Then I practiced pediatrics in the suburbs of Philadelphia for a year or two. I practiced with Bill Rashkind, who was a pediatrician and physiologist. Bill developed the Rashkind procedure, which saved the lives of thousands of babies with congenital heart disease. He became a professor of pediatrics full-time at Children's Hospital, and suddenly I was left with a practice. I discovered that a lot of the parents who were coming in to see me, mothers, were coming because they were themselves anxious or depressed. These were suburban housewives, and in those days they didn't

have jobs. A lot of my discussions were around psychological issues, so I took a psychiatric residency. In those days, the government was subsidizing general practitioners and pediatricians to go into psychiatry because they thought we needed more psychiatrists. I was going to be a child psychoanalyst. I went to St. Christopher's to begin a child psychiatry residency and discovered that because I made house calls I knew more about family structure and dynamics than the psychiatry professors. I could go into people's homes and size up the family, whereas in the clinic the mother would bring the child, the child would talk to a psychiatrist, the mother would talk to a social worker, and the father would somehow get evaluated. In a half hour in a home you learn much more than in that whole intake procedure.

I was very unhappy with the training, and the theoretical basis of child psychoanalysis didn't satisfy me. I kept thinking, "How many of these kids who are coming in with learning problems have lead poisoning?" The inner city we served at St. Christopher's had a lot of lead. People thought that was a crazy idea. In my psychiatry residency, I turned to the laboratory. I did some stuff with invertebrates: *planaria* and morphine addiction.

I had another formative experience. I was in the community psychiatry program in North Philly, the inner city. I was the Director of Consultation Education, which was the outreach part of the community health center. I gave a talk at a black church one night to a group of adolescents—mostly boys. At the end of the talk, a kid came up to me and started telling me about his ambitions. He was a very nice kid, but he was obviously brain-damaged. He had trouble with words, with propositions and ideas. I thought, how many of these kids who are coming to the clinic are in fact a missed case of lead poisoning? My office looked out on a school playground. I watched the kids every morning line up and go to school. I said, "I'm going to go into that school and identify the children who have elevated lead and see what their IQs are." Then it occurred to me that the blood lead at 6 years of age might be normal if the exposure occurred at less than 2 years of age. So I began to think: "What can I use to read back in their exposure history?" I went up to Boston to see a guy named Louis Kopito about hair lead levels. But hair lead has serious methodological issues: you can't tell how much is deposited from the outside and how much reflects what's in the blood and brain. Fingernails have the same problem. Fingernails are keratin, high-protein. They have a lot of lead in them, but they also have a lot of external dirt. Lead goes to bone and it's treated like calcium, but you can't do a bone biopsy. Then it occurred to me there's a way to do a spontaneous bone biopsy. It's universal, spontaneous, and painless. You just have to catch a deciduous tooth. There actually had been one paper in the 1960s on high tooth lead levels in kids who had been poisoned.² I collaborated with a dentist in the dental school. [We] collected a lot of teeth from inner-city and suburban kids. The sources were a suburban periodontist and two inner-city dental clinics. The tooth lead levels in the inner city kids were five times what they were in the suburban kids. We got a medical student, Bruce Dobkin, who went to St. Christopher's Hospital and obtained the names of children who had been discharged with lead poisoning. We identified five kids with lead poisoning, and we paid \$5 per tooth. The lowest lead concentration in

the poisoned children was 63 parts per million (ppm), and the highest was something like 120. Twenty percent of the kids in the whole sample in the inner city had levels higher than 63 ppm. The exposure prevalence was very high. We published a very short paper in *Nature*.³

PHR: It seems like this work originated in large part from your clinical experience of doing home visits.

My practice in pediatrics was in Mainline Philadelphia, an upper-middle-class area. I did have experience visiting homes in the inner city, and I did my pediatric residency in the middle of the black ghetto in South Philadelphia—17th and Bainbridge streets. I was single then and would walk back through there after a date. I knew that neighborhood pretty well, and I knew the quality of the homes.

