A. Lead Paint—The “Ubiquitous Contaminant”

Virtually all persons in industrial civilizations have some lead in their bodies due to the ubiquitous nature of lead in the environment. Lead is found in many consumer products manufactured in the United States and in foreign countries. Products containing lead include jewelry, toys, cribs, high chairs, cosmetics, and lunchboxes. In fact, in the 2000s, many toys were recalled by the Consumer Product Safety Commission when they were found to be contaminated with lead. Consumers and children can also be exposed to lead through imported pottery, candies, or Mexican or Asian folk remedies, which may contain high concentrations of lead. Additionally, lead particles or dust can be brought into the home from contaminated work clothes and lead to exposure. Despite all these potential sources of contamination, however, most lead paint litigation arises out of the exposure of children to housing built before 1950 when lead was banned in residential (as opposed to industrial) paint.

In some jurisdictions, litigation has been brought against paint manufacturers for allegedly placing an injurious product on the market and for failure to warn of the risks of exposure. In other jurisdictions, such as in the state of New York, litigation is generally brought against landlords, landowners, and residential management companies for exposure to lead-based paint applied to the surfaces of older apartments and rented homes. In most of those cases, the plaintiffs are children or young adults who live in older housing often located in economically-depressed communities. It is important to note, however, that lead-based paint can also be found in older private housing in higher-income neighborhoods. However, far less litigation arises out of exposure to lead-based paint in private homes. In some cases,
this is a result of prevailing laws that prevent such lawsuits; in other cases, it is a result of the lack of a deep-pocketed defendant or relevant insurance policy.

In 1991, the Centers for Disease Control (CDC) noted that “it is unlikely that there is a single predominant source of lead exposure” for young children. Nevertheless, experts for plaintiffs in lead paint litigation claim the most common cause of exposure is very small particles of lead-based paint peeling from walls, ceilings, and other surfaces and combining with household dust. This dust contaminates all surfaces, including a mobile child’s hands, fingers, skin, scalp, clothes, and toys and, through normal hand-to-mouth activity is absorbed into the child’s bloodstream. All other potential sources of exposure, such as exposure in utero, recent immigration from countries still using leaded gasoline, or use of ceramics or cosmetics containing lead are often dismissed by plaintiffs’ experts as significant exposure sources.

Although the medical community refers to a level above the “reference point,” or 10 micrograms per deciliter, as an “elevated blood lead level,” plaintiffs’ testifying experts routinely diagnosis “lead poisoning,” and testify that a child has been “lead poisoned” and is “brain damaged” based upon a single elevated blood lead level (EBLL) reading. In an August 2005 statement, the CDC noted the 10 mcg/dl level of concern, “which was originally intended to trigger community wide prevention activities, has been misinterpreted frequently as a definitive toxicologic threshold.” Additionally, the CDC addressed newer studies that showed evidence of adverse health effects in children with blood lead levels (BLLs) of less than 10 mcg/dl. As of the date of the report, the studies were based on a sample of “fewer than 200 children whose BLLs were never above 10 ug/dL.” In the CDC’s view, “questions remained about the size of the effect.” Thus, the CDC declined to lower the level of concern set in 1991 because, among other reasons,

no effective clinical or public health interventions have been identified that reliably and consistently lower BLLs that already are < 10 ug/dL, no threshold for adverse effects have been demonstrated, the adverse health effects associated with elevated BLLs are subtle, and establishing a level of concern substantially < 10 ug/dL probably would be accompanied by a sharp increase in misclassification of children as having an elevated BLL.

Commonly, defense experts contend that an EBLL reading above 10 mcg/dl does not have an adverse effect on every child.

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B. Acute Exposure

There are two types of lead exposure: acute and chronic. Acute exposure occurs over a short period of time or from a single exposure or ingestion. Experts retained by plaintiffs testify that an acute exposure results in a rapid rise in blood lead levels lasting from a little as seven days to roughly two to three weeks. Because lead is a neurotoxin, experts may testify that in every case, lead poisons a child’s brain and central nervous system and causes irreparable and permanent cognitive damage. In cases where the plaintiff’s pediatrician or a pediatric neurologist has run a battery of neurological tests with normal findings, plaintiff’s experts may nevertheless testify that once any amount of lead enters the brain, it remains in the brain for a two- to three-year period. This opinion is often proffered at trial, although it is in direct contradiction to the CDC’s March 2002 statement that “leaded dust generally causes an elevated blood lead level only after a significant duration of exposure.”

