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AIRBORNE LEAD REDUCTION ACT OF 1984

HEARING

BEFORE THE

COMMITTEE ON ENVIRONMENT AND PUBLIC WORKS UNITED STATES SENATE

NINETY-EIGHTH CONGRESS

SECOND SESSION

ON

S. 2609

A BILL TO AMEND THE CLEAN AIR ACT WITH REGARD TO MOBILE SOURCE EMISSION CONTROL

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levels are explained by the seasonal fluctuation in lead gasoline usage. Seasonal fluctuations in blood lead levels have been noted previously, but this is the first time that they have ever been adequately explained.

So the gasoline lead-blood lead relationship that explained the overall 4-year decline in blood lead levels also explained the increase in blood lead levels that occured from the winter to the summer months.

For children 6-month mean blood lead levels were highly correlated with 6-month mean gasoline lead levels. This high correlation did not appreciably change even after accounting for the effects of virtually every conceivable variable that could influence these results. The probability that chance could account for this relationship between blood lead levels and gasoline lead is less than one in 10,000.

The NHANES II blood lead level results also predicted the average length of time that a change in blood lead levels would lag behind the gasoline lead exposure by approximately 1 month. A similar value was determined independently by isotopic measurements.

In addition, the New York City and Chicago lead screening programs have measured the blood lead levels of a large number of children. Analysis of the blood lead levels of New York and Chicago children from 1970 to 1976 showed a strong association between gasoline lead and blood lead. Just as with the NHANES II data, the decline in blood lead levels are explained by gasoline lead usage and also explained the short-term seasonal fluctuations in blood lead.

Another study conducted by Dr. Needleman's group in Boston of umbilical cord blood lead levels between April 1979 and April 1981 showed a downward trend and the seasonal pattern of blood lead levels were found. Average monthly mean blood lead levels were strongly associated with the local monthly gasoline usage.

The implication of gasoline lead as a significant contributor to the umbilical cord blood lead levels is noteworthy, since the yet-unborn child is probably the most vulnerable to the neurotoxic effects of lead.

The NHANES II study, the New York City, the Chicago, and the Boston blood lead data are four large independent data sets, each demonstrating the same strong relationship between gasoline lead and children's blood lead levels. In each of these studies, gasoline lead usage not only explained long-term decreases in blood lead levels but simultaneously accounted for the short-term increases in blood lead levels that occur from winter to summer months.

The excellent agreement of these four independent studies strongly supports a causal relationship between changes in gasoline lead use and changes in blood lead.

In an effort to quantify the amount of lead in human blood which comes from gasoline, a large-scale field study was undertaken in Italy in 1974. The gasoline of an entire region in northern Italy was 90 percent replaced with gasoline which contained lead from a foreign source that could be isotopically traced. Gasoline lead was found to contribute at least 5 to 6 micrograms per deciliter to the blood lead levels.

Estimates from the NHANES II are that gasoline lead contributed approximately 8 micrograms per deciliter to the blood lead levels, which is in good agreement with the Italian experiment.

For a typical child in the United States, current gasoline lead exposure accounts for 25 to 50 percent of the total blood lead level. In pockets of excessive lead exposure from gasoline, this contribution is likely to be higher.

Calculations from CDC's lead screening program indicate that if no lead had been allowed in gasoline since 1977, there would have been approximately 80 percent fewer children identified with lead toxicity. That is, 80 percent of the children found to have lead toxicity required the contribution of both gasoline lead and nongasoline lead sources to reach the toxic effects.

Compounding the gasoline lead exposure problem is that the gasoline lead that is dispersed into the environment accumulates. General lead accumulation in our environment from all sources has already amassed to such a degree that the typical adult body burdens of lead in the United States are about 500 times higher than those persons who lived in our preindustrial era. Even though the amount of lead in gasoline has been reduced, as of the beginning of 1984, lead is still being dispersed into the United States environment at a rate of 53,000 tons per year, or 6.1 tons per hour, an unnecessary contamination of our environment by a very highly toxic material that has been demonstrated to have adverse effects on humans.

