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DISTRICT 1

APPEAL NO. 07AP2873

CITY OF MILWAUKEE,
Plaintiff-Appellant,

v.

NL INDUSTRIES, INC.
Defendant-Respondent

and

MAUTZ PAINT CO.
Defendant.

Appeal from the Circuit Court for Milwaukee County,
The Honorable John A. Franke, Circuit Court Judge,
Presiding
Circuit Court Case No.: 01CV003066

DEFENDANT-RESPONDENT'S APPENDIX

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Preventing Lead Poisoning in Young Children



A STATEMENT BY THE CENTERS FOR DISEASE CONTROL — OCTOBER 1991

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES / Public Health Service

Chapter 1. Introduction

Summary

New data indicate significant adverse effects of lead exposure in children at blood lead levels previously believed to be safe. Some adverse health effects have been documented at blood lead levels at least as low as 10 micrograms per deciliter of whole blood ($\mu\text{g/dL}$).

The 1985 intervention level of 25 $\mu\text{g/dL}$ is, therefore, being revised downwards to 10 $\mu\text{g/dL}$.

A multitier approach to followup has been adopted.

Primary prevention efforts (that is, elimination of lead hazards before children are poisoned) must receive more emphasis as the blood lead levels of concern are lowered.

The goal of all lead poisoning prevention activities should be to reduce children's blood lead levels below 10 $\mu\text{g/dL}$. If many children in the community have blood lead levels ≥ 10 $\mu\text{g/dL}$, communitywide interventions (primary prevention activities) should be considered by appropriate agencies. Interventions for individual children should begin at blood lead levels of 15 $\mu\text{g/dL}$.

Childhood lead poisoning is one of the most common pediatric health problems in the United States today, and it is entirely preventable. Enough is now known about the sources and pathways of lead exposure and about ways of preventing this exposure to begin the efforts to eradicate permanently this disease. The persistence of lead poisoning in the United States, in light of all that is known, presents a singular and direct challenge to public health authorities, clinicians, regulatory agencies, and society.

Lead poisoning is one of the most common and preventable pediatric health problems today.

Lead is ubiquitous in the human environment as a result of industrialization. It has no known physiologic value. Children are particularly susceptible to lead's toxic effects. Lead poisoning, for the most part, is silent: most poisoned children have no symptoms. The vast majority of cases, therefore, go undiagnosed and untreated. Lead poisoning is widespread. It is not solely a problem of inner city or minority children. No socioeconomic group, geographic area, or racial or ethnic population is spared.

Previous lead statements issued by the Centers for Disease Control (CDC) have acknowledged the adverse effects of lead at lower and lower levels. In the most recent previous CDC lead statement, published in 1985, the threshold for action was set at a blood lead level of 25 $\mu\text{g/dL}$, although it was acknowledged that adverse effects occur below that level. In the past several years, however, the scientific evidence showing that some adverse effects occur at blood lead levels at least as low as 10 $\mu\text{g/dL}$ in children has become so overwhelming and compelling that it must be a major force in determining how we approach childhood lead exposure.

This document provides guidelines on childhood lead poisoning prevention for diverse groups. Public health programs that screen children for lead poisoning look to this document for guidance on screening regimens and public health actions. Pediatricians and other health-care practitioners look to this document for information on screening and guidance on the medical treatment of poisoned children. Government agencies, elected officials, and private citizens seek guidance about what constitutes a harmful level of lead in blood—what the current definition of lead poisoning is and what blood lead levels should trigger environmental and other interventions.

It is not possible to select a single number to define lead poisoning for the various purposes of all of these groups. Epidemiologic studies have identified harmful effects of lead in children at blood lead levels at least as low as 10 $\mu\text{g}/\text{dL}$. Some studies have suggested harmful effects at even lower levels, but the body of information accumulated so far is not adequate for effects below about 10 $\mu\text{g}/\text{dL}$ to be evaluated definitively. As yet, no threshold has been identified for the harmful effects of lead.

Because 10 $\mu\text{g}/\text{dL}$ is the lower level of the range at which effects are now identified, primary prevention activities—communitywide environmental interventions and nutritional and educational campaigns—should be directed at reducing children's blood lead levels at least to below 10 $\mu\text{g}/\text{dL}$. Blood lead levels between 10 and 14 $\mu\text{g}/\text{dL}$ are in a border zone. While the overall goal is to reduce children's blood lead levels below 10 $\mu\text{g}/\text{dL}$, there are several reasons for not attempting to do interventions directed at individual children to lower blood lead levels of 10-14 $\mu\text{g}/\text{dL}$. First, particularly at low blood lead levels, laboratory measurements may have some inaccuracy and imprecision, so a blood lead level in this range may, in fact, be below 10 $\mu\text{g}/\text{dL}$. Secondly, effective environmental and medical interventions for children with blood lead levels in this range have not yet been identified and evaluated. Finally, the sheer numbers of children in this range would preclude effective case management and would detract from the individualized followup required by children who have higher blood lead levels.

The single, all-purpose definition of childhood lead poisoning has been replaced with a multitier approach, described in Table 1-1. Community prevention activities should be triggered by blood lead levels ≥ 10 $\mu\text{g}/\text{dL}$. Medical evaluation and environmental investigation and remediation should be done for all children with blood lead levels ≥ 20 $\mu\text{g}/\text{dL}$. All children with blood lead levels ≥ 15 $\mu\text{g}/\text{dL}$ should receive individual case management, including nutritional and educational interventions and more frequent screening. Furthermore, depending on the availability of resources, environmental investigation (including a home inspection) and remediation should be done for children with blood lead levels of 15-19 $\mu\text{g}/\text{dL}$, if such levels persist. The highest priority should continue to be the children with the highest blood lead levels.

Other differences between the 1985 and 1991 statements are as follows:

Screening test of choice. Because the erythrocyte protoporphyrin level is not sensitive enough to identify children with elevated blood lead levels below about 25 $\mu\text{g}/\text{dL}$, the screening test of choice is now blood lead measurement.

Universal screening. Since virtually all children are at risk for lead poisoning, a phase in of universal screening is recommended, except in communities where large numbers or percentages of children have been screened and found not to have lead poisoning. The full implementation of this will require the ability to measure blood lead levels on capillary samples and the availability of cheaper and easier-to-use methods of blood lead measurement.

Table 1-1. Interpretation of blood lead test results and follow-up activities: class of child based on blood lead concentration

Class	Blood lead concentration ($\mu\text{g/dL}$)	Comment
I	≤ 9	A child in Class I is not considered to be lead-poisoned.
IIA	10-14	Many children (or a large proportion of children) with blood lead levels in this range should trigger communitywide childhood lead poisoning prevention activities (Chapter 9). Children in this range may need to be rescreened more frequently.
IIB	15-19	A child in Class IIB should receive nutritional and educational interventions and more frequent screening. If the blood lead level persists in this range, environmental investigation and intervention should be done (Chapter 8).
III	20-44	A child in Class III should receive environmental evaluation and remediation (Chapter 8) and a medical evaluation (Chapter 7). Such a child may need pharmacologic treatment of lead poisoning (Chapter 7).
IV	45-69	A child in Class IV will need both medical and environmental interventions, including chelation therapy (Chapters 7 and 8).
V	≥ 70	A child with Class V lead poisoning is a medical emergency. Medical and environmental management must begin immediately (Chapters 7 and 8).

Primary prevention. Efforts need to be increasingly focused on preventing lead poisoning before it occurs. This will require communitywide environmental interventions, as well as educational and nutritional campaigns.

Succimer. In January, 1991, the U.S. Food and Drug Administration approved succimer, an oral chelating agent, for chelation of children with blood lead levels over 45 $\mu\text{g/dL}$.

Childhood lead poisoning prevention programs have had a tremendous impact on reducing the occurrence of lead poisoning in the United States. Because of these programs, deaths from lead poisoning and lead encephalopathy are now rare. These programs have targeted high-risk children for periodic screening; provided education to caretakers about the causes, effects, symptoms, and treatments for lead poisoning; and ensured medical treatment and environmental remediation for poisoned children. Screening and medical treatment of poisoned children will remain critically important until the environmental sources most likely to poison children are eliminated.

Federal regulatory and other actions have resulted in substantial progress in reducing blood lead levels in the entire U.S. population. In the last two decades, the virtual elimination of lead from gasoline has been reflected in reductions in blood lead levels in children and adults. Lead levels in food have also decreased since most manufacturers stopped using leaded solder in cans and since atmospheric deposition of lead on food crops declined as a result of reductions of lead in gasoline. In 1978, the Consumer Product Safety Commission banned the addition of lead to new residential paint.

Nevertheless, important environmental sources and pathways of lead remain. Lead-based paint and lead-contaminated dusts and soils remain the primary sources and pathways of lead exposure for children. In addition, children continue to be exposed to lead through air, water, and food, as well as occupations and hobbies of parents and caretakers. The focus of prevention efforts, therefore, must expand from merely identifying and treating individual children to include primary prevention—preventing exposure to lead before children become poisoned. This will require a shared responsibility among many public and private agencies. Public agencies will have to work with pediatric health-care providers to identify communities with childhood lead-poisoning prevention problems and unusual sources of lead and to ensure environmental followup of poisoned children. Public housing and economic development agencies will have to integrate lead paint abatement into housing rehabilitation policies and programs. Health-care providers will need to phase in virtually universal screening of children. Public and private organizations must continue to develop economical and widely-available blood lead tests to make such screening possible. Public and private housing owners must bear a portion of the financial burden for abatement.

The changes in this statement are not meant to create an enormous burden on primary pediatric health-care providers. These changes will only be useful if public health and other agencies effectively complement health-care providers' activities. Several efforts have begun to increase federal support of childhood lead poisoning prevention programs and of followup activities. Ongoing efforts to develop infrastructure and technology by the public and private sectors include 1) the development of inexpensive, easy-to-use portable methods for measuring blood lead levels; 2) the development of training and certification programs for lead paint inspectors and abatement contractors; and 3) the development and testing of new abatement methods, including encapsulants. The changes in this statement are also not meant to increase the emphasis on screening of children; the long-term goal of this statement is **prevention**. Until primary prevention of childhood lead poisoning can be achieved, however, increased screening and followup of poisoned children is essential.

Elimination of Childhood Lead Poisoning

Will require efforts from both the private and public sectors.

Will require a shift in emphasis to primary prevention.

Will take time and resources.

Should proceed in a rational manner, with the highest risk children being made the highest priority.

Can be achieved.

In February 1991, the U.S. Department of Health and Human Services released a *Strategic Plan for the Elimination of Childhood Lead Poisoning* (HHS, 1991). This plan describes the first 5 years of a 20-year societywide effort to eliminate this disease. It places highest priority on first addressing the children at greatest risk for lead poisoning. The U.S. Department of Housing and Urban Development (HUD, 1990) and the Environmental Protection Agency (EPA, 1991) have both released plans dealing with the elimination of lead hazards. To eliminate this disease will require a tremendous effort from all levels of government as well as the private sector, but we believe that the benefits to society will be well worth it. We look forward to the day when childhood lead poisoning is no longer a public health problem.

References

CDC (Centers for Disease Control). Preventing lead poisoning in young children: a statement by the Centers for Disease Control. Atlanta: CDC, 1985; CDC report no. 99-2230.

EPA (Environmental Protection Agency). Strategy for reducing lead exposures: report to Congress. Washington (DC): EPA, 1991.

HHS (U.S. Department of Health and Human Services). Strategic plan for the elimination of childhood lead poisoning. Atlanta: CDC, 1991.

HUD (U.S. Department of Housing and Urban Development). Comprehensive and workable plan for the abatement of lead-based paint in privately owned housing: report to Congress. Washington (DC): HUD, 1990.

Chapter 2. Background

Summary

The blood lead level considered to indicate lead toxicity has progressively shifted downwards.

In general, children are more at risk for lead exposure than adults.

Large numbers of children in the United States continue to have blood lead levels in the toxic range.

This chapter describes the health effects of lead on children and fetuses, the metabolism of lead, and the demographics of lead exposure in the United States. It explains why the definition of childhood lead poisoning is being revised.

EFFECTS OF LEAD ON CHILDREN AND FETUSES

Lead affects virtually every system in the body.

The blood lead level considered to indicate lead poisoning has fallen steadily since the 1970s.

Blood lead levels at least as low as 10 µg/dL are associated with adverse effects.

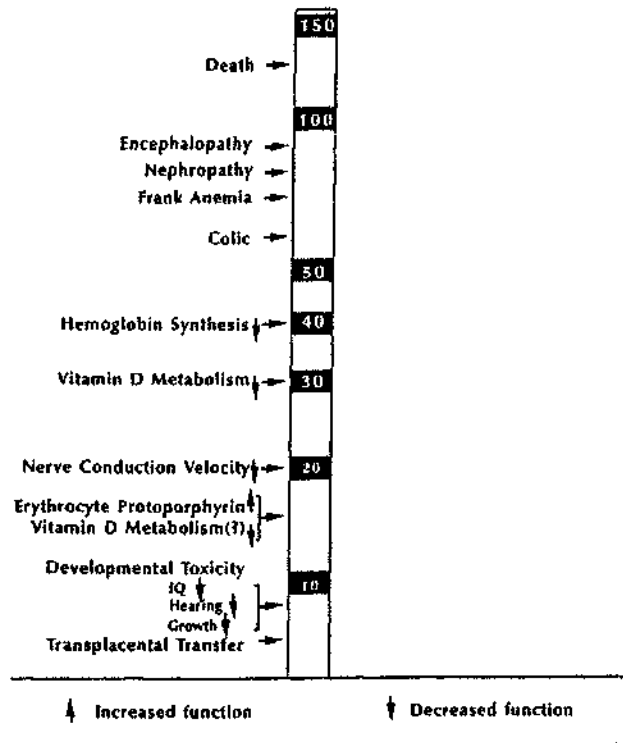
Although the effects of low-level lead exposure may not seem severe in the individual child, on a population basis they are extremely important.

Lead is a poison that affects virtually every system in the body. It is particularly harmful to the developing brain and nervous system of fetuses and young children. The adverse effects of lead on children and adults are summarized in Figure 2-1.

The risks of lead exposure are not based on theoretical calculations. They are well known from studies of children themselves and are not extrapolated from data on laboratory animals or high-dose occupational exposures.

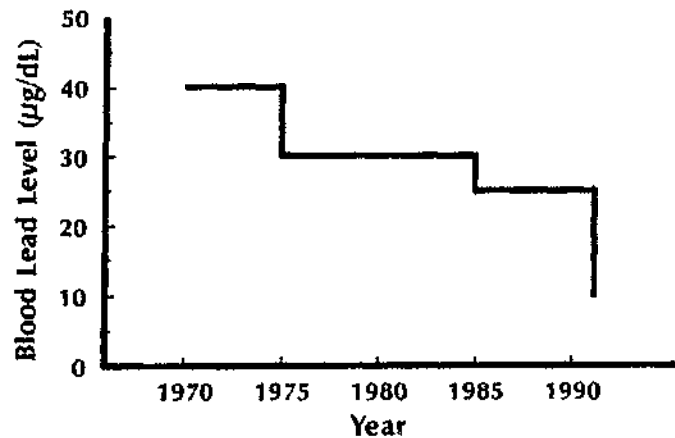
Levels of Concern

Since 1970, our understanding of childhood lead poisoning has changed substantially. As investigators have used more sensitive measures and better study designs, the generally recognized level for lead toxicity has progressively shifted downward. Before the mid-1960s, a level above 60 µg/dL was considered toxic (Chisolm and Harrison, 1956). By 1978, the defined level of toxicity had declined 50% to 30 µg/dL. Figure 2-2 shows how the federal definition of an elevated blood lead level has changed over the years.

Figure 2-1. Lowest observed effect levels of inorganic lead in children*

*Note: The levels in this diagram do not necessarily indicate the lowest levels at which lead exerts an effect. These are the levels at which studies have adequately demonstrated an effect.

Source: ATSDR, 1990.

Figure 2-2. Blood lead levels considered elevated by the Centers for Disease Control and the Public Health Service

Range of Effects of Lead

Very severe lead exposure in children (blood lead levels ≥ 80 $\mu\text{g/dL}$) can cause coma, convulsions, and even death. Lower levels cause adverse effects on the central nervous system, kidney, and hematopoietic system. Blood lead levels as low as 10 $\mu\text{g/dL}$, which do not cause distinctive symptoms, are associated with decreased intelligence and impaired neurobehavioral development (Davis and Svendsgaard, 1987; Mushak et al., 1989). Many other effects begin at these low blood lead levels, including decreased stature or growth (Schwartz et al., 1986; Bornschein et al., 1986; Shulka et al., 1989), decreased hearing acuity (Schwartz and Otto, 1987), and decreased ability to maintain a steady posture (Bhattacharya et al., 1988). Lead's impairment of the synthesis of the active metabolite 1,25-(OH)₂ vitamin D is detectable at blood lead levels of 10-15 $\mu\text{g/dL}$. Maternal and cord blood lead levels of 10-15 $\mu\text{g/dL}$ appear to be associated with reduced gestational age and reduced weight at birth (ATSDR, 1988). Although researchers have not yet completely defined the impact of blood lead levels <10 $\mu\text{g/dL}$ on central nervous system function, it may be that even these levels are associated with adverse effects that will be clearer with more refined research.

Studies of Low-Level Lead Effects on the Central Nervous System

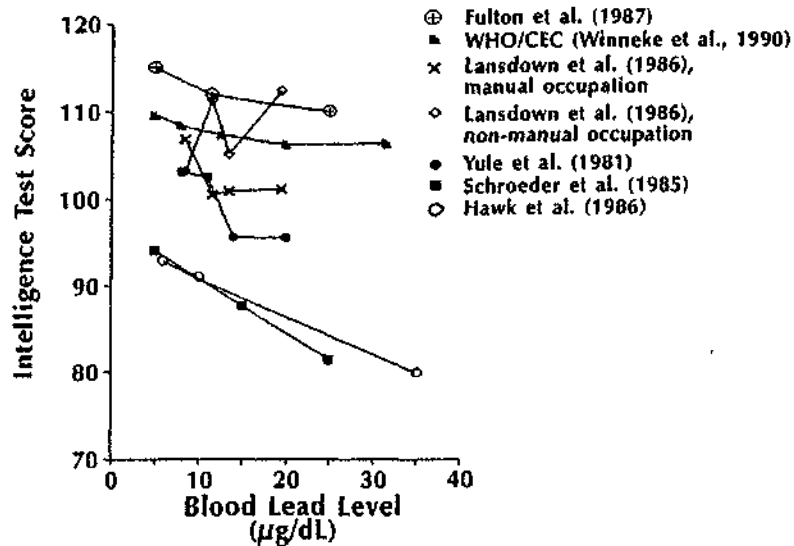
The concern about adverse effects on central nervous system functioning at blood lead levels as low as 10 $\mu\text{g/dL}$ is based on a large number of rigorous epidemiologic and experimental studies. In particular, recent cross-sectional and prospective studies have provided new evidence about the association between low-level lead exposure and child development.

Several well-designed and carefully conducted cross-sectional and retrospective cohort studies in many different countries have been conducted (Lansdown et al., 1986; Fulton et al., 1987; Fergusson et al., 1988; Silva et al., 1988; Bergomi et al., 1989; Hansen et al., 1989; Hatzakis et al., 1989; Winneke et al., 1990; Lyngbye et al., 1990; Needleman et al., 1990; Yule et al., 1981; Lansdown et al., 1986; Hawk et al., 1986; Schroeder et al., 1985). Figure 2-3 shows the mean intelligence quotient (IQ) scores (in most cases adjusted for potential confounding factors) achieved by children with different blood lead levels from several of these studies. Some inconsistencies can be found in the results of these studies, but the weight of the evidence clearly supports the hypothesis that decrements in children's cognition are evident at blood lead levels well below 25 $\mu\text{g/dL}$. No threshold for the lead-IQ relationship is discernable from these data.

Most investigators report lower IQ scores among the more highly exposed children but these differences have not uniformly reached statistical significance (that is, $p < .05$). One way to synthesize the data from different studies is meta-analysis. Recent evaluation of 24 major cross-sectional studies provides strong support for the hypothesis that children's IQ scores are inversely related to lead burden (Needleman and Gatsonis, 1990).

Although available evidence is not sufficient to conclude that lead-associated deficits are irreversible, a recent follow-up study reported that the educational success of a cohort of young adults was significantly inversely associated with the amount of lead in teeth they shed as first and second graders (Needleman et al., 1990). In this study, dentine lead levels above 20 ppm were associated with a seven-fold risk of not graduating from high school, a six-fold risk of having a reading disability, deficits in vocabulary, problems with attention and fine motor coordination, greater absenteeism, and lower class ranking. Although dentine lead levels did not correspond in any simple way to blood lead levels, the available preschool blood lead levels

Figure 2-3. Blood lead levels and IQ scores of children, from cross-sectional and retrospective cohort studies*



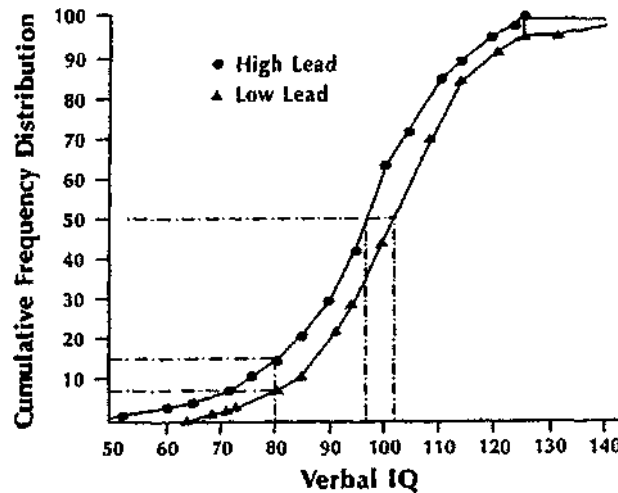
*Data from prospective studies are not included.

of the more highly exposed children averaged 35 µg/dL (Needleman et al., 1979). Increased circumpulpal dentine lead levels (>16 ppm) have been linked to higher rates of learning disabilities in a recent Danish study as well (Lyngbye et al., 1990).

To address methodological limitations of cross-sectional studies of lead and child development, a number of prospective studies were begun during the 1980s. Blood lead measurements were begun during the prenatal period and continued for several years, along with assessment of development. In several but not all cohorts, prenatal exposures have been associated with slower sensory-motor and delayed early cognitive development (Bellinger et al., 1987; Bellinger et al., 1991; Dietrich et al., 1987; Ernhart et al., 1986; Dietrich et al., 1991). With low postnatal exposures and favorable socioeconomic conditions, some of these early associations may attenuate as children grow older (Bellinger et al., 1991). In addition, several studies have noted that children's cognitive performance in the preschool period may be associated with early postnatal lead exposures (McMichael et al., 1988; Bellinger et al., 1991). It will be necessary for these prospective studies to follow their respective cohorts into the school-age years in order for the full implications of these early patterns to become clear.

Questions are frequently raised about the practical significance of the difference frequently observed between the IQ scores of more exposed and less exposed children. For the previously described population of children studied by Needleman et al. (Needleman et al., 1979), a shift in mean IQ score of 4-6 points as a result of lead exposure was associated with a substantial increase in the prevalence of children with severe deficits (that is, IQ scores less than 80) (Figure 2-4). Similarly, in this population the shift was associated with an absence of children who achieved superior function (that is, IQ scores greater than 125).

Figure 2-4. Cumulative frequency distribution of verbal IQ scores in children with high and low tooth lead levels



Source: Needleman et al., 1979.

ABSORPTION OF LEAD

Children are at higher risk for lead exposure because
 They have more hand-to-mouth activity than adults.
 They absorb more lead than adults.

Many factors can affect the absorption, distribution, and toxicity of lead. Children are more exposed to lead than older groups because their normal hand-to-mouth activities may introduce many nonfood items into their gastrointestinal tract (Lin-Fu, 1973). The efficiency of gastrointestinal absorption of lead in food and beverages in children has been estimated to be around 40% (Ziegler et al., 1978). From experimental studies, gastrointestinal absorption of lead from nonfood sources is decreased in the presence of food (Rabinowitz, 1980). Efficiency of absorption is probably also affected by the particle size and form of lead (Barltrop and Meek, 1979). Deficiencies in iron, calcium, protein, and zinc are related to increased blood lead levels and perhaps increased vulnerability to the adverse effects of lead (Mahaffey, 1981; Mahaffey and Michaelson, 1980).

BLOOD LEAD LEVELS IN THE UNITED STATES

Large numbers of children continue to have blood lead levels high enough to cause adverse effects.

Substantial progress has been made, however, in reducing blood lead levels in the United States.

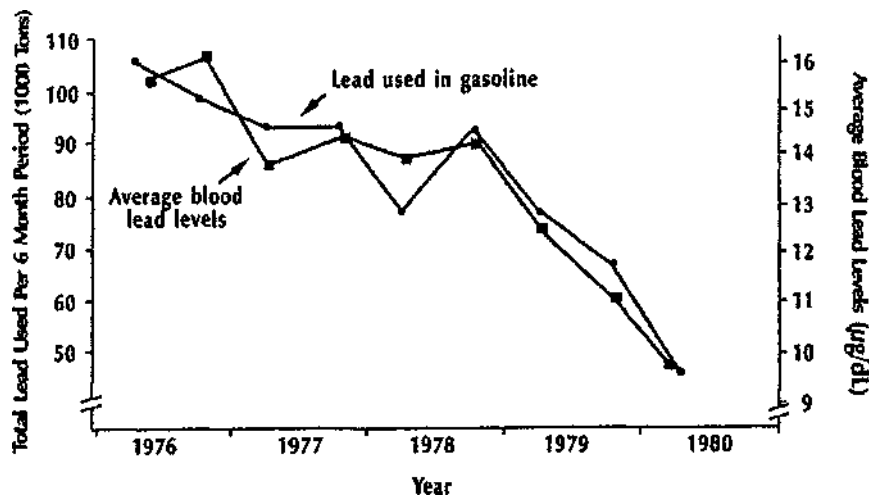
Lead-based paint remains the major source of high-dose lead poisoning in the United States.

The Agency for Toxic Substances and Disease Registry estimated that in 1984, 17% of all American preschool children had blood lead levels that exceed 15 $\mu\text{g}/\text{dL}$ (ATSDR, 1988). Although all children are at risk for lead toxicity, poor and minority children are disproportionately affected. Lead exposure is at once a by-product of poverty and a contributor to the cycle that perpetuates and deepens the state of being poor.

Substantial progress has been made in reducing blood lead levels in U.S. children. Perhaps the most important advance has been the virtual elimination of lead from gasoline. Close correlations have been demonstrated between the decline in the use of leaded gasoline and declines in the blood lead levels of children and adults between 1976 and 1980 (Annest, 1983) (Figure 2-5). Levels of lead in food have also declined significantly, as a result both of the decreased use of lead solder in cans and the decreasing air lead levels.

Lead-based paint remains the major source of high-dose lead poisoning in the United States. Although the Consumer Products Safety Commission (CPSC) limited the lead content of new residential paint starting in 1978, millions of houses still contain old leaded paint. The Department of Housing and Urban Development estimates that about 3.8 million homes with young children living in them have either nonintact lead-based paint or high levels of lead in dust (HUD, 1990).

Figure 2-5. Change in blood lead levels in relation to a decline in use of leaded gasoline, 1976-1980



Source: Annest JL, 1983.

INTERPRETATION OF BLOOD LEAD LEVELS

In interpreting blood lead levels, the provider should

Understand the scientific basis for concern.

Understand the degree of imprecision and inaccuracy in blood lead measurements.

Explain carefully why followup is or is not needed.

The studies which form the basis of our concern about childhood lead poisoning are described in Chapter 2. These studies suggest that adverse effects of lead occur at blood lead levels at least as low as 10 $\mu\text{g}/\text{dL}$. The following paragraphs provide guidance on what might be told to a parent, depending on the blood lead levels of the child.

Blood lead level <10 $\mu\text{g}/\text{dL}$. A blood lead level <10 $\mu\text{g}/\text{dL}$ is not considered to be indicative of lead poisoning.

Blood lead level 10-14 $\mu\text{g}/\text{dL}$. Children with blood lead levels in this range are in a border zone. Since the laboratory tests for measuring blood lead levels are not as accurate and precise as we would like them to be at these levels, many of these children's blood lead levels may, in fact, be <10 $\mu\text{g}/\text{dL}$. Although a detailed environmental history should be taken since an obvious remediable source of lead may be found, it is unlikely that there is a single predominant source of lead exposure for most of these children. Thus, a full home inspection is not recommended. It is, however, prudent to try to decrease exposure to lead with some simple interventions (Page 30). (The required education can be done face-to-face or by distributing brochures or other written materials.) In addition, these children should receive followup blood lead testing in about 3 months. The adverse effects of blood lead levels of 10-14 $\mu\text{g}/\text{dL}$ are subtle and are not likely to be recognizable or measurable in the individual child. It is important to make sure that these children's blood lead levels do not go up.

Example: Johnny was a 12 month old child without any risk factors for high-dose exposure. A capillary blood lead test was performed, and his blood lead level was 14 $\mu\text{g}/\text{dL}$. His pediatrician told his mother that Johnny's blood lead test was in a kind of "border" zone, but that it was high enough to require careful followup. The pediatrician explained that laboratory test results have some inaccuracy and imprecision, but, nevertheless, suggested some housekeeping and nutritional interventions to reduce Johnny's exposure. Johnny had a venous blood lead measurement three months later, which was 7 $\mu\text{g}/\text{dL}$. Three months after that, when Johnny was 18 months old, his blood lead level was 5 $\mu\text{g}/\text{dL}$. His blood lead level was measured one year later and was 5 $\mu\text{g}/\text{dL}$, and he received no further followup.

Blood lead level 15-19 $\mu\text{g}/\text{dL}$. Children with venous blood lead levels 15-19 $\mu\text{g}/\text{dL}$ need more careful followup. The pediatric health-care provider should take a careful history, asking about sources of lead exposure (Chapter 3). Parents should receive guidance about interventions to reduce blood lead levels (Page 30). Children with blood lead levels in this range are at

risk for decreases in IQ of up to several IQ points and other subtle effects. The effects of lead at these levels are significant enough that the health-care provider should emphasize to parents the importance of followup screening to make sure the levels do not increase. The provider should also discuss interventions to reduce the blood lead levels. In addition, these children should receive followup testing (Chapter 6). If their blood lead levels persist at $\geq 15 \mu\text{g/dL}$, environmental investigation and remediation should be completed, if resources permit. In some communities, childhood lead poisoning prevention programs may be able to manage the environmental investigation and remediation.

Blood lead level 20-69 $\mu\text{g/dL}$. Children with venous blood lead levels in this range should have a full medical evaluation (Chapter 7). This includes a detailed environmental and behavioral history (asking about reading or other learning disabilities, language development, pica, etc.), a physical examination, and tests for iron deficiency. Particularly for children needing urgent medical followup (that is, blood lead level $\geq 45 \mu\text{g/dL}$), pediatric health-care providers with limited experience in treating lead poisoning should consider referring such children to a clinic with experience in managing childhood lead poisoning. These children should also have complete environmental investigations so that lead hazards can be reduced. The local public childhood lead poisoning prevention programs will often work as a team with the pediatric health-care provider and the child's family to ensure appropriate environmental followup.

Blood lead level $\geq 70 \mu\text{g/dL}$. Children with blood lead levels this high constitute a medical emergency that preferably should be managed by someone with experience in treating children who are critically ill with lead poisoning. Medical and environmental management must begin immediately (Chapters 7 and 8).

EDUCATING PARENTS ABOUT REDUCING BLOOD LEAD LEVELS

What can parents do to reduce blood lead levels?

Housekeeping interventions to reduce exposure to dust.
Interventions to reduce exposure to other sources of lead.
Attention to nutrition.

There are many interventions parents can use to help reduce blood lead levels. These interventions are not a substitute for lead hazard abatement.

Housekeeping Interventions

Particularly in older homes, which may have been painted with lead-based paint, interventions to reduce exposure to dust may help reduce blood lead levels. These include:

- Make sure your child does not have access to peeling paint or chewable surfaces painted with lead-based paint. Pay special attention to windows and window sills and wells.

EX14

October 2004

**EVALUATION OF THE EFFECTIVENESS OF THE MILWAUKEE
LEAD HAZARD CONTROL ORDINANCE**

Summary Report

**Prepared by
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Elizabeth Slone, and John Menkedick**

**Battelle
505 King Avenue
Columbus, OH 43201**

**Contract No. C-OPC-21357
Task Order 2**

**Peter Ashley, DrPH, Government Technical Monitor
U.S. Department of Housing and Urban Development
Washington, D.C. 20410**

0065-WRR-000042925

Battelle Final Report v.2

October 2004
Contract C-OPC-21357 - Task Order 2

Exh 207

P1234

R-App 0015

Table 2-5 presents descriptive statistics on children's blood-lead concentrations. Table 2-6 displays model-based unadjusted geometric means of blood-lead concentrations and proportion of children with elevated blood-lead levels. For those housing units with multiple children, these models account for the anticipated correlation between those children. Without adjusting for potential differences in sample recruitment between treatment and control neighborhoods and other potential explanatory covariates, the results indicate that:

In the North Neighborhoods:

- There are no statistically significant differences between the control neighborhood and its associated treatment neighborhood at Phase I, in terms of the geometric mean of children's blood-lead levels or proportion of children with elevated blood-lead levels.
- In the treatment neighborhood, although the geometric mean of children's blood-lead levels and the proportion of children with elevated blood-lead levels both decrease between Phase I and Phase II, these differences are not statistically significant.
- In the control neighborhood, the geometric mean of children's blood-lead levels and the proportion of children with elevated blood-lead levels (PbB $\geq$ 10 μ g/dL) at Phase II were significantly lower than at Phase I.
- The effects of neighborhood-wide lead poisoning prevention treatments in reducing geometric means of children's blood-lead levels or the proportion of children with elevated blood-lead levels were not statistically significant.

In the South Neighborhoods:

- The control neighborhood had a significantly higher geometric mean blood-lead concentration than its associated treatment neighborhood at Phase I for children identified from the STELLAR database (p-value=0.01), as well as for the combined group (p-value=0.015).
- The effects of lead poisoning prevention treatments in reducing geometric means of children's blood-lead levels or proportion of children with elevated blood-lead levels were not statistically significant.

0065-WRR-000042934

Effectiveness in Achieving Lower Blood Lead Levels

Although the treatments conducted under the Milwaukee Pilot Ordinance were relatively effective in bringing window dust lead loadings below those in comparable untreated units, the effects on children's blood lead levels did not meet original expectations. The study compared the probability of having an elevated blood-lead level (i.e., blood lead concentrations at or above 10 µg/dL) by age between the Scheduled Screening study children and Prospective Study children, and found no significant differences, with or without accounting for other factors of interest. Twenty-six percent of children age 18 months in the Prospective study group had elevated blood-lead levels (reducing to 13% at Phase IV), while 27 percent of children in Phase II (median age 24 months) of the Scheduled Screening group had elevated levels.

To better understand why the treatments did not result in the anticipated improvements in children's health, a number of potential confounding factors were explored. The study had earlier found that children living in the North side of Milwaukee tended to have higher blood lead levels than children in the South side of the city, so it was hypothesized that neighborhood factors may have influenced the probability of elevated blood lead levels. The study also looked at whether the time when the Ordinance went into effect in a community had an impact on the blood lead levels. Although both the neighborhood and the time when the Ordinance went into effect influenced the blood lead levels, after controlling for these factors, there were still no significant differences in the likelihood of having an elevated blood-lead level between the two study groups and no differences between the geometric mean blood lead levels in the two groups.

One factor that appears to have had an effect on the blood lead levels between the Prospective Study group and the Scheduled Screening group was the floor dust lead loading. Children in the Prospective Study were divided into two groups: those living in homes where all visits had floor dust lead levels less than 20 µg/ft² and those living in homes with at least one higher dust lead result. Prospective Study children living in a home with the lower floor dust lead loadings had proportionally fewer elevated blood lead levels as they got older compared to all children in the Scheduled Screening group (regardless of floor dust-lead levels) when other factors were not considered, as seen in Table 3-5. In addition, children living in treated dwellings with lower floor lead levels had significantly lower blood lead levels than children in the untreated dwellings, at 6, 12, and 18 months of age; at 24 months, the blood lead levels were marginally lower (p-value=0.059).

These findings suggest that had the treatments been able to result in lower floor dust lead levels than in the Scheduled Screening homes, then the impact on the blood lead levels may have been greater. This observation is further supported by the findings that after controlling for age and seasonal effects, floor dust lead levels were significantly related (p-value=0.006) to the blood lead concentration of children in the Prospective Study, while window sill and trough levels were only marginally related (p-value=0.058 and 0.069, respectively). Of all the factors that influenced floor dust lead loadings (season, inspector, rental vs. owner-occupied, interior deterioration), interior deterioration is one factor that is both a possible cause and could be influenced by treatment.

0065-WRR-000042954

Potential Limitation of the Study

The observed trends in blood lead levels for the two groups were also unexpected. Previous studies have demonstrated that children's blood lead levels tend to rise from birth until early in the second year and then decline. It would have been anticipated that children in the Scheduled Screening group would have exhibited this positive rate of increase in blood lead levels from age 6 months to 24 months, while children in the Prospective study would have had a lower rate of increase or ideally no significant increase. Instead, there was no significant change in blood lead levels for the Scheduled Screening group after controlling for other factors, while there was a significant upward trend for the Prospective Study group. It raises the question of why children in the untreated dwellings would not experience the same upward trend of blood lead levels that is observed with children in other studies and whether it is appropriate to use this population as a quasi-control population.

At age 6 months, the predicted blood lead level for children in the Scheduled Screening group was almost twice the level seen in children in the Prospective Study group. As noted earlier, because household exposure sources would be expected to be less of an influence on younger less mobile children, the magnitude in this difference was not anticipated. If our assumption is wrong about household exposure at a young age, then this suggests that the treated homes may have a dramatic effect on children at a young age, but the effect disappears by age two. However, if the original assumption about household exposures is correct, the finding may further suggest that children in the Scheduled Screening group had different non-housing lead exposure levels before 6 months (and possibly before birth) than children in the Prospective Study. The finding could also mean that the scheduled screening study represents a biased sample of children. For example, despite the fact that the Milwaukee Standard of Care includes the proactive screening of children for elevated blood-lead levels at 12 months of age, the population of children that actually receive this screening may be biased towards higher levels of lead exposure.

Conclusions

Whether the Scheduled Screening group served as an optimal control group or not, the fact that children in the treated dwellings had a significant increase in blood lead levels from age 6 months to 24 months and that over a quarter of these children had elevated blood lead levels by the end of the study suggests that improvements to the Milwaukee Pilot Ordinance (e.g., more focus on exterior lead sources such as soil that can be tracked into the homes) should be considered. The program was very successful in reducing window dust lead loadings, an exposure source that proved to be marginally related to the children's blood lead levels. However, it was not successful in reducing floor dust lead levels, which was strongly influential as an exposure source. The strong relationship observed between interior deterioration and dust lead levels suggests the importance of correcting interior deterioration and maintaining properties over time. Adding components to the city's program that would be expected to result in lower floor dust-lead loadings and to keep floor dust lead loadings low, would be recommended to improve the benefits of the program.

0065-WRR-000042955

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October 2004
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Exh 207

P1234.031

R-App 0018

	A	B	C	D	E	F	G	H	I
1	Incidence (<18 yrs)			Incidence	Incidence				
2	Year	>=10 ug/dL		# children <6 yrs >=10 ug/dL	# children <6 yrs >=20 ug/dL				
3	1997	22.0%		3464	518				
4	1998	16.7%		2670	375				
5	1999	15.2%		2255	304				
6	2000	12.6%		1961	260				
7	2001	12.2%		2038	248				
8	2002	11.4%		1787	244				
9	2003	9.6%		1462	209				
10	2004	8.4%		1380	157				
11	2005	7.2%		1117	157				
12	2006	6.1%		884	147				
13				19016	2619				
14				Total in last 10 years	Total in last 10 years				
15	Prevalence (<18 yrs)								
16	Year	>=10 ug/dL							
17	1997	31.9%							
18	1998	28.5%							
19	1999	23.9%							
20	2000	17.2%							
21	2001	15.8%							
22	2002	13.8%							
23	2003	11.2%							
24	2004	9.6%							
25	2005	8.1%							
26	2006	6.4%							
27									
28	Prevalence (<6 yrs)			<6 yrs only	<6 yrs only		<6 yrs only	<6 yrs only	
29	Year	>=10 ug/dL		# children >=10 - numerator	# children - denominator		# children >=20	% of children >=20	
30	1997	31.9%		6550	20506		1409	6.9%	
31	1998	26.4%		5208	19719		1032	5.2%	
32	1999	23.5%		4198	17893		780	4.4%	
33	2000	19.3%		3658	18992		684	3.6%	
34	2001	16.5%		3563	21565		620	2.9%	
35	2002	14.1%		3217	22813		526	2.3%	
36	2003	11.3%		2588	22815		447	2.0%	
37	2004	9.8%		2364	24007		364	1.5%	
38	2005	8.3%		1865	22510		304	1.4%	
39	2006	6.6%		1414	21440		236	1.1%	
40									
41	Testing Penetration (1 & 2 yrs only)								
42	Year								
43	1997	25.0%							
44	1998	37.7%							
45	1999	46.9%							
46	2000	48.8%							
47	2001	52.6%							
48	2002	49.7%							
49	2003	50.6%							

Exh 209

P1247

R-App 0019

	A	B	C	D	E	F	G	H	I
50	2004	53.0%							
51	2005	49.9%							
52	2006	46.7%							

Exh 209

P1247.002

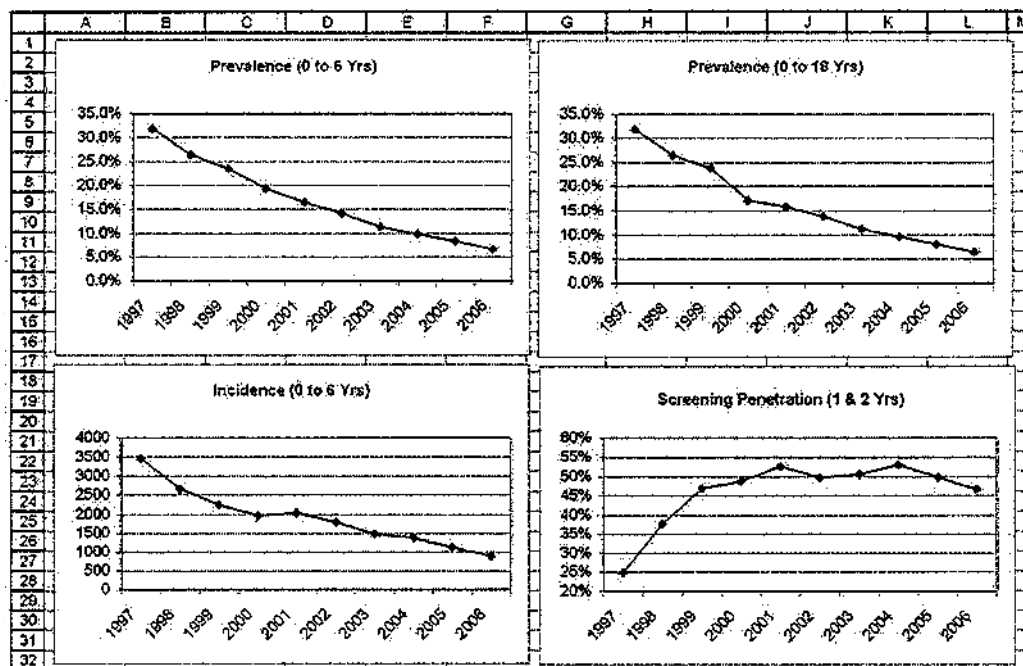
R-App 0020

	A	B	C	D	E	F	G
1	Prevalence (0 to 6 yrs)			0-6 yrs only	0-6 yrs only		0-6 yrs only
2	Year	>=10 ug/dL		# children - numerator	# children - denominator		# children >=20
3	1991	83.3%*		3217	3864		1242
4	1992	78.5%*		7229	9445		2685
5	1993	57.6%*		5977	10375		2240
6	1994	63.7%*		7426	11653		2028
7	1995	46.9%*		7156	15288		1703
8	1996	34.8%		6840	19635		1485
9							
10							
11	*Note: Data prior to 1996 must be viewed critically. State law with respect to reporting lead test results						
12	was enacted in the fall of 1994. It took a while for State and local Health authorities to work						
13	with private clinical laboratories to educate them to the law and implement systems for						
14	reporting lead results. Prior to 1996 (or mid 1995), reporting from private laboratories or						
15	medical providers was sporadic at best. In the early 90's, results 20 or greater were thought						
16	to be sufficient for reporting, and later 10 or greater was the presumed cutoff for reporting.						
17	This results in inflated (or minimally questionable) prevalence rates due to smaller denominators.						

Exh 209

P1247.003

R-App 0021



Exh 209

P1247.004

R-App 0022

Exh 404

ANNUAL LEAD GRANT EXPENDITURES
By funding source
1992-2005

	SOURCE	1992	1993	1994	1995	1996	1997	1998	1999	2000	2001	2002	2003	2004	2005	TOTAL
EVALUATION GRANT (EPA)	Federal					45,187	45,804	48,880	7,554							146,535
ENVIRONMENTAL JUSTICE GRANT (EPA)	Federal					114,260	157,440	82,280								353,980
LEAD PAINT WORKER TRAINING GRANT (EPA)	Federal					49,920										49,920
MUD ROUND 1	Federal			290,816	1,143,588	1,428,140	145,070	474,330	766,824							3,648,868
LEAD BASED PAINT HAZARD (LHD) R	Federal						1,068,113	1,624,212	1,290,860	812,520	66,937					4,967,842
LEAD POISONING PREVENTION (LPP)	Federal	222,560	215,290	258,489	394,626	540,363	636,455	676,044		640,540	532,943	904,018	470,283	520,863	723,643	7,421,322
LEAD HAZARD REDUCTION (LHD) R	Federal									286,185	1,581,307	534,518	1,870,543	718,151	18,229	5,090,300
LEAD PACT (LHD) R	Federal									815,739	1,487,568	271,774				3,000,300
LEAD URBAN INITIATIVES (LHD)	Federal													1,325,606	973,925	2,299,531
LEAD (LHD/INDO)	Federal													84,848	223,311	308,159
CONDUCT LEAD (LHD)	Federal									600,000	540,438	650,365	629,848	783,727	807,128	3,912,406
LEAD DETECTION/ELIMINATION	State	175,577	132,978	190,208	319,111	332,509	315,930	344,678	359,582	274,489	250,594	311,521	310,540	344,326	300,372	4,409,143
LEAD OUTREACH (LHD)	Federal															542,857
LEAD PAINT HAZARD CONTROL (LHD 11)	Federal															2,367,454
LEAD DEMONSTRATION	Federal															2,342,142
LEAD POISONING PREVENTION EDUCATION	State	11,001	20,453	30,451	28,269											137,184
GRN	City	57,791	256,285	285,232	124,068	182,104	209,156	182,889	541,738	694,622	698,201	696,007	675,142	617,913	551,948	8,595,282
Indirect Costs	City	64,428	118,438	273,006	411,303	588,428	298,260	436,012	160,813	219,006	104,722	130,445	87,812	128,911	153,829	3,956,630
TOTAL		\$921,288	\$772,884	\$1,258,966	\$2,471,187	\$3,349,074	\$3,568,214	\$3,877,744	\$3,347,964	\$4,432,464	\$3,592,363	\$3,480,426	\$4,420,316	\$4,422,028	\$8,778,138	\$53,036,828

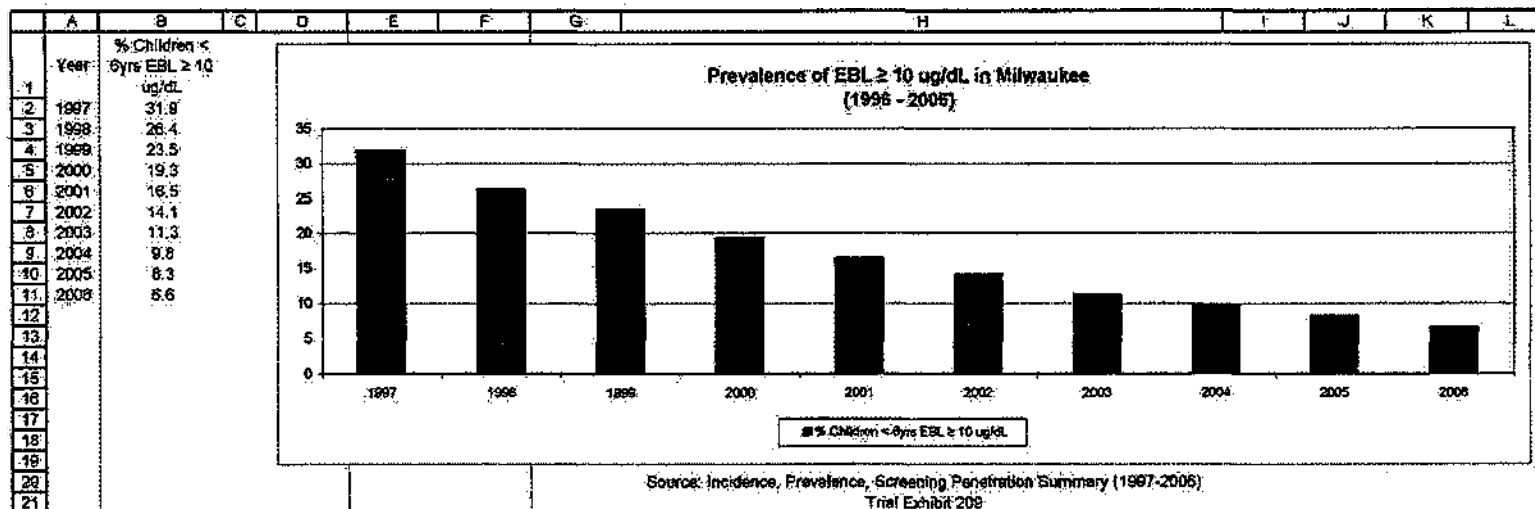
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BUS TOTAL	\$53,036,828
Subtract 0.74% for Non-Paint Investigations**	(\$397,473)
FINAL TOTAL	\$52,639,355

** Since its inception, the Childhood Lead Poisoning Prevention Program has spent its resources addressing lead hazards caused by the presence of lead paint. During the 15-year lifetime of the program, there have been exceptional instances in which the Program investigated non-paint sources of lead. The LIS database shows that from 01/01/97 through 12/31/05, the LIS tracked over 100,000 samples for the presence of lead, and non-paint sources of lead were found to determine the cause of a child's elevated blood level of 30+ mcg/dL. The STELLAR database shows that during the same period, at least 5,014 investigations were conducted by the Program to determine the cause of a child's elevated blood lead. As such, approximately 0.74% percent of the Program's resources have been expended on non-paint sources of lead. This amount is deducted from the subtotal of the final program figure.

ML06CMHT-053540

P2272.004



Exh 409

P2277

R-App 0024



Levels of Lead Poisoning

_____ lead level is _____
 (child's name) (lead result)

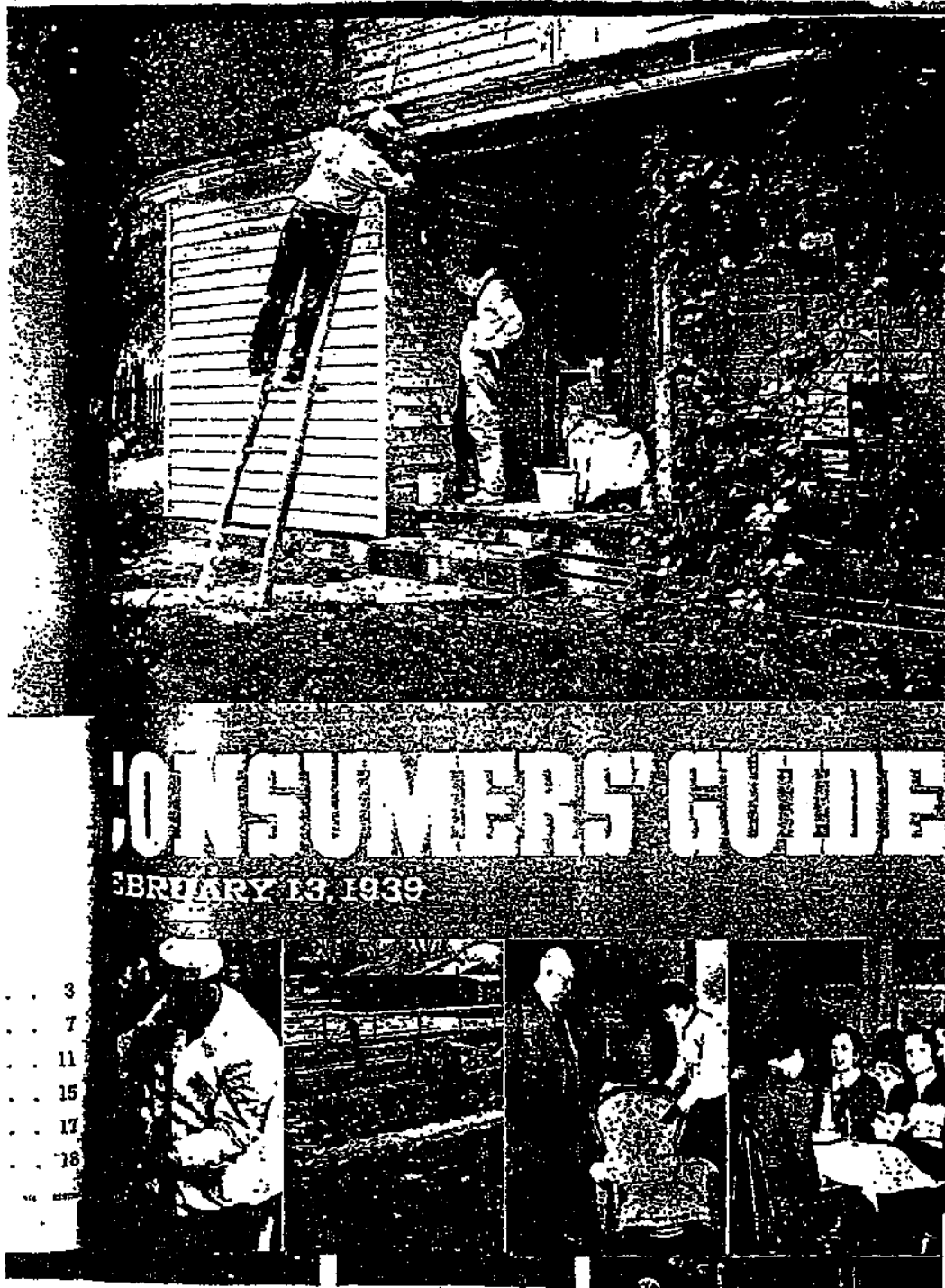
LEAD LEVEL	MEANING	NEXT ACTIONS
0 – 9	SAFE no lead poisoning	• Have lead test done once each year.
10 – 19	EXPOSED to lead	SEE YOUR DOCTOR • Retest* in 3 – 4 months
20 – 24	Lower Level LEAD POISONING	SEE YOUR DOCTOR • Retest* in 3 – 4 months
25 – 44	LEAD POISONED	SEE YOUR DOCTOR • Retest* in 1 – 3 months • Home will be inspected for lead hazards by Health Dept.
45 and higher	EXTREME LEAD POISONING	• Child may be hospitalized to remove lead from the body (chelation) • X-rays may also be taken • Home will be inspected for lead hazards by Health Dept. • Retest* child frequently after chelation

* Retest should be a venous test (blood is drawn from child's arm.)

LEAD LEVELS FOR: _____ Date of Birth _____

LAB RESULTS				NEXT TEST DUE	OTHER NOTES
DATE DONE	TESTING PLACE	LEAD	OTHER		

This information will be helpful to share with your doctor.



0049-EXH-044453

Exhibit 542

R-App 0027

6 inactive opaque pigments. They are the titanium pigments and the zinc sulfide pigments (lithopone). Transparent pigments, most of which are chemically inactive, do not give paint its essential hiding power. The most important ones are silica, magnesium silicate, calcium carbonate (whiting, chalk), barium sulfate (barytes, blanc fixe), and kaolin (white china clay). Inexpensive, they are used legitimately to give greater volume of pigment at low cost, but used to excess they become adulterants. Unfortunately there is no simple rule to give consumers for telling where legitimate use ends and adulteration begins. It's a matter of cost versus durability.

REPORTS by the Forest Service of the Department of Agriculture show that an important index to the serviceability of a paint is the total pigment content. Most house paints serve best, experts from this agency say, when, in the finish coat the total pigment is between 27 and 32 percent by volume of the nonvolatile matter (pigment plus drying oil), or roughly about 75 percent by weight and in the undercoat between 33 and 40 percent or, roughly again, about 80 percent by weight.

No less important in a paint than the pigment is the liquid in which the pigments are suspended. This liquid in white and tinted paints consists of drying oil, volatile thinner, and drier. The drying oil is usually linseed oil, but suitable drying oils may also be made by mixing soybean oil or perilla oil with the linseed oil. In house paints the drying oil is usually either raw or boiled.

Bodied oil is added to paints sometimes to control the paint consistency. Too much of it gives the paint certain enamel qualities that make paint difficult to apply properly.

Tung oil is useful in white or tinted paints only in combination with other oils and then only in relatively moderate amounts.

The percentage of volatile thinner in paint gives warning to consumers

when there is too much bodied oil in the vehicle. When the volatile thinner amounts to more than 35 percent of the liquid part of the paint by weight, consumers are receiving a clear signal to watch out. Conversely no less than 85 percent of the liquid part of the paint by weight should be drying oil; that is, linseed oil with perhaps a small admixture of tung, perilla, or soybean oil.

Volatile thinners, whose function in a paint is to dilute it so that it spreads easily, evaporate after the paint is applied. Hence the volatile in their name. For this purpose most paint makers use the petroleum products, mineral spirits, and varnish and paint makers' naphtha. For consumers, however, the safest paint thinner is turpentine. Water, which is sometimes used for this purpose in soapy solution, serves the purpose badly and has no proper place in a paint.

PAINT IDENTIFICATION depends upon the consumer's ability to interpret the complicated statement of composition, or formula, often printed on the label. A number of States, following the lead of North Dakota, require all house paint sold within them to reveal their formulas on the label.

Indicating, however, that some consumers who want informative labeling have not been shouting from the housetops for nothing, the National Paint, Varnish, and Lacquer Association at its convention last year passed a resolution urging the paint industry "in consideration of the interests of consumers of paint products" to adopt formula labeling for most of the paints consumers buy.

Acting upon the resolution, the association soon afterward released a publication in which it indicated the kind of formula labeling it thought desirable.

In this pamphlet recommended standards of identity are defined for each paint ingredient. One of these definitions in particular will be of interest to consumers. "Water," the association said, "is the only word" that should be used to de-

scribe water. Do not use "moisture," "emulsion," "fed oil," or "any other term" for water.

Under the association's recommendation the composition of paint would be given by the weight of each ingredient.

This resolution and the recommendations for putting it into effect are a step forward. Better still than may even be paint labels in store for consumers which tell all in simple, non-technical language that can be understood by all. What most consumers want is a paint label which says that this paint is excellent, fair, or good for particular purposes.

TACKLING the complex problem of securing informative labeling for paint consumers, the Forest Products Laboratory of the United States Forest Service has finally worked out a practicable method of paint identification which answers the question consumers ask most frequently about paints, "Which one shall I use?" After studying the paint problem that have puzzled consumers for years, it is now proposing a classification of house paints by type and division, much as lumber has long been classified for sale by species and the species subdivided into grades.

The unfamiliar and technical-looking types for paints recommended by the Forest Service are in fact neither technical nor difficult. Arranged in five classes they enable every consumer to buy the kind of paint he needs to meet his purpose. Under the classification system the most important types of white-tinted house paints of high grade would be:

Type L: The pigment consists entirely of white lead except for small additions of colored pigments to make the tinted paints. This paint, if used exclusively, remains the best choice for house owners who wish to allow very long intervals, longer than the durability of any other white or tinted paint, to elapse between paint jobs. When used

(Continued on page 11)

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SHOPPING FOR PAINT

Continued from Page 9

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not a coating consisting wholly of white lead on woods that hold out well will still go through a long period of neglect during which it looks shabby but not unkempt. If such neglect, a white lead surface can easily be repaired without extra expense. Paint of type L is recommended also for houses in which moisture at times collects around the painted woodwork. The paint will blister as readily as other paints but, if the coating consists entirely of white lead paint, the blisters usually disappear when the wood dries out without leading to a subsequent peeling and scaling.

Type TL, high content of white lead. The pigment consists of titanium pigment and white lead, with a zinc oxide. This makes a very wet paint that behaves in many respects like paint of Type L.

Type TLZ, high content of white lead, medium content of zinc oxide.

Type TLZ, high content of white lead, low content of zinc oxide, plus titanium.

Type TLZ, medium content of white lead, medium content of zinc oxide, plus titanium.

Type TLZ, medium content of white lead, low content of zinc oxide.

Type TLZ, low content of white lead, medium content of zinc oxide.

The pigment in these five types of paint is a mixture of titanium pigment, white lead, and zinc oxide, in the relative proportions indicated.

The white paint in the "first grade" of most manufacturers of pre-

pared paint is now of one of these types. The TLZ paints remain

clearer and whiter through a greater part of their useful life than paints of other types. When tinted, however,

the color begins to fade fairly early.

When painting is done consistently with any one type of TLZ paint, the paint is of excellent durability that is free from checking or cracking. The relatively long time are

obtained. However, if repainting is delayed too long, conspicuous cracking and eventually curling and scaling develop which leave a difficult and uncertain surface for repainting. Paint jobs ordinarily should be spaced at 4 to 5 year intervals.

Type TZ, medium content of zinc oxide: The pigment consists of titanium pigment and zinc oxide, with no white lead. Paints of TZ type remain cleaner than any other house paints on the market. When applied under unfavorable conditions such as painting in unseasonable weather, they are less reliable in behavior than paints containing white lead. For use in localities where there is hydrogen sulfide in the air the TZ paints are advantageous because white lead blackens under these conditions.

Type LZ, very high content of white lead, high content of zinc oxide.

Type LZ, very high content of white lead, medium content of zinc oxide.

Type LZ, very high content of white lead, low content of zinc oxide.

Type LZ, high content of white lead, high content of zinc oxide.

Type LZ, high content of white lead, medium content of zinc oxide.

The pigment contains white lead and zinc oxide, with or without transparent pigments. Paints of the LZ types are apparently becoming obsolete as white prepared paints but they are still very widely used for tinted paints because they are thought to retain the original color somewhat longer than other paints. The relative seriousness of fading and soiling with dirt is largely a matter of individual preference.

Zinc sulfide pigments are used in many house paints but nearly all of them are manufacturers' second- or third-grade paints. On the other hand the great majority of cheap white and tinted paints contain lithopone or other zinc sulfide pigments. Though technical men may disagree about the suitability of zinc sulfide pigments for making

high-grade house paints, consumers may well assume that any house paint containing such pigments is not of the best quality. The zinc sulfide pigments, however, are entirely suitable for high-grade interior paints, particularly wall paints. They are widely used for this purpose.

APPLIED PROPERLY and given reasonable care, all of the paints listed here will give good service for specific purposes if made well. A paint surface, however, isn't quite as imperishable as the memory of a good woman. Periodically it must be renewed. When it is renewed it should be renewed with the same type of paint originally used. There are some exceptions to the rule, but consumers should make the exceptions only when their own experience has shown that one kind of paint goes over another kind of paint satisfactorily. To make sure that they do not waste their money, consumers should make a note of the formulas of the paints they use so that when they go buying again they can purchase the same kind of paint.

Experts in the United States Forest Service who have studied more than 500 cases of unsatisfactory paint service within the last few years learned that most paint failures that are caused by faulty application or maintenance come from (1) the painter's ignorance of the kind of paint he is using, (2) the failure to space painting in accordance with the kind of paint used, and (3) from using different kinds of paint for successive paint jobs.

CAREFUL STUDY of paint formulas on paint cans, and the keeping of careful records as to the date of each paint job and kind of paint used can help consumers reduce these failures. Consumers, however, will find their problems greatly simplified when a simple and practical method of classifying and grading paints is adopted by paint manufacturers.

19

FEBRUARY 13, 1939

0049-EXH-044460

I CERTIFY THIS TO BE A TRUE
AND CORRECT COPY OF THE
PRINTING OF THIS DOCUMENT

7 AUG 1966

Date

Signature

TT-P-56b
CANCELLATION
June 7, 1966

FEDERAL SPECIFICATION

**PRIMER COATING (PRIMER-SEALER), PIGMENTED OIL,
PLASTER AND WALLBOARD**

*This cancellation was approved by the Commissioner,
Federal Supply Service, General Services Administration.*

Federal Specification TT-P-56b, dated February 23, 1954, has been superseded by TT-S-179a,
Sealer Surface: Pigmented Oil, Plaster and Wallboard, dated June 7, 1966.

102-502-26

FSC 7920

U.S. GOVERNMENT PRINTING OFFICE: 1964

Exhibit 544

R-App 0030

Copy for E. F. Hickson

TT-P-56b

22 FEBRUARY 1954

SUPERSEDED
Fed. Spec. TT-P-56a
10 December 1945

FEDERAL SPECIFICATION

PRIMER COATING (PRIMER-SEALER), PIGMENTED OIL,
PLASTER AND WALLBOARD

This specification was approved by the Commissioner, Federal Supply Service,
General Services Administration, for the use of all Federal agencies.

1. SCOPE

1.1 This specification covers oil-type pigmented wall sealer for siding interior wall surfaces.

TT-W-251—White-Lead; Basic-Carbonate, Dry, Paste-in-Oil, and Semi-paste Containing Volatile Thinner.

UU-W-101—Wallboard; Composition.

2. APPLICABLE SPECIFICATIONS AND STANDARD

2.1 The following specifications and standard, of the issues in effect on date of invitation for bids, form a part of this specification:

Federal Specifications:

TT-D-651—Drier; Paint, Liquid.

TT-P-141—Paint, Varnish, Lacquer, and Related Materials; Methods of Inspection, Sampling, and Testing.

TT-P-143—Paint, Varnish, Lacquer, and Related Materials; General Specification for Packaging, Packing, and Marking.

TT-P-381—Pigments-in-Oil, (Tinting Colors).

TT-T-801—Turpentine; Gum Spirits and Wood (Steam-Distilled and Sulfate) (for Use in Organic Coatings).

TT-V-71—Varnish, Interior, Floor and Trim.

(Copies of Federal Specifications and the Index of Federal Specifications and Standards may be obtained upon application accompanied by check, money order, cash, or Government Printing Office coupons, to the General Services Administration, Business Service Center, Region 3, Seventh and D Streets, NW, Washington 25, D. C. This office will also honor deposit account numbers issued by the Government Printing Office. Prices may be obtained from the Index of Federal Specifications and Standards or from the GSA Regional Offices. Single copies of this specification and other product specifications required for bidding purposes are available without charge at the GSA Regional Offices in Boston, New York, Atlanta, Chicago, Kansas City, Mo., Dallas, Denver, San Francisco, Los Angeles, Seattle, and Washington, D. C.)

Military Standard:

MIL-STD-129—Marking of Shipments.

(Copies of Military specifications, standards, and drawings required by contractors in connection with specific procurement functions should be obtained from the procuring agency or as directed by the contracting officer.)

2.2 Specifications and standards applicable only to individual departments are listed in section 7.

ST1016-64-(210)

1

PHW:JEMc
V-1

September 24, 1928.

MEMORANDUM FOR FILES:

The attached letter of September 19, 1928 from the Board of Education, City of New York, should be jointly answered by Divisions V and IX. A great part of this letter is devoted to specifications for plaster. It is, therefore, suggested that Division IX answer the letter inserting the following:

In regard to the painting of plaster, we quote the following from page 44 of Technologic Paper 274:

"Too much emphasis can not be laid on the thorough drying of all plaster, concrete, cement, etc., before painting. It must be remembered that such structures when new contain much water, and unless sufficient time, under proper conditions of temperature and humidity, is allowed for thorough drying the water remaining in the wall will eventually seriously injure any paint coating. It should be pointed out that a wall may appear to be dry on the surface but be still damp in the interior. Even when properly dried, new structures of this kind may contain free lime; hence, it is advisable when the structure is less than a year old, even if it is known to be dry, to apply before painting, an aqueous solution of zinc sulphate (3 to 4 pounds crystallized zinc sulphate to 1 gallon of water). Allow this to thoroughly dry before applying paint."

It is noted that the plastered surfaces are painted two or three months after the plaster is applied. Apparently the plaster is frequently applied directly to the tile or brick wall. We very much doubt the advisability of attempting to paint plaster that is applied directly on brick wall. The plaster should have air space back of it so as to assure drying.

In regard to the painting procedure, it is noted that you propose to use lead and oil paint for the second and third coats. There is nothing better that can be used for these coats. The specification for the first or primer coat is indefinite. There is no reason why if lead and oil paint is used for the second and third coats, that it should not also be used for the priming coat.

PL04031

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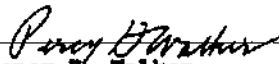
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Exhibit 559


-2-

The correspondent is referred to Technologic Paper 274, page 36, for formula for priming coat for plaster using white lead.

We are enclosing a copy of our Letter Circular 59, which lists the Bureau's publications on paint and varnish and gives information as to how they may be obtained.


Percy E. Walker.

Enclosures:
Letter.
LC 59

PL04032

0035AWP0000362D

REPRODUCED AT THE NATIONAL ARCHIVES

50092

OFFICE

CITY OF MILWAUKEE, WISCONSIN

DEPARTMENT OF PUBLIC WORKS

BUREAU OF BRIDGES AND PUBLIC BUILDINGS

SPECIFICATIONS

Consisting Of

Part 3 - Bureau Requirements

Part 4 - Detail Specifications

Governing The

PAINTING WORK

in the

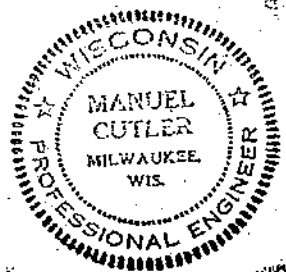
~~THE WISCONSIN MUSEUM~~

Located At

818 West Wisconsin Avenue

Milwaukee, Wis.

JUNE, 1946



472-001

00070

Exhibit 567

PART 4

DETAIL SPECIFICATIONS

For The

PAINTING WORK

In The

MILWAUKEE PUBLIC MUSEUM

Located At

818 West Wisconsin Avenue

Milwaukee, Wisconsin

(See also "General Specifications of the Department of Public Works")

FORM OF BID:- This Contractor shall submit an aggregate bid for the entire painting and wall washing in accordance with the schedule given on Pages 7, 8, 9, & 10 and these specifications.

SCOPE OF WORK:- This Contractor shall be required to furnish all materials, labor, equipment, scaffolding, etc. necessary to make a complete job of this work. He should carefully read the entire specifications, as well as the General Specifications of the Department of Public Works, as he will be required to do all the work specified herein.

The Contractor is advised to view the premises so as to familiarize himself with existing conditions.

MATERIALS:- The Contractor for the painting must provide all necessary materials of every kind for the work, including paint, oil, putty, varnish, etc., and all ladders, ropes, scaffolds, covering cloths and tools of every kind to properly do the work, promptly as wanted and without unnecessary delay.

All the materials of the several kinds must be of the best quality, and the work must be done in a skillful and workmanlike manner.

CLEANING:- All the plaster walls and ceilings, also all the wood work around the window and door openings, the sash and metal or wood shades attached to the sash, shall be washed clean before any painting is done.

This Contractor shall use a paint cleaning compound in the washing water so as to remove all dirt, grease, etc., from the walls and ceilings.

PAINTING:- Paint all plastered walls, ceilings, and beams, also panels in windows where now painted on the inside,

-5-

472-001

00071

one coat of "Dutch Boy", or equal, paste white lead and flatting oil as per formula.