I did another study in Philadelphia. It was with Irving Shapiro, a pediatrician whom I knew, and Ed Sewell, who was the head doctor of the school system. Ed collaborated with us because he wanted to use the school system to do health research in children. We collected teeth from six or seven inner-city schools and three or four schools in Northeast Philadelphia, which had experienced a population explosion and building boom after World War II. Because the city had a contract with the Catholic schools, Ed asked me to include some Catholic schools in the project. This was very important.

The differences in teeth from the inner city and Northeast Philadelphia were so great that I used to play a little game. Irving Shapiro would collect teeth, analyze them, and send me the results. I could guess the kids' race and where they lived. If [the lead level] was over 20 ppm, it was a black kid from the inner city. If it was under 5, it was a white kid from Northeast Philadelphia. It was so clear-cut—the separation was extraordinary. Then a bunch of teeth came in that were very high from kids with Italian or Irish names who all lived on three streets in East Kensington. So Irving and I went to St. Anne's School, which was right next to the National Lead Company. These kids were living in the shadow of the NL stacks. NL had a factory that spanned both sides of a huge street. The children in St. Anne's were getting as much lead from industrial emissions as the inner-city kids who got lead from paint. Of course it caused a little hullabaloo, but the city didn't do a thing about it. I thought this was going to open the doors to lead control.

PHR: How did you publicize your findings?

We talked to the city, and we published in the *New England Journal of Medicine*.⁴ Years later there was a lawsuit on behalf of the people who lived there. A Washington law firm won an award of a million dollars.

PHR: Did the community get involved at the time of your study?

It turns out that the [community] residents knew that there was a lot of lead; they were not surprised by it. I had another experience: I was collecting lead from the gutters in these neighborhoods to see what the levels were. The workmen came out of the [National Lead] factory, and they were very menacing: "What are you doing here?" "I'm from the city," I said, "just collecting some samples." They told me, "Get the hell out of here."

This work got me an invitation to Boston Children's Hospital [and Harvard Medical School] to do this work. In 1976, I finally got a big grant. I collected teeth from school systems in Somerville and Chelsea, Massachusetts. In those days, those were white, industrial, working-class neighborhoods. I collected something like 3,000 teeth from 2,500 children. The teachers were terrific, very cooperative, very well motivated.

PHR: How did it work? Did the teachers ask the children to bring in their teeth?

We had posters placed around the city in store windows. My secretary's husband was a commercial artist. He drew us a tooth that looked like Mickey Mouse. It had a missing tooth, and it said, "I gave." The communities were aware of the campaign. We gave as a reward a little kit—toothbrush, toothpaste—and a badge.

In Philadelphia, [we had given out] solid silver Kennedy half dollars, which were quite rare. The kids, after they gave the tooth, were given this half dollar and a certificate by the dental clinic. I discovered that some of the dentists were giving the kids two quarters and keeping the half dollars. I spoke at a community meeting and I said, "How'd you like that Kennedy half dollar?" And [the kids] said, "What do you mean? I got two quarters." This was my first experience with the corrupting power of cash in science.

[In the Somerville and Chelsea study], instead of cash we gave a toothbrush and a badge. We got some funny things from the kids. We got some dogs' teeth and some adult molars that they'd found in the house. Even some white stones. The toothpaste and badge were a powerful motivator. The teachers were wonderful. They each had a shoe box filled with coded envelopes. On the envelope was a diagram of the human mouth with a big smile. They would look in the kids' mouth, find the space, and then mark the space on the envelope. When my chemist would open the envelope, he would look at the type tooth and the space and see if they were consistent. At the peak, we were able to do, I think, 60 teeth per week. We had no idea what a normal tooth lead level was or what the range was. We had to develop a rolling standard. As we did 100 teeth, we'd see what the mean was and then establish the upper 90th percentile and the lowest 10th percentile. But then we would do another 100, and the mean would move around a little bit. This is important because this is one of the issues raised during the investigation of scientific misconduct. I had to make up the rules myself as to which children were classifiable and who were not. Initially I said that if a child gave four samples, then three out of four had to be consistent. Otherwise we'd say "unclassified" and exclude the child. But that excluded too many children. We made it two out of three. That was raised in the investigation, and I was a little hazy on it; I couldn't remember. At any rate, we identified, I think, 270 kids who were at the highest or lowest end of the distribution and brought them into Boston Children's Hospital. I would interview the mother, give her an IQ test and a medical questionnaire. The kid had a four-hour examination by well trained psychometricians, and then we'd get the data crunched.