C. Chronic Exposure

Chronic lead poisoning occurs over a longer period of time—several weeks, months, or years. Sources of exposure other than lead paint include soil (residual lead from decades of using leaded gasoline), dust, drinking water, food, ceramics, cosmetics, occupations and hobbies, air, food, travel to developing countries still utilizing lead-based gasoline, traditional medicines, and (more recently) toys from China. These sources are routinely discounted by plaintiffs’ experts as relevant pathways for exposure. For example, experts may describe the existence of lead in drinking water as “small or limited” and may rule it out as a source of lead intoxication.

Whether acute or chronic exposure is alleged, once a child is removed from the source of exposure or the exposure source is remediated, the BLL should gradually decrease. This is referred to as the “half-life” of lead in the blood. Nevertheless, plaintiffs’ experts often testify that permanent and irreversible damage has already occurred. They may testify that while the lead in the blood may decrease, lead in the brain (which cannot be measured by any techniques in modern medicine) will remain constant for approximately two or more years and that lead in the bones will also remain for many years. Moreover, they opine that lead in the skeleton releases during bone growth, returning lead into the blood stream, particularly in children from five to seven years old or during adolescent growth spurts.

D. The Physical Manifestations of Elevated Blood Lead Levels in Young Children

The current reference point or “level of concern” set by the CDC is 10 micrograms per deciliter, which, as the graph reveals, is aligned with the CDC’s consistent decreasing of what it considers to be an elevated blood lead level.

It is often alleged that whether exposure is acute or chronic, children with an EBLL (> 10 mcg/dl), or even lower blood lead levels consistent with generalized environmental exposure (< 5 mcg/dl), have inevitably suffered permanent and irreversible brain damage. Based on medical and epidemiological studies, plaintiff’s experts have largely testified that the younger the child is, the more severe the adverse health effects of lead exposure will be. The critical time is said to be when an infant begins to crawl; this is the period of most vulnerability if the home contains lead-based paint or contaminated dust. Thus, the current CDC level of concern is focused on children aged six and younger.

Neuroscientists and pediatricians retained by plaintiffs often testify that the developing brain of a child between the ages of two and three mistakes lead for calcium. Calcium is used by the brain’s blood vessels and cells to produce certain chemicals that the brain requires to develop and function normally. Lead limits calcium in the brain by replacing calcium and interfering with the brain’s chemistry,

4. In 1991, the CDC revised the 1985 intervention level of 25 ug/dl to 10 ug/dl. “In 1984, the last year for which estimates are available, it is believed that between 3 and 4 million children younger than age 6 years (17% of all U.S. children in this age group) had blood lead levels above 15 ug/dl.” See Preventing Lead Poisoning 1991, supra note 1, Chapters 1 and 6.

5. Id., Figure 2-2.
resulting in cognitive deficits. Physicians may also testify that damage to the central nervous system is proof of an injury.

Moreover, plaintiffs’ experts may testify that lead impairs nerve cells’ ability to communicate with one another. Lead also interferes with mitochondria, which are organelles within brain cells. Mitochondria produce energy for brain cells, allowing them to function normally. Lower energy means lower brain functionality.

In any event, even though the CDC did not recommend lead testing for children over six, there are occasional cases brought on behalf of children older than six claiming brain damage and a litany of associated injuries. In 2004, some states (such as New York) increased the age of concern to encompass children aged seven or younger. This is because lead is said to target the developing brain; mature brain cells are much less vulnerable to the effects of lead.6

Physical manifestations of lead poisoning such as convulsions, stomach pain, loss of appetite, and even coma occur at BLLs greater than 70 mcg/dl. Chelation, an inpatient therapy to rapidly remove lead from the blood stream, is not medically appropriate at BLLs less than 45 mcg/dl. In fact, most “treatments” for an EBLL consist of advisory guidance by the pediatrician concerning the sources of lead, cleaning the home, and nutrition, as well as supplementing the child’s iron and calcium intake.

As discussed previously, “half-life” is the amount of time it takes for a quantity of lead to decrease by 50 percent. Some experts testify that while the range may widen slightly at either end of the spectrum, the half-life of lead in humans is approximately 30 to 35 days. Over time, the amount of lead will decrease, but lead in the bones or teeth may take years, if not decades, to be eliminated.