The finish or color of the various rooms shall be as specified herein.

All painting to be in color as shown in schedule.

All paint shall be according to the following formula:

100 lbs. "Dutch Boy" or equal, white lead paste.
2-1/2 gals. "Dutch Boy", or equal, flatting oil.

Should white lead paste or flatting oil be unobtainable then this Contractor shall base his bid on an approved flat paint in the white, with introduction of the proper ground in oil colors to produce the colors as specified. Paint shall be equal to Pittsburgh Wallhide Interior Flat, S.W. oil type flat finish, or Patek Bros. Intensocote.

All inside woodwork around windows and door openings that have stained woodwork shall be washed and receive three (3) coats of paint in color as specified for walls.

HEATING AND VENTILATING GRILLES:- All grilles to be cleaned and steel brushed and to receive one good coat of black enamel.

WOOD BASE:- The wood base on walls, where visible in these rooms, shall be washed and painted three (3) good coats of paint in color as directed.

EXHIBIT CASES:- The exhibit cases along the walls, etc. will not be moved and the Contractor is to paint around them.

Cases shall be covered with suitable drop cloths during this work. These cases to be wiped clean and all dust removed, especially on the top, before painting around them.

Free standing cases, in the various rooms, will not be moved and the Contractor shall cover same with cover cloths.

The City will remove all hanging framed pictures which are now on walls. Wherever paintings or murals form part of wall surfaces or are permanently fastened to same, they will remain in place and the Contractor shall carefully cover same with drop cloths, so as to protect them during progress of the work.

SAMPLES:- Samples of each shade and class of finish shall be prepared immediately after contract is signed and shall be submitted for approval of the Museum Board or its representative.

All finished work at the building shall match the approved samples.

-6-

472-001

00072

25

City of Milwaukee, Wisconsin
Department of Public Works
Bureau of Bridges and Public Buildings

OFFICIAL NOTICE
INSTRUCTIONS TO BIDDERS
and
SPECIFICATIONS
GOVERNING THE CONSTRUCTION
of the
JOHNSTON EMERGENCY HOSPITAL
AT 8TH AND WINDLAKE AVENUES
in the
8TH WARD OF THE CITY OF MILWAUKEE

1929

0067

Exhibit 586

METAL WINDOW FRAMES:- All metal windows shall be painted two (2) coats, using lead and oil paint on exterior surfaces and metal trim enamel on interior surfaces.

PRESSED STEEL:- All pressed steel doors as specified in carpenter specifications will be furnished in baked enamel by the manufacturer of doors. All other pressed steel work, including all interior door frames, transom frames, transom bars, partition window frames, stops, mullions, etc. as specified in carpenter work shall be painted two coats of metal trim enamel.

KALAMEIN COPPER, BRONZE AND IRON:- All Kalamein copper and bronze work referred to in Sheet Metal, Copper Work and Kalamein Work specifications, shall be cleaned thoroughly and given one coat of raw linseed oil, allowed to dry and all surplus oil wiped off. All Kalamein iron doors and trim referred to in Sheet Metal, etc. specifications shall be painted two (2) coats of metal trim enamel. Paint clothes chute doors and frames two (2) coats of paint.

WOOD WINDOW FRAMES:- All wood window frames, including sash shall be painted two coats of lead and oil paint on all exterior surfaces and four coats on all interior surfaces including all casings, stops, etc. as shown on plans and sections. Four coat job shall consist of two (2) coats of enamel undercoater, one (1) coat composed of 1/2 undercoater and 1/2 enamel and a final coat of enamel (semi-gloss finish.)

Picture mouldings to be 4-coat job same as above.

WOOD DOORS, SASH, ETC.:- All wood doors and wood interior sash as specified in carpenter specifications will be delivered at the job, finished with one coat of stain, one coat of shellac and one coat of varnish, said finish included in carpenter contract.

After the doors and sash have been fitted, retouch with stain all parts where stain has been removed and give such parts a coat of shellac and one coat of varnish. After this retouching has been completed, apply one coat of varnish over all surfaces, including sides, bottom and top edges. Sandpaper between coats.

Last coat rubbed down with pumice stone and oil to a satin finish.

Finish all closet shelves fittings, hook strips and shoe shelves with one coat of stain, one coat of shellac and one coat of varnish. Finish all wood cabinets shown on plans and details with one coat of stain, one coat of shellac and two coats of varnish. Sandpaper between coats and rub down last coat with pumice stone and oil to an even satin finish.

PAINTING PLASTER WALLS, CEILINGS, PARTITIONS, ETC.:- See plastering specifications and plans for extent of plaster surfaces.

Previous to painting, all plaster surfaces shall be treated with a solution made by dissolving two pounds of zinc sulphate in one gallon of water. After the zinc sulphate has been applied, sufficient time must be allowed for the plaster to dry before applying priming.

All painting, except as otherwise specified, to be in three (3) coat work, that is a priming coat, second coat, and third coat, and shall be according to the following formula:

Priming Coat:

100 lbs. Dutch Boy or equal white lead.
7 gals. pure boiled linseed oil.
(or 7-gals. pure raw linseed oil and 3 pints pure drier.)

1 Gal. pure turpentine.

Second Coat:

100 lbs. Dutch Boy or equal white lead
2 to 3 gals. Dutch Boy or equal flatting oil.

Third Coat:

100 lbs. Dutch Boy or equal white lead.
2 to 3 gals. Dutch Boy or equal flatting oil.

The rooms shall be painted in colors as directed. The addition of all tinting materials shall be confined to the last two coats of paint to be applied. The body coat shall be tinted to approximately the required color; the finishing coat shall be tinted to final color selected by the Bureau of Bridges and Public Buildings.

The color shall be in three tones for each room, that is, a light color ceiling, deeper color for body, and a medium for trim.

Paint the plastered walls and ceilings with paint as specified above, in the following basement rooms: Lobby, Corridors, Stair Hall, Nurses and Doctors' Dining Room, Help Dining Room, Serving Room, Help Rest Room, Ladies' Toilet Room, Men's Toilet Room and Morgue. Also the ceilings of Kitchen, Food Wagon Room, Dishwashing Room and Preparation Room.

The plastered walls and ceiling of entire 1st, 2nd, and 3rd story rooms including stair walls and corridors shall be painted three (3) coats as specified above and as per formula, except such rooms as are otherwise specified.

The plastered walls and ceiling of basement laboratory shall be given two coats mill white undercoating, one coat composed of 1/2 undercoater and 1/2 of fume-proof Laboratory Enamel, and then one (1) coat of fume-proof Laboratory Enamel, making a total of four coats.

Paint Manual

WITH PARTICULAR REFERENCE TO
FEDERAL SPECIFICATIONS

By Percy H. Walker and Eugene F. Hickson

BUILDING MATERIALS AND STRUCTURES
REPORT BMS105

Issued October 11, 1945

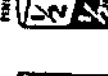


U. S. DEPARTMENT OF COMMERCE
Henry A. Wallace, Secretary
NATIONAL BUREAU OF STANDARDS, Lyman J. Briggs, Director

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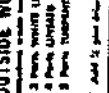
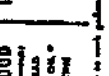
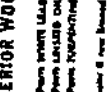
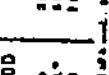
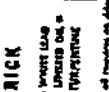
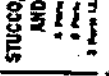
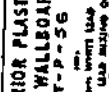
Exhibit 592

REPAINT WORK

OUTSIDE WOOD (INCLUDING WOOD TRIMMERS) FIRST COAT  	BRICK, STUCCO, CONCRETE AND STONE FIRST COAT  	INTERIOR PLASTER, WALL BOARD AND WOODWORK FIRST AND FINISH COATS (SLAT FINISH) 

NOTE: Add 1/2 gal. of white lead per gal. of paint. If more than 1 gal. of paint is used, the amount of white lead should be increased proportionately. The above formula is for use on new work. No other material should be used.

NEW WORK

OUTSIDE WOOD (INCLUDING WOOD TRIMMERS) FIRST COAT  	INTERIOR WOOD FIRST COAT  	BRICK FIRST COAT  	STUCCO, CONCRETE AND WALLBOARD FIRST COAT  

USE PROPER PRIMING COAT BELOW FOLLOWED BY TWO COATS MIXED AS PER DIAGRAMS ABOVE

FIGURE 1.—Volume-mixing directions for soft-paste white lead TT-R-5516.

process). Made by either process, it may differ in color and oil absorption. Hence, a purchaser should have an agreed-upon sample for color, color strength, and oil absorption. Zinc oxide alone with linseed oil gives a paint that chalks less readily than most other linseed-oil paints (see glossary) but dries to a hard film that tends to check and crack. It is added to the chalking type of pigments to harden the film, to help retain the color of light tints, and to retard the formation of mildew. Paints mixed-on-the-job may contain either zinc-oxide or low-leaded zinc-oxide (see glossary) pastes-in-oil. In general, not more than 1 lb of either type of zinc-oxide paste-in-oil should be used with 3 lb of white-lead paste or semipaste.

By weight, an average zinc-oxide paste-in-oil meeting the specification contains 83 percent of pigment and 17 percent of oil; by volume, 44 percent of pigment and 56 percent of oil; and 1 gal weighs about 25 lb.

(c) **Red Lead; Dry and Paste-in-Oil**
TT-R-191a, July 5, 1938
Amendment 2, May 28, 1943

Red lead is used in paint for priming iron and steel surfaces. Federal Specification TT-R-191a covers two types and three grades as follows:

TYPE I, DRY	TYPE II, PASTE-IN-OIL
Grade A.....85 percent.	Grade B.....85 percent.
Grade B.....85 percent.	Grade C.....87 percent.
Grade C.....87 percent.	

Type I, dry red lead, may vary somewhat in specific gravity, becoming slightly lighter as the true red lead increases. Grades A, B, and C have a specific gravity of about 8.9, and 1 gal of dry red lead weighs about 74 lb with a bulking value of about 0.0135 gal/lb.

Type II, paste-in-oil, has an average specific gravity of 6.37 and shows an average weight per gallon of about 45 lb. Formulas for mixing red-lead paints on the job are given in tables 6 and 8. The priming paint in table 6 weighs 25 lb/gal and has a PV of 30. The formulas given in table 8 produce paints that have a PV of 36 and dry somewhat harder and with less gloss, and allow recoating earlier than paints whose vehicle is high in oil (as in table 6).

Lead Poisoning in Young Children

By HUNTINGTON WILLIAMS, M.D., EMANUEL KAPLAN, Sc.D.,
CHARLES E. COUCHMAN, and R. R. SAYERS, M.D.

Lead poisoning in young children associated with eating lead-containing paint has been increasingly recognized until it ranks as one of the most common causes of child mortality due to poisoning. However, lead poisoning is not a reportable disease and, therefore, there is a lack of adequate morbidity data (1). The widespread occurrence of lead poisoning throughout the United States and Canada is evident from reports of cases in which eating lead-containing paints was mentioned as the cause of poisoning in infants and young children (2-37). Although 19 different communities are mentioned, most of the cases were from large cities such as Baltimore and Boston, where children's hospitals or local health authorities were especially interested in the problem. During the period 1931-40, the city of Baltimore alone reported 24.3 percent of all the child deaths from lead poisoning reported from the entire United States registration area (1). From January 1, 1931, to June 30, 1951, a total of 293 cases of lead poisoning was reported in Baltimore children. Of these, 83 died (2).

The most common cause of lead poisoning is

apparently the habit of chewing paint from cribs, toys, furniture, woodwork such as window sills, and the eating of painted plaster and fallen paint flakes. The tendency to put things in the mouth, though normal in the first year of life, is considered abnormal if continued into the latter part of infancy, and is referred to as pica, or perverted appetite (6, 30). Pica is the usual forerunner of lead poisoning.

Although pica does not exist on a seasonal basis, a striking number of lead-poisoning cases resulting from this habit occur in the hot summer months. For this, no satisfactory explanation is available, although several investigators have commented on the tendency for lead poisoning to occur in children during the warmer weather (2, 21, 26, 37-40).

Diagnosis, Prognosis, and Sequelae

Unrecognized plumbism, lead poisoning, in children may explain many obscure nervous conditions and convulsions of undetermined etiology (12, 22). Errors have been made in operating on cases presenting symptoms indicating a need for surgery but which were caused by lead intoxication (14, 31).

Lead poisoning is cumulative. Some weeks or months following the continued ingestion of small amounts of lead, symptoms begin to appear. Early symptoms may be only irritability, fretfulness, or disturbed gastrointestinal function characterized by lack of appetite, constipation, vomiting, or cramps. A secondary anemia with resulting pallor is often present. More severe intoxication results in lead en-

Dr. Williams is commissioner of health of the Baltimore City Health Department; Dr. Kaplan is chief of the division of chemistry; Mr. Couchman is director of the bureau of industrial hygiene; and Dr. Sayers, a medical director (retired) of the Public Health Service, is senior medical supervisor of occupational diseases. For several years he was director of the Bureau of Mines, Department of the Interior.

cephalitis due to increased intracranial pressure because of cerebral edema. The acute stages of the disease are manifested in changes in mental state, ataxia, persistent vomiting, muscle weakness or paralysis, delirium, stupor, coma, convulsions, and, not infrequently, death.

Diagnosis involves correlation of a history of paint-eating or pica with the physical findings, laboratory and X-ray data. The importance of eliciting a history of pica cannot be over-emphasized in the early recognition of the disease. Examination of blood smears often shows stippling of the red blood cells. Porphyrinuria is frequently found. The demonstration by roentgenogram of an increased density in the growing ends of the long bones is a cardinal sign of lead poisoning (34). In recent years, the quantitative determination of lead in blood or urine as an index of abnormal lead absorption has proved an invaluable aid in diagnosis (41, 42). Lead poisoning in children differs considerably from the disease in adults. Central nervous system involvement or encephalopathy, rarely seen in adults, is common in children, whereas peripheral neuritis, lead line on the gums, and colic are usually absent.

The prognosis in lead encephalitis in children is poor; the high mortality rate, as well as the incidence of severe, lifelong, residual nervous system injury, has been commented on by many investigators (25). The mental development of even the less severe cases may be seriously impaired (16).

Baltimore Experience

Thomas and Blackfan (3) of the Johns Hopkins Hospital were the first to point out in American pediatric literature the frequency of occurrence of lead encephalopathy in children. Subsequently, studies at the same institution provided pioneer information on the diagnosis (4, 26, 34, 42) and treatment (25, 40) of plumbism in infancy. The Baltimore City Health Department in 1932 began studies of nonindustrial lead poisoning in children in an investigation of cases resulting from the use of storage battery casings for fuel (43). Afterwards, all cases of lead poisoning brought to

the attention of the department were routinely investigated to ascertain the source of the lead.

Blood-Lead Laboratory Service

As an aid in diagnosis, since early in 1935, the bureau of laboratories of the Baltimore City Health Department has maintained a free routine analytical service for the quantitative estimation of lead in the blood of cases of suspected plumbism (41). The dithizone method is used. Especially prepared lead-free blood specimen collection containers known as "blood-lead outfits" are distributed to the local hospitals and physicians in the same manner as outfits regularly provided for specimens in cases of communicable diseases.

Since 1935, almost 3,000 specimens of blood from about 1,800 children have been tested for lead. Increase in this service, as well as the increased number of cases diagnosed during the last 4 years in contrast to the preceding 13 years, is shown in the accompanying table. Undoubtedly, the improved educational activities in lead poisoning prevention in recent years have been a prominent factor in this increase.

Comparison of blood-lead laboratory service tests and cases of lead poisoning in Baltimore children for the periods 1948-51 and 1935-47

Period	Number children given lead test		Diagnosed cases of lead poisoning	
	Total	Average per year	Total	Average per year
1948-51 (4 years) ---	1,007	252	166	41
1935-47 (13 years) --	772	59	161	12.4

Field Investigation

Field work, associated with a follow-up of the blood-lead laboratory service, has enabled the Baltimore City Health Department to acquire relatively accurate data concerning the incidence of lead poisoning in the community (1). A report of each blood analysis was forwarded to the bureau of industrial hygiene, which investigated cases in which the blood showed an abnormal absorption of lead. The

Lead poisoning cases according to month of report

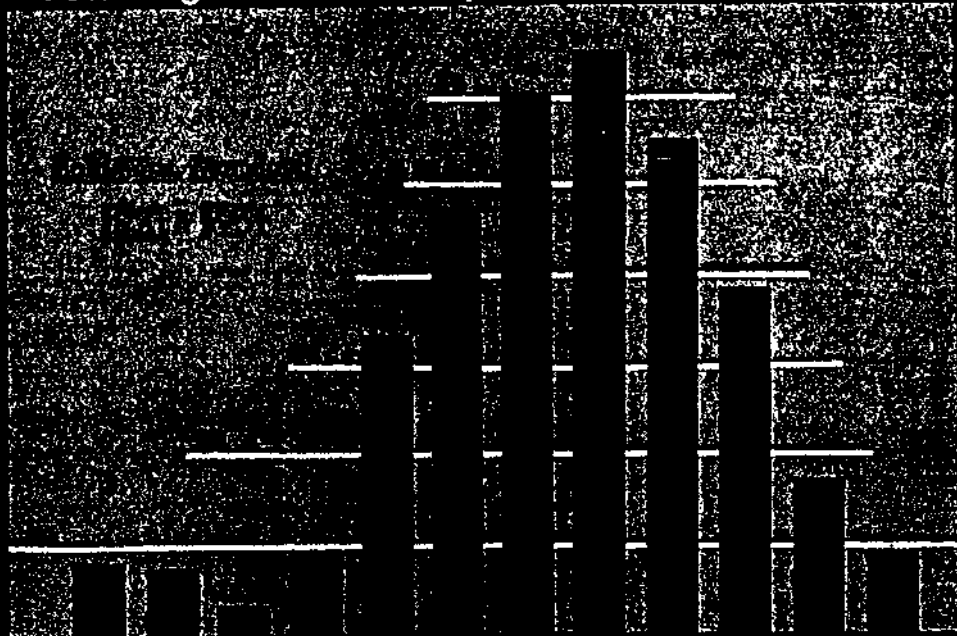


Figure 1. Lead poisoning cases reported in Baltimore, according to month of report, 1931-51.

upper limit of normal is considered to be 0.05 to 0.06 mg. percent of lead. Adequate clinical and laboratory data were usually available because nearly all of the children were diagnosed and treated in hospitals.

After learning the medical history, a field worker visited the home of the affected child to obtain pertinent information, particularly concerning exposure to lead. Almost without exception, the cause of poisoning was found to be pica associated with the ingestion of paint. This was confirmed at the time of the home visit by obtaining for analysis a sample of paint scrapings, approximately 0.5 to 1 gm., from surfaces where the child had chewed paint.

Lead Poisoning in Children

Over the past two decades, 347 cases of lead poisoning have been diagnosed in Baltimore

children. These do not include the storage battery cases referred to earlier. Of the 347 cases, 54 have been reported since June 30, 1951. A study of these cases has led to the discovery of interesting patterns in the seasonal incidence of the disease, age and color distribution of the children, and the types of houses involved. It is evident from figure 1 that more cases were reported in July and August than in any other months. There was no significant difference in incidence between sexes. Sixty percent of the cases occurred among children in their second year of life, at teething age, when they have a greater tendency to put things into the mouth. Only 2.3 percent of the cases were in children above 5 years of age.

The annual attack rate for the age segment under 5 years during the period 1931-51 was 7.5 times as high among the Negro population (71 per 100,000) as it was among the white

population (9.5 per 100,000). When the more recent experience of the past 4 years is considered, the attack rates for both white and Negro children as well as the difference between races are significantly elevated above the average experience cited. The high rates among Negro children are a problem of considerable public health significance since 30 percent of Baltimore's preschool population is Negro. The racial difference in incidence is believed to be due to environmental factors probably resulting chiefly from economic disadvantage.

For the past few years information has been collected on home ownership in neighborhoods where child lead poisoning cases occurred. Almost 90 percent of the houses were tenant-occupied. In the early years of the study some of the cases arose in well-kept property, but with the continued community education by press and radio, cases in this category have become relatively rare. The problem in Baltimore at present involves chiefly slum or blighted-area properties. The cases are concentrated in two areas which are of known slum status and where the houses are old and have had many coats of paint, usually lead paint, applied throughout several decades. A typical home where a case of lead poisoning occurred is shown in figure 2.

Methods of Prevention

Education and Publicity

In an effort to prevent lead poisoning, repeated public warnings about this child health hazard have been given by the Baltimore City Health Department in the press, by radio, and on television. The *Baltimore Health News*—mailed each month to over 10,000 persons, including 1,800 local physicians and 6,000 school teachers—devoted a number of issues (2, 46, 47) to the subject. The bureau of child hygiene issued a leaflet (45) entitled "Lead Poisoning in Children, a Disease You Can Prevent." The leaflet directs attention to 220 cases and 78 deaths from lead poisoning in Baltimore during the past 18 years, makes suggestions to parents for preventing children from contracting the disease, lists the warning sig-

nals to be watched for, and stresses the importance of early diagnosis and treatment.

It is not unusual now for a mother to take a child to a physician and to volunteer information on pica and suggest that the child may be suffering from lead poisoning.

The Public Health Nurse

One of the most promising advances in the prevention of child plumbism was the assignment several years ago of a public health nurse supervisor to investigate lead poisoning cases. With the knowledge gained by intimate association with the problem, the supervisor was able to interest other public health nurses. They not only make home visits and disseminate information in the most-affected areas of the city, but may take part in well-baby clinics, where mothers are told of the dangers connected with pica.

Lead Paint Removal

For the past 4 years, landlords of properties where lead poisoning has occurred have been notified, in accordance with the Baltimore ordinance on the hygiene of housing (44), to remove lead paint from the surfaces where there is flaking or where a child has chewed. Of 96 such notices during this period only 2 were not complied with in the time allotted. Both owners were summoned to the Magistrate's Court, where they were found guilty and fined; only then did they fully comply with the health department's orders. Many sanitarians on the health department staff who inspect property, primarily on the basis of other types of complaints or because of rodent and housing surveys, also require correction of a flaking paint condition in the notice sent to the property owner. The sanitarians are instrumental in the distribution to slum dwellers of the leaflet on the lead poisoning prevention (45).

Legislation

Because of the danger to small children, manufacturers of cribs and toys have for many years (9, 17, 48) used paints free of lead pigment.

For various reasons legislation against the use of lead paint has existed in widely separated jurisdictions for a number of decades. Germany has had a national law on the matter



Figure 2. House doorway in home of patient with lead poisoning.

since about 1900. Regulations prohibiting the use of lead-containing paints on toys, children's furniture, and for interior work have been enforced in France since 1917 (17). As early as 1922, the nations adhering to the International Labor Office in Geneva proposed a convention prohibiting the use of white-lead paint in interior painting of buildings as a health measure affecting painters (49). Since 1932, factory legislation in Ontario has required all lead-containing paints supplied to plants manufacturing children's toys and furniture to be so labeled (17, 48). No cases of lead poisoning related to chewing on new furniture or painted toys have been reported in recent times. When such objects are involved, the source of lead has been repainted furniture—parents frequently use lead-base paints for repainting jobs. Nevertheless, the Maryland State Legislature in 1949 enacted chapter 517 of the Acts of 1949, which

made it compulsory to affix a label to any toy or to any children's furniture decorated with paint or other material containing lead or any other poisonous substance, stating clearly the poisonous nature of the paint or decoration. Unenforceable, the law was repealed a year later.

On June 27, 1951, regulation No. 17 was adopted by the commissioner of health of Baltimore under the ordinance on the hygiene of housing. The text follows:

Interior Painting. No paint shall be used for interior painting of any dwelling or dwelling unit or any part thereof unless the paint is free from any lead pigment.

The wording was studied carefully so as not to prohibit the use of paints containing either lead driers, usually present in amounts corresponding to less than 1 percent of lead in the finished paint, or pigments contaminated with

traces of lead. The use of the term "lead-free paint" was purposely avoided, since it is doubtful if the usual commercial product could be made without having a detectable amount of lead present. This regulation has had pronounced salutary effect as shown by an increasing interest on the part of home owners, health agencies, and local paint manufacturers, some of whom have recently advertised paints free from lead pigment.

Lead Content of Paint

The health department's suggestions to parents interested in the purchase of "lead-free" paint emphasizes a selection based upon the labeled composition of the product. Although there is no Maryland law on the subject, many of the paints sold locally contain a statement of composition on the label. Such labels provide information on the presence of lead-bearing compounds and are adequate except in those instances where the terms "chrome yellow," "chrome green," or "chrome orange" camouflage the fact that these pigments contain substantial amounts of lead chromate. For this reason, when inquiry is made, it has been recommended that no yellow, green, or orange colors be used in refinishing articles of furniture intended for use by children unless the pigment composition as declared on the label clearly excludes the presence of lead.

Summary

Lead poisoning in children caused by ingesting lead from surfaces coated with lead-containing paint is apparently widespread throughout many parts of the Nation.

The disease has a high rate of incidence in the city of Baltimore, where it occurs in children of teething age living in old, run-down rented properties where lead paint had been used indoors for many years.

Public health education, coupled with a "lead consciousness" on the part of physicians and the pediatric clinics of local hospitals, and with a blood-lead laboratory service offered by the city health department has resulted in a marked increase in case recognition.

It is hoped that the application of principles

involving education and the enforcement of measures regulating the use of lead-containing paints will result in a material reduction and the eventual eradication of child lead poisoning in Baltimore City.

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REFERENCES

- (1) McDonald, J. M., and Kaplan, E.: Incidence of lead poisoning in the city of Baltimore. *J. A. M. A.* 119: 870-872 (1942).
- (2) Williams, Huntington: Lead poisoning killed 83 children. *Baltimore Health News* 28: 113-116 (1951).
- (3) Thomas, H. M., and Blackfan, K. D.: Recurrent meningitis due to lead in a child of five years. *Am. J. Dis. Child.* 8: 877-880 (1914).
- (4) Blackfan, K. D.: Lead poisoning in children with especial reference to lead as a cause of convulsions. *Am. J. Med. Sc.* 153: 877-887 (1917).
- (5) Strong, R. A.: Meningitis caused by lead poisoning in a child of nineteen months. *Arch. Ped.* 37: 532-537 (1920).
- (6) Ruddock, J. C.: Lead poisoning in children with special reference to pica. *J. A. M. A.* 82: 1682-1683 (1924).
- (7) McKhann, C. F.: Lead poisoning in children. *Am. J. Dis. Child.* 32: 386-392 (1926).
- (8) McKhann, C. F.: Lead poisoning in children. The cerebral manifestations. *Arch. Neurol. Psychiat.* 27: 294-304 (1932).
- (9) McKhann, C. F., and Vogt, E. C.: Lead poisoning in children. *J. A. M. A.* 101: 1131-1135 (1933).
- (10) Donnelly, H. H., Schutz, O. A., and Nimetz, A.: Chronic lead poisoning in early childhood. *Virginia Med. Monthly* 62: 88-89 (1935).
- (11) Nicholson, W. W.: Lead poisoning in children. *Kentucky Med. J.* 33: 180-181 (1935).
- (12) Cushing, H. B.: Diagnosis of lead poisoning in children. *Internat. Clin. 44th Series* 1: 189-195 (1934).
- (13) Gant, V. A.: Lead poisoning. Chicago, Industrial Health Book Co., 1939, pp. 39-42.
- (14) Bucy, P. C., and Buchanan, D. N.: The simulation of intracranial tumor by lead encephalopathy in children. *J. A. M. A.* 105: 244-250 (1935).
- (15) Kowaloff, I.: Lead encephalopathy. *Am. J. Dis. Child.* 61: 547-556 (1941).

- (16) Byers, R. K., and Lord, E. E.: Late effects of lead poisoning on mental development. *Am. J. Dis. Child.* 66: 471-494 (1943).
- (17) Ross, R. J., and Brown, A.: Poisonings common in children. *Canad. Pub. Health J.* 26: 237-243 (1935).
- (18) Parker, G. M. (Division of Industrial Hygiene, Toronto): Personal communication.
- (19) Taylor, H. W., and Schram, M.: Abdominal symptoms of lead poisoning in children. *Arch. Ped.* 53: 182-188 (1936).
- (20) Anonymous: Lead toys—lead paint—lead poisoning. *Safety Education* 22: 74 (1942).
- (21) Rapoport, M., and Rubin, M. I.: Lead poisoning. A chemical and experimental study of the factors influencing the seasonal incidence in children. *Am. J. Dis. Child.* 61: 245-255 (1941).
- (22) Netzeley, R. E.: Lead encephalitis precipitated by acute infection. *California West. Med.* 46: 306-308 (1937).
- (23) Golden, J. T.: Watch out for lead poison. *The Cincinnati Inquirer*, Sunday, August 12, 1951, p. 3.
- (24) Goettisch, E., and Mason, H. H.: Glycosuria in lead poisoning. *Am. J. Dis. Child.* 59: 119-128 (1940).
- (25) Ennis, J. M., and Harrison, H. E.: Treatment of lead encephalopathy with BAL (2,3-dimercaptopropanol). *Pediatrics* 5: 853-868 (1950).
- (26) Blackman, S. S., Jr.: The lesions of lead encephalitis in children. *Bull. Johns Hopkins Hosp.* 61: 1-51 (1937).
- (27) Dannenberg, A. M., Wideman, Arnold H., and Friedman, Paul S.: Ascorbic acid in the treatment of chronic lead poisoning. *J. A. M. A.* 114: 1439-1440 (1940).
- (28) Haverfield, W. T., Bucy, Paul C., and Elonen, Anna S.: The surgical treatment of lead encephalopathy. *J. A. M. A.* 114: 2432-2437 (1940).
- (29) Wilkinson, E. E.: Sodium citrate in treatment of lead encephalopathy. *Texas State J. Med.* 43: 442-445 (1947).
- (30) Stetson, C. A.: Pica: Its relationship to lead poisoning in children. *J. Maine Med. Assoc.* 38: 10-12 (1947).
- (31) Sibley, W. L.: Lead poisoning in infancy. A case in which two operations were performed on the stomach. *South. Surgeon* 15: 427-436 (1949).
- (32) Akelaitis, A. J.: Lead encephalopathy. A clinicopathological study. *J. Nerv. & Ment. Dis.* 93: 313-331 (1941).
- (33) McGovern, J. P., and Simmons, E.: Chronic lead poisoning associated with eosinophilia and splenomegaly. *Clin. Proc. Child. Hosp.* 6: 33-38 (1950).
- (34) Park, E. A., Jackson, Deborah, and Kajdi, Laslo: Shadows produced by lead in the X-ray pictures of the growing skeleton. *Am. J. Dis. Child.* 41: 485-499 (1931).
- (35) Selfert, H. E. (Virginia Bureau of Industrial Hygiene): Personal communication.
- (36) McKhann, C. F.: Lead poisoning. In Brenne-mann's *Practice of Pediatrics*, Hagerstown, Md., W. F. Prior Co., Inc., 1948, vol. 1, ch. 18, p. 1-8.
- (37) Feldman, H. T.: Lead poisoning. Salient features in its diagnosis and treatment. *Clin. Proc. Child. Hosp.* 7: 194-205 (1951).
- (38) Brienl, A., and Young, W. J.: The occurrence of lead poisoning among North Queensland children. *Ann. Trop. Med.* 8: 575-589 (1914-15).
- (39) Fukushima, M., and Matsumoto, H.: Statistics of 298 cases of infantile lead poisoning. *Oriental J. Dis. Infants* 3: 27-31 (1928).
- (40) Shelling, D. H., and Hopper, K. B.: Calcium and phosphorus studies. *Bull. Johns Hopkins Hosp.* 58: 197-211 (1936).
- (41) Kaplan, E., and McDonald, J. M.: Blood-lead determinations as a health department laboratory service. *Am. J. Pub. Health* 32: 481-486 (1942).
- (42) Blumberg, H., and Scott, T. F. Mc.: The quantitative spectrographic estimation of blood lead and its value in the diagnosis of lead poisoning. *Bull. Johns Hopkins Hosp.* 56: 276-293 (1935).
- (43) Williams, Huntington, Schulze, W. H., Rothchild, H. B., Brown, A. S., and Smith, F. R., Jr.: Lead poisoning from the burning of battery casings. *J. A. M. A.* 100: 1485-1489 (1933).
- (44) Baltimore ordinance on the hygiene of housing. No. 384, approved March 6, 1941, as amended by No. 902, approved March 29, 1943.
- (45) Lead poisoning in children—A disease you can prevent. *Baltimore City Health Department Leaflet*, 1949.
- (46) Lead poisoning in children. *Baltimore Health News* 14: 109-110 (1937).
- (47) Lead poisoning in children is preventable and can be fatal. *Baltimore Health News* 26: 122-124 (1949).
- (48) Jephcott, C. M.: Lead in certain colored chalks and their danger to children. *Canad. Pub. Health J.* 28: 391-393 (1937).
- (49) Hamilton, Alice: Recent changes in the painters' trade. U. S. Department of Labor, Division of Labor Statistics, *Bulletin No. 7*, 1937, p. 40.

Maximum Daily Intake of Lead Without Excessive Body Lead-Burden in Children

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Three hundred micrograms of elemental lead is considered to be the maximum daily permissible intake (MDPI) from all sources for children. As the average intake increases above this value, the entire amount cannot be excreted and accumulation in the body begins. This will increase progressively as long as undue ingestion continues. The MDPI has been established on the basis of levels of lead in the blood of nonexposed and exposed children including those with frank lead poisoning; results of experimental lead ingestion by adults; fecal lead output in children; initial biologic effects of increased lead intake; rates of increase in lead in the blood of exposed children, and sequelae of lead poisoning. The aim of the MDPI is prevention of disease and preservation of health in children.

Knowledge of the limiting value for lead intake is essential for the control of lead poisoning in children. Accordingly, an ad hoc committee was appointed to establish a daily permissible intake (DPI) that would serve as a reference base in developing measures for prevention of lead poisoning in children. Intake is defined as the amount of lead ingested in the diet and nonfood substances, and the amount retained in respiratory exchange. The conclusion of the ad hoc committee is that the value for a daily permissible total

lead intake from all sources for children is 300 µg.

The DPI is based upon such data as are available. These include levels of lead in the blood of nonexposed and exposed children and of those with frank lead poisoning; the results of experimental lead ingestion by adults; measurements of fecal output of lead in exposed and nonexposed children; the initial effects of increased lead intake; rates of increase in levels of lead in the blood of exposed children; and sequelae of lead poisoning.

Criteria for Establishing DPI

The DPI should preclude accumulation of lead (increase in the total body burden) in a child between 12 months and 36 months of age, as well as in older children. The most critical age for exposure of a child through ingestion of paint, putty, and other lead-containing nonfood materials is usually to 3 years of age.

Levels of lead in the blood of .00 ml of whole blood and above considered evidence of undue absorption of lead. Levels of lead in the blood are stated in terms of the

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Members of the ad hoc committee who assisted in the development of the proposed daily permissible intake were Roger Challop, MD, Bureau of Community Environmental Management, Department of Health, Education and Welfare (DHEW); Julian Chisolm, MD, Johns Hopkins University Medical School; Jane Lin-Fa, MD, Maternal and Child Health Service, DHEW; Herbert Stohlgren, PhD, Bureau of Occupational Safety and Health, DHEW; Herbert Stohlgren, PhD, Bureau of Occupational Safety and Health, DHEW; Lloyd Tepper, MD, Kettering Laboratory, Cincinnati; Barry G. King, PhD, Committee Chairman, Bureau of Community Environmental Management, DHEW.

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Exhibit 619

Table 1.—Some Comparative Measurements: Child and Adult

Biological Measurement	Columns				
	a	b	c	d, e, %	f, g, %
Age	2 yr	3 yr	Adult		
Weight (kg)	12.5	14.6	70	18	21
Height (cm)	87.5	96.2	170	51	57
Basal metabolic rate (cal/kg m hr)*	52	51	40	130	128
Caloric intake (Calories/24 hr)	1,280	1,190	2,700	47	52
Gastric juice (pH)	1.2-2.0	1.4-2.0	1.4-4.36	...	...
Ventilation (cu m/24 hr)	6	6	15	...	...
Pb diet (ug/24 hr) (from Tepper) ¹	...	...	225	...	...

*Cal = large calorie (kilogram calorie).

Table 2.—Estimated Child's Intake of Lead From Food, Water, and Air

Kehoe's estimate ¹	Mean (ug Pb)		Range (ug Pb)	
Pb in 50% adult diet, day	163		135 to 200	
Pb in respiratory intake, one half ventilation rate of 5 cu m/day at 2.4 ug Pb/cu m of air*	6		6 to 6	
Total	171		138 to 206	
Tepper's estimate ¹				
Pb in 50% adult diet per day	112		100 to 140	
Pb in respiratory intake, one half ventilation rate of 6 cu m per day at 2.4 ug Pb/cu m of air	6		6 to 6	
Total	118		106 to 146	

* Fifty percent of the retained inspired air.

weight of lead per unit volume or per unit weight of blood. Specific gravity of blood of normal children is from 1.043 to 1.050.

Levels of Lead in the Blood of Unexposed Children

The lead intake for unexposed children—that is, children living in dwellings free from hazardous sources of lead—is derived from their normal diet, and from air with a lead content of about 1ug/cu m to 3ug/cu m.

Concentrations of lead in the blood of these children have been reported to range from 15ug/100 ml of 40ug/100 ml of whole blood, with a median level of 25ug/100 ml whole blood. The mean reported for children in an urban population was

25ug/100 ml, and for rural children 13ug/100 ml whole blood. The question of normal concentrations of lead in the blood of children should be reviewed. It may be that the presently available data include measurements from biased samples. This is suggested by the recent finding of Tepper that the median level of lead in the blood of adult urban women is not more than 20ug/100 ml whole blood.

Estimate of Lead Ingested and Inspired in Unexposed Children

Some measurements are given for comparing metabolism in children and adults (Table 1). The caloric and food intake of 2- and 3-year-old children is approximately 50% of that of an adult. Kehoe reports that oral in-

gestion of lead by normal adults amounts to 300ug to 400ug/day. Based on the relative food intake, the oral intake of lead for a child would be 150ug to 200ug/day. Tepper, in a recent study, found that lead content of the diet of the adult is approximately one third less than observed in earlier studies. Based upon Tepper's observations, the average intake in the diet would be from 100ug to 140ug of lead/day. The lead content in the air may amount to 1ug cu m to 3ug cu m. The air exchange by children 12 to 36 months of age in a 24-hour period is estimated at about 6 cu m/24 hours, assuming a ventilation about twice the resting volume. The contribution of inspired air to the estimated totals for lead intake is relatively small—2.9% to 3.7% (Table 2).

On the basis of Kehoe's findings, Tepper's more recent values for lead content of diet, and a value of 2ug Pb/cu m air, the minimal values for total lead intake are approximately 106ug to 138ug/day. With the investigators' maximum values, the daily intake would range from about 146ug to 206ug/day.

If it is assumed that 90% of the ingested lead and 50% of the retained inspired lead are excreted, lead excretion in the feces would be expected to be 92ug to 143ug/day; fecal excretion for the range of maximal values would be from 135ug to 159ug/day. The latter values are in close agreement with the findings reported by Chisolm in 1956: 120ug to 175ug/24 hours, with a mean value of 132ug for unexposed children. Bartrop reported a mean normal value of 130ug per day for healthy children; the observed range was 125ug to 159ug/day.

Supplemental Lead Ingestion

Kehoe has reported the results of

experimental ingestion of lead on four subjects. In addition to the lead intake in a normal diet, daily doses of aqueous solutions of lead salts from 300 μ g to 3,000 μ g were administered for various periods of time. Findings on two other young adults who inadvertently ingested quantities of lead estimated to have been between 5,000 μ g and 10,000 μ g/day for a period of 30 days also have been reported by Kehoe. The estimated equivalent doses for children from 2 to 3 years of age are one half of those for the adult (Table 3).

The DPI is aimed toward the preservation of health as well as the prevention of disease. As the average intake increases above the DPI, the entire amount cannot be excreted so that accumulation of an excessive body lead burden begins and will increase progressively as long as undue ingestion continues. Chisolm states the following:

Arithmetic increases in blood-lead concentration above 40 μ g Pb/100 gm whole blood are correlated statistically with exponential increases in the daily urinary

output of delta-aminolevulinic acid (ALA) and with exponential increases in the "chelatable lead" as measured by the ethylenediamine tetra-acetic acid (EDTA) mobilization test for lead. There is a wide body of experimental and clinical data which indicates that the increase in ALA in urine is correlated with the inhibition of the activity of delta-aminolevulinic acid dehydratase (ALAD) in the tissues. Consequently, the increase in urinary excretion of ALA provides evidence of an adverse metabolic effect; namely, interference in porphyrin metabolism and in the formation of heme. Heme is the prosthetic group of hemoglobin and the respiratory enzymes; i.e., the cytochromes found in all tissues. This adverse metabolic effect is observed to increase as blood-lead

concentration rises above 40 μ g Pb/100 gm whole blood.

Such changes occur as total daily ingestion of lead is increased from 300 μ g to 650 μ g.

Observations on three groups of children exposed to sources of lead ingestion were reported by Chisolm and Harrison in 1956; fecal lead excretion was related to signs associated with increased lead intake. Eleven children, with lead levels in the blood less than 60 μ g/100 gm whole blood and known to have ingested lead or to have been exposed to sources of lead, had an average value of 582 μ g fecal

Table 3.—Experimental Data on Supplemental Lead Ingestion (From Kehoe*)

Added Supplement (μ g Pb/Day)	Total Lead Ingestion		Period of Ingestion, mo	Adult Lead in the Blood (μ g/100 ml)
	Adult (μ g Pb/Day)	Child (Estimated) (μ g Pb/Day)		
350	600	300	(indefinitely)	(no rise)
1,000	1,200	450	4	55
			9	65
2,000	2,300	1,150	12	70
3,000	3,300	1,650	4	80
			8	60
			24	75
				After chelation
	5,000	2,500	1	80

Table 4.—Increases and Intervals Between Measurements of Lead in the Blood

Initial Measurement Range μ g Pb/100 ml Whole Blood	Second or Third Measurement							
	Range 50 μ g-75 μ g Pb				Range 80 μ g-110 μ g Pb			
	No. Cases	Interval, mo	Change μ g Pb/100 ml Blood	No. Cases	Interval, mo	Change μ g Pb/100 ml Blood	No. Cases	Interval, mo
<40	1	11 $\frac{1}{2}$	(32 - 53)	2	5 $\frac{1}{2}$	(25 - 89)	1	1
40-49	2	1 $\frac{1}{2}$	(46 - 61)	12		(94 - 110)		
50-59	2	1	(75 - 77)	5	3	(58 - 110)	5	2
		6 days	(60 - 79)		1	(62 - 110)	1	
		4 $\frac{1}{2}$	(50 - 90)		1	(56 - 110)	7	
		4 $\frac{1}{2}$	(74 - 81)		7 $\frac{1}{2}$	(59 - 89)	2 $\frac{1}{2}$	
		3 $\frac{1}{2}$	(62 - 110)		3	(59 - 92)	1 $\frac{1}{2}$	
					3 $\frac{1}{2}$	(55 - 110)		
					2 $\frac{1}{2}$	(53 - 93)		
					5 $\frac{1}{2}$	(59 - 91)		
60-69				1*	1 $\frac{1}{2}$		5	4 $\frac{1}{2}$
								6 days
								6 days
								2
								11 $\frac{1}{2}$
								10 $\frac{1}{2}$
>110							2*	
								8

* Increase within same range.

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lead per 24 hours; the median value was 451 μ g and the range 37 μ g to 1,830 μ g/24 hours.

A second group of five children with levels of lead in the blood greater than 60 μ g/100 ml—roentgenographic evidence of lead in bones and positive qualitative tests for coproporphyrin in urine—had a mean fecal excretion of lead of 2,160 μ g/24 hours with a median value of 1,110 μ g and a range of 116 μ g to 9,600 μ g/24 hours. The third group included six children with lead intoxication either without encephalopathy or with mild or severe encephalopathy. The fecal lead content of this group ranged from 3,040 μ g to 10,400 μ g.

Baltrop published case reports on three children, with histories of pica of four to six months' duration, admitted to the hospital for treatment of clinical lead poisoning. The fecal lead in the first stool of two of the children was 1,900 μ g and 1,300 μ g; the lead levels in the blood were 85 μ g/100 gm whole blood and 68 μ g/100 gm whole blood, respectively. In the case

of the third child, pica had ceased five days before admission. The lead content of the first and third specimen following admission was 170 μ g and 370 μ g, respectively. His level of lead in the blood was 92 μ g/100 gm.

Mild symptoms compatible with symptomatic lead intoxication may be found in the presence of lead values between 60 and 80 μ g Pb/100 ml whole blood. Minimal to severe brain damage and serious residual effects can occur with levels greater than 80 μ g/100 ml; this is clearly documented in cases of acute encephalopathy. Impairment of health possibly including adverse effects on the developing nervous system may occur at lower levels, particularly in the presence of other medical conditions; eg, anemia.

Mean and minimum blood-lead levels and medical conditions in children associated with these levels have been observed by Chisolm in his studies.

Three factors must be considered in the evaluation of lead levels above 40 μ g/100 ml. If the child has anemia

the health consequences at any given level of blood-lead may be greater than those affecting a child with a normal hemoglobin value. For example, a lead level of 40 μ g/100 ml in the blood of a child whose red blood cell volume is reduced to half of its normal level may be equivalent in clinical effect to a higher lead level in the blood of a child with a normal red blood cell volume. Anemia is a condition frequently resulting from, or associated with, lead poisoning. The second and third factors are even more significant.

It is important to note that increases in A.L.A. coproporphyrin and chelatable lead are exponential with respect to lead concentration in the blood. In consequence, the margin of safety decreases at an exponential rate as lead concentration in the blood increases arithmetically.

The third factor is the rapid rate of increase in lead level in the blood that can occur. This is illustrated by data from Chisolm's studies (Table II). The data result from analyses performed by the Baltimore City Health Department using the dithizone technique. The maximum analytic variation between pairs of approximately 40 blind duplicate samples was 6 μ g Pb/100 gm whole blood. The standard deviation between pairs has been $\pm$ 2.5 μ g Pb/100 gm whole blood. Further, when the data were being collected, only rises of 10 μ g Pb/100 gm whole blood or greater were included.

Intoxication	Mean, 176 μ g/100 ml blood low, 63 μ g/100 ml (41 children)
Mild encephalopathy	Mean, 328 μ g/100 ml; low, 88 μ g/100 ml (69 children)
Severe encephalopathy	Mean, 336 μ g/100 ml; low, 100 μ g/100 ml (31 children)
Fatalities	Mean, 327 μ g/100 ml; low, 138 μ g/100 ml (99 children)

References

1. Steinfeld JL: *Surgeon General's Policy Statement on Medical Aspects of Childhood Lead Poisoning*. Public Health Service, 1970.
2. Robinson NJ, Karpinski FE, Brieger K: The concentration of lead in plasma, whole blood and erythrocytes of infants and children. *Pediatrics* 24:792-797, 1958.
3. Haggerty CJ, Noeland RA: An inquiry into certain aspects of lead poisoning in children and community problem. Department of Pediatrics report, Marquette Medical School, 1949.

4. Tepper LB: Statement before the DPE ad hoc committee meeting, Cincinnati, 1971.
5. Kebab SA: Harben Lectures. *J Roy Inst Public Health* 24:31-47, 101, 129-143, 177-203, 1961.
6. Chisolm JJ Jr, Harrison RE: The exposure of children to lead. *Pediatrics* 18:942-957, 1956.
7. Baltrop D, Killala NJP: Fecal excretion of lead by children. *Lancet* ii:1017-1018, 1967.
8. Chisolm JJ: Statement before the DPE ad hoc committee meeting, Cincinnati, 1971.

SURGEON
GENERAL
1931

0049-EXH-044255

Exhibit 632

CHILD WELFARE

it comes from the consciousness of possessing the ability requisite for what one undertakes. Civilization today rests upon self-confidence. The whole principle of an attractive personality lives in this sentence—"A fine manner pleases; a coarse manner repels." Cultivate the art of being agreeable; and cultivate self-confidence. These qualities will help you as nothing else will; they will call out your success qualities. Long after teachers and classmates have forgotten the marks you attained and long after the diploma has faded you will be remembered for your school personality.

There are so many things to write about. Girls and how they help and hinder a boy in his studies; automobiles which can transport you to school or carry you to the devil; studies which will help you vocationally to earn a living and studies which will assist you avocationally; leisure hours and your training for them; the choice of a vocation with all the problems of whether you want a profession or business career;—there's no end to the things we could talk about.

YOUR LOVING DAD

§ § §

Important Art Commission

SOME months ago President Hoover appointed a research committee to study social conditions in the United States. Among the divisions making up such a study is one on the Arts, under the general direction of Frederick P. Keppel, President of the Carnegie Corporation. One essential phase of the art study deals with art education. Mr. Keppel has called upon Mr. Royal D. Farnum, Educational Director of the Rhode Island School of Design, to assist in the educational side of the arts. The whole study will be completed in 1932, with the results to be published in a comprehensive report supplemented by monographs. The undertaking is regarded as "the most significant exploration of the social trends ever undertaken in this country."

—From *Everyday Art*.

25 (1932) 608

Lead Poisoning from Toys

THE SURGEON GENERAL COUNCILING of the United States Public Health Service has been informed of the occurrence of occasional cases of lead poisoning in infants and children, apparently due to biting lead paint from cribs, toys, etc. The Public Health Service has previously called attention to this possible source of lead poisoning, but it is likely that more cases occur than become known. Children with perverted appetites would be particularly likely to encounter the hazard. Though lead paint has wide fields of usefulness, the painting of babies' toys and cribs is not one of them. Generally manufacturers of these articles are seeing to it that lead paint is not used for this purpose, but warning is necessary that parents, especially in repainting cribs, should use paint which are free from lead, namely, quick-drying lacquers or enamels sold for interior use.

Although in recent years it has been found that the human body takes up lead from its surroundings much more frequently than was formerly supposed, and although there are still important industrial sources of lead poisoning which need correction, the incidence of lead poisoning does not seem to be on the increase. The recent development of quick-drying lacquers and enamels has doubtless kept down the number of cases of this disease. One of the most prolific sources yet remaining is the painter's trade, and it is believed that by the dust arising from scraping or sandpapering dry paint may be more important than brush painting. Even the hazard in removing paint may be avoided by the use of new methods.—United States Public Health Service.

FRESHLY baked bread is not as readily digestible as stale bread, says Hyatt. The fresh bread remains in a solid form when it is swallowed, and not so much of its face is exposed to the digestive secretions.

June, 1921

Ca
Fore.

By R

SCHOOL mellow fragrant maples to hundreds of blossoms. Overhead birds, mingle squirrels; and unsheathing of In this happy Toronto for six months to October children be fifteen years are While open-air Toronto has the High Park at the other in Both schools Education and the week (ex-holidays. All clear except on tr

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AM. JOURNAL OF
DISEASES OF CHILDREN
1914

Vol 8
377-380

RECURRENT MENINGITIS, DUE TO LEAD, IN A
CHILD OF FIVE YEARS*

HENRY M. THOMAS, M.D., AND KENNETH D. BLACKFAN, M.D.
BALTIMORE

Recurrent
The case was seen in Dr. Howland's service in the Harriet Lane Home of the Johns Hopkins Hospital. It was that of a boy, between 5 and 6 years old, who was admitted Aug. 22, 1913, having been transferred from the Home of the Friendless in a state of coma.

Little was learned of his early history except that he had had in the eight preceding months two attacks of otitis media, and that for two years he had been subject to rather frequent and severe bleedings from the nose.

He seemed as well as usual up to five days before admission, when he began to complain of pain in his face and head, to be restless at night, and to look ill. The next day stiffness of the neck was noted, and the child vomited on several occasions. He became rapidly worse, and convulsions and coma developed the day before he was brought to the hospital.

On admission he was comatose. His head was retracted, and his arms and legs were extended and spastic. Kernig's sign was, however, not present. There were recurrent, general convulsions. The left eye was deflected inward. The child's temperature was practically normal. There was a leukocytosis of 27,600.

Spinal puncture was done on four occasions—directly after admission, the next day, then in three days, and after an interval of two weeks. The results of these punctures were all very similar, except that the pressure, which was very high on the first occasion, was not above normal on the subsequent punctures. The fluid was always clear and sterile. It contained from twenty to forty cells, mostly mononuclear. It was positive to the Noguchi globulin test, reduced Fehling's solution, and the Wassermann reaction was negative.

The von Pirquet skin test was negative.

The right optic nerve was swollen and its edges obscured. There were numerous hemorrhages in the retina about it. The left optic nerve was practically normal. As noted on admission, the left external rectus was weak. There was nothing else abnormal discovered in the nervous system.

The convulsions, the rigidity of the neck, the optic neuritis, and the spinal fluid findings, suggested a meningitis. It was considered to be a serous meningitis of unknown cause.

Improvement began after the first lumbar puncture, and in a week the patient seemed nearly well, complaining only of headache at night, abdominal pain, and occasionally vomiting. He was discharged September 20, after an examination which revealed nothing abnormal. The spinal fluid, however, showed an increase in the cells and a positive globulin test. The right optic nerve was reported by the ophthalmological department to be quite normal, although there was some difference of opinion in regard to this.

The child was returned to the care of the Home of the Friendless, but was seen from time to time in the out-patient department. The condition remained excellent for five months.

In January, 1914, he had a number of attacks of epistaxis, for which he was treated in the throat department.

*Read at the Annual Meeting of the American Neurological Association held at Albany, New York, May 9, 1914.

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On March 1 he began to suffer from headache and vomited. The convulsions recurred and in two days he was again in coma. He was readmitted to the Harriet Lane Home on March 3, in a condition which was almost identical to that of his first admission, six and a half months before. The neck was stiff but there was no definite Kernig sign, and the limbs were not held so stiffly as on the former admission. There was choked disk on the right side, with some retinal hemorrhages; on the left side the disk was sharp-cut—indeed, looked slightly atrophic. He had moderate anemia, hemoglobin of 55 per cent., red blood-cells 4,480,000, white blood-cells 23,100. Lumbar puncture yielded fluid very similar to that found at first admission. The fluid, however, did not seem to be under any noticeably increased pressure.

The recurrence of such a condition naturally aroused a great deal of interest, and the case was studied most carefully from every side. Nothing, however, was discovered that threw light on the etiology, until our attention was attracted to a slight discoloration about a tooth, and a more careful inspection revealed the presence of a very fine but typical lead-line on the gums surrounding many of the teeth. The blood showed well-marked stippling of the red cells (Grawitz's granules), and confirmed us in our view that poisoning by lead was the cause of the trouble.

The urine was examined but no lead was demonstrated in it.

We were much puzzled as to the source of the lead, until he was found with his mouth covered with white lead paint which he had bitten from the railings of his crib. Before this discovery we had investigated the conditions and the other inmates of the institution in which he lived and had found nothing that aroused our suspicion, but the fact that the white paint on the patient's bedstead in the dormitory was chipped off much more than from the others.

After we knew of his peculiar habit, we found that he would gnaw any painted object unless he was most carefully watched.

In this second attack he developed a weakness of the external rectus muscle—this time of the right eye—three or four days after his entrance to the hospital, and he complained from time to time of abdominal pain.

The patient again made a remarkably quick and uneventful recovery. He was discharged on May 1, having been up and around the ward for more than a month, quite recovered except for the persistence of an increase in cells and positive globulin reaction in the spinal fluid.

He remained in good health for three weeks when on May 25 he had a severe convulsion and died. No post-mortem examination was obtained.

Lead-poisoning in children does not seem to be very common, although it is well recognized. That there is a special form of meningitis due to lead has been insisted on only by the French observers. That lead affects the central nervous system has been long known, and in certain fatal cases of lead encephalopathy anatomic changes have been noted in the meninges together, at times, with increase of the fluid in the ventricles or in the subarachnoid space. Felix Aussendorf¹ has collected reports of a number of such cases, among them Spiller's² important case.

French clinicians, true to their historic interest in lead poisoning, have, in the last ten years, been much occupied as to the effects of the metal on the brain and its coverings. Among the numerous articles

1. Aussendorf, Felix: Zur Kenntnis der pathologischen Anatomie des Zentralnervensystems bei Encephalopathia saturnina. Inaug. Diss., Leipzig, 1911.

2. Spiller: Jour. Med. Research, August, 1903, x, No. 1.

THOMAS AND BLACKFAN—LEAD POISONING 379

those written by Mosny, in association with several collaborators, are the most important. In the chief of these, Mosny and Malloizel³ give the histories of forty-seven cases of lead-poisoning, mostly from their own experience, in almost all of which lumbar punctures were done and the cerebrospinal fluid studied.

They point out that in acute lead colic there is very generally evidence of involvement of the meninges as shown by the fact that the cerebrospinal fluid contains an increased number of cellular elements, mostly lymphocytes. In these so-called abortive cases (*la méningite fruste*) the only symptom of such meningeal irritation may be headache, but the severity of this is not always proportionate to the laboratory findings.

In chronic lead-poisoning they rarely found any change in the cerebrospinal fluid.

In other cases classed as acute lead meningitis, the symptoms of involvement of the central nervous system are pronounced. In these the meningeal reaction, as revealed by lumbar puncture, is always very marked, persistent, and may be as intense as in tuberculous meningitis. It is in this group that our case would be classed, together with many cases of the so-called lead encephalopathy.

Mosny and Malloizel feel that the term "lead encephalopathy" is far too general, and they have attempted to divide the cases into more definite clinical groups, in all of which, however, there is more or less evidence of involvement of the meninges, as shown by the results of spinal puncture.

It is to be regretted that there is no record in their excellent article of any ophthalmoscopic examination.

Plate⁴ reports a typical clinical case of lead meningitis in a young man of 20. Four lumbar punctures were done, giving a decreasing cell-count from 90 to 14. The author thinks his is the first case reported in Germany.

Mass⁵ reports a number of cases in which the diagnosis of acquired hydrocephalus, due to serous meningitis, was made, and in which he thought he could assume lead as the cause. These cases were seen in the chronic stage or at necropsy, and in none of them was lead proved to be more than a possible etiological factor.

Involvement of the optic nerves and of the motor nerves of the eyeballs in the course of lead-poisoning, although certainly not fre-

3. Mosny and Malloizel: *La méningite saturnine*, 1907, xvii, 506.

4. Plate: *Ueber einen Fall von Meningitis saturnina*, München, med. Wchnschr., 1913, ix, 2343, 2345.

5. Mass: *Ueber eine besondere Form der Encephalopathia saturnina (Meningitis serosa)*, Monatschr. f. Psychiat. u. Neurol., 1911, xxx, 207.

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quently noted in America, is well known. Wilbrand and Saenger⁶ have collected reports of most of the cases. It would seem that optic neuritis may occur primarily, or through the direct toxic action of lead on the nervous system, or by the production of interstitial neuritis, or through the alterations of the blood-vessels in the retina and optic nerves, or secondarily through intercranial changes or changes in the kidney. Paralysis of one or more of the external eye muscles is not infrequently associated with optic neuritis, or may occur alone.

J. Lockhart Gibson⁷ has for a number of years repeatedly called attention to a most remarkable group of cases seen in children of Queensland, Australia. The case which we have reported seems closely allied to these. In that country children in general seem peculiarly liable to lead-poisoning, and perhaps especially to its effects on the eyes. Gibson has seen as many as nine cases of what he calls plumbic ocular neuritis in a single year. This condition usually affects children below the age of 8, and is characterized by the development of squint, due to weakness of one or both external recti, and associated with marked optic neuritis. There is often no other symptom of lead-poisoning, but at times more serious symptoms of lead encephalopathy are present. Gibson believes the source of the lead to be usually due to the children's getting dried paint from the railings of long-painted verandas or garden fences on their hands and then into their mouths. Children who suck their fingers or bite their fingernails are more liable to the disease. He regards the condition as a neuritis, but points out in his last article the great importance of an early lumbar puncture, and the almost constant finding of an increased pressure in the cerebrospinal fluid. He regards the withdrawal of the fluid as the essential therapeutic measure, surely indicating that meningeal irritation must be an essential factor in the condition.

We have found no record of any examination of the withdrawn fluid, either chemically or microscopically.

The case which we have reported, the results of lumbar punctures in acute lead colic, the results of certain of the autopsies of patients dying of lead encephalopathy, the cases reported by Gibson, all force us to the conclusion that lead appears to have a special effect on the meninges and the central nervous system, and that it may not infrequently be the unsuspected cause of so-called serous meningitis.

1228 Madison Avenue—Johns Hopkins Hospital.

6. Wilbrand and Saenger: Die Neurologie des Auges, 1913, v.

7. Gibson, J. Lockhart: Australian Med. Gaz., 1897 to 1912; Brit. Med. Jour., 1908.

Cerebr

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TEN RULES FOR HEALTHFUL LIVING.

Do you want to be healthy?

By following these rules most everyone can become and remain healthy:

1. Food—Milk, a quart a day for children; cereals; vegetables every day, including leafy ones such as lettuce, spinach, beet tops, to get the necessary vitamins; fruits every day; eggs; meat in moderation; sweets in moderation; 8 glasses of water a day.
2. Posture—Stand and sit straight; stand tall, keep head up, chin in, chest out, abdomen in, back straight, shoulders back; walk largely on balls of feet, with feet straight—not turned out.
3. Exercise—Enough every day to sweat freely. Walk 3 miles a day if you can't do anything better. Play in the open air.
4. Rest—When tired. Never eat a hearty meal when tired. Sleep at least 8 hours with bedroom windows open.
5. Mouth—Brush the teeth after meals and at bedtime. Keep the mouth clean. Salt and water is a good mouth wash.
6. Bowels—At least one good movement a day, preferably after breakfast. Many a headachy, ailing individual owes his trouble to constipation. Coarse cereals, vegetables, fruits like apples, prunes, etc., and plenty of water will usually ward off constipation. Don't use drugs.
7. Baths—A cold bath every day if it makes you feel good, otherwise a tepid bath; a warm cleansing bath once a week.
8. Clothing—Suitable to the season: It should not be too heavy.
9. Communicable Disease—Practice good health habits; avoid the careless spitter and sneezer, the common drinking cup or towel; eat and drink only clean foods. Keep your fingers away from your mouth.
10. Mental Hygiene—In many ways most important of all. Refuse to worry and hurry. Be calm. Control your emotions.

Be Cheerful. Be Friendly. Be Independent.

Follow Emerson's advice and work with your own hands, stand on your own feet, and think your own thoughts. Thus you will acquire poise, the truest sign of a real man or woman.

MASSACHUSETTS DEPARTMENT OF PUBLIC HEALTH.

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MONTHLY BULLETIN HEALTH DEPARTMENT



CITY OF BOSTON

FRANCIS X. MAHONEY, M. D., Health Commissioner.

Communications relating to this publication should be addressed to the
EDITOR MONTHLY BULLETIN, HEALTH DEPARTMENT, BOSTON.

VOL. 10. 2

BOSTON, NOVEMBER, 1927.

No. 11

VITAL STATISTICS FOR MEDICAL MEN.

(The following is the first of a series of articles and notes on vital statistics by Dr. Frederick L. Hoffman, consulting statistician, that will appear in every issue of the "Bulletin." These articles will be so written as to make them readable and attractive to medical men, public health workers, and laymen, and in such a manner that they will show their direct relation and value to the prevention of disease and prolongation of life.)

The Essential Purpose.

Vital statistics serve essentially the purpose of measuring with approximate accuracy the collective experience of large numbers of people in matters of life, death and survival. They are represented chiefly by statistics of births and deaths collected by authority, primarily for legal purposes to identify the person, but secondarily for public health and medical purposes in the furtherance of efforts to prevent disease and to prolong human life.

The Basic Data.

The basic data in vital statistics are certificates of births and deaths which have a legal status before the courts in a variety of judicial proceedings. They are usually made out by the attending physician who certifies to the occurrences of births and deaths and they therefore involve a function of the greatest possible responsibility.

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SUMMARY.

Cases under investigation October 1, 1927	48
New case reported during month of October	73
Total	121

DISPOSITION OF CASES.

Placed under treatment	26
Unable to locate	27
False address given	10
Moved out of town	4
Under investigation October 31, 1927	54
Total	121
Visits by investigator	282

VENEREAL COMPLAINTS AND SOURCES OF INFECTION.

Placed under treatment	2
Unable to locate	4
Total	6

LEAD POISONING IN EARLY CHILDHOOD.

For this year there has been reported to this department on death certificates four deaths of children from lead poisoning, all such causes of death having been certified by the medical examiner. These deaths were due to a childhood habit of sucking paint from cribs with the consequent ingestion of lead and subsequent lead poisoning and death. It would thus be well for mothers and others entrusted with the care of children in cribs to watch for this common habit in early child life and to take steps to correct it wherever it is found.

A clean cloth or other clean material attached to the side of the crib or other part which a child with this habit is likely to reach with its mouth is a good preventive of this paint-sucking habit. The cloth or other material should be attached in such a temporary manner that it will readily admit of being easily changed so that it may at all times be kept clean.

Here is one cause of death in early childhood that can positively be prevented if we watch the habits of the child with its crib, high chair, or other painted article which comes within its reach.

(264)

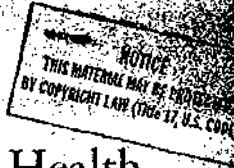
DIVISION OF TUBERCULOSIS, OCTOBER, 1927.

Districts.	Total Newborn Cases at Clinics.			Total Newborn New Cases.		
	Adult.	Children.	Total.	Adult.	Children.	Total.
South End	274	179	453	44	31	75
East Boston	33	178	211	18	18	36
North End	98	79	177	7	4	11
West End	92	88	180	8	8	16
South Boston	87	46	133	19	24	43
Hyde Park	29	26	55	8	7	15
Codman square	20	11	31	8	—	8
Charlestown	10	8	18	3	2	5
Old Town Hall	15	3	18	8	—	8
Total	640	603	1,243	103	80	183

Districts.	Van Pelt.	Wasserman.	Lamp.	X-Ray. (Recommended.)
South End	22	33	79	89
East Boston	9	9	20	29
North End	4	9	29	23
West End	8	3	3	4
South Boston	15	7	3	26
Hyde Park	—	3	—	—
Codman square	—	3	—	—
Charlestown	—	—	—	—
Old Town Hall	2	4	—	3
Total	57	60	148	143

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Blood Lead Determinations as a Health Department Laboratory Service*

EMANUEL KAPLAN, Sc.D., AND JOHN M. McDONALD,
M.D., D.P.H.

Chief, Division of Chemistry, Bureau of Laboratories; and Director, Bureau of Occupational Diseases, City Health Department, Baltimore, Md.

THE Baltimore City Health Department first became interested in lead poisoning in 1929 in an investigation of several cases of industrial lead poisoning. This interest was broadened in 1932 when an outbreak of 40 non-fatal cases of lead poisoning occurred mostly in children as a result of the inhalation of fumes and dust arising from the use of discarded storage battery casings for fuel.¹ Subsequently, for a period of more than four years all cases of lead poisoning which came to the attention of the Health Department were routinely investigated in order to ascertain if possible the source of the lead exposure in both adults and children. Early in 1935, at the request of Dr. Edwards A. Park, Pediatrician-in-Chief at the Johns Hopkins Hospital, the Bureau of Laboratories provided facilities for the quantitative estimation of lead in blood in cases of suspected plumbism. These facilities were soon expanded into a routine service. The usefulness of the quantitative blood lead analysis as an index of abnormal lead absorption had already been demonstrated at that time by the extensive studies of Blumberg and Scott at the Johns Hopkins Hospital² as well as by other investigators.^{3,4} Subsequent investigations have further confirmed the value of this

analysis in providing evidence of lead absorption in cases of suspected lead intoxication.⁵⁻¹⁴

Since July, 1935, we have routinely examined more than 1,400 specimens of blood from over 1,000 individuals. The blood lead service has been used by more than 20 local hospitals and by more than 80 private physicians. At the present time approximately 30 specimens of blood are examined each month. No charge is made for this work. It is the purpose of the present report to describe this unique laboratory service and to call attention to the valuable assistance which it has given to our Division of Industrial Hygiene and Bureau of Occupational Diseases in locating lead hazards and in evaluating the extent of lead poisoning in the community.

COLLECTION OF SPECIMENS

Ten gram samples of whole clotted blood are used in these examinations. In order to minimize contamination, only specimens submitted in prepared containers known as "blood lead outfits" (Figure 1) are accepted for analysis. The outfit consists of a lead-free glass test tube (17 mm. x 91 mm.) stoppered with a clean new XXXX quality Armstrong cork. The tube is first cleaned, then rinsed with nitric acid and lead-free water, and dried. The tube, together with an identification card requesting information concerning the

* Read before the Industrial Hygiene Section of the American Public Health Association at the Seventieth Annual Meeting in Atlantic City, N. J., October 15, 1941.

[4813]



FIGURE 1

name, address, age, sex, color, occupation, and length of exposure to lead of the patient is packed in a distinctive cardboard box bearing the "blood lead outfit" label. Directions for collecting the blood specimen are printed on the reverse side of the card. Recently, the outfit was further improved by the provision of a sterile, lead-free, rustless, 19 gauge, Petroff needle (Becton Dickinson, Number 497N) contained in a small paper envelope. The needle is suitable for adult use only and eliminates the need for a syringe. The cost to us of the blood lead outfit including the needle is 7½ cents. The outfits are distributed by the Bureau of Laboratories to the local medical profession and hospitals in the same manner as outfits regularly provided for the collection of specimens in cases of infectious diseases.

METHOD OF ANALYSIS

It is now well established that traces of lead are normally present in the blood. The difference between normal and abnormal amounts of lead in circulating blood is a quantitative and not a qualitative distinction and requires the use of highly sensitive and accurate means of lead estimation. In this work the modification of the dithizone method described by Wilkins, Willoughby, *et al.*^{5,12} was used. The method was further modified to include

the neutral wedge photometer in accordance with the technic of Clifford and Wichmann.¹⁶

Meticulous supervision is required. The method is practical only when a sufficient volume of samples is anticipated. In our laboratory, reagents and equipment are available for the simultaneous examination of 6 specimens. This equipment necessitated an outlay over a period of 4 years of more than \$400. However, the average cost of a single examination is estimated at about 25 cents. The figure given does not include the cost of labor. When analyses are made in multiple, the average time required of the analyst for a single analysis is slightly more than 1 hour.

REPORTING OF RESULTS

Blood lead concentrations are reported as mg. lead per 100 gm. of blood. Recently, for convenience, the following summary of the interpretation of the blood lead value was printed on the report form which is mailed to the physician.

INTERPRETATION OF RESULTS

It is now recognized that in the general population the average lead content of the blood is about 0.03 mg. per 100 gm. In persons with no history of exposure to lead, the blood lead level may range as high as 0.05 to 0.06 mg. per 100 gm. of blood. Higher blood lead values are evidence of abnormal absorption of lead and suggest the relative severity of the recent lead exposure.

Lead absorption is not synonymous with lead poisoning. Experience has demonstrated that individuals in industrial lead exposures may have abnormal blood lead levels with no accompanying symptomatology. A high blood lead level is not, of itself, diagnostic of lead poisoning but must be correlated with other findings, both clinical and laboratory, as well as with a history of definite exposure to lead.

No direct correlation exists between the degree of elevation of the blood lead value and the severity of the symptoms. It should be further noted that in convalescence from lead poisoning the blood lead value may remain in the abnormal range for a considerable period after symptoms have disappeared.

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BLOOD LEAD DETERMINATIONS

483

TABLE 1
Initial Blood Lead Values in Normal Individuals and in Cases of Suspected Lead Poisoning

Mg. Lead per 100 gm. Blood	Normal Controls	Suspected Lead Poisoning			
		Positive Clinical Diagnosis		Negative Clinical Diagnosis	
		Adults	Children	Adults	Children
0.00	2	..	..	1	..
0.01	8	..	..	10	9
0.02	25	..	..	31	17
0.03	41	..	..	68	40
0.04	39	..	..	79	50
0.05	8	1	..	57	22
0.06	1	6	..	23	19
0.07	1	3	..	8	15
0.08	1	8	1	3	7
0.09	..	9	6	2	..
0.10-0.20	..	46	26	5	..
0.21-0.30	..	3	22	1	..
0.31-0.40	..	5	7	..	..
0.41-0.50	..	..	8	..	..
0.51-0.60	..	1	3	..	..
0.61-0.70	..	..	1	..	..
TOTAL (750)	126*	82	74	291	177

* Mean normal value 0.031 with standard deviation ± 0.017 .