We'd pay to have all of the teachers dismissed for half a day and hire substitute teachers. All they had to do was fill

out a questionnaire for every child in the class, whether they were in the study or not. The questions were very simple: Is the child distractible? Yes or No. Disorganized? Yes or No. Follow simple directions, complex directions, etc. There were 11 questions like that. We had 2,146 good datasets, that is, a good tooth analysis and a good questionnaire. Then we arranged the subjects in six groups of 42 ascending tooth levels. Class 1 was the lowest, Class 2, up to 6. We just counted the negative reports by teachers for each of the six groups. As tooth lead went up, the rate of bad reports went up, too. It was extraordinary. The teachers, who didn't know [the kids'] lead levels, could identify all these non-adaptive behaviors [that were] in direct relationship to the level of lead in the teeth. That convinced me that I was right. The evidence came out of the computer; there it was. So we published it with the IQ and language data in 1979 in the *New England Journal*.¹

I had a very good organic chemist, Neil Maher, who was doing the tooth analysis. In 1976, I got a call from David Schoenbrod, a lawyer at the Natural Resources Defense Council. He had sued EPA to write an air lead standard. EPA had drafted the first version, and he asked me to take a look at it. It was such a bad piece of work that it was clear to me that it was an industry pass-through. Probably an industry scientist had written it and given it to EPA and they had incorporated it. Neil and I wrote a rather strong report, and we both went down to Crystal City, Virginia, as part of the Clean Air Science Advisory Committee's (CASAC's) review of this document. The chairman of the CASAC was Roger McClellan. He later was the head of the Chemical Industry Institute of Toxicology. A very nice fellow, but very pro-industry. Most of the people on the CASAC were pro-industry except Sam Epstein from Boston Children's, Ruth Diamond, who was the Dean of Boston University School of Public Health, and Bailus Walker, who is now at Howard University College of Medicine. After a very strenuous debate and at the conclusion of two days, the CASAC totally rejected the document and decided not to revise it—that is they decided to get rid of it, start again, and get some new people involved.

They got a new coordinator for CASAC to produce the EPA criteria document. They wrote a thicker one; it was better, but it still wasn't good enough, and CASAC again said this needs to be tightened up and we need new consultants. I was appointed, and then Sergio Piomelli from Columbia, who was a pediatrician, was appointed as well. We went down to North Carolina. It was the year of the big brownout in New York City, 1977. We went down just after that. We spent three or four days in North Carolina, and it was terribly hot. They had also appointed two pro-industry consultants—Emmett Jacobs, who was the vice president for petroleum affairs at Dupont, and a young guy named Ed McCabe.

PHR: What did the pro-industry people say?

They really weren't on firm ground. They didn't have the background. McCabe was appointed because he had participated in one epidemiologic study that had measured blood leads across the country.⁵ He was not the senior author. He didn't design the study. He became a consultant to the industry—wrote letters to the editor and that kind of stuff. Jacobs was a smart cookie, but he was no pediatrician or biologist. I said to him, "You have these PhDs, these smart

chemical engineers, why don't you develop a better anti-knock agent [one without lead]?" And he said, "Well, Herb, to tell you the truth, our economists are looking at the gasoline market. It's beginning to flatten out. There's not going to be the same kind of demand. And we're not going to put 100 million dollars into R and D." This is what he said. This was my post-graduate education. That all this b—s—in the criteria document [about the lack of any danger from lead in gasoline] didn't mean anything. Dupont's scientific position was determined by the company's economists.