In summary, in spite of some other views which you may hear this morning—and I predict you will hear other views—evidence is overwhelming that the gasoline lead is a major controllable source of lead exposure. Eliminating lead in gasoline would not only immediately decrease the blood lead levels, but would also decrease the background lead levels for future generations.

Thank you very much.

Senator STAFFORD. Thank you very much, Doctor.

And now we would be pleased to hear from Dr. Needleman.

STATEMENT OF DR. HERBERT L. NEEDLEMAN, DIRECTOR, BEHAVIORAL SCIENCES DIVISION, CHILDREN'S HOSPITAL OF PITTSBURGH

Dr. NEEDLEMAN. Good morning, Mr. Chairman. I am Herbert L. Needleman. I am chief of psychiatry at the Children's Hospital of Pittsburgh. I am also a pediatrician, and I believe that I may be the only witness here today who has actually treated lead poisoning. So I speak about my research and the research of others from the vantage of somebody who has had to deal with the acute consequences of lead intoxication and the remedial education of children who are brain-damaged as a result of their exposure.

I want to congratulate this committee and Senator Durenberger for taking the leadership in what I think is one of the most important public health issues of the decade for American children.

I will not read my testimony because of time. I gather my assignment is to review in capsule form the health effects of lead at low dose. I will concentrate on the work of my group, but I don't want to convey that the burden of the evidence resides just with the

studies we have done. I simply know them the best. There is a convergence of inference on the basis of most health scientists that lead at low dose is toxic. The great exception to that covergence comes from the lead industry and its surrogates in the academy who are sponsored by them.

Now let me turn to the issue of epidemiologic studies of lead at low dose. I treated a lot of lead poisonings as a resident in pediatrics in Philadelphia, and when I went into community psychiatry in the late 1960's, I spoke to principals, school teachers in the inner city of Philadelphia and found that the most vexing problems for them were children who were behavior-disordered, learning-disabled, or mentally retarded. And parents reported the same concerns.

It seemed to me that since this was the same area in which lead exposure was endemic, perhaps some of this was related to a preventable exposure to a poison. Forty years ago, my mentor, Dr. Randolph Byers, a neurologist for the Boston Children's Hospital, had followed up 20 children who had recovered from lead poisoning, and found that 19 out of 20 had severe behavior disorders or learning disabilities.

In 1943 Dr. Byers asked, "How many children in the city who are learning-disabled are that way because of exposure to lead?" That was a prophetic question that we have been struggling to answer ever since.

In order to look back in the history of exposure to children with lead, because blood lead is the short-term storage system, I developed a new method, lead storage in deciduous teeth. We collect a lot of baby teeth. It turned out that the tooth was in fact a very good marker for past exposure.

I went to Boston then to see if this classifier would enable us to see if differential exposure made any difference in children's functions. I will tell you quickly about that study.

We collected teeth from about 2,000 children in Summerville and Chelsea, MA, classified them according to the amount of lead in their teeth, brought in children with the highest and the lowest lead into the Boston Children's Hospital, examined a large number of non-lead covariates which could affect development: socioeconomic status, mother's IQ, et cetera. Controlling for those, we evaluated children's psychologic performance under blind conditions. My research assistants did not know the children's lead levels.

And when we then corrected for all the covariates which did differ between exposure, we found that children with high lead in their teeth, all of whom were diagnosed by society as normal enough to attend ordinary first and second grade, had a mean IQ difference 4 points below those with low lead in their teeth. None of these children had diagnoses of lead poisoning.

Now, the lead industry has said, "Well, 4 points is not a very big difference." But if you plot the actual distribution of differences, a 4-point shift in the mean predicts a fourfold increase in the percentage of children with severe deficits.