If the findings in this report indicate a doubtful result or an abnormal absorption of lead, an additional specimen of blood and also a specimen of urine should be examined. Because of the time and expense involved in making these analyses, physicians must communicate directly with the Director, Bureau of Laboratories, in order to make arrangements for repeat tests.

This interpretation is based on the available literature cited in the above references as well as upon our own experience.

SUMMARY OF BLOOD LEAD FINDINGS

At the outset the laboratory service was intended merely to provide practitioners with an aid in the diagnosis of lead poisoning particularly in cases involving infants and young children. The cooperation of the hospitals and private physicians who made use of the laboratory service was solicited in order to make available the clinical histories of those individuals whose bloods had been examined for lead. This investi-

gative work was expanded after the creation of the Bureau of Occupational Diseases in the Baltimore City Health Department in 1936. In this way, an excellent source of information was provided as to the occurrence of both industrial and nonindustrial lead poisoning in the city. In addition a study of the case histories permitted a correlation of the chemical blood lead determination with the clinical signs and symptoms, the history of exposure, and the final diagnosis made in each instance. Case histories of 624 individuals (373 adults and 251 children) have thus far been examined. A distribution of the blood lead values of this group in relation to the clinical diagnoses made is shown in Table 1.

Initial blood lead values only are recorded in this table; values obtained in convalescence or after treatment are not shown. The table also includes a series of specimens from 31 adults and 95 children (126 in all) with no history

of industrial or other exposure to lead. These specimens were obtained through the cooperation of the local infectious disease hospital. The blood lead values in this "normal" group were not significantly different for the adults and children. Since these individuals were not "normal" with respect to health, it is conceivable that metabolic disturbances associated with infectious disease may have slightly elevated the blood lead values of some members of the series.

In the group diagnosed as clinical lead poisoning our data include 82 adults, all but one of whom contracted lead poisoning in the course of their occupations; and 74 children of an average age of $2\frac{1}{2}$ years, practically all of whom had a history of pica associated with the chewing of objects painted with lead containing paints. In many of the children's cases, analyses were made of paint scrapings to confirm the source of lead. Specimens from all but one of the 251 children were submitted from hospitals. On the other hand, specimens from 30 per cent of the adults were submitted by private physicians.

In plumbism, the most commonly occurring blood lead values in both adults and children was in the range of 0.1 to 0.2 mg. per 100 gm. of blood. Values in excess of 0.2 mg. occurred far more frequently in affected children than in adults.

The group tabulated as "negative clinical diagnosis" consists of individuals whose clinical histories did not show a diagnosis of lead poisoning. Many of the children in this group had a history of pica. Almost all of the adults were engaged in occupations wherein some abnormal absorption of lead might be suspected. Those adults in this category who showed blood lead values in excess of 0.07 mg. were definite examples of asymptomatic lead absorption. These men were engaged

in such occupations as the manufacture of lead paint pigments, the manufacture of lead arsenate, scrap lead reclamation, and lead burning.

USEFULNESS OF THE BLOOD LEAD SERVICE AS A HEALTH DEPARTMENT METHOD

The local medical profession is accustomed to asking for a blood lead determination by the Baltimore City Health Department in cases of suspected lead poisoning encountered in their practice. A copy of the result of each analysis is forwarded to the Bureau of Occupational Diseases for investigation. In this way the majority of the actual lead hazards in local industry have been defined and numerous corrections of lead exposures were obtained.

With the reports from the blood lead service as a clue, numerous instances have occurred in the past 6 years wherein outbreaks of industrial lead poisoning were immediately called to the attention of the Bureau of Occupational Diseases and the Division of Industrial Hygiene. An excellent example which has already been cited related to an incident involving several cases of lead poisoning in a manufacturing plant. A cross-connection in plumbing permitted a solution of lead compound to enter the water supply of a drinking fountain in the plant. Numerous other incidents have occurred which involve such diverse occupations as the manufacture of lead arsenate, the manufacture of lead paint pigments, acetylene torch burning in shipbreaking, enamelling with lead-containing frit and the cleaning of tanks which had been used for the storage of leaded gasoline.

In recent years, largely as a result of studies associated with a follow-up of the blood lead laboratory service, it has been possible for the Baltimore City Health Department to acquire relatively accurate information concern-

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TABLE 2

Reporting of Non-fatal Lead Poisoning in Baltimore City, 1931-1940

Year	Total Non-Fatal Cases	Cases Reported		Cases Not Reported But Ascertained Through Follow-up of Laboratory Service	
		Adult	Child	Adult	Child
1931	..	..	..	..	..
1932	49	12	37	..	..
1933	2	2	..	..	..
1934	4	..	4	..	..
1935	12	1	4	4	3
1936	26	3	..	12	11
1937	24	..	..	16	8
1938	11	2	..	2	7
1939	18	..	..	11	7
1940	26	3	..	18	5
TOTAL	177	23	45	63	41

ing the incidence of lead poisoning in the community.¹⁴ During the past 10 years, all of the known cases of fatal lead poisoning (49 children and 7 adults) were reported to the Bureau of Vital Statistics. However, there has been little tendency to report cases of non-fatal lead poisoning. This fact is strikingly demonstrated in Table 2.

The increased incidence of reported lead poisoning in 1932 is due to the storage battery outbreak mentioned earlier.¹ Beginning in 1935, it will be noted that the Health Department became aware of a large number of unreported cases of non-fatal lead poisoning. This resulted directly from the follow-up of the laboratory service. Only those cases are listed in which an investigation with regard to the history of exposure, signs, symptoms, and laboratory examination furnished evidence to substantiate the diagnosis of lead poisoning.

SUMMARY

A description has been given of a blood lead laboratory service which the Bureau of Laboratories of the Baltimore City Health Department has made available to the physicians and

hospitals of Baltimore during the past 6 years. The service has proved invaluable to the Division of Industrial Hygiene and the Bureau of Occupational Diseases in locating lead hazards and in evaluating the extent of both industrial and nonindustrial lead poisoning in the community.

REFERENCES

1. Williams, H., et al. Lead Poisoning from the Burning of Battery Cables. *JAMA*, 100:1485, 1933.
2. Blumberg, H., and Scott, T. F. McN. The Quantitative Spectrographic Estimation of Blood Lead and Its Value in the Diagnosis of Lead Poisoning. *Bull. Johns Hopkins Hosp.*, 56:216-223, 1935.
3. Litane, S., and Weyrauch, F. Relationship of the Lead Content of the Blood and Urine to the Onset of Clinical Symptoms. *Med. Klin.*, 29:13-15, 1933.
4. Bass, E. The Relation of Clinical Symptoms in Lead Poisoning to the Lead Content of the Blood and Urine. *Deutsche med. Wochenschr.*, 59:1665-1668, 1933.
5. Willoughby, C. E., and Wilkins, E. S., Jr. The Lead Content of Human Blood. *J. Biol. Chem.*, 124:639-654, 1938.
6. Smith, F. L., Rathmell, T. K., and Marcell, G. E. The Early Diagnosis of Acute and Latent Plumbism. *Am. J. Clin. Path.*, 8:471-514, 1938.
7. Kaplan, E., and McDonald, J. M. The Blood Lead Value as an Aid in the Diagnosis of Lead Poisoning. *J. Pharmacol. & Exper. Therap.*, 63:17, 1938.
8. Teisinger, J. A Rapid Micro Polarographic Method for the Quantitative Estimation of Lead in Blood. *Ztschr. f. d. ges. exper. Med.*, 98:520, 1936.
9. Tzege, H., and Schmitt, F. Quantitative Estimation of the Lead Content of Blood and Excreta in Normal Individuals and in Cases of Lead Poisoning. *Ztschr. f. d. ges. exper. Med.*, 100:717-735, 1937.

10. Tompsett, S. L., and Anderson, A. D. The Lead Content of Human Tissues and Excreta. *Biochem. J.*, 29:1831-1854, 1935.
11. Fell, A. The Importance of the Blood and Urinary Lead Examination in the Diagnosis of Plumbism. *Pract. med.*, 45:328, 1931.
12. Kehoe, R. A. Industrial Lead Poisoning and the Medical Control of Industrial Lead Exposure. *Symposium on Industrial Health, Medical College of Virginia, Richmond, Va.*, 1940, pp. 19-29.
13. Freese, W. C., et al. The Control of the Lead Hazard in the Storage Battery Industry. *Pub. Health Bull. No. 262*. Government Printing Office, Washington, D. C., 1941.
14. Chalmers, J. N. M. Lead Content of Human Blood. *Lancet*, 138:447-450, 1940.
15. Wilkins, E. S., Jr., Wilboughby, C. E., Kraemer, E. O., and Smith, F. L. Determination of Minute Amounts of Lead in Biological Materials. *Indus. & Engin. Chem. Anal. Ed.*, 7:33-36, 1935.
16. Clifford, P. A., and Wichmann, J. H. Dithione Methods for the Determination of Lead. *J. Assoc. Official Agr. Chem.*, 19:150-156, 1936.
17. Baltimore City Health Department. Plumbing Cross Connection Results in Acute Lead Poisoning. *Baltimore Health News*, 13:52, 1936.
18. Kaplan, E., and McDonald, J. M. The Incidence of Lead Poisoning in the City of Baltimore. In press.

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GACMV

Children's Health | Article

IQ and Blood Lead from 2 to 7 Years of Age: Are the Effects in Older Children the Residual of High Blood Lead Concentrations in 2-Year-Olds?

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Increases in peak blood lead concentrations, which occur at 18–30 months of age in the United States, are thought to result in lower IQ scores at 4–6 years of age, when IQ becomes stable and measurable. Data from a prospective study conducted in Boston suggested that blood lead concentrations at 2 years of age were more predictive of cognitive deficits in older children than were later blood lead concentrations or blood lead concentrations measured concurrently with IQ. Therefore, cross-sectional associations between blood lead and IQ in school-age children have been widely interpreted as the residual effects of higher blood lead concentrations at an earlier age or the tendency of less intelligent children to ingest more leaded dust or paint chips, rather than as a causal relationship in older children. Here we analyze data from a clinical trial in which children were treated for elevated blood lead concentrations (20–44 µg/dL) at about 2 years of age and followed until 7 years of age with serial IQ tests and measurements of blood lead. We found that cross-sectional associations increased in strength as the children became older, whereas the relation between baseline blood lead and IQ attenuated. Peak blood lead level thus does not fully account for the observed association in older children between their lower blood lead concentrations and IQ. The effect of concurrent blood level on IQ may therefore be greater than currently believed. **Key words:** chelation, developmental testing, IQ, lead poisoning, longitudinal studies. *Environ Health Perspect* 113:597–601 (2005). doi:10.1289/ehp.7625 available via <http://dx.doi.org/> [Online 2 February 2005]

In 1994, two meta-analyses of the relationships between childhood lead exposure and IQ appeared (Pocock et al. 1994; Schwartz 1994). Both dealt with a problem that arises in the interpretation of prospective studies in which the child's blood lead concentration peaks at about 2 years of age, but IQ can only be measured stably (with higher test-retest correlation) in children ≥ 4 years of age (Sternberg et al. 2001). Both meta-analyses focused on the strength of association between IQ at school age and either blood lead concentration measured at 2 years of age or derived measures of lead exposure, such as average blood lead up to 3 years of age, which was found to be correlated strongly with 2-year blood lead concentration.

One meta-analysis (Pocock et al. 1994) included cross-sectional studies in a separate analysis; those studies also showed an inverse association of blood lead concentration with IQ at ≥ 6 years of age. The other meta-analysis (Schwartz 1994) included cross-sectional studies in the overall model, but additional analysis of cross-sectional studies showed findings similar to those in the prospective studies. Both meta-analyses concluded that the prospective design was more reliable in studying blood lead–IQ associations, because reverse causality, in which impaired children ingest more leaded paint or dust, was less likely to produce an association. Neither meta-analysis emphasized the importance of

cross-sectional associations in older children, and both implied that the interpretation most consistent with the overall results was that peak blood lead concentration, achieved at about 2 years of age, was most likely responsible for the cognitive effects, even though these effects were not reliably detectable until ≥ 4 years of age.

These meta-analyses as well as the findings from the individual prospective studies, especially Boston (Bellinger et al. 1992), have had a strong effect on the interpretation of both longitudinal and cross-sectional studies. One consequence is that if we wish to know the minimum concentration in blood at which lead affects cognition, then we are obliged to follow children starting at 2 years of age. School-age children, in whom IQ is relatively stable and testable, have lower blood lead concentrations. A cross-sectional study among school-age children might reflect latent damage done by higher blood lead concentrations at 2 years of age but detectable only at school age because IQ can be more reliably tested with greater precision at that age. This interpretation has guided screening programs, which focus on 1- and 2-year-olds; clinical trials, which have treated 2-year-olds; and the interpretation of the cross-sectional literature. Thus, the report in 2000 (Lanphear et al. 2000) that school-age children studied in the third National Health and Nutrition Examination Survey had an

inverse relationship between their concurrent blood lead concentrations and IQ even at blood lead concentrations < 10 µg/dL, the current Centers for Disease Control and Prevention (CDC) level of concern, was interpreted by some to reflect the effects of the children's higher blood lead concentrations when they were 2 years old. However, after publication of prospective data showing that even peak blood lead concentrations < 10 µg/dL in toddlers were associated with decrements in IQ (Canfield et al. 2003), changes in policy were quickly considered.

It is not clear that only the peak blood lead concentration matters. In the prospective Boston study that examined children with very low lead exposure, blood lead concentration at 2 years of age, but not at 57 months or 10 years of age, was significantly associated with IQ at 10 years of age (Bellinger et al. 1992). Other prospective studies of children with relatively high lead exposure, however, found concurrent (Dietrich 1995; Dietrich et al. 1993) or lifetime average blood lead concentration (Tong et al. 1996) to be more strongly associated with IQ measurement at school age than were peak levels. Another study modeling relative contribution of prenatal and postnatal (0–2 years, 2 to 3–7 years) blood lead also found increases in blood lead after 2 years of age associated with IQ deficits after 2 years of age (Wasserman et al. 2000).

Other analyses have asked whether greater declines in blood lead concentration from the 2-year peak are associated with higher IQ later. Two studies with analyses that adjusted for 2-year cognitive test score found that children whose blood lead concentrations fell more after 2 years of age had increased IQ (Liu et al.

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from an earlier blood lead concentration, both prior and concurrent blood lead concentration were included in the models (blood lead concentration at 2 and 5 years on IQ at 5 years of age, blood lead concentration at 2 and 7 years on IQ at 7 years of age, blood lead concentration at 5 and 7 years on IQ at 7 years of age). The model thus includes later IQ as the dependent variable, and earlier blood lead concentration, later blood lead concentration, and covariates as the independent variables. We also tried additional adjustment for earlier IQ, which theoretically tests for change in IQ by change in blood lead concentration. This allows us to compare results with previous studies using a similar approach (Liu et al. 2002; Ruff et al. 1993).

Quantitative analysis of both prior and concurrent blood lead concentration in the same model, even without adjustment for prior IQ, still faces the problem of collinearity between the two blood lead measurements. A qualitative alternative may reduce the precision in estimation but should show the effect on school-age IQ by blood lead concentration with less distortion. Therefore, we used the median of earlier blood lead concentration and the median of later blood lead concentration as cutoff points to reduce earlier and later blood lead concentrations to binary variables. This does not eliminate the inherent correlation between earlier and later blood lead concentrations (children with lower earlier blood lead concentrations tend to have lower later blood lead concentrations), but it reduces the impact of collinearity on the stability of the

estimated regression coefficients and produces a model that is easy to interpret.

To see whether the association between blood lead concentration and IQ score varied by treatment group (succimer or placebo), we repeated these regression models by treatment group.

We used SAS (version 9.0; SAS Institute Inc., Cary, NC) for statistical analysis. All tests are two sided. Because of the difference in the number of children tested for each follow-up measurement, the sample sizes of the different regression models vary slightly.

Results

Study subjects. Four centers were involved in the recruitment, treatment, and follow-up of a total of 780 children in the TLC study: Baltimore had 213, Cincinnati had 194, Newark had 208, and Philadelphia had 165. Three hundred ninety-six children were randomly assigned to the succimer group, and 384 to the placebo group. There were no differences between groups in age, sex, race, socioeconomic status, and blood lead concentration of children at recruitment. The children were mainly African American (77%), from households speaking English (95%), with a single parent (72%), and receiving public assistance (97%).

Blood lead concentration and IQ scores. The blood lead concentrations and IQ scores of TLC children at baseline and at 5 and 7 years of age are shown in Table 1. The correlation coefficients of blood lead concentrations at baseline and 5 or 7 years were 0.40 and

0.27, respectively; that between measurements at 5 and 7 years was 0.78. The correlation coefficients of cognitive scores at baseline and 5 or 7 years were 0.54 and 0.41, respectively; that between 5 and 7 years was 0.78. All of these correlation coefficients were statistically significant (all *p*-values < 0.001). Although mean blood lead concentration declined over time, the standard deviations remained similar. As shown in Table 1, the ages at IQ tests and blood lead concentration measurements were very close. For example, at about 7 years of age, 94% of children had IQ and blood lead tests on the same day.

Heterogeneity between succimer and placebo groups. There were no statistical differences between succimer and placebo groups in either blood lead concentrations or cognitive scores at the time points under consideration (all *p*-values > 0.10). The association between blood lead concentration and IQ score was homogeneous between treatment groups except for baseline blood lead concentration and baseline MDI: 10- μ g/dL increment at baseline had a coefficient of -1.6 (SE, 1.3) MDI points in the succimer group and -4.6 (SE, 1.4) MDI points in the placebo group. In the regression models including both prior and concurrent blood lead concentrations without additional adjustment for prior IQ, the results were similar in the succimer and placebo groups.

Associations of blood lead concentration and IQ. Figure 1 displays the mean IQ at current and subsequent ages by quartiles of blood lead measured at 2, 5, and 7 years of age. Although blood lead concentrations decreased over time and IQ test scores first declined and then increased, the inverse association between concurrent blood lead concentration and IQ test scores remained constant.

Table 2 shows the estimated difference in cognitive test scores for each 10- μ g/dL change in prior, concurrent, peak, or average blood lead concentration. Overall, the concurrent blood lead concentration always had the strongest association with IQ, and as the children aged, the relationship grew stronger. The peak blood lead concentration from baseline to 7 years of age was not associated with IQ at 7 years of age. The average blood lead concentration from baseline to 5 or 7 years of age was inversely associated with IQ at 5 or 7 years of age.

Is the cross-sectional association at a later age a residue of effects attributable to earlier blood lead concentrations? In the model including both prior and concurrent blood lead concentrations, concurrent blood lead was always more predictive of later IQ scores (Table 3). Adjustment for prior IQ did not significantly change the strength of association with concurrent blood lead concentration. Alternatively, categorizing prior and

Table 2. Adjusted estimates (95% confidence intervals) of the effect of a change of 10 μ g/dL in five measures of lead exposure on change on IQ test scores measured at 2, 5, and 7 years of age.

Outcome variable: IQ score (points)	Independent variable: blood lead concentration (per 10- μ g/dL increment) ^a				
	2 years of age	5 years of age	7 years of age	Peak ^b	Average ^c
MDI at baseline	-2.9 (-4.7 to -1.0)				
IQ at 5 years of age	-2.3 (-4.1 to -0.5)	-3.5 (-5.3 to -1.7)			-2.9 (-4.8 to -1.0)
IQ at 7 years of age	-1.1 (-2.9 to 0.7)	-2.9 (-4.8 to -1.1)	-5.4 (-7.8 to -2.9)	-0.7 (-2.1 to 0.7)	-3.3 (-5.4 to -1.1)

^aEach combination of outcome and independent variables was modeled separately; all were adjusted for clinic center, race, sex, language, parent's education, parent's employment, single parent, caregiver's IQ, and exact age at blood lead concentration measurement. ^bPeak blood lead concentration between baseline and 7 years of age. ^cAverage blood lead concentration from baseline to 5 years of age for IQ at 5 years of age, and average blood lead concentration from baseline to 7 years of age for IQ at 7 years of age.

Table 3. Adjusted estimates (95% confidence intervals) of the effect of a 10- μ g/dL change in prior and concurrent blood lead concentrations on IQ test scores at 5 and 7 years of age.

Outcome variable: IQ score	Additional adjustment for baseline MDI or 5-year IQ score	Independent variable: blood lead concentrations (per 10- μ g/dL increment) ^a		
		2 years of age	5 years of age	7 years of age
IQ at 5 years of age	None	-1.2 (-3.1 to 0.7)	-2.9 (-4.9 to -0.9)	
	Baseline MDI	-0.1 (-1.8 to 1.5)	-2.4 (-4.1 to -0.7)	
IQ at 7 years of age	None	0.1 (-1.8 to 2.0)		-5.0 (-7.8 to -2.4)
	Baseline MDI	0.4 (-1.4 to 2.1)		-3.8 (-6.2 to -1.4)
	None		-1.2 (-4.1 to 1.7)	-3.9 (-7.4 to 0.6)
	5-year IQ		1.7 (-0.3 to 3.6)	-3.7 (-6.3 to -1.1)

^aEach row of results was from separate models; all were adjusted for clinic center, race, sex, language, parent's education, parent's employment, single parent, caregiver's IQ, and exact age at both blood lead measurements.

concurrent blood lead concentrations by the corresponding medians in the regression model had similar results (Table 4). Among children with both prior and concurrent blood lead concentrations below the corresponding medians as the reference group, those with prior blood lead concentrations at or above median but concurrent blood lead concentrations below the median did not have a decrease in school-age IQ score. In contrast, children with concurrent blood lead concentrations at or above the median had roughly similar IQ decreases, irrespective of their prior blood lead concentration.

Discussion

In these data we found a stronger relationship between blood lead concentration at 7 years and IQ at 7 years than between IQ at 7 years and the higher 2-year-old blood lead concentration; the association of blood lead concentration at 5 years and IQ at 5 years is also stronger than that between IQ at 5 years and blood lead at 2 years. The strength of the cross-sectional association increases over time (from 2 years of age to 5–7 years of age), despite lower blood lead concentrations in older children. This analysis supports the idea that lead exposure continues to be toxic to children as they reach school age, and does not support the interpretation that all the damage is done by the time the child is 2 or 3 years of age.

We found previously that children with relatively greater declines in blood lead have improved IQ; but that finding was limited to children given placebo (Liu et al. 2002). The analysis that yielded that result was modeled closely on that of a previous report (Ruff et al. 1993) in which declining blood lead concentration was associated with increased IQ independent of chelation treatment, because our primary goal was to attempt to replicate their

findings. We now suspect that the difference in the results for placebo and succimer groups was caused by unexpected heterogeneity in the relationship at baseline between blood lead and IQ in the placebo and succimer groups. Removing baseline MDI from the model that was previously used reduced the difference between placebo and succimer groups. In neither analysis did we see evidence in the overall group or in the placebo group that blood lead at 2 years of age determined IQ at 7 years of age. Nevertheless, modeling two change measures (IQ change and blood lead change) in the same model was complicated by intraindividual tracking in blood lead concentration and IQ over time as well as correlation between baseline lead and baseline IQ (Dietrich et al. 1993; Ernhart 1993). Theoretically, putting baseline IQ in the regression model would yield an estimate of the effect of blood lead concentration change on individual IQ change over time; however, the baseline IQ may be on the causal path. Also, IQ at an early age has less stability (Sternberg et al. 2001), and the use of different IQ test measures at 2 years of age and school age (Liu et al. 2002; Ruff et al. 1993; Tong et al. 1998) may actually introduce more variance. Thus, although it is attractive to look at change in IQ by change in blood lead when considering the effect of an intervention, modeling those changes simultaneously is complex and can produce results that are difficult to interpret. A simpler approach is to model IQ at school age by prior and concurrent blood lead concentration: If higher concurrent blood lead concentrations are associated with lower IQ, then it is plausible that we should attempt to keep blood lead concentrations low.

It is prudent to ponder whether greater association between concurrent blood lead and IQ score at school age was due to reverse causality. This could happen if the lower IQ

preceded higher lead concentrations. In this data set, baseline MDI was not associated with blood lead concentration at 7 years, with or without adjustment of baseline blood lead. This supports the findings of the longitudinal Port Pirie study (Tong et al. 1996) and supports the notion that lead exposure causes cognitive deficiency in children within approximately normal intelligence range, but not the reverse, although the association between much lower IQ than seen in these study subjects and later high blood lead through pica cannot be tested here. Another way in which the strengthening association over time may be produced is to have a fixed effect of the higher blood lead concentrations at 2 years of age concentrated in a more restricted range of blood lead concentrations at 7 years of age. In the TLC study, however, the range of blood lead concentration at entry was 24 µg/dL, and the range at 7 years of age was 26 µg/dL, with similar standard deviations. The restricted range of blood lead concentrations in TLC at enrollment might produce an imprecise estimate of the slope of the relationship between IQ and blood lead concentration, which would stabilize as the children led different lives and their blood lead concentrations dispersed. The slope of blood lead concentration at 2 years of age versus IQ at 5 years of age is similar to that seen in the meta-analyses, and the cross-sectional associations are similar to those seen in studies previously reviewed (Pocock et al. 1994; Schwartz 1994). It seems unlikely that the slope would be substantially steeper if more children with lower values at 2 years of age were added, because the range of 10–20 µg/dL is the range most stably estimated from the meta-analyses, and the TLC estimate is already similar to it.

This finding implies that cross-sectional associations seen in older children, such as the school-age children in the National Health and Nutrition Examination Survey data, should not be dismissed as representing a residual from early high lead exposure. Breslau et al. (2001) recently showed that urban Detroit, Michigan, children had declines in IQ from 6 to 11 years of age, whereas suburban children showed no change in the inherently age-adjusted scores. The data suggested that growing up in a racially segregated and disadvantaged community might contribute to a decline in IQ scores in the early school years. However, urban dwelling may serve as a surrogate of individual exposure, such as lead exposure. TLC children had similar communities of origin as the urban children in the study by Breslau et al. (2001). It could be that lead exposure is causally related to this phenomenon, or it could be that factors leading to the families' inability to improve their housing led to both higher lead levels and lower IQ.

Table 4. Adjusted estimates of regression coefficients (95% confidence intervals) of categorical blood lead concentrations in models for IQ scores.

Category of blood lead concentration (µg/dL) ^a	No.	Outcome variable			
		IQ at 5 years ^b		IQ at 7 years ^b	
		Mean score	Comparison	Mean score	Comparison
2 years	5 years				
<24.9	<11.4	83.7	Referent		
<24.9	≥11.4	78.8	-2.9 (-5.8 to 0.1)		
≥24.9	<11.4	82.4	0.4 (-2.5 to 3.3)		
≥24.9	≥11.4	77.8	-4.0 (-6.8 to -1.5)		
2 years	7 years				
<24.9	<7.2			89.3	Referent
<24.9	≥7.2			84.6	-3.6 (-6.4 to -0.7)
≥24.9	<7.2			88.9	-0.0 (-2.8 to 2.7)
≥24.9	≥7.2			84.0	-3.7 (-6.2 to -1.3)
5 years	7 years				
<11.4	<7.2	244		89.4	Referent
<11.4	≥7.2	52		85.9	-2.3 (-5.9 to 1.3)
≥11.4	<7.2	62		89.2	0.3 (-3.1 to 3.7)
≥11.4	≥7.2	255		83.9	-3.8 (-6.0 to -1.6)

^aMeasured at 2 and 5 years of age, 2 and 7 years of age, and 5 and 7 years of age. ^bAdjusted for clinic center, race, sex, language, parent's education, parent's employment, single parent, caregiver's IQ, and exact age at both blood lead measurements.

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Lead Exposure, IQ, and Behavior in Urban 5- to 7-Year-Olds: Does Lead Affect Behavior Only by Lowering IQ?

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Exhibit 765

tion, employment, income, and receiving public assistance) at recruitment. Overall, the children were mostly black (77%), spoke English (95%), with a single parent (72%), and with parent(s) receiving public assistance (97%). Girls accounted for 44% of children, 40% of children had parents with <12 years of education, and 58% of children had neither parent employed.

Blood Lead Concentrations

At baseline, the mean blood lead concentration was 26 $\mu\text{g}/\text{dL}$. It declined to 12 $\mu\text{g}/\text{dL}$ (range: 2–35 $\mu\text{g}/\text{dL}$) at 36 months of follow-up and to 8 $\mu\text{g}/\text{dL}$ (range: 0–26 $\mu\text{g}/\text{dL}$) at 60 months of follow-up (Table 1). There were no differences in blood lead concentrations between succliner and placebo groups at these 3 age points (mean $\pm$ SD age: 2.0 $\pm$ 0.5, 5.0 $\pm$ 0.5, and 7.0 $\pm$ 0.2 years, respectively).

Cognitive Tests

The cognitive scores (mean $\pm$ SD) in TLC children at baseline, age 5, and age 7 are shown in Table 1. Caregiver's IQ of these children had a mean of 80 and an SD of 11. Again, these cognitive scores of both children and caregivers did not differ by treatment group.

Behavioral Tests

The behavioral test scores (mean $\pm$ SD) at 5 and 7 years are shown in Table 1. Also shown are the correlation

coefficients of these test scores with IQ and with blood lead concentration measured at the same age.

Lead and Behavior Association

We first plotted concurrent blood lead concentration and behavior scores and did spline regression. As examples, results of CPRS-R at age 5 and teacher-rated BASC (at age 7) are shown in Figs 2 and 3. Behavioral problems tend to increase with increasing blood lead concentration at both ages 5 and 7 in the unadjusted data, but the estimates of CPRS-R at age 5 and of BASC internalizing problems (both teacher and parent rated) at age 7 were not statistically significant. In the path analysis for the CPRS-R, there were no statistically significant direct effects of blood lead level at age 2 or age 5; indirect effects were small and not consistent (Table 2). At age 7, there were no statistically significant direct or indirect effects of blood lead concentration at age 2 (data not shown). Blood lead concentration at age 7 had a statistically significant direct effect on BASC-TRS behavioral symptoms index, externalizing problems, and school problems and BASC-PRS externalizing problems (Figs 4 and 5). There were indirect effects of blood lead concentration at age 7 on all of the measurements except BASC-TRS externalizing problems and BASC-PRS internalizing problems. In the logistic regression analysis examining those at risk of or having clinically significant behavior problems, a 10 $\mu\text{g}/\text{dL}$ elevation in 7-year lead was asso-

TABLE 1 Mean and SD of Blood Lead Concentrations, IQ, and Non-IQ Outcomes and the Correlation Coefficients Between IQ and Blood Lead Concentrations and Non-IQ Outcomes at Specific Age in TLC Children

Age, y	Variables	n	Mean $\pm$ SD	Correlation With Concurrent IQ ^a	Correlation With Concurrent Lead ^b
2	Blood lead concentration, $\mu\text{g}/\text{dL}$	780	26.2 $\pm$ 5.1	–0.20	NA
5	Blood lead concentration, $\mu\text{g}/\text{dL}$	731	12.0 $\pm$ 5.2	NA	–0.20
	IQ	727	80.6 $\pm$ 11.3		
	CPRS-R				
	Oppositional index	721	57.6 $\pm$ 13.0	–0.14	0.03
	Hyperactivity index	721	61.6 $\pm$ 13.7	–0.14	0.06
	ADHD index	721	59.9 $\pm$ 12.8	–0.25	0.06
	Behavioral index	721	60.4 $\pm$ 12.0	–0.20	0.05
7	Blood lead concentration, $\mu\text{g}/\text{dL}$	622	8.0 $\pm$ 4.0	–0.23	NA
	IQ	643	86.7 $\pm$ 13.3	NA	–0.23
	BASC-TRS				
	Adaptive skills	530	46.3 $\pm$ 9.4	0.39	–0.13
	Behavioral symptoms index	540	54.5 $\pm$ 11.5	–0.28	0.18
	Externalizing problems	539	55.2 $\pm$ 13.0	–0.18	0.18
	Internalizing problems	541	52.4 $\pm$ 10.1	–0.17	0.08
	School problems	541	56.2 $\pm$ 12.2	–0.47	0.23
	BASC-PRS				
	Adaptive skills	647	45.7 $\pm$ 11.0	0.33	–0.11
	Behavioral symptoms index	647	55.6 $\pm$ 14.9	–0.21	0.13
	Externalizing problems	647	58.0 $\pm$ 15.4	–0.23	0.16
	Internalizing problems	647	50.1 $\pm$ 12.1	–0.06	0.06

NA indicates not applicable.

^a All $P < .05$ except for parent-rated internalizing problems at age 7.

^b All $P < .05$ except for CPRS-R scores and BASC internalizing problems (both teacher and parent rated).

children). Although it may be that effects of lead on behavior occur only in children with relatively high exposures, such as those in TLC, experience with lead and IQ is not reassuring in that the dose-response curve seems to be at least linear and may be steeper at lower levels of exposure.¹⁴ Still, it would be interesting to see whether the association exists in children with background levels of exposure at approximately age 2 years, because they probably do not have a marked decline of blood lead levels by age 7 years. Nonetheless, in the eastern US cities where TLC was done, poor housing, lead exposure, poverty, and other social stressors are strongly confounded, and so the causal nature of the lead and behavior association cannot be proven absolutely by observational study.

With the regulation of leaded gasoline and paint, the mean blood lead concentration in US children declined remarkably in the past 3 decades. However, the best estimate of the number of children aged 1 to 5 years with blood lead concentration $>10 \mu\text{g/dL}$ in 1999–2002 was 1.6% (~310 000 children).¹⁵ If lead effects on behavior are independent of its effects on IQ, the adverse effects of lead exposure in children would be larger than currently thought, but the benefits of prevention of lead exposure would also be magnified. Additional studies are needed to study the associations between baseline and concurrent blood lead levels and neuropsychological function and behavior problems among children older than 7 years and into adolescence. How exposure to urban violence or other stressors associated with poverty impact lead-exposed children as they enter adolescence is yet to be determined, although there are now ≥ 2 studies in which lead exposure in early life is associated with later delinquent behavior.^{14,17}

CONCLUSIONS

We have found, in children with relatively high lead exposure, that concurrent blood lead concentration was associated with externalizing and school problems at 7 years old, and the effect was not entirely mediated through the lead effect on IQ. On the other hand, higher blood lead concentration at 2 years of age was not associated with behavior at 7 years of age. Finding both direct and indirect effects of concurrent blood lead concentration on behavior among school-aged children lends further urgency to the necessity of preventing lead exposure in children, preferably continuing into school age.

ACKNOWLEDGMENTS

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REFERENCES

1. Bellinger DC, Stiles KM, Needleman HL. Low-level lead exposure, intelligence and academic achievement: a long-term follow-up study. *Pediatrics*. 1992;90:855–861.
2. Baghurst PA, McMichael AJ, Wigg NR, et al. Environmental exposure to lead and children's intelligence at the age of seven years. The Port Pirie Cohort Study. *N Engl J Med*. 1992;327:1279–1284.
3. Dietrich KN, Berger OG, Succop PA, Hammond PB, Bornschein RL. The developmental consequences of low to moderate prenatal and postnatal lead exposure: intellectual attainment in the Cincinnati Lead Study Cohort following school entry. *Neurotoxicol Teratol*. 1993;15:37–44.
4. Pocock SJ, Smith M, Raghuram P. Environmental lead and children's intelligence: a systematic review of the epidemiological evidence. *BMJ*. 1994;309:1189–1197.
5. Schwartz J. Low-level lead exposure and children's IQ: a meta-analysis and search for a threshold. *Environ Res*. 1994;65:42–55.
6. Lanphear BP, Dietrich K, Auinger P, Cox C. Cognitive deficits associated with blood lead concentrations $<10 \mu\text{g/dL}$ in US children and adolescents. *Public Health Rep*. 2000;115:521–529.
7. Canfield RL, Henderson CR Jr, Cory-Slechta DA, Cox C, Jusko TA, Lanphear BP. Intellectual impairment in children with blood lead concentrations below $10 \mu\text{g/dL}$. *N Engl J Med*. 2003;348:1517–1526.
8. Lanphear BP, Hornung R, Khoury J, et al. Low-level environmental lead exposure and children's intellectual function: an international pooled analysis. *Environ Health Perspect*. 2005; 113: 894–899.
9. Needleman HL, Gunnoe C, Leviton A, et al. Deficits in psychologic and classroom performance of children with elevated dentine lead levels. *N Engl J Med*. 1979;300:689–695.
10. Munoz H, Romieu J, Palazuelos B, Mancilla-Sanchez J, Mendez-Gonzalez F, Hernandez-Avila M. Blood lead level and neurobehavioral development among children living in Mexico City. *Arch Environ Health*. 1993;48:132–139.
11. Leviton A, Bellinger D, Alford RN, Rabinowitz M, Needleman H, Schoenbaum S. Pre- and postnatal low-level lead exposure and children's dysfunction in school. *Environ Res*. 1993;60: 30–45.
12. David OJ, Hoffman SP, Clark J, Grad G, Sverd J. The relationship of hyperactivity to moderately elevated lead levels. *Arch Environ Health*. 1983;38:341–346.
13. Yuthill RW. Hair lead levels related to children's classroom attention-deficit behavior. *Arch Environ Health*. 1994;51: 214–220.
14. Needleman HL, McFarland C, Ness RB, Flenberg SB, Tobin MJ. Bone lead levels in adjudicated delinquents. A case control study. *Neurotoxicol Teratol*. 2002;24:711–717.
15. Dietrich KN, Berger OG, Succop PA. Lead exposure and the motor developmental status of urban six-year-old children in the Cincinnati Prospective Study. *Pediatrics*. 1993;91:301–307.
16. Winneke G, Altman L, Kramer U, et al. Neurobehavioral and neurophysiological observations in six year old children with low lead levels in East and West Germany. *Neurotoxicology*. 1994;15:705–713.
17. Almans L, Sveinsson K, Kramer U, Winneke G, Wiegand H. Assessment of neurophysiologic and neurobehavioral effects of environmental pollutants in 5- and 6-year-old children. *Environ Res*. 1997;73:125–131.

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LEAD POISONING—RUDDOCK.

JOUR. A. M. A.
MAY 24, 1924

made with a refined antitoxin or globulin, according either to the old or to the new formula.

Freezing of a three L + dose mixture of toxin-antitoxin containing 0.3 per cent. tricresol, prepared with an antitoxin concentrated without the use of heat, resulted in an increase in toxicity of the mixture.

Freezing of mixtures of toxin-antitoxin made with unconcentrated antitoxin and containing three L + doses of toxin, in which tricresol or phenol is used as a preservative, results in an increase in toxicity of the mixture.

Freezing of such mixtures in which chlorbutanol or chloroform is used as a preservative, or in which no preservative is used, does not cause an increase in toxicity of the mixture.

Only highly refined antitoxin or globulin should be used in the preparation of diphtheria toxin-antitoxin mixtures.

Mixtures of diphtheria toxin-antitoxin containing one-tenth L + doses should be the only formula permitted.

195 College Avenue.

✓ LEAD POISONING IN CHILDREN

WITH SPECIAL REFERENCE TO PICA

JOHN C. RUDDOCK, M.D.

LOS ANGELES

Pica, or parorexia, is a craving for unnatural articles of food—a depraved appetite. It has been observed in hysteria, chlorosis, insanity and pregnancy, but more particularly among children in the latter half of infancy, children from 1 to 2 years of age. It sometimes begins in later childhood when from any cause the general health fails, and the child becomes anemic.

Still¹ states that he has seen fourteen cases. In some of his cases, mud and mortar were especial favorites; in others, coal, cinders and gravel were preferred. In nine cases, the habit began in the second year of life; in one, it began at 8 months.

Thomson,² in a study of pica and its causes, divides this morbid craving in children into two groups:

1. Those instances in which the habit begins at any age, often associated with some marked deterioration of the general health (of which anemia is usually a symptom), and lessens or passes off as the general condition improves.

2. A larger group in whom the morbid craving develops in early infancy, as opportunity for its indulgence offers, apart from any very noticeable cachexia, and without anemia, passing off gradually in most cases, even if untreated, when the child is about 3 years old.

Patients in the former, or cachectic, class may be suffering from a variety of curable and incurable conditions, such as rickets, bronchitis, roundworms, apparently idiopathic anemia, malignant tumor, tuberculosis and other chronic maladies.

Koplik³ believes that this peculiar condition in children is an exaggeration of the normal habit in young infants of invariably placing everything within reach in their mouths. In children who suffer from pica, the sense of wholesomeness is lacking, and they crave sand,

dirt, gravel, cinders, wall plaster, paper, paint and other substances.

Although a child with pica craves many different types of things, it is very fortunate that most of these things are inert, and, although they occasionally lead to minor ailments, such as constipation, fissure-in-ano and other intestinal disorders, they do not cause any serious disturbance of the health of the child.

There is, however, one type of poisoning which these pica children acquire, and that is lead poisoning. This may easily be overlooked, because the average physician has never had his attention called to the fact, and also because the clinical picture is usually very different from that in similar poisoning in adults. There are many ways in which the child may acquire lead poisoning which the average parent could easily prevent.

Some children affected with pica have a morbid craving to gnaw painted objects, such as window sills, white furniture, crib railings, porch railings and other articles around the home within their reach. A child lives in a lead world.

There are many other ways in which lead may be introduced by mouth: in paint from toys; in buns and candies colored with lead chromate; in printers' type used as a plaything; in ointment of lead oxid (litharge), and in water and milk that have stood in lead receptacles. Or the child may become poisoned by improper medication, i. e., by taking lead acetate solution in treatment of poison oak; by sucking paint from the mother's face; by nursing after the use of lead ointment on the mother's breast, or by swallowing leaden objects, such as BB shot.

Holt⁴ reported one case due to inhalation of dust that contained lead. He also stated that the number of authentic cases of lead poisoning in young infants was very small, although much has been written on congenital lead poisoning in extensive studies of the families of workers in lead, in which, in many instances, both parents were suffering from lead poisoning.

Before taking up the general discussion and symptomatology of lead poisoning in children, I shall report two cases, which have come under my observation during the last year:

REPORT OF CASES

CASE 1.—A boy, aged 3 years, normal at birth and always well except for nervousness, caused by irregular sleeping hours and careless parents, for six months previous to observation had complained of severe abdominal cramps associated with vomiting, loss of weight and appetite, listlessness, irritability, night cries, bed wetting and progressive anemia. The abdominal cramps were usually in the afternoon and evening, and were becoming more frequent.

The child had been observed by several physicians in their offices, and in one instance the diagnosis of appendicitis was made. The correct diagnosis, however, might have been made by any one who called at the child's home, because every bit of paint on porch railings, window sills, crib, bureau, chairs and even white enameled door casings, as high as the child could reach, had been gnawed off.

The child was fairly well nourished, and was pale and pot-bellied, but of normal height. There was a distinct lead line on the gums. The abdomen was relaxed, and no tenderness was elicited. The spleen was just felt. There was no paralysis.

The laboratory data were: white blood corpuscles, 15,400; red blood corpuscles, 4,000,000; hemoglobin, 50 per cent. A differential count was normal. There was no stippling, and the red cells showed only the usual evidence of a secondary anemia.

Lead was recovered from both the urine and the feces.

4. Holt, L. E., Jr.: Lead Poisoning in Infancy, *Am. J. Dis. Child* 25: 229 (March) 1923.

1. Still, G. F.: *Common Disorders and Diseases of Childhood*, London, Henry Frowde, 1915, pp. 779-782.

2. Thomson, John: On Pica, or Dirt Eating, in Children, *Edinburgh Hosp. Rep.* 31: 81-94, 1895.

3. Koplik, Henry: *The Diseases of Infancy and Childhood*, Philadelphia, Lea & Febiger, 1918, p. 791.

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LEAD POISONING—RUDDOCK

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As this child would not gnaw varnished objects, it was easy to remove the source of lead by re-finishing the entire house; within two weeks, except for some anemia, the child was normal.

CASE 2.—A girl, aged 2 years, normally developed, who had always been well, during the last month had had three series of convulsions. The mother stated that the child gnawed the paint from the crib and the window sills, and had an uncontrollable desire to chew any painted object.

Examination was negative, except for a slight degree of internal strabismus of the left eye, which the mother had not noted before.

The red blood corpuscles numbered 4,520,000 per cubic millimeter; the hemoglobin was 60 per cent. No stippling was seen. No lead was found in the urine, but there were traces of lead in the feces on two examinations.

Since the source of lead has been removed, this child has made an uneventful recovery and is now normal.

SYMPTOMATOLOGY³

The history of the case is very important and may lead to an easy diagnosis after all other methods have proved failures. The appearance of the child is usually striking in the chronic cases of lead poisoning, on account of the degree of secondary anemia concomitant with it.

In the early cases, a change of disposition is often the first symptom noticed. The child becomes fretful, peevish and restless at night; the appetite becomes poor; the breath is foul, and frequently hemorrhages occur from the gums. The child may complain of pains in the epigastrium and the legs.

In chronic cases, the pains in the abdomen become continuous and more severe. Constipation is present. Vomiting is rare. There is usually a degree of anemia present, but occasionally the parents overlook all manifestations until the development of cerebral disturbances. Very few cases reported show the characteristic wrist-drop so common in adults, but paralysis of the cranial nerves, particularly the facial and motor oculi nerves, is common. Gibson⁴ reported a group of eye symptoms, which he referred to as "plumbic ocular neuritis."

The temperature, as a rule, is normal except in the fatal cases and in those with cerebral disturbances, in which it may reach 105 or 106 F. before the death of the patient.

Of the various forms of cerebral disorders produced by lead, convulsive seizure is by far the most common. There is no way, however, to distinguish the convulsions due to lead poisoning from those due to other causes. All types of convulsions are seen. They may be local or general; they may be mild or severe. The convulsions do, however, have one characteristic, and that is that they are very persistent and are attended by a high mortality.

Colic has been more frequently observed than paralysis, but more common than either are irritability, restlessness and a blue line on the gums. It should be remembered, however, that a continuous lead line is seldom found. The characteristic appearance is the presence of minute black dots in the margin of the mucous membrane around the teeth, which are covered with tartar. It is difficult to see, and a hand lens should be used.

The blood picture, except for a secondary anemia, usually tells us nothing, although this is determined by the duration of the poisoning. If the disease has been

present for some time, stippling of the red blood corpuscles may occur.

Direct evidence of lead poisoning is obtained by the demonstration of lead in the urine and feces. There is, however, always a larger amount in the feces than in the urine, and it is often present in the feces when it cannot be demonstrated in the urine.

CLASSIFICATION INTO GROUPS

Because of the variability of the sensitiveness of children to lead, a classification into definite groups is difficult, and when made must be based largely on the severity of the predominating system.

Turner divides lead poisoning in children into four groups:

1. Paralytic cases showing symmetrical wrist-drop, foot-drop, spasm of the calf muscles and, as a secondary lesion, a persistent talipes equinus.

2. Cases characterized by pains in the abdomen and limbs, concomitant with habitual constipation, and occasionally a blue line on the gums. These children are usually irritable and neurotic.

3. Children suffering from ocular neuritis; viz., a neuritis involving the optic, oculomotor, abducens and trochlear nerves.

4. Eclamptic cases with severe and persistent convulsions, which often end fatally.

We are indebted to Australian observers for most of the literature regarding lead poisoning in children, and for an historical account of lead poisoning in the children of Queensland, from the first recorded cases in 1892 to 1917. A recent article⁵ records the probable success of efforts to have an act passed prohibiting the use of white lead paint on veranda railings and outside surfaces within the reach of children's fingers.

TREATMENT⁶

The treatment of this condition may be considered from three points of view:

1. Prophylactic. Careful supervision of children during the age (from 1 to 3 years) in which pica is most prevalent. Wide publicity and education through periodicals and home journals, in order to educate the parents.

2. Symptomatic. For the spasms, chloroform inhalation is the only agent that is efficacious; for the anemia, the various forms of iron and arsenic; for the local palsies, massage and electricity, and for the permanent palsies, surgery should be resorted to, if possible.

3. Medicinal. For the removal of lead from the system, potassium iodid, from 3 to 5 grains (0.2 to 0.3 gm.) three times daily with a morning purge of magnesium sulphate may be tried. This is the usual method, although in severe cases it is not well to give the iodids, since, according to some writers, the liberation of the lead that has been deposited in the tissues may increase the severity of the symptoms.

Gibson,⁴ in 1919, reported successful treatment by the two-bath ionization system. The patient places both hands on an aluminum plate immersed in a salt solution, which is connected with the negative pole, and both feet likewise in a similar bath, which is connected with the positive pole. From 7½ to 8½ volts and 25

3. A complete description of the symptomatology of lead poisoning in children is given by Blackfan, K. D.: *Lead Poisoning in Children*, Am. J. M. Sc. 232:877 (June) 1917.

4. Gibson, J. L.: *The Diagnosis, Prophylaxis and Treatment of Plumbic Ocular Neuritis Amongst Queensland Children*, M. J. Australia 24:201 (Sept. 3) 1917; *On the Importance of De-Ionization in the Treatment of Plumbism in Queensland Children*, ibid. 1:272 (April 5) 1919.

5. Considerable urine is evaporated to dryness, and 50 c.c. of fuming nitric acid is added; after the reaction has subsided, it is allowed to simmer over the free flame for one-half hour, and then 25 c.c. more acid is added three times each fifteen minutes. The fluid is then evaporated to a small volume, neutralized with sodium hydroxid, filtered and the lead tested with hydrogen sulphid, which will give a brown precipitate.

6. An Historical Account of the Occurrence and Causation of Lead Poisoning Among Queensland Children, M. J. Australia 1:148 (Feb. 11) 1922.

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INJURY OF RECTUM—MARBURY

JOHN A. M. A.
MAY 24, 1924

milliamperes of current given for one-half hour, daily. The lead is deposited on the aluminum plate in the negative bath.

CONCLUSIONS

1. Pica is one of the most important etiologic factors in lead poisoning in children.

2. There are many mild cases of lead poisoning in children, manifested by spasms or colic, the true nature of which are never suspected.

3. Energetic prophylactic measures should be employed with children who habitually eat painted articles.

4. The feces and the urine should be examined for lead in all children with convulsions in which the etiologic factor is not clear.

1002 Pacific Mutual Building.

TRAUMATIC INJURY OF THE RECTUM

REPORT OF CASE *

W. B. MARBURY, M.D.
WASHINGTON, D. C.

In looking over the literature, including that at the Surgeon General's Library, on injuries to the rectum, I have been unable to find a case similar to the one reported here. Gant epitomizes sixty-four cases that fell under his personal observation. These may be classified as:

1. Those due to injury from foreign bodies, either during instrumentation or as result of accident. Among these, fish-hooks, gunshot wounds, falling on palms or stabs, and use of the speculum and dilators play a conspicuous part.

2. Rupture of the sphincter due to pederasty, or of the perineum during parturition. Rupture of the rectovaginal septum during copulation.

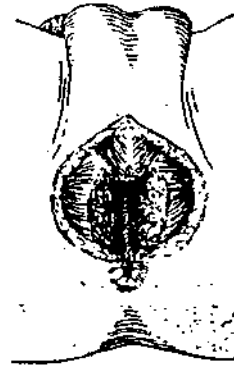


Fig. 1.—Muscles in front of the rectum torn and retracted, with the latter practically intact.

3. Rectovaginal rent induced by rectal distention with an air-filled rubber bag.

4. Perforation of the rectum by foreign bodies left in the abdomen.

5. Ulceration due to phenol (carbolic acid) or turpentine.

The anatomic structures most usually affected are the rectum or sigmoid, urethra, one or both sphincters, the levator ani muscle, and the rectovaginal septum.

Only in one case was there mention of any injury to the bony structure, and that was when the coccyx was fractured as the result of a kick during a fight.

The unique features about the case reported here are that all the muscles about the rectum were torn and retracted, leaving the intestine intact; rupture of the urethra, and fracture of the descending ramus of the pubes.

REPORT OF CASE

L. S., a colored boy, aged 4 years, while riding on the running board of a truck, Aug. 11, 1923, was thrown as the

* Read before the Virginia, Maryland and District of Columbia Medical Society, Dec. 3, 1923.

truck entered an alley, and crushed between it and the side of a brick wall. He was brought soon afterward to the emergency hospital. The pupils were equal and reacted to light and in accommodation. The tongue was coated. The teeth and gums were in good condition. The tonsils were slightly enlarged, but the pharynx was otherwise normal. There were several palpable cervical glands. The chest was firm, and expansion was good and equal on the two sides. The percussion note was resonant throughout. The breath sounds were clear. The heart was not enlarged, but there was a soft systolic murmur at the apex not transmitted to the axilla. The abdomen was slightly distended and tender in the region of the bladder. About the rectum, running anteriorly, there was a laceration, which seemed to go through the sphincter. There was much bleeding, some of which appeared to come from high up in the rectum. An attempt was made to pass a catheter, but this was unsuccessful and only a little blood was obtained. On further examination it was found that the laceration at the rectum included both sphincters and part of the perineal raphe. The finger could be passed upward anterior to the mucous membrane of the bowel, and the sharp edges of the broken ramus on the right side could be felt. Also the urethra was torn in this region, and urine was passed through the opening. Whenever the patient's legs were moved in any way, he experienced great pain in the region of the symphysis. Once when we were moving the patient, the whole pelvis was seen to rock irregularly, and definite crepitus was obtained. There was no evidence of injury to the extremities.

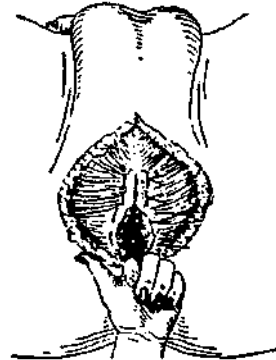


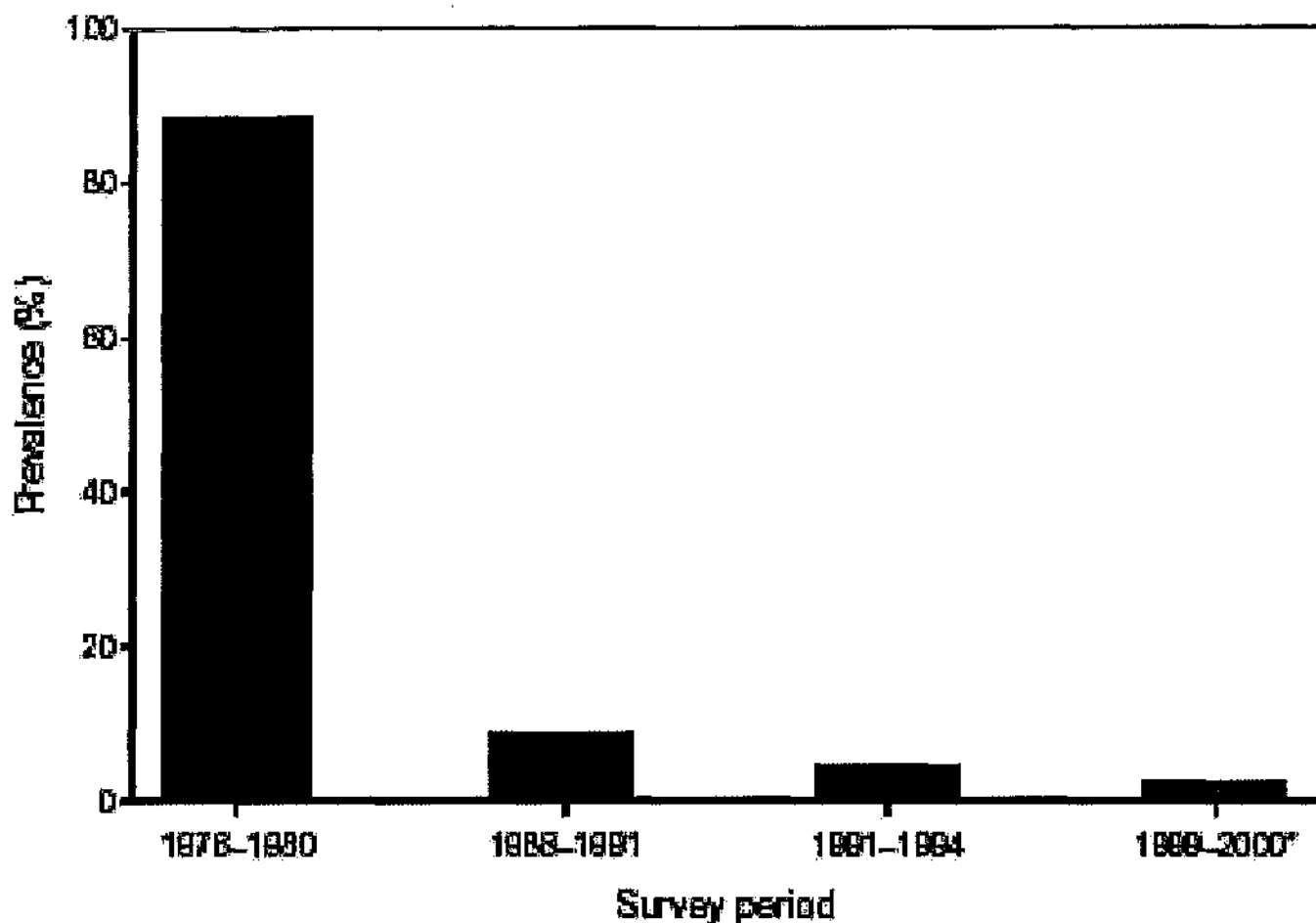
Fig. 2.—Muscles being sutured over the rectum en masse; area between sutured muscle and skin packed lightly with gauze (not shown).

The patient was sent to the operation room. Under general anesthesia, and with the patient in the lithotomy position, a more thorough examination was made. The rectal tube was found practically intact, but the muscles anterior to it were torn and retracted on both sides so as to appear only laterally, and retracted far back toward the posterior surface. The rough edges of the fractured descending ramus of the pubes could be felt. The opening in the urethra was not made out. The torn sphincter muscles and the levator ani were brought together over the midline anteriorly and sutured with chromic catgut. This procedure was facilitated by keeping one finger in the rectal tube. The area above this was left open, and a small amount of packing was placed in the space for drainage and to control oozing. A cast was applied from the knees to the costal margin.

Dr. Groover reported that examination of the lumbar spine, pelvis and hips showed the presence of a dislocation of the right innominate bone, with a fracture of the descending ramus of the pubes on the right side; the pubic symphysis was separated approximately 1 inch and there was, of course, necessarily some compensatory damage in the sacro-iliac joints.

The following day the patient seemed a little better, though very restless. There was some abdominal distention, and he complained of the cast being too tight. This was loosened. There was copious drainage of pus from the peroneal wound, and the temperature rose to 102.2 F. He voided involuntarily and through the urethral rent. Two hundred cubic centimeters of physiologic sodium chlorid solution was given by hypodermoclysis. August 14, he had an involuntary defecation. He had a frequent desire to urinate by the normal route, but without success. August 28, or seventeen days after admission, there were some signs of the patient's regaining control of the bowels, but the urethral fistula persisted.

FIGURE 1. Blood lead levels ≥ 10 $\mu\text{g/dL}$ among children aged 1–5 years — United States, 1976–1980, 1988–1991, 1991–1994, and 1999–2000*



Source: National Health and Nutrition Examination Surveys (NHANES).

Note: In 1991, NHANES III Phase 1 was completed and Phase 2 was begun.

* Data for 1999–2000 are highly variable (relative standard error $>30\%$).

Exhibit 875

TABLE 1. Estimated prevalence* of children aged 1–5 years with blood lead levels (BLLs) ≥ 10 $\mu\text{g/dL}$ [†] — United States, 1976–1980, 1988–1991, 1991–1994, and 1999–2000

Survey	Prevalence of BLLs ≥ 10 $\mu\text{g/dL}$		Children with BLLs ≥ 10 $\mu\text{g/dL}$	
	(%)	(95% CI) [§]	No.	(95% CI)
1976–1980	88.2	(83.8–92.6)	13,500,000	(12,800,000–14,100,000)
1988–1991	8.6 [¶]	(4.8–12.4)	1,700,000	(960,000–2,477,000)
1991–1994	4.4	(2.9–6.6)	880,000	(590,000–1,330,000)
1999–2000	2.2 ^{**}	(1.0–4.3)	434,000 ^{**}	(189,000–846,000)

Source: National Health and Nutrition Examination Surveys (NHANES).

Note: In 1991, NHANES III Phase 1 was completed and Phase 2 was begun.

* Estimated number of children aged 1–5 years with BLLs ≥ 10 $\mu\text{g/dL}$ divided by estimated population of children aged 1–5 years.

[†] CDC has determined BLL ≥ 10 $\mu\text{g/dL}$ is a level of concern.

[§] Confidence interval.

[¶] Estimate differs slightly from that published previously because of updates in coding and weighting of survey data.

^{**} Data for 1999–2000 are highly variable (relative standard error $>30\%$).

Exhibit 876

From: Richard Gaeta
To: Amy Murphy
Date: 5/4/05 9:34AM
Subject: Aldermanic Approach

The slow down in applications can put us in trouble if we don't come up with some innovative ways to stimulate more demand. In looking at our target area, it is clear that Alderman McGee covers the largest portion of our target area. His district also has the second highest prevalence rate in the city (23.7%).

I would like permission to approach the alderman to discuss what he can do with us to address this problem. In particular, we could draft a letter on his behalf and send it to property owners that own property in his district. We would send it out from our office and he would lots of kudos. This approach was so successful with Witkowiak, that 6 months after the letter went out we were still receiving applications from that mailing. The Hines letter that Catrina received is also netting lots of applications.

If Vivian wants to accompany me to the alderman's office, it may be a good opportunity for Vivian to take a more active role in promotion of our program. Ideas?

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CC: Mary Smith; Peter Skiba

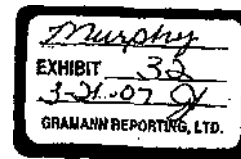


Exhibit 897

MKE 0014750

jgfinal

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Scene	Designation	Source	Tx Duration	Elapsed	Remains	Barcode
1	6:13 -6:20	Girard, James 2006-07-28	00:00:10	00:00:00	01:09:15	jgfinal.1
		6:13 Q: Good morning, Dr. Girard.				
		6:14 A: Good morning, Mr. Lopata.				
		6:15 Q: We've met before in another case,				
		6:16 correct?				
		6:17 A: That's correct.				
		6:18 Q: You understand that you are under oath				
		6:19 today?				
		6:20 A: I do.				
2	11:18 -12:16	Girard, James 2006-07-28	00:00:40	00:00:10	01:09:05	jgfinal.2
		11:18 Q: Dr. Girard, Exhibit Number 82 is				
		11:19 entitled report of Dr. James E. Girard, is that				
		11:20 accurate?				
		11:21 A: It is.				
		11:22 Q: Is this your expert report in the City				
		11:23 of Milwaukee case?				
		11:24 A: It is.				
		11:25 Q: Does the report marked as Exhibit 82				
		12:1 contain all of the opinions that you intend to				
		12:2 offer at trial?				
		12:3 A: It does.				
		12:4 Q: So it would be accurate to say that				
		12:5 you do not intend to offer any opinions that are				
		12:6 not already stated in this report, correct?				
		12:7 A: Correct.				
		12:8 Q: Okay. This is your final report,				
		12:9 correct?				
		12:10 A: Correct.				
		12:11 Q: Okay. Were there any prior drafts of				
		12:12 this report?				
		12:13 A: No, there weren't.				
		12:14 Q: Okay. Did you draft this report?				
		12:15 A: I -- I drafted this together with				
		12:16 Mr. Lewis.				
3	16:7 -16:12	Girard, James 2006-07-28	00:00:16	00:00:50	01:08:25	jgfinal.3
		16:7 Q: Okay.				
		16:8 A: I might want to add that some part of				
		16:9 what paint is and the life cycle of paint will				
		16:10 also involve pigments and what pigments were				
		16:11 available, just to make certain that you're				
		16:12 clear on what I'm telling you.				

Exhibit 960

jgfinal

4 19:21-20:7 Girard, James 2006-07-28 00:00:17 00:01:06 01:08:09 jgfinal.4

19:21 (Girard Exhibit No. 84 was marked for
19:22 identification.)
19:23 BY MR. LOPATA:
19:24 A: copy of your current CV is what has
19:25 been marked 84?
20:1 A: Yes, sir.
20:2 Q: In the lower right-hand corner, it has
20:3 a date designation of June 2006, correct?
20:4 A: Correct.
20:5 Q: Is this the most recent copy of your
20:6 CV?
20:7 A: It is.

5 26:16-26:24 Girard, James 2006-07-28 00:00:48 00:01:23 01:07:52 jgfinal.5

26:16 Q: What -- well, let's start this way:
26:17 What lead pigments do you plan on testifying
26:18 about at trial?
26:19 A: Well, at the time that I wrote the
26:20 report, I planned to talk about white lead
26:21 carbonate, zinc oxide, mixed lead/zinc pigments,
26:22 titanium dioxide pigments. And, since many of
26:23 these things contain silicates, probably
26:24 silicates as well.

6 28:9-28:13 Girard, James 2006-07-28 00:00:17 00:02:11 01:07:04 jgfinal.6

28:9 Q: Okay. As far as nonlead pigments go,
28:10 which nonlead pigments will you be testifying
28:11 about at trial?
28:12 A: Zinc oxide, titanium dioxide, and I
28:13 think that will be it.

7 34:7-34:16 Girard, James 2006-07-28 00:04:20 00:02:28 01:06:47 jgfinal.7

34:7 Q: Okay. Dr. Girard, I'm going to ask
34:8 you whether you recognize certain writers as
34:9 authoritative and reliable experts in the field
34:10 of paint chemistry. I'm going to start with
34:11 your own reliance materials. Do you understand?
34:12 A: Okay.
34:13 Q: Looking at Exhibit 1, do you recognize
34:14 Alvah Sabin as an authoritative and reliable
34:15 expert in the field of paint chemistry?
34:16 A: Yes, I do.
34:17 Q: Okay. You were unfamiliar with
34:18 Mr. Sabin at your 2005 deposition, correct?
34:19 A: Correct.
34:20 Q: Since that time, counsel has provided

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34:21 you with a copy of Mr. Sabin's book on white
34:22 lead, correct?
34:23 A: Correct.
34:24 Q: Okay. And you now recognize Mr. Sabin
34:25 as authoritative and reliable?
35:1 A: Yes.
35:2 Q: Okay. You continue to recognize
35:3 Frederick Brown --
35:4 A: Yes.
35:5 Q: -- as an authoritative and reliable
35:6 source in paint chemistry, correct?
35:7 A: I do.
35:8 Q: Do you continue to recognize his
35:9 employer, the Forest Products Laboratory, in
35:10 Madison as an authoritative and reliable source
35:11 in paint chemistry?
35:12 A: I do.
35:13 Q: And do you recognize, generally, the
35:14 higher government body, the U.S. Department of
35:15 Agriculture, as an authoritative and reliable
35:16 source in paint chemistry?
35:17 A: I do.
35:18 Q: Okay. Do you recognize the Bureau of
35:19 Chemistry within the Department of Agriculture
35:20 as an authoritative and reliable source in the
35:21 field of paint chemistry?
35:22 A: Yes.
35:23 Q: Okay. And do you recognize the Office
35:24 of Price Administration as an authoritative and
35:25 reliable source in the field of paint chemistry?
36:1 A: I do.
36:2 Q: Okay. Exhibit 2 is portions of G.P.A.
36:3 Turner's book. Do you recognize G.P.A. Turner
36:4 as an authoritative and reliable expert in the
36:5 field of paint chemistry?
36:6 A: Yes, I do.
36:7 Q: Okay. And referring to Exhibit 3,
36:8 E.W. Boughton, do you recognize Mr. Boughton as
36:9 an authoritative and reliable expert in the
36:10 field of paint chemistry?
36:11 A: Yes.
36:12 Q: Mr. Boughton worked for the U.S.
36:13 Government National Bureau of Standards,
36:14 correct?
36:15 A: Correct.
36:16 Q: Do you recognize the National Bureau

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36:17 of Standards as an authoritative and reliable in
36:18 the field of paint chemistry?
36:19 A: I do.
36:20 Q: Do you recognize Eugene F. Hickson,
36:21 also of the National Bureau of Standards, as an
36:22 authoritative and reliable expert in the field
36:23 of paint chemistry?
36:24 A: I do.
36:25 Q: Do you recognize Percy Walker, also
37:1 with the National Bureau of Standards, who often
37:2 wrote with Mr. Hickson, as an authoritative and
37:3 reliable expert in the field of paint chemistry?
37:4 A: I do.
37:5 Q: Okay. And do you recognize George
37:6 Burgess, director of the National Bureau of
37:7 Standards, as an authoritative and reliable
37:8 expert in the field of paint chemistry?
37:9 A: I do.
37:10 Q: Do you recognize the federal General
37:11 Services Administration as authoritative and
37:12 reliable in the field of paint chemistry?
37:13 A: I have to be honest to say I don't
37:14 remember them.
37:15 Q: So you don't know one way or the
37:16 other?
37:17 A: I just don't know one way or the
37:18 other.
37:19 Q: Okay. Referring to Exhibit 10, you
37:20 identify a source from the U.S. Bureau of Mines.
37:21 Do you recognize the U.S. Bureau of Mines as
37:22 authoritative and reliable in the field of paint
37:23 chemistry?
37:24 A: I certainly think they're reliable in
37:25 what they do in terms of -- I think Exhibit 10
38:1 is a mineral resources document.
38:2 Q: Yes, it is.
38:3 A: And I certainly -- I certainly think
38:4 with regard to that, the Bureau of Mines is
38:5 quite reliable.
38:6 Q: Are there areas where you think the
38:7 Bureau of Mines is not reliable?
38:8 A: Well, to be honest with you, I don't
38:9 know how much they have done in paint. That's
38:10 the only thing that I'm -- I'm hedging on. In
38:11 terms of pigments, I'm sure -- and minerals, I'm
38:12 sure they are very authoritative.

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38:13 Q: So you would recognize the U.S. Bureau
38:14 of Mines as authoritative when it comes to
38:15 testimony about pigments, correct?
38:16 A: Correct.

8	39:6-44:15	Girard, James 2006-07-28	00:04:20	00:06:48	01:02:27	jgfinal.8

39:6 Q: Okay. Would you agree that a primary
39:7 function of paint includes protecting surfaces?
39:8 A: I would.
39:9 Q: Would you agree that a primary
39:10 function of paint includes making surfaces
39:11 washable for hygiene and good health?
39:12 A: I do.
39:13 Q: Would you agree that consumers want
39:14 paints to last?
39:15 A: I do.
39:16 Q: Would you agree that consumers
39:17 especially want outside paints to last?
39:18 A: Yes.
39:19 Q: Would you agree that some paints are
39:20 formulated to achieve durability?
39:21 A: I do.
39:22 Q: Would you agree that other paints are
39:23 formulated to achieve certain colors?
39:24 A: Yes.
39:25 Q: Would you agree that some paints are
40:1 formulated to achieve drying time?
40:2 A: Dry –
40:3 A: specific drying time?
40:4 A: Yes.
40:5 Q: Would you agree that some paints are
40:6 formulated to achieve a certain gloss?
40:7 A: Yes.
40:8 Q: Would you agree that some paints are
40:9 formulated to achieve certain opacity?
40:10 A: Yes.
40:11 Q: Do you agree that outside paints are
40:12 formulated different than inside paints?
40:13 A: Yes.
40:14 Q: Would you agree that one of the most
40:15 important traits for an exterior architectural
40:16 paint is durability of the paint against
40:17 sunlight and rain?
40:18 A: Yes.
40:19 Q: Do you agree that outside paints are
40:20 more expensive, all other things being equal.