I had worked at Dupont when I was in medical school. Between my first and second years as a medical student, I worked as a laborer at Dupont's Deepwater plant, where they made tetraethyl lead. I didn't know anything about it at that time. I shoveled chemicals: backbreaking, awkward, dangerous work. We carried cigarettes in a plastic case because if you didn't you would dissolve the tobacco—you sweated so much. It was so hot there, I would walk out into a summer evening and it would feel like I was walking into air-conditioning. We weren't allowed to carry matches. We were allowed to smoke at 10, lunch time, and 2. The smoking whistle would blow and all these guys would pour out of the different buildings. They had a wooden shack where they would have two cigar lighters and a Coke machine. Everyone would smoke two cigarettes back-to-back and drink Coke, then go back to work. There was a group of workers who always sat in one place in the corner. They didn't talk to anyone. They just stared out in to space. They were obviously out of contact. So I said to some of the old guys, "What's going on?" and one said, "Oh, they're from the House of Butterflies [where tetraethyl lead was fabricated]." I knew nothing about the House of Butterflies; I just knew these guys were brain-damaged.

When I left that job (after two months), the sector head—he was a PhD, I guess a chemical engineer—asked me to come up and talk to him. He asked me, "What do you do think about this job?" I said, "I don't think any human should do this work. I mean it's hot, dangerous, and nasty." I said, "I don't think any animal should do this work. Nobody with a nervous system should be exposed to this kind of work." He was kind of shocked. It was just horrible, but it put some money in my pocket for school.

I worked in what was called the sulfonating house. We were always moving. You would have to wear a hat and goggles and gloves all the time. Hard toe shoes. You would go in to work and change your clothes and in about 30 minutes you were soaked. Absolutely drenched with sweat. At the end of the day you'd shower and go home. I couldn't eat when I had that job. I'd drink a quart of milk at lunch and some crackers. I would go home and drink and drink and drink. My thirst would be enormous. I would lose like 13 pounds a day and then gain it back. As I said, I don't think anything with a nervous system should be doing that work.

PHR: Were there any issues with the industry other than at government meetings?

In 1979, when I published that paper [on the Somerville and Chelsea study],¹ the lead industry was silent. They didn't say anything for about six months. I expected that there would be a big response, but there was nothing. Then Jerome Cole of the International Lead Zinc Organization called and

wrote a letter to the editor—the usual stuff. Then they started to call for my data, my printouts, and I said, “No. I’ll share them with any legitimate scientist, but I’m not going to share them with the lead industry because they don’t qualify.”

PHR: How did they request your data?

In public. Then in the writing of the final EPA criteria document, I testified and was questioned about my work. Claire Ernhart testified, presented, and was questioned about her work. It was a very strange thing. Lester Grant had been at the University of North Carolina, but then he went over to EPA. He asked me to criticize her work and her to criticize my stuff. I thought that was kind of strange to set up this kind of duel. So I presented my stuff, and Ernhart raised questions about uncontrolled variates, etc.

PHR: Can you explain?

Claire Ernhart is a psychologist who published in 1974 what was the best paper at that time in the *Journal of Learning Disabilities*.⁶ She and Joseph Perino examined the IQ scores in children on Long Island whose blood leads were over 40 or under 30. It was a more sophisticated analysis than anything that had been done before because she used multiple regression analysis and included a number of variables, including maternal IQ. There was a significant effect; the high lead subjects had significantly lower IQ scores than the low lead subjects. She said that while this may not be visible in the clinic, it has important effects on IQ, and public health authorities should pay attention to it. Then, in 1981, at the American Association for the Advancement of Science in Toronto, Ellen Silbergeld, Debbie Rice, and I were part of a symposium on lead toxicity. Ernhart got up in the audience and said she was publishing a paper [showing] that if there is any effect it is “minimal.” Jerome Cole was on the panel; he was the head of the International Lead Zinc Research Organization. Six months later she had a grant from ILZRO and became their principal spokesperson.

In her later paper, Ernhart presented her data in an idiosyncratic way.⁷ She did not present *r* and *p* values, which is the customary way. She gave some other metric to it that can be transformed, which I did and there was a significant effect. It just was blurred out by her.