Because there are no outward or physical manifestations of injury caused by lead exposure, physical and neurological examinations (even by plaintiff’s experts) are usually negative or unremarkable. This is said to be the case because, in a developing system, injuries often do not manifest until more complex cognitive processes are required to function normally. This has been called the “lag effect”; cognitive deficits and intellectual impairments become apparent as the child gets older. Even specific skills that are presently unimpaired may become impaired in the future.

The lack of present physical manifestations does not prevent a female infant-plaintiff and her experts from arguing that lead stored in her body will have a negative impact on future pregnancies if released lead from the maternal skeleton crosses the placenta and is ingested by the developing baby.

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6. A new study concludes “6 year blood lead level is more strongly associated with cognitive and behavioral development than is blood measured in early childhood.” Richard Hornung, Bruce P. Lanphear, & Kim N. Dietrich, Age of Greatest Susceptibility to Childhood Lead Exposure: A New Statistical Approval, 117 ENVIRONMENTAL HEALTH PERSPECTIVES (August 2009).
E. The Cognitive Manifestations of Elevated Blood Lead Levels in Children

Expert witnesses for both plaintiff and defense rely on the findings of a neuropsychologist who has performed a battery of comprehensive neurological tests. Based on neuropsychological testing, the plaintiff’s expert may testify to cognitive or intellectual deficits in the form of a low IQ, delayed speech (often seen in male children), or deficiencies, or that variances (or scatter) in one or more of the tested areas reflect brain damage. On the other hand, defense experts will point to the findings as being representative of results found in the population of children who have certain neurological or medical disorders unrelated to toxic lead.

For the plaintiffs’ bar and its experts, any weakness detected by testing may be said to be evidence of brain injury or central nervous system dysfunction. Because there are often no other medical events that could have caused the deficits (such as head trauma), plaintiff’s expert may testify that any deficits in skills such as expressive language, impulse control, fine motor functioning, memory, or problem solving are strictly the result of brain damage caused by lead poisoning. Defense counsel must review the medical records and history for other, more common causes of cognitive and learning disorders, such as maternal smoking, birth trauma, and poor prenatal care (all of which can and often do contribute to later developmental problems).

In many cases in which a child is alleged to have suffered elevated blood lead levels, the child may indeed be experiencing problems in school or with his or her behavior. Often there are other contributing factors. In some cases the child is being raised in a home in which English is not the dominant language spoken. Many children thrive in bilingual homes; however, it may be difficult for a child to learn predominantly in English, especially if he or she is unable to seek help at home. A child with a reading disorder such as dyslexia may demonstrate problems in school and comprehension. Often, this type of condition is hereditary; however, some courts are loath to admit evidence of familial disorders or medical history. Therefore, a jury may never hear that, for example, a child’s parents and siblings (who have never allegedly been exposed to lead) have similar reading problems as the infant-plaintiff claiming to have been injured by lead exposure.

The admissibility of familial evidence differs from state to state, and, notably in New York, among the judicial departments. In the First Department, a defendant was precluded from compelling the mother of the plaintiff to undergo an IQ test. Defendants in the First Department largely have also been prevented from compelling information regarding an infant-plaintiff’s sibling’s medical, develop-

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ment, or academic history. Conversely, such information is discoverable in the Fourth Department. Where the parent of the infant-plaintiff was also a plaintiff, employment records and IQ tests of the mother were found to be likely lead to the discovery of relevant or admissible evidence. The academic records of non-party siblings were also sought. Because those records likely would contain privileged material, the court ordered an on-camera inspection.

Additionally, infant-plaintiffs who have been diagnosed with autism or related disorders have been more prevalent in recent years. Plaintiff’s counsel may argue that the effects of lead on an autistic brain are even more devastating than on a “normal” brain, as the autistic brain is already fragile. However, because autism is a condition that usually manifests during a child’s early development, the parent may not realize their child is not developing normally until around age two. Because age two is also when a child becomes mobile, a diagnosis of autism and exposure to toxic lead paint with EBLLs can occur simultaneously. Plaintiff’s counsel will claim the injury is indivisible at trial. This may prove to be a very persuasive argument, as there are no “hallmarks” of the effects of lead on the brain. Certainly the fact that autism is still an idiopathic disease (i.e., from an unknown source) allows plaintiff’s counsel to go just far enough to suggest that exposure to lead may be a factor, despite the fact that there is no medical or scientific support for such a theory.