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40:21 than paints for the inside of a home?
40:22 A: I'm not certain.
40:23 Q: You don't know one way or the other?
40:24 A: Right.
40:25 Q: Would you agree that outside paints
41:1 use more expensive ingredients, including
41:2 pigments, than paints made for the inside of
41:3 homes?
41:4 A: I would agree.
41:5 Q: Would you agree that you would not
41:6 typically expect someone to use an expensive
41:7 outside -- pigmented paint for the outside on
41:8 the inside of a home?
41:9 A: I would agree with that.
41:10 Q: Okay, Do you agree that some paints
41:11 are formulated to achieve a certain spreading
41:12 power?
41:13 A: Yes, I do.
41:14 Q: Would you agree that some paints are
41:15 formulated to achieve a certain degree of grind?
41:16 A: Could you spell that word, please.
41:17 Q: G-R-I-N-D.
41:18 A: Yes.
41:19 Q: Are you familiar with what grind is in
41:20 paint formulation?
41:21 A: The way I understand it, pigments are
41:22 ground to give a certain size particle that is
41:23 being suspended in the -- in the vehicle.
41:24 Q: Do you know what degree of grind is in
41:25 paint formulation?
42:1 A: I would have to look it up.
42:2 Q: You agree that some paints are
42:3 formulated to achieve a certain method of
42:4 application?
42:5 A: Yes.
42:6 Q: Do you agree that some paints are
42:7 formulated to achieve a certain shelf life?
42:8 A: Yes.
42:9 Q: Do you agree that some paints are
42:10 formulated to achieve a certain flow?
42:11 A: Yes.
42:12 Q: Do you agree that some paints are
42:13 formulated to achieve a certain consistency?
42:14 A: Yes, I would.
42:15 Q: Do you agree that some paints are
42:16 formulated to achieve a certain cost?

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42:17 A: Yes.
42:18 Q: Do you agree that some paints are
42:19 formulated to achieve a certain hiding power?
42:20 A: Yes.
42:21 Q: Would you agree that some paints are
42:22 formulated to achieve a certain amount of
42:23 lightfastness?
42:24 A: Yes.
42:25 Q: Do you understand what lightfastness
43:1 means?
43:2 A: I would -- my understanding of that is
43:3 that it has to do with its ability to fade.
43:4 Q: A paint that is lightfast is one that
43:5 does not fade, correct?
43:6 A: Correct.
43:7 Q: Would you agree that some paints are
43:8 formulated to achieve a certain chalking
43:9 tendency?
43:10 A: Yes.
43:11 Q: Do you agree that some paints are
43:12 formulated to achieve certain wetting
43:13 characteristics?
43:14 A: Yes.
43:15 Q: The bottom line of all the questions I
43:16 just asked you is that there are trade-offs that
43:17 need to be made in formulating paints, correct?
43:18 A: Correct.
43:19 Q: Sometimes you can trade the price of a
43:20 paint for durability when formulating, correct?
43:21 A: Yes.
43:22 Q: Other times you have to trade hiding
43:23 power for color, correct?
43:24 A: Correct.
43:25 Q: Paint manufacturers provide options
44:1 for painters and consumers. And then those
44:2 people get to choose what kind of paint they
44:3 want for a particular purpose, correct?
44:4 MR. LEWIS: Object to foundation.
44:5 Scope. Please answer.
44:6 THE WITNESS: Consumers can choose
44:7 that, yes.
44:8 BY MR. LOPATA:
44:9 Q: Okay. And paint manufacturers provide
44:10 the options so that consumers or painters can
44:11 choose the type of paint that meets their needs,
44:12 correct?

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44:13 MR. LEWIS: Foundation and scope.
44:14 Please answer.
44:15 THE WITNESS: Yes.

9	59:18-61:11	Girard, James 2006-07-28	00:01:54	00:11:08	00:58:07	jgfinal:10

59:18 Q: Dr. Girard, let me ask first a
59:19 question of clarification. My question to you
59:20 is not whether or not zinc oxide was used as a
59:21 paint pigment earlier in the century, okay?
59:22 A: Okay.
59:23 Q: My question to you was whether or not
59:24 you would agree -- do you agree that exterior
59:25 paints historically contained lead pigment long
60:1 after interior paints, because it took more
60:2 decades -- many more decades for paint chemists
60:3 to learn how to make a durable exterior paint
60:4 without lead pigment?
60:5 MR. LEWIS: Objection. Foundation and
60:6 scope and speculation. Please answer.
60:7 THE WITNESS: I wouldn't disagree with
60:8 that statement.
60:9 BY MR. LOPATA:
60:10 Q: Okay. What you were saying earlier
60:11 was that zinc oxide was used as a pigment,
60:12 correct?
60:13 A: Correct.
60:14 Q: Okay. Zinc oxide cannot be used alone
60:15 in an exterior paint, correct?
60:16 A: I think that traditionally it wasn't,
60:17 yes.
60:18 Q: Okay. In fact, if lead-free zinc
60:19 oxide was used as a paint pigment with a vehicle
60:20 such as linseed oil, what would happen is the
60:21 paint would prematurely crack, correct?
60:22 A: It was a much harder material and it
60:23 would crack, yes.
60:24 Q: Okay. You were not trying to
60:25 suggest -- strike that.
61:1 Paint made from lead and oil possesses
61:2 unique qualities that occur when the lead and
61:3 oil react with one another, correct?
61:4 A: Correct.
61:5 Q: If a consumer wanted to use an
61:6 oil-based paint, then some amount of lead was
61:7 desirable, correct?
61:8 MR. LEWIS: Object to foundation.

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61:9 Scope. Speculation. Please answer.
 61:10 THE WITNESS: For the performance of
 61:11 the paint, yes.

10	122:5 - 122:15	Girard, James 2006-07-28	00:00:29	00:13:02	00:56:13	jgfinal.11
	122:5	Q: Okay. One of the reasons that lead				
	122:6	has -- was historically combined with zinc oxide				
	122:7	was that lead provided some of the good soft				
	122:8	traits that, when combined with the zinc oxide,				
	122:9	created a good durable paint film, correct?				
	122:10	A: It made the -- it made the film more				
	122:11	elastic, yes.				
	122:12	Q: It made the film -- leaded zinc oxide				
	122:13	made the paint more elastic than it would have				
	122:14	been without the lead, correct?				
	122:15	A: Yes.				
11	124:5 - 130:5	Girard, James 2006-07-28	00:06:26	00:13:31	00:55:44	jgfinal.12
	124:5	Q: Chalking is -- when paint is about to				
	124:6	start chalking, it's visible to the human eye,				
	124:7	correct?				
	124:8	A: Yes.				
	124:9	Q: And when somebody wants to maintain				
	124:10	their home, and they see that the paint on the				
	124:11	outside of their home is about to start				
	124:12	chalking, they can prepare the surface and				
	124:13	repaint over, correct?				
	124:14	A: They can do that.				
	124:15	MR. LEWIS: Objection to foundation.				
	124:16	Calls for speculation. Please answer.				
	124:17	BY MR. LOPATA:				
	124:18	Q: Once old paint is covered over, the				
	124:19	chalking process stops, correct?				
	124:20	MR. LEWIS: Object to foundation.				
	124:21	Speculation. Please answer.				
	124:22	THE WITNESS: It no longer -- chalking				
	124:23	no longer occurs on the surface.				
	124:24	BY MR. LOPATA:				
	124:25	Q: Okay. Chalking doesn't occur				
	125:1	underneath the surface because it no longer has				
	125:2	access to oxygen or sunlight, correct?				
	125:3	A: Correct.				
	125:4	Q: Okay. And chalking is an exterior				
	125:5	phenomenon, correct?				
	125:6	A: Correct.				
	125:7	Q: Okay. Chalking does not happen on the				
	125:8	inside of homes, correct?				

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125:9 A: Right.
125:10 Q: And when we look at the possible
125:11 causes that are listed, they say use of an
125:12 interior paint for an outdoor application,
125:13 erosion of factory finished metal siding, and
125:14 use of a lower quality highly pigmented paint.
125:15 What, if anything, would you be saying
125:16 about that other than sort of what that states?
125:17 A: Nothing.
125:18 Q: Okay. It would be wrong to apply an
125:19 interior paint to an -- to the outside of a
125:20 home, correct?
125:21 A: Yes.
125:22 Q: It would be wrong to apply an outside
125:23 paint to the interior of a home, correct?
125:24 A: Correct.
125:25 Q: And it would be -- that would be
126:1 essentially the misuse of a product, correct?
126:2 MR. LEWIS: Objection to scope. Calls
126:3 for legal conclusion. Please answer.
126:4 THE WITNESS: People make mistakes,
126:5 you know, but it would be using the wrong
126:6 product for the wrong application.
126:7 BY MR. LOPATA:
126:8 Q: Okay. It would not be -- something
126:9 like that would not be -- sorry. Let me
126:10 rephrase the question. Use of the paint for a
126:11 purpose not intended would be -- if it failed,
126:12 would not be the fault of the paint, it would be
126:13 the fault of the person applying the paint,
126:14 correct?
126:15 MR. LEWIS: Objection to foundation.
126:16 Scope of expertise. Calls for speculation.
126:17 Please answer.
126:18 THE WITNESS: Yes, it would be the
126:19 cause of the person that applied it.
126:20 BY MR. LOPATA:
126:21 Q: Is there anything else that you would
126:22 talk about chalk rundown? With the exception of
126:23 we're going to go to the next page on chalking.
126:24 A: Okay. That's it.
126:25 Q: Okay. What if anything would you be
127:1 saying -- well, strike that.
127:2 This picture of chalking again you
127:3 can't really see, can you?
127:4 A: That's right.

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127:5 Q: Okay. We've gone over some of the
127:6 chalking issues over the last couple of minutes.
127:7 Is there anything else that you would be saying
127:8 about chalking?

127:9 A: You know, I would be talking about the
127:10 fact that it was well known that lead-based
127:11 paints chalked and that, you know, it was an
127:12 inevitable process that took place, as you said
127:13 yourself, because of sunlight, because of
127:14 exposure to air and water. And I would describe
127:15 to the jury how it takes place.

127:16 Q: Okay. Now, when you said inevitable
127:17 process, the exception there is if it's
127:18 covered -- you're just referring to a paint that
127:19 is left to stand indefinitely, correct?

127:20 A: Yes.

127:21 Q: Okay. You're not referring to
127:22 chalking being inevitable if the paint is well
127:23 maintained, covered over and repainted over
127:24 time, correct?

127:25 A: Well, certainly when you repaint, you
128:1 cover over the layer and you begin anew, okay.
128:2 So I would agree with you that repainting stops
128:3 the process.

128:4 Q: Okay. And so when you said the word
128:5 inevitable process, the assumption built into
128:6 that is that the paint is not maintained,
128:7 correct?

128:8 A: Well, I guess you and I are -- maybe
128:9 it's a semantic issue, okay. I would contend
128:10 that chalking is a process that takes place over
128:11 time and that, as it begins, it may not be
128:12 obvious the day it begins. And so chalking
128:13 progresses to a point at which it becomes
128:14 obvious. And once it becomes obvious, then
128:15 repainting is something that would stop it.

128:16 Q: We've already -- I don't want to go --
128:17 I want to make sure I'm clear. You've already
128:18 agreed that paint -- it is -- as paint is about
128:19 to start chalking, it is visible to the human
128:20 eye, correct? You've already testified to that?

128:21 MR. LEWIS: Objection.

128:22 Mischaracterizes his testimony. Please answer.

128:23 THE WITNESS: I think what I testified

128:24 to is that if you look at a surface that's

128:25 chalked, you can tell by looking at it, okay. I

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129:1 disagree that I actually stated you could, you
129:2 know, that the instant it starts to happen that
129:3 I can tell by visually inspecting it.
129:4 BY MR. LOPATA:
129:5 Q. If you maintain a -- if you go and
129:6 look at the outside of your house, the first
129:7 thing that happens before paint starts to chalk
129:8 is it starts to lose its gloss or shine?
129:9 A: Flattening starts to happen. Okay.
129:10 But that is a gradual process. It's not -- it's
129:11 not something that happens overnight.
129:12 Q: I hear you. You would agree -- and
129:13 this is us referring back to Frederick Brown --
129:14 that prior to chalking paint does visually start
129:15 to change in appearance. It begins to flatten,
129:16 correct?
129:17 A: Yes.
129:18 Q: Okay. And at that time, somebody who
129:19 is maintaining their home could choose to
129:20 repaint their home, correct?
129:21 MR. LEWIS: Objection to scope,
129:22 expertise, calls for speculation. Please
129:23 answer.
129:24 THE WITNESS: Homeowner can choose to
129:25 paint their house at any time. So, you know, if
130:1 they chose to do it then, that would be good.
130:2 BY MR. LOPATA:
130:3 Q. I want to make sure I understand this
130:4 inevitable word.
130:5 A: Okay.

12	130:6-132:10	Girard, James 2006-07-28	00:02:15	00:19:57	00:49:18	jgfinal.13
130:6	Q: Would you agree -- strike that.					
130:7	Inevitable suggests to me -- and I					
130:8	want to make sure we're on the same page, that					
130:9	it will always happen. It is inevitable, it					
130:10	will eventually happen.					
130:11	What I think I thought I heard you					
130:12	already testify was in fact chalking can be					
130:13	retarded and stopped. Would you agree in the					
130:14	first instance that chalking can be retarded and					
130:15	stopped?					
130:16	A: I would agree.					
130:17	Q: Okay. In instances where somebody					
130:18	either maintains the paint or paints over, those					
130:19	are instances where chalking is not inevitable.					

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130:20 correct?

130:21 A: Intervention will stop it.

130:22 Q: Okay.

130:23 A: I agree.

130:24 Q: Okay. Paint companies -- paint does

130:25 not last forever, correct?

131:1 A: Absolutely.

131:2 Q: The purpose of paint is to take the

131:3 brunt -- especially exterior paint, to take the

131:4 brunt of sunlight, rainwater, the elements, so

131:5 that the underlying foundation, such as the

131:6 wood, doesn't get deteriorated, correct?

131:7 A: Correct.

131:8 Q: Okay. When paint companies sell

131:9 paint, the people to whom they're selling know

131:10 that they will eventually have to repaint,

131:11 correct?

131:12 A: Yes.

131:13 MR. LEWIS: Object to foundation.

131:14 Scope of expertise, calls for speculation.

131:15 BY MR. LOPATA:

131:16 Q: One of the reasons that paint

131:17 companies provide information about paint

131:18 failure modes is because paint companies

131:19 recognize and teach consumers that they will

131:20 have to repaint, correct?

131:21 MR. LEWIS: Foundation and scope.

131:22 Please answer.

131:23 THE WITNESS: Yes, they do.

131:24 BY MR. LOPATA:

131:25 Q: Okay. Homeowners -- it would --

132:1 homeowners expect that paint will have a limited

132:2 lifespan, correct?

132:3 A: Yes.

132:4 Q: And homeowners expect that they will

132:5 have to repaint someday, correct?

132:6 A: Yes.

132:7 Q: And homeowners expect that repainting

132:8 is necessary to protect their investments,

132:9 correct?

132:10 A: Yes.

13	132:21-133:10	Girard, James 2006-07-28	00:00:31	00:22:12	00:47:03	jgfinal.14
	132:21	Q: Generally or typically, lead-based				
	132:22	paints don't fail by cracking, correct?				
	132:23	A: Most generally, yes.				

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132:24 Q: Okay. Generally or typically,
132:25 lead-based paints don't fail by flaking.
133:1 correct?
133:2 A: Yes.
133:3 Q: Correct?
133:4 A: Yes. Correct.
133:5 Q: Okay. Zinc oxide alone is a paint
133:6 that fails by cracking and flaking, correct?
133:7 A: Yes.
133:8 Q: And titanium dioxide is a paint that
133:9 fails by cracking and flaking, correct?
133:10 A: Yes.

14	144:20-145:6	Girard, James 2006-07-28	00:00:52	00:22:43	00:46:32	jgfinal.34

144:20 Q: Okay. Can I ask you to pull out your
144:21 expert report, and ask you to turn to the second
144:22 page, first paragraph, that reads: Some lead
144:23 paint has documented formulations that contain
144:24 more than 30 percent lead by weight, 30,000 ppm.
144:25 Lead paint surfaces even as it -- at a very low
145:1 end, 1 milligram per centimeter squared, would
145:2 burden a typical home with hazardous lead dust
145:3 in situations where the lead paint was sanded
145:4 and turned into dust.
145:5 Did I read that accurately?
145:6 A: You did.

15	147:7-148:14	Girard, James 2006-07-28	00:01:40	00:23:35	00:45:40	jgfinal.35

147:7 Q: Would it be fair to say that -- well,
147:8 let me ask you this: You're making a
147:9 quantitative decision that 1 milligram per
147:10 centimeter squared would burden a typical home,
147:11 correct?
147:12 A: Yes.
147:13 Q: And that's only when the paint is
147:14 sanded and turned into dust, correct?
147:15 A: I mean, it depends on who you read.
147:16 If you read Jacobs, for instance, he says that
147:17 definitely when it's sanded, that that is
147:18 certainly the case.
147:19 But in his paper, he also describes
147:20 either intact or nonintact painted surfaces that
147:21 he think -- he thinks are hazards.
147:22 Q: Okay. I guess I'm trying to
147:23 understand. What is it that -- with respect to
147:24 this paragraph, what is it that you're going to
147:25 actually testify about?

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148:1 A: Well, first off, I'm going to testify
148:2 about the amount of lead in paint. That's what
148:3 the first sentence is about, okay?
148:4 Q: Let's focus on the second sentence for
148:5 now.
148:6 A: Okay. The second sentence,
148:7 essentially what that sentence says is that,
148:8 even though some paint contained as much as
148:9 30 percent, you know, other lead paint surfaces
148:10 have a much lower concentration, such as 1
148:11 milligram per square centimeter. And that even
148:12 with these paints that have a low concentration
148:13 of lead, sanding them will turn the paint into
148:14 dust and contaminate the house.

16 149:11-149:12 Girard, James 2006-07-28 00:00:04 00:25:15 00:44:00 jgfinal.36

149:11 Q: The Jacobs article is Exhibit 9,
149:12 A: Okay. Good.

17 149:13-150:9 Girard, James 2006-07-28 00:01:31 00:25:19 00:43:56 jgfinal.37

149:13 Okay. If you turn to page A-605,
149:14 okay, the right-hand column, first full
149:15 paragraph that starts: This study. Okay? And
149:16 start with the second sentence, which I will
149:17 read.
149:18 Use of lead-safe work practices on
149:19 surfaces with lead-based paint is essential in
149:20 order to minimize dust, paint chips and
149:21 contaminated soil that may be generated during
149:22 maintenance and housing rehabilitation
149:23 activities, because only a small amount of
149:24 lead-based paint is needed to produce very high
149:25 dust lead levels.
150:1 For example, if sanded and turned into
150:2 contaminated dust that is spread across an
150:3 average size room, only 1 square foot of paint
150:4 at a lead concentration of 1 milligram per
150:5 square centimeter (the federal standard) is
150:6 needed to produce a settled dust level of
150:7 9,300 micrograms per square foot, several orders
150:8 of magnitude above current dust level standards
150:9 (U.S. HUD 1995).

18 164:23-167:14 Girard, James 2006-07-28 00:02:29 00:26:50 00:42:25 jgfinal.15

164:23 Q: You may testify about lithopone?
164:24 A: Yes.
164:25 Q: Okay. Do you know how lithopone is

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165:1 manufactured?
165:2 A: You know, the -- the methodology for
165:3 it?
165:4 Q: Uh-huh.
165:5 A: No, I don't.
165:6 Q: Would you agree, based on your
165:7 readings, that lithopone is not easily
165:8 manufactured?
165:9 A: I would agree on that.
165:10 Q: Okay. Would you agree that, today,
165:11 nobody uses lithopone in paint, correct?
165:12 A: Yes.
165:13 Q: Okay. Lithopone historically has had
165:14 shortcomings that have made it undesirable for
165:15 use in paint for certain purposes, correct?
165:16 A: Some of the literature that I have
165:17 reviewed indicates that, yes.
165:18 Q: Okay. Lithopone has poor durability
165:19 when it's exposed to outdoor sunlight, correct?
165:20 A: It is more I would say photochemically
165:21 active than some of the others, yes.
165:22 Q: Okay, so --
165:23 A: I'm agreeing with you, yes.
165:24 Q: -- take it one at a time. Lithopone
165:25 has poor durability when it's exposed to the
166:1 sun, correct?
166:2 A: Yes.
166:3 Q: Okay. It actually changes color from
166:4 white to gray to black when it's exposed to
166:5 sunlight, correct?
166:6 A: It can, yes.
166:7 Q: Okay. And in the existence of
166:8 sunlight, lithopone will wear very badly,
166:9 correct?
166:10 A: It has a poor wear function, yes.
166:11 Q: Okay. Lithopone paint, when it's used
166:12 outdoors, will flake-off almost immediately,
166:13 correct?
166:14 A: I wouldn't agree with almost
166:15 immediately, but it does flake more easily.
166:16 Q: Premature paint failure when lithopone
166:17 is used in exterior paint, correct?
166:18 A: It tends to fail faster.
166:19 Q: Lithopone was typically used as a flat
166:20 wall paint for the insides of homes, correct?
166:21 A: Yes, it was.

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166:22 Q: And if a consumer wanted to use or
166:23 paint a wall on the inside of their home that
166:24 had gloss, they couldn't use lithopone, correct?
166:25 A: I don't believe so.
167:1 Q: You're agreeing with me or you're not
167:2 sure?
167:3 A: I'm not absolutely certain. From
167:4 all -- that might be outside of the -- the
167:5 material that I've reviewed, I certainly know
167:6 it was used internally in flat paint. I don't
167:7 know about the glossy finished paint.
167:8 Q: Okay. Lithopone did not have a gloss
167:9 finish, correct?
167:10 A: That's one of its problems.
167:11 Q: Okay. And lithopone is not suitable
167:12 for windows because lithopone can't withstand
167:13 ultraviolet light, correct?
167:14 A: Yes.

19	168:8 -168:18	Girard, James 2006-07-28	00:00:55	00:29:19	00:39:56	jgfinal.31
	168:8	Q: Okay. Could I ask you to pull out				
	168:9	Exhibit 1, which is the book by Dr. Sabin --				
	168:10	A: Okay.				
	168:11	Q: -- on white lead.				
	168:12	A: I have to apologize. I've really got				
	168:13	this thing messed up. Can I put the expert				
	168:14	report aside now for a while?				
	168:15	Q: Sure. If I can ask you to turn to				
	168:16	page 54.				
	168:17	MR. LEWIS: Fifty-four?				
	168:18	MR. LOPATA: Fifty-four.				

20	168:19 -169:20	Girard, James 2006-07-28	00:01:05	00:30:14	00:39:01	jgfinal.16
	168:19	BY MR. LOPATA:				
	168:20	Q: Do you see that this is the section on				
	168:21	lead-free zinc oxide?				
	168:22	A: Yes.				
	168:23	Q: Can I ask you to look on page 55, the				
	168:24	sentence -- and tell me when you find the				
	168:25	sentence that starts naturally.				
	169:1	A: Got it.				
	169:2	Q: Okay. Would you -- do you agree that				
	169:3	naturally 1-1/2 times as many coats of zinc				
	169:4	paint as of lead are needed to get the same				
	169:5	covering effect, and this makes the former more				
	169:6	expensive to use, for the labor of applying a				
	169:7	coat of paint is two-thirds to three-quarters				

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169:8 the cost of the job. Furthermore, it takes more
 169:9 labor and more skill to use the white zinc.
 169:10 Do you agree with that?
 169:11 A: Except that there's a typo where it
 169:12 says naturally once and a half.
 169:13 Q: Okay. Other than the typo?
 169:14 A: Other than saying 1-1/2, which I'm
 169:15 sure you read -- you inferred the correct
 169:16 meaning of the sentence, I'm not disagreeing
 169:17 with that, but yes.
 169:18 Q: Would you agree that zinc oxide paint
 169:19 covers less surface area than lead-based paint?
 169:20 A: Yes, I would.

21	169:21 -171:13	Girard, James 2006-07-28	00:03:21 00:31:19 00:37:56	jgfinal.38

169:21 Q: Would you agree that zinc oxide paint
 169:22 requires more labor to --
 169:23 A: You know, just hang on a minute now.
 169:24 There is a document here. Let me just think for
 169:25 a minute. If we -- I know this is your
 170:1 deposition, too. But actually if you go to
 170:2 Cruikshank, okay?
 170:3 Q: Which exhibit is that?
 170:4 A: That's a good question. Whoever finds
 170:5 it first, yell out the number.
 170:6 Q: It's 39.
 170:7 A: Thirty-nine.
 170:8 Okay. Yes. On page 47. That's what
 170:9 I remember.
 170:10 Q: Uh-huh.
 170:11 A: Okay. So on page 47 of Exhibit 39,
 170:12 there's a table, which unfortunately doesn't
 170:13 have a number. And it's -- it's entitled table
 170:14 showing the comparative durability figures of
 170:15 various protective paints.
 170:16 Now, if you look at column D, it
 170:17 has -- I have to get out my magnifying glass
 170:18 unfortunately. Estimated number of times
 170:19 painted in a year -- in 20 years, excuse me.
 170:20 Estimated number of times painted in 20 years.
 170:21 So it says that for oxide of zinc,
 170:22 it's four. And for white lead, it's five.
 170:23 Okay?
 170:24 So this -- this data suggests that
 170:25 that oxide of zinc is more durable than lead,
 171:1 number one, okay. Because you -- you know, you

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171:2 only do it four times in 20 years versus five.
 171:3 And then if you go to the far -- I
 171:4 think it's a right column -- this is very hard
 171:5 to read and I apologize for this. It has all
 171:6 kinds -- okay. So E is the material cost. F is
 171:7 the labor cost. And G is the estimated overall
 171:8 cost. Okay?
 171:9 And he says that it's 87 -- I don't
 171:10 know if we're talking about pounds here or
 171:11 dollars, okay. But nonetheless, his data says
 171:12 that the overall cost of the oxide of zinc is
 171:13 less than lead. If I'm reading this right.

22 174:2-174:12 Girard, James 2006-07-28 00:00:33 00:34:40 00:34:35 jgfinal.39

174:2 Q: Okay. Separate from what he stated,
 174:3 what I want to know, in your opinion, do you
 174:4 believe that zinc oxide as a sole pigment used
 174:5 in paint was more durable than white lead
 174:6 carbonate?
 174:7 MR. LEWIS: Object to foundation and
 174:8 vagueness and time frame. Please answer.
 174:9 THE WITNESS: I mean, I think you're
 174:10 trying to put things in boxes: More/less,
 174:11 on/off. Okay. I certainly think it was a
 174:12 competitive product.

23 176:10-176:17 Girard, James 2006-07-28 00:00:20 00:35:13 00:34:02 jgfinal.17

176:10 Q: Okay. Do you agree that, if the
 176:11 characteristic properties of oxide of zinc are
 176:12 to be retained in the paint, ordinary raw
 176:13 linseed oil is quite inadmissible as a vehicle,
 176:14 by reason of its marked yellow color, which
 176:15 readily stains the oxide of zinc. Do you agree
 176:16 with that?
 176:17 A: Yes, I do.

24 178:9-178:15 Girard, James 2006-07-28 00:00:20 00:35:33 00:33:42 jgfinal.18

178:9 Q: Okay. Do you agree that the keenest
 178:10 supporter of oxide of zinc will, if he be honest
 178:11 and intelligent, find frequent occasion to
 178:12 recommend white lead for some specific purpose?
 178:13 Do you agree with that?
 178:14 A: I do. He seems to be a very balanced
 178:15 author.

25 178:16-179:3 Girard, James 2006-07-28 00:00:35 00:35:53 00:33:22 jgfinal.19

178:16 Q: Could you turn to page 31, and let me
 178:17 know when you're there.

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178:18 A: I'm there.
 178:19 Q: Do you agree, looking at the last
 178:20 sentence of the first paragraph, with the
 178:21 statement that it is a mistake on the part of
 178:22 manufacturers to imagine that oxide of zinc
 178:23 paints can or will be used in preference to
 178:24 other white pigments for every conceivable
 178:25 purpose in painting?
 179:1 A: I see that.
 179:2 Q: Do you agree with that?
 179:3 A: Yes.

26	179:4-180:3	Girard, James 2006-07-28	00:01:09	00:36:28	00:32:47	jgfinal.20
	179:4	Q: Okay. Could you turn to page 39.				
	179:5	Second paragraph that starts: On the other				
	179:6	hand.				
	179:7	A: Yes.				
	179:8	Q: Do you agree that a disadvantage of				
	179:9	many ready-mixed oxide of zinc paints is their				
	179:10	comparatively low opacity, which necessitates				
	179:11	several coats being applied before a colored				
	179:12	surface can be obscured. Did I read that				
	179:13	correctly?				
	179:14	A: You did.				
	179:15	Q: Do you agree with that?				
	179:16	A: I do.				
	179:17	Q: Okay. Could you turn to the next				
	179:18	page, page 40. Do you see in the second full				
	179:19	paragraph, it states, startling One, do you agree				
	179:20	that one of the most serious objections advanced				
	179:21	against oxide of zinc paint by the practical				
	179:22	painter is the difficulty of getting these				
	179:23	materials dry within a reasonable period, and it				
	179:24	must be admitted that considerable trouble has				
	179:25	arisen on this score when the painter has used				
	180:1	oxide of zinc for the first time.				
	180:2	Do you agree with that?				
	180:3	A: Yes.				
27	180:4-180:23	Girard, James 2006-07-28	00:00:42	00:37:37	00:31:38	jgfinal.21
	180:4	Q: Could you turn to page 53, please, and				
	180:5	let me know when you're there. Second full				
	180:6	paragraph.				
	180:7	A: Okay. It is evident?				
	180:8	Q: Do you agree that it is evident from				
	180:9	what has been stated above that white lead is in				
	180:10	itself a drier and consequently, if we grind a				

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180:11 sample of white lead and a sample of zinc oxide.
 180:12 respectively and linseed oil and painted them
 180:13 side by side, the white lead sample would be dry
 180:14 long before the zinc oxide sample.
 180:15 Do you agree?
 180:16 A: Yes.
 180:17 Q: That's one of the reasons why lead had
 180:18 to be used with zinc oxide paints, correct?
 180:19 MR. LEWIS: Objection to foundation,
 180:20 calls for speculation, outside the scope of
 180:21 expertise. Please answer.
 180:22 MR. LOPATA: That's why a drier had to
 180:23 be used with it.

28	182:5-182:24	Girard, James 2006-07-28	00:01:01	00:38:19	00:30:58	jgfinal.22
	182:5	Q: Okay. And can I ask you to turn to				
	182:6	page 56 of the Sabin article -- of --				
	182:7	A: Fifty-six of Sabin.				
	182:8	Q: At the bottom of the page that starts				
	182:9	the third.				
	182:10	A: Yes.				
	182:11	Q: Are you with me?				
	182:12	A: Yes.				
	182:13	Q: Okay. Do you agree that another				
	182:14	objection to zinc is that it forms a very hard				
	182:15	and inelastic film which is liable to crack				
	182:16	through to the wood, and then the moisture gets				
	182:17	in behind it and throws it off in larger flakes.				
	182:18	Do you agree with that?				
	182:19	A: Well, we've already talked about the				
	182:20	fact that it's more inelastic. And because it's				
	182:21	more inelastic, it's going to crack. If it				
	182:22	cracks through to the wood, then moisture is				
	182:23	going to get behind it. So I don't have any				
	182:24	problem with that.				
29	182:25-183:3	Girard, James 2006-07-28	00:00:13	00:39:20	00:29:55	jgfinal.23
	182:25	Q: Okay. Could you turn to page 37 of				
	183:1	Sabin, please, and let me know when you're				
	183:2	there.				
	183:3	A: I'm here.				
30	183:4-185:1	Girard, James 2006-07-28	00:02:07	00:39:33	00:29:42	jgfinal.24
	183:4	Q: I lost my place. I apologize. I'm				
	183:5	sorry. Back to 57. I had a typo in my outline.				
	183:6	A: Okay.				
	183:7	Q: Let me know when you're there.				

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183:8 A: It happens. I'm back to 55.
183:9 Q: Fifty-seven.
183:10 A: Oh, excuse me. Fifty-seven.
183:11 Q: Do you see where Dr. Sabin writes:
183:12 Painters who use such mixtures generally use two
183:13 or three parts of white lead to one of white
183:14 zinc. If enough lead is used this defect
183:15 disappears.
183:16 A: And so we're talking about the
183:17 inelastic problem here, right?
183:18 Q: That's right.
183:19 A: I see that, yes.
183:20 Q: Okay. Zinc oxide on its own was
183:21 defective, correct?
183:22 MR. LEWIS: Object to
183:23 characterization. Calls for a legal conclusion.
183:24 Please answer.
183:25 THE WITNESS: He uses the word
184:1 defective. I mean, it is a -- it is a property
184:2 that disappears is what I would say.
184:3 BY MR. LOPATA:
184:4 Q: Okay. When lead is added to zinc
184:5 oxide to create a mixed pigment paint, the
184:6 cracking defect in zinc oxide is remedied,
184:7 correct?
184:8 A: That's what he said. Correct.
184:9 Q: And you agree with that?
184:10 A: And I agree with that.
184:11 Q: Okay. Could you turn to page 31,
184:12 please. Let me know when you're there.
184:13 A: I'm here.
184:14 Q: Okay. This is -- you can tell from
184:15 the top of the page this is the section on white
184:16 lead and opacity, correct?
184:17 A: Correct.
184:18 Q: It, referring to white lead, is the
184:19 most opaque white pigment used in paint, as is
184:20 well known. Did I read that accurately?
184:21 A: You did.
184:22 Q: And you would agree that lead -- white
184:23 lead is the most opaque white pigment used in
184:24 paint, correct?
184:25 A: Well, the next sentence says: White
185:1 zinc is perhaps as opaque.

31	185:12 -186:12	Girard, James 2006-07-28	00:00:54	00:41:40	00:27:35	jgfinal.32

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185:12 Q: Doctor, would you agree with the first
 185:13 sentence indicating that lead is the most opaque
 185:14 white pigment used in paint?
 185:15 A: I will agree with that.
 185:16 Q: Now, in fairness, the second sentence
 185:17 states that white zinc is perhaps as opaque,
 185:18 particle for particle, but it requires from
 185:19 1-1/2 to twice as much oil to make a paint;
 185:20 hence the film has less pigment in it, and it is
 185:21 generally allowed that it takes from three to
 185:22 four coats of it to equal two of lead, Did I
 185:23 read that accurately?
 185:24 A: You did.
 185:25 Q: And do you agree with that?
 186:1 A: I do.
 186:2 Q: Okay. Zinc oxide requires more
 186:3 linseed oil, correct?
 186:4 A: Yes.
 186:5 Q: Zinc oxide paint does not hide prior
 186:6 coats of paint as well as lead, correct?
 186:7 A: Yes. That's correct.
 186:8 Q: And zinc oxide paint requires three or
 186:9 four coats, whereas lead paint only requires
 186:10 about two coats, correct?
 186:11 A: That's what it says, yes. And I agree
 186:12 with it.

32	195:18-195:25	Girard, James 2006-07-28	00:00:10 00:42:34 00:26:41	jgfinal.40
	195:18	Q: Sure. One of the things that you plan		
	195:19	to talk about is the life cycle of paint,		
	195:20	correct?		
	195:21	A: Correct.		
	195:22	Q: Part of the life cycle of paint is		
	195:23	deterioration, as you've testified previously,		
	195:24	correct?		
	195:25	A: Correct.		
33	199:16-204:2	Girard, James 2006-07-28	00:04:47 00:42:44 00:26:31	jgfinal.25
	199:16	Q: Okay. Do you agree with that, that		
	199:17	anatase titanium dioxide pigments produce the		
	199:18	weakest paint films of the white opaque		
	199:19	pigments?		
	199:20	A: That's what paint companies report. I		
	199:21	don't disagree with it.		
	199:22	Q: Okay. And would you agree that the		
	199:23	titanium pigments all fail by erosion or		
	199:24	sloughing of the film?		

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199:25 A: I think I would have to read the whole
200:1 article to agree or disagree with that.
200:2 Q: Okay. Do you know how titanium
200:3 dioxide paints fail?
200:4 A: The mechanisms -- I mean, the
200:5 mechanisms are somewhat the same of what we were
200:6 talking about before.
200:7 Q: Would you agree that titanium dioxide
200:8 paints chalk?
200:9 A: They would.
200:10 Q: Okay. Titanium dioxide paints, in
200:11 less -- when used as a sole pigment, chalk
200:12 excessively, correct?
200:13 A: They chalk. Okay. I'm not
200:14 comfortable going into excessively or not.
200:15 Q: Are you aware whether or not titanium
200:16 dioxide paints are treated with anything to stop
200:17 the chalking?
200:18 A: I'm sure they are.
200:19 Q: Okay. Do you know what titanium
200:20 dioxide paints are treated with to slow down the
200:21 chalking process?
200:22 A: No. I'm not, sir.
200:23 Q: Okay. Titanium dioxide paints are the
200:24 poorest of the white pigments in terms of tint
200:25 retention, correct?
201:1 A: Yes.
201:2 Q: And what that means is that titanium
201:3 dioxide paints have films that fade, correct?
201:4 A: Yes.
201:5 Q: Rutile titanium dioxide, R-U-T-I-L-E,
201:6 do you know what that is?
201:7 A: Well, there's two forms of titanium,
201:8 anatase and rutile.
201:9 Q: Do you know the differences between
201:10 anatase and rutile?
201:11 A: I don't know all the specifications,
201:12 no.
201:13 Q: Do you know generally what the
201:14 difference is between anatase and rutile
201:15 titanium dioxide?
201:16 A: If we're talking about paint --
201:17 Q: Uh-huh.
201:18 A: -- generally the rutile makes a better
201:19 paint.
201:20 Q: Do you know why rutile titanium

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201:21 dioxide makes a better paint?
201:22 A: I think that generally accepted that
201:23 it makes a stronger film.
201:24 Q: Okay.
201:25 A: more resistant film.
202:1 Q: Okay. Do you know the difference
202:2 between anatase and rutile titanium dioxide?
202:3 A: I'm not certain at this moment, no.
202:4 Q: One of the problems with all titanium
202:5 dioxides are their photogenic yellowing,
202:6 correct?
202:7 A: Yes. It's one of the problems.
202:8 Q: What that means in lay terms is that
202:9 titanium dioxide tends to turn yellow when it's
202:10 exposed to sunlight?
202:11 A: Yes.
202:12 Q: And another problem with titanium
202:13 dioxides is that they cannot achieve bright
202:14 colors, correct?
202:15 A: It's harder.
202:16 Q: To date, in 2006, you cannot get
202:17 bright colors with titanium dioxide-only paints,
202:18 correct?
202:19 A: Yes.
202:20 Q: Okay. Another problem with titanium
202:21 dioxide is that it is susceptible to mildew,
202:22 correct?
202:23 A: It is more susceptible, yes.
202:24 Q: One of the reasons that lead was used
202:25 in pigments was because it repels water,
203:1 correct?
203:2 A: I don't know if it repels water. It
203:3 stops mildew, if that's where we were at the
203:4 last sentence.
203:5 Q: Lead --
203:6 A: Changing the --
203:7 Q: First we were talking about titanium
203:8 dioxide and how it is susceptible to mildew,
203:9 correct?
203:10 A: Right.
203:11 Q: And one of the reasons that titanium
203:12 dioxide mildews so readily is because it takes
203:13 in water readily, correct?
203:14 A: Yes.
203:15 Q: In contrast, one of the benefits of
203:16 lead is that it repels water, correct?

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203:17 A: It repels water more effectively than
 203:18 does the titanium pigment.
 203:19 Q: Okay. Lead repels water better than
 203:20 titanium dioxide pigment, correct?
 203:21 A: Yes.
 203:22 Q: Lead repels water better than
 203:23 lithopone, correct?
 203:24 A: Yes.
 203:25 Q: Lead repels water better than zinc
 204:1 oxide, correct?
 204:2 A: I'm not certain.

34 204:9-205:2 Girard, James 2006-07-28 00:00:58 00:47:31 00:21:44 jgfinal.33

204:9 Q: Okay. Do you recall agreeing that
 204:10 zinc oxide, lead-free zinc oxide takes in 20
 204:11 times as much water as a white lead carbonate
 204:12 pigment?
 204:13 A: I don't remember that -- that
 204:14 testimony.
 204:15 Q: Okay.
 204:16 A: I remember we were -- we were talking
 204:17 about that. We were talking about the hygienic
 204:18 properties, about it being used internally. And
 204:19 I think that I would have to review the Brown
 204:20 article to go back and say it was 20 times.
 204:21 Q: Okay. Generally speaking, without
 204:22 assigning a value, you would agree that lead
 204:23 pigments are better at water resistance than
 204:24 lead-free zinc oxide, correct?
 204:25 A: I would agree with that.
 205:1 Q: Okay. One of the reasons -- strike
 205:2 that.

35 205:3-206:22 Girard, James 2006-07-28 00:02:03 00:48:29 00:20:46 jgfinal.26

205:3 (Girard Exhibit No. 94 was marked for
 205:4 identification.)
 205:5 BY MR. LOPATA:
 205:6 Q: I'm showing you an article by
 205:7 Frederick Brown that was authored in 1949. This
 205:8 was not part of your reliance materials, but I
 205:9 believe you have in fact seen this article
 205:10 before, haven't you?
 205:11 A: Yes, I have.
 205:12 Q: Okay. If I could ask you to turn to
 205:13 page 628, second column after the -- the
 205:14 paragraph after the break.
 205:15 A: Of the many ways?

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205:16 Q: Yes. Can I ask you to skip down to
 205:17 the sentence that starts: Old-fashioned pure
 205:18 white lead. Let me know when you're there.
 205:19 A: Yes.
 205:20 Q: Okay. Do you agree that old-fashioned
 205:21 pure white lead paint is more reliable in
 205:22 performance and wears out by fine crumbling that
 205:23 makes it stand postponement of repainting longer
 205:24 than any other white paints?
 205:25 Do you agree with --
 206:1 A: I -- you know, that's -- that's -- I
 206:2 think Brown's a very reasonable scientist. And
 206:3 I would accept his statement --
 206:4 Q: Okay.
 206:5 A: -- and agree with it.
 206:6 Q: Okay. Continuing in the next
 206:7 sentence: On the other hand, the more recent
 206:8 paints made with titanium dioxide, zinc oxide
 206:9 and white lead, together with other necessary
 206:10 pigments and liquids, remain relatively clean
 206:11 and bright in appearance. They do not, however,
 206:12 retain color so well. And if repainting is
 206:13 postponed beyond the proper time, they look
 206:14 shabbier and are more troublesome to prepare for
 206:15 repainting than white lead paint. Did I read
 206:16 that accurately?
 206:17 A: You did.
 206:18 Q: Okay. Do you agree that paints that
 206:19 are made from a combination of titanium dioxide,
 206:20 white lead and zinc oxide do not retain color as
 206:21 well as white lead paint, correct?
 206:22 A: That's correct.

36	209:21 -210:10	Girard, James 2006-07-28	00:00:25 00:50:32 00:18:43	jgfinal.27

209:21 Q: Homeowners who plan their maintenance
 209:22 programs carefully and stick to them can take
 209:23 full advantage of the newer paints. But those
 209:24 who may neglect their repainting would be wiser
 209:25 to use white lead paint. Would you agree with
 210:1 that?
 210:2 A: That clearly states what you just
 210:3 said.
 210:4 Q: Okay.
 210:5 A: Okay.
 210:6 Q: And you would agree with that?
 210:7 MR. LEWIS: Object to foundation.

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210:8 Scope of expertise. Please answer.
210:9 THE WITNESS: That's what Brown said,
210:10 okay, and I don't disagree with him.

37 215:2-220:10 Girard, James 2006-07-28 00:06:24 00:50:57 00:18:18 jgfinal.41

215:2 Q: You state in the report that paint
215:3 will fail no more than 4 to 7 years after
215:4 application of the paint.
215:5 A: Okay. We're on page 2?
215:6 Q: Bottom of the page.
215:7 A: Yes. Yes. Okay.
215:8 Q: How did you come up with the 4 to
215:9 7-year time period?
215:10 A: I think that's -- you know, in all
215:11 these depositions we've had, we've talked about
215:12 how many years the life cycle goes. And what I
215:13 tried to do is take the numbers that I have seen
215:14 and try to extrapolate them to very generous
215:15 estimates.
215:16 Q: So the 4 to 7 years isn't necessarily
215:17 tied to a particular document; it's sort of a --
215:18 an extrapolation that you have made?
215:19 A: It's an extrapolation I have made, you
215:20 know, from the body of information I have read
215:21 and the great number of products that are out
215:22 there.
215:23 Q: Okay. Do you -- is the 4 to 7 years
215:24 related to any particular period in time?
215:25 A: You know, one of the things that I
216:1 have -- I have kind of realized is that, in
216:2 reading documents over almost a period of
216:3 100 years, that that period tends to be pretty
216:4 much the same over the period.
216:5 We can go back to the Cruikshank
216:6 article we were talking about earlier, 1909,
216:7 they're talking 4 or 5 years between
216:8 reapplications.
216:9 And I -- I think that even if you get
216:10 into like the modern -- I think it actually
216:11 states on the -- like the modern-day Web sites
216:12 that -- that these -- this type of period is
216:13 a -- is a life cycle period for paint.
216:14 Q: So the 4 to 7-year period, in your
216:15 opinion, would relate to the life of paint
216:16 regardless of whether we're talking about a
216:17 paint from the 18th century or a paint from the

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216:18 21st century, correct?

216:19 A: And what I would -- what I would say

216:20 there is it's in that period with the older

216:21 paint being on the shorter end. Hopefully the

216:22 more modern paint being on the longer end.

216:23 Q: Okay.

216:24 A: But again, we have cheap

216:25 manufacturing -- I don't want to say cheap

217:1 manufacturing, but inexpensive formulations that

217:2 don't last as long as expensive formulations,

217:3 even today.

217:4 Q: Okay. So there's a variance in the

217:5 life cycle of paint, which is why you provided a

217:6 range, correct?

217:7 A: That's correct.

217:8 Q: Okay. And the 4 to 7-year range that

217:9 you provided on the life of paint is one that

217:10 would apply regardless of whether the paint was

217:11 manufactured in the 18th century or the 20th --

217:12 21st century, correct?

217:13 A: Let's say 19th at the -- let's say

217:14 from 1900 to the modern time.

217:15 Q: Okay. So you would agree that your

217:16 estimate of the 4 to 7-year life cycle of paint

217:17 would apply regardless of whether the paint was

217:18 made in the 19th century, the 20th century or

217:19 the 21st century?

217:20 A: Right.

217:21 Q: Okay. Are you referring just to

217:22 exterior paints when you make that estimate, of

217:23 4 to 7 years?

217:24 A: No. I mean, I think all paint

217:25 ultimately fails. Interior paint fails as well.

218:1 Q: Okay. Are you saying that interior

218:2 paint will typically fail after 4 to 7 years?

218:3 A: I think in -- generally speaking I

218:4 would say that interior paint lasts longer. It

218:5 is less exposed to environmental conditions.

218:6 Q: Okay. So the 4 to 7-year period would

218:7 relate to exterior paints.

218:8 A: It would definitely relate to exterior

218:9 paint.

218:10 Q: Okay. Do you have an estimate as to

218:11 how long interior paints last?

218:12 A: I think when you have interior paint,

218:13 it really depends on location. Because interior

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218:14 paints are much more subject to wear cycles
218:15 of -- of -- let's say it's a residential paint,
218:16 of the wear cycle of the family. And where it's
218:17 located, if it's in a traffic or nontraffic
218:18 zone. If it's in a wet area or dry area.
218:19 Whether it's an area exposed to light from the,
218:20 you know, coming through the windows. Whether
218:21 it's in a friction creating place like a -- like
218:22 a window. You know, it's hard to put all
218:23 interior paints into one -- one category.
218:24 Q: Okay. Would the life of interior
218:25 paint also depend on whether the paint is
219:1 maintained?
219:2 A: Well, I -- I -- are you saying does it
219:3 depend on whether it's painted over?
219:4 Q: We can start there,
219:5 A: I mean I don't know -- I've owned a
219:6 home for a long time and --
219:7 Q: That's fair. The estimate that you
219:8 have of the lifetime of paint relates to paints
219:9 that have not been painted over, correct?
219:10 A: Yes.
219:11 Q: Okay. Paints that are properly
219:12 maintained can last indefinitely, correct?
219:13 MR. LEWIS: Object to foundation.
219:14 Please answer.
219:15 THE WITNESS: I would contend that
219:16 your usage of the word maintained is a
219:17 reapplication of a new paint, okay? So I
219:18 wouldn't say -- you're building up layers, so,
219:19 you know, you are, in essence, covering over and
219:20 keeping that undersurface from whatever attack
219:21 it was -- it had when it was on the surface.
219:22 So I -- I would call it covering over,
219:23 repainting rather than maintenance.
219:24 BY MR. LOPATA:
219:25 Q: Okay. So covering over or repainting
220:1 of paint can prolong the life of paint in -- the
220:2 undercoats indefinitely, correct?
220:3 MR. LEWIS: Object to foundation.
220:4 Please answer.
220:5 THE WITNESS: In an ideal world.
220:6 They -- it can -- it can definitely extend it.
220:7 I mean, there are processes that take place at
220:8 the -- at the boundary between the substrate and
220:9 the paint, which will cause all the layers to

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220:10 fail and fall off.

38 221:24-222:18 Girard, James 2006-07-28 00:00:47 00:57:21 00:11:54 jgfinal.42

221:24 Q: Do you know what the term lead-safe

221:25 means?

222:1 A: I mean, I think in Jacobs' word, he

222:2 uses it all the time. He's defined it by HUD.

222:3 Okay? There's lead-safe work practices.

222:4 There's lead-safe areas. You know.

222:5 Q: If a surface is considered lead-safe,

222:6 do you have an understanding of what that means?

222:7 A: That it's less than 1 milligram per

222:8 square centimeter concentration, or 0.5 percent.

222:9 A: surface is considered lead-safe if

222:10 the lead is intact, correct?

222:11 MR. LEWIS: Objection. Misstates his

222:12 prior testimony.

222:13 THE WITNESS: No. I don't think so.

222:14 BY MR. LOPATA:

222:15 Q: Okay.

222:16 A: Because I think if you go to Jacobs,

222:17 he contends that even intact paint is a lead

222:18 hazard.

39 224:8-224:17 Girard, James 2006-07-28 00:00:32 00:58:08 00:11:07 jgfinal.43

224:8 Q: Perhaps the first layer attached to

224:9 the substrate.

224:10 MR. LEWIS: Objection to form.

224:11 Objection to foundation. Objection to scope.

224:12 Please answer.

224:13 THE WITNESS: You know, I think that

224:14 lead in paint, intact or not, will ultimately

224:15 someday fail and will become a hazard.

224:16 So although it might not be a hazard

224:17 today, it will be a hazard later.

40 226:2-227:5 Girard, James 2006-07-28 00:01:01 00:58:40 00:10:35 jgfinal.44

226:2 Q: I'm asking you whether or not you

226:3 believe that if a paint lasts for the lifetime

226:4 for which it's supposed to exist, that the paint

226:5 is considered to have been successful.

226:6 MR. LEWIS: Object to foundation.

226:7 Please answer.

226:8 THE WITNESS: I'm saying that paint

226:9 has a life cycle of 4 years. I think your

226:10 description is one in which it's lived its life

226:11 cycle, and it then falls off the wall, and it

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226:12 was successful in staying on the wall 4 years.
226:13 BY MR. LOPATA:
226:14 Q. Okay. So if a paint lasts for 4 to
226:15 7 years, the paint is considered to have been
226:16 successful?
226:17 MR. LEWIS: Asked and answered.
226:18 BY MR. LOPATA:
226:19 Q. Correct?
226:20 MR. LEWIS: Same objections. Please
226:21 answer.
226:22 THE WITNESS: I think that's what I
226:23 just said.
226:24 BY MR. LOPATA:
226:25 Q. You would agree?
227:1 A: Well, I mean, I really am having
227:2 trouble with 'successful' in this terminology.
227:3 I mean when -- when the paint has fallen off the
227:4 wall, it's hard to say it hasn't
227:5 catastrophically failed.

41 228:3-228:15 Girard, James 2008-07-28 00:00:36 00:59:41 00:09:34 jgfinal.45

228:3 Q. Okay. Regardless of whether a paint
228:4 is 4 years or 10 years or even a 20-year paint,
228:5 if it fulfills its purpose, it's considered to
228:6 have been a successful paint, correct?
228:7 MR. LEWIS: Object to foundation and
228:8 scope. Please answer.
228:9 THE WITNESS: I mean at the point that
228:10 this stuff falls off and becomes a problem, I
228:11 have a problem saying that it's a success. We
228:12 keep dancing around this problem. I think you
228:13 understand where I'm at. And -- you know, I
228:14 kind of feel like you're putting words in my
228:15 mouth.

42 231:15-232:2 Girard, James 2008-07-28 00:00:35 01:00:17 00:08:58 jgfinal.28

231:15 Q: Do you agree that it was not until the
231:16 1960s that alkyd-based white house paints began
231:17 to be popular?
231:18 A: Yes.
231:19 Q: Okay. Do you agree that this was due,
231:20 in part, to a disastrous experience by a large
231:21 paint company in the 1930s. They introduced a
231:22 white alkyd-based house paint that contained
231:23 zinc oxide, lead-free, and experienced
231:24 widespread intercoat peeling problems that
231:25 forced these alkyd paints off of the market. Do

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232:1 you agree with that?

232:2 A: Yes.

43 270:4-270:16 Girard, James 2006-07-28 00:00:41 01:00:52 00:08:23 jgfinal.46

270:4 Q: Do you consider this, I'll call it the

270:5 Dutch Boy salesman manual, to be an

270:6 authoritative and reliable source of information

270:7 about paints and pigments?

270:8 A: Yes, it's a reliable source, given the

270:9 fact that it is a salesman's brochure.

270:10 Q: Okay.

270:11 A: And, you know, I think that part of a

270:12 salesman's job is to accentuate the positive and

270:13 to maybe, you know, not worry as much about the

270:14 negative. So I see this as a sales kind of

270:15 document that isn't quite as even-handed as some

270:16 other kind of a treatise might be.

44 278:2-282:23 Girard, James 2006-07-28 00:06:30 01:01:33 00:07:42 jgfinal.29

278:2 Q: Okay. I understand you actually have

278:3 an original Handbook on Painting book?

278:4 A: Yes.

278:5 Q: How did you come to possess that book?

278:6 A: I bought it.

278:7 Q: Where did you buy it?

278:8 A: I think I bought it on eBay.

278:9 Q: Do you know the date of that book?

278:10 A: I'd have to look.

278:11 Q: Have you read other portions of the

278:12 book other than Chapter 11?

278:13 A: Yes.

278:14 Q: Have you read the entire book?

278:15 A: I can't say that I honestly read the

278:16 entire thing. I've read -- you know, I skimmed

278:17 the entire book. I read excerpts from it.

278:18 Q: Okay. How does the excerpt here,

278:19 which is Chapter 11, titled Common Painting

278:20 Defects, Their Cause and Cure, how does that

278:21 relate to your opinions we've spoken about

278:22 today?

278:23 A: Well, to go back to what we talked

278:24 about earlier, you know, in terms of the life

278:25 cycle of paint and how paint degrades, the --

279:1 excuse me -- the title of the chapter is Common

279:2 Painting Defects, Their Cause and Cure. And it

279:3 goes through some of the things we talked about

279:4 earlier today, like checking, alligating,

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279:5 cracking and scaling.
279:6 And it's -- you know, it's this little
279:7 tiny book that I'm sure is meant to be carried
279:8 in a painter's pocket to give practical
279:9 suggestions about their trade.
279:10 Q: Do you agree that the Handbook on
279:11 Painting published by National Lead Company is
279:12 an authoritative and reliable source of
279:13 information concerning paints and pigments?
279:14 A: I do.
279:15 Q: Do you agree that the Handbook on
279:16 Painting is an authoritative and reliable source
279:17 concerning surface preparation before painting?
279:18 A: I do.
279:19 Q: Do you agree that the Handbook on
279:20 Painting is an authoritative and reliable source
279:21 concerning defects with paint film and the
279:22 causes of those defects?
279:23 A: Yes.
279:24 Q: And do you agree that the Handbook on
279:25 Painting is an authoritative and reliable source
280:1 concerning methods to remedy defects or problems
280:2 in paint film?
280:3 A: Yes.
280:4 Q: And do you agree that the Handbook on
280:5 Painting is an authoritative and reliable source
280:6 of information concerning qualities of white
280:7 lead pigment?
280:8 A: What do you mean by qualities, sir?
280:9 Q: Well, let me ask you a few specific
280:10 questions from the chapter.
280:11 A: Okay. Sure.
280:12 Q: First, before we get into that
280:13 specifics, a few more general questions.
280:14 Do you disagree with any of the
280:15 discussion in this chapter about the types of
280:16 defects or problems that occur in paint film?
280:17 A: No. I don't believe so.
280:18 Q: Do you disagree with any of the
280:19 recommendations in this chapter about how to
280:20 remedy problems or defects in paint film?
280:21 A: I don't think so. No.
280:22 Q: Okay. Please look at page 97, which
280:23 is the first page in chapter 11.
280:24 A: Okay.
280:25 Q: The first full paragraph, second

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281:1 sentence. It states that: Pure white lead
281:2 paint properly mixed and skillfully applied to a
281:3 surface fit to receive it and exposed to
281:4 ordinary atmosphere conditions always does what
281:5 it is advertised to do, last long, look well
281:6 during its life and wear away gradually, leaving
281:7 a perfect surface for repainting.
281:8 Do you agree with that statement?
281:9 MR. LEWIS: What page was that?
281:10 THE WITNESS: Ninety-seven.
281:11 MR. PAUKA: It's page 97.
281:12 MR. LEWIS: Thank you.
281:13 BY MR. PAUKA:
281:14 Q. Do you agree with that statement,
281:15 Professor Girard?
281:16 A: I think as a general statement, it's
281:17 fine. I don't agree or disagree with it. I
281:18 don't have any information to base that it's not
281:19 a true and accurate statement.
281:20 Q: Okay. So it's fair to say that you do
281:21 not disagree with the statement, correct?
281:22 A: I have no basis on which to disagree.
281:23 This, as I understand it, was in a time when,
281:24 you know, professional painters mixed their
281:25 paint on site. It was before prepackaged paint.
282:1 And so, you know, like they say, if you do it
282:2 right and you prepare the surface, and -- it's
282:3 not for the common homeowner. It's for a
282:4 professional painter to follow these rules.
282:5 Q: And your understanding was, at the
282:6 time of this Handbook on Painting, which I
282:7 believe is dated 1936, white lead pigment was
282:8 used by master painters?
282:9 A: Uh-huh. Yeah.
282:10 Q: Was that a yes to the date or yes
282:11 to --
282:12 A: I don't know. Yes. That was a
282:13 complex question. Okay.
282:14 Q: Okay.
282:15 A: So the date was '36.
282:16 Q: Right.
282:17 A: Okay.
282:18 Q: And my statement was: Do you agree,
282:19 around the time that this Handbook on Painting
282:20 was published, which is in the 1930s, that white
282:21 lead pigment was typically used by master

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282:22 painters and not by ordinary homeowners?

282:23 A: Yes.

45	282:24 - 283:19	Girard, James 2006-07-28	00:01:13	01:08:02	00:01:13	jgfinal.30

282:24 Q: Would you please turn to page 108.

282:25 A: I'm there.

283:1 Q: It's the section that begins, washing.

283:2 A: Okay.

283:3 Q: The second paragraph in that section,

283:4 the sentence that states: White lead never

283:5 develops water soluble compounds and surfaces

283:6 painted with properly mixed white lead paint

283:7 will never give this trouble,

283:8 And you're welcome to read that entire

283:9 section if you need to. My question is: Do you

283:10 agree with that statement?

283:11 A: Well, the paragraph before it talks

283:12 about a problem where water contacts the

283:13 surface. Water soluble materials from the

283:14 pigments become soluble in the water and it

283:15 streaks down the side of the wall.

283:16 And it says that white lead never

283:17 develops water soluble compounds. It's quite

283:18 true that lead carbonate is not a water soluble

283:19 material. So I agree with the statement.

Play Time for this Script: 01:09:15

Total time for all Scripts in this report: 01:09:15

Egan Vol. 2

0163

**IN THE CIRCUIT COURT OF MILWAUKEE COUNTY
STATE OF WISCONSIN**

CITY OF MILWAUKEE, a
municipal corporation,

Plaintiff,

v. Case No. 01- CV-3066

NL INDUSTRIES, INC., a
foreign corporation, and
MAUTZ PAINT CO., a Wisconsin
corporation,
Defendants.

Deposition of JOHN EGAN (Volume 2)

May 22, 2007

9:15 a.m.

at

733 North Van Buren Street
Milwaukee, Wisconsin

Reported by Lynn M. Bayer, RPR/CM

Egan Vol. 2

1 Deposition of JOHN EGAN, a witness in the
2 above-entitled action, taken at the instance of the
3 Defendants, pursuant to notice and agreement, before
4 Lynn M. Bayer, RPR/CMR, Notary Public in and for the
5 State of Wisconsin, at 733 North Van Buren Street,
6 Milwaukee, Wisconsin, on May 22, 2007, commencing at 9:15
7 a.m. and concluding at 3:55 p.m.

8

9 **A P P E A R A N C E S**

10 Cohen, Milstein, Hausfeld & Toll, PLLC, by
11 Mr. George Farah and
12 Ms. Kathleen Konopka
13 1100 New York Avenue, N.W.
14 Suite 500, West Tower
15 Washington, D.C. 20005
16 Appeared on behalf of the Plaintiff.

17

18 Bartlit, Beck, Herman, Palenchar &
19 Scott, LLP, by
20 Mr. John M. Hughes
21 1899 Wynkoop Street, 8th Floor
22 Denver, CO 80202
23 Appeared on behalf of the Defendant NL
24 Industries.

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Egan Vol. 2

1 that accounts are properly charged and time is
2 properly recorded for various city reporting
3 including grants. Internal control measures would
4 basically -- are designed to ensure that these
5 performance measures were properly charged.

6 BY MR. HUGHES:

7 Q. What is your basis that even one dollar was charged
8 to a paint related expenditure?

9 A. These performance measures have been identified by
10 the health department as performance measures that
11 were used related to lead paint abatement. Based
12 upon that information that these performance measures
13 are related to lead paint abatement, my assumption is
14 that they were properly -- that that's what they were
15 used for.

16 Q. Who told you these were related to paint?

17 A. These were -- performance measures, Jim Bauer
18 basically who has been the person who oversees any
19 health department grant has -- in our office has
20 reviewed this information and also information from
21 the health department that he received would indicate
22 that these performance measures are related to lead
23 paint.

24 Q. Let me just make sure, I want to have -- because I'd
25 like to move on. None of the performance measures

Egan Vol. 2

1 include the word paint. But you say they're related
2 to lead paint. And I want to know all the
3 information that you're aware of that indicates these
4 performance measures are related to lead paint and
5 not just lead generally?

6 A. Information I can speak to which is produced related
7 to our financial system, these performance measures
8 have been related to -- my understanding is that
9 these performance measures based upon information
10 from the health department are related to lead paint
11 abatement.

12 Q. Did somebody tell you that?

13 A. This information came from an accountant in our
14 office who's very familiar with the lead paint
15 abatement programs, Jim Bauer told me that, correct.

16 Q. Jim Bauer is the accountant who's familiar with lead
17 paint abatement programs?

18 A. Correct.

19 Q. And the programs that he identified were these
20 programs that don't mention the word paint; correct?

21 A. Correct.

22 Q. And is there -- other than what Jim Bauer told you,
23 is there any other basis for your testimony that 100
24 percent of the expenditures that you've identified
25 here relate to lead paint?

Egan Vol. 2

1 A. I don't recall all the communications, you know,
2 related to performance measures from the health
3 department staff; but it's a correct statement to say
4 that I'm relying on Jim Bauer who works with the lead
5 abatement program from the city, reviews those lead
6 abatement expenditures and costs that are booked
7 within our city -- you know, does the internal review
8 for our office. Based upon that, you know, I feel
9 with a high level of confidence that these are
10 related to lead paint abatement.

11 Q. So your testimony is that each of the dollar figures
12 in the past damages spreadsheet, the latest one which
13 I believe is Egan Deposition Exhibit 10, the third
14 amended statement of damages, as well as the figures
15 in Egan Deposition Exhibit 11, that 100 percent of
16 that money was spent on lead paint?

17 A. I feel that internal controls within the city, that
18 in every material aspect, you know, all these -- that
19 these costs were spent on lead paint.

20 MR. FARAH: Can I ask a question for
21 clarification. When you're asking about these
22 spreadsheet figures are you also including the
23 footnote deduction?

24 MR. HUGHES: We'll get to that later.

25 MR. FARAH: But is that what you meant in

Egan Vol. 2

1 that question?

2 MR. HUGHES: I didn't really think about
3 the footnote when I asked that question. I'm ready
4 to move on. We'll come back to the footnote.

5 Q. Okay. So let's get back to the Egan Deposition 9
6 spreadsheet and the Egan Deposition Exhibit 10
7 spreadsheet which are the spreadsheets that relate to
8 the second and third amended statement of damages.
9 And I think that for the -- on the third amended
10 statement of damages you had deleted O and M costs
11 for 1992, 1993 and 1994; is that correct?

12 A. That is correct.

13 Q. And on the second amended statement of damages for
14 1993 you had claimed \$606,000 -- \$606,904; correct?

15 A. For 1993?

16 Q. \$606,000?

17 A. That is correct.

18 Q. And then 1994 \$685,000?

19 A. Correct.

20 Q. And on April 17 of 2007, you claimed no dollars for
21 those categories; correct?

22 A. Correct.

23 Q. And now for 1993 you claim \$256,000, 1994 \$201,000
24 approximately; correct?

25 A. Right.

Egan Vol. 2

1 Q. In 1995 -- strike that. What are indirect costs?

2 A. There is two types of indirect costs. One is related
3 to central service costs which are basically the
4 indirect overhead of the city. This includes, for
5 example, a comptroller's office, some costs within
6 administration and personnel costs. It also --
7 indirect costs within a department itself which is,
8 again, the administration of the particular
9 department, the budgeting, the personnel that they do
10 within a department. So the indirect costs basically
11 makes up two parts. The city wide indirect overhead
12 and the department's indirect overhead.

13 Q. Are the figures identified as indirect cost figures
14 in the spreadsheet for Egan Deposition Exhibit 10,
15 are those indirect cost figures a combination of both
16 city indirect costs and health department indirect
17 costs?

18 A. Generally speaking, yes.

19 Q. Is there an exception to that?

20 A. There is an exception. In later years where indirect
21 costs within the department, if those indirect costs
22 produce a higher rate than the average rate of
23 indirect costs in city departments city wide, the
24 indirect costs in those -- in the health department
25 let's say would be lower to the city wide average of

Hearing

1 STATE of WISCONSIN : CIRCUIT COURT : MILWAUKEE COUNTY
2 BRANCH 25

3 -----

4 CITY OF MILWAUKEE,

5 Plaintiff,

6 vs. Case No. 01CV003066

7 NL INDUSTRIES INC.,

8 Defendant,

9 -----

10 MOTION HEARING

11 -----

12 May 15, 2007 HON. JOHN FRANKE

13 Circuit Court Judge, Presiding

14

15

16 APPEARANCES:

17

18

19 RICHARD LEWIS

20 ATTORNEY AT LAW

21 appearing for the Plaintiff.

22 CHRISTOPHER RIORDAN

23 ATTORNEY AT LAW,

24 appearing for the Plaintiff..

25 KATHLEEN KONOPKA

 Attorney at Law

 appearing for the Plaintiff.

 JAMES PIZZIRUSSO,

 Attorney at Law,

 appearing for the Plaintiff.

 25

Hearing

1 TERRY NELLIS
 Attorney at Law
2 appearing for the Plaintiff.
3 JAMES MURRAY
 Attorney at Law
4 appearing for the Defendant.
5 DONALD SCOTT
 Attorney at Law
6 appearing for the Defendant.
7 ANDRE PAUKA
 Attorney at Law
8 appearing for the Defendant
9 MICHAEL WIRTH
 Attorney at Law
10 appearing for the Defendant.
11 JOHN HUGHES
 Attorney at Law
12 appearing for the Defendant.
13 BRIAN PRESTES
 Attorney at Law
14 appearing for the Defendant.
15
16
17
18
19
20 Official Reporter - Mary R. Hermann
21
22
23
24
25

Hearing

1 With respect to motion in limine No. 1 to
2 exclude evidence regarding utility or policy, I'm
3 denying the motion. I perhaps should note that in my
4 view, any ruling on a motion in limine is not
5 necessarily a final ruling. It's essentially a ruling
6 in advance upon objection, but it doesn't preclude
7 objections at trial based on all sorts of problems,
8 including foundation for testimony, and it doesn't
9 open the door to any and all questions about a
10 particular area. And in this case, I am not
11 concluding that this jury is going to be told that
12 they should balance the utility of lead paint against
13 the harms that may have been established.

14 The City is seeking to exclude evidence
15 about the value or utility of paint, the value --
16 specifications that government entities may have made
17 about paint and certain housing policies that I'm not
18 entirely clear on. I think there are purposes for
19 this evidence that are really outside of this notion
20 of balancing utility versus injury. And to the extent
21 that the cases cited, the Pennoyer case and the Jost
22 case do support the plaintiff's view in some
23 respects -- Pennoyer is P-E-N-N-O-Y-E-R. These cases
24 deal with private nuisance cases and primarily or at
25 least partly the maintenance of a nuisance, and at

Hearing

1 based on negligent conduct, the defenses available for
2 a tort of that type are available to the defendant.

3 Now that presents some difficult questions
4 here, but a lot of it just has to do with the nature
5 of the conduct that's alleged, and the complexity of
6 the cause and effect here, and the time that has
7 passed. And I'm frankly not sure what kind of showing
8 is going to be required to get other entities, or
9 persons, or groups of people on the verdict form. But
10 as a general principle of law, if the jury finds that
11 the defendant's conduct was negligent, then I believe,
12 assuming the evidence supports it, the defendants are
13 entitled to pursue defenses based on the negligence
14 and liability of others. If the jury answers no to
15 the question about whether the defendant's conduct was
16 negligent, things may well be over, but it's also a
17 possibility they have to be asked additional questions
18 regarding whether NL's conduct was reckless or
19 abnormally dangerous.

20 And to some extent because of those
21 conclusions about what the jury will need to consider
22 and find in this case, I am going to deny the City's
23 motion for partial summary judgment and all of the
24 motions in limine. That is 1 through 6. I'm putting
25 aside 7 for the time being.

Hearing

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2 exclude evidence regarding utility or policy, I'm
3 denying the motion. I perhaps should note that in my
4 view, any ruling on a motion in limine is not
5 necessarily a final ruling. It's essentially a ruling
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21 that the cases cited, the Pennoyer case and the Jost
22 case do support the plaintiff's view in some
23 respects -- Pennoyer is P-E-N-N-O-Y-E-R. These cases
24 deal with private nuisance cases and primarily or at
25 least partly the maintenance of a nuisance, and at

Hearing

1 least arguably are limited to circumstances where
2 there is an intentional nuisance. Intentional conduct
3 causing the nuisance. No real dispute about whether
4 the defendant is producing some noxious substance or
5 causing some trouble that is invading or making things
6 hard for a neighbor.

7 I don't see the doctrine as having
8 application in this case, although I've indicated that
9 I'm not inclined to believe this jury should be
10 allowed to balance or be told that they should balance
11 the value of lead paint in whatever decade might be at
12 issue versus the harms that it has caused or the
13 threat of harm that it contained. There are questions
14 regarding the reasonableness of the defendant's
15 conduct, the unreasonableness of the interference,
16 issues regarding lack of ordinary care, which will
17 come up under the negligence question, possibly issues
18 regarding the negligence of others for which these
19 pieces of evidence have at least some arguable
20 purpose, and I'm going to deny the motion to exclude
21 those as subjects of evidence.

22 For similar reasons, I'm going to deny the
23 second motion in limine. This was to exclude evidence
24 about the property owner's failure to maintain
25 properties and the City's lack of property specific

Hearing

1 product identification.