On the handout there is a graph that depicts this that was published in the New England Journal of Medicine.

We see that moving the mean a small amount down quadruples the amount of children with severe deficits.

In addition, we asked teachers, who also did not know the children's lead levels, to evaluate the kids on 11 yes or no items: Is the child distractable, yes or no; is the child able to persist at his work; is the child hyperactive, et cetera.

When we plotted dentine lead levels against the proportion of bad scores on each one of those items, there was a regular smooth increase in bad scores, step by step, with the increase in lead levels.

So something was going on in the classroom which affected behavior, and was recognized by the teachers, which was directly correlated with dentine lead levels.

This study received a lot of attention, and a great deal of skepticism. Bill Yule, who is a psychologist in London, was funded by the Medical Research Council to disprove this. He admits that. He found the same thing in English school children, using the same scale. This finding doesn't depend purely on my own work.

Now, this is an issue which has gathered a great deal of controversy. The bulk of the conclusions from pediatricians and public health authorities is that lead at doses below those which bring children to the hospital is associated with impaired mental function.

The lead industry and certain of their academic consultants have raised canons for proof in epideniologic studies of lead which are impossible to fulfill in real-world investigations. In this regard, their behavior resembles that of the tobacco industry, which still maintains in the face of overwhelming evidence that the health case against smoking is unproved. The parallels are extraordinary.

Now, school-age children may not be the most sensitive target group. Lead crosses the placenta. A good friend of mine, Dr. Jack Scanlon, in 1972 measured umbilical cord blood lead—which is infant's blood, by the way—and showed in a small Boston sample that suburban children, infants of suburban mothers, had lower umbilical cord blood lead levels than urban mothers.

We collected, as Dr. Houk and Dr. Goldstein indicated, 12,000 umbilical cord blood leads from BH Hospital women in over 2 years. We measured the monthly mean umbilical cord blood lead and from proprietary sources got the total sales of alkyl gasoline in Massachusetts. The correlation between the decrease over those 2 years month-by-month was 0.75. A perfect correlation would be 0.1; no correlation would be zero. The coefficient of determination for amount of lead in umbilical cord blood is 50 percent. That study which was published in Lancet in 1983.

We then took 5,000 mother-infant pairs, and on the first day after birth we asked the mother a large series of questions about past medical history, alcohol, tobacco use, et cetera, and looked at a number of birth outcomes. In last week's Journal of the American Medical Association, we published an article which showed that the incidence of minor malformations, none of which are compromising to the baby's health, increases in dose-dependent fashion in relation to the amount of lead in the umbilical-cord blood, controlling for all the other covariates in the model which could affect malformation.

The mean umbilical cord blood lead in that sample was 6.7 micrograms per deciliter. As you go from the lowest you can measure

to the median, the risk for minor malformations almost doubles. And as you go to the 98.8 percentile, it almost triples.

So lead may be involved, as it has been demonstrated in animals, in affecting the developmental program.

We have also followed forward from that group of individuals 250 children and seen them at every 6-month intervals, and we're measuring their development. And in an article which has been accepted for publication, we demonstrate, controlling for 120 nonlead covariates, that umbilical-cord-blood lead predicts infant IQ as measured by Bailey scales at 6 and 12 months of age.

So it may be that a determinant of infant outcome at 6 and 12 months of age is established by lead exposures while the fetus is in utero. That corresponds very nicely with animal studies conducted over the last 15 years.

In summary, I think a prudent and neutral evaluator of the body of data, not just my own group's, could not escape the conclusion that low-dose effects of lead affect children's brains and forward studies of lead show an effect on the development and the incidence of minor malformation.

I think the larger mystery here is why our society has been so slow to recognize this threat—it's been 40 years since Dr. Byers' paper in the American Journal of Disease in Children—and why it's so slow to deal with it effectively and promptly.

I congratulate you and the committee, sir, for seizing the leadership on this vital public health problem and would be prepared to answer your questions.