The claimed damage in infant cases is brain damage. Plaintiff’s experts may opine that when a child’s brain is injured, the child’s IQ decreases. While experts acknowledge that results of IQ testing can vary if testing is repeated or based on the test-takers mood and that brain injury cannot be determined by IQ testing alone, experts may still maintain that any and all poor results or impairments are evidence of brain injury. For example, if a child’s performance on the visual IQ test varies from the performance IQ test, the differential (according to plaintiff’s expert) is not simply the result of the child’s natural strengths and weaknesses. Or, if a child has a 90 in English but a 75 in science, or is noted to be “doing very well” in some areas and “struggling” in others, this too (according to plaintiff’s expert) is a sign of brain damage. For most physicians (not involved in litigation), parents, and educators, this would be a sign of more interest or proficiency in one subject matter over another; for plaintiff’s experts, this can be evidence that the child’s overall IQ has decreased as a result of lead poisoning. If the child is “average,” the expert may testify the child should have been “high average”; if the child is “low average,” but for lead poisoning, the child should have been “average”; and so on.

Such testimony is allegedly based on studies purportedly demonstrating that the main loss in IQ at BLLs less than 10 mcg/dl is about six IQ points; for each

10 mcg/dl increase, there is a loss of an additional two IQ points. Another study found that the major loss of IQ points occurs at BLLs less than 10 mcg/dl. This study has resulted in enlarging the scope of lead paint litigation. Other reasons for a low IQ or learning disabilities such as maternal IQ, family history of learning disability, inability to speak English in an English-speaking school setting, autism or associated conditions on the autism spectrum, low socioeconomic status, and poor school attendance are largely discounted as irrelevant by plaintiffs’ experts, but are valid issues for the performance of a differential diagnosis by an expert.

F. The Claim for Damages

Plaintiffs seek compensatory and special damages for past and future pain and suffering as well as monetary awards to pay for future tutors and/or private schools. These schools can provide academics for special-needs students, occupational therapy, and psychological support (and could cost more than $35,000 a year).

In addition, plaintiffs usually call a forensic economist at trial to project the child’s future earning capacity (using projections based on different educational scenarios) and lost earnings. First, the economist will project the child’s earnings as a minimum wage earner, a high-school graduate, a two-year college graduate, and a four-year college graduate. Then, using the child’s age, plaintiff’s economist will calculate when the child is expected to enter the labor force and the child’s work life expectancy (the number of years an individual is expected to be in the labor force). The point of the exercise is to highlight that the more education a child receives, the more career opportunities he or she will have, the more the child will earn, and the longer the child, as an adult, will ultimately remain in the labor force. All of this is impaired by brain injury as a result of exposure to toxic lead.

In some cases, the expert takes into account periods when an individual may be out of the labor market (layoffs, unemployment, periods of bearing and raising children, periods of disability, etc.); in other cases, the economist makes no such adjustments. Based on the wage scenario (minimum wage, high-school graduate, and so forth) and the work life expectancy (using governmental statistical tables) and factoring in inflation and growth rates (using historical data), the economist projects the child’s alleged total lifetime earnings to a reasonable degree of economic certainty. These sums routinely add up to millions of dollars in claimed damages.

In a typical case, the plaintiff’s attorneys will call a pediatrician, a public health sanitarian and/or lead paint inspector, a neurologist, a neuropsychologist, and an economist. The defense may call a pediatric neurologist, a neuropsychologist, a risk assessor, and, in rare cases, an economist.

Unlike brain injury cases where there is a definable injury based on PET scans or other tests, in lead poisoning cases, injuries are more speculative. Such cases often involve learning disabilities and behavioral issues that are not uncommon in the
general population of children who have not sustained EBLLs. Nevertheless, based on the economist’s projections and calculations, plaintiffs are often awarded sums in the high six figures, as well as millions of dollars in compensation for “invisible” damages.

G. Adult Lead Exposure

Generally, adult lead poisoning cases arise out of occupational exposure. Most cases of occupational lead poisoning occur through inhalation. That is, plaintiffs’ lead poisoning is allegedly a direct result of unsafe work conditions, the negligence of the defendant, or violation of various individual state statutes that require employers to provide adequate protection for workers to avoid exposure to lead. Even in occupational cases, it is important to explore whether the plaintiff could have been exposed by any other source, including hobbies such as pottery/ceramics, auto repair, stained glass/painting, printmaking, hunting/target shooting, and playing games with lead figurines.

In construction cases, plaintiffs will often claim that provisions such as periodic equipment inspections, respiratory equipment, containment structures, air monitoring, ventilation systems