2 This jury is going to be asked to visit,
3 perhaps revisit, the history of lead paint over a long
4 period of time to make judgments about the
5 reasonableness of things which it simply can't make
6 without some knowledge and understanding of the
7 circumstances that eventually lead to the nuisance and
8 the various causal steps that we -- that things went
9 through from marketing and selling paint, through the
10 retail process, to its use in homes and application
11 there, to becoming dust and chips, and eventually
12 ingested by children. And I agree with the defense
13 that it is entitled in the context of the verdict
14 questions that I anticipate here to introduce
15 otherwise admissible evidence about things that
16 contributed to the ultimate harm that necessarily
17 underlies the City's claim that it has to abate this
18 nuisance.

19 For similar reasons, I am granting motion
20 No. 3 with respect to excluding evidence as to other
21 sources of lead.

22 Motion No. 5 seems to me to be --

23 MR. SCOTT: Excuse me, Your Honor. You
24 indicated you were granting No. 3?

25 THE COURT: I'm sorry, denied, No. 3. Thank

Hearing

1 you.

2 Motion No. 4 has apparently been withdrawn.

3 I won't struggle with it now.

4 The fifth motion seems to me to be silly and
5 unnecessary. I don't know what evidence or argument
6 we're talking about here. I assume it is going to be
7 obvious to the jury that NL lacks meaningful control
8 over the nuisance in the years at issue. And to the
9 extent that it might be necessary, I could instruct
10 the jury that this is not a defense. But to the
11 extent that this all has relevance to the jury's
12 evaluation of the intentional conduct, knowledge at
13 certain points in time and the causation issues here,
14 I don't see how the evidence can be limited in any
15 meaningful way. I just can't imagine this is going to
16 take any time, and I assume it's going to be obvious
17 to the jury about the extent to which NL parted with
18 this product and did not have ownership of or control
19 over it at particular points in time.

20 Finally, I'm going to deny motion No. 6 with
21 respect to excluding evidence about collateral source
22 payments. I also think this is primarily a nonissue.
23 There doesn't appear to be any dispute about the
24 application of the rule, but we often lose sight of
25 the difference between the rule and the evidentiary

Hearing

1 corollary that goes with it. The rule seems to apply
2 here. And to the extent necessary, I can instruct the
3 jury that they may not reduce any award because of any
4 payments from a third party that may have been
5 received.

6 The general rule, of course, is that
7 evidence shouldn't come in if it's offered for that
8 purpose, but it just seems incredibly impractical to
9 me to think that we're going to try this case without
10 reference to federal programs or federal grants or the
11 jury somehow isn't going to be aware that there was at
12 least a few dollars of state or federal money
13 involved. I can't imagine working with these exhibits
14 for more than a few minutes and somehow redacting them
15 so that the jury doesn't find out about it, and it
16 just seems to me to be totally unnecessary. I can't
17 imagine the plaintiff expected to try this case
18 without having some reference by its witnesses to
19 federal grants or federal programs. I see no reason
20 to try to exclude some references at this point when
21 it seems absolutely clear that they're going -- there
22 is going to be a great deal of reference to federal
23 grants and federal programs. And Mr. Lewis talked
24 earlier today about the evidence of federal funding.
25 I just don't see how we can avoid it. And to the

Hearing

1 extent that there is a concern about the collateral
2 source rule, the jury can be instructed in that
3 regard.

4 I'm not sure how much I need to say about
5 the City's motion for partial summary judgment, but I
6 need to break and take up a case that's waiting, if it
7 is indeed ready to go. I'll confer with those
8 attorneys. It will take a few minutes.

9 (Whereupon the case was passed.)

10 THE COURT: Let's take a 15 minute recess.

11 (Whereupon a short recess was taken.)

12 THE COURT: I've brought out NL's motions in
13 limine, but I want to finish with respect to ruling
14 regarding the City's motion for partial summary
15 judgment.

16 The motion is based on the argument that
17 undisputed facts establish that the presence of lead
18 paint in Milwaukee homes substantially interferes with
19 the activities of the entire community, or perhaps
20 alternatively undisputed facts establish that the
21 existence of lead paint in these homes unreasonably
22 interferes with such activities.

23 Taking issues regarding substantial
24 interference and unreasonable interference away from a
25 jury is difficult under most circumstances and

Hearing

1 certainly difficult here. And in trying to apply
2 summary judgment methodology here, it seems to me that
3 many of the arguments the defense has made could be
4 applied to the question of whether there is a prima
5 facie showing that undisputed facts warrant summary
6 judgment. But for purposes of this really, I will
7 assume that plaintiff's motion and the materials in
8 support of its motion set forth a prima facie showing
9 that the presence of lead paint substantially or
10 unreasonably interferes with the activities of an
11 entire community.

12 I find, however, that the materials filed in
13 opposition to the motion raise a genuine issue of fact
14 with respect to this question, and that there is a
15 genuine issue of fact as to whether the existence of
16 paint in the homes necessarily interferes with the
17 activities of the community in a manner that could be
18 said to be substantial or unreasonable.

19 Therefore, the motion is denied on the first
20 ground asserted by the plaintiff. Until a nuisance is
21 established, I'm not sure how I could consider whether
22 undisputed facts establish that NL intentionally
23 created a nuisance. The nuisance has to be
24 established first, but I believe there are even more
25 disputed issues of fact and genuine issues regarding

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Transcript of Proceedings - 5/25/2007

Vol. 5

1 STATE OF WISCONSIN : CIRCUIT COURT : MILWAUKEE COUNTY

2 -----
3 CITY OF MILWAUKEE,
4 a municipal corporation,

5 Plaintiff,

6 vs.

Case No. 01-CV-3066

7 NL INDUSTRIES, INC.,
8 a foreign corporation,

9 Defendants.

10 Before the Honorable

11 JOHN FRANKE, Circuit Court Judge

12 Branch 25, presiding

13
14 Friday, May 25, 2007

15 VOLUME 5

16 9:20 a.m.

17 A P P E A R A N C E S:

18 RICHARD LEWIS, TERRY NILLES, KATHLEEN KONOPKA,
19 Attorneys at Law, appeared on behalf of the Plaintiff.

20 JAMES T. MURRAY, JR., ANDREW M. PAUKA, Attorneys at
21 Law, appeared on behalf of the Defendant.

22
23 ORIGINAL
24
25

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Vol. 5

1 other evidence that it's going to hear, that it would
2 be of any real surprise or significance to know that
3 a company that markets a product and has been
4 defending its safety or relative safety in other
5 areas also lobbied government agencies. It just
6 seems so obvious as to be unimportant.

7 Now, if particular critical statements
8 are made in that context, issues may arise. But as a
9 general matter, I just don't see this as critical or
10 important. I may be wrong about the defense position
11 about what the industry's attitude was, but they were
12 clearly selling lots of paint. And the simple fact
13 alone that they lobbied against regulatory control is
14 not helpful or relevant. And someone could believe
15 quite strongly that something should not be sold or
16 should be limited in a certain way or should have
17 certain warnings on it but still believe the
18 government should not be the person doing it. That
19 raises a host of other issues about government
20 regulation and what it means to say, in one forum,
21 that the government should not act.

22 So I'm trying to make it clear that
23 the simple act of lobbying in and of itself, of
24 taking a position, particularly if it's not NL
25 directly, but really if it's anybody, is not directly

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Vol. 5

1 relevant to the issues this jury has to decide. And
2 any use of lobbying evidence needs to be restricted
3 in the same way, under the same principles that
4 agency statements may need to be restricted, and
5 restricted also to what's truly relevant to the job
6 that this jury has to do.

7 The jury may have to make decisions
8 about what the industry knew and what this defendant
9 knew and when it knew it. To the extent that
10 documents related to lobbying shed light on that,
11 they may have some purpose here, but the mere act of
12 lobbying alone is not something that I think is
13 relevant or needs to take time in this case.

14 But the motion, as it stands now, is
15 denied.

16 The fourth part seeks to exclude
17 evidence in argument about what NL had -- what NL
18 Industries has or has not done to abate lead paint in
19 Milwaukee. I think this may be more about argument
20 than evidence.

21 As I mentioned in an earlier hearing,
22 I think it would be reasonably clear to the jury that
23 nothing major was done. But ultimately, I think it's
24 irrelevant, and I'm going to grant Part 4 of the
25 motion.

1 Whether this defendant voluntarily
2 chose to abate lead paint or set up some fund to deal
3 with the medical problems that result is simply not
4 relevant to whether they were legally responsible for
5 their conduct many, many years ago.

6 This is somewhat tough, because I
7 suspect that if NL Industries had set up some huge
8 trust fund to perhaps deal with the medical problems,
9 they would want to get that in; but they would want
10 to get it in for the same reasons the plaintiff does.
11 And they aren't good reasons, because it's -- appeals
12 to sympathy or emotion, not because it's really
13 relative to whether this defendant is legally
14 responsible for what it did to cause this alleged
15 nuisance. That occurred some time ago, and whether
16 they voluntarily chose to address certain issues
17 after that point is not relevant. And part of the
18 motion, Part 4 is granted.

19 Part 5 has been conceded, I believe,
20 by the plaintiff. I will simply grant Part 5. And
21 as with any motion, it means granted unless or until
22 there's further proceedings outside the hearing of
23 the jury. If something changes in some material way,
24 I'll have to perhaps reconsider issues, but for now,
25 it's -- it's granted and it's off limits.

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Transcript of Proceedings - 5/25/2007

Vol. 5

1 I'm going to take a break and then
2 rule on the fourth NL motion concerning Dr. Lanphear.

3 Is there anything else that is pending
4 or that -- particularly that you feel needs to be
5 addressed before opening statements on Tuesday?
6 Mr. Lewis?

7 MR. LEWIS: Your Honor, I spoke with
8 defense counsel this morning about getting some kind
9 of list of their witnesses, some kind of indication
10 of the order of their witnesses at some point. I've
11 provided one to the Court and revised it recently.
12 Mr. Pauka said he would do his best to get me
13 something, and I would just like to have the Court
14 require that I receive something and not have it be
15 kind of floating as it has been for several days.

16 THE COURT: I assume you have perhaps a
17 series of witness lists. And the problem with
18 ordering witness lists is that lawyers can put
19 down -- can always protect themselves by putting down
20 every possible witness and then putting paragraphs at
21 the end that I don't think create any rights but
22 lawyers do, and they say I reserve the right to call
23 any other witness listed in the Milwaukee phone book
24 who might know something about this.

25 This needs to be a good-faith process.

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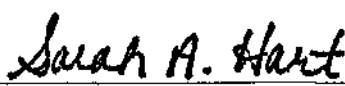
Vol. 5

Transcript of Proceedings - 5/25/2007

1 STATE OF WISCONSIN)
2) SS:
3 MILWAUKEE COUNTY)
4

5 I, Sarah A. Hart, RPR/RMR/CRR and Notary
6 Public in and for the State of Wisconsin, do hereby
7 certify that the foregoing proceedings was recorded
8 by me and reduced to writing under my personal
9 direction and that it is a true and correct
10 transcript of said proceedings had in said matter to
11 the best of my knowledge.

12 In witness whereof, I have hereunto
13 set my hand and affixed my seal of office on this
14 25th day of May, 2007.

15 
16 SARAH A. HART, RPR/RMR/CRR
17 Notary Public

18 My commission expires September 30th, 2007.
19
20
21
22
23
24
25

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Transcript of Proceedings - 5/29/2007

Vol. 6

1 STATE OF WISCONSIN: CIRCUIT COURT: MILWAUKEE COUNTY

2 CITY OF MILWAUKEE,
3 a municipal corporation,

4 Plaintiff,

5 -vs-

Case No. 01-CV-3066

6 NL INDUSTRIES, INC.,
7 a foreign corporation,

8 Defendants.

9
10 BEFORE THE HONORABLE

11 JOHN FRANKE, Circuit Court Judge

12 Branch 25, presiding

13
14 Tuesday, May 29, 2007

15 VOLUME 6

16 9:43 a.m.

17 A P P E A R A N C E S:

18
19 RICHARD LEWIS, Attorney-at-Law, appeared on behalf
20 of the Plaintiff.

21 RUDY CONRAD, City Attorney's Office.

22 DONALD SCOTT, JAMES T. MURRAY, JR., MICHAEL WIRTH,
23 ANDRE PAUKA, JOHN HUGHES, Attorneys-at-Law, appeared on
24 behalf of NL.
25

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Transcript of Proceedings - 5/29/2007

Vol. 6

1 A Shall I read the quote for the record?

2 Q Only if it helps you explain it to the jury. I'd
3 rather you just explain to the jury.

4 A Well, it explains that abatement of lead-based
5 paint hazards is essential if we're going to
6 eliminate childhood lead poisoning, because
7 lead-based paint is the most concentrated source of
8 lead to children, historically the source most
9 closely linked to lead poisoning in children.

10 That is not to deny that there are
11 other sources of lead exposure in children, but
12 that lead -- especially for children with the
13 highest blood-lead levels, lead-based paint is of
14 particular and primary importance.

15 Q Thank you. I'd like to turn to the question of --
16 the public health question of the level or the
17 number of children affected by childhood lead
18 poisoning.

19 MR. LEWIS: And, once again, I'd like to
20 turn to the strategic plan document, Exhibit 169,
21 and publish a page to the jury.

22 Q Dr. Foldy, in terms of the scope of the problem,
23 could you, in reference to what the CDC consensus
24 statement says, explain this issue to the jury.

25 A Well, based on the various estimates of the number

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Vol. 6

1 of children affected, CDC made the statement that
2 it has already affected millions of children and
3 could affect millions more. It also says that the
4 impact is real, even if it is relatively silent,
5 that is to say, not obvious, and that both the
6 number and the impact have driven the decision
7 towards a strategic plan to eliminate poisoning.

8 Q Thank you. Can you explain to the members of the
9 jury how, just in a practical sense, how a small
10 child or an infant or toddler actually becomes lead
11 poisoned?

12 A Yes. As was mentioned during the introduction,
13 there had been some thought early on that children
14 become lead poisoned by eating paint chips. But as
15 our evolution of understanding of lead poisoning
16 has continued, we understand that children can get
17 toxic levels of lead by ingesting what is basically
18 a microscopic amount of lead every day.

19 And so this is not necessarily
20 chewing on the lead painted surfaces, like you
21 heard about before, but, in fact, that children
22 between the ages where they begin to crawl, through
23 the first couple of years of life, are very
24 actively exploring with their hands constantly,
25 getting dust on their hands, and hand-to-mouth

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Vol. 6

1 activity, as anyone who has watched a thumb sucker
2 or tried to take away, for example, a set of keys
3 from a teething baby, is a very common, ordinary
4 part of childhood.

5 So as a result, both the
6 contamination of hands through the routine
7 exploring of the child's environment, it's what
8 children are supposed to do, and the sticking of
9 those hands, as well as toys and other objects into
10 the mouth, creates, if you will, a continuous
11 conveyor belt of microscopic quantities of lead
12 into the child's body. Because the child retains
13 the lead in the body, it gradually accumulates to
14 toxic levels.

15 Q Does this behavior of putting the hands in the
16 mouth, do the pediatricians have a name for that?

17 A It's called hand-to-mouth behavior, and it's
18 considered quite a normal part of growing up.

19 Q Doctor, I'm showing you Exhibit 197, the 2005
20 statement from the American Academy of Pediatrics
21 addressing this issue. Could you explain what the
22 statement is intended to communicate.

23 A They say much more succinctly what I had said
24 earlier, that children can ingest lead-laden dust
25 through normal mouthing behaviors by simply placing

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Transcript of Proceedings - 5/29/2007

Vol. 6

1 their hands or an object in their mouth.

2 Q Doctor, as a family practitioner, what do you tell
3 parents -- is there a way to have parents stop
4 their children from hand -- what's called
5 hand-to-mouth behavior?

6 A I'd say none that's been found effective. Again,
7 it's considered a normal part of the child's
8 behavioral development.

9 Q Doctor, when you came to Milwaukee to assume the
10 position as medical director in 1996, had the
11 Centers for Disease Control published guidance or
12 recommendations to local health departments and
13 local public health agencies on how to respond to
14 childhood lead poisoning?

15 A Yes, they had, and these have been refined over
16 time.

17 MR. LEWIS: Your Honor, I'd like to
18 publish a page from a document that's been
19 exhibit -- that's been admitted with those
20 recommendations.

21 MR. SCOTT: Your Honor, with --

22 THE COURT: What's the exhibit number?

23 MR. LEWIS: 170, Your Honor.

24 MR. SCOTT: I'm frankly a bit concerned
25 that we have some things being shown, we have no

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Vol. 6

1 Q Dr. Foldy, can you identify that, please. It's on
2 your screen, I believe.

3 A Yes, the exhibit I'm looking at now is a printout
4 from the City of Milwaukee's database that
5 tracks -- that creates estimates based on the
6 numbers of children screened, and also the numbers
7 of children reported to have high blood lead
8 levels, as well as -- yes, that is what is on this
9 screen. And, also, the proportion of children ages
10 one to two years old who have been screened during
11 given calendar years.

12 Q Doctor, is this the kind of report you would
13 request and review while you were serving as health
14 commissioner overseeing the childhood lead
15 poisoning program?

16 A Yes. In fact, I requested that many of these
17 reports be created, both for my benefit and for the
18 community's benefit.

19 MR. LEWIS: Your Honor, may this be
20 admitted?

21 MR. SCOTT: No objection to 209.

22 THE COURT: 209 is received.

23 BY MR. LEWIS:

24 Q All right. I'd like to work with Bill to make some
25 of this readable and ask you a couple questions. I

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1 think right now the print is too small.

2 MR. LEWIS: Bill, if we could go to the
3 top in the second and third column. The second --
4 over one to the right. The second and third
5 column.

6 Q Dr. Foldy, can you identify what information is
7 contained in this chart?

8 A This is a raw count of the number of children who
9 were tested for blood lead who were less than six
10 years old and who were found to have, it says here,
11 greater than 10 micrograms per deciliter in their
12 blood. Unfortunately, the years are not shown in
13 the rows.

14 Q Let me interrupt and ask that we expand the view to
15 the side so the years will be shown. Please
16 continue.

17 A So as you now can see, the column I was speaking
18 about was the middle column, or D, less than six
19 years at greater than 10 micrograms per deciliter.
20 See, the calendar year is listed in Column A.

21 So you can see, for example, in
22 1997, 3,464 children fell into this category. By
23 2006, this had gone down. And then a total is
24 given in Row 13 of under-six-year-old children with
25 levels greater than 10 of over 19,000 children

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1 during that period.

2 Q And is this limited to the City of Milwaukee?

3 A It is my understanding that this data has been
4 edited to exclude lead results received for
5 children living outside the city limits.

6 Q And Column E refers to incidence, children less
7 than six years, greater than or equal to
8 20 micrograms. Can you explain that to the jury?

9 A Yes. And let me say thank you for pointing out, I
10 did not see the equal sign, greater than or equal.
11 So the first column I talked about were children
12 who had a level of 10 or higher.

13 The level in Column E, these are
14 children with higher lead levels, 20 or higher.
15 These are levels at which a higher intensity of
16 public health intervention is recommended by the
17 Centers for Disease Control and also recommended by
18 state programs.

19 Q And what was the total number of children during
20 the ten-year period '97 to 2006 that were measured
21 to have that level of lead in the blood?

22 A 2,619 children.

23 Q I'd like to turn to a different part of the slide.
24 In the left-hand column, the third box down, it's
25 called prevalence, less than six years. The third

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1 one down, next one down.

2 I'd like to focus on the
3 six-year-old data. Thank you.

4 Dr. Foldy, can you explain to the
5 members of the jury what the word "prevalence"
6 means?

7 A First, to distinguish it from what you were looking
8 at before, I need to say that the way the first
9 column was created was to look for any child who
10 had -- was newly found to have an elevated blood
11 lead in that category, hence, the incidence or
12 onset of a case of lead poisoning.

13 Prevalence looks at all children
14 found to have the elevated blood lead level,
15 whether or not they had previously been detected.
16 So it simply gives us a measure in a given calendar
17 year how many children are walking around with this
18 elevated lead level, whether or not they were
19 previously detected.

20 This is important again because with
21 lead being a chronic disease and a chronically
22 absorbed poison, it's not an event that only
23 happens once. And it's an event that occurs and
24 that continues to persist in a large proportion of
25 children over time, as you see in this slide.

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1 document.

2 THE COURT: Yes, I'm looking at -- was it
3 291?

4 MR. LEWIS: I believe so, Your Honor.
5 It's the Lead-Safe Housing Registry.

6 MR. SCOTT: I have no objection to
7 displaying a portion of it for now, but I just want
8 to look at it first.

9 THE COURT: If you want to display a
10 portion or offer that portion --

11 MR. LEWIS: I don't need to display it,
12 Your Honor.

13 THE COURT: All right. Well, I'll
14 reserve a ruling on its admissibility. It appears
15 to be a lengthy document with lots of charts in it.

16 MR. LEWIS: Thank you. Can you show --

17 THE COURT: Now, what's in front of the
18 jury is --

19 MR. LEWIS: That is Exhibit 408, Your
20 Honor. And I've asked that that be admitted also.

21 THE COURT: I believe that was.

22 MR. SCOTT: Yes.

23 THE COURT: 408 is in.

24 MR. LEWIS: Thank you. Please show Your
25 Honor and the witness Exhibit 409, just to the

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1 court and the witness first.

2 BY MR. LEWIS:

3 Q Dr. Foldy, can you identify Exhibit 409?

4 A Yes. This is a graph, again, of the percentage of
5 children found to have elevated blood leads that is
6 greater or equal to 10 micrograms per deciliter per
7 calendar year. It's a graphic representation of
8 some of the numbers we saw earlier.

9 Q And is this an accurate summary of the kind of data
10 we reviewed earlier?

11 A Yes.

12 Q And can you explain just what's happening in the
13 bar chart from the time 1997 to 2006?

14 A Well, through the combined efforts of everybody
15 involved, we see a consistent decline year by year
16 in the number of children who present with elevated
17 blood leads. However, as late as 2006, what looks
18 like a small number of 6 percent of course reflects
19 1 in 20 children in the City of Milwaukee.

20 MR. LEWIS: Your Honor, I ask that 409 be
21 admitted.

22 MR. SCOTT: No objection.

23 THE COURT: Received.

24 MR. LEWIS: And if you could please show
25 Your Honor and the witness Exhibit 405.

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1 Q Can you identify that for the record, please.

2 A Again, this is a graphic representation where the
3 bars represent the number of chelation events that
4 I believe -- well, no, I'm sorry, in an earlier
5 exhibit, only one year's data was shown. This
6 shows data over a number of years starting in 1993
7 and ending in 2006.

8 Q And is this an accurate summary of the kind of data
9 that is in the chelation database?

10 A Yes.

11 MR. LEWIS: Your Honor, I ask that 405
12 be admitted.

13 MR. SCOTT: No objection.

14 THE COURT: Received.

15 BY MR. LEWIS:

16 Q Could you explain this to the jury, please, Doctor.

17 A Well, what this means -- I do consider it good
18 news -- is that the number of children with
19 blood-lead levels who are high enough to require
20 treatment with medications, chelation, has gone
21 down over time. It would be nice if we could
22 chelate lead even out of these children, but
23 there's no evidence that those drugs work at levels
24 below a certain blood-lead level, so the medicines
25 become ineffective under that level. But this is

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1 STATE OF WISCONSIN)
2) SS
3 MILWAUKEE COUNTY)
4

5 We, JULIE A. POENITSCH, RPR/RMR/CRR, JULIE K.
6 LYLE, RPR/RMR/CRR, and STEPHANIE JACKSON, RPR/RMR, in
7 and for the State of Wisconsin, do hereby certify that
8 the foregoing proceedings were recorded by us and
9 reduced to writing under our personal direction and that
10 it is a true and correct transcript of said proceedings
11 had in said matter to the best of our knowledge.

12 In witness whereof, we have hereunto set our
13 hand at Milwaukee, Wisconsin, on this 29th day of May,
14 2007.
15
16
17
18

19 JULIE K. LYLE

JULIE A. POENITSCH

20
21 STEPHANIE JACKSON
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23
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25

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Transcript of Proceedings - 5/30/2007

Vol. 7

1 STATE OF WISCONSIN : CIRCUIT COURT : MILWAUKEE COUNTY

2 -----

3 CITY OF MILWAUKEE,
4 a municipal corporation,

5 Plaintiff,

6 vs.

Case No. 01-CV-3066

7 NL INDUSTRIES, INC.,
8 a foreign corporation, et al.,

9 Defendants.

10 Before the Honorable

11 JOHN FRANKE, Circuit Court Judge

12 Branch 25, presiding

13

14 Wednesday, May 30, 2007

15 VOLUME 7

16 9:31 a.m.

17 A P P E A R A N C E S:

18 MICHAEL HAUSFELD, RICHARD LEWIS, RICHARD SERPE, TERRY
19 NILLES, SARAH SCHUBERT, Attorneys at Law, appeared on
20 behalf of the Plaintiff.

21 JAMES T. MURRAY, JR., DONALD SCOTT, ANDRE PAUKA,
22 MICHAEL WIRTH, Attorneys at Law, appeared on behalf of the
23 Defendant.

24

25

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1 screen, please. Do you recognize the Milwaukee
2 Health Department newsletter?

3 A. The lead poisoning prevention newsletter, yes.

4 MR. SCOTT: I offer 804, Your Honor.

5 THE COURT: Any objection?

6 MR. LEWIS: No objection, Your Honor.

7 THE COURT: Received.

8 (Whereupon, Exhibit No. 804 was
9 received into evidence.)

10 BY MR. SCOTT:

11 Q. And on the third page -- may I have the screen?

12 Thank you. That the brochure that we were just
13 looking at is among the materials that is made
14 available to the public, Lead-Safe Procedures and
15 Home Improvement. Is that true?

16 A. Yes. This was from 2005. It appears to -- I can't
17 see -- it says that it is "material available from
18 the health --" I presume the next word is
19 "department". Yes.

20 Q. Okay. And is a guide for property owners explaining
21 how to use lead-safe procedures for home improvement
22 and renovation?

23 A. Yes.

24 Q. So it is the Milwaukee Health Department's public
25 information of how homeowners can do property

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1 renovation in a lead safe manner for themselves and
2 their families?

3 A. The text that you read from the pamphlet itself
4 previously stated work that is done by yourself or
5 by contractors.

6 Q. You have a choice? Yes? It refers to a choice,
7 work done by yourself or by contractor?

8 A. Right. And informs the consumer of steps that need
9 to be taken by anyone working around lead-based
10 paint.

11 Q. Yes. Now, let's turn more to routine maintenance
12 than renovation practices in these last two
13 exhibits. Off the screen I would like you to look
14 at Exhibit 728. And do you recognize an
15 informational brochure for parents that is
16 distributed by the Milwaukee Health Department?

17 A. I have not seen it previously.

18 Q. All right. Then have you seen Exhibit 507 before,
19 the sorry I missed you document?

20 A. Not to my recollection.

21 Q. From your experience as the commissioner, did the
22 Health Department advise parents on how they could
23 protect children from lead poisoning?

24 A. Yes.

25 Q. Which included the advice to remove or cover any

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1 chipped paint, right?

2 A. Yes. I can't -- I don't know the origin of what
3 you're showing me but I think the answer to the
4 question is yes.

5 Q. I'm just using this to remind myself and going by
6 your memory. Advise parents that they could protect
7 their children by removing or covering chipped
8 paint, right?

9 A. I do not recall suggesting to parents that they
10 remove paint.

11 Q. Scrape it off? Just cover it?

12 A. Wet wiping deteriorated areas was definitely
13 recommended.

14 Q. How about preventing children from eating chips and
15 cleaning up chips?

16 A. Yes. Those would have been recommended.

17 Q. A lot of washing hands frequently?

18 A. Absolutely.

19 Q. Giving them a proper diet high in calcium and iron?

20 A. Yes.

21 Q. And washing toys?

22 A. Yes.

23 Q. And running water for a while before using it so
24 there's not lead in the water?

25 A. Yes.

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1 Q. Now, you recognize, Dr. Foldy, that there are
2 millions of homes in the United States that do have
3 lead-based paint and have children and that children
4 don't get elevated blood levels. You know that?

5 MR. LEWIS: Objection, Your Honor.

6 THE COURT: Overruled.

7 THE WITNESS: Can you restate the
8 question?

9 Q. (By Mr. Scott) Even though there are millions of
10 homes in the United States that have lead-based
11 paint and they have children and children do not get
12 elevated blood levels?

13 A. I believe that's true.

14 Q. And, in fact, your home and homes have been among
15 them, correct?

16 A. Correct.

17 Q. You testified earlier this morning with Mr. Lewis
18 that you believe intact lead-based paint was a
19 hazard. Do you recall that?

20 A. That it represents a potential hazard.

21 Q. Potential hazard.

22 A. And can create dust, yes.

23 Q. Now, you have some personal experience living in
24 older homes and following the kinds of practices the
25 Health Department has recommended to people?

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1 STATE OF WISCONSIN)
2) SS
3 MILWAUKEE COUNTY)

4 We, JULIE A. POENITSCH, RPR/RMR/CRR, STEPHANIE
5 JACKSON, RPR/CMR, and SARAH A. HART, RPR/RMR/CRR, Notaries
6 Public in and for the State of Wisconsin, do hereby
7 certify that the foregoing proceedings were recorded by us
8 and reduced to writing under our personal direction and
9 that it is a true and correct transcript of said
10 proceedings had in said matter to the best of our
11 knowledge.

12 In witness whereof, we have hereunto set our
13 hand at Milwaukee, Wisconsin, on this 30th day of May,
14 2007.

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JULIE A. POENITSCH

STEPHANIE JACKSON

SARAH A. HART

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Transcript of Proceedings - 5/31/2007

Vol. 8

1 STATE OF WISCONSIN : CIRCUIT COURT : MILWAUKEE COUNTY

2 -----
3 CITY OF MILWAUKEE,
4 a municipal corporation,

5 Plaintiff,

6 vs.

Case No. 01-CV-3066

7 NL INDUSTRIES, INC.,
8 a foreign corporation,

9 Defendants.

10 Before the Honorable

11 JOHN FRANKE, Circuit Court Judge

12 Branch 25, presiding

13
14 Thursday, May 31, 2007

15 VOLUME 8

16 9:00 a.m.

17 A P P E A R A N C E S:

18 MICHAEL HAUSFELD, RICHARD LEWIS, RICHARD SERPE, TERRY
19 NILLES, SARA SCHUBERT, Attorneys at Law, appeared on
20 behalf of the Plaintiff.

21 JAMES T. MURRAY, JR., DONALD SCOTT, ANDRE PAUKA,
22 MICHAEL WIRTH, JOHN HUGHES, Attorneys at Law, appeared on
23 behalf of the Defendant.
24
25

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1 BY MR. SCOTT:

2 Q Dr. Markowitz, do you remember that I've asked you
3 that question in the past?

4 A I think you did. I don't remember.

5 MR. SCOTT: Your Honor, may I give the
6 witness a copy, before I show on the screen, of
7 testimony from December of 2005?

8 THE COURT: Yes.

9 BY MR. SCOTT:

10 Q If you would look at the testimony beginning at line
11 nine.

12 Your Honor, may I display the
13 testimony?

14 MR. HAUSFELD: Your Honor, may we see it
15 first?

16 MR. SCOTT: I only have the one copy.

17 THE COURT: Can you put it on the screens
18 that we have --

19 MR. SCOTT: Sure.

20 THE COURT: -- besides the jury screen?
21 Any objection?

22 MR. HAUSFELD: I'm just trying to see if I
23 can get the page before and the questions after.

24 THE COURT: Well, within reason, anyone can
25 place published deposition testimony in context, but

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1 I'll permit Mr. Scott to proceed.

2 MR. SCOTT: May I?

3 THE COURT: Including publishing this to
4 the jury.

5 MR. SCOTT: Would you turn that on, please.

6 Q Doctor, do you recall that in December of 2005, I
7 asked you this question beginning at line nine:

8 "You have no knowledge of any medical
9 or scientific information known to the National Lead
10 Company that was concealed from or hidden from either
11 the public health community or the public, do you?"

12 And your answer was, "That is
13 correct."

14 Was that your testimony at that time?

15 A It was.

16 Q Did you have any qualifications or doubt about the
17 meaning of the question?

18 A Well, I think I did.

19 Q Would you agree, Doctor, that as far as you know,
20 there is no medical or scientific information known
21 to the Lead Industries Association that was ever
22 concealed or hidden from the public or from the
23 public health community? Is that true?

24 A It depends what you mean by "concealed" or "hidden
25 from."

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1 Q Do you remember answering exactly that question when
2 I asked it of you in the past?

3 A I do.

4 Q Line four, right above the one we just read. Was
5 this your testimony at that time about a
6 year-and-a-half ago:

7 "Question: You have no knowledge of
8 any medical or scientific information that was known
9 by the Lead Industries Association that was hidden
10 either from the public or from the public health
11 community, do you?

12 "Answer: I have no such knowledge,
13 no."

14 Was that your testimony at that time?

15 A Yes, it is, that there was no medical or scientific
16 information that was actively hidden. It does not
17 mean that it was provided to them.

18 Q Would you agree -- you've researched the question
19 whether there was any information about lead paint or
20 lead poisoning that was known to the manufacturers
21 like National Lead and was not actually in the
22 published literature. You've looked at that
23 question, too; haven't you?

24 A I have.

25 Q And have you given testimony in the past that in all

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1 chance to look at it before it's published?

2 THE COURT: Yes.

3 MR. HAUSFELD: Thank you, Your Honor.

4 MR. SCOTT: Ready? Counsel?

5 There will be a series of questions on
6 this, Your Honor, not just the one question and
7 answer. It does carry over to the next page.

8 THE COURT: Is there an objection to the
9 publication of the question and answer beginning on
10 line 14?

11 MR. HAUSFELD: How far are we going, Your
12 Honor?

13 MR. SCOTT: I'm going to continue
14 questioning what's on the next page of the
15 deposition.

16 MR. HAUSFELD: Just so I can get a sense of
17 where you're going and whether or not there's any
18 objection. What lines were you -- where are you
19 intending to stop?

20 MR. SCOTT: I'm going to question on the
21 subject matter on the next page or two, but I don't
22 know what questions I'll ask until I hear the
23 answers.

24 MR. HAUSFELD: I guess we'll just have to
25 do it, Your Honor, on a question-by-question basis.

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1 THE COURT: Unless there's some challenge
2 to the authenticity of the transcript, I'll permit
3 Mr. Scott to cross-examine the published testimony.
4 If there's an objection along the way, you can make
5 it. If other testimony from the same or a different
6 deposition ought to be published along this -- along
7 with this, we can deal with that.

8 Go ahead, Mr. Scott.

9 MR. SCOTT: Thank you.

10 Q Beginning on line 19, Dr. Markowitz. And
11 recognizing, as you and I both know, this subject
12 continues on the next page.

13 A Correct.

14 Q Okay. Recognizing that --

15 A Correct.

16 Q -- in your deposition, were you asked this question:
17 "Previously, I believe you've testified that there's
18 no information about lead paint or lead poisoning
19 known to the manufacturers that was not in published
20 literature; is that correct?

21 "Answer: I think that's correct."

22 Was that your answer to that
23 particular question at that time?

24 A It was.

25 Q Now, in fact, you pointed out -- if we could go off

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1 the screen for a moment -- that in further research,
2 you and your colleague, Dr. Rosner, had found
3 something from 1959; right?

4 A Correct.

5 Q And you brought up that one item as an exception to
6 the previous testimony you had given?

7 A I don't think I said it was an exception.

8 Q You brought up the 1959 item; right?

9 A I did bring it up, yes.

10 Q Yes. And that was a report in 1959, something at
11 a -- an NPVLA meeting that concerned an area of
12 Cleveland; is that right?

13 A That is correct.

14 Q And even on that point, you testified that you didn't
15 know that it had not been published. You just didn't
16 know for sure whether it had been; right?

17 A I -- I don't remember what my exact answer was, but I
18 think it was that I wasn't sure if it had been
19 published or not.

20 Q And are you still not sure whether it was published
21 or not?

22 A I have not found any reference to its being
23 published.

24 Q Okay. You're talking --

25 A I'm not -- I'm not -- I'm sorry.

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1 A Now, you're talking about any specific decision on a
2 specific job, rather than -- rather than general
3 influence that the LIA may have -- may have had on
4 the use of lead in -- in -- in government jobs.

5 Q I'll come to general influence in a minute. But
6 you're not aware of -- that any agency of the federal
7 government ever made a decision to specify lead paint
8 instead of a different kind of paint because of the
9 LIA.

10 A That I -- I have no evidence about that.

11 Q Let's talk first about the U.S. Forest Products
12 Laboratory. The jury heard them referred to during
13 opening statement.

14 The U.S. Forest Products Laboratory is
15 an agency of the federal government, a part of the
16 U.S. Department of Agriculture?

17 A That's correct.

18 Q Formed in about 1918?

19 A I think that is correct.

20 Q And probably still in existence today, but for our
21 purposes, they then stayed in business for many
22 decades.

23 A Yes, and they were in existence for many decades.

24 Q And they have laboratories over in Madison that have
25 always done their own research, scientific research

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1 on paints?

2 A Yes. In fact, I think when I was at the University
3 of Wisconsin, I passed by the Forest Products
4 Laboratory on a number of occasions.

5 Q So they were studying paints from 1918 on, which is
6 10 years before the LIA was even created.

7 A That is correct.

8 Q And you're aware that, at least from the early 1920s,
9 the Forest Products Laboratory was recommending the
10 use of more lead in paint.

11 A I'm not sure what you mean by "more lead in paint."

12 Q They were recommending the use of lead-based paint.

13 A They were recommending the use of lead-based paint,
14 yes.

15 Q Let's look at one example.

16 Oh, Your Honor, I didn't mean to leave
17 that up but -- while I did --

18 Exhibit 542. Are you familiar with an
19 article written by the Forest Products Laboratory and
20 published in the Consumers Guide of 1939?

21 A I don't remember it right now.

22 Q Okay. Then I won't put it -- I won't put it in
23 through you.

24 Have you read publications by the U.S.
25 Forest Products Laboratory from the 1920s and '30s

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1 recommending the use of lead paint?

2 A It seems to me that I have seen some publications in
3 which they talked about using lead paints and nonlead
4 paints.

5 Q My question was whether they recommended.

6 A I can't -- I can't remember specifically if they
7 recommended lead in paint or not. They may have.
8 I -- I just don't remember right now.

9 Q Let me try to refresh your recollection then from
10 this document.

11 This is Exhibit 542. And page nine is
12 on the screen before you, column three. The U.S.
13 Forest Products Laboratory discussing their Type L
14 lead paint. Just read that to yourself and see if it
15 refreshes your recollection on the position taken by
16 this agency.

17 A Yes. Could I just look at that page as a whole for a
18 second?

19 Q Here you go.

20 A Great.

21 Q Does this help?

22 A Great.

23 Q The question is, does it help you remember maybe
24 seeing this magazine before, with the article from
25 the Forest Products Lab?

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1 BY MR. SCOTT:

2 Q There you go.

3 A Yes. Do you want me to read the second page as well?

4 Q It goes on for three pages, actually. Do you
5 remember the document?

6 A I do. This -- I mean, I -- sorry. I remember this
7 page. And certainly what he is saying in this page
8 is that white lead and oil is a reliable product
9 because it is standard, not it is -- unlike prepared
10 paints, which may have different characteristics
11 according to which brand is involved, white lead and
12 oil is -- is reliable and -- you know, and his
13 concern is that if pure white lead prepared paint
14 is -- is gone into, that that would destroy that
15 reliability.

16 Q Do you remember that Dr. Brown, for those reasons,
17 among others, supported the use of more lead pigment
18 in paint for higher quality?

19 A As I recall, he did say that, yes.

20 Q And he said that in 1938, which was the same time as
21 the LIA white lead promotion campaign that you talked
22 about on direct examination; right?

23 A Yes, that's -- that's true. He is not -- again, he
24 is a chemist talking about the physical properties of
25 the paint. He is not talking about the potential

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1 dangers of the paint.

2 Q Are you suggesting he didn't know?

3 A No. I think he did know that -- that -- that there
4 were these potential dangers. But as a chemist, that
5 was not his, you know, concern. His concern and the
6 Forest Products Laboratory concern was comparing the
7 painterly qualities of different pigments in
8 different kinds of paints. And that's what he did,
9 and that's what he wrote about, and that's what he
10 was very good at.

11 MR. SCOTT: Your Honor, may I now display
12 this letter from Exhibit 840? It's a three-page
13 letter, and I'll offer those pages when we come to
14 that but --

15 THE COURT: Yes.

16 MR. SCOTT: Okay. Here we have --

17 MR. HAUSFELD: Your Honor, may I just
18 preserve an objection to the production of letters by
19 other doctors to the LIA?

20 MR. SCOTT: May I proceed, Your Honor?

21 THE COURT: Let me see how this is going to
22 be used. Go ahead.

23 BY MR. SCOTT:

24 Q Now, Dr. Markowitz, you recognize this as a letter
25 from the Forest Products Laboratory to Felix Wormser

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1 at the LIA in 1939; right?

2 A It's from F.L. Brown, right, at the Forest Products
3 Laboratory to Felix Wormser.

4 Q Okay. Fair enough. The chief chemist.

5 A Correct.

6 Q And here in his second paragraph, he wrote to
7 Mr. Wormser, "You know that we have long emphasized
8 certain important advantages of pure white lead paint
9 that commend it strongly to any paint users." Do you
10 remember that generally being Dr. Brown's position?

11 A Yes, that was.

12 THE COURT: Excuse me.

13 THE WITNESS: I'm sorry.

14 THE COURT: Is there an objection?

15 MR. HAUSFELD: I think there was possibly a
16 misreading. It was "many" as opposed to "any."

17 MR. SCOTT: Many paint users. If I misread
18 that, I didn't mean to.

19 Q Was that generally his position?

20 A Yes, for the reasons that I indicated before and
21 which he discusses in the next few lines.

22 Q You've already explained that. And do you have an
23 opinion about "we" referring to -- you talked about
24 who "we" meant with the LIA. Does "we" here mean the
25 U.S. Forest Products Laboratory, "we"?

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1 A I think that is true, that he's talking about the
2 Forest Products laboratory.

3 Q Now, I won't spend more time today on Dr. Brown, but
4 I do want to ask this: You made it a specific point
5 to research whether or not the industry had some
6 undue influence or impact on Dr. Brown's
7 recommendations; did you not? You looked into that
8 specifically.

9 A Well, looked into the Forest Products laboratory and
10 gathered materials from the Forest Products
11 Laboratory and examined their records, yes.

12 Q And what you found was there were scientific
13 communications between scientists in the government
14 and industry; correct?

15 A That is correct.

16 Q But you did not find even an attempt by the industry
17 scientists to influence the recommendations of the
18 Forest Products Laboratory.

19 A I can't recall, I mean, all the things that we found.
20 But basically what we found is that the evidence was
21 absent about any influence or lack of influence that
22 the representatives of the lead industries had on the
23 Forest Products Laboratories. The Forest Products
24 Laboratory was mostly doing paint tests. They put up
25 fences, and they painted those fences with many

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1 different kinds of paints and pigments, and then they
2 evaluate --

3 Q Doctor, my question --

4 THE COURT: Hold on.

5 BY MR. SCOTT:

6 Q My question was simply whether or not you found any
7 evidence of influence upon the Forest Products
8 Laboratory or an attempt to influence?

9 MR. HAUSFELD: Your Honor, I think the
10 witness should be allowed to finish, and then he can
11 ask another question, if he would like.

12 MR. SCOTT: I really hate to interrupt,
13 Your Honor, but --

14 THE COURT: Hold on. I believe the last
15 answer was or had become nonresponsive, and
16 Mr. Scott's proceeded with a question. You can
17 answer the question, if you understand it.

18 BY MR. SCOTT:

19 Q Was there an attempt to influence?

20 A I was not able to find evidence of an attempt to
21 influence or -- or not. There was certainly a lot of
22 communication and cooperation between members of the
23 industry and the chemists at the Forest Products
24 Laboratory.

25 Q Let me turn now to the National Bureau of Standards,

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1 which is an organization of the U.S. government that
2 issued building specifications; right?

3 A Correct.

4 Q Part of the U.S. Department of Commerce?

5 A Correct.

6 Q And the National Bureau of Standards was issuing
7 building specs, specifications, from the 1920s for
8 decades until the agency names changed; right?

9 A Correct.

10 Q And have you researched what they were recommending
11 be used -- specifying be used in government
12 buildings?

13 A Yes.

14 Q And do you remember finding that the National Bureau
15 of Standards recommended and specified the use of
16 lead-based paint for many of their buildings?

17 A In a variety of cases they did, yes.

18 Q And they did that, from your research, because of the
19 durability of lead paint; right?

20 A I think that -- that that was one of the -- the
21 factors. I -- I can't remember all the -- the bases
22 upon which they made a decision about recommending
23 white lead paint.

24 Q Now, do you recall that not only was the Bureau of
25 Standards specifying paint be used for government

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1 buildings, but they were also answering questions
2 received from the public, from school boards?

3 A I seem to recall that, yes.

4 Q And do you remember that the Bureau of Standards,
5 when asked about paint, recommended to the school
6 boards the use of more lead-based paint?

7 A Yes. Again, they were recommending, as -- as you
8 indicated before, on the basis of durability and not
9 on the basis of other factors.

10 Q Do you remember they made those recommendations to
11 the school board for the City of New York?

12 A I think that is correct.

13 Q And Minneapolis?

14 A I don't recall Minneapolis specifically.

15 Q Okay.

16 A But --

17 Q You know that that was their position when asked, use
18 more lead-based paint?

19 A Well, sometimes. We don't --

20 Q For durability?

21 A For durability, yes. I mean, they were recommending
22 it on very narrow grounds.

23 Q And these were chemists also, like Dr. Brown.

24 A Yes, they were chemists, and that's -- that's what
25 their concern was, was how long it lasted, how well

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1 it lasted, and not -- they were not concerned about
2 the health effects.

3 Q Dr. Markowitz, you know that the people in the
4 National Bureau of Standards spoke about childhood
5 lead poisoning issues in the 1930s with experts like
6 Alice Hamilton?

7 A I recall that they spoke with Alice Hamilton. I
8 cannot recall that they spoke with her about
9 childhood lead poisoning. They did speak with
10 someone else about childhood lead poisoning, but I
11 don't recall their speaking -- they -- they did see
12 her, and the evidence that I saw was ambiguous about
13 whether she was talking to them about childhood lead
14 poisoning or not.

15 Q You're referring now to Dr. Ella Oppenheimer at the
16 Children's Bureau?

17 A Correct.

18 Q And she certainly had an interest in children, and
19 she came over to talk to the National Bureau of
20 Standards in the 1930s about the health issues that
21 related to their work?

22 A Well, I think she went there for a very specific
23 purpose. She went there because there was a concern
24 about lead on cribs and toys. And she had sent out a
25 letter to a variety of furniture manufacturers, and I

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1 exceptions.

2 THE COURT: Well, I believe the witness
3 indicated that this was a document he has reviewed.
4 I'll permit you to question him about it. If there's
5 any -- the hearsay objection is overruled.

6 BY MR. SCOTT:

7 Q Here we see on the screen, April 10 -- that's 1935 --
8 Dr. Ella Oppenheimer, Children's Bureau, Department
9 of Labor pays a visit, re: danger of lead poisoning
10 from painted toys, et cetera. Correct?

11 A Yes. And the context -- do you want me to talk about
12 the context for this?

13 Q We'll cover our ground faster, Doctor, if I just ask
14 you questions.

15 A I'm sorry.

16 Q That's all right. And here we don't know what the et
17 cetera means, but we do know that she was coming over
18 to talk to the National Bureau of Standards about
19 issues -- something related to the danger of lead
20 poisoning and children because that's her job. We
21 know that much.

22 MR. HAUSFELD: That is the problem, just --

23 THE COURT: Sustained. If that's an
24 objection, it's sustained.

25 MR. HAUSFELD: Yes, Your Honor.

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1 BY MR. SCOTT:

2 Q All right. She paid a visit concerning the danger of
3 lead poisoning from painted toys and maybe something
4 else?

5 MR. HAUSFELD: The document says what it
6 says, and I don't believe the witness -- excuse me,
7 counsel can read any more into it than what appears
8 on the document on its face.

9 THE COURT: I would like to avoid a sidebar
10 at this point.

11 MR. SCOTT: Your Honor, I'll just offer it
12 for what it says on its face.

13 THE COURT: Do you expect to offer some
14 further foundation for this particular document
15 beyond what's been presented through this witness?

16 MR. SCOTT: Not at that -- not at this time
17 through this witness, Your Honor, so I'll -- I'll
18 move on to something else, and we'll do more another
19 time.

20 THE COURT: All right.

21 MR. SCOTT: Okay.

22 THE COURT: Let's move on.

23 BY MR. SCOTT:

24 Q Now, you do know, Dr. Markowitz, that the National
25 Bureau of Standards continued to specify the use of

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1 lead-based paint for home use through the 1930s,
2 1940s at least; correct?

3 A I can't recall specifically, but I think that's
4 correct, yes.

5 Q Let me -- okay. And they recommended it and
6 specified it for home use for both interiors and
7 exteriors during that period of time?

8 A I think that is correct.

9 Q And I stop at 1950, because the agency names changed
10 then. You're aware of that.

11 A Yes.

12 Q Now, do you know of any evidence, sir, that NL had
13 any influence on the views of the Department of
14 Commerce's National Bureau of Standards regarding the
15 paint specifications?

16 A Well, certainly the -- the industry and NL, part of
17 the industry, that is, the lead industry, was in
18 constant communication with the Department of
19 Commerce and the Bureau of Standards. They were
20 talking to each other all the time, they had a
21 cooperative relationship. The Bureau of Standards
22 depended upon members of the industry for information
23 about their products and about what was being used in
24 those products and the qualities of those products.

25 And so there really was a -- a very

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1 much a cooperative relationship here. It was not a
2 situation like we have today where you have an agency
3 that's regulating another, you know, industry and
4 the -- they're, you know, in conflict with each
5 other. This is -- this is a cooperative enterprise
6 between the Bureau of Standards and the paint
7 manufacturers and the lead industry.

8 Q Do you remember my question was about the National
9 Lead Company?

10 A I was trying to put that into a -- into a broader
11 context. I would put National Lead Company in
12 that -- in that broader context.

13 Q Would you agree that you don't know of any influence
14 that the National Lead Company had upon the Bureau of
15 Standards in issuing paint specifications?

16 A As I recall, the LIA talks about having an influence
17 or trying -- I shouldn't say having an influence.
18 They talk about trying to influence specifications by
19 the government, and that would include the National
20 Bureau of Standards.

21 Q Would you agree that you don't know of any
22 specifications of the National Bureau of Standards
23 that were influenced by the LIE -- LIA?

24 A I don't know of any specific examples. I went
25 through the National Bureau of Standards' records,

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1 Q Doctor, on page 884 of this exhibit do you see that
2 Dr. Blackfan's case number one is a child who -- who
3 died who was found to have -- at his orphanage have
4 eaten all of the paint off the crib?

5 A That is correct.

6 Q That's the -- that's the kind of finding that the
7 American doctors were getting in the 19 teens; is
8 that right?

9 A Yes, I think that's true, in the 19 teens.

10 Q And case number two -- actually two and three are
11 actually two brothers who are reported by their
12 father to have gnawed paint and ruined an entire set
13 of parlor furniture?

14 A Correct.

15 Q Do you recall that?

16 A I do.

17 MR. SCOTT: Your Honor, I offer Exhibit 676
18 to the extent published.

19 THE COURT: Any objection?

20 MR. HAUSFELD: No, Your Honor.

21 THE COURT: Received.

22 BY MR. SCOTT:

23 Q Dr. Markowitz, are these kinds of cases reported by
24 Dr. Blackfan fairly typical of the kinds of lead
25 poisoning of children from paint that doctors found

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1 at that time in the teens?

2 A I -- no, I don't think that's true, because in
3 Australia they were finding many different kinds of
4 lead poisoning cases other than what you've described
5 here.

6 Q Okay. In Queensland in the tropical area?

7 A Well, it was in Queensland.

8 Q Yeah, we'll come to that in the morning. The
9 American reports, these are typical of the American
10 reports of doctors at that time?

11 A Again, we're talking about the reports of some
12 specific cases. There were not very many specific
13 cases in the teens, but there were some. And I
14 don't -- I can't recall if all of them were of this
15 character or not. There may be others that were less
16 severe.

17 Q So you're not comfortable saying whether they were
18 typical of the cases of that time or not?

19 A I can't remember all the cases, and so I would rather
20 not give an opinion about whether they were typical
21 or not.

22 Q Are you comfortable though confirming for the jury
23 that the cases reported in those times involved what
24 today we consider very large amounts of lead paint?

25 A I think that -- that the -- yes, that the cases at

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1 that time were causing death and severe convulsions
2 and talking about children ingesting large amounts of
3 lead paint.

4 Q And at that time the doctors did not even have the
5 technology to measure blood lead as we know it today;
6 right?

7 A Correct.

8 Q So we don't know what kind of blood lead was involved
9 in these cases?

10 A Right. We know that they were suffering convulsions
11 and dying, so we know that the blood leads were very
12 high.

13 Q Okay. Fair enough. And you recall that Dr. Ruddock
14 reported similar cases in 1924?

15 A I can't remember the specific cases that Dr. Ruddock
16 was reporting on in 1924.

17 Q Okay. Would you look on your screen, Exhibit 9. Do
18 you recognize Dr. Ruddock's report "Lead Poisoning in
19 Children" from the Journal of the American Medical
20 Association in 1924?

21 A I do recognize that, yes.

22 Q All right. And then if we look at case number one --
23 Your Honor, may I display as I go
24 along?

25 THE COURT: Yes.

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1 BY MR. SCOTT:

2 Q So here we have Ruddock, the Journal of the AMA in
3 1924, Lead Poisoning in Children, With Special
4 Reference to Pica. He's a doctor in Los Angeles.
5 And then in his report of cases -- it's a little hard
6 to read. Let me read this. "The child had been
7 observed by several physicians in their offices, and
8 in one instance the diagnosis of appendicitis was
9 made. The correct diagnosis" --

10 A However.

11 Q -- "however, might have been made by anyone who
12 called the child's home, because every bit of paint
13 on porch railings, window sills, crib, bureau, chair,
14 and even white enameled door casings as high as the
15 child could reach had been gnawed off."

16 Was that the kind of cases Dr. Ruddock
17 was finding?

18 A Well, he was only reporting on two cases. So -- and
19 he in the course of this article talks more generally
20 about the issue of childhood lead poisoning. And
21 it's -- is not talking about -- you know, giving a
22 whole list of cases, but saying that the problem is
23 lead, not -- not the children who are gnawing off,
24 you know, all of this furniture.

25 Q Doctor, I didn't say the problem was the children. I

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1 asked if these are the kinds of cases Dr. Ruddock was
2 reporting.

3 A He only reported on two cases.

4 Q Okay. And the cases -- the cases that were reported
5 involved very large amounts of paint in the child;
6 correct?

7 A Yes, because to cause death and convulsions it had to
8 involve very large amounts of lead paint.

9 Q And do you remember what recommendation was given by
10 Dr. Ruddock to address this problem in the Journal of
11 the American Medical Association?

12 A I can't recall specifically what he recommended.

13 MR. SCOTT: I'm displaying a section called
14 "Treatment," Your Honor, paragraph one. "The
15 treatment of this condition may be considered from
16 three points of view: One, prophylactic, careful
17 supervision of children during the age from one to
18 five -- to three years in which pica is most
19 prevalent. Wide publicity and education through
20 periodicals and home journals in order to educate the
21 parents." Then he goes on to talk to doctors about
22 symptoms.

23 For prevention, was that the advice
24 given by Dr. Ruddock as I read it?

25 A Yes, it was.

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1 Q And pica as the term was used at that time was used
2 in reference to children the doctors believed for a
3 period of time during their development put lots of
4 unnatural items in their mouth. That's what doctors
5 believed at the time.

6 A Well, there was a lot of disagreement about what pica
7 meant. Dr. Rosner and I went through many articles
8 that mentioned pica, and some described it that way,
9 others described it as simply an exaggeration of the
10 normal habit of children putting at a certain age
11 everything in their mouth. That's -- that's what
12 children do --

13 Q Did doctor --

14 A -- at a certain age. My apologies.

15 Q Did Dr. Ruddock describe it the way I have?

16 A He described it the way you did, and he cited a
17 Dr. Koplic as part of his definition. And Dr. Koplic
18 describes it more as a -- as a bad habit.

19 THE COURT: Are you able to spell
20 Dr. Koplic.

21 THE WITNESS: K-O-P-L-I-C.

22 THE COURT: All right.

23 MR. SCOTT: Your Honor --

24 THE COURT: I'm looking for a point to
25 break. If we're going to -- if there's more with

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1 STATE OF WISCONSIN)
2) SS
3 MILWAUKEE COUNTY)
4

5 We, STEPHANIE JACKSON, RPR/CMR, and SARAH A.
6 HART, RPR/RMR/CRR, Notaries Public in and for the State of
7 Wisconsin, do hereby certify that the foregoing
8 proceedings were recorded by us and reduced to writing
9 under our personal direction and that it is a true and
10 correct transcript of said proceedings had in said matter
11 to the best of our knowledge.

12 In witness whereof, we have hereunto set our
13 hand at Milwaukee, Wisconsin, on this 31st day of May,
14 2007.
15
16
17
18

19 SARAH A. HART

STEPHANIE JACKSON

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Vol. 9

1 STATE OF WISCONSIN : CIRCUIT COURT : MILWAUKEE COUNTY

2 -----

3 CITY OF MILWAUKEE,
4 a municipal corporation,

5 Plaintiff,

6 vs. Case No. 01-CV-3066

7 NL INDUSTRIES, INC.,
8 a foreign corporation,

9 Defendants.

10 Before the Honorable

11 JOHN FRANKE, Circuit Court Judge

12 Branch 25, presiding

13

14 Friday, June 1, 2007

15 VOLUME 9

16 9:30 a.m.

17 A P P E A R A N C E S:

18 MICHAEL HAUSFELD, RICHARD LEWIS, RICHARD SERPE,
19 TERRY NILLES, SARA SCHUBERT, KATHY KONOPKA, Attorneys at
20 Law, appeared on behalf of the Plaintiff.

21 JAMES T. MURRAY, JR., DONALD SCOTT, ANDRE PAUKA,
22 MICHAEL WIRTH, JOHN HUGHES, Attorneys at Law, appeared
23 on behalf of the Defendant.

24

25

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1 "White Lead," this states "The use of basic
2 carbonate of white lead in the paint, varnish, and
3 lacquer industry for interior paints is
4 negligible."

5 A And this is what I was referring to earlier.

6 THE COURT: Wait for a question, please.

7 THE WITNESS: Oh, I'm sorry.

8 BY MR. SCOTT:

9 Q My question is, do you know or not know whether
10 that statement is correct as of 1944 when it was
11 written?

12 A I don't know if it's correct or not.

13 Q You showed the jury with Mr. Hausfeld Exhibit 43 --
14 no, I got the wrong -- do you remember -- I have
15 the wrong number here.

16 Do you remember that in your direct
17 examination two days ago that you showed a picture
18 of paint cans, mixing instructions by the National
19 Lead Company in the late 1940s?

20 A I think it was 1950s, yes.

21 Q 1950, like one-half white lead, one-half -- do you
22 remember that picture?

23 A Yes, for interior wallboard or -- or walls, yes.

24 Q Have you read a -- off the screen, please -- a
25 document from the federal government

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1 U.S. Department of Commerce paint manual with
2 reference to federal specifications? Do you know
3 this one, 1945?

4 A I'm sorry, I don't know this one.

5 Q Do you know who the co-authors are, Percy H. Walker
6 and Eugene F. Hichson, H-I-C-H-S-O-N?

7 A From what I remember, they're with the Bureau of
8 Standards.

9 Q The National Bureau of Standards of the
10 U.S. Department of Commerce?

11 A Thank you for that correction, yes.

12 Q And they're chemists?

13 A That's my recollection, yes.

14 Q And for quite a number of years, they were the ones
15 principally responsible for writing federal
16 government building specifications?

17 A Yes.

18 Q Your Honor, may I display cover and this portion
19 for counsel?

20 THE COURT: Unless there's objection, you
21 may proceed.

22 BY MR. SCOTT:

23 Q Here's the cover, sir, on the screen. And what we
24 have here are Paint Manual, Federal Specifications,
25 Issued October 1945, U.S. Department of Commerce,

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1 National Bureau of Standards, that we've been
2 talking about; is that right, Doctor?

3 A That is correct.

4 Q And then going to the 14th page of the exhibit,
5 which is page 18 of the manual, do you recognize
6 the government's instructional pictures on how to
7 make lead paint for interiors and exteriors in
8 1945?

9 A Yes, this is taken directly from National Lead's
10 Handbook of Painting.

11 Q Which is a how-to-do-it book?

12 A Yes, it is.

13 Q Now, in 1945, and as you remember, the federal
14 government was still specifying leaded oil paint
15 for its own buildings for outside in 1945; is that
16 right?

17 A Yes, that is correct.

18 Q And for interior in 1945; is that right?

19 A Correct.

20 Q Both for repaint work and for new work?

21 A Correct.

22 Q Both for outdoors and indoors?

23 A Correct.

24 Q Now let's come to the 1950s. Specifically,
25 Exhibit 38 in 1953. I'll blow that up in a moment,

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1 just one moment, Doctor. This is one page you were
2 shown before. Now, context, 1953, at this date,
3 the City of Baltimore has already now issued an
4 ordinance concerning the use of interior lead paint
5 within the City; right?

6 A Yes.

7 Q That was the 1951 ordinance issued by Dr. Williams,
8 the commissioner of health of the City of
9 Baltimore?

10 A Huntington Williams, yes.

11 Q Huntington Williams. And that essentially banned
12 the use of lead pigment in paint for interiors
13 within the City of Baltimore?

14 A That is correct.

15 Q And it was the only restriction or ban of its type
16 in 1951; correct?

17 A That is correct.

18 Q And that resulted from a discovery of an unexpected
19 number of children who were eating peeling paint in
20 some poor areas of inner-city Baltimore after the
21 war?

22 A Not just peeling paint. Chewing on window sills
23 was probably the most important source, but --

24 Q And plaster? And plaster?

25 A And plaster.

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1 were other -- there were a few other organizations
2 and several organizations representing the lead
3 industry and the paint industry.

4 Q Now, in the 1950s, both before and after this 1955
5 new standard, articles were published by physicians
6 concerned about the issue of childhood lead
7 poisoning; is that right?

8 A Oh, yes.

9 Q There were articles by Dr. Randolph Byers from
10 Boston Children's?

11 A Correct.

12 Q By Dr. Huntington Williams, the commissioner of
13 health in Baltimore?

14 A Correct.

15 Q By Dr. Julian Chisolm at Johns-Hopkins in
16 Baltimore?

17 A Correct.

18 Q And by others?

19 A Correct.

20 Q Would you agree, Doctor, that during that period of
21 time, even in the 1950s, that well-intentioned
22 people took different positions on whether to
23 continue using lead-based paint?

24 A I -- I'm not in a position to know which people
25 were well intentioned and which people weren't well

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1 intentioned. I didn't know them. And I know them
2 only from reading reports by and about them.

3 Q Well, after the Baltimore ordinance of 1951, is it
4 true that for over the next 20 years, no agency of
5 the United States government urged or proposed a
6 ban on lead in house paint, 20 years?

7 A I think that's probably true, yes.

8 Q Including the Children's Bureau?

9 A I think that's true.

10 Q United States Public Health Service?

11 A Yes.

12 Q None of the federal agencies who were writing
13 building specifications?

14 A Yes.

15 Q Nor the surgeon general of the United States?

16 A Correct.

17 Q In fact, the surgeon general issued a report on
18 childhood lead poisoning as late as 1971. You're
19 familiar with that document, I believe?

20 A I think I have seen that, yes.

21 Q And in that document in 1971 the surgeon general
22 recommended protective measures for children but
23 did not urge a ban or restriction on the use of
24 lead in house paint; is that correct?

25 A I think that is correct.

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1 MR. SCOTT: Your Honor, I pass the
2 witness.

3 THE COURT: Mr. Hausfeld, you may proceed
4 with any redirect.

5 MR. HAUSFELD: Thank you, Your Honor.

6 REDIRECT EXAMINATION

7 BY MR. HAUSFELD:

8 Q Good afternoon, Doctor.

9 A Good afternoon --

10 MR. HAUSFELD: Good afternoon, ladies and
11 gentlemen.

12 THE WITNESS: -- Mr. Hausfeld.

13 MR. HAUSFELD: May we display
14 Exhibit 464. And could we have the next page,
15 please.

16 Q Doctor, have you seen this before?

17 A I have.

18 Q And it is from who, Doctor?

19 A This is a document from Robert Kehoe, and this
20 first page is reimbursement of \$25 for his
21 reviewing a workers' compensation case against the
22 National Lead Company. And it's from 1954.

23 MR. HAUSFELD: And may we turn the page,
24 please.

25 Q And have you -- can you identify this for us,

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Vol. 9

1 STATE OF WISCONSIN)
2) SS
3 MILWAUKEE COUNTY)

3

4 I , JULIE K. LYLE, RPR/RMR/CRR, Notary Public in
5 and for the State of Wisconsin, do hereby certify that
6 the foregoing proceedings were recorded by me and
7 reduced to writing under my personal direction and that
8 it is a true and correct transcript of said proceedings
9 had in said matter to the best of my knowledge.

10 In witness whereof, I have hereunto set my
11 hand at Milwaukee, Wisconsin, on this 1st day of June,
12 2007.

13

14

15

16

17

JULIE K. LYLE

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Vol. 10

1 STATE OF WISCONSIN : CIRCUIT COURT : MILWAUKEE COUNTY

2 -----
3 CITY OF MILWAUKEE,
4 a municipal corporation,

5 Plaintiff,

6 vs.

Case No. 01-CV-3066

7 NL INDUSTRIES, INC.,
8 a foreign corporation,

9 Defendants.

10 Before the Honorable

11 JOHN FRANKE, Circuit Court Judge

12 Branch 25, presiding

13
14 Monday, June 4, 2007

15 VOLUME 10

16 10:55 a.m.

17 A P P E A R A N C E S:

18 MICHAEL HAUSFELD, RICHARD LEWIS, RICHARD SERPE,
19 TERRY NILLES, CHRISTOPHER RIORDAN, Attorneys at Law,
20 appeared on behalf of the Plaintiff.

21 JAMES T. MURRAY, JR., DONALD SCOTT, MICHAEL
22 WIRTH, Attorneys at Law, appeared on behalf of the
23 Defendant.
24
25

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1 of Public Accountants; and I also maintain about 40
2 hours of continuing education a year, mainly in
3 government accounting and auditing.

4 Q Mr. Egan, would you like a glass of water?

5 A Sure.

6 THE COURT: Well, don't use last week's
7 water over there. Here's a new cup.

8 MR. HAUSFELD: I just asked if he'd like
9 it. I couldn't offer him any because we don't have
10 it.

11 Q What is your job description, Mr. Egan?

12 A I'm a special deputy comptroller and director of
13 accounts.

14 Q And for how long have you been with the City of
15 Milwaukee?

16 A I've been with the City since May of 1985.

17 Q And what has been your job since then?

18 A From 1985 to 2000, I was in the comptroller's
19 financial advisory division, from 2000 to 2004 I was
20 deputy comptroller, and since 2004 I've been special
21 deputy comptroller, director of accounts.

22 Q And what has that entailed in terms of your job
23 duties?

24 A Currently there's 43 people who report to me. I'm in
25 charge of divisions of payroll; general accounting;

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1 accounts payable; cost and revenue, which includes
2 grant administration; and also system support.

3 Q What is the general procedure by the City with regard
4 to auditing expenditures for programs?

5 A Any expenditure that's paid by the City is first
6 approved by the department. You have an independent
7 clerk who basically would enter it into the financial
8 system. You would have two approved persons within
9 the department that would sign, or in this case it
10 would have been done on the computer, approve that
11 signature. That comes to our department, including
12 any paperwork.

13 Every payment is reviewed by a staff
14 member in our department and approved and then
15 submitted to our payroll manager to review and
16 actually sent to the treasurer's office for payment.

17 Q Did there come a time, Mr. Egan, when you were asked
18 to review the expenditures by the City programs for
19 childhood lead prevention?

20 A Yes.

21 Q And could we please display Exhibit 392. I'm going
22 to ask if we can enlarge this exhibit.

23 Well, before we enlarge it, can you
24 identify this for us, please.

25 A These are expenditures from 1992 to 2006.

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1 MR. HUGHES: Excuse me, Mr. Egan. I think
2 we need some foundation here first. I would object.

3 THE COURT: I assumed that's what we're
4 going to hear.

5 MR. HAUSFELD: Yes, it's hard to get a
6 foundation before you identify what the document is.

7 THE COURT: Why don't you lay at least some
8 foundation for this before the jury sees it. I don't
9 know that they could read the little print, but if
10 it's going to be enlarged, let's hear the foundation
11 first and have a decision about whether this should
12 be received or not. And then the jurors can see it
13 if it is.

14 BY MR. HAUSFELD:

15 Q How did you go about compiling the expenditures for
16 the City programs for childhood lead poisoning?

17 A In late summer or early fall it was, in 2006, we met
18 with the business manager of the Health Department
19 that provided us with some preliminary damages in
20 this lawsuit that were put together by them. That
21 also was also identified to us, all the programs and
22 projects that were related to lead-paint abatement
23 that were identified for this lawsuit by the Health
24 Department.

25 We went through our audited accounting

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1 system project by project and looked at all the
2 detail of each project and basically verified or
3 changed the numbers that were earlier provided as to
4 the Health Department related to the costs for this
5 lawsuit.

6 Q What, if anything, did you do to verify that the
7 programs that were referred to you for purposes of
8 calculation all related to Milwaukee Childhood
9 Lead-Poisoning Prevention Programs?

10 A It was more reasonableness. The accountant who spent
11 a lot of time looking at this has been involved in
12 looking at lead paint or paint expenditures from the
13 Health Department since 1990. We recently looked at
14 all the -- all the projects and programs that were
15 identified and -- but mostly relied on the Health
16 Department's expertise in determining whether the
17 programs were lead-paint related.

18 Q In relying on the Health Department's expertise, did
19 you do anything to verify the accuracy of that
20 expertise as expressed to you?

21 A No.

22 Q Did you do anything in addition to verifying the
23 programs in connection with the computation of
24 expenditures? Did you verify the numbers as well?

25 A We verified all the numbers against our financial

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1 information management system.

2 Q How many pieces of paper, if you can estimate, did
3 you review in putting together this compilation
4 that's now Exhibit 392?

5 A Well, most of the information now in our new
6 financial system is done electronically. But
7 there's -- so we basically took a lot of our
8 information off our audited financial system. But
9 there were probably thousands of pieces of paper and
10 millions of pieces of data that we looked at.

11 Q And did you complete that review to a matter of
12 accounting satisfaction?

13 MR. MURRAY: Objection, Your Honor.

14 Mr. Egan has not been named as an expert.

15 THE COURT: You may answer that question
16 yes or no.

17 THE WITNESS: Yes.

18 THE COURT: I assume there's an objection
19 to further testimony about this?

20 MR. HUGHES: Yes, Your Honor. Mr. Egan was
21 not named as an expert, and we have not been put on
22 notice that he would offer anything but fact-witness
23 testimony.

24 THE COURT: Just a moment. Overruled.

25 Well, there was an answer. I asked about a

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1 continuing objection. At least at this point that
2 objection is overruled. You may proceed.

3 MR. HAUSFELD: Your Honor, at this point we
4 would request that Exhibit 392 be displayed.

5 THE COURT: Are you going to be offering
6 this exhibit at some point?

7 MR. HAUSFELD: Yes.

8 THE COURT: Is there an objection to its
9 receipt.

10 MR. HUGHES: There is an objection. I
11 think Mr. Egan already testified that he has no
12 personal knowledge that the money --

13 THE COURT: Don't argue it at this point.
14 At this point the objection to the receipt of this
15 exhibit is sustained.