When Ernhart criticized my work in the EPA hearings, she said something about inadequate control of confounders. When I criticized her, I said, “You didn’t even control for socioeconomic status, which is traditional.” She said, “Well, that’s because all of my subjects were of the same socioeconomic status.” I said, “Well, I’ve read your paper and apparently I know your paper better than you. I have a copy of it here, and it says the parents of these subjects were teachers, postal workers, and welfare mothers.” This was kind of dramatic.

There was one incident that was most revealing about Ernhart. It involved a lawsuit about a kid from Cleveland. I was asked by a lawyer for the plaintiff if I would be an expert witness for the case. I read the case and said absolutely. I thought this was an open-and-shut case. This was a child named Danita R. She was described as singing nursery rhymes, dancing, being a very bright kid. Then she got sick. She was taken to Rainbow Children’s Hospital with a fever

and sore throat. She was stuporous. A neurosurgeon looked at her and thought she had a brain tumor because she had signs of increased intracranial pressure. They took her to the OR as an emergency. On the way up, they drew her blood for lead. In the OR, they opened her head, and they saw severe swelling of the brain and some dead cerebellar tissue, which they excised. They closed her up, and she had a very stormy post-op period. Then they inserted a shunt to decrease the intracranial pressure. After recovery, she had hyperactivity, attention deficit disorder, and a low IQ. Her blood lead [level] returned while she was in the OR and was as high as they could measure. It was over 100. That was the ceiling of their measurement. So here was a case of a kid with an extraordinarily high blood lead and evidence of dead brain tissue. I said, “Sure, I’ll testify.” Claire Ernhart testified for the defense.

PHR: When was this trial?

Mid-1980s. Then in 1991 I was approached by a guy named Benjamin Fisherow, a senior attorney at the Department of Justice. He asked if I would be the principal medical witness in a suit against the owners of a mill in Midvale, Utah. The owners were Sharon Steel, a local Pittsburgh steel company; Gulf Resources; and a third group. The suit was not for damages to people but to get the owners to pay to clean up the place. They had mined and smelted the lead there and left a mountain of tailings. Houses were built on them. It was felt to be a hazardous waste dump. Fisherow prepared a very good case. Lots of good witnesses on the environmental side. I was deposed here in town at Jones Day. Twenty lawyers in a room where the conference table is like a bowling alley. Claire Ernhart was there for my deposition.

PHR: She was in the room?

In the room. She was seated and taking notes. A few months later, a lawyer from Philadelphia sends me a copy of a submission to the National Institutes of Health accusing me of scientific misconduct.

Sandra Scarr, who had worked as a consultant to EPA during the drafting of the [1986] criteria document, had been a member of a special ad hoc committee sent by Lester Grant to interview me and Claire Ernhart. The committee wrote a report which says you can not make any conclusions from the data of Needleman or Ernhart. The report contained 11 factual errors. The deal was that they could come and I’d give them the data and they could ask any questions they wanted but I would have a chance to see this and comment on it before it was published. It was sent to me the day of publication. I wired Grant that if he didn’t correct all these errors, I would make him send an errata sheet to the entire distribution.

PHR: This was being published where?

Distributed by EPA as an addendum to the 1983 criteria document. So Lester corrected all of those things because they were factual, but he didn’t change the conclusion; it was still left a little vague. However, then the CASAC met in North Carolina and I was asked to come down and comment. Ernhart and Scarr were there. I got up and said the report was erroneous and here are the facts. In the mean-

time, the EPA had given us some money to reexamine the data and sent two EPA staffers to help with it: Joel Schwartz and Hugh Pitcher. They analyzed the data and got the same results. They recorded that the conclusions I drew and published were accurate. The final version in that 1986 criteria document says this is pioneering work and it does support the conclusion that low levels of lead affect children's IQ, etc. It also said that Ernhart's data support that, too; they looked at it and found the same thing I did.

[In 1991], I got a brief that accused me of scientific misconduct. It was submitted by a guy named David Geneson. He is an attorney with Hunton and Williams. Hunton and Williams is interlocked with Ethyl Corporation of America through its board of trustees. So he was the person who sent the charges down to NIH. The next thing I know, I was called by a reporter from *Science* magazine. I said, "Come on, this is just the industry trying to get me." I didn't realize how serious it was. The university called me and said, this is nothing to worry about. It will pass. The next thing I know they're going to have an inquiry.