Senator STAFFORD. Thank you, Doctor.

Now Dr. Cole.

STATEMENT OF DR. JEROME F. COLE, PRESIDENT, INTERNATIONAL LEAD ZINC RESEARCH ORGANIZATION, INC., ACCOMPANIED BY DR. ROBERT BORNSCHEIN, ASSOCIATE PROFESSOR, UNIVERSITY OF CINCINNATI

Dr. COLE. Thank you, Senator. My name is Jerome F. Cole. I am president of the International Lead Zinc Research Organization, and I am appearing here today at the request of the Lead Industries Association, which is the trade association for the lead industry in the United States. With me is a consultant to ILZRO, Dr. Robert Bornschein. Dr. Bornschein is a researcher, an expert on studies pertaining to neurobehavioral effects of lead. He is an associate professor at the University of Cincinnati and is very familiar with the scientific studies pertaining to the neurobehavioral effects that Dr. Needleman was referring to. He is here to answer any questions that you may have. He is also prepared to answer any questions regarding the current ongoing prospective studies which were the subject of the EPA-sponsored meeting in Cincinnati that was referred to earlier by Dr. Goldstein, and also of course his own work, which is a very large study being sponsored by the U.S. National Institutes of Environmental Health Sciences.

The research that ILZRO, our organization, and others have conducted indicate that lead that has been used in paint in the past and is still present in old deteriorating housing is far and away the primary cause of elevated blood lead levels in children.

Frankly, we think that over the past decade the Government has been shooting at the wrong target when they have been so active in attempting to get the lead out of gasoline. Lead has been used in gasoline for over 60 years. Over that time, despite extensive research efforts on the part of industry, and academia, and the Government, there is simply no evidence that anyone in the general public has ever been harmed by this usage.

We are aware, of course, of the claims and the arguments of those who would further restrict or ban the use of lead in gasoline. These generally include an inflated estimate of the contribution of leaded gasoline to lead absorption as measured by the lead content in blood, and further, a onesided, and I think an alarmist, view of the medical and health evidence pertaining to the impact of low-level-lead exposure.

Over the years there have been a number of studies that have attempted to estimate the contribution of leaded gasoline to lead in blood. Virtually none of these studies have been definitive. However, a picture has emerged which is fairly consistent; and that is that the contribution of leaded gasoline to lead in blood is relatively small.

The most recent thorough review of this issue was carried out by Dr. Peter Elwood of the U.K. Medical Research Council epidemiology unit in Cardiff, Wales. And I have attached a copy of Dr. Elwood's review to my statement, and I would hope that it would be included as well.[1]

Dr. Elwood's conclusion is that while it is impossible to make a reasonable estimate of the total contribution of gasoline lead to blood lead, all the studies put together indicate that both from air and other routes of exposure, the contribution of gasoline lead is small.

I should also point out at this stage that Dr. Elwood is not alone in that view. Others have also indicated that the contribution of lead in gasoline is relatively small. In fact, a study that was carried out in Dallas—there has been a large lead controversy going on in Dallas—back in 1982, and I believe Dr. Houk was a part of that, indicated that in areas of heavy traffic, the contribution of lead in gasoline, as indicated by traffic density, was 1 microgram per deciliter. That's considerably lower than the levels that he was talking about here earlier today based on the NHANES II data.

We have heard a lot about the NHANES II data and the correlation between the reduction of lead in gasoline and lead in blood over the same period, 1976 to 1980. It's true, of course, that lead in gasoline was reduced over that period of time and so did blood leads come down over that same period of time.

However, to suggest that all, or even most, of the decline in blood lead was caused simply by a reduction of lead in gasoline is, in our view, simplistic and dangerous. I think it's simplistic because the analysis ignores the impact of other, concurrent private and governmental initiatives. In fact, there are some 14 governmental regulations on the books right now that require control of lead in the environment.

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[1] Retained in committee files.