16 BY MR. HAUSFELD:

17 Q Mr. Egan, did you make a calculation as to the
18 expenditures for the City's childhood lead prevention
19 programs?

20 A Yes.

21 Q And for what years?

22 A From 1992 to 19 -- from 1992 to 2006.

23 Q And that was for expenditures actually incurred in
24 pursuit of the programs?

25 A Yes.

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1 Q And did you arrive at an aggregate figure for those
2 expenditures?

3 A Yes.

4 Q And could you testify, please, as to what that figure
5 was.

6 MR. HUGHES: Objection.

7 THE COURT: Sustained.

8 Members of the jury, I'm going to
9 excuse you for a moment. Please wait in the jury
10 room. We'll either bring you back shortly or give
11 you an estimate of when we will be ready to proceed.

12 Jurors are excused. Counsel should
13 remain.

14 THE CLERK: All rise.

15 (Jury dismissed.)

16 THE COURT: The witness can remain at the
17 witness stand for the moment.

18 All I have immediately available in
19 terms of a description of this witness's testimony is
20 contained in the City's pretrial report filed May
21 2nd which had a summary of witness testimony. And
22 for Mr. Egan, it's a short paragraph that basically
23 says he will testify about facts he offered in his
24 deposition testimony. I assume that was an adverse
25 deposition. I'm gathering that there was no report

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1 filed for this witness, that he was not named as an
2 expert.

3 MR. LEWIS: The only report, Your Honor, is
4 the spreadsheet of damages which he has calculated.

5 THE COURT: I think I can guess, but I'm
6 trying to figure out how this -- figure out how this
7 compares to the more detailed spreadsheets I've seen
8 in the past that listed many individual programs.

9 MR. LEWIS: Yes, Your Honor. It's the same
10 total. And rather than organizing the expenditures
11 by grant, they are organized here by the four basic
12 areas of expenditure on the program: Primary
13 prevention, secondary prevention, and indirect costs.
14 I apologize, I don't have the document in front of me
15 for the fourth column. It's an aggregate of exactly
16 the same data as the spreadsheet that went grant by
17 grant and year by year.

18 Community outreach was the fourth
19 category, Your Honor, which is included as one of the
20 grant programs.

21 THE COURT: Well, let's deal with the
22 issues separately here, the expert witness objection
23 and the perhaps other foundation objections to the
24 exhibit. The reason I overruled the objection, even
25 though I was a little surprised he wasn't named as an

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1 is entitled "Annual Lead Grant Expenditures by
2 Program Activity," is a document we were supplied a
3 few weeks ago. When I deposed Mr. Egan on May 22nd
4 of 2007, I showed him that document. I asked him,
5 "Mr. Egan, I will hand you a document that I will
6 designate Egan Deposition Exhibit 26, the document
7 entitled 'Annual Lead Grant Expenditures by Program
8 Activity.' Have you seen this document before?"

9 "Answer: No.

10 "Question: So you didn't help prepare
11 this document?

12 "Answer: No."

13 So that's what I have to say about
14 that document.

15 The other point I would raise is that
16 in his deposition, I can point you to the pages that
17 I'd like Your Honor to read. Mr. Egan testified that
18 he had no personal knowledge that even \$1 was spent
19 on a paint-related activity as distinct from what the
20 City has identified as its lead program activities,
21 when pressed on whether or not he knew if money was
22 being spent on paint-related activities, he fell back
23 to "somebody told me."

24 I think that, therefore, there would
25 be no foundation as a fact witness for him to testify

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1 that any of the money that the City is claiming as
2 damages was spent on a paint-related activity. If
3 you like, I can point you to that section of the
4 deposition transcript.

5 THE COURT: Is this something different
6 than what you just pointed me to?

7 MR. HUGHES: Well, the part that you read
8 earlier was his admission that he had nothing to do
9 with preparing the document that the City is trying
10 to show to the jury to get admitted into evidence
11 right now, and that is on page 320 of his deposition,
12 lines 18 through 24.

13 And then on page 218 of his
14 deposition, I ask him what is -- page 218, line 7, I
15 asked him, "What is your basis that even \$1 was
16 charged to a paint-related expenditure?"

17 And then we have a two- or three-page
18 discussion where he admits over and over it's based
19 on someone telling him that, not on any personal
20 knowledge.

21 As a fact witness, I think he can only
22 testify as to what he has personal knowledge of. He
23 has no personal knowledge that any money was spent on
24 lead paint, so there's no foundation for his
25 testimony.

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1 THE COURT: How long is the deposition
2 transcript?

3 MR. HUGHES: This section I'm referencing
4 you to?

5 THE COURT: The whole thing.

6 MR. HUGHES: It goes from page 164 to --
7 this was his second deposition -- 323. So what, is
8 that 170 pages?

9 THE COURT: It's not a part of the court
10 record at this point. I don't know that the whole
11 thing needs to be. And you've characterized or
12 quoted from certain parts of this.

13 Mr. Lewis or Mr. Hausfeld, who's going
14 to argue why this exhibit ought to come in on the
15 existing foundation?

16 MR. LEWIS: Your Honor, Mr. Egan has
17 verified the accuracies of these numbers and can
18 testify to the procedures in the Health Department
19 and for compliance with federal grants that the
20 dollars spent in these programs were spent for the
21 purpose intended, for a legitimate purpose, childhood
22 lead-poisoning prevention. He has verified the
23 numbers in a different spreadsheet when it was
24 organized by grant -- they're the same numbers.
25 Since that time, he has reviewed this spreadsheet --

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1 MR. HUGHES: Yes.

2 THE COURT: Mr. Hughes.

3 MR. HUGHES: Yes.

4 THE COURT: A couple of weeks ago you
5 received Exhibit 392. Do you know exactly when?

6 MR. HUGHES: We -- I believe we received
7 it -- obviously before Mr. Egan's deposition. That's
8 why I was able to ask him about it at the deposition.

9 I think, as Your Honor referenced
10 before, all along, the way that the City has
11 identified its damages has been in a spreadsheet with
12 a breakdown of expenditures under different grants
13 and different categories of spending; and it has --
14 and when it produced that -- I guess they would call
15 it a summary of evidence chart -- each of -- this was
16 accompanied by a document that identified, for each
17 cell within that spreadsheet, the back-up
18 documentation that we could look at to determine
19 where the number came from. And if we want an
20 example of this kind of spreadsheet, it's Exhibit
21 404, fourth page, on your laptop.

22 And so that, I would gather, is the
23 kind of summary evidence chart that would be
24 appropriate because, as the rule provides, they did
25 provide us the underlying documentation to try to

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1 substantiate those numbers.

2 As far as Exhibit 392 is concerned, I
3 have no idea, and they have not identified any
4 documents which would show us how to break down
5 between expenditures on primary prevention, secondary
6 intervention, community outreach. As far as I know,
7 this document was not accompanied by a reference to
8 materials that would allow us to make the calculation
9 in that way. And certainly Mr. Egan, when asked at
10 his deposition what is primary prevention, he didn't
11 know.

12 So I don't see how -- and this
13 obviously was created before that deposition. So I
14 do not think there is any -- this is not a summary of
15 evidence chart because we have not been provided the
16 methods for calculating the numbers here in this
17 spreadsheet.

18 The other point I would like to make
19 is that I know -- I think it's probably proper as a
20 fact witness for Mr. Egan, if he wants to go to the
21 spreadsheet that you're looking at that we have had
22 an opportunity to review and say this is what the
23 lead program spent in each of these years and here's
24 how the comptroller's office is involved and here are
25 the kind of audits that take place, his personal

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1 knowledge of that all, that's fine. I'm talking
2 about Exhibit 404, page 4.

3 But what he has no basis to testify to
4 based on personal knowledge is whether any of that
5 money was spent on lead paint, and we would object to
6 any testimony from Mr. Egan on that subject.

7 MR. LEWIS: Your Honor, may I respond?

8 THE COURT: All right.

9 MR. LEWIS: Your Honor, 404 has the exact
10 same numbers. 404 has the names of the grants. In
11 light of the Court's consideration of making an
12 instruction to the jury on collateral source at the
13 close of evidence, we preferred not to list the
14 grants in this exhibit. But I agree with Mr. Hughes;
15 it's the identical information. The witness is
16 familiar with it. The witness can explain the
17 procedures as to these numbers. And we did provide
18 to the defendants the back-up for every single cell
19 in this spreadsheet, and to get from this spreadsheet
20 to 392 is a mathematical exercise.

21 We would be happy to examine the
22 witness on 404 as opposed to 392, because the bottom
23 line is we want to ask this witness what the
24 procedures are and what the number is, and both
25 documents have the same information.

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1 THE COURT: I'm looking at one copy of
2 Exhibit 404 on the laptop, and then the one that's on
3 our common screen here I believe is Exhibit 392.

4 MR. LEWIS: That's correct, Your Honor.

5 THE COURT: And I'm having trouble reading
6 any of the numbers precisely, but it's clear that 392
7 has four categories in the total, and 404 has many
8 more categories and then a total.

9 Are you saying, Mr. Lewis, that those
10 total numbers running along the bottom are all
11 exactly the same?

12 MR. LEWIS: No, Your Honor. I'm saying the
13 total of \$53,036,838 and the final total of
14 52,644,365 is exactly the same.

15 THE COURT: And did you explain to
16 Mr. Hughes in some way how one does the math and
17 takes all the categories on 404 and comes up with the
18 numbers on 392, or did you just provide him with this
19 and say this is our latest exhibit?

20 MR. LEWIS: Your Honor, these are the four
21 basic categories of expenditures that come out of the
22 spreadsheet 404. I wasn't present at the deposition
23 that Mr. Hughes is reading from, and I don't recall
24 all the questioning about 404.

25 MR. HAUSFELD: Your Honor, in order to

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1 A Yes.

2 Q And then you have a final total.

3 A Yes.

4 Q And what does that final total represent?

5 A That represents, based upon our review of all the

6 financial information on the City's financial system,

7 the total amount after a reduction of -- for possible

8 non-paint-related properties, the total amount that

9 they'll -- in our best professional judgment relates

10 to non-paint-related expenditures for the City.

11 Q Did you mean the final total represents the

12 paint-related or the non-paint-related?

13 A The paint-related. I'm sorry.

14 MR. HAUSFELD: Okay. Your Honor, we would

15 request that Exhibit 404 be displayed to the jury.

16 THE COURT: Are you moving it into

17 evidence?

18 MR. HAUSFELD: Yes, Your Honor.

19 THE COURT: Any objection?

20 MR. HUGHES: Foundation.

21 THE COURT: Objection's sustained.

22 You want to pursue this further,

23 Mr. Hausfeld?

24 MR. HAUSFELD: Yes.

25 THE COURT: Members the jury, I'm going to

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1 dismiss you for another break. Please wait in the
2 jury room. We'll either bring you back or give you a
3 time to be ready. You're excused.

4 THE CLERK: All rise.

5 (Jury excused.)

6 THE COURT: Let's have the witness step
7 down. I keep slipping at these two screens, and I
8 think it would be easier if I just paused here and
9 either found or received a hard copy of each of
10 these. Some of my laptop exhibits, I can enlarge
11 them. For some reason, I can't enlarge page 4 of
12 404, and I don't know that I have handy a copy of
13 392, although I think I've been shown one.

14 So let's just go off the record for a
15 moment.

16 MR. LEWIS: Your Honor, I could hand out
17 paper copies if you like.

18 THE COURT: Thank you.

19 MR. LEWIS: Thank you. 404 and 392, Your
20 Honor.

21 (A discussion was held off the record.)

22 THE COURT: All right. Mr. Hughes, what's
23 the basis for your objection?

24 MR. HUGHES: The basis for our objection is
25 that the witness has no personal knowledge that any

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1 of this money was spent on lead paint. And I think
2 what would be illuminated -- and if Your Honor would,
3 I would like you to consider his deposition testimony
4 where he repeatedly states that the only basis for
5 his testimony that money was spent on lead paint is
6 based on what somebody else told him. Somebody from
7 the Health Department told him, too.

8 Would you like to see that portion of
9 the deposition?

10 THE COURT: Yes. And specifically what
11 parts do you want me to look at?

12 MR. HUGHES: I've got them marked here for
13 you. It's from the May 22nd, 2007, deposition,
14 beginning on page 218, starting on line 7 and going
15 through to page 219. Actually, I'd say through
16 middle of 220, line 10 of 220, to capture everything.

17 THE COURT: All right. Why don't you give
18 that to the clerk.

19 Mr. Hausfeld?

20 MR. HAUSFELD: Yes, Your Honor.

21 THE COURT: Do you want to be heard on why
22 there is sufficient foundation to receive this
23 exhibit?

24 MR. HAUSFELD: The materials were prepared
25 under his supervision at his request. The request

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1 specifically went to the expenditures incurred by the
2 City in connection with lead-paint aspects of the
3 childhood lead prevention program. The information
4 was compiled by the people who ran the program.

5 And by the way, there may be a
6 definitional issue, but with regard to the childhood
7 lead prevention program, all of the efforts were
8 designed or related to paint issues. So everything
9 within the program dealt with paint under the
10 abatement program. When we did try to identify those
11 items which were not paint-based, he deducted them
12 from the calculation of what was incurred on an
13 annual basis. They are verified -- the expenditures
14 are verified in the initial instance, by normal City
15 procedures, and then were again subject to a second
16 verification by him after receipt of all the
17 information. There was no mathematical
18 extrapolation. It was a pure condition of the
19 expenditures incurred over time.

20 Dr. Foldy I think was asked, "Could
21 anyone make the calculation of how much in total was
22 expended by the City with regard to the lead-paint
23 aspects of the childhood lead prevention program?"
24 And he said, "Yes, anyone in the City's comptroller's
25 department," and that was what was done.

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1 reserve the right to recall the witness.

2 THE COURT: Is there other evidence
3 available today?

4 MR. HAUSFELD: Yes.

5 THE COURT: What would that be?

6 MR. HAUSFELD: Dr. Nichols.

7 THE COURT: Mr. Hughes, this exhibit or
8 these exhibits appear to go to the heart of the
9 defense -- or, rather, the plaintiff's damage case,
10 so I'm going to give them some chance to get these
11 things in.

12 Do you care whether we do this now or
13 proceed with something else?

14 MR. HUGHES: It doesn't matter to me. I
15 would only reiterate that, even if they were able to
16 lay a foundation this money was spent by the lead
17 program, I think the deposition testimony makes
18 crystal clear that Mr. Egan has no basis to testify
19 this money was spent on paint. He has no personal
20 knowledge of that.

21 There's a difference between saying
22 this is what the lead program spent and that the
23 money was spent on paint. And we have objections to
24 both, the foundation for both at this point. We
25 think that the foundation is insurmountable on the

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1 second point based on the deposition testimony.

2 MR. HAUSFELD: Your Honor, may I have a
3 moment?

4 THE COURT: I'm assuming that underlying
5 data here, the things that are not in evidence in the
6 trial, were all available to the defense. Is that
7 fair to assume, Mr. Hughes?

8 MR. HUGHES: Yes, Your Honor.

9 THE COURT: All right. Was there something
10 else, Mr. Hausfeld?

11 MR. HAUSFELD: May I have a moment, Your
12 Honor?

13 THE COURT: Go ahead.

14 MR. HAUSFELD: Your Honor, after
15 deliberation among counsel, if it's still available,
16 we would like to reserve our right with regard to
17 Mr. Egan and go on to the next witness at this time.

18 THE COURT: I think that would be the
19 wisest choice. So let's do that. Let's bring the
20 jurors down.

21 Is Dr. Nichols available and in Court?

22 MR. HAUSFELD: Yes.

23 (Discussion off the record.)

24 THE CLERK: All rise.

25 (Jury present.)

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1 THE CLERK: Please be seated.

2 THE COURT: Members of the jury, we are
3 going to interrupt the testimony of Mr. Egan and
4 proceed with a different witness.

5 Mr. Hausfeld, you may call the next
6 witness.

7 MR. HAUSFELD: Dr. Nichols.

8 THE COURT: Sir, would you please raise
9 your right hand?

10 DONALD NICHOLS, Ph.D., called as a witness
11 herein, having been first duly sworn on oath, was
12 examined and testified as follows:

13 THE COURT: Please be seated.

14 THE WITNESS: Thank you.

15 THE COURT: State and spell your name.

16 THE WITNESS: My name is Donald Nichols.

17 Nichols is N-I-C-H-O-L-S.

18 THE COURT: You may proceed.

19 DIRECT EXAMINATION

20 BY MR. HAUSFELD:

21 Q Good afternoon, Doctor.

22 A Good afternoon.

23 Q It is doctor, is it not?

24 A It is.

25 Q And you have a doctorate in what field?

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1 STATE OF WISCONSIN)

2) SS
3 MILWAUKEE COUNTY)

4 We, CONSTANCE L. BAUER, RPR/RMR/CRR, and
5 JULIE K. LYLE, RPR/RMR/CRR, Notaries Public in and for the
6 State of Wisconsin, do hereby certify that the foregoing
7 proceedings were recorded by us and reduced to writing
8 under our personal direction and that it is a true and
9 correct transcript of said proceedings had in said matter
10 to the best of our knowledge.

11 In witness whereof, we have hereunto set
12 our hand at Milwaukee, Wisconsin, on this 4th day of June,
13 2007.

14

15

16

17

18

19 JULIE K. LYLE

CONSTANCE L. BAUER

20

21

22

23

24

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1 STATE OF WISCONSIN : CIRCUIT COURT : MILWAUKEE COUNTY

2 -----

3 CITY OF MILWAUKEE,
4 a municipal corporation,

5 Plaintiff,

6 vs. Case No. 01-CV-3066

7 NL INDUSTRIES, INC.,
8 a foreign corporation,

9 Defendant.

10 Before the Honorable

11 JOHN FRANKE, Circuit Court Judge

12 Branch 25, presiding

13

14 Tuesday, June 5, 2007

15 VOLUME 11

16 9:40 a.m.

17 A P P E A R A N C E S:

18 RICHARD LEWIS, RICHARD SERPE, SARA SCHUBERT,
19 Attorneys at Law, appeared on behalf of the Plaintiff.

20 JAMES T. MURRAY, JR., DONALD SCOTT, ANDRE
21 PAUKA, JOHN HUGHES, Attorneys at Law, appeared on behalf
22 of the Defendant.

23

24

25

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1 Q Doctor, have you had occasion to sit on advisory
2 committees to the Centers for Disease Control in
3 their efforts to study childhood lead poisoning?

4 A I have served as a consultant to such committees,
5 yes, sir.

6 MR. LEWIS: Can we have Exhibit 170 for
7 the witness and the Court.

8 Q Can you identify the document on your screen,
9 please.

10 A Yes. This is the cover of the CDC report entitled
11 "Preventing Lead Poisoning in Young Children,"
12 which was released by the Centers for Disease
13 Control in 1991.

14 MR. LEWIS: Your Honor, this has been
15 admitted. May I publish to the jury?

16 THE COURT: Yes.

17 MR. SCOTT: I believe it was admitted in
18 part, Your Honor, as published.

19 THE COURT: Apparently it may be subject
20 to further publication. Are you able to quickly
21 identify what you want to present?

22 MR. LEWIS: Yes, I am, Your Honor. In
23 the listing of the consultants and advisors for the
24 CDC, can you turn to the third page and enlarge Dr.
25 Landrigan.

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1 Q Dr. Landrigan, can you identify your role serving
2 as a consultant to the Centers for Disease Control
3 when they published their 1991 report, "Preventing
4 Childhood Lead Poisoning"?

5 A Yes. That's my name listed in the book with my
6 first name misspelled.

7 Q And what was involved -- what kind of work did you
8 do as a consultant to the Centers for Disease
9 Control on this issue?

10 A Well, I went repeatedly -- I can't recall precisely
11 how many times, but repeatedly to Atlanta to where
12 CDC is headquartered to sit in on these meetings
13 and to participate in a very robust discussion
14 about how this nation can best prevent lead
15 poisoning in young children.

16 Q And what kinds of professionals were you meeting
17 with in Atlanta?

18 A It was mixed, but most of them were medical. Some
19 were public health officials. Some were people
20 representing various interests that were concerned
21 about lead poisoning.

22 Q And why did the Centers for Disease Control call
23 together scientists and doctors from around the
24 country to look at lead poisoning at this time in
25 the early 1990s?

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1 A Well, first of all, the reason the CDC called
2 people in at all is the fact that the Centers for
3 Disease Control is the prevention arm of the U.S.
4 Public Health Services. Other people do research,
5 but CDC is focused on prevention. And so it's
6 their duty from time to time to look at the major
7 diseases that confront people in this country and
8 decide what to do about it.

9 And in 1991, we were in an interesting
10 situation in terms of lead poisoning in children in
11 this country. On the one hand, we had -- we were
12 in the midst of a heroic effort to reduce
13 blood-lead levels in this country. We had started
14 in 1976 to get lead out of gasoline. By 1991 that
15 process of getting lead out of gasoline was well
16 underway to completion. Blood-lead levels had
17 fallen dramatically in children, largely as a
18 consequence of getting lead out of gasoline.

19 But at the same time -- at the same time
20 as we were making this great success in reducing
21 kids' exposure to lead, new scientific information
22 was coming in which made it plain to us that lead
23 could cause damage to children at quite low levels
24 of exposure, levels that we had previously thought
25 were safe for children.

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1 Q And as a result of the work of the scientists who
2 came together under the supervision of the CDC,
3 what was the final product of that effort?

4 A Well, the final product was this report. And --
5 and the central recommendation in that report is
6 that the so-called action level for dealing with
7 lead in children, the level of lead in blood in
8 children, which is defined as the action level,
9 this committee decided to lower that level from the
10 previous value, which was 25 micrograms per
11 deciliter of blood down to a level of 10 micrograms
12 per deciliter.

13 Q Just to be clear for the jury, 10 micrograms per --
14 of what per deciliter?

15 A 10 micrograms of lead in a deciliter, which is a
16 tenth of a liter of whole blood.

17 Q Doctor, are you board certified in any medical
18 disciplines?

19 A Well, I already mentioned pediatrics. And then
20 beyond pediatrics, in two others, preventive
21 medicine and occupational medicine.

22 Q I want to go through those briefly. Can you give
23 us a brief description of what pediatric medicine
24 means.

25 A Pediatrics is the study of disease in children and

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1 it encompasses diagnosis, treatment, prevention of
2 disease in children.

3 Q And you also mentioned occupational medicine. What
4 is that?

5 A Occupational medicine is the study of diseases of
6 the workplace, diseases of workers, and it also --
7 in more recent years, the specialty has expanded to
8 encompass diseases caused by toxic agents in the
9 environment. And so people who are certified in
10 occupational medicine are qualified to diagnose, to
11 treat, and prevent diseases that are caused by
12 toxic chemicals in the workplace and toxic
13 chemicals in the general community environment.

14 Q Doctor, in your capacity as a physician practicing
15 occupational medicine, were you called upon to
16 investigate health effects in the workers at the
17 World Trade Center after the destruction of those
18 buildings?

19 A Yes. We -- before 9/11, we already had the largest
20 occupational medicine program in New York City and,
21 in fact, in the whole -- in the whole northeast.
22 And so quite naturally in the aftermath of 9/11,
23 New York City officials, state and federal
24 officials, turned to us to -- to lead the effort to
25 diagnose and follow and treat the firefighters, the

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1 railings, on the windows, the gutters of the house.

2 Then the committee goes on in the fourth
3 line there to say that residences with exterior
4 lead paint are more than three times as likely to
5 have elevated levels of lead in the surrounding
6 soil. By that they mean levels that are greater
7 than 500 parts per million, as compared to
8 dwellings that have no exterior lead paint.

9 And finally, they speak to the issue of
10 deterioration. So you can have lead paint, and
11 then you can have deteriorated lead paint. They
12 say for buildings that have deteriorated exterior
13 lead paint, soil contamination is eight times more
14 common. So it's found in 48 percent of those
15 homes, as compared to residences that have no
16 exterior lead paint.

17 Q Thank you, Doctor. In terms of epidemiology, which
18 we talked about earlier, have epidemiologists in
19 this country studied the disease of childhood lead
20 poisoning?

21 A Oh, yes, extensively.

22 Q And have they identified the important risk factors
23 for that disease?

24 A Yes, they have.

25 Q And what are those risk factors?

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1 A Well, the single most important source of lead that
2 causes lead poisoning in children in this country
3 is -- is lead paint. And then as we just covered
4 in this -- in the last two minutes, this lead paint
5 contributes in turn to lead in house dust, lead in
6 soil. And the pathways by which lead gets from the
7 environment into children is basically 90 percent,
8 99 percent hand to mouth, which is to say that
9 little infants, one, two, three years old
10 predominantly, crawling in a home that has lead on
11 the floor, in the dust, or playing in the yard that
12 has lead in the soil, through the normal stuff that
13 little kids do, picking up a toy and putting it in
14 their mouth, picking up their finger and licking it
15 off, through those normal activities of early
16 childhood, little children transfer lead from the
17 dust -- transfer lead from the dust and from the
18 soil into their mouths, then they swallow it, it
19 enters their bloodstreams, it gets into their
20 bodies and causes a whole series of damages that we
21 refer to as lead poisoning.

22 Q Doctor, as a pediatrician, is it sound medical
23 advice to mothers and fathers to tell them to
24 eliminate hand-to-mouth behavior in infants and
25 toddlers?

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1 A Oh, it's a wonderful theory. Trouble is, it
2 doesn't work. Unless you tie a child's hands
3 behind them, there's no way that anybody who's ever
4 cared for children has been able to keep a child
5 from putting toys and fingers into their mouth.
6 It's just not possible.

7 Q Would you give that medical advice as a
8 pediatrician to moms and dads?

9 A No, never. Because it's impossible. I don't
10 believe in giving advice that people can't follow.

11 Q Would you blame moms and dads for cases of
12 childhood lead poisoning if the parents don't
13 eliminate that behavior?

14 A No.

15 MR. SCOTT: Objection, your Honor.

16 THE COURT: Ground?

17 MR. SCOTT: It's already been answered.

18 THE COURT: All right. The answer may
19 stand. Go ahead. Mr. Lewis.

20 MR. LEWIS: I'm sorry, did the jury hear
21 the answer?

22 THE WITNESS: The answer was no.

23 BY MR. LEWIS:

24 Q Have epidemiologists looked at age of housing as a
25 risk factor for childhood lead poisoning?

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1 thought you were going to say something.

2 THE COURT: I'm not sure what it is.

3 This is some sort of summary that someone prepared
4 based on 285?

5 MR. LEWIS: Yes, Your Honor.

6 MR. SCOTT: Could we have some foundation
7 as to who prepared it?

8 THE COURT: You want to voir dire the
9 witness, or let's have Mr. Lewis do it. Why don't
10 we have Mr. Lewis do it.

11 BY MR. LEWIS:

12 Q Doctor, were you able to review data on the level
13 of childhood lead poisoning for the year 1996 in
14 the city of Milwaukee and in the combined target
15 areas?

16 A Yes, I was.

17 Q And is that the same data that you reviewed for the
18 last exhibit, except then it was for the year 2005?

19 A Yes. Same data set. Different years.

20 MR. LEWIS: May I publish 2002 to the
21 jury, Your Honor?

22 MR. SCOTT: No objection.

23 THE COURT: Go ahead.

24 BY MR. LEWIS:

25 Q And what did you find when you looked at the 1996

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1 data?

2 A Well, in 1996, it's clear that the rates of
3 elevated blood-lead levels are a great deal higher
4 than they were in 2005. So city-wide, as you can
5 see here, it's about 35 percent in 1996, and as we
6 saw a couple of minutes ago, by a decade -- about
7 11 years later, it had fallen from 35 percent to
8 about 8 percent in the whole city. And then
9 looking at the target areas, you can see it's about
10 44, 45 percent in 1996. And you'll recall from the
11 previous graph, that by 2005, it had fallen from
12 this very high rate down to a rate of about
13 13 percent.

14 So I would summarize this as saying it's
15 a good news/bad news drawing. The good news is
16 that lead levels have certainly come down in all
17 the children in Milwaukee, city-wide and in the
18 target areas both. But the bad news is that the
19 gap has persisted. Ten years ago, 11 years ago,
20 and last year, in both instances, the kids who
21 lived in the target areas had substantially higher
22 levels of lead than children in the rest of the
23 city.

24 Q Dr. Landrigan, if you can put yourself back into
25 the time period 1996, if you were evaluating this

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1 data then from the city of Milwaukee, from a public
2 health point of view how would you characterize
3 this level of childhood lead poisoning?

4 A Well, I'd call it an epidemic. There is no other
5 word. The correct diagnosis of a disease that
6 affects this many children in a population is
7 epidemic. There's no other word that would be
8 appropriate.

9 MR. LEWIS: Could you put Exhibit 365 on
10 the screen, please. Your Honor, I apologize. I
11 should have asked earlier that 181 and 197, as
12 published, be admitted.

13 MR. SCOTT: 181 and 197? No objection as
14 published, Your Honor.

15 THE COURT: That's 181 and 197?

16 MR. LEWIS: Correct, Your Honor.

17 THE COURT: Received.

18 MR. LEWIS: Can you make this a little
19 larger?

20 Q Doctor, how do epidemiologists go from spreadsheets
21 of data regarding childhood lead poisoning to maps?

22 A Well, basically they do it one child at a time or
23 one house at a time, and from the spreadsheet, they
24 identify the address at which a child lives, and in
25 the old days, we used to put a pin in the map

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1 corresponding to that address. Today you would use
2 a computer graphic. But in any case, you build up
3 a map that shows the distribution of cases of lead
4 poisoning in a community.

5 Q Is this a standard tool of epidemiologists?

6 A Oh, yes. It was -- The father of epidemiology was
7 a man named John Snow, who studied cholera in
8 London in the 1850s, and he did this. So this is a
9 technique that's been around for 150, 160 years.

10 Q Now, this is the 1996 data for the City of
11 Milwaukee, with the target areas outlined in red.
12 I apologize on this issue. We're creating a new
13 document, which will be here shortly, to deal with
14 the color issue.

15 But as an epidemiologist studying
16 childhood lead poisoning, what can one learn about
17 what the conditions were like in 1996 when this
18 data was generated?

19 A Well, there's several conclusions you can draw from
20 this map. The first -- the first one is that
21 there's just a lot of cases, just a great number of
22 dots.

23 The second is that they're clearly
24 concentrated in these two areas of the city, which
25 I know are called the north and south target areas,

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1 is yes. I don't know why he'd want to leave one of
2 the great cities of the Northeast, but apparently
3 he does. Yes, we'll do our best.

4 MR. LEWIS: Thank you, Your Honor.

5 THE COURT: But I'd ask you to try to
6 have that all done by five, unless there's some
7 very good reason why it's going to take more than
8 that amount of time to finish this.

9 MR. SCOTT: What time did you ask us to
10 come back, Judge?

11 THE COURT: 1:20.

12 (A lunch recess was taken.)

13 THE CLERK: All rise for the jury.

14 (Jury present.)

15 THE CLERK: You can be seated. Court is
16 back in session.

17 THE COURT: Good afternoon and welcome
18 back to all. We will continue with testimony.
19 We're still on direct. Mr. -- this is testimony
20 from Dr. Landrigan.

21 Mr. Lewis, you may proceed.

22 MR. LEWIS: Thank you, Your Honor.

23 Q Good afternoon, Doctor.

24 A Mr. Lewis.

25 MR. LEWIS: May we have Exhibit 171 on

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1 the screen, please. This was admitted. And can
2 you enlarge findings two through five.

3 Q Doctor, do you recognize this as the findings from
4 the Title X law "Residential Lead-Based Paint
5 Hazard Reduction Act of 1992"?

6 A Yes, I do. These are -- these are some more of the
7 findings at the beginning of that piece of
8 legislation.

9 Q We covered the first finding this morning. The
10 second finding reads: "At low levels, lead
11 poisoning in children causes intelligence quotient
12 deficiencies, reading and learning disabilities,
13 impaired hearing, reduced attention span, and
14 hyperactivity and behavioral -- and behavior
15 problems."

16 Can you explain those findings, please.

17 A Yes. All of those -- all of those findings are
18 caused by the brain damage that is produced in
19 children --

20 Q Doctor, could you put the microphone a little --
21 thank you.

22 A Sorry. All of those findings are caused by the
23 brain damage that is produced in children by lead
24 which they have swallowed and taken into their
25 bodies. We know from a whole series of studies and

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1 were reaffirmed on the CDC consensus document, the
2 National Academy of Sciences consensus document,
3 the American Academy of Pediatrics consensus
4 document that lead, even at quite low levels of
5 exposure, has the capacity to cause brain injury in
6 children. And the way that brain injury shows up
7 when doctors and psychologists evaluate a child is
8 in these ways here: Diminished intelligence, which
9 is measured as decreased IQ; problems with reading
10 and learning and math; hearing problems; shortening
11 of their attention span so they can't focus as well
12 as other kids on a task; hyperactivity, which goes
13 along with shortening of the attention span; and
14 all kinds of behavior problems, including the one
15 that's been commented on over the years by some of
16 the best pediatric writers is that children who
17 have been exposed to lead tend to be hyperactive
18 and aggressive. That's sort of the -- it's not
19 seen by all the writers, but it's seen rather
20 commonly.

21 Q Doctor, the third finding from Title X regards
22 housing. It says that "Pre-1980 American housing
23 stock contains more than 3 million tons of lead in
24 the form of lead-based paint, with the vast
25 majority of homes built before 1950 containing

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1 substantial amounts of lead-based paint."

2 Doctor, what types of locations or cities
3 in America have a predominance of homes built
4 before 1950?

5 A Well, it's what I said before lunch. It's the
6 older houses concentrated in the Northeast and the
7 upper Midwest, concentrated in the inner cities.

8 Q Doctor, the fourth finding reads: "The ingestion
9 of household dust containing lead from
10 deteriorating or abraded lead-based paint is the
11 most common cause of lead poisoning in children."

12 Doctor, can you explain how the words
13 "deteriorating or abraded" are used in the context
14 of childhood lead poisoning.

15 A Okay. Let me -- there's a couple of things here.
16 The first point in this Bullet No. 4 is the
17 statement that it's ingestion. Ingestion, simply
18 means the swallowing of lead-based paint by
19 children, is the principal route by which the lead
20 gets into the children's body. They certainly
21 reaffirm that paint is the main source.

22 And then those two words, "deteriorating
23 and abraded," they refer to two different things.
24 So deteriorating lead paint is what it is. It's
25 paint that's in bad repair, it's chipping, it's

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1 statistics for children suffering from childhood

2 lead poisoning in Milwaukee?

3 A Yes, I have.

4 Q And the third line recounts that from 1993 to 2007,
5 there were 850 chelation events here in Milwaukee.

6 Do you see that?

7 A Yes, I do.

8 Q And what is the blood lead range where children are
9 chelated over this time period?

10 MR. SCOTT: Your Honor, I will object.

11 There's no foundation for Dr. Landrigan to talk
12 about the blood level at which children were
13 chelated in Milwaukee on this chart.

14 THE COURT: Sustained.

15 BY MR. LEWIS:

16 Q In general, Dr. Landrigan, based on recommendations
17 from medical organizations, at what level of blood
18 lead poisoning are children chelated?

19 A Generally children are not chelated until the blood
20 level has risen to above 40 or 45 micrograms per
21 deciliter.

22 Q Now, as to those children who experience those
23 chelation events, would you expect that they would
24 recover from lead poisoning and be fine today?

25 A They will recover from the acute lead poisoning

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1 because the chelating agent will rapidly remove the
2 lead from their bodies, but they will not be fine
3 today. They may appear fine from across the room,
4 but as soon as they go in for any kind of testing,
5 IQ testing, behavioral testing, it will become
6 evident very quickly that they have problems, the
7 kind of problems that we've just been talking
8 about, reduced intelligence, shortening of
9 attention span, hyperactivity.

10 Q Doctor, as to the 19,016 cases of elevated blood
11 lead above ten reported in Milwaukee from 1997 to
12 2006, would you expect that the children in that
13 group, in that population, would have fully
14 recovered from any effects of lead exposure?

15 A No, they won't fully recover. Now, to be quite
16 fair, it's important to say that the children who
17 have a blood level of 40 are going to be worse off
18 than the children who had a blood level of 20. And
19 those children in turn are going to be worse off
20 than the children who had a level of ten. It's
21 related to the dose. That makes -- that's
22 perfectly logical.

23 But even the children whose blood level
24 is above ten are going to be left with residual
25 damage in the form of reduced IQ and problems in

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1 behavior.

2 Q Dr. Landrigan, as to the children identified in
3 this summary, could their cases of childhood lead
4 poisoning have been avoided by eating lots of
5 vegetables, lots of spinach?

6 A No. Unfortunately, no.

7 Q Could their cases of lead poisoning be cured by
8 stop -- cessation of eating junk food or food from
9 McDonald's?

10 A No, no.

11 Q Why not?

12 A Because, unfortunately, the lead is already in
13 those children, the lead has already damaged their
14 brains. And whatever they do subsequently may have
15 effects on their health, it apparently will, but
16 it's not going to reverse the lead poisoning.

17 Q Could these 19,000 cases have been avoided by
18 housecleaning, such as a bucket and a mop?

19 A No. You may have been able to reduce the numbers
20 slightly, but you can't prevent -- you can't fully
21 prevent lead poisoning with a bucket and a mop.

22 Q What about washing hands? If these 19,000 kids had
23 washed their hands more, would that have prevented
24 childhood lead poisoning in Milwaukee?

25 A Not -- not in the great majority of them, because

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1 ultimately the source of the lead was the lead in
2 the dust and the soil that came from the paint that
3 the children transferred into their -- into their
4 mouths off their little fingers. And it's simply
5 not possible to follow around behind a child and
6 wash his hands every minute. It's just not
7 possible.

8 Q Doctor, earlier in your testimony, you talked about
9 the Centers for Disease Control action level of
10 10 micrograms of lead in a child's blood, which was
11 set in 1991.

12 A Yes.

13 Q Has the recent American Academy of Pediatrics
14 report indicated the extent to which children are
15 at risk below 10 micrograms per deciliter?

16 A Yes, it has.

17 MR. LEWIS: Can we look at Exhibit 197,
18 please.

19 And can we go to the abstract on the
20 first page. You can enlarge that. It's in bold
21 print. And can you enlarge the abstract at the
22 beginning.

23 Q Doctor, the second sentence of this paragraph
24 reads: "Evidence continues to accrue that commonly
25 encountered blood lead concentrations, even those

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1 the break.

2 MR. LEWIS: I'm sorry, Mr. Scott, one
3 quick housekeeping. May I move for the admission
4 of 197, 2000, 2001, and 2002, Your Honor. You had
5 reserved earlier in the direct.

6 MR. SCOTT: 197 is the American Academy.
7 No objection as published. I don't remember what
8 the other ones were.

9 MR. LEWIS: They were the three charts,
10 one comparing Milwaukee to the United States --

11 MR. SCOTT: Can I just take --

12 THE COURT: What were the numbers again?

13 MR. LEWIS: 2000, 2001, and 2002.

14 MR. SCOTT: I haven't had a chance to
15 compare those with the data yet, Your Honor. We
16 can take that up later on.

17 THE COURT: All right.

18 CROSS EXAMINATION

19 BY MR. SCOTT:

20 Q Dr. Landrigan, can you hear me okay from here?

21 A Mr. Scott, I can.

22 Q Thank you very much. Good to see you again.

23 A Likewise.

24 Q You've described for us at the beginning your
25 academic credentials. You are being paid as an

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1 expert witness to testify in this case; are you
2 not?

3 A Yes, I am.

4 Q And you are charging at the rate of 4- to \$500 an
5 hour to do so?

6 A That is correct.

7 Q Let's begin in the 1960s with your residency and
8 try to get up to current date as quickly as we can
9 in the next couple of hours. First, we do have to
10 go back to 1912. You mentioned an article by Dr.
11 McKhann in Boston. Do you remember what the lead
12 level was of the child or children who were
13 diagnosed at that time?

14 A No, I don't remember the precise level, but
15 probably would have been -- and of course, you have
16 to understand that the techniques for measuring
17 blood lead were very imprecise at that time
18 compared to today. But it would almost certainly
19 have been 80 or 100 micrograms per deciliter.

20 Q Do you remember what was the level recognized as
21 being toxic in 1912?

22 A At that time, there was no definition that I'm
23 aware of.

24 Q Then let's go to your residency, your pediatric
25 residency. You graduated in 1967, and in that

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1 period of time, you found, did you not, that the
2 main concern then for children in lead paint was
3 not dust, but whether or not they had pica?

4 A That's correct.

5 Q And by pica, you mean the idea that children would
6 actually eat chips of paint.

7 A That's right.

8 Q And that, in fact, was the prevailing opinion of
9 the experts in the 1950s and '60s, that pica was
10 the sole source, the sole route for children's
11 exposure to lead paint.

12 A That's right. Concepts of dust started to come out
13 in -- as best as I can recall, in the 1970s.

14 Q 1970s. Now, you have been writing articles on lead
15 and lead poisoning since the 1970s; haven't you?

16 A Yes, sir.

17 Q And just to place us in time, I'd like to show on
18 your screen, a 1981 article that you wrote with
19 Dr. Needleman.

20 A Yes.

21 Q And is Dr. Needleman a friend of yours?

22 A Oh, yes.

23 Q And a recognized authority in the field?

24 A He is both.

25 Q And what I want to ask -- so we can see where

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1 things stood in 1981. That's why I have you
2 looking at this article, how things stood in 1981.

3 Your Honor, may I display this and a
4 portion at page 293?

5 MR. LEWIS: Your Honor, no exhibit
6 number. I don't know that I've seen this.

7 MR. SCOTT: I'm sorry, this is 883.

8 MR. LEWIS: And has that been provided?
9 No objection. No objection, Your Honor.

10 MR. SCOTT: We believe it has been, yes.

11 MR. LEWIS: No objection.

12 THE COURT: You want to display part of
13 Exhibit 883?

14 MR. SCOTT: Yes.

15 THE COURT: Okay. Go ahead. Did you say
16 you wanted to compare it to some other exhibit?

17 MR. SCOTT: No.

18 THE COURT: Okay. Go ahead.

19 BY MR. SCOTT:

20 Q I want to see where the knowledge stood in 1981.
21 And, Doctor, if we could just look at this portion
22 of what you wrote in 1981 and see if this was the
23 state of knowledge. "Lead has been used by man
24 since antiquity. Knowledge of its toxicity at high
25 dose is old and widely acknowledged. Only recently

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1 has attention been directed to the question of
2 whether low doses of lead also produce adverse
3 health effects."

4 Did you write that as of 1981?

5 A I did write that.

6 Q And was that the state of the knowledge at that
7 time?

8 A Yes. This fits precisely into that progression of
9 knowledge that I was describing in the last minutes
10 of my direct examination, that each decade we've
11 learned more about the toxicity of lead at lower
12 levels. And this question of whether low doses of
13 lead produce adverse effects first came out in the
14 1940s in the work of Dr. Byers and Dr. Lord in
15 Boston and then was reemphasized in the '70s by the
16 work I did in El Paso in the mid-'70s and the work
17 that Dr. Herb Needleman did in Boston in the late
18 '70s. And so this report, written in 1981, was
19 recapping that history.

20 Q Would you agree, sir, that as late as 1969, 1970, a
21 lead level as high as 60 micrograms was considered
22 safe in children?

23 A It was, yes.

24 Q Then into the mid-1970s, a lead level of as high as
25 40 was considered safe in children.

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1 A Yes, that is correct.

2 Q And it was in that time period, in the middle
3 1970 -- middle 1970s, that you did a study
4 involving children exposed to smelter emissions
5 down in El Paso, Texas.

6 A That's correct.

7 Q And what you found then was that -- at the time you
8 wrote in 1975 that the question whether there were
9 adverse health effects in children in the range 40,
10 as high as 80 micrograms was still an unresolved
11 medical question.

12 A That's not quite true. What the -- we certainly
13 understood that in the range between 40 and 80,
14 some children could develop symptoms of lead
15 poisoning. So in the case of those children, there
16 was no question. The question that was unresolved
17 at that time was whether lead could cause damage in
18 children who had no obvious symptoms.

19 Q Would you agree, Doctor, that as of the mid-1970s,
20 it was an unresolved issue whether blood-lead
21 levels of 40 to 80 micrograms adversely affected
22 the nervous system?

23 A No, that's -- that's too simplistic. It was clear
24 in some children who had symptoms at blood levels
25 of 70 or 75, that in those children, lead had

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1 advertently affected their nervous systems. As I
2 say, the unresolved question concerned the children
3 who had no obvious symptoms. And the question
4 precisely was, did lead cause damage in children
5 who had no obvious symptoms.

6 Q What was unresolved was in children who had no
7 symptoms, whether they were harmed at all at the
8 level of 40 to 80 micrograms.

9 A That was unresolved at that time.

10 Q That was unresolved.

11 A Yeah.

12 Q Now, we've just been talking about what was
13 considered safe or not in the 1970s. Now let's
14 look at what the blood-lead levels of children were
15 at that time. You talked in direct examination
16 about the national survey, and the jury has seen
17 this before, N-H-A-N-E-S, NHANES.

18 A Yes.

19 Q And the first survey of that kind came out -- well,
20 it covered the years 1976 to 1980.

21 A That's right. It was actually the second survey
22 but the first one that included lead.

23 Q First one to include lead. And this was called the
24 National Health and Nutrition Examination Survey?

25 A Correct.

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1 Q You have seen CDC, Centers for Disease Control,
2 publications that summarize the findings on
3 children's blood leads based upon the NHANES
4 surveys.

5 A Yes, I have.

6 Q This might be the most convenient: I'm going to
7 show you from the CDC's current website such a
8 summary.

9 May I approach the witness, Your Honor?

10 THE COURT: Yes.

11 THE WITNESS: Thanks.

12 BY MR. SCOTT:

13 Q Dr. Landrigan, I have two tabs on that paper, that
14 website, which I believe you're familiar with. If
15 you just look at those and confirm that this is the
16 data you know about that reflects the historical
17 measurements of children's blood leads in the
18 NHANES surveys.

19 A Yes, these are.

20 Q I would like to publish a -- from Table 1 --

21 May I publish, Your Honor?

22 THE COURT: Unless there's objection, you
23 may.

24 MR. LEWIS: None, Your Honor.

25

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1 BY MR. SCOTT:

2 Q Dr. Landrigan, let's, before we look at the
3 details, just see what we're looking at here. This
4 data is the prevalence of blood-lead levels of 10
5 micrograms or greater; is that correct?

6 A That's correct, national data.

7 Q National data from NHANES. And that prevalence
8 means the percentage of children tested who have
9 that level compared to all children who were
10 tested.

11 A That is correct.

12 Q And do you see the result in the first such survey
13 that you and I spoke about a moment ago?

14 A Yes. At that time 88.2 percent of children in the
15 entire United States had a blood-lead level greater
16 than 10. Which was 13.5 million children.

17 Q I couldn't hear you, Doctor.

18 A Which was 13.5 million children.

19 Q And then there were a number of changes, including
20 the phase-out of leaded gasoline from virtually all
21 uses in this country.

22 A Correct.

23 Q And thereafter then, these numbers came down, as
24 shown here, in 1988 to '91, to a level of
25 8.6 percent; then for 1991 to '94, to a level of

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- 1 4.4 percent; from 1999 to 2000, to a level of
2 2.2 percent. Is that all correct?
- 3 A All that's correct, yes.
- 4 Q And still declining?
- 5 A And still to this day.
- 6 Q Now, the decline, particularly the first drop, from
7 the 88 percent down, was the result of the removal
8 of lead from gasoline.
- 9 A In large measure. There may have been a few other
10 factors, like taking lead out of tin cans and the
11 abatement of a certain number of houses. But I
12 think most of us agree that the removal of lead
13 from gasoline was the principal driver.
- 14 Q And you, in fact, have seen graphs where the CDC
15 charts out the amount of lead from gasoline being
16 used and the children's blood leads. You've seen
17 those; haven't you?
- 18 A Yes, and the two come down in very close parallel,
19 yeah.
- 20 Q Okay. And then this is shown in a graph at
21 Figure 1 of the same document, which is
22 Exhibit 875. The previous one was Exhibit 874.
23 And this displays the same information graphically
24 rather than numerically; does it not, sir?
- 25 A Yes, it does indeed. We like to refer to it as one

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1 of the great triumphs of American public health in
2 the past century.

3 MR. SCOTT: We offer Exhibits 874 and
4 875, which are excerpted from the CDC website as
5 displayed.

6 MR. LEWIS: No objection, Your Honor.

7 THE COURT: Received.

8 BY MR. SCOTT:

9 Q Now let's talk about the sources of exposure for
10 children. And we'll stay with the question of
11 gasoline for a few minutes. You would agree that
12 lead from gasoline was being emitted into the air,
13 pumped into the air for decades beginning back to
14 the 1920's.

15 A From the late '20s, early 1930s, until the
16 mid-1970s, that's right.

17 Q And that lead in the air initially would then fall
18 to the ground.

19 A That's correct.

20 Q And where it entered yards and dirt and grass, it
21 stayed there.

22 A For a time at least, yes.

23 Q Do you agree, Dr. Landrigan, that lead from leaded
24 gasoline has caused more exposure for people to
25 lead than any other source of lead worldwide?

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1 next sentence.

2 Q "At the same time the available evidence does not
3 refute" -- that means disprove; right?

4 A Yes, sir.

5 Q -- "refute the interpretation that these
6 associations are at least in part causal."

7 Is that the additional sentence you
8 wanted?

9 A Yes, sir, correct. Thank you.

10 Q So you would agree that the available evidence
11 neither proves nor disproves whether those
12 associations are causal, cause and effect, at this
13 time.

14 A Yeah, I think -- I think what the -- the work group
15 is obviously struggling here with new science,
16 which is what scientists do all the time. It took
17 many years for it to become accepted fact that
18 blood-lead levels between 10 and 20 micrograms
19 caused brain damage to children. Now we have
20 several new studies looking at blood-lead levels
21 below 10. Also, if you go up just above Question
22 2, there's -- you can see how they're struggling
23 with this, because right above Question 2, it says,
24 "Though the number of studies providing direct
25 evidence of associations is limited, and most are

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1 cross-sectional, they provide supporting evidence
2 of an association in the context of the much larger
3 number of studies that relate slightly higher
4 levels of lead in blood to impairments in
5 children's health."

6 I think a lot of times what happens in
7 science is there's this sort of a tipping point
8 phenomenon. Evidence accumulates, more
9 accumulates, and finally the weight of opinion
10 swings over, it tips, and I think right now we're
11 poised at the point and -- and this very
12 prestigious group says they want a little more
13 evidence before they conclude that blood levels
14 below 10 cause brain damage in kids. They
15 recognize the association. They're not quite ready
16 to sign off on causality. I'm okay with that.

17 Q Do you agree, Dr. Landrigan, that, in fact, the
18 whole science of lead poisoning has been changing
19 dramatically throughout the 20th century?

20 A Yes. Yes --

21 Q And right up to the present day.

22 A Up to the present day --

23 Q Nothing further.

24 A -- as new tools have come along, we've made no
25 advances.

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1 MR. SCOTT: I have nothing further.

2 THE COURT: Mr. Lewis, any redirect?

3 MR. LEWIS: Yes, Your Honor. Mr. Scott,
4 can we stay with that exhibit that you had up
5 there, the 2005 CDC.

6 MR. SCOTT: I think what happens here is
7 I'm trapped here by my cords.

8 MR. LEWIS: Can I have that, guys? Can I
9 have page 1 of that document?

10 MR. SCOTT: I can go back to my seat or I
11 can stand here. Whatever you want me to do.

12 MR. LEWIS: This is going to be short, so
13 if I can impose on you. If we can go to page 1.

14 MR. SCOTT: With the Court's permission.

15 THE COURT: Sure.

16 MR. LEWIS: That's the cover page. And
17 I'd like to go to page 1 of the 2005 CDC report.
18 I'd like to enlarge the bottom paragraph on that
19 page. Thank you. That's all I need. Thank you
20 very much.

21 THE COURT: We went to the cover page a
22 moment ago.

23 MR. LEWIS: Yes, that was --

24 THE COURT: And now we've moved to page 1
25 of some document within that?

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1 STATE OF WISCONSIN)
2) SS
3 MILWAUKEE COUNTY)
4

5 I, JULIE A. POENITSCH, RPR/RMR/CRR, Notary
6 Public in and for the State of Wisconsin, do hereby
7 certify that the foregoing proceedings were recorded by
8 me and reduced to writing under my personal direction
9 and that it is a true and correct transcript of said
10 proceedings had in said matter to the best of my
11 knowledge.

12 In witness whereof, I have hereunto set
13 my hand at Milwaukee, Wisconsin, on this 5th day of
14 June, 2007.
15
16
17
18

19 _____
20 JULIE A. POENITSCH
21
22
23
24
25

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1 STATE OF WISCONSIN : CIRCUIT COURT : MILWAUKEE COUNTY

2 -----
3 CITY OF MILWAUKEE,
4 a municipal corporation,

5 Plaintiff,

6 vs.

Case No. 01-CV-3066

7 NL INDUSTRIES, INC.,
8 a foreign corporation,

9 Defendant.

10 Before the Honorable

11 JOHN FRANKE, Circuit Court Judge

12 Branch 25, presiding

13
14 Wednesday, June 6, 2007

15 VOLUME 12

16 9:20 a.m.

17 A P P E A R A N C E S:

18 RICHARD LEWIS, RICHARD SERPE, CHRIS RIORDAN,
19 KATE KONOPKA, Attorneys at Law, and SARA SCHUBERT,
20 appeared on behalf of the Plaintiff.

21 JAMES T. MURRAY, JR., DONALD SCOTT, ANDRE PAUKA,
22 JOHN HUGHES, Attorneys at Law, appeared on behalf of the
23 Defendant.
24
25

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1 A I was responsible for developing and overseeing all
2 of the activities of the Childhood Lead Poisoning
3 Prevention Program. That included testing of young
4 children, managing our data, assuring laboratory
5 services, assuring public health nurse case
6 management, community education, and environmental
7 inspection and abatement.

8 Q And when you say abatement, what kind of abatement
9 are you talking about?

10 A Our focus was controlling lead-based paint.

11 Q In 2006, could you estimate approximately how many
12 other employees were on your staff when you were
13 managing the Lead Poisoning Prevention Program.

14 A Yeah. The number of staff varied over the years. In
15 general, it ranged from 35 to 45, with probably an
16 average of 40 in the later years.

17 Q And in the earlier years, in the '90s, what would the
18 average have been then?

19 A When I began as the manager of the program, we had
20 seven employees and steadily built the program
21 through various projects. But we held pretty steady
22 over 30 employees for the last ten years.

23 Q And as a general matter, what kind of work were those
24 30 to 40 employees involved in at the program?

25 A We had a very diverse staff. We employed nurses,

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1 public health assistants, or community outreach
2 workers. We employed environmental inspectors that
3 were trained and certified by the State of Wisconsin
4 as risk assessors, and we had various managers and
5 individuals that even managed our surveillance or our
6 database.

7 Q And was there a common aspect as to all the
8 employees' work at the Childhood Lead Poisoning
9 Prevention Program?

10 A Yes. All of these disciplines and staff worked
11 together to focus on preventing childhood lead
12 poisoning and ultimately eliminating childhood lead
13 poisoning through controlling lead-based paint in the
14 City of Milwaukee.

15 Q When you were manager of the program, did you have
16 occasion to work with other managers from other
17 cities and other doctors and scientists involved with
18 childhood lead poisoning?

19 A Absolutely. Starting in the late -- in 1980s, early
20 1990s, there was a huge national movement toward an
21 increased focus toward childhood lead poisoning
22 prevention, and I worked with various individuals
23 from other programs, as well as national agencies.

24 Q And were you asked to sit on any national advisory
25 committees who were dealing with childhood lead

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1 poisoning?

2 A I did. I served on the CDC advisory committee on
3 childhood lead poisoning for two years, and as a part
4 of those responsibilities, I chaired the primary
5 prevention work group, we published a document, and
6 served as a member of the screening subcommittee work
7 group.

8 Q You said CDC. What does that stand for?

9 A That's the Centers for Disease Control.

10 Q And where is the Centers for Disease Control?

11 A They're located in Atlanta, Georgia.

12 Q And is that a national governmental organization?

13 A Yes. They're part of the Department of Health &
14 Human Services, and they are the health or medical
15 arm dedicated to the prevention of childhood lead
16 poisoning, among their various other duties.

17 Q And what was the nature of the training and skills of
18 the other persons who sat with you on that advisory
19 committee at CDC?

20 A The vast majority of the other individuals on the
21 committees were either M.D.s or Ph.D.s, and they were
22 experts in things like medical management or
23 toxicology or the environmental area of the program.

24 Q And was all the work of the CDC advisory committee
25 concerning childhood lead poisoning?

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1 Q In your position as Childhood Lead Poisoning
2 Prevention Manager from 1992 to 2006, did you manage
3 projects?

4 A Yes, I managed multiple projects.

5 Q And what -- what kind of projects did you manage?

6 A Uh-huh. We managed projects that focused on the
7 testing of children, we managed projects focused on
8 the inspection and identification of lead paint
9 hazards and the control or abatement of those lead
10 hazards. We had many, many programs that provided
11 education and outreach to the community and working
12 with communities most affected by the problem.

13 Q Could we have Exhibit 2009, please.

14 Ms. Murphy, that exhibit will appear
15 on your screen and on the Court's screen first, and
16 I, just as a matter of ground rules, I'll ask you to
17 identify exhibits, and then some of those exhibits
18 will then appear on the large screen. Do you have
19 2009 before you?

20 A Yes.

21 Q Can you tell me what it is?

22 A This is a list of titles of the various projects
23 under my management from 1992 through 2006 as a part
24 of the Childhood Lead Poisoning Prevention Program.

25 MR. LEWIS: Your Honor, I ask that 2009 be

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1 admitted and published to the jury.

2 MR. MURRAY: No objection, Your Honor.

3 THE COURT: Received.

4 BY MR. LEWIS:

5 Q Ms. Murphy, the first 16 items on this list describe
6 projects. What kind of projects were these?

7 A Uh-huh. These projects focused on testing of young
8 children, focused on assuring services to
9 lead-poisoned children and their families to prevent
10 further exposure. These projects focused on
11 targeting lead paint in high-risk properties before a
12 child was poisoned, and some of them focused on
13 community outreach education, and working with
14 communities most affected.

15 Q Let's look at the first one, the evaluation grant,
16 EPA. What does that mean?

17 A This project took place in the mid-1990s. It was a
18 collaboration with the Environmental Protection
19 Agency. It funded our data manager, which was really
20 the cornerstone of our surveillance and data
21 collection activities, as well as the linchpin to
22 assure that services were provided. In collaboration
23 with the EPA, this project supported evaluation on
24 the effectiveness of our various interventions so we
25 could continue to improve what we were doing.

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1 Q When you talk about interventions, what does that
2 mean?

3 A Intervention is a service or an activity aimed at
4 either providing services to lead-poisoned children
5 and their families, children we know are poisoned, or
6 in preventing poisoning from happening in the first
7 place by concentrating and focusing on lead paint.

8 Q Let's look at the second one. It's called The
9 Environmental Justice Grant, EPA. What does that
10 mean?

11 A Well, the Environmental Justice Grant, again, took
12 place in the mid-1990s. This was our first attempt.
13 It was basically a demonstration project for a
14 two-year period to try to control lead paint hazards
15 before a child was poisoned, rather than identifying
16 the child and providing follow-up activities once the
17 exposure was identified and some damage had been
18 done. We outposted staff to one of our highest risk
19 communities in Milwaukee and tried to target the
20 homes before a child was poisoned.

21 Q Let's go to the fifth item on Exhibit 2009 --

22 A Yes.

23 Q -- called Lead Poisoning Prevention, CDC.

24 A Yes.

25 Q What does that mean?

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1 A This project, we received funding initially for this
2 project in 1992, and we still are receiving this.
3 This project is still going on at the Health
4 Department.

5 This is annual funding from the
6 Centers for Disease Control that the City of
7 Milwaukee receives on a competitive basis every year,
8 and the services are primarily focused, for this
9 project, on the health aspects of childhood lead
10 poisoning prevention, screening, education, public
11 health nurse case management.

12 Q And the next project is called Lead Hazard Reduction,
13 HUD 8.

14 A Yes.

15 Q What does that mean?

16 A This was one of our larger HUD grants focused on the
17 abatement or the control of lead paint hazards in
18 housing located in our target areas.

19 Q And going down a ways to the 14th item, it says,
20 Lead Paint Hazard Control, HUD 11.

21 A Yes.

22 Q How is that different from what you just described?

23 A Well, it was a subsequent grant from HUD. The HUD
24 grants that we received or the HUD projects were time
25 limited, and then we were able to apply for

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1 additional funds toward the ends of like when HUD 8
2 was near its end, we applied and received money under
3 HUD 11. So this represents rounds or years in which
4 HUD provided funding.

5 Q As to these 16 programs, the first 16 lines of
6 Exhibit 2009, was there a common aspect, a common
7 goal of these projects?

8 A Yes. The entire focus of all of these projects
9 collectively was to prevent lead poisoning in
10 children through focusing on lead paint.

11 Q And do you know that from personal experience from
12 1992 to 2006?

13 A I do.

14 Q Under these projects listed in Exhibit 2009, did the
15 City make expenditures?

16 A Yes, we did.

17 Q And by expenditures, I mean did the City spend money?

18 A Yes, under each one of these projects.

19 Q And what kind of -- what were the -- what were the
20 basic items that the City was spending money on under
21 these projects?

22 A The basic categories was staff or personnel that
23 really represented the various disciplines we
24 discussed earlier. We funded abatement, we funded
25 community education, supplies, materials, training.

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1 THE COURT: Ma'am, slow down just a little
2 bit.

3 THE WITNESS: Okay.

4 THE COURT: After abatement, can you repeat
5 the list?

6 THE WITNESS: Sure. Supplies, materials,
7 training.

8 THE COURT: Go ahead, Mr. Lewis.

9 BY MR. LEWIS:

10 Q What does training have to do with preventing
11 childhood lead poisoning?

12 A In the case of our risk assessors, or our inspectors,
13 they were required to be trained and then certified
14 by the state to fulfill their duties.

15 Q And was their central duty to inspect and assess
16 hazards from lead paint?

17 A Yes, that was their primary duty.

18 Q And why was it important to have supplies in order to
19 have a program to prevent childhood lead poisoning
20 from lead paint?

21 A Well, supplies could be anything from the piece of
22 paper that documents the inspection result or the
23 dust wipe test used to document the hazard in the
24 household.

25 Q Did the Childhood Lead Poisoning Prevention Program

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1 Q And if there was an irregularity, would CDC ask you a
2 question or inquire or write a letter to you?

3 A If there was an irregularity, yes, but I really don't
4 ever recall one happening.

5 Q Do you recall any irregularities being brought to
6 your attention by CDC regarding the projects that you
7 did with them?

8 A I don't recall any irregularity.

9 Q When you were manager of the program from 1992 to
10 2006, how did you know whether an expenditure under
11 the program was related to a lead paint activity?

12 A Well, first of all, it's important to remember that
13 all of our activities were focused on preventing
14 childhood lead poisoning through the major source
15 that we knew of, which was lead paint.

16 I coordinated and directed and managed
17 a management team, which, in turn, supported our
18 field staff and our direct service staff. We worked
19 together on a daily basis to assure that everything
20 we were doing, with frankly our limited resources,
21 were focused on lead paint hazards.

22 Q And did you do -- did you review the expenditures
23 contemporaneously in 1992?

24 A Yes.

25 Q And did you review the expenditures contemporaneously

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1 in 1993?

2 A Yes.

3 Q And did you do that for every year from 1992 to 2006?

4 A Yes. I had to either approve or oversee every
5 expenditure and activity of the Childhood Lead
6 Poisoning Prevention Program.

7 Q The other levels of approval that you mentioned, were
8 they also done contemporaneously beginning in the
9 year 1992?

10 A Yes.

11 Q Was approval of expenditures by the Childhood Lead
12 Poisoning Prevention Program conditional on the fact
13 that the expenditure was being made to prevent
14 childhood lead poisoning and to focus on the source
15 being lead paint?

16 A Yes. The stated purpose of each one of these
17 projects and the funding was conditional on the
18 prevention and ultimate elimination of childhood lead
19 poisoning through a focus on lead paint and lead
20 paint hazards.

21 Q Was the approval of these expenditures done
22 contemporaneously?

23 A Yes.

24 Q Was the recording of the approval of the expenditure
25 done contemporaneously?

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1 A Yes. Purchases and activities that required funding
2 were done on a daily basis, and each one of those was
3 reviewed at the program level with the managers of
4 the lead program and me, as well as the business
5 operations or accounting arm of the Health Department
6 and the City comptroller's office.

7 Q Were the actual approved expenditures recorded in
8 City financial records contemporaneously?

9 A Yes, they were.

10 Q And do those records exist today?

11 A They do.

12 Q And in the course of this case, have you been asked
13 to identify those records that you know about and
14 provide them to the lawyers?

15 A Yes.

16 Q Are you familiar with the accounting term "invoice"?

17 A I am.

18 Q Can you explain to the members of the jury what that
19 means in your program.

20 A Right. An invoice is used to request payment for a
21 good or a service. The invoice -- an invoice
22 individually would be prepared to pay a lead
23 abatement contractor for the cost of lead abatement
24 of a housing unit or the purchase of supplies and
25 materials needed to support those activities.

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1 describing the work of the various personnel assigned
2 to the project.

3 Q And is this a typical budget proposal for the 16
4 projects that were identified in Exhibit 2009 from
5 1992 to 2006?

6 A Yes, it's representative.

7 MR. LEWIS: Your Honor, similar to the
8 photographs, I would, I could go on with more
9 examples, if the Court would like. There are
10 literally tens of thousands of these budgets or
11 budget line items, but I'd rather just do this by
12 example and move on, if that's acceptable with the
13 Court.

14 THE COURT: That's acceptable to the Court.

15 MR. LEWIS: Thank you, Your Honor.

16 Q Can we turn to Exhibit 325, please. Start at page 6.

17 Ms. Murphy, before we go to that
18 exhibit, the last exhibit was a budget. Can you
19 explain to the members of the jury that -- whether or
20 not a budget is a projection of money to be spent or
21 an accounting of money already spent?

22 A A budget is a projection or a plan on the money to be
23 spent over the life of the project.

24 Q And as a general matter, over the 14 years that you
25 managed the program, did you receive the budgetary

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- 1 funds that you requested, for example in the one
2 example we looked at?
- 3 A Yes.
- 4 Q And did you spend the money in a manner consistent
5 with what the budget proposal called for?
- 6 A Yes, we did.
- 7 Q And did you also have a responsibility after you
8 spent the money to identify that and report it to the
9 agency that was managing that project?
- 10 A Yes, we did. We provided regular reports to those
11 various agencies.
- 12 Q And what do you mean by regular?
- 13 A In some cases, we provided quarterly updates on --
14 every three months on every activity and our
15 expenditures for that particular quarter, and to
16 date. In some cases, it's only an annual report.
17 But regularly, the program would receive reports from
18 the business operations arm of the Health Department,
19 where we would get an accounting or an update on the
20 status of our expenditures.
- 21 Q And those reports would show actual expenditures,
22 dollars that had already been spent?
- 23 A Yes. And we'd reconcile them with our internal
24 information at the program level.
- 25 Q And did you request a report for each year, 1992 to

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1 2006, for each project on Exhibit 2009 that we
2 reviewed this morning?

3 A Yes.

4 Q Do you now have 325 in front of you?

5 A Yes.

6 Q Can you identify that, please.

7 A Yes. This is a report to the U.S. Department of
8 Housing and Urban Development for a project that we
9 referred to as HUD Round 8. It was a multi-year
10 project and at the end of each one of these projects,
11 we would have to provide a closeout report. The
12 closeout report would represent our achievements, our
13 lessons learned, as well as a financial documentation
14 of the project.

15 MR. LEWIS: Can this be admitted into
16 evidence, Your Honor, and published to the jury?

17 MR. MURRAY: Well, Your Honor, we have no
18 objection to this page. I don't know how many pages
19 follow, and if other pages follow, what's on them.
20 As to this -- what we're looking at right now, we
21 have no objection.

22 THE COURT: Is this Exhibit 325?

23 MR. LEWIS: Yes, it is, Your Honor. It's
24 beginning at page 6 of that exhibit.

25 MR. SERPE: And 6 and 18.

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1 MR. LEWIS: And pages 6 and 18 are the ones
2 we wish to have admitted and published to the jury.

3 THE COURT: My Exhibit 325 on the laptop
4 begins with something else, but does it get to
5 what -- yes. Page 6 --

6 MR. LEWIS: Page 6 and 17, Your Honor. I
7 made a mistake. I said 18.

8 MR. MURRAY: 6 is a blank page. This is
9 September 23rd?

10 MR. LEWIS: Yes, it is. Your Honor, as to
11 those pages, can this be admitted and published to
12 the jury?

13 THE COURT: Pages?

14 MR. LEWIS: 6 --

15 THE COURT: And 7 of the exhibit.

16 MR. LEWIS: 6 and 17, Your Honor. Your
17 Honor, may I hand up a hard copy to the Court to
18 assist this?

19 THE COURT: You may, but this one's working
20 fine --

21 MR. LEWIS: Okay.

22 THE COURT: -- on the screen.

23 MR. MURRAY: We have no objection to those
24 pages, Judge.

25 THE COURT: All right. Exhibit 325 --

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1 The second paragraph states that the
2 City of Milwaukee Health Department Childhood Lead
3 Poisoning Prevention Program requested and received a
4 total of \$5,000,000 of funding under HUD's Lead-Based
5 Paint Hazard Control Program for Round 8 activities.

6 Do you see that?

7 A Yes.

8 Q What was the central purpose of Round 8 activities?

9 A To identify and target and control lead-based paint
10 hazards in high-risk housing.

11 Q And what was the central purpose of expenditures made
12 under Round 8?

13 A To control lead-based paint hazards and to protect
14 children from them.

15 Q And was the \$5,000,000 identified in this exhibit
16 received and expended to accomplish the purposes you
17 mentioned?

18 A Yes, they were.

19 Q Can we turn to page 17, please. Can you blow up the
20 part that says "Expenditures-Financial Summary"?
21 That's good.

22 Ms. Murphy, can you explain to the
23 members of the jury what an Expenditures-Financial
24 Summary section of a closeout report is?

25 A Yes. The financial summary, in lump sum fashion,

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1 provides a rationale or a description of how the
2 funds were spent. This particular paragraph
3 summarizes and sums up the costs for direct labor or
4 what we refer to as personnel or staff costs.

5 Q And are there -- I'm sorry. Were records created
6 contemporaneously to identify that each of the
7 expenditures identified in the financial summary was
8 made for a purpose related to lead-poisoning
9 prevention?

10 A Yes. That was done through the time entry system.

11 Q And is this Exhibit 325 a typical closeout report
12 that you did for each project from 1992 to 2006 in
13 relation to the organization that you were
14 coordinating with to expend those funds?

15 A This was a typical closeout report to the Department
16 of Housing and Urban Development. Different funding
17 agencies and different agencies involved in childhood
18 lead poisoning prevention required different formats
19 of final reports.

20 Q But for expenditures under each program, from 1992 to
21 2006, under your supervision, was a closeout report
22 done and provided?

23 A Yes, a closeout or a final report.

24 Q And does a closeout or final report --

25 THE COURT: Excuse me. I'm not sure about

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1 A Yes, I can. When I began with the program in 1992,
2 our budget or our expenditures hovered somewhere
3 around half a million dollars. After two to three
4 years, we broke the million dollar mark for our
5 annual budget, and then through the 1990s and early
6 2000s, our annual budgets were somewhere between 3,
7 4, and 5 million. In 2006 was our highest budget
8 year, where our annual budget exceeded \$7,000,000.

9 Q And you described the budget amounts. Would the
10 expenditures amounts be the same?

11 A The budget was our proposed or our projected
12 expenditures. That was different, but not very
13 different, from the final expenditures under a
14 specific project.