The NIH referred the investigation to the university. That's their procedure. My files were locked, and I could only look at my data in the presence of a representative of the Office of Scientific Integrity of the university. I had to call her up and say I wanted to look at some data: can you come and unlock the files? They put bars on my file cabinets. The inquiry committee was composed of three people from the University of Pittsburgh: two epidemiologists and a statistician. They looked at my data tapes and regressions and got the same results. They reported that they found no evidence of scientific misconduct but they could not rule out scientific misconduct. But the university said there was enough reason to go ahead with an investigation, which is the second phase of a scientific misconduct inquiry. It's like the grand jury deciding whether there is a reason to go forward, and what the university found was that there was no misconduct but they should go forward anyway.

PHR: Do you have any sense on who was pressing them on this?

Yes, I do. I think it was the guy who recruited me to the university. I think I displeased him because there was certain research he wanted me to do that I said, "No, that's not my bag and I won't do it." A lot of this is surmise, but I told him no and think I made a serious enemy. Also, Sharon Steel is a local firm, and I had cost them \$20 million in the environmental lead lawsuit that I had testified in. There are local industry connections to the university. So I think those two things together are adequate to explain why this thing was pulled off.

PHR: What was it like for you during this period?

Horrible. It was absolutely horrible. I was so angry, and it's not good to be that angry and worried; it's bad for your health. I was mostly furious because I thought, they're not going to find anything because there isn't anything to find. What I discovered is that not only did the university not come to defend me but they wouldn't give me an even playing field. I went to the dean, and I said, "OK, I want the investigation to be public. I want to have scientists, the press,

and my colleagues here at the university monitor this. The university guidelines for scientific misconduct state that that the university can bring in outside experts. I want you to bring the top people in lead toxicology and neurotoxicology and put them on the panel." The dean refused my request to open it up and bring in appropriate experts who knew the field. He said, "We don't need them. We have our own experts." This is hard to believe, but one of their experts was Robert McCall, a psychologist who had worked on American Psychological Association panels with Sandra Scarr. I brought this up. I said this guy has a conflict. He knows her and has been working with her. Another was Herbert Rosencranz, a toxicologist who had been head of environmental health at Case Western Reserve, where Claire Ernhart was. So I said he should not be on this panel either. They responded, "We know about that, and there is no conflict of interest."

PHR: Did you have a group who you were supported by? Other professors and medical people?

Well, it is a very clarifying moment when this happens. You learn who your friends are. My friends were not people in the medical school, but the faculty in the university at large, in the liberal arts and sciences, etc. They really stood behind me. The major issue was having an open hearing. I knew that if we went in to executive session, I was through—I mean, just judging by the report that the inquiry committee wrote. I campaigned to get it open, and the university faculty senate was behind me 100%. It became a big issue here. The chancellor was challenged in public. About 400 scientists from around the country petitioned. The hearings were then declared open, at which point Sandra Scarr and Claire Ernhart said they would not come. They did not want to be questioned in public.

All we knew, my lawyer and I, was that there were meetings between the investigation committee and the administration, the science integrity officer, and Scarr and Ernhart. They were having discussions and they finally persuaded them to come, because if they didn't they would have had to drop the whole thing. If I couldn't confront my accusers, then there would be no case. The deal was that they would come but they could refuse to answer any questions that they didn't want to answer. So how do you confront somebody when they can say, I'm not going to answer that? I had a lawyer, a very good lawyer, but he was not allowed to speak. He could only sit there and whisper in my ear.

PHR: How long was the hearing?

A day and half. It should have been longer. Really, we should have just pursued them. We should have said, you have to answer that question. They accused me of not controlling for age in my study. But the IQs are normed for age. So I asked, did you control for age in your paper such-and-such? They answered, that's not relevant. So that was the kind of thing that went on. The main issue was that they said I'd chosen my subjects knowing who had high lead and low IQ. So when I got my printouts out and read through them again, I saw on the front page of every data run a piece of computer code in SPSS which said, select the subjects if the lead is high or low. That was in the computer code. I asked Scarr, "Did you see this code?" She said, "I don't know." I

said, “Do you know that it’s at the front page of every sub-routine of the data you examined?” She wouldn’t answer.

It went on for a day and a half. The press was very favorable and kind. It took a long time for the committee to turn it around. They said there was no evidence of scientific misconduct in terms of false application or plagiarism; however, the way I reported my control group was misrepresented. That was important because I had brought this up to them at the beginning. I said there is an error in reporting the range of tooth lead levels in my control group. There were a couple of things. One was where I changed in the middle from three out of four teeth to two out of three. That was unclear, but it was not dishonest. It had no impact [on the results]. As I told you, I was doing it in a kind of rolling sequence of admitted subjects. I said, yeah, I was uncertain about that. It was the first time that this had ever been done, and we were only doing 60 teeth a week, so the values changed with time. But the industry trumpeted that I had deliberately misrepresented the data.

PHR: So you are at the university and some of your colleagues have abandoned you—what’s happening?

At that point, I spent most of my time with my staff. They were very helpful to me. The PhD research coordinator helped me a great deal. Two younger people did a lot of research and brought stuff together; they prepared it for me for my hearing. I had asked for help from the Tenure and Academic Freedom Committee (TAFC). The chair, Richard Tobias, who was a former president of the faculty senate, a professor of English, was a great support, and the TAFC was supportive of me. The faculty senate really backed me up completely. I felt I had friends. The Dean of the School of Public Health at that time, Don Mattison, was a good friend of mine. I had known him for a long time. His interests and mine were similar. After this was all over, he called me up because he had a research project he wanted me to participate in. Months had gone by of absolute silence, and now he took me out to lunch and we talked. I said, “Hey, Don, how come you never spoke to me when I was in the middle of all that melodrama?” He said, “Well, my wife thought I should, but I guess I was afraid.” At least that was honest.

PHR: So, in your relationships now with the faculty, have there been lingering issues?

No. Because of this experience, because they were so helpful to me, I ran for a position on the TAFC. I served for many years. I was chairman for four years.

I’m going to tell you another story. There was a guy named Erdem Cantekin who was a whistle-blower—a biomedical engineer who was the science director for an ear, nose, and throat research project. A huge grant. Millions of dollars to study the antibody treatment of otitis media, a common infectious disease in childhood. Halfway through the study, the [researchers] stopped the data collection and did an analysis and found a marginal improvement for their drug over the control group. Very small difference. They wanted to submit it to the *New England Journal*, but Cantekin wouldn’t sign off on it. He said, “First of all, we broke the code. We said we were going to do 1,000 subjects but we did 500,” and a lot of other things. Suddenly Cantekin became

persona non grata. He had tenure; they couldn’t fire him, but they dismissed him from the head of this project. By the way, it turned out the principal investigator was taking money from Glaxo at the same time he was accepting federal support and not reporting it. He was found guilty of scientific misconduct, but he survived. Erdem was sent to an office over what used to be the Giant Eagle grocery market with a filing cabinet and a phone. He sued and won a big, big settlement. No one would talk to Erdem. I used to go to lunch with him once a week in the cafeteria. Anyhow, I joined the TAFC and I became the chairman, and I’ve been involved in those kinds of arbitration since.

PHR: So what happened in 1991, after the investigation?

The investigation committee found no misconduct. They just let [the inquiry committee’s findings] stick. I continued to get grants after that. I was allowed to apply for grants because only if you’re found guilty of scientific misconduct do they say you are barred from research, but that never happened.

PHR: You were a hero outside of the university.

The reason I told you the Erdem Cantekin story is because it contains a diagnostic episode. When the principal investigator was found guilty of scientific misconduct, the medical director of Children’s Hospital wrote a public letter to the editor in his defense. I wrote a letter subsequent to that in which I said that the real hero was Erdem Cantekin, who was punished and should have been applauded for his courage. Also that the university had to be very careful about doing a minuet with the drug companies. I wanted Erdem to know that he wasn’t totally alone. I got an anonymous letter from a faculty member thanking me for that editorial. That tells you what the climate was. He didn’t even sign his name.