15 Q Ms. Murphy, you described a range of figures for
16 the -- for the closeout or the actual expenditures in
17 each year. What would be an average number from 1992
18 to 2006 for the actual expenditures made by the
19 Childhood Lead Poisoning Prevention Program on lead
20 poisoning prevention and lead hazard abatement?

21 A On average, over those 14, 15 years, the average
22 budget was somewhere -- annual budget was somewhere
23 between 3- and \$4,000,000.

24 Q And would that have been the annual amount of
25 expenditures?

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1 A Yes.

2 Q On average?

3 A Yes.

4 Q And can you estimate over the course from 1992 to
5 2006, the total number of expenditures made for the
6 purpose of preventing childhood lead poisoning and
7 abating lead paint hazards?

8 A Somewhere between 50- and \$55,000,000.

9 Q And is that based on your personal experience?

10 A Yes, it is.

11 Q And is that based on your role in supervising the
12 budgets for every project from 1992 to 2006?

13 A Yes.

14 Q And is that based on your role and your personal
15 experience in supervising the closeout reports for
16 every project for every year from 1992 to 2006?

17 A That's correct.

18 Q Were there any external controls, outside of the
19 Health Department, on expenditures to assure that
20 expenditures were made for the purposes of the 16
21 projects listed on 2009?

22 A Yes. The external controls were with the Health
23 Department business operations arm, the City
24 comptroller, and then eventually the agency that
25 provided funding for the project.

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1 Q When you needed to obtain expenditure data to make a
2 report, where did you get it?

3 A From the business operations accounting arm of the
4 City of Milwaukee Health Department.

5 Q And does the comptroller have access to that same
6 data?

7 A Yes.

8 Q And could the comptroller give a more precise figure
9 of the expenditures from 1992 to 2006 that were made
10 by the program for childhood lead poisoning
11 prevention and lead paint abatement?

12 MR. MURRAY: Objection, speculation.

13 THE COURT: Just a moment. Overruled. You
14 may answer.

15 THE WITNESS: Could you repeat the
16 question?

17 BY MR. LEWIS:

18 Q Yes. Would the comptroller be able to make a more
19 precise estimate than you did -- I'm sorry. Let me
20 withdraw that.

21 Would the comptroller be able to make
22 a precise calculation of the amount of funds expended
23 by the Childhood Lead Poisoning Prevention Program
24 from 1992 to 2006 on the activities related to
25 childhood lead poisoning and abatement of lead paint

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1 MR. LEWIS: Thank you very much. "Only 10
2 percent of the housing units in the target area
3 report substantial deferred maintenance, which may
4 result in costs higher than the estimated average."

5 THE COURT: Mr. Murray, go ahead.

6 MR. MURRAY: Thank you, Your Honor.

7 THE COURT: Is this a -- Are we done with
8 these exhibits?

9 MR. MURRAY: We are done with these
10 exhibits.

11 THE COURT: Then let's break until 1:30.

12 Members of the jury, do not discuss
13 the case during the noon recess. Please be back in
14 the jury room at or before 1:30.

15 All rise for the jury.

16 (Jury excused.)

17 THE COURT: Anything that needs to be put
18 on the record at this point, Mr. Lewis?

19 MR. LEWIS: Nothing, Your Honor.

20 THE COURT: Mr. Murray?

21 MR. MURRAY: No, Your Honor.

22 THE COURT: All right. We'll recess until
23 1:30.

24 (A lunch recess was taken.)
25

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1 THE CLERK: All rise.
2 (Jury present.)
3 THE CLERK: Please be seated.
4 THE COURT: Good afternoon and welcome back
5 to all. We'll proceed with the cross-examination of
6 the witness, Ms. Murphy, and Mr. Murray.
7 MR. MURRAY: Thank you, Your Honor.
8 THE COURT: You may continue.
9 MR. MURRAY: Good afternoon, ladies and
10 gentlemen.
11 JURY PANEL: Good afternoon.
12 BY MR. MURRAY:
13 Q Could you display, Don, to the witness and to Court
14 and counsel Exhibit 897, please.
15 Would you take a look at that,
16 Ms. Murphy.
17 A Yes.
18 Q This is a memorandum that was provided to you by
19 Richard Gaeta on May 4th, 2005; correct?
20 A That's correct.
21 Q And the subject -- Well, strike the question.
22 Why don't you tell us who Richard
23 Gaeta is, please.
24 A Richard Gaeta is the environmental prevention
25 manager/supervisor, and he supervises other

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1 environmental managers and field staff.

2 Q Within your department.

3 A Within the Childhood Lead Poisoning Prevention
4 Program.

5 Q And in that capacity, he was providing this
6 memorandum to you on a topic called "Aldermanic
7 Approach"; correct?

8 A That's correct.

9 MR. MURRAY: May we display this to the
10 jury, please, Your Honor?

11 THE COURT: Any objection?

12 MR. LEWIS: None, Your Honor.

13 MR. MURRAY: Could you highlight the first
14 paragraph, please, Don.

15 Q Now, Ms. Murphy, in this memorandum to you, Mr. Gaeta
16 indicates that "The slowdown in applications can put
17 us in trouble if we don't come up with some
18 innovative ways to stimulate more demand."

19 The applications that he is referring
20 to are applications for window treatments?

21 A Yes, for our Primary Prevention Program.

22 Q And we had testimony here a few days ago by
23 Ms. Zollicoffer, who was working through the
24 Dominican Center for Women, and talked about the fact
25 that she would go canvassing in the neighborhoods and

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1 try to generate enthusiasm for your programs; you
2 understand that to be so?

3 A Yes.

4 Q But apparently, as of this time at least, the number
5 of people who were applying to volunteer in this
6 program was down; is that correct?

7 A What this indicates, yes, is that there's a slowdown
8 in applications.

9 Q And that was of concern to you and to Mr. Gaeta
10 because it would impact on your funding; is that
11 true?

12 A Under our various funding agreements, we agreed to
13 conduct lead abatement in a certain number of homes,
14 and, yes, a slowdown in applications can impact our
15 projection of the number of lead paint control
16 abatements.

17 Q Projections to whom?

18 A To Housing and Urban Development.

19 MR. MURRAY: Okay. I'd offer the portion
20 of this exhibit, Exhibit 897, that has been
21 published, Your Honor?

22 MR. LEWIS: No objection, Your Honor.

23 THE COURT: Received.

24 BY MR. MURRAY:

25 Q Could we go, please, to Exhibit 896. Do you have

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1 that before you, Ms. Murphy?

2 A I do.

3 Q And do you recognize it?

4 A I do.

5 Q It's titled "House and Lead Poisoning Summary,
6 May 2006," with data that apparently was current as
7 of 2005; is that true?

8 A That's correct.

9 MR. MURRAY: May we publish this, Your
10 Honor?

11 MR. LEWIS: No objection.

12 THE COURT: Go ahead.

13 BY MR. MURRAY:

14 Q Now, this is a little bit hard to see, but we're
15 going to focus on a couple points, and maybe we can
16 highlight those. The first section -- the first half
17 of this document -- talks about basically housing
18 statistics in Milwaukee; correct?

19 A That's correct.

20 Q And what I'd like to focus on are the pre-1950
21 housing, which is broken down into two parts of this,
22 pre-1940 and 1940 to 1949. So if we could highlight
23 those. Thank you.

24 We have to do a little math here, but
25 what this basically tells us is that with respect to

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1 A Yes, you've read that correctly.

2 Q And then at 20 micrograms per deciliter and higher,
3 the explanation is, "This means the child may have
4 lead poisoning. This result, along with the child's
5 lead history and the type of this test, will be used
6 to determine necessary medical management and
7 follow-up by the Childhood Lead Poisoning Prevention
8 Program. The longer the exposure and the higher the
9 lead level increases the probability of lasting
10 adverse health affects."

11 Have I read that correctly?

12 A You have.

13 MR. MURRAY: And so, Your Honor, I would
14 offer this three-page 2007 excerpt from the Health
15 Department's Web page.

16 MR. LEWIS: No objection.

17 THE COURT: Received.

18 MR. MURRAY: Your Honor, that con --

19 THE COURT: The number again is?

20 MR. MURRAY: 514.

21 THE COURT: 514.

22 MR. MURRAY: Your Honor, that concludes my
23 questioning of Ms. Murphy, but I would like to just
24 do a little housekeeping, two exhibits I don't
25 believe I offered when I made reference to them, and

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1 those were 404, page 4, and 503 as published.

2 THE COURT: Any objection to 404, page 4,
3 spreadsheet?

4 MR. LEWIS: No, Your Honor.

5 THE COURT: Received.

6 MR. LEWIS: I'm sorry. 404, page 4 --

7 THE COURT: It's a spreadsheet.

8 MR. LEWIS: -- is being moved into
9 evidence. No objection.

10 THE COURT: And the other was?

11 MR. MURRAY: 503.

12 MR. LEWIS: Is that as published?

13 MR. MURRAY: As published.

14 THE COURT: I believe this has been
15 received to the extent published. Is that it?

16 MR. MURRAY: That's it.

17 THE COURT: Any redirect?

18 MR. MURRAY: Thank you, Ms. Murphy.

19 MR. LEWIS: Yes, Your Honor.

20 THE COURT: Go ahead, Mr. Lewis.

21 MR. LEWIS: May I have 897 back on the
22 screen?

23 MR. SCOTT: From me or from --

24 MR. LEWIS: I'd like your assistance. And
25 I believe the first paragraph was highlighted?

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1 MR. SCOTT: Do you want me to do that
2 again?

3 MR. LEWIS: Yes, please. Thank you. Can
4 you make it a little larger?

5 MR. SCOTT: Not really much.

6 MR. LEWIS: Okay.

7 MR. SCOTT: It stretches across.

8 MR. LEWIS: Thank you.

9 REDIRECT EXAMINATION

10 BY MR. LEWIS:

11 Q Ms. Murphy, you have before you Exhibit 897; can you
12 see that?

13 A I can.

14 Q And the highlighted material that you discussed with
15 Mr. Murray, you looked at the first sentence. The
16 second sentence says, "In looking at our target area,
17 it is clear that Alderman McGee covers the largest
18 portion of our target area. His district also has
19 the second highest prevalence rate in the City,
20 23.7 percent."

21 Do you see that?

22 A I do.

23 Q And can you explain what a prevalence rate is?

24 A A prevalence rate represents the percentage of
25 children tested in a given year with a lead level

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1 STATE OF WISCONSIN)

2) SS
3 MILWAUKEE COUNTY)

4 I, CONSTANCE L. BAUER, RPR/RMR/CRR, Notary
5 Public in and for the State of Wisconsin, do hereby
6 certify that the foregoing proceedings were recorded by me
7 and reduced to writing under my personal direction and
8 that it is a true and correct transcript of said
9 proceedings had in said matter to the best of my
10 knowledge.

11 In witness whereof, I have hereunto set my
12 hand at Milwaukee, Wisconsin, on this 6th day of June,
13 2007.

14

15

16

CONSTANCE L. BAUER, RPR/RMR/CRR

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Vol. 14

1 STATE OF WISCONSIN : CIRCUIT COURT : MILWAUKEE COUNTY

2 -----
3 CITY OF MILWAUKEE,
4 a municipal corporation,

5 Plaintiff,

6 vs.

Case No. 01-CV-3066

7 NL INDUSTRIES, INC.,
8 a foreign corporation,

9 Defendant.

10 Before the Honorable

11 JOHN FRANKE, Circuit Court Judge

12 Branch 25, presiding

13
14 Friday, June 8, 2007

15 VOLUME 14

16 9:12 a.m.

17 A P P E A R A N C E S:

18 RICHARD LEWIS, RICHARD SERPE, CHRIS RIORDAN,
19 Attorneys at Law, and SARA SCHUBERT, appeared on behalf of
20 the Plaintiff.

21 DONALD SCOTT, ANDRE PAUKA, JOHN HUGHES, MICHAEL
22 WIRTH, Attorneys at Law, appeared on behalf of the
23 Defendant.
24
25

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1 indirect effects, which I haven't explained, but they
2 were small and not consistent. At age 7, there were
3 no statistically significant direct or indirect
4 effects on blood lead concentration at age 2. They
5 don't show the data here. It seemed to them so clear
6 that there was no effect at the age 2 exposure that
7 they didn't even present the data.

8 Q All right. If I could just go back then to the
9 abstract to look at what conclusion the authors drew
10 in their results. I won't do this, Doctor, because
11 it's chopped off on the left-hand side. It won't
12 display right. Let me go instead to page 655,
13 Column 1, right here.

14 Would you -- would you read this for
15 us, Doctor, and tell us if you agree, based upon the
16 data in the study as you've reviewed it.

17 A Yes, no effect on -- in seven-year-olds from blood
18 lead at age 2. There is an effect on seven-year-olds
19 from their blood levels at age 7.

20 Q There is an effect -- Okay.

21 A Yes.

22 Q Uh-huh. So the relationship between the lead level
23 the child has at seven and the behavior at age 7.

24 A Yes.

25 Q No relationship between the lead level at two and the

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1 behavior at two --

2 A Correct.

3 Q -- at seven.

4 A And the behavior at seven, that's correct.

5 Q Doctor, just in conclusion, looking at the series of
6 NIH-sponsored TLC studies, Liu and the two Chen
7 articles, tell us whether or not they find that blood
8 lead does matter to the outcomes in a child while the
9 lead is high at age 5 and age 7? Does it matter?

10 A They find that the concurrent level has an influence
11 on IQ and behavior at ages 5 and 7. I think it's a
12 clear finding, a negative effect on IQ and some of
13 the measures of behavior.

14 Q Doctor, I'd ask you to give any opinions to a
15 reasonable degree of scientific certainty and ask
16 you, in your opinion, do the TLC studies sponsored by
17 the NIH show that there are continuing or persisting
18 effects from lead exposure at age 2 upon child's
19 behavior or IQ in the school years?

20 A They do not show that. They show that there are no
21 continuing, or as they call it, residual effects from
22 the early exposure in this TLC -- we call the seven
23 TLC-related studies, yes.

24 Q Now, looking at the entire body of scientific
25 literature that you have found on the subject of

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1 whether effects continue, do you have an opinion to a
2 reasonable degree of scientific certainty whether the
3 science, as of this point in time, tells us whether
4 early exposure around age 2 does or does not have
5 residual or persisting effects into the school years?
6 Do you have an opinion?

7 A I do.

8 Q What's your opinion?

9 A That the available data, the weight of the available
10 evidence, especially that from what I see is to be
11 the best study on this issue, show that there is not
12 a persisting effect on IQ from early life, that is
13 age 2 exposures. There are effects from continuing
14 exposures, but not from the early exposure.

15 MR. SCOTT: Your Honor, I'm concluding. I
16 do want to offer the exhibits as published, the
17 series of articles as published, and I'll need to
18 make a list of those numbers. May I do that after I
19 get a list from my notes?

20 THE COURT: Yes, you may. With respect to
21 articles to the extent they've been published, is
22 there any objection to receipt?

23 MR. SERPE: Your Honor, no extent to the
24 articles themselves, but we would object to the
25 demonstrative, which was being put up periodically,

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1 with comments on it, as being admitted into evidence.

2 MR. SCOTT: I'm not offering that, Your
3 Honor.

4 THE COURT: I think we're just talking
5 about the articles, and to the extent --

6 MR. SERPE: To the extent published, no
7 objection.

8 THE COURT: -- published, they are
9 received, and we'll need to make sure the clerk has
10 those numbers.

11 MR. SCOTT: I'll write them down right now
12 from my notes.

13 THE COURT: All right. We're going to go
14 until about 12:20 so who's going to handle the
15 cross-examination?

16 MR. SERPE: I am, Your Honor.

17 THE COURT: Mr. Serpe, you may proceed.
18 You can do it from there, or if you want to use the
19 table or the podium or whatever.

20 (Discussion off the record.)

21 MR. SERPE: Your Honor, it'll take me only
22 about 60 seconds to get organized, if you want to
23 indulge me. Thank you.

24 May it please the Court. Good late
25 morning, Dr. Rodricks. Good morning, ladies and

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Vol. 14

1 STATE OF WISCONSIN)

2) SS
3 MILWAUKEE COUNTY)

4 I, CONSTANCE L. BAUER, RPR/RMR/CRR, Notary
5 Public in and for the State of Wisconsin, do hereby
6 certify that the foregoing proceedings were recorded by me
7 and reduced to writing under my personal direction and
8 that it is a true and correct transcript of said
9 proceedings had in said matter to the best of my
10 knowledge.

11 In witness whereof, I have hereunto set my
12 hand at Milwaukee, Wisconsin, on this 8th day of June,
13 2007.

14
15
16 CONSTANCE L. BAUER, RPR/RMR/CRR
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Transcript of Proceedings - 6/11/2007

Vol. 15

1 STATE OF WISCONSIN : CIRCUIT COURT : MILWAUKEE COUNTY
2 -----

3 CITY OF MILWAUKEE,
4 a municipal corporation,

5 Plaintiff,

6 vs.

Case No. 01-CV-3066

7 NL INDUSTRIES, INC.,
8 a foreign corporation,

9 Defendant.

10 Before the Honorable

11 JOHN FRANKE, Circuit Court Judge

12 Branch 25, presiding

13
14 Monday, June 11, 2007

15 VOLUME 15

16 11:05 a.m.

17 A P P E A R A N C E S:

18 MICHAEL HAUSFELD, RICHARD LEWIS, RICHARD SERPE,
19 TERRY NILLES, Attorneys at Law, and SARA SCHUBERT,
20 appeared on behalf of the Plaintiff.

21 JAMES T. MURRAY, JR., DONALD SCOTT, ANDRE PAUKA,
22 JOHN HUGHES, Attorneys at Law, appeared on behalf of the
23 Defendant.
24
25

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1 obviously in the popular press about the dangers or
2 perceived dangers of trans fat. So it's a field
3 that continues to change.

4 Q All right. Let's turn to childhood lead paint
5 poisoning and the question of the public health
6 consensus in the 20th century. Let's begin at the
7 beginning, 1900. In America, was there a public
8 health consensus about an issue of childhood lead
9 paint poisoning?

10 A No.

11 Q What was known at that point in general about
12 childhood cases of lead poisoning?

13 A There were children who were lead poisoned in the
14 19th and early part of the 20th century. The most
15 common cause of lead poisoning was contaminated
16 water and contaminated food. There was also a
17 variety of other instances, such as getting into
18 medicines that had lead, getting into cosmetics,
19 especially worn by some Asian women that had that
20 very white -- and sometimes babies could get too
21 much of that. But for the most part, it dealt with
22 contaminated food and contaminated water supplies.

23 Q That was 1900. Let's move forward now to 1920. As
24 of that time, 1920, was there a public health
25 consensus in this country about a problem of

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1 childhood lead paint poisoning?

2 A No.

3 Q What was known by 1920 about the question of lead
4 paint poisoning in children?

5 A There had been some -- some case reports, so -- and
6 there were two that came out of Baltimore with --
7 Thomas and Blackfan were the authors in 1914, and
8 then Blackfan expanded on that in 1917.

9 Q I'm going to display for you Exhibit 675. And can
10 you identify this article?

11 A Yes, that's -- this is the 19 -- excuse me, this is
12 the 1914 first case report.

13 Q And what was reported in -- I'm sorry. It's on the
14 screen now.

15 What was reported in the 1914
16 article case report by Thomas and Blackfan?

17 A A child who was -- was being reared in an orphanage
18 suffered seizures and coma, was brought to the
19 Johns-Hopkins Hospital, and doctors there were
20 puzzled about what the problem was. Eventually,
21 after a long period of time, they settled that it
22 was a case of lead poisoning. They queried that
23 because most lead poisoning in 1914 was still
24 industrial, and so obviously a child was not in any
25 kind of industrial setting. And so they visited

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1 the home, the orphanage where he was being reared,
2 and they noticed that he had chewed all of the lead
3 paint off of the slats of the crib in which he
4 was -- which he was living.

5 Q Was that finding described in the article that we
6 have on the screen?

7 A Yes.

8 Q Okay. Was there any indication what level of blood
9 lead the child may have had in the Thomas and
10 Blackfan article?

11 A No, this was still 20-some-odd years before
12 blood-lead levels were available clinically.

13 Q You also mentioned the 1970 -- 1917 article by the
14 second author, Dr. Blackfan?

15 A Yes.

16 Q And I'll be putting on the screen Exhibit 8.
17 That's the wrong one.

18 A I think that's the same one.

19 Q That was the same article. Actually, the jury's
20 previously seen anyway the Blackfan article of
21 1917. Tell us what was reported at that time in
22 the second article in this country.

23 A Well, what happens in case reports, oftentimes in
24 the -- they are followed by additional case reports
25 by the same author. That's a frequent thing that

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1 happens. And so what happened in the case of
2 Dr. Blackfan, three years later he had seen three
3 more children, and so he described again his first
4 case and then three more cases. And then in
5 particular, he described the source of paint of two
6 brothers who came into his care in visiting and
7 figuring out what the home and -- and what had
8 happened to those two brothers was that they had
9 gnawed all of the paint off of a set of furniture.

10 Q And 1917, there still was not a blood-lead test
11 available?

12 A That's correct.

13 Q As an historian, Dr. English, what was the
14 influence, if any, of these early Baltimore cases
15 reported by Thomas and Blackfan upon the
16 development of a public health response?

17 A Well, they seemed to have caught the attention of
18 other pediatricians around the country, because in
19 a short period of time, in the early 1920s,
20 additional case reports occurred in cities around
21 the country. So there were some from Florida,
22 there was one from New Orleans, an important one in
23 1924 from Los Angeles by John Ruddock.

24 MR. SCOTT: If we could look at
25 Dr. Ruddock's article. It's Exhibit 798.

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1 Q And where and when was this published?

2 A 1924 in the Journal of the American Medical
3 Association, and that came from Los Angeles.

4 Q Okay. Dr. Ruddock's name. What did Dr. Ruddock
5 report here?

6 A Dr. Ruddock added an important new finding. And
7 what he said was that the children -- he described
8 a couple of more children -- a couple more case
9 reports of children who had been lead poisoned, but
10 what he did was he -- he made the observation that
11 the -- the lead had been acquired through a very
12 abnormal eating pattern which doctors call pica.

13 And what pica -- he defined it right
14 there. Pica is a craving for unnatural articles of
15 food, a depraved appetite. And pica is different
16 from the normal hand-to-mouth activity that all
17 infants have. And those of you who have children
18 or grandchildren, have seen that, know that from
19 six months, a child will often pick up something,
20 put it in their mouth, put it down. And over the
21 course of time your parent keeps on saying -- I
22 still remember my mother, I can hear her now,
23 saying get that out of your mouth. And that's just
24 part of what happens. That's not pica. Pica --
25 and what Dr. Ruddock described, was a very

1 aggressive, very -- an eating that would -- tended
2 to devour all of the paint. It would strip the
3 paint off of furniture or woodwork.

4 Q What influence did Dr. Ruddock's 1924 article have
5 on the development of public health perceptions of
6 this problem?

7 A Well, one of the things is this notion of an
8 aggressive eating problem, pica, formed part of the
9 public health community's understanding of the
10 disease from -- from the very beginning of the
11 first consensus that happened in the 1930s through
12 into the 1970s.

13 Q Dr. English, can you identify in the period of the
14 1920s any particular center or place of activity on
15 the question of childhood lead paint poisoning in
16 the country?

17 A You mean a geographic spot?

18 Q Yes, please.

19 A Yes. It would probably be Boston. And the reason
20 for that is after these first case reports, which
21 were sort of scattered around the country, the
22 first time that there were a significant number of
23 cases in one location that were described was in
24 Boston. And Charles McCann was the pediatrician
25 who -- who wrote those articles, bringing those

1 cases to the attention of -- of the American
2 physicians.

3 Q What was Dr. McCann's affiliation?

4 A He was with Harvard, associated with Harvard in
5 Boston.

6 Q I'm showing Exhibit 10. And could you identify
7 this article, please, Doctor.

8 A Yes. This is his first article on -- on childhood
9 lead poisoning.

10 Q And do you recall when he published it?

11 A 1926, I believe.

12 Q I think it was last week, Dr. Landrigan was
13 testifying for the City, and he referred to a first
14 report in this country of childhood lead paint
15 poisoning by McCann in 1912. Are you aware of any
16 such article?

17 A I -- I think that's not correct. This is his first
18 paper. And I don't know what Dr. Landrigan was
19 referring to.

20 Q What was the influence -- well, first of all, what
21 did Dr. McCann report here in his 1926 piece?

22 A Well, what he did was he -- first of all, he began
23 to incorporate what other people had done. So he
24 mentioned pica, too. He picks up Dr. Ruddock's
25 observation. And then he went on to -- in this

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1 think I mentioned that what they were interested in
2 doing is actually tracing the lead from when it
3 entered the body and moved through it and
4 eventually was excreted from the body in the urine
5 or feces. That required being able to measure lead
6 in very, very small amounts in body tissues. And
7 that's one of the things that they did first,
8 because there was just no technique available for
9 doing that. And Dr. Fairhall did that. And what
10 he was able to do was be able to find like one part
11 in a billion, which was something practically
12 unheard of in the first part of the 20th century.

13 Q Doctor, we've talked about National Lead Company's
14 role during the 1920s. Did there come a time when
15 a public health consensus first emerged in our
16 country concerning the problem of childhood lead
17 paint poisoning?

18 A Yes.

19 Q You mentioned earlier a report by the Boston Health
20 Department. Before we look at any particular
21 writings, I'm going to ask you what that consensus
22 was. You prepared a summary slide to follow you as
23 you describe this for the jury?

24 A I have.

25 Q All right. I'm going to let this follow you, not

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1 lead you.

2 So, first of all, we'll just put it
3 up after you tell us, what was the public health
4 consensus that you found in the literature of the
5 late 1920s?

6 A Right. 1920s, and it extended into the 1930s in a
7 number of public health organizations. And what
8 these public health organizations concluded, based
9 on the case reports that had been published so far,
10 and including Dr. McCann's studies in Boston, was
11 that children in the United States were being lead
12 poisoned in two specific environments, and then --
13 and in response to those two environments, they
14 made two recommendations about what the American
15 public should do about it.

16 The first environment was a child
17 who was in a crib for a long period of time who
18 would chew lead paint from the slats of the crib or
19 would chew lead paint from toys which were in the
20 crib that the child was confined in. That was
21 environment number one.

22 The second environment was the
23 toddler --

24 Q Let me pause, Dr. English. What did the public
25 health experts recommend for the infant in the crib

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1 or highchair in the first setting?

2 A In this first setting in cribs and toys, the
3 recommendation was that cribs and toys should not
4 be painted with lead paint.

5 Q Okay. I think you were going to turn to the second
6 situation.

7 A Yes, the second was an older child, a toddler,
8 roaming through the houses of the 1920s, who came
9 into contact with -- with lead paint on woodwork.

10 Q And what did the public health experts recommend
11 for the child, the toddler-age child in the second
12 situation?

13 A They recommended that parents be vigilant, parents
14 supervise their child in a way that they were
15 supervising the children for other hazards found in
16 a household.

17 Q Okay. Next I want to show you some of the
18 literature you've identified for me on these two
19 issues identified in the '20s and early '30s. If
20 we could begin with the infants on -- I'm going to
21 display Exhibit 679. And would you tell us what we
22 are looking at, Dr. English.

23 A This is that first -- that first consensus
24 statement from the Health Department in Boston in
25 1927.

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1 Q And what is discussed --

2 I'm displaying the second page, Your
3 Honor. It is printed as page 286, Lead Poisoning
4 in Early Childhood. And the first paragraph of
5 that section.

6 A Okay. After the heading, Lead Poisoning in Early
7 Childhood, "For this year, there have been reported
8 to this department on death certificates four
9 deaths of children from lead poisoning, all -- all
10 such causes of death having been certified by the
11 medical examiner. These deaths were due to a
12 childhood habit of sucking paint from cribs with a
13 consequent ingestion of lead and subsequent lead
14 poisoning and death. It would be well for mothers
15 and others entrusted with the care of children in
16 cribs to watch for this common habit in early child
17 life and to take steps to correct it whenever it is
18 found" -- I'm sorry, "wherever it is found."

19 Q And did the Public Health Service get involved in
20 this issue at or about the same time?

21 A Yes.

22 Q Would you describe, before I use any documents,
23 what the Surgeon General or the Public Health
24 Service said on this subject of the lead poisoning
25 in the infants.

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1 A Yes. In 1931, the Surgeon General issued a
2 statement on it.

3 Q Okay. Exhibit 632, there's an article,
4 Dr. English. Can you give us a date and tell us
5 whatever you know about this publication.

6 A It's 1931, and Child Welfare is a publication of
7 the Parent and Teacher Association. So this was a
8 message that was directed to a broad audience of
9 people concerned with children.

10 Q We are displaying in the right column an article
11 called Lead Poisoning from Toys. And begins
12 "Surgeon General Cumming of the United States
13 Public Health Service."

14 And would you continue, Dr. English,
15 and read for us what the Surgeon General said about
16 the problem in infancy as of 1931.

17 A "Surgeon General Cumming of the United States
18 Public Health Service has been informed of the
19 occurrence of occasional cases of lead poisoning in
20 infants and children, apparently due to biting lead
21 paint from cribs, toys, et cetera. The Public
22 Health Service has previously called attention to
23 this possible source of lead poisoning" -- that's
24 an article that appeared, and I'll talk about that
25 later.

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1 Q Is that the one in U.S. Daily?

2 A Yes, it is.

3 Q You may continue.

4 A -- "but it is likely that more cases occur than
5 become known. Children with perverted appetites
6 would be particularly likely to encounter the
7 hazard. Though lead has wide fields of usefulness,
8 the painting of babies' toys and cribs is not one
9 of them. Generally, manufacturers of these
10 articles are seeing to it that lead paint is not
11 used for this purpose, but warning is necessary
12 that parents, especially in repainting cribs,
13 should use paints which are free from lead, namely,
14 quick-drying lacquers or enamels sold for interior
15 use."

16 Q And how does this advice attributed to the Surgeon
17 General relate to the public health of infants that
18 you described a few moments ago?

19 A Well, he also identifies the source of the problem
20 as babies' toys and cribs. And he's also making
21 the recommendations that these items should not be
22 painted with lead paint.

23 Q Incidentally, Dr. English, do you know
24 approximately how large the Public Health Service
25 was reporting to the Surgeon General at that point

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1 in time?

2 A It was a relatively large organization. I've read
3 the annual report of the Surgeon General's office,
4 and there were, oh, something like 11,000 employees
5 that reported to the Surgeon General.

6 Q Now, these articles have focused on the infant
7 situation you described. Are there articles also
8 addressing more directly the toddlers?

9 A Yes, there are.

10 Q I'm going to show you Exhibit 757. And before we
11 look at any detail, this -- although it's blurry in
12 the copying -- says the United States Daily. What
13 is this publication?

14 A The United States Daily was -- in the 1930s was a
15 daily newspaper format that dealt with issues that
16 were distributed widely to government employees and
17 bureaus and also read by the informed public.

18 That changed over the course of time
19 in that the U.S. Daily became a magazine, a weekly
20 magazine, around 1940, it was called U.S. News.
21 And that changed again in 1948 and became U.S. News
22 and World Report, a magazine that's still in
23 existence today. So like many publications, it
24 changed over the course of the 20th century.

25 Q This particular edition is dated September 12th,

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1 program and so that -- like I showed the radio
2 address, but that was just one of many things that
3 he did, lots of publications.

4 The third thing he did was really
5 quite innovative, and that was the nurse home
6 visit. And what he did, when a child was
7 identified as being lead poisoned, he assigned a
8 nurse to go into the home who would then try to
9 identify the sources of lead in the child's home
10 and then educate the parents about what they should
11 be doing in order to prevent further exposure to
12 lead.

13 Q What value did the home nurse visits have to the
14 development of public health knowledge on this
15 issue?

16 A I think they were crucial, because up until that
17 point, the -- most of the case reports had relied
18 on the parents' sort of recollection or sort of
19 their own appraisal of where the child might have
20 gotten into lead. But the home visitor was able to
21 have a professional come in, the nurse could come
22 in and actually document that. And what -- what
23 they documented was that children were getting lead
24 poisoned in precisely the same way that parents had
25 been saying, and that is the two settings that

1 we've talked about.

2 Q Now, in addition to the work you've described in
3 Baltimore Health Department under Dr. Huntington
4 Williams, was there work done at a scientific level
5 by the Baltimore experts?

6 A Yes. They developed, in the 1930s, the first
7 clinical test to measure the blood level, and
8 before that it had been an experimental test. But
9 what Huntington Williams did was had his Bureau of
10 Laboratory develop a test that could be used by
11 clinicians.

12 Q And those were blood-lead measurement. And did
13 they use the same vocabulary we use about
14 micrograms or milligrams per deciliter of blood?

15 A No, they didn't. They used milligrams per 100 cc's
16 of whole blood. I tend to convert those when I see
17 the old just because I'm used to the microgram,
18 but --

19 Q What was the importance in this story, Doctor, of
20 the development of blood-lead testing technology
21 for the first time in Baltimore?

22 A Well, what it does is it gives some precision to
23 what -- the extent of the exposure and so we have
24 an idea of how much lead these children had who
25 were being designated as being poisoned in the

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1 1930s. And -- oh, and additionally, from a
2 historian's point of view, it gives me a tool in
3 order to go back and look at some of those case
4 reports and see the symptoms that the child had and
5 make a reasonable estimate of what the blood
6 level -- lead levels would have had to have been in
7 some of those children who were poisoned before the
8 test came into being.

9 Q I'm showing you Exhibit 684. And can you identify
10 this article, sir.

11 A Yes. This is one of several articles from the
12 Baltimore Health Department that described their
13 experience with this new blood-lead test.

14 Q This is by Kaplan and McDonald. And what do they
15 do in this article? I'm going to display a table
16 in a moment, but first, put us in the context.
17 What are they reporting here?

18 A Well, this article came out in 1942, and the test
19 was developed in 1935. And so this gave the first
20 several years of experience that they had had with
21 their test. Now, up until that point, the
22 diagnosis -- and really, up until really into the
23 1960s, the diagnosis of lead poisoning was based on
24 symptoms. And so you'll see in the table that it's
25 divided into the proven cases and not proven cases.

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1 And, of course, they were also interested in
2 adults, not just children. So we'll see a column
3 for children, and we'll see a column for adults.
4 But what it does, it gives a spectrum, it gives a
5 sense of what they considered normal and what they
6 considered to be abnormal.

7 Q This is 1942?

8 A '42, yes.

9 Q Exhibit 684, yes, reporting on the early years of
10 testing.

11 We'll go to the fourth page -- third
12 page, Table 1. And, Doctor, would you describe for
13 us what we are looking at in this table.

14 A Initial blood-lead values in normal individuals and
15 in suspected cases of blood-lead -- suspected cases
16 of lead poisoning.

17 Q All right. Here, pause for a moment. The normal
18 controls, what are they?

19 A These are people who are -- who are not suspected
20 of lead poisoning who served as a comparison group.
21 I see a value -- the high end of the value for the
22 normal is .08.

23 Q What does that translate into in modern --

24 A That's 80 micrograms.

25

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1 Q Then could you tell us what is shown in this column
2 called normal controls.

3 A Right. What it shows is that really all but two
4 are above the level of ten micrograms. Or maybe
5 it's all but ten. I'm not exactly sure what the
6 break point would be, but ten would be the current
7 level of concern. All of what they considered
8 normal had blood-lead levels higher than that, with
9 the average being oh, I'd guess -- would look like
10 it would be somewhere around 30, 35, something like
11 that.

12 THE COURT: When you say 30, 35, 30 to 35
13 what?

14 THE WITNESS: Micrograms per deciliter of
15 whole blood.

16 BYMR. SCOTT:

17 Q Let me clarify this, Doctor, in response to the
18 Court's question. The measurement in this table as
19 of 1942 is expressed in what fashion?

20 A In milligrams per hundred grams of blood. And a
21 gram of blood is a cc. 100 cc's is a deciliter,
22 and that's why -- that's how the nomenclature is
23 now.

24 Q Does that translate just by decimal points into
25 today's nomenclature?

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1 A Yes. Yes.

2 Q So this .08 milligrams per 100 what?

3 A Grams.

4 Q Grams, is 80 micrograms per deciliter.

5 A Micrograms per deciliter, right.

6 Q Now, there's a column -- there's a set of columns
7 over here called "Suspected Lead Poisoning." And
8 in children, focusing on children, at what levels
9 of blood lead did the Baltimore Health Department
10 confirm a lead poisoning diagnosis?

11 A So in that -- in the positive column there -- and
12 we're not looking at the adults for a moment, we're
13 looking at the children. You can see that the
14 lowest level that they confirmed as a positive case
15 was 80, and that most of the children who were lead
16 poisoned had blood-lead levels of between 100 --
17 that's the 10 -- the .1 column -- up to 300. And
18 then there were others that were still higher.

19 Q You're talking about here?

20 A Yes, that's the hundred. And then the next one is
21 200 to 300.

22 Q And you're saying that's where most of them were?

23 A That's where most of them were, yes.

24 Q And over here on the right there's a negative
25 clinical diagnosis. What does that refer to with

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1 specific reference to the children?

2 A What this represents is that when a child who had a
3 suspicious symptom came into the emergency room,
4 they were given the diagnosis of suspected lead
5 poisoning. And then, after the physicians did
6 their assessment, they came out with those who were
7 considered to be positive for lead poisoning and
8 those who were not. But they all got blood-lead
9 levels because they were of the suspicion of
10 blood-lead levels.

11 Q What is the blood-lead level of the children who
12 were -- whom they suspected of lead poisoning who
13 were found not to have it?

14 A Well, you can see there's a -- there's a range
15 there that goes from roughly 20 micrograms up to --
16 up to 80.

17 Q Doctor, from this table, are you able to tell us,
18 as of 1942, what was considered the beginning, the
19 threshold, blood lead for lead poisoning in
20 children?

21 A It was 80.

22 Q 80 micrograms per deciliter?

23 A 80 micrograms, yeah.

24 Q Now, you touched on this earlier. Did this kind of
25 data from the Baltimore Health Department allow an

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1 historian or a public health official to look back
2 on the earlier cases like those reported by Ruddock
3 and Blackfan?

4 A I think it does.

5 Q And can you make a judgment historically and
6 medically as to what levels of blood lead were
7 involved with children having the symptoms in those
8 cases?

9 MR. HAUSFELD: Objection. As an
10 historian, he can testify as to what was done by
11 others, but not what he does looking back.

12 THE COURT: The testimony of -- the
13 historical testimony we've heard in a variety of
14 contexts could have a number of different purposes.
15 The truth or accuracy of the underlying data that's
16 being referred to, the truth or accuracy of the
17 opinions that were reached back then, and thirdly,
18 what was the state of scientific understanding at
19 the time. And if it's necessary to isolate those
20 various purposes and give limiting instructions,
21 I'll consider that. I'm not sure as a practical
22 matter that can be done. I'll sustain the
23 objection, though, and ask you to make clear what
24 you're asking this witness to opine about.

25 BYMR. SCOTT:

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1 BYMR. SCOTT:

2 Q Let's come to the American Academy of Pediatrics in
3 just a moment. But you also said that Dr. Anna
4 Baetjer did something with this report after she
5 got it?

6 A Anna Baetjer sent it to Katherine Bane who was a
7 pediatrician in the U.S. Public Health Service. So
8 what that did, it brought this new concept to the
9 attention of the Public Health Service. This was
10 in 1951. And after receiving that, the U.S. Public
11 Health Service decided to invite Huntington
12 Williams to publish his findings, his emerging
13 findings, in their journal, Public Health Reports,
14 which is the -- one of the leading, if not the
15 leading, public health journals in this country.

16 Q I have Exhibit 602 on the screen from the Public
17 Health Reports. Is this the journal that you were
18 referring to --

19 A Yes.

20 Q -- from the Public Health Service?

21 A Yes, it is.

22 Q And is this a -- tell us what this is.

23 A Well, this is -- this is his new conclusions that
24 he's reaching, but it's also a paper I think that
25 shows the transition. In other words, remember, he

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1 had presided in many ways over the old consensus.
2 And we can see him moving to a new consensus here
3 in this 1952 paper.

4 Q Let's look at just a couple of excerpts from this
5 paper. In the first paragraph, he writes that
6 "Although 19 different communities are mentioned,
7 most of the cases were from large cities such as
8 Baltimore and Boston, where children's hospitals or
9 local health authorities were especially interested
10 in the problem."

11 Does Dr. Williams and his co-authors
12 do anything here to pull together the existing
13 reports from around the country?

14 A Yes. I think that the first -- what is it -- it
15 was the second to 37th footnote there are most of
16 the case reports of childhood lead-paint poisoning
17 in this country.

18 Q And then you were talking just a moment ago about
19 showing the transition. Going to the second
20 paragraph, I believe this may help you show what
21 Dr. Williams was talking about. Would you explain
22 these -- what he was talking about --

23 A Right --

24 Q -- in going from the old consensus to the new one.

25 A Well, he says here, "The most common cause of lead

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1 poisoning is apparently the habit of chewing paint
2 from cribs, toys, furniture, and woodwork, such as
3 windowsills." That's the old consensus. And then
4 he adds, "and the eating of painted plaster and
5 fallen paint flakes." And then he continues, "The
6 tendency to put things in the mouth, though normal
7 in the first year of life, is considered abnormal
8 if continued into the latter part of infancy and is
9 referred to as pica, or perverted appetite. Pica
10 is the usual forerunner of lead poisoning."

11 So he's continuing that concern
12 about pica. But the main thing about the source of
13 lead is that after the comma, "and the eating of
14 painted plaster and fallen paint flakes." That's
15 what's new.

16 Q Does Dr. Williams in this article report the
17 housing conditions where this is occurring?

18 A He does.

19 Q Looking at page 233 of Exhibit 602, Column 1.
20 Okay. Would you point out to us, Dr. English, what
21 is reported in this article about the kinds of
22 housing where this is happening.

23 A "For the past" -- "For the past few years,
24 information has been collected on home ownership in
25 neighborhoods where child lead-poisoning cases

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1 occurred. Almost 90 percent of the houses were
2 tenant-occupied. In the early years of the study,
3 some of the cases arose in well-kept property, but
4 with the continued community education by press and
5 radio, cases in this category have become
6 relatively rare. The problem in Baltimore at
7 present involves chiefly slum or blighted-area
8 properties. The cases are concentrated in two
9 areas which are of known slum status and where the
10 houses are old and have had many coats of paint,
11 usually lead paint, applied throughout several
12 decades," a typical home where a lead case occurs
13 is in Figure 2.

14 Q And what consequences followed publication of
15 Dr. Williams' article in 1952?

16 A Well, he's describing a -- the -- he's showing
17 that -- a new consensus of where children are
18 getting lead poisoned. And that's -- that's part
19 of the new consensus. That's one part.

20 Q Were there more publications in the 1950s
21 concerning the kind of problem that Dr. Williams
22 describes in Baltimore?

23 A Yes. There were. And, in fact, I look at this
24 article by Dr. Williams as sort of like a case
25 report. In other words, he -- he's showing a

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1 was to Randolph Byers. And the grant of \$10,000 to
2 the Johns-Hopkins Hospital, that was a Julian
3 Chisolm study that was not published until 1956.

4 Q Harvard is Dr. Byers, B-Y-E-R-S?

5 A Yes, B-Y-E-R-S.

6 Q And Johns-Hopkins, Dr. Chisolm?

7 A Yes.

8 Q Would those physicians proceed to publish in the
9 1950s?

10 A Yes. Byers went on to publish in the 1950s at
11 least three more studies, and Chisolm began a
12 career, a very distinguished career, in the field
13 of childhood lead poisoning. That would be his
14 first paper in 1956. And he continued publishing
15 into the 1980s.

16 Q What else was he --

17 A Maybe even into the early 1990s. He may have one
18 paper into the 1990s.

19 Q Just briefly, what thing was the LIA doing in
20 response to these reports?

21 A Another thing they were doing -- this was another
22 way -- another clearinghouse activity. There was a
23 new medication, a way of treating quite
24 successfully childhood lead poisoning. And again,
25 as it -- you know, as hospitals around the country

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1 were using this drug for the first, second, and
2 third time, so before they would be actually
3 publishing it, he would keep -- Bowditch would keep
4 track of the results. And this was a way of
5 communicating this new therapy to other doctors in
6 advance of being published. These -- these
7 publications began, I think -- oh, I think there
8 may have been one in '52, a couple in '53, but most
9 of them are after 1954.

10 Q Doctor, let us not take more time on Exhibit 38,
11 because the jury has seen it before in individual
12 paragraphs and it's in the record.

13 A Okay.

14 Q Let's move on to what you called the new public
15 health consensus in 1955. What was the new public
16 health consensus?

17 A Okay. There are -- there are two prongs to this
18 new consensus. The first consensus is what I call
19 the old paint problem -- consensus or problem. And
20 that is the identification of -- that the new
21 source of lead poisoning in children came from
22 dilapidated houses built before 1940 where lead
23 paint had been put on over many years and many
24 coats that had come under poor maintenance and
25 then, therefore, come under disrepair. So the

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1 edification of the cite had been pointing at
2 dilapidated, pre-1940 houses.

3 The second part of that, of the old
4 paint problem, was that it was -- do you want me to
5 deal with the responses to it?

6 Q Let's hold off for one second.

7 A Okay.

8 Q In this slide you created, "New Problem: Peeling
9 and flaking interior paint in dilapidated housing,"
10 and I believe you're quoting from one of the
11 writers in the field there?

12 A Yes, I believe that's Chisolm.

13 Q And what was the representation with respect to the
14 old paint that was already on the walls?

15 A First of all, recognize the hazard. So they had to
16 educate the public on what the hazard was. And
17 then the hazard had to be -- had to be addressed,
18 which meant the peeling and flaking paint simply
19 had to be cleared away.

20 Q And what was the other part of the problem and the
21 recommendation in 1955 with respect to new paint?

22 A With new paint. And that was that what should
23 happen with -- with the new paint that was going to
24 be applied on these walls, or even walls on new
25 houses. And that was -- and that was -- that was a

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1 new standard, a voluntary standard, had to be
2 achieved, which culminated in 1955 with a 1 percent
3 standard, 1 percent of lead content in paint
4 standard.

5 Q How did the new 1 percent standard get put in
6 place?

7 A As I -- as I mentioned, Manford Bowditch had -- had
8 gotten George Wheatley of the American Academy of
9 Pediatrics interested in this problem, and so
10 Bowditch brought George Wheatley and the American
11 Academy of Pediatrics together with the American
12 Standards Association. And this is an organization
13 that sets appropriate standards for a whole variety
14 of consumer products. Got them together to discuss
15 what a -- a safe level of interior paint, what a
16 safe concentration, what a safe amount of lead in
17 interior paint would be. And so these were the
18 umbrella organizations, the LIA, the American
19 Academy of Pediatrics and American Standards
20 Association, but they had a much broader committee
21 that came together to discuss these issues in 1953
22 and 1954.

23 Q All right. And when was the new standard issued?

24 A It was issued in 1955.

25 Q I'm showing you Exhibit 601. What is this, Doctor?

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1 A This is the publication of the standard. So you
2 can see the sponsor is the American Academy of
3 Pediatrics.

4 Q And who approved it?

5 A It was approved by -- first of all, by the
6 subcommittee that came up with the standard, and
7 then by the -- the larger group.

8 Q Let's look at the larger group. What organizations
9 participated in this effort?

10 A Okay. We've already mentioned the American Academy
11 of Pediatrics, and George Wheatley there is serving
12 as chairman. And then as we go down the list, you
13 can see a whole variety of organizations, the
14 American Hospital Association, the American Medical
15 Association. There were business groups like the
16 Petroleum Institute; the American Public Health
17 Association; the American Society for Testing
18 Materials, of course they would need that; some
19 insurance companies; the Boy Scouts; the Child
20 Study Association, that's child psychiatry; HEW was
21 Department of -- that was the precursor of Health
22 and Human Services, so the federal government was
23 involved; the Food and Drug Administration; Good
24 Housekeeping. There are quite a few that -- then
25 some of the organizations, the Lead Industry

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1 did any of the groups involved in the development
2 of this standard oppose it in 1955?

3 A No, the --

4 Q I want to move forward to the beginning of the
5 1970s, Dr. English, and ask you first, was there a
6 change in the public health consensus in America
7 about childhood lead-paint poisoning between 1955
8 and 1970?

9 A No.

10 Q Did there come a time when federal law was put in
11 place dealing with lead house paint?

12 A Yes. In -- in 1971 for the first time.

13 Q And you have a -- a slide to summarize the key
14 dates --

15 A I do.

16 Q -- of the federal work? What happened in 1971?

17 A 1971, the federal government passed a law that said
18 that 1 percent paint had to be used on all federal
19 housing.

20 Q That's limited to federal housing now?

21 A Federal housing, yes.

22 Q Was there any federal law enacted concerning homes
23 in the country, private homes?

24 A No, not at this point.

25 Q When did that happen?

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1 A That happened the following year. The jurisdiction
2 for the -- this was shifted to the Food and Drug
3 Administration, and in 1972, they listed -- they
4 had a ruling that -- that pertained not only to
5 federal housing but to all housing, that lead
6 should not have -- the lead content should be less
7 than a half a percent.

8 Q I know during this trial we and the Court and the
9 jury have heard these different dates, 1972, 1978.
10 What happened in 1978?

11 A 1978, the jurisdiction shifted again to another
12 federal agency, it's the Consumer Product Safety
13 Commission. But in 1978, the Consumer Product
14 Safety Commission limited the concentration of lead
15 in all household paint to 0.06 percent.

16 Q Dr. English, looking back from the 1970s, from
17 these laws, back to the early decades of the 20th
18 century, standing there in 1971 looking back, have
19 you found historical evidence that any agency of
20 our federal government opposed the use of lead
21 house paint, leaded house paint, before these dates
22 on the screen?

23 A I have not found. In fact, quite to the contrary,
24 they recommended the use of lead paint.

25 Q Which agencies recommended the use of lead paint

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1 over those years?

2 A One was the National Bureau of Standards.

3 Q When did they start recommending lead paint?

4 A I found recommendations for lead paint that go back
5 at least until 1924.

6 Q And let's look at Exhibit 939. Can you tell us
7 what this is, Doctor.

8 A Yes, this is one of the standards. This is T 274,
9 which was for the use of paints, and this
10 particularly suggested the use of lead-based paint.

11 Q And the agency, again, that issued this, the
12 National --

13 A The National Bureau of Standards.

14 Q Bureau -- and who were the authors? Who worked for
15 the government?

16 A Percy Walker and E.F. Hichson.

17 Q In your research, have you found their names over a
18 period of years?

19 A Yes. They continued to be involved into the '30s
20 and into the '40s.

21 Q Did the National Bureau of Standards of the federal
22 government recommend lead paint to people outside
23 the federal government?

24 A They did. So it was both a regulation of what the
25 government should purchase on government buildings,

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1 and then when people would write in, such as a
2 school system or something, or a hospital, they
3 would recommend the use -- usually we would refer
4 them to a -- a standard like this one here, the
5 T 274, which encouraged the use of lead paint.

6 Q I'd like you to look at Exhibit 559, which is
7 September 1928. And the second page bears the
8 signature of Percy H. Walker. What is this
9 document?

10 A This is a letter that Percy Walker wrote to the
11 Board of Education in New York in response to a
12 letter basically what should we paint our schools
13 with.

14 Q And did Walker of the government answer that
15 question what they should paint the schools with?

16 A Yes. He advised using lead paint.

17 Q Would you read for us the final paragraph on the
18 first page.

19 A "In regard to the painting procedure, it is noted
20 that you propose to use lead-in-oil paint for the
21 second and third coats. There is nothing better
22 that can be used for these coats. The
23 specification for the first or primer coat is
24 indefinite. There is no reason why if lead-in-oil
25 is used for the second and third coats, that it

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1 should not also be used for the priming coat."

2 Q Are you familiar with the next document,
3 Exhibit 592?

4 A Yes. This is a paint manual with all the painting
5 specifications of the National Bureau of Standards
6 that was published in 1945, just after the war.

7 Q 1945, again by Walker and Hichson?

8 A Yes, the same -- the same men.

9 Q And the agency is the National Bureau of Standards
10 in the U.S. Department of Commerce?

11 A Correct.

12 Q Okay. What kind of paint did they recommend?

13 A Lead paint still.

14 Q On page 18 of this exhibit, can you confirm that
15 this is one of the pages of this 1945 government
16 paint manual?

17 A It is.

18 Q What kind of paint do they recommend for indoor
19 use, Doctor?

20 A Lead paint.

21 Q What kind do they recommend for exterior use?

22 A Lead paint.

23 Q Did any other agencies of the U.S. government --
24 we're here -- 1970s looking back over those
25 decades. Any other agencies of the federal

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1 government recommend that folks use lead-in-oil
2 paint?
3 A Yes, the Forest Products Laboratory.
4 Q We've heard about them before. They've always been
5 over in Madison?
6 A I believe so.
7 Q If you look with me at Exhibit 542. And what is
8 this magazine?
9 A This is a publication of the -- of the Forest
10 Products Laboratory.
11 Q And there's a chapter in here --
12 A "Shopping for Paint."
13 Q Right here, okay. Just an overview, what does the
14 agency recommend to consumers in this -- let's see,
15 what year this?
16 A I think it's '39, '37. I'm not -- I can't recall
17 precisely.
18 Q We've got February 13, 1939 here. Does that sound
19 right?
20 A '39, okay.
21 Q Yeah, what do they recommend?
22 A They recommended the use of lead paint.
23 Q Going over to what's called page 6, going through
24 the paints, would you read U.S. Forest Products'
25 recommendation to the consumer -- to the consumer

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1 in 1939?

2 A "The pigment consists entirely of white lead except
3 for additions of colored pigments that" -- "that
4 make the" --

5 Q I'm sorry, it's in the fold of the magazine. This
6 is the only copy. "This paint"?

7 A "This paint, if used exclusively, remains the best
8 choice for homeowners who" --

9 Q "House owners." I think it's house.

10 A Yeah, "of house owners who wish to allow very long
11 intervals, longer than the durability of any other
12 white or tinted paint, to" -- and I can't make out
13 that word. I might be able to see it actually in
14 the smaller.

15 Q "Elapse"?

16 A Yeah -- "elapse between paint jobs."

17 Q Now, this is 1939. We saw the National Bureau of
18 Standards paint manual in 1945. When Huntington
19 Williams issued his ban on interior lead-based
20 paint in Baltimore City in 1951, what did the
21 federal government do in its specifications?

22 A They didn't -- they didn't change. They continued
23 to recommend the use of lead-based paint.

24 Q 1954 -- just remind us, in 1954, what is Manford
25 Bowditch of LIA doing with the American Academy of

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1 Pediatrics?

2 A What he's doing then is he's serving as the
3 secretary of the subcommittee that's developing the
4 1 percent standard for interior paints in this
5 country.

6 Q Let's look at Exhibit 544. What is this document,
7 sir?

8 A This is a specification for paint from 1966.

9 Q All right. This is a cancellation. Do you see
10 that?

11 A Yes.

12 Q If we go to the attached document, what is the date
13 of the issuance?

14 A Oh, that's right, it's -- it's February 23rd, 1954.

15 Q What kind of paint was the federal government
16 recommending for interior wall surfaces in this
17 specification of 1954?

18 A Lead paint.

19 Q TT-W-251?

20 A Right, basic carbonate of lead. That's basic white
21 lead paint.

22 Q When the government canceled that specification in
23 1966, what was the new one they issued for interior
24 house paints?

25 A There was a new one, that came out in -- following

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1 1967, which permitted the use of lead paint, it
2 didn't -- as long as it was labeled as lead paint.

3 Q It's Exhibit 538 issued in 1967. Is this the one
4 you mean?

5 A Yes.

6 Q You say it didn't require lead paint but what?

7 A But permitted it.

8 Q And did the government say what needed to be done
9 on lead paint if it was used?

10 A It had to have a label indicating that it had lead
11 paint in it -- lead in it.

12 Q Going to page 6 of this specification.

13 A Yes.

14 Q What was the government's requirement for interior
15 paint?

16 A "Paints containing lead pigment shall be marked on
17 each container with the following: Contains lead
18 pigment."

19 Q That's 1967?

20 A That's 1967.

21 Q Four years before the first ban on your list here?

22 A That is correct.

23 Q What was the government doing on specifications for
24 exterior paint after Baltimore in 1951?

25 A They continued to recommend lead paint.

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1 Q Exhibit 534. Exterior, 1965, what did the
2 government specify?

3 A Lead paint.

4 Q TT-W-251?

5 A That's correct.

6 Q Going back now to the early 1970s, we have seen
7 what the laws were at that point. When these laws
8 were enacted in the early 1970s by the federal
9 government, did the public health community, the
10 experts at that time, view the risks of lead paint
11 as we do today?

12 A Oh, absolutely not.

13 MR. HAUSFELD: Objection.

14 THE WITNESS: Quite differently.

15 THE COURT: Hold on, there's an
16 objection.

17 MR. HAUSFELD: Foundation.

18 MR. SCOTT: Based on his research.

19 THE COURT: Just a moment. Overruled.
20 You may answer.

21 THE WITNESS: No, they did not view it in
22 the same way. They viewed it in a considerably
23 different way.

24 BYMR. SCOTT:

25 Q I would like you to look at Exhibit 619, 6-1-9.

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1 What is this article, sir?

2 A This is an article published by a group of leading
3 lead researchers which aimed to calculate how much
4 lead a child could consume on a daily basis and be
5 considered safe.

6 Q Let's pause on the -- who is writing this. It's
7 under the name of Mr. Barry G. King, Ph.D. Who was
8 King?

9 A He was a researcher at Cincinnati.

10 Q And this was reporting from a committee?

11 A Yes. And you can see that there's government
12 Department of Health and Human Services, there's
13 Julian Chisolm from Johns-Hopkins, there's Jane
14 Lin-Fu, and she was a -- an important health
15 educator and pediatrician of the maternal health
16 services of HEW. So this was a -- this was a broad
17 group of experts.

18 Q Did this broad group of experts in 1971 take the
19 position that any intake of lead by a child was
20 harmful?

21 A Oh, no. What they did was they -- they determined
22 that a child could consume up to 300 micrograms of
23 lead every day and still fall under the blood-lead
24 level of 40, which was the federal government's
25 level of concern in 1970.

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1 Q You've spoken about the public health consensus in
2 1955. Was there ever a new public health consensus
3 after that on the nature of childhood lead-paint
4 poisoning?

5 A The -- the next one was in 1978.

6 Q 1978.

7 A '8, yes.

8 Q Let's look again at -- this is when the -- I'll
9 call the third law, but the 1978 version, there was
10 already a law about lead in house paint generally,
11 but this dropped the limit.

12 A Right. That was one of the components of the
13 public health response to a newer -- to the -- in
14 1978.

15 Q But what was new in 1978, Dr. English?

16 A A couple -- three things, I think. One was that
17 the level of concern of lead in the blood dropped
18 from roughly 60 at the beginning of the -- of the
19 decade in 1970 to 30 in 19 -- a report from the CDC
20 in 1978.

21 The second difference was that for
22 the first time, they recognized that there were
23 some case reports, the first one was in 1974, that
24 household dust might be an additional source of
25 lead of -- of -- for children. And that was an

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1 STATE OF WISCONSIN)
2) SS
3 MILWAUKEE COUNTY)
4

5 I, JULIE K. LYLE, RPR/RMR/CRR, Notary Public
6 in and for the State of Wisconsin, do hereby certify
7 that the foregoing proceedings were recorded by me and
8 reduced to writing under my personal direction and that
9 it is a true and correct transcript of said proceedings
10 had in said matter to the best of my knowledge.

11 In witness whereof, I have hereunto set my
12 hand at Milwaukee, Wisconsin, on this 11th day of June,
13 2007.
14
15
16
17

18 _____
19 JULIE K. LYLE
20
21
22
23
24
25

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Transcript of Proceedings - 6/12/2007

Vol. 16

1 STATE OF WISCONSIN : CIRCUIT COURT : MILWAUKEE COUNTY

2 -----

3 CITY OF MILWAUKEE,
4 a municipal corporation,

5 Plaintiff,

6 vs.

Case No. 01-CV-3066

7 NL INDUSTRIES, INC.,
8 a foreign corporation,

9 Defendant.

10 Before the Honorable

11 JOHN FRANKE, Circuit Court Judge

12 Branch 25, presiding

13
14 Tuesday, June 12, 2007

15 VOLUME 16

16 9:30 a.m.

17 A P P E A R A N C E S:

18 MICHAEL HAUSFELD, RICHARD LEWIS, RICHARD SERPE, TERRY
19 NILLES, Attorneys at Law, SARA SCHUBERT, appeared on
20 behalf of the Plaintiff.

21 JAMES T. MURRAY, JR., DONALD SCOTT, ANDRE PAUKA,
22 Attorneys at Law, appeared on behalf of the Defendant.

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1 aside the issue of lead paint on cribs, toys, and
2 furniture, were there any other public reports in
3 Milwaukee about lead toxicity?
4 A They're scattering. Again, they tend to be
5 industrial concerns. There's a few hazards that the
6 state public health authorities investigate at the
7 request of the City of Milwaukee. One has to do with
8 whether or not the welding in a dairy apparatus may
9 have lead in it. And the state looks into that and
10 makes a decision that's not true, and that's
11 reported.

12 There's also a concern briefly in the
13 1940s about whether chalk being used in schools has
14 lead in it. And that's looked into by the state
15 public health officials, and they decide that it's
16 not a hazard, and that's reported as well.

17 But apart from the consistent
18 reporting of the toy hazard and the -- I have to
19 say -- I don't want to say it's regular, but there's
20 a consistent story of industrial poisonings that
21 continue, although much less frequent than the
22 earlier part of the century. That's all that's being
23 mentioned up through the 1940s and early '50s.

24 Q Dr. Sharpless, I would like to move on to a different
25 topic. It's the third area that you researched.

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1 What did you learn about the -- or
2 strike that.

3 What -- what did you learn about the
4 use of lead paint by government agencies in
5 Milwaukee?

6 A I looked at specifications and purchasing for the
7 City of Milwaukee's public agency's specifications
8 for various buildings, museums, libraries, buildings,
9 on playgrounds, courthouses. And the specifications
10 would be issued for public bids for construction or,
11 in some cases in the 1930s, when the city was
12 actually building some of its own buildings during
13 the Depression, specifications for its own use on the
14 building. And these specifications go into detail on
15 every aspect of construction, electricity, plumbing,
16 but they also go into detail on the paint that's to
17 be used, both interior and exterior.

18 Q What types of paints were specified?

19 A Rather consistently from 1900 up to the 1960s, lead
20 paint is being specified in one way or another.

21 Q In what types of structures was lead paint specified
22 to be used?

23 A For nearly every conceivable public building:
24 Schools, libraries, museums, play pavilions on
25 playgrounds, hospitals.

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1 Q I would like to display for Dr. Sharpless
2 Exhibit 586.

3 Dr. Sharpless, can you identify 586?

4 A Yes, this is -- this is a typical building spec for
5 the City of Milwaukee Department of Public Works.
6 This is 1929, and it's for the Johnston Hospital.

7 MR. PAUKA: Your Honor, may I display it to
8 the jury?

9 THE COURT: Any objection?

10 MR. SERPE: No objection, Your Honor.

11 THE COURT: Go ahead.

12 MR. PAUKA: Your Honor, I apologize. I did
13 forget to ask to move into evidence Exhibit 610,
14 which is the Lullaby Furniture circular. May that be
15 moved into evidence?

16 THE COURT: It's received. I don't know
17 that anyone cares about all the other stuff in the
18 Lullaby Furniture catalog and whether it just needs
19 to be restricted to the portion published, but I'll
20 receive it to the extent published.

21 MR. SERPE: Your Honor, all objections to
22 that exhibit are noted.

23 MR. PAUKA: Thank you, Your Honor. Please
24 display 586 for the jury, please.

25 Q Dr. Sharpless, I'll start at the top here. And can

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1 of a government, down to 10 crayons for 50 cents
2 purchased in January of 1923. Paste --
3 Q Can I interrupt you there, Dr. Sharpless, and display
4 for Dr. Sharpless Exhibit 928.
5 Dr. Sharpless, what is Exhibit 928?
6 A That's -- actually, that's just a typical front page
7 out of the whole series. Proceedings of the board of
8 school directors, Milwaukee, Wisconsin.
9 Q And are these the minutes of the proceedings that you
10 found in your research?
11 A They're not only the minutes of the school board
12 itself, they're the minutes of the subcommittees. And
13 there are the detailed monthly expenditures of the
14 school board.
15 MR. PAUKA: Your Honor, may I display for
16 the jury?
17 MR. SERPE: No objection.
18 BY MR. PAUKA:
19 Q And I interrupted you when you were telling us what
20 type of information is found in these minutes.
21 A The level of detail in these monthly reports is
22 astonishing. Clearly, the school board wanted
23 everybody in the city to know that they were spending
24 the last nickel properly. And literally down to
25 boxes of crayons are listed here by item, by vendor,

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1 and by expenditure.

2 Q How many pages of these proceedings did you find in
3 your research?

4 A A couple thousand.

5 Q I had not put 1,000 pages plus into this document.
6 I've only taken 10 pages out. I would like to go
7 through some of those pages now.

8 And before we get there, what did you
9 discover about the school board's use or purchase of
10 white lead?

11 A For 50 years, they regularly purchased the
12 ingredients for the mixing of lead paint. They
13 purchased white lead and oil in bulk, turpentine and
14 mineral spirits in bulk, and tints and additions to
15 the paint in bulk.

16 Q You said for 50 years. Was this every year, or how
17 frequently did they make the purchases?

18 A There's a purchase of some sort just about every
19 year. There are some years where there are
20 exceptionally large purchases and they -- when I read
21 through the minutes, they seem to be associated with
22 the building of a new school or the renovation of a
23 school. So they would purchase more in years where
24 they were going to build something.

25 Q I'm going to the second page of this exhibit. Can

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1 you tell us the date of this document.

2 A 1902.

3 Q That was April 8th, 1902?

4 A Yes.

5 Q I'm going to go down to line 254, and I will enlarge
6 that.

7 Can you tell us what is reported in
8 line 254?

9 A In line 254 is the name of the vendor. Patek was a
10 local vendor of paint products in the city of
11 Milwaukee. And it tells the item purchased, white
12 lead and oil. And it gives the amount that's being
13 spent on that purchase.

14 Q How much was spent?

15 A \$212.04.

16 Q Did you see other purchases of white lead by Patek?

17 A Yes.

18 Q Approximately how many?

19 A Early on in the century, four, five, six. They seem
20 to fall out of competition some place in the 1920s,
21 cease to appear in the '30s.

22 Q Let's move to the next page of the exhibit, which --
23 can you tell us what the date of this exhibit is?

24 A May 7th, 1912.

25 Q I'm going to go down to line 589 and enlarge -- I

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Vol. 16

1 STATE OF WISCONSIN)
) SS
2 MILWAUKEE COUNTY)

3

4 I, SARAH A. HART, RPR/RMR/CRR, Notary Public in
5 and for the State of Wisconsin, do hereby certify that the
6 foregoing proceedings were recorded by me and reduced to
7 writing under my personal direction and that it is a true
8 and correct transcript of said proceedings had in said
9 matter to the best of my knowledge.

10 In witness whereof, I have hereunto set my hand
11 at Milwaukee, Wisconsin, on this 12th day of June, 2007.

12

13

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16

SARAH A. HART

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Vol. 18

1 STATE OF WISCONSIN : CIRCUIT COURT : MILWAUKEE COUNTY

2 -----
3 CITY OF MILWAUKEE,
4 a municipal corporation,

5 Plaintiff,

6 vs.

Case No. 01-CV-3066

7 NL INDUSTRIES, INC.,
8 a foreign corporation,

9 Defendant.

10 Before the Honorable

11 JOHN FRANKE, Circuit Court Judge

12 Branch 25, presiding

13
14 Thursday, June 14, 2007

15 VOLUME 18

16 9:24 a.m.

17 A P P E A R A N C E S:

18 RICHARD LEWIS, RICHARD SERPE, CHRIS RIORDAN,
19 Attorneys at Law, SARA SCHUBERT, appeared on behalf of
20 the Plaintiff.

21 JAMES T. MURRAY, JR., DONALD SCOTT, JOHN
22 HUGHES, Attorneys at Law, appeared on behalf of the
23 Defendant.
24
25

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1 KATHY: 959.

2 MR. SCOTT: 959? Okay. For the record.

3 THE COURT: I just need the exhibit
4 numbers again. And the list that I have doesn't
5 include the 510, 511. And I think there was a
6 third exhibit you started with.

7 MR. SCOTT: Yeah, 207 is one report of
8 the Battelle results. That was introduced probably
9 back during Dr. Foldy. As displayed it was
10 introduced.

11 THE COURT: I believe portions of this
12 have been received.

13 MR. SCOTT: Yes.

14 THE COURT: What about 510?

15 MR. SCOTT: 510 --

16 THE COURT: Has that been received in
17 part?

18 MR. SCOTT: Yes, I think it may have been
19 used under a different number, for which I
20 apologize. We'll have to coordinate on that. It
21 may have had plaintiff's number on it at that time.

22 THE COURT: Okay. You may proceed.

23 MR. SCOTT: I'll straighten that out
24 while Dr. Magee is here.

25 Q Okay. Now, Dr. Magee, could you tell us first what

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- 1 information was being sought in these three
2 studies -- no, let me start with a different
3 question. Who sponsored these studies?
- 4 A These studies were sponsored by HUD, and the
5 Battelle study was HUD and the City of Milwaukee
6 and I believe another agency, CDC maybe. I forgot,
7 I think there are three logos on the front page.
8 The HUD 14 Grantee study is HUD for sure, and the
9 HUD did the six-year follow-up study.
- 10 Q So they're all HUD sponsored. Do any or all of
11 them contain data for Milwaukee housing?
- 12 A Yes, the Battelle study is solely a study of the
13 Milwaukee program. The 14 Grantee study is a more
14 general, larger study, and Milwaukee was one of the
15 14 HUD grantees.
- 16 Q And in general what kind of information was HUD
17 seeking in sponsoring these studies?
- 18 A In all of these studies, they were seeking to
19 determine whether various treatment or abatement
20 options in homes would or would not be successful
21 in reducing children's blood-lead levels.
- 22 Q Now, to review the three conclusions that you've
23 arrived at, what is the first one, please, based
24 upon the three studies?
- 25 A This is based on all three of them. Window

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1 treatments do not reduce children's blood-lead
2 levels more than paint stabilization and cleaning.
3 I.e., there was no difference between the various
4 treatment options that included or did not include
5 window treatments.

6 Q And the second one?

7 A The window treatments are very effective at
8 reducing window dust lead levels.

9 Q Is that where children are found to be getting the
10 blood lead?

11 A Well, that's --

12 MR. LEWIS: Your Honor, object to
13 leading.

14 THE COURT: Hold on. Sustained.

15 BY MR. SCOTT:

16 Q Did they have a finding on where children are
17 getting blood lead or what's correlated with their
18 blood lead in these studies?

19 A Yes, they did.

20 Q And what was that?

21 A That is floor dust levels are correlated with
22 children's blood-lead levels in all of these
23 studies, but floor dust levels are not affected by
24 window dust levels. So you can work on the
25 windows, and it will do a great job at reducing the

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1 dust on the windows, but it doesn't improve
2 children's blood-lead levels because that is
3 affected by what's on the floor, not what's on the
4 window.

5 Q In these documents, do the writers express surprise
6 over what they find?

7 A Yes, there are quite a few statements where they
8 say, our anticipated outcome was not found or we
9 were surprised to find that dot, dot, dot.

10 Q Begin with Battelle, and would you describe for us
11 briefly what the -- what the nature of this study
12 was.

13 A Yes. Now, Battelle is a study that is specifically
14 about Milwaukee. It's a study of the pilot
15 ordinance that was carried out in the late '90s, I
16 guess, which was specifically focusing on the types
17 of abatement procedures that they were carrying
18 out. So this is a Milwaukee-only study, and it was
19 broken into two substudies that I'm sure you'll ask
20 me to talk about.

21 Q I will. The portion I've highlighted on page 5 of
22 the exhibit refers to a Milwaukee ordinance. Is
23 that the pilot ordinance you just mentioned?