PHR: It raises the question of what effect you think the assault on you had. Was it meant to scare younger scholars away from doing controversial research?

I wrote about that in a piece in *Pediatrics*.⁸ If this is what happens to me, what is going to happen to somebody who doesn’t have tenure? I’m worried that people who are trying to get a niche and don’t have tenure are asked—and I’ve seen it as a member of the TAFC—to do things that they question the ethics of. They are intimidated. It’s a real force.

PHR: What were the repercussions after 1991? Were you able to continue your work?

I think, all in all, that throwing light on [my experience] was healthy for the medical community—to see the way that certain people operate. So I think that was good.

PHR: Has there been any effort to apologize to you?

You know, it says in the faculty handbook that if someone is found not guilty of scientific misconduct, the university will make a public statement. But they never did. It got lost in a committee. Subsequent to that, however, I did win the Chancellor’s Award for Community Service—\$2,000 and a handshake.

PHR: Do you think we are ever going to find a threshold below which lead has no effect on children?

Most of the damage is done at very low levels, which is what we showed in our study in 1987.⁹ It is what Joel Schwartz has shown in his meta-analysis.¹⁰ It's a very intriguing physiological problem. Why is it that the toxic effect of lead is stronger at lower doses? I have a couple of ideas. I think there is an early mechanism that is important and powerful that can be saturated by only a little bit of lead; you do that damage and then you need more lead to get the other targets activated. I think that's what some smart molecular biologist will be able to show. As a matter of fact, Jay Schneider has shown that lead at picogram concentrations influences the length of branches of nerves in tissue cultures.¹¹ I think that at very small doses, these things happen because you don't need much. Then the next damage occurs on a different mechanism at a different level. All along there are different mechanisms that come into play that end in the neurophysiologic deficit.

I don't think there is a threshold. Barry Commoner, who made me see this, says that we've had a billion years to adapt to natural molecules. We've had a couple thousand years to adapt to lead. Fifty years to adapt to pesticides. All of these are toxic at some level. We have developed no adaptive biological mechanism for lead, which has no purpose at all in the body. Nobody has ever been able to discover an enzyme that is activated or influenced by lead. There is no biological function, so any amount is going to be deleterious.

We are now able to look at effects at lower doses for a couple of reasons. One is that we have better statistics, analytic methods, especially since the removal of lead from gasoline has now given us comparison groups with blood leads of 1 or below. We never had that before. When I did my study in the 1970s, my control group had a mean blood lead of 15. Now we have a large number of people walking around with blood leads of 1 or below.

PHR: So you're still working away. You're now 75 years old. You certainly started a school.

I didn't start it. There were maybe six or seven papers before mine. Phil Landrigan had a nice paper in the 70s.¹² Claire Ernhart's paper was a nice piece of work for the time.⁶ A woman in Virginia, Bridgette de la Burde, a pediatrician, looked at some kids with high lead levels.¹³ But above all, there was Randolph Byers in 1943 and after, who said he wondered how many of the kids walking around with school or behavior problems were lead-poisoned. That was really where it began. What I did was develop a tooth assay, which was very useful. I had a very good epidemiologist in Boston, Alan Leviton, who helped me develop a rigorous study. It answered the questions that were around at that time.

PHR: Does that explain in some sense why you became such a focus for the industry?

Yes! Sure. It's very clear to me that in 1990 there were now 30 papers from around the world all saying the same thing—

except for Claire Ernhart. The [lead industry] couldn't contest that, so what were they going to do? If they could discredit my work, the whole thing would collapse or be fundamentally revised. I'm sure that was it. That's why they kept saying they had to have my original data because they had planned to make a concerted attack on [my findings]. Then all the other work that grew out of it would be . . .

PHR: Suspect? Discredited.

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Editor's note: For a selected bibliography of related literature, see http://www.publichealthreports.org/userfiles/120_3/Appendix_to_Needleman.pdf