24 A Yes, that's what the document says very clearly in
25 the first couple of pages.

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1 you're away from the microphone now, so please keep
2 your voice up.

3 THE WITNESS: This is the north target
4 area, this is the south target area. This is more
5 or less a cartoon. We don't know exactly what
6 streets these are. We wanted to just show you
7 there was a particular part of the north target
8 area that they designated the treatment area,
9 houses that received the treatments.

10 BY MR. SCOTT:

11 Q Let's pause on that. The treatments are what?

12 A The treatments include some general maintenance and
13 some window work.

14 Q Pursuant to the Milwaukee ordinance?

15 A Milwaukee pilot ordinance.

16 Q Go ahead, please.

17 A In the south they did the same thing. There was
18 part of the south area that was defined as the
19 treatment area that received the treatment. And
20 the control area was a portion of the neighborhood
21 that would not receive the treatment.

22 Q And what were the findings in the first of the
23 studies they called general screening?

24 A Briefly, Phase I and Phase II simply means before
25 and after the treatment. So we're looking at the

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1 blood-lead levels of the children in each of these
2 four areas before and after. Before and after.

3 In the north, we actually see that the
4 control group did better at reducing the blood-lead
5 levels. So at best, there's no difference, but, in
6 fact, this was a statistically significant
7 difference. Not doing anything caused the
8 children's blood-lead levels to go down more than
9 doing the treatment. In the south, there was no
10 difference when you did the statistical test.

11 Q They were declining everywhere, though?

12 A They were declining everywhere, not different
13 statistically in the south. The control group was
14 statistically better than the treatment group in
15 the north.

16 Q Go back, please. Going back to Exhibit 207 in the
17 report. That's page 8 of the document itself. In
18 the north neighborhoods, what was the conclusion
19 for the north neighborhoods?

20 A I'll just read directly. "The effects of
21 neighborhood-wide lead-poisoning prevention
22 treatments in reducing geometric means of
23 children's blood-lead levels or the proportion of
24 children with elevated blood-lead levels were not
25 statistically significant."

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- 1 Q You said that the -- this is talking about the
2 prevention treatments compared to the control is
3 not statistically significant. Was there -- in
4 fact, when you flip it around, the controls
5 compared to the treatment groups, was there a
6 significant improvement?
- 7 A There was a significant improvement. That's in
8 another bullet in the document.
- 9 Q In the south neighborhoods, what was the conclusion
10 from the general screening study?
- 11 A Again, I'll just read their conclusions. I'm just
12 reporting what they are reporting. "The effects of
13 lead-poisoning prevention treatments in reducing
14 geometric means of children's blood-lead levels or
15 proportion of children with elevated blood-lead
16 levels were not statistically significant."
- 17 Q Now, there was a second study within the same
18 document, was that right?
- 19 A Yes, that's correct.
- 20 Q And going back to the paragraph we had up earlier,
21 that's called the "Prospective Longitudinal Study."
22 What was that?
- 23 A This is a study where they were not doing a
24 snapshot of simply before and after, but they were
25 going to take the before measurement, do the

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1 treatments, or not, in the control group, and then
2 follow children over time for up to 24 months, I
3 believe they did.

4 Q And what did they find in the prospective
5 longitudinal study?

6 A Well, they found essentially the same thing. They
7 found that the treatment did not significantly
8 reduce children's blood-lead levels to any greater
9 degree than not doing the treatment. So there was
10 no difference.

11 Q We're on page 26 of the study, page 30 of the
12 overall exhibit.

13 A Um-hum.

14 Q Let's take this one piece at a time, Doctor. The
15 first paragraph, first sentence, reads: "Although
16 the treatments conducted under the Milwaukee pilot
17 ordinance were relatively effective in bringing
18 window dust lead loadings below those in comparable
19 untreated units, the effects on children's
20 blood-lead levels did not meet original
21 expectations."

22 Did I read that correctly?

23 A You read that the way I see it, yes.

24 Q And how does that relate to the three conclusions
25 you put in the summary slide a few moments ago?

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1 STATE OF WISCONSIN)

2) SS

3 MILWAUKEE COUNTY)

4 I, JULIE A. POENITSCH, RPR/RMR/CRR,
5 Notary Public in and for the State of Wisconsin, do
6 hereby certify that the foregoing proceedings were
7 recorded by me and reduced to writing under my personal
8 direction and that it is a true and correct transcript
9 of said proceedings had in said matter to the best of my
10 knowledge.

11 In witness whereof, I have hereunto set
12 my hand at Milwaukee, Wisconsin, on this 14th day of
13 June, 2007.

14

15

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17

18

19 JULIE A. POENITSCH

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Vol. 21

1 STATE OF WISCONSIN : CIRCUIT COURT : MILWAUKEE COUNTY

2 -----
3 CITY OF MILWAUKEE,
4 a municipal corporation,

5 Plaintiff,

6 vs.

Case No. 01-CV-3066

7 NL INDUSTRIES, INC.,
8 a foreign corporation,

9 Defendant.

10 Before the Honorable

11 JOHN FRANKE, Circuit Court Judge

12 Branch 25, presiding

13
14 Tuesday, June 19th, 2007

15 VOLUME 21

16 9:16 a.m.

17 A P P E A R A N C E S:

18 RICHARD LEWIS, MICHAEL HAUSFELD, KATE KONOPKA,
19 and TERRY NILLES, Attorneys at Law, and AMY MURPHY,
20 appeared on behalf of the Plaintiff.

21 DONALD SCOTT, JAMES T. MURRAY, JR., JOHN
22 HUGHES, and ANDRE PAUKA, Attorneys at Law, appeared on
23 behalf of the Defendant.

24
25
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1 that would be stopped. If that meant telling
2 customers and school boards that the product was
3 safe, that's what would happen. That one goal was
4 followed no matter what happened, no matter what
5 their own advisors told them, no matter what the
6 science told them, no matter what they saw.

7 And that goal was followed until 1978
8 when it couldn't be followed anymore because lead
9 paint was banned. It was made illegal to sell
10 because the federal government found it was in the
11 public interest to ban that product to protect
12 children.

13 So the core question, after all this
14 time, after all these witnesses, the core question,
15 ladies and gentlemen of the jury, is was National
16 Lead's conduct acceptable? Was it acceptable to
17 respond to the lead-paint crisis in Milwaukee that
18 they created in the first place by selling that
19 product without any restrictions for all those
20 decades and promoting it as safe, was it acceptable
21 in Milwaukee to do nothing? Was it acceptable, and
22 is it acceptable today, for National Lead to take
23 no responsibility, none, zero, and instead, to
24 shirk any responsibility and to foist it off onto
25 moms and dads and pediatricians and landlords and

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1 health departments, to take no responsibility,
2 leave it to them, leave it to somebody else? And
3 what about the kids? Leave it to the kids.
4 They're the ones in the end of the day who walk
5 around with the damage.

6 They left it on many communities, they
7 left all that on the community in Milwaukee.

8 And as the judge instructed you
9 yesterday, responsibility is very important in this
10 case. The instruction that the judge reviewed on
11 what substantial factor means -- substantial factor
12 in creating the public nuisance talks about
13 responsibility, and that's important in all of
14 those questions, 2, 3, 4, and 5, when you must
15 deliberate and make a decision about the conduct of
16 National Lead, the responsibility of National Lead.

17 There was a table of responsibility in
18 every community. And in this community in
19 Milwaukee, when it comes to childhood lead-paint
20 poisoning, the parents have been sitting at that
21 table caring for their children. The pediatricians
22 have been sitting at that table. The Shonia
23 Zollicoffers from the Dominican center, organizing
24 in the community, have been sitting at that table.
25 Amy Murphy from the Health Department, since 1992,

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1 has been sitting at that table. Landlords have
2 been paying their share to make homes lead-safe,
3 sitting at the table.

4 There's an empty chair at the table, just
5 like that empty chair over there. National Lead
6 has never sat at the table of responsibility. This
7 is the first time, through our legal process, that
8 they've had to do that.

9 We ask you in your deliberations to look
10 at this conduct and decide, is it acceptable to
11 decide that it -- no, it's not acceptable. It's
12 unacceptable to foist this responsibility on
13 everyone else but yourself when you created the
14 product, created the market, and promoted the
15 product as safe all those years.

16 So the responsibility instruction is very
17 important, and so is the one that the judge
18 reviewed about causation. And the judge said
19 repeatedly yesterday, for all those questions, that
20 if the jury finds -- if you, the jury, find that
21 National Lead is a cause of an interference with a
22 public right, such as health, if you find that they
23 are a cause, then they have responsibility.

24 They don't have to be the only cause.
25 There can be more than one cause. But as the judge

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1 said for each question, if you find that National
2 Lead is a cause in creating the interference with
3 public health in Milwaukee that we call childhood
4 lead-paint poisoning, then they have to answer for
5 that.

6 And we ask you, when you consider all the
7 evidence, the fundamental evidence about what has
8 happened to children in this city and the knowledge
9 that this company had over the decades when they
10 promoted this product, that you return a verdict, a
11 judgment, about that responsibility, a judgment
12 that says that was unacceptable.

13 And you have to come to the table of
14 responsibility. We ask you to find in your
15 judgment a verdict for the City of Milwaukee and to
16 reimburse that Health Department in the amount of
17 the \$52.6 million that they've spent on this
18 problem alone from 1992 until the end of 2006.

19 I'd like to look for a moment at those
20 instructions that I just mentioned, starting with
21 the one about responsibility. Can we put that up?

22 As Your Honor said, this is when the
23 Court defined substantial factor, which is
24 something that the jury has to look at in
25 determining causation.

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1 So childhood lead poisoning and lead is
2 an extremely well-studied disease and well-studied
3 chemical. As the experts explained, there are more
4 than a thousand studies about what lead does to
5 children, and they come from four continents. They
6 come from Australia, they come from Yugoslavia,
7 they come from Mexico, they come from eastern
8 Europe. They come from Boston, they come from
9 Milwaukee, they come from Cincinnati; all those old
10 cities in the United States have studied this
11 problem. And there's differences among the
12 studies, but there's one thing that every study of
13 children exposed to lead paint shows, that lead
14 hurts kids, that lead hurts children's brains.

15 As the American Academy of Pediatrics
16 stated -- Exhibit 197, Bill -- 2005, the most
17 recent statement from the doctors in this country
18 who take care of these kids, if we could go to
19 their statement.

20 Thank you very much.

21 Here they're talking about lead in
22 children. In 2005 they say, "Evidence continues to
23 accrue that commonly encountered blood-lead
24 concentrations, even those less than 10 micrograms
25 of lead per deciliter of blood of the child, may

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1 impair cognition, the mental development of the
2 child, and there is no threshold yet identified for
3 this effect."

4 So after all these studies, after all
5 these years, the American Academy of Pediatrics
6 cannot say there's any threshold, any safe level.
7 That's why abatement, a permanent solution, is so
8 important.

9 I'd like to look at the dot map. We have
10 a larger one here today. Your Honor, this is a
11 large version of a document put in evidence that we
12 called the dot map.

13 And when we talk about a community,
14 ladies and gentlemen of the jury, we're talking
15 about the city of Milwaukee because, back in 1996
16 when Dr. Foldy began to describe how -- what they
17 encountered, the problem is occurring throughout
18 the city.

19 These are the children. And if we look
20 at the key down here, 7,636, this is every report
21 of an elevated blood lead 10 micrograms or greater,
22 the level that the CDC says is the level of concern
23 across the city. But where is the problem most
24 concentrated? In what the Health Department has
25 defined today as the 2006 target area in these

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1 communities.

2 So the Health Department has responded
3 city-wide to any case of childhood lead poisoning,
4 and they've worked with parents to educate them.
5 But this is where they've focused the effort to try
6 and stop the damage, the tragedy of childhood lead
7 poisoning in this city.

8 And you recognize the location, I
9 believe, from -- from the lake and from your own
10 experience. But these children have been
11 identified; they've been followed. This is where
12 the chelations have also occurred. This is the
13 problem of childhood lead poisoning in the city of
14 Milwaukee in 1996. And as we look at 2005,
15 although there have been significant
16 improvements -- thank you -- even in 2005, more
17 than 2000 children have been reported with an
18 elevated blood lead. Those are the communities,
19 those are the target areas for the city of
20 Milwaukee.

21 So the question is, Is lead paint present
22 in those homes, in those communities, an
23 interference with public health? Does it
24 constitute a public nuisance? That is the first
25 question on the verdict sheet.

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1 Now, the law of public nuisance has
2 identified foul odors from egg farms, has
3 identified spilling waste into a creek. That law
4 is quite old. It's more than a hundred years old;
5 it goes way back in time.

6 And we would suggest to you, ladies and
7 gentlemen of the jury, that those problems pale --
8 foul odors from an egg farm, it's important, but it
9 pales in comparison to the threat of brain damage
10 across this community. Absolutely pales in
11 comparison.

12 Dr. Landrigan called this a public health
13 crisis. Dr. Foldy, who took an oath, the gentleman
14 from the Milwaukee Health Department, former
15 commissioner -- maybe we could look at his
16 photograph to remind the jury who he was --
17 Dr. Foldy, who took an oath to protect the public
18 health in Milwaukee, found that the only way to do
19 that was to create the Childhood Lead-Poisoning
20 Prevention Program and put 35 to 45 people to work
21 full time, full time, to try and eradicate this
22 disease.

23 Now, the defendant, National Lead, does
24 not dispute that childhood lead poisoning is bad
25 for kids. They do not dispute that lead paint is a

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1 cause of childhood lead poisoning. And there are
2 no public health experts that dispute that, and
3 they didn't call any as witnesses because those
4 facts are undisputed.

5 The judge instructed you also as to the
6 credibility of witnesses, to use your own sound
7 judgment. And we would ask that when you consider
8 the testimony of the physicians who have spent
9 their life studying this problem, studying
10 epidemics, Dr. Landrigan; Dr. Foldy, a public
11 health professional, as a physician; Ms. Murphy,
12 since 1992 practicing public health, those
13 witnesses told you about their day-to-day
14 experience. They didn't have to make up something
15 for this case. They told you about their career,
16 their lifetime of experience with childhood lead
17 poisoning and the lead-paint hazards. They came in
18 here to explain that to you.

19 And the reason that the Centers for
20 Disease Control and the National Academy of
21 Sciences and HUD and the President's Task Force
22 have all said that lead paint is the major cause of
23 childhood lead poisoning in America is because it's
24 true. They have no other agenda. They said it
25 because it's true. And we ask you to find that it

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Vol. 21

1 STATE OF WISCONSIN)

2) SS
3 MILWAUKEE COUNTY)

4 I, JULIE A. POENITSCH, RPR/RMR/CRR,
5 Notary Public in and for the State of Wisconsin, do
6 hereby certify that the foregoing proceedings were
7 recorded by me and reduced to writing under my personal
8 direction and that it is a true and correct transcript
9 of said proceedings had in said matter to the best of my
10 knowledge.

11 In witness whereof, I have hereunto set
12 my hand at Milwaukee, Wisconsin, on this 19th day of
13 June, 2007.

14
15
16
17
18 _____
19 JULIE A. POENITSCH
20
21
22
23
24
25

ORIGINAL - 6-19-2007

STATE OF WISCONSIN

CIRCUIT COURT
CIVIL DIVISION, BRANCH 25

MILWAUKEE COUNTY

CITY OF MILWAUKEE,

Plaintiff,

v.

Case No. 01-CV-3066

NL INDUSTRIES, INC.,

Defendant.

Jury Instructions

A. Introductory Instructions**OPENING INSTRUCTIONS [100, 115, 120]**

This case will be submitted to you in the form of a special verdict consisting of ** questions. It is your duty to answer those questions which, according to the evidence and these instructions, it becomes necessary for you to answer to arrive at a completed verdict. It then becomes my duty to direct judgment according to law and according to the facts as you have found them.

The evidence in this case consists only of the following:

1. The sworn testimony of the witnesses, unless stricken by the court. (This includes any depositions or portion of depositions received.)
2. Any exhibits which have been received into evidence.

During the trial, I ordered that certain testimony be stricken, and you must disregard any stricken testimony. Anything you may have seen or heard outside of the courtroom is not evidence.

The lawyers in a case have a duty to object to what they feel are improper questions or answers. You should not draw any conclusion for either side from the fact that an objection was made and you must not let any objections or rulings by the court affect your evaluation of the evidence.

You are to decide the case solely on the evidence offered and received at trial. You are to be guided by these instructions and your own sound judgment in considering the evidence in this case and in answering the verdict questions. You should not concern yourselves about whether your answers will be favorable to one party or to the other nor with what the final result of this lawsuit may be.

I have not intended to give any member of the jury the impression that I have any feeling as to how you should decide this case. If any one of you has such an impression, you should disregard it entirely and decide the issues of fact solely as you view the evidence. You are the sole judges of the facts; the court is the judge of the law only.

BURDEN OF PROOF: QUESTIONS 1 - 4 [200]

Questions 1 through 5 in the verdict ask that you answer the questions "yes" or "no". The plaintiff in this case, the City of Milwaukee, has the burden of proving that "yes" should be your answer to these questions. This burden is to satisfy you by the greater weight of the credible evidence, to a reasonable certainty, that "yes" should be your answer.

The greater weight of the credible evidence means that the evidence in favor of a "yes" answer has more convincing power than the evidence opposed to it. Credible evidence simply means evidence that you believe in light of reason and common sense.

"Reasonable certainty" means that you are persuaded based upon a rational consideration of the evidence. Absolute certainty is not required, but a guess is not enough to meet the burden of proof.

B. Nuisance Claim

The City claims that the presence of lead-based paint in and on private homes in Milwaukee constitutes a public nuisance and that NL Industries was responsible for causing this nuisance. Questions 1 through 3 address this claim.

Question 1: Existence of Public Nuisance

Question 1 asks: Between 1992 and the end of 2006, was the presence of lead-based paint in and on houses in the City of Milwaukee a public nuisance?

A public nuisance is a condition that unreasonably interferes with a right common to the general public. A public nuisance requires an injury to the community or the general public, in contrast to a *private nuisance*, which requires an interference with a person's use or enjoyment of private property.

A condition does not become a public nuisance simply because it interferes with the use and enjoyment of land by a large number of persons or causes injury to many persons. There must be some interference with a public right, such as an interference with the health or safety of the general public. However, a public nuisance need not directly affect all members of a community, as long as there is an interference with a right common to the general public.

Question 2: Intentionally and Unreasonably Causing a Nuisance

Question 2 asks: Did NL Industries intentionally and unreasonably engage in conduct that was a cause of the public nuisance? Before you may answer this question "yes," you must be satisfied that NL Industries acted intentionally and unreasonably, and that its intentional and unreasonable conduct was a cause of the public nuisance.

"Intentionally." An interference with a public right is deemed to be "intentional" if the defendant acted for the purpose of causing it or knows that it is resulting or is substantially

certain to result from his conduct. Thus, NL Industries acted intentionally if it intended to create the public nuisance at issue in this case, or if it knew that its conduct was substantially certain to cause such a nuisance.

“Unreasonably.” In determining whether NL Industries acted unreasonably, you may consider all of the circumstances related to the defendant’s conduct, the creation of the public nuisance at issue, and the extent of any harm or injury that was a consequence of the public nuisance. In determining whether NL Industries acted unreasonably, you may consider whether the conduct of NL Industries was in compliance with existing laws. However, the fact that the defendant’s conduct may not have been in violation of any existing law does not necessarily relieve the defendant of liability in this case. You may find that the defendant’s otherwise lawful conduct was unreasonable under all of the circumstances presented by the evidence in this case.

“A Cause.” This question does not ask about “the cause” but rather “a cause” because a public nuisance may have more than one cause. NL Industries is responsible for causing the alleged public nuisance if you find that its intentional and unreasonable conduct was *a substantial factor* in producing the public nuisance. A public nuisance may be caused by the conduct of a single person or entity or the combined conduct of any number of people or entities.

“Substantial factor,” as that term is used here, means that NL Industries’ misconduct had such an effect in producing the public nuisance and resulting injury as to lead reasonable people to regard it as a cause, in the sense that the use of the term “cause” brings to mind the idea of responsibility. A substantial “cause” does not include every one of a great number of events without which the alleged public nuisance and alleged injury in question would not have occurred.

Question 3: Negligently Causing a Nuisance

Question 3 asks: Did NL Industries negligently engage in conduct that was a cause of the public nuisance?

"Negligently." A business entity is negligent when it fails to exercise ordinary care. Ordinary care is the degree of care that a reasonable business would use in the same or similar circumstances. A business is not using ordinary care and is negligent, if the business, without intending to do harm, does something or fails to do something that a reasonable business would recognize as creating an unreasonable risk of injury or damage to a person or property. "Similar circumstances" include the time period in which a party acts or fails to act, and this standard of ordinary care must be applied to NL Industries as of the time NL Industries was alleged to have been negligent.

"A Cause." Again, this question does not ask about "the cause" but rather "a cause" because a public nuisance may have more than one cause. NL Industries is responsible for causing the alleged public nuisance if you find that its negligent conduct was *a substantial factor* in producing the public nuisance. A public nuisance may be caused by the conduct of a single person or entity or the combined conduct of any number of people or entities. The term "substantial factor" has the same meaning as set forth on page 4.

C. Conspiracy Claim

The City of Milwaukee claims that NL Industries conspired with others to create the public nuisance at issue in this case. Questions 4 and 5 address this claim.

Question 4: Conspiracy [2800, 2806]

Question 4 asks: Did NL Industries engage in a conspiracy with one or more others that was a cause of the public nuisance?

Conspiracy. A conspiracy is a combination of two or more persons acting together to accomplish some unlawful purpose or to accomplish some lawful purpose by unlawful means. The essence of the conspiracy alleged here is a combination or agreement which had as its purpose the creation of a public nuisance.

Mere similarity of conduct among various persons and the fact that they may have associated with each other, and may have assembled together and discussed common aims and interests, does not necessarily establish the existence of a conspiracy.

The evidence need not show that the members entered into any express or formal agreement or that they directly, by words spoken or written, stated between themselves what their objectives or purposes were, or the details of them, or the means by which the objectives or purposes were to be accomplished. A conspiracy may be established by evidence that the members in some way or manner, or through some contrivance, positively or without it being openly expressed, came to a mutual understanding to try to accomplish a common and unlawful plan.

The evidence need not show that all the means or methods claimed by the plaintiff were agreed upon to carry out the alleged conspiracy; nor that all means or methods which were agreed upon were actually used or put into operation. Nor need the evidence show that all persons alleged to have been members of the claimed conspiracy were indeed members.

In determining whether a conspiracy existed, you are not to isolate various acts and events and determine separately whether each was illegal, unfair, or unreasonable. Rather, you are to consider all of the evidence -- all of the acts and events -- as a whole in determining whether a conspiracy existed and, if so, its character and effects.

Acts in and of themselves lawful may nevertheless constitute or be a part of an illegal conspiracy if they are part of a concerted plan. Even lawful agreements, legal actions, and business activities may help make up a pattern of conduct that is unlawful.

“A Cause.” Before you may answer this question “yes,” you must be satisfied not only that NL Industries engaged in a conspiracy with one or more others, but that such conspiracy was a cause of the public nuisance. Once again, this question does not ask about “the cause” but rather “a cause” because a public nuisance may have more than one cause. The conspiracy was a cause of the public nuisance if it was *a substantial factor* in producing the public nuisance. A public nuisance may be caused by the conduct of a single person or entity or the combined conduct of any number of people or entities. The term “substantial factor” has the same meaning as set forth on page 4.

Question 5: Conspiracy – Overt Acts [2810]

Question 5 asks: Did NL Industries or one of its co-conspirators perform any act in furtherance of that conspiracy?

You will answer this question only if you have found that two or more parties entered into an unlawful conspiracy to create the public nuisance at issue in this case. Before you may answer this question “yes,” you must be satisfied by evidence which is clear, satisfactory, and convincing, to a reasonable certainty, that one of the parties to the conspiracy performed an act in furtherance of or for the purpose of carrying out the conspiracy.

Lobbying Activities

Evidence was received in this case indicating that NL Industries, and the LIA, may have lobbied legislators or regulators about matters in which they had an interest. You may consider this evidence in resolving the issues of fact in this case, except that you may not consider any lobbying activities as “conduct” or “acts” or “methods” in determining whether

the City has met its burden of proof with respect to Questions 2, 3, 4 and 5. Parties have a constitutionally protected right to communicate with legislators or regulators about their interests, and to petition the government in furtherance of those interests. Because of this right under the United States Constitution, you may not consider evidence of lobbying activity in determining whether the conduct of NL Industries should be found liable for causing or conspiring to create a public nuisance.

D. Damage Question

Question 6 asks: What sum of money, if any, will fairly and reasonably compensate the City of Milwaukee for past expenditures incurred to abate the public nuisance?

The City claims damages for the costs of responding to the alleged public nuisance of lead paint in and on homes in the City. Your answer to Question 6 should be the sum of money you find has reasonably and necessarily been incurred by the City of Milwaukee as the natural consequence of the public nuisance.

The amount of damages, if any, found by you should in no way be influenced or affected by any of your previous answers to questions in the verdict. Your answer to the damage question should not be affected by sympathy or resentment; nor should you make any deductions because of a doubt in your minds as to the liability of any party to this action.

In considering the amount to be inserted by you in answer to question number six, the burden of proof rests upon the City to satisfy you by the greater weight of the credible evidence, to a reasonable certainty, that the City sustained damages and the amount of the damages. The greater weight of the credible evidence means that the evidence in favor of an answer has more convincing power than the evidence opposed to it. Credible evidence means evidence you believe in light of reason and common sense. "Reasonable certainty" means

that you are persuaded based upon a rational consideration of the evidence. Absolute certainty is not required, but a guess is not enough to meet the burden of proof.

The amount you insert in answer to question number six is for you to determine from the evidence. What the attorneys ask for in their arguments is not a measure of damages. The opinion or conclusions of counsel as to what damages should be awarded should not influence you unless it is sustained by the evidence. Examine the evidence — carefully and dispassionately — and determine your answer from the evidence in the case.

Collateral Source of Funds

Evidence in this case has indicated that the City received grants from the federal government or other sources to help pay for some of the costs of its abatement program. However, any issues with respect to the distribution of any damages awarded are not a part of the jury trial in this matter, and this evidence may not affect your answer to the damage question. You may not reduce your award of damages because the City may have received funds for some costs from another source.

E. Evaluation of Evidence

215 CREDIBILITY OF WITNESSES; WEIGHT OF EVIDENCE

You are the sole judges of the credibility of the witnesses and the weight to be given to their testimony. In weighing the evidence, you should use your common sense, and you may take into account matters of your common knowledge and your observations and experiences in the affairs of life.

In determining the credibility of witnesses and the weight you give to the testimony of each witness, you may consider their interest or lack of interest in the result of this trial, their conduct and demeanor on the witness stand, their bias or prejudice if any has been shown, the clearness or lack of clearness of their recollections, and their opportunity for observing and

knowing the matters about which they testified. You may also take into consideration the reasonableness of the witness' testimony, possible motives for falsifying, and all other facts and circumstances which tend either to support or to discredit the testimony of that witness. Ultimately, you should give to the testimony of each witness such weight and credit as you believe it is fairly entitled to receive.

230 CIRCUMSTANTIAL EVIDENCE

It is not necessary that every fact in this trial be proved directly by a witness or an exhibit. A fact may be proved indirectly by other facts or circumstances, from which it usually and reasonably follows according to human experience. This is called circumstantial evidence, and you should give it such weight as you believe it deserves.

Direct evidence is not necessarily better or worse than circumstantial evidence. Either type of evidence can prove a fact.

260 EXPERT TESTIMONY

Usually witnesses can testify only to facts they know. However, witnesses with expertise in a particular area are sometimes permitted to give an opinion in their field of expertise. In determining the weight to be given such an opinion, you should consider the qualifications and credibility of the expert and whether reasons for the opinion are based on facts in the case. Opinion evidence was admitted in this case to help you reach a conclusion. You are not bound by any expert's opinion.

In resolving any conflicts in expert testimony, weigh the different expert opinions against each other and consider the relative qualifications and credibility of the experts and the reasons and facts supporting their opinions.

110 ARGUMENTS OF COUNSEL

You should consider carefully the closing arguments of the attorneys, but keep in mind their arguments, conclusions, and opinions are not evidence. Ultimately, you must draw your own conclusions and your own inferences from the evidence and answer the questions in the verdict form based on your view of the evidence and my instructions on the law.

F. Closing Instructions**180, 190 CLOSING INSTRUCTIONS**

This case is now ready to be submitted to you for your deliberation. In your deliberations you must examine the evidence with care and caution. Act with judgment, reason, and prudence. You are to decide this case fairly and impartially, without fear or favor, and without passion or prejudice. Give each question in the verdict form your careful and conscientious consideration. In answering each question, free your minds of all feelings of sympathy, bias, or prejudice. Let your verdict speak the truth, whatever the truth may be.

Agreement by ten or more jurors is sufficient to become the verdict of the jury. If you can do so consistently with your duty as a juror, at least the same ten jurors should concur in all of your answers. I ask you to be unanimous if you can. At the foot of the verdict, you will find a place provided where dissenting jurors, if there be any, will sign their names and state the answer or answers with which they do not agree.

As jurors, you each have a duty to consult with one another and to deliberate for the purpose of reaching agreement. At all times you must keep an open mind. You must be receptive to and consider carefully the thoughts and views of other jurors. In the course of your deliberations, you must not hesitate to re-examine your own views and to change your opinion if convinced that it is erroneous. On the other hand, you must not surrender your

honest opinion as to the weight or effect of the evidence in order to accommodate other jurors or simply for the purpose of reaching a verdict.

When you retire to the jury room, your first duty will be to elect a jury foreperson, who will preside over your deliberations and write in the answers you have agreed upon. The vote of the foreperson is entitled to no greater weight than the vote of any other juror. When your deliberations are concluded, and your answers inserted in the verdict, the presiding juror will sign and date the verdict, and all of you will return with the verdict into the court.

While you may ask to see exhibits that are not initially sent up to the jury room, it is up to me to decide whether and to what extent you may review exhibits received into evidence. While it is up to you to decide what weight is to be given to any of the evidence in this case, it is important that physical evidence not be given greater weight than testimony simply because it can be reviewed in the jury room.

OFFICIAL - 6-19-2007

STATE OF WISCONSIN

CIRCUIT COURT
CIVIL DIVISION, BRANCH 25

MILWAUKEE COUNTY

CITY OF MILWAUKEE,

Plaintiff,

v.

Case No. 01-CV-3066

NL INDUSTRIES, INC.,

Defendant.

SPECIAL VERDICT

Public Nuisance Claim

QUESTION 1. Between 1992 and the end of 2006, was the presence of lead-based paint in and on houses in the City of Milwaukee a public nuisance?

ANSWER: YES
(yes or no)

If you have answered Question 1 "no," your verdict is complete. Do not answer any further questions. If you have answered Question 1 "yes," then answer the following questions, unless the instructions indicate that you should not answer a question.

QUESTION 2. Did NL Industries intentionally and unreasonably engage in conduct that was a cause of the public nuisance?

ANSWER: No
(yes or no)

QUESTION 3. Did NL Industries negligently engage in conduct that was a cause of the public nuisance?

ANSWER: No
(yes or no)

Conspiracy Claim

QUESTION 4. *[Answer this question only if you have answered "yes" to Question 2 OR Question 3.]* Did NL Industries engage in a conspiracy with one or more others that was a cause of the public nuisance?

ANSWER: _____
(yes or no)

QUESTION 5. *[Answer this question only if you have answered "yes" to Question 4.]*
Did NL Industries or one of its co-conspirators perform any act in furtherance of that conspiracy?

ANSWER: _____
(yes or no)

Damages

QUESTION 6: *[Answer this question only if you have answered "yes" to Question 2 OR Question 3 OR Question 5.]* What sum of money, if any, will fairly and reasonably compensate the City of Milwaukee for past expenditures incurred to abate the public nuisance?

ANSWER: \$ _____

Dated at Milwaukee, Wisconsin, this 22 day of June, 2007.

Signed: Patricia EASON REED (Foreperson)

Dissenting juror: EASON-REED, Margene As to question(s) 1

Dissenting juror: Vandenberg, Obrian As to question(s) 3

STATE OF WISCONSIN CIRCUIT COURT MILWAUKEE COUNTY
CIVIL DIVISION, BRANCH 25

CITY OF MILWAUKEE,

Plaintiff,

v.

Case No. 01-CV-3066

NL INDUSTRIES, INC.,

Defendant.

OCT - 8 2007

**Memorandum Decision
Denying Plaintiff's Motion for a New Trial**

Following the jury trial in this matter, the City of Milwaukee filed a motion to set aside the verdict and order a new trial.¹ Most of the issues raised in this motion were discussed before or during trial, some on several occasions and some to a considerable extent. For reasons set forth below as well as reasons set forth in connection with earlier rulings, the City's motion will be denied.²

The motion for a new trial raises seven challenges to the instructions, grouped under Section A of the City's motion and brief. In addition, the City seeks a new trial based on violations of the collateral source rule (see Section B of the City's motion); the trial court's decision not to submit a nuisance theory based on recklessness, (Section C); and the court's grant of summary judgment on the issue of future costs (Section D). Finally, the City contends that the court improperly denied three of its motions in limine (Section E).

¹ Plaintiff also filed a separate motion to change the jury's answer to Verdict Question 2, which was addressed in a separate decision.

² Because more than 90 days have passed since the verdict, the post-trial motions are considered denied pursuant to Wis. Stat. § 805.16(3). This decision only confirms that denial.

The City's principal objections to the jury instructions concern the instructions related to the claim of an intentional public nuisance, and the legal basis for those instructions will be discussed below. I will also discuss the City's requested instruction regarding agency and its claim that the City was prejudiced by evidence of collateral source payments. As to the other issues raised by the City, I will rely on the record made in connection with pretrial motions and rulings during the trial.

I. Jury Instructions on the Claim of Intentional Public Nuisance

A. Introduction

There is no Wisconsin case that defines the intentional conduct element of a claim based on the intentional creation of a public nuisance. The court's instructions were drawn from *Vogel v. Grant-Lafayette Elec. Co-op.*, 201 Wis. 2d 416 (1996), a private nuisance case that addressed the concept of intent, and the Restatement of Torts (Second). Wisconsin cases have long cited with favor to the Restatement, which attempts to distill the vast, "impenetrable jungle" of nuisance law into some basic principles of law.³

The Restatement emphasizes that a *public* nuisance has its historical origins in crimes against the property and rights of the Crown, a tort that is completely distinct from the notion

³ This criticism of nuisance law is from Prosser and Keeton on Torts:

There is perhaps no more impenetrable jungle in the entire law than that which surrounds the word 'nuisance.' It has meant all things to all people, and has been applied indiscriminately to everything from an alarming advertisement to a cockroach baked in a pie. There is general agreement that it is incapable of any exact or comprehensive definition. Few terms have afforded so excellent an illustration of the familiar tendency of the courts to seize upon a catchword as a substitute for any analysis of a problem; the defendant's interference with the plaintiff's interests is characterized as a "nuisance," and there is nothing more to be said.

W. Page Keeton, et al., Prosser and Keeton on Torts, at 616 (5th ed., Lawyers ed., 1894). This indictment is frequently quoted by frustrated appellate courts, including our own Supreme Court in *Milwaukee Metropolitan Sewerage Dist. v. City of Milwaukee*, 277 Wis. 2d 635, ¶ 24 (2005).

of a *private* nuisance. Restatement of Torts (Second), Section 821B, Comment a. These torts share the name of "Nuisance" only through of an "accident of historical development":

This word, which in its origin is merely the French word for harm, has come, by reason of nothing more than the mere use of the same term to describe two quite different things, to cover two types of invasion of the interests of others. They have little or nothing else in common, and are quite unrelated . . .

Id., Chapter 40, Introductory Note. The Restatement defines a public nuisance as an "unreasonable interference with a right common to the general public." *Id.*, § 821B. A private nuisance is "a nontrespassory invasion of another's interest in the private use and enjoyment of land. *Id.*, § 821D.

Beyond this important distinction as to the nature of the interest that is invaded, however, it is difficult to identify principles of *private* nuisance liability that do not have some applicability to a claim of *public* nuisance. In the Restatement topic entitled "Private Nuisance: Elements of Liability," the comments repeatedly refer to public nuisances and indicate, expressly or implicitly, that the rules set forth apply to a public nuisance as well. Wisconsin public nuisance cases often cite to private nuisance cases, without apology or distinction, and in the briefs filed in this case, the parties have relied primarily on private nuisance cases.

B. The Court's Instructions

Verdict Question 1 addressed the existence of the public nuisance as it pertained to both the City's *intentional* and *negligent* public nuisance claims. Question 1 on the verdict form, which the jury answered "Yes," read as follows:

QUESTION 1. Between 1992 and the end of 2006, was the presence of lead-based paint in and on houses in the City of Milwaukee a public nuisance?

ANSWER: Yes

Verdict Question 2 then addressed the City's *intentional* public nuisance theory:

QUESTION 2. Did NL Industries intentionally and unreasonably engage in conduct that was a cause of the public nuisance?

ANSWER: No

The jury instructions regarding the terms "intentionally" and "unreasonably" as used in Question 2 were as follows:

"Intentionally." An interference with a public right is deemed to be "intentional" if the defendant acted for the purpose of causing it or knows that it is resulting or is substantially certain to result from his conduct. Thus, NL Industries acted intentionally if it intended to create the public nuisance at issue in this case, or if it knew that its conduct was substantially certain to cause such a nuisance.

"Unreasonably." In determining whether NL Industries acted unreasonably, you may consider all of the circumstances related to the defendant's conduct, the creation of the public nuisance at issue, and the extent of any harm or injury that was a consequence of the public nuisance. In determining whether NL Industries acted unreasonably, you may consider whether the conduct of NL Industries was in compliance with existing laws. However, the fact that the defendant's conduct may not have been in violation of any existing law does not necessarily relieve the defendant of liability in this case. You may find that the defendant's otherwise lawful conduct was unreasonable under all of the circumstances presented by the evidence in this case.

The City has two objections to these instructions. First, it contends that the court should have defined "intentionally" by reference to "public injury or public harm" instead of "the public nuisance at issue." Secondly, it contends that the instructions incorrectly required the plaintiff to prove that NL's conduct was unreasonable.

C. The Element of Intent

In most nuisance cases, both public and private, there is no real issue regarding intent, because the connection between the defendant's conduct and the offensive condition and the resulting injury is direct, immediate and unmistakable. The lead paint nuisance at issue here is quite different, and rests on a uniquely indirect connection between conduct and injury.

Unlike the invasions so apparent in a classic nuisance – the smoke spewed forth by the factory or the sulfur dioxide discharged into the air by the defendant in *Jost v. Dairyland Power Co.* – NL did not simply spray lead paint over neighboring houses or pour it into the water supply.⁴ Instead it marketed and sold lead paint that, by subsequent choices made by any number of suppliers, retailers and homeowners, found its way into homes as an invited and welcomed guest, a guest that only much later and only under certain circumstances, became dangerous and quite unwelcome. The evaluation of intent is further complicated by the long period of time that lapsed between the defendant's conduct and the development of the public nuisance.

There was no dispute that the City had to prove that *at the time it promoted and sold lead paint*, the defendant “intended” some consequence related to childhood lead poisoning. There was no dispute that this element could be satisfied either by showing that this consequence was NL's actual purpose or that that NL “knew that its conduct was substantially certain to cause” such a consequence. The evidence did not suggest that NL sold paint for the purpose of causing childhood lead poisoning and thus, as with virtually all nuisance claims, “intent” is really a question of the defendant's knowledge and awareness, that is, whether the defendant knew that the harmful consequences were reasonably certain to happen.

The City's objection to the instructions is directed at the description of the consequence that must be anticipated by NL. In its requests submitted just before and after the close of the evidence, the City proposed that the anticipated consequence be described as “an injury or harm, that is, childhood lead paint poisoning, or the threat of childhood lead paint poisoning”:

NL's conduct was intentional if, without any desire to cause injury or harm, NL nonetheless had knowledge that its activities were a cause

⁴ *Jost v. Dairyland Power Co-op.*, 45 Wis.2d 164 (1969).

of an injury or harm, that is, childhood lead paint poisoning, or the threat of childhood lead paint poisoning, or if NL was substantially certain that its activities would cause such injury or harm.

Plaintiff's Proposed Jury Instructions, filed June 12, 2007, Request 10, at page 11; *Plaintiff's Proposed Jury Instructions*, filed June 15, 2007, Request 5, at page 6.

Both the *Restatement* and the *Vogel* decision make it clear that knowledge of *some* harm or injury is not enough. Such a theory would mean strict nuisance liability for any manufacturer of products known to cause injury and death – such as children's bicycles or products containing mercury – if other unanticipated problems developed. In the context of the City's nuisance claim against NL, identifying the harm as "childhood lead poisoning" does not correct the problem, because the phrase begs a critical question. Does it include the harm to children who chewed on toys and crib rails that was reported in the 1930's? It certainly seems to. In fact, it would seem to include even one instance of lead poisoning. Under such an instruction the City might be entitled to a directed verdict, since there was at least some period when NL knew that its paint was causing some children to become lead poisoned.⁵

The court's instruction was drafted from the *Restatement's* definition of an intentional invasion:

An invasion of another's interest in the use and enjoyment of land or an interference with the public right, is intentional if the actor

(a) acts for the purpose of causing it, or

(b) knows that it is resulting or is substantially certain to result from his conduct.

⁵ Such a theory would have raised other difficult questions, given the extraordinarily long time between the defendant's conduct and the public nuisance. Would it be enough for the jury to find that NL anticipated that "some childhood lead poisoning" would occur at the time of its conduct, say, in the 1940's, or would the jury be asked to determine whether NL knew in the 1940's that the problem would still exist 50 years later, when the City was injured by this nuisance?

Restatement, § 825. The pronoun "it" clearly refers to "an interference with the public right" and the Restatement comments make it clear that "it" means *the* interference that is alleged to be the nuisance.

Vogel confirms this interpretation. In that case, the court considered negligence and private nuisance claims of dairy farmers who alleged that their herd was adversely affected by stray voltage from the particular system that distributed electricity to their farm. The problems with the herd and the decline in milk production were first noticed in 1970, although it was not until 1986 that the Vogel's suspected stray voltage was the problem and contacted the Grant-Lafayette Electrical Cooperative. The trial court declined to submit an intentional nuisance theory, and the Supreme Court affirmed this decision. Even though the defendant knew that stray voltage would result from the type of delivery system used on the farm, liability for intentional nuisance required knowledge of the unreasonable levels of stray voltage that were harming the cows. *Id.*, at 431-32. As the court concluded:

In order for a nuisance to exist in this fact situation, there must be an *unreasonable* amount of stray voltage that affects the person's interest in the private use and enjoyment of land. Therefore, GLEC may be liable for an intentional invasion under the continuing invasion rationale expressed in the Restatement if it continued to impose excessive levels of stray voltage onto the Vogels' farm that might endanger their cows after it had *knowledge of the problem*. However, that is not the case here. In fact, the record indicates the opposite.

Id., at 433, *emphasis added*.

In *Vogel*, "the problem" was clearly excessive stray voltage that harmed cows. It was not simply *some* stray voltage, nor was it the electrical distribution equipment that had apparently caused "the problem." In this case, characterizing "the problem" is not so easy, because the "injury" was characterized in so many different ways, including the threat of harm created by the mere existence of lead paint, the more immediate harm created by the lead dust that accumulates, the elevated blood levels found in so many children, the injuries

suffered by these lead poisoned children, the larger public health problems that resulted from all of these symptoms, and reduced property values. Because this *public* nuisance claim was brought by the City, its counsel focused on the need to pay for lead-abatement programs:

The injury is the need to abate the lead paint on the walls. This is what the Court of Appeals is saying that it's not just -- they are saying that it's not the kids with poisoning. It's the injury to the City. It's that injury which this case is about. So the harm is the need to abate the lead paint.

Tr., Motion Hearing, May 15, 2007, at 25. What was "the problem" that NL must have anticipated in order for it to be liable for causing a public nuisance? Was it some of these injuries? All of them?

After the court had proposed that "the problem" be described as "the public nuisance at issue in this case," the plaintiff requested that the court instead refer to "public injury or public harm." *Plaintiff's Memorandum Regarding Proposed Verdict Questions and Jury Instructions*, filed June 18, 2007, at 11.⁶ In the context of this case, "public injury" would appear to be a reference to the public nuisance as that was defined for the jury:

A public nuisance is a condition that unreasonably interferes with a right common to the general public. A public nuisance requires an injury to the community or the general public. . . .

Jury Instructions, at 2. Under that interpretation, there is no material difference in meaning between the court's instruction and the City's request. If the phrase was intended to refer to a different or broader "public injury or public harm," some additional explanation was needed.

Of course, it was not necessary that the jury find that NL anticipated the precise nature and scope of the problem as it unfolded in the 1990's. It is enough that NL have foreseen the

⁶ June 18 was essentially the second full day of the jury instruction conference. The evidence had been completed towards the end of the day on Thursday, June 14, and because of the extensive issues that remained as to the appropriate jury instructions, the jury was excused until Monday, June 18, at 10:30 a.m. Both parties submitted new proposed verdict forms and jury instructions on Friday, and the court and parties spent most of that day on issues related to the jury instructions. Substantial issues remained when the court adjourned for the weekend at 5:30 p.m., and these issues were discussed for most of the day on Monday, June 18. The court did not begin instructing the jury until late in the day, and closing arguments did not begin until Tuesday morning.

general problem that was outlined by the City's evidence and that the problem would rise to the level of an injury to the community. I considered adding jury instructions along these lines, but did not do so because no party requested them; because any such instructions would likely have presented contentious drafting issues; and because it was obvious to the jury that the law did not require the City to prove that NL knew precisely what was going to happen 50 years in the future.

D. Unreasonableness

Perhaps no instruction issue was the subject of more discussion than the question of how the notion of unreasonableness should be addressed. The City's proposed instructions permitted the jury to consider whether the *harm* caused by the nuisance was unreasonable, but did not permit consideration of whether NL's *conduct* was unreasonable. I concluded that under the Restatement rules the unreasonableness of the defendant's conduct is clearly an issue in any claim of public nuisance. Less clear is where within the framework of legal instructions the jury should be told to consider such unreasonableness.

As noted earlier, a public nuisance is defined as an "unreasonable interference with a right common to the general public." *Id.*, § 821B. Following this definition, § 821B list three circumstances that might support a finding of unreasonableness, all of which reference the defendant's conduct:

(2) Circumstances that may sustain a holding that an interference with a public right is unreasonable include the following:

(a) whether the conduct involves a significant interference with the public health, the public safety, the public peace, the public comfort or the public convenience, or

(b) whether the conduct is proscribed by a statute ordinance or administrative regulation, or

(c) whether the conduct is of a continuing nature or has produced a permanent or long-lasting effect, and, as the actor knows or has reason to know, has a significant effect upon the public right.

Id., § 821B.

The notion of unreasonableness is also linked to an intentional "invasion" in § 822, which distinguishes nuisances based on intentional conduct from those based on negligent or other tortious conduct:

One is subject to liability for a private nuisance if, but only if, his conduct is a legal cause of an invasion of another's interest in the private use and enjoyment of land, and the invasion is either

(a) *intentional and unreasonable*, or

(b) unintentional and otherwise actionable under the rules controlling liability for negligent or reckless conduct, or for abnormally dangerous conditions or activities.

Id., § 822, *emphasis added*. As used in the Restatement, the terms "invasion" and "interference" encompass both the nuisance itself and the defendant's tortious conduct, and § 822 does not specify whether one or both must be *intentional and unreasonable*. In the typical nuisance case, the wrongful conduct is contemporaneous with the creation of the injury, and the distinction may be less important or perhaps its importance is less apparent. Moreover, the term *intentional* can only be applied to the defendant's conduct, and the requirement that the invasion be "*intentional and unreasonable*" may be viewed as directed primarily at the defendant's behavior. Earlier comments about the meaning of "Unreasonable interference" following § 821B support this conclusion:

In each of these categories [of tort liability], some aspect of the concept of unreasonableness is to be found. If the interference with the public right is intentional, it must also be unreasonable. *[parenthetical reference omitted]* If the interference was unintentional, the principles governing negligent or reckless conduct, or abnormally dangerous activities all embody in some degree the concept of unreasonableness.

Id., § 821B, Comment c.

The question of whether an "invasion" is both intentional and unreasonable is further complicated by the "imperative distinction" between a nuisance and liability for a nuisance, emphasized in *Milwaukee Metropolitan Sewerage Dist. v. City of Milwaukee*, 277 Wis. 2d 635 (2005) and based on citations to the Restatement:

. . . it is imperative to distinguish between a nuisance and liability for a nuisance, as it is possible to have a nuisance and yet no liability. *A nuisance is nothing more than a particular type of harm suffered; liability depends upon the existence of underlying tortious acts that cause the harm. The Restatement (Second) of Torts illustrates this point: [F]or a nuisance to exist, there must be harm to another or the invasion of an interest, but there need not be liability for it. If the conduct of the defendant is not of a kind that subjects him to liability . . . the nuisance exists but he is not liable for it.*

Restatement (Second) of Torts § 821A cmt. c (emphasis added).

Much of the confusion in nuisance law results from a "[f]ailure to recognize that . . . nuisance has reference to the interest invaded and not to the type of conduct that subjects the actor to liability." Restatement (Second) of Torts § 822 cmt. b. Thus, a cause of action in nuisance is predicated upon a particular type of injurious consequence, not the wrongful behavior causing the harm. (citation omitted).

Id., ¶¶ 25-26. To invite the jury to consider the reasonableness of the defendant's conduct in determining the existence of the nuisance would violate this distinction. It would ask the jury to consider and weigh the same factors that would bear on whether a defendant's conduct was intentional or negligent or otherwise tortious.

The jury instructions proposed by the City did not permit the jury to consider or determine the reasonableness of the *defendant's conduct* as to either the existence of the nuisance or the defendant's conduct. The notion of unreasonableness appears only in the proposed definition of public nuisance:

A public nuisance is a condition which substantially or unreasonably interferes with a right common to the general public such as health or safety.

Plaintiff's Proposed Jury Instructions, filed June 12, 2007, Request 3, at page 4; *Plaintiff's Proposed Jury Instructions*, filed June 15, 2007, Request 1, at page 1. Since these instructions allowed the jury to find *either* a substantial or an unreasonable interference, a finding that the interference was "substantial," as it quite obviously was, meant that the notion of unreasonableness had been written out altogether.

To the extent that jury might consider whether the interference was unreasonable, the City's instructions discussed this only in terms of the injury component of the nuisance:

An interference is unreasonable if it materially affects or impairs, or threatens to affect or impair the health of a community or neighborhood.

• • •

When you consider the unreasonableness of the interference, you may consider a number of factors including the nature of the harm, the number of people in the community who may be affected by it, the extent of the harm, the permanence of the injuries, or the potential for likely future injuries or harm.

Plaintiff's Proposed Jury Instructions, filed June 12, 2007, Request 4, at page 5; *Plaintiff's Proposed Jury Instructions*, filed June 15, 2007, Request 2, at page 3. This definition of "unreasonable interference" and the suggested considerations focus entirely on the "injurious consequences" as they existed between 1992 and 2006. The instructions allow for no consideration of the reasonableness of the defendant's conduct, all of which occurred before 1978.

The City's instructions incorrectly omitted any consideration of the reasonableness of NL's conduct. Based on the rules and comments of the Restatement, and on the "imperative distinction" between a nuisance and liability for the nuisance, the jury was properly instructed on the theory of a public nuisance based on intentional conduct.

II. The City's Requested Instruction on the Law of Agency

Over the defendant's objections, a great deal of evidence was received that reflected statements by persons affiliated with the Lead Industries Association. These statements reflected a variety of opinions and discussions about health issues related to lead and lead paint and also the exchange of information on such issues. Their principal purpose was to establish what was known within the lead industry generally and by NL in particular. In that respect, the evidence was not significant for its "truth content," but was admissible to demonstrate what NL was likely to have known about the dangers of lead paint during the first half of the 1900's. While this evidence was received without limitation, the City objects to the court's failure to instruct the jury about whether it must or could "attribute" the statements to NL Industries. Specifically, the City asked that the jury be instructed that all statements of the Lead Industries Association prior to 1961 should be "attributed" to NL Industries, because the LIA was then an unincorporated association, and that statements after 1961 could be "attributed" to NL if the jury found that the LIA was NL's agent or co-conspirator.

The defendant has contended that the requested instruction did not correctly summarize the law regarding the jury's evaluation of such evidence. I will put those issues aside because, in the context of this case, the proposed instruction is both confusing and unnecessary. I received this evidence because it was relevant to the determination of NL's state of mind during the many decades in which the conduct that allegedly caused the nuisance had occurred. I found that it was relevant for this purpose regardless of whether an agency foundation had been laid for all of the time periods and all of the declarants reflected in this evidence. Since the evidence was received without limitation, the question is whether

the City was entitled to an instruction intended to guide the jury as to the "attribution" of these statements.

The conduct for which NL was alleged to be responsible was the promotion and sale of lead pigment and paint. There was no doubt that that NL engaged in such conduct, and the "agency" statements at issue did not bear on such conduct by NL or others in any material way. The evidence reflected who knew what and who said what to whom within the lead paint industry. Of course, the statements of an agent can be important verbal acts, such as where an agent accepts an offer or speaks words that are negligent. The statements at issue here, however, were not material to liability-forming conduct and it was not necessary for the jury to determine whether NL was "responsible" for such the statements. Under these circumstances it would be confusing and unnecessary to ask the jury to determine whether the legal elements of agency had been satisfied as to each of the many statements and each of the many speakers.

III. Collateral Source Issue

The evidence at trial disclosed to the jury that most of the lead abatement programs at issue were funded by various "grants." Even though the jury became aware of this primarily through evidence elicited by the City, the City contends that this was violation of the collateral source rule and that it is entitled to a new trial. In addition to arguments raised at trial, the City argues that the recent decision in *Leitinger v. DBart, Inc.*, 2007 WI 84, entitles it to relief.⁷

⁷ Citing language from *Leitinger*, the City questions the validity of my distinction between the collateral source rule and its "evidentiary corollary." Whether the evidentiary component of the collateral source rule is viewed as a "rule" or a "corollary" does not affect its application in this case. Based on a footnote in the *Leitinger* decision, however, it appears that the better term is "evidentiary analogue." *Id.*, at ¶ 30, fn 24.

Lettinger was a personal injury case, and addressed an issue that has arisen in the effort to determine the reasonable value of medical expenses in the complex and seemingly unreasonable world of health care pricing. Specifically:

The issue of law presented on review is whether, in light of the collateral source rule, evidence of the amount actually paid by a plaintiff's health insurance company for the plaintiff's medical treatment is admissible in a personal injury action for the purpose of establishing the reasonable value of the medical treatment rendered.

Id., ¶ 4. Apparently on the theory that evidence of what was actually paid is at least as probative of reasonable value as evidence of the amount billed, the trial court permitted the defendant to introduce evidence of the amounts actually paid by the plaintiff's insurance company. The Supreme Court disagreed, finding that the collateral source rule applied and concluding that the evidence was not admissible for that purpose.

In our case, NL *did not* seek to offer evidence of federal grant amounts for the purpose of establishing the reasonable value of lead abatement programs. In fact, *it was the City* that chose to use such evidence as the principle measure of damages. It did so for very good reasons, but it cannot then claim unfair prejudice and demand a new trial based on the collateral source rule or the holding in *Lettinger*.

Based on the exhibits to be used by the City and representations made in pretrial proceedings, it was clear that the damages case could not be explained and defended before the jury without reference to the grant programs. The testimony of Amy Murphy and other witnesses bore this out. The damages figures were directly based on grant expenditures and the City sought to use the grant review and oversight procedures as evidence that the expenditures were in fact related to the abatement of lead paint. NL also sought to use this evidence for legitimate purposes, such as challenging the credibility of HUD studies and challenging the City's policy choices in attacking the problems created by lead paint.

While the jury did not reach the question of damages, it was carefully instructed that it could not reduce its damage award based on this evidence:

Evidence in this case has indicated that the City received grants from the federal government or other sources to help pay for some of the costs of its abatement program. However, any issues with respect to the distribution of any damages awarded are not a part of the jury trial in this matter, and this evidence may not affect your answer to the damage question. You may not reduce your award of damages because the City may have received funds for some costs from another source.

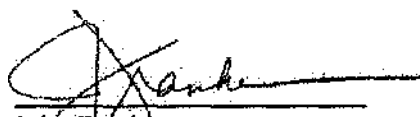
Jury Instructions, at 9. The City, however, now raises an issue not mentioned at trial, that is, that collateral source evidence prejudiced the plaintiff on issues related to liability. For reasons discussed above, the City has waived any claim of prejudice. Moreover, while there may be cases in which a jury's decision on liability could be affected by knowledge that the plaintiff has been reimbursed from other sources, this is simply not such a case. I see no meaningful risk that this jury would have altered or compromised its liability decision because of evidence that other government entities may have borne some of the costs.

Conclusion

For these reasons, and for reasons set forth before and during the trial, the plaintiff's motion for a new trial is without merit. Because more than 90 days have passed since the verdict, the motion must be denied, and pursuant to Wis. Stat. § 805.16(3) it is ordered that judgment shall be entered for the defendant.

Dated: October 7, 2007.

By the Court:


John Franke
Circuit Judge

STATE OF WISCONSIN CIRCUIT COURT MILWAUKEE COUNTY
CIVIL DIVISION, BRANCH 25

CITY OF MILWAUKEE,

Plaintiff,

v.

Case No. 01-CV-3066

NL INDUSTRIES, INC.,

SEP 25 2007

Defendant.

Memorandum Decision
Denying Plaintiff's Motion to Change the Special Verdict

Following the jury trial in this matter, the City of Milwaukee filed a motion to change the jury's answer to Question 2 in the verdict form from "No" to "Yes."¹ The City contends that the evidence required any reasonable jury to answer this question "Yes." For reasons set forth below, as well as reasons set forth in various rulings on related issues during the trial, I disagree.

In answering "Yes" to verdict Question 1, the jury had found that the presence of lead-based paint in and on houses in the City of Milwaukee between 1992 and 2006 was a condition that unreasonably interfered with a right common to the general public. Question 2 on the verdict form, which the jury answered "No," read as follows:

QUESTION 2. Did NL Industries intentionally and unreasonably engage in conduct that was a cause of the public nuisance?

ANSWER: No

¹ Plaintiff also filed a separate motion to set aside the entire verdict and order a new trial, which is addressed in a separate decision.

Although the form of Question 2 is not at issue, it should be noted that it combined the element of intentional conduct and the issue of cause into a single question. This was done for reasons discussed on the record and with agreement of both parties. See *Trial Tr.*, Vol. 19, at 3148-3149, and Vol. 20, at 3174-75.²

For purposes of this motion, I will accept the City's argument that if one looks at NL's conduct between 1900 and 1978, or even between, say, 1940 and 1978, such conduct was clearly a substantial factor in producing the nuisance. I will also assume that if a jury were to find that NL knew that it was creating the type of public health problem that led to the City's abatement efforts, that the jury should necessarily find that such conduct was unreasonable.

The real issue was whether NL had sufficient knowledge to satisfy the element of intent. In that regard the jury was instructed that "NL Industries acted intentionally if it intended to create the public nuisance at issue in this case, or if it knew that its conduct was substantially certain to cause such a nuisance." *Jury Instructions*, at 4. As with virtually all nuisance cases, there was no claim that the offending party had acted for the purpose of causing the nuisance, and so the question turned on what NL knew was reasonably certain to follow from its conduct. The factual circumstances of this nuisance, however, presented significant problems for the finder of fact in attempting to determine what NL knew was "substantially certain" to follow from its conduct. Unlike the typical nuisance case, the

² The reason for the broad scope of Question 2 was the conceptual confusion that would result if, as both parties requested, a jury was first asked to consider whether defendant intentionally *created* a nuisance and then to consider whether such conduct *caused* the nuisance. This problem was discussed again when NL Industries objected to a similar combined format for the negligence questions, at which point I described a "backup plan" that would allow the jury to somehow first consider the intentional *creation* of the nuisance and then the *legal cause* of the nuisance. *Trial Tr.*, Vol. 20, at 3174-3175. I did not pursue this idea primarily because the parties had agreed to a combined question and because any such alternative approach would present its own significant drafting problems. Although I noted that a combined question made it harder to determine precisely what a jury meant by the answer "No," I considered that there was really no issue as to whether NL's contributions were "a cause" of the nuisance as that nuisance had been defined for the jury, and that this case was primarily about the level of knowledge necessary to satisfy the intent element of a public nuisance.

“conduct” that contributed to the nuisance occurred over a period of more than 75 years, during which time NL’s knowledge was evolving in significant respects. Unlike the typical nuisance case, there was a large gap between the last of the conduct, which ended completely by 1978, and the injury suffered by the City beginning in 1992. The gap between most of the conduct and the emergence of the *public* nuisance was enormous.

It was the City’s legal theory that it need only to prove that NL anticipated that its conduct would cause “childhood lead paint poisoning, or the threat of childhood lead paint poisoning.” See *Plaintiff’s Proposed Jury Instructions, filed June 12, 2007*, at 11, and *Plaintiff’s Proposed Jury Instructions, filed June 15, 2007*, at 6. Its factual theory was that at least by the 1930’s, NL was aware that children were suffering severe injury and death from ingesting lead paint, and was therefore quite aware that paint was poisoning children. The problem with both theories is that the evidence in the case identified two related yet quite different lead poisoning problems. More importantly, it was overwhelmingly clear that the problem identified in the first half of the century was *not the reason* why the presence of lead-based paint in 1992 constituted a *public* nuisance and was *not the reason* why the City undertook its abatement efforts.

During the 1930s, it became apparent that the toxicity of lead was not simply a health problem for those who worked in the lead industry or with lead paint. It was causing serious injury and death to children who, apparently attracted to the taste, ingested paint by chewing on toys or cribs or other painted surfaces. Decades later a much more subtle and insidious problem was identified, as it became clear that the mere presence of lead paint inevitably resulted in the presence of lead dust and chips and that, through the ordinary hand to mouth activities of very young children, lead was being ingested. It further became clear that this

process was sufficient to cause great damage at blood levels much lower than were previously thought dangerous.

This led to an outright prohibition on the sale of lead paint by January of 1978, although even then the full extent of the health problems was not recognized -- in part because some of the circumstances that led to the crisis were not yet in place. For a variety of reasons about which we heard much testimony at trial, circumstances that included the simple activities of opening and closing windows and the sanding of old paint surfaces, lead paint was gradually poisoning a staggering number of children. It was this problem that caused the Court of Appeals to refer to a "community wide injury to public health" in this case and a "public health catastrophe" in another.³ It was this problem that caused the City of Milwaukee to identify a *public* health crisis and target certain areas for abatement. It was this problem that caused the injury that was at the core of the City's nuisance claim, as described by its counsel:

The injury is the need to abate the lead paint on the walls. This is what the Court of Appeals is saying that it's not just -- they are saying that it's not the kids with poisoning. It's the injury to the City. It's that injury which this case is about. So the harm is the need to abate the lead paint.

Tr. May 15, 2007, at 25. As serious as the earlier childhood lead poisoning problems may have been, they were only historical background for the public nuisance of 1992. That public nuisance would have existed, perhaps in a much more virulent form, had no child ever been poisoned by chewing on toys or crib railings.

I rejected the legal theory embodied in the City's requested instructions because the phrase "childhood lead paint poisoning" clearly included the poisoning identified in the 1930's, which was vastly different in nature and scope than the lead dust crisis identified by

³ *City of Milwaukee v. NL Industries, et al*, 278 Wis. 2d 313, ¶ 15 (Cl. App. 2005); *Thomas ex rel. Grambling v. Mallett*, 285 Wis. 2d 236, 558 (2005).

the testing done in the 1980's and 1990's. It was the latter tragedy that the City alleged had risen to the level of an *interference with a public right*, not the earlier instances of children eating lead paint.

The basis for the specific instructions given by the court is further discussed in connection with the City's motion for a new trial, but under any reasonable view of intent, the City was required to do more than show that NL anticipated the sort of childhood lead poisoning that was well-documented during the first half of the 20th Century. Relying on the Restatement (Second) of Torts and *Vogel v. Grant Lafayette Elec. Cooperative*, 201 Wis. 2d 416 (1996), I concluded that it was not enough for the jury to find that a defendant anticipated "a harm" or "some harm." Such a doctrine would impose strict liability for nuisance whenever some harm could be anticipated even though nothing close to a *public* nuisance was foreseeable. The plaintiff must establish that the defendant anticipated "the harm" that actually occurred.

Under this legal standard, the City's factual theory failed because the childhood lead poisoning of the 1930's and the childhood lead poisoning problem that triggered the City's abatement efforts in 1990 were ultimately two very different things. Proof that NL was aware of the earlier problem, even buttressed by anecdotal evidence that at times NL acted in its narrow self-interest and with corporate insensitivity, did not establish the NL anticipated the public nuisance found by the jury in its answer to Question 1.

Given the many decades over which NL promoted and sold lead paint, it is not surprising that the evidence regarding NL's sense of corporate responsibility was mixed. Evidence offered by the defense indicated that at times the lead industry, and NL in particular, acted responsibly and in the public interest as to matters related to the toxicity of lead. The evidence also suggested that the lead industry, including NL, was very concerned about the

public image of lead products, and that at times the lead industry preferred to have bad news cast in the best possible light, did not always share information that reflected badly upon lead products, and sought to blame others for health problems related to lead. Ultimately, this evidence said little or nothing about NL's ability to foresee the lead paint crisis that was quietly and gradually gathering in what became the target areas for the City's lead abatement efforts.

The City claimed, in pretrial submissions and before the jury, that evidence would show that NL and the lead industry covered up important information about the dangers of lead, presumably evidence related to the foreseeability of a public nuisance. No such evidence materialized. The most the City could point to in this respect was that in 1952, the Lead Industries Association had gathered lead poisoning data from health departments in several cities and did not take steps to disseminate that information. *See Exhibit 39*. It is difficult to find a meaningful secret here. Twenty-six children reported poisoned by lead paint in Baltimore, four of whom died, is a problem whether or not a similar number of victims is identified elsewhere. Even if we assume that public health officials were not aware of what was happening elsewhere, common sense suggests they would not have been surprised to learn that similar cities had similar problems. This sketchy and incomplete data added little to what was already known, and the numbers only serve to emphasize the gulf between what was known in the 1950's and what was later discovered in the 1980's.

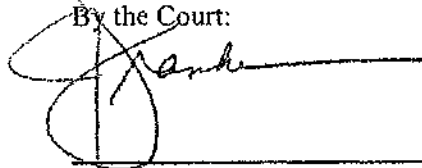
While the historical background was interesting, it said nothing by itself about whether anyone at NL anticipated the public health crisis that brought about the City's abatement efforts in the 1990's. In fact, the evidence demonstrated that despite widespread concern about the toxicity of lead and about its effect on children, *no one* anticipated the "public health catastrophe" that eventually led to the injury suffered by the City in this case. Thus,

whether the intent instruction referred to "the public nuisance at issue in this case," or some other description of the public health crisis that was at the heart of the City's public nuisance theory, the jury was entitled to find that the City's evidence was woefully insufficient and to answer the question "No."

It might be sound public policy under some circumstances to require an industry to pay for injuries caused by its products, even when the injuries were not anticipated. There may be other legal claims that establish liability for an injury that is not foreseen. The law of nuisance, however, does not impose such liability. Therefore, it is ordered that the City's motion to change the jury's answer to Question 2 in the verdict form is denied.

Dated: September 24, 2007.

By the Court:

A handwritten signature in black ink, appearing to read "John Franke", written over a horizontal line.

John Franke
Circuit Judge

WISCONSIN COURT OF APPEALS
DISTRICT 1

APPEAL NO: 07AP2873

CITY OF MILWAUKEE,

Plaintiff-Appellant,

v.

NL INDUSTRIES, INC,

Defendant-Respondent

and

MAUTZ PAINT CO.

Defendant

Appeal from the Circuit Court for Milwaukee County,
the Honorable John A. Franke, Circuit Court Judge, Presiding
Circuit Court Case No: 01CV003066

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liability to NL “without ever trying to find out if our product, Dutch Boy, is in any of those homes”); *see also*, Trial Tr. 6/19/07 at 3404:6-15, A-App-157 (blaming landlords and property owners for creating a private nuisance). In light of this Court’s previous ruling on these precise issues, NL’s “product identification” and “property maintenance” arguments were prejudicial and should have been excluded.

The City asked the trial court to mitigate such prejudice with the following instruction: “To find that NL’s conduct was a substantial factor in causing the alleged public nuisance, you need not find that NL’s lead pigment or lead paint was on any specific property in the City.” Pltf’s. Prop. Verdict Questions and Jury Instructions (R.579) at 4. The trial court refused to offer this instruction, however, which also constituted error.

C. The trial court improperly prevented the jury from considering reckless conduct liability, the City’s conspiracy claim, and future costs.

1. Reckless conduct

In addition to proving liability for nuisance through intentional or negligent conduct, Wisconsin courts have also recognized that liability for nuisance may be based on the

rules governing reckless conduct or abnormally dangerous conditions. See *Milwaukee Metro. Sewerage*, 277 Wis. 2d 635, ¶ 32, 691 N.W.2d 658 (citing Restatement (Second) of Torts § 822).

At the close of the Plaintiff's case in chief, NL moved for a directed verdict as to Plaintiff's nuisance claim on all four tortious conduct theories: intentional, negligent, reckless, and abnormally dangerous conduct. In ruling on reckless conduct, the trial court found that the evidence showed that "there was at least some risk, arguably a great risk, that some child would be harmed by the continued use of lead paint." Trial Tr. 6/8/07 at 2145:9 - 2146:6, A-App-133-135. Despite this finding, however, the trial court granted NL's motion as to reckless conduct. *Id.* at 2146:23 - 2147:10, A-App-136-137, but denied NL's motion for directed verdict as to intentional conduct. Reckless conduct is a lesser standard than intentional conduct which the trial court should have let the jury consider. As the comments to the Restatement (Second) of Torts § 500 note:

Reckless misconduct differs from intentional wrongdoing in a very important particular. While an act to be reckless must be intended by the actor, the actor does not intend to cause the harm which results from it. It is enough

that he realizes or, from facts which he knows, should realize that there is a strong probability that harm may result, even though he hopes or even expects that his conduct will prove harmless. *However, a strong probability is a different thing from the substantial certainty without which he cannot be said to intend the harm in which his act results.*

See Cmt. f (emphasis added). Thus, had the jury been allowed to consider this question, they could have found that there was a “strong probability” (as opposed to “substantial certainty”) that NL knew its conduct was causing harm. The City was prejudiced by the jury’s failure to consider this claim.

2. Conspiracy

Over the City’s objection, the trial court instructed the jury in the Special Verdict to answer Question 4, “Did NL Industries engage in a conspiracy with one or more others that was a cause of the public nuisance,” only if the jury had previously found that NL either “intentionally and unreasonably,” or “negligently” “engage[d] in conduct that was a cause of the public nuisance.” Special Verdict (R.585) at 1-2, A-App-201-202. This instruction precluded the jury from considering NL’s vicarious liability for acts that it “promoted, but did not commit[.]” *Segall v. Hurwitz*, 114

when the City "chose" to abate. Thus, the trial court should have denied NL's motion. To the extent this Court orders a new trial, the jury should be allowed to consider the City's future damages, as well.

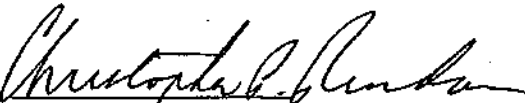
CONCLUSION

For the foregoing reasons, the City of Milwaukee respectfully requests that this Court change the jury's conclusion as to Special Verdict Question Number Two to find that NL intentionally caused the public nuisance the jury found to exist. Additionally, Appellant requests that the Court grant a partial new trial due to the trial court's numerous errors which, individually and cumulatively, prejudiced the City.

Dated this 24th day of March, 2008

von BRIESEN & ROPER, S.C.

By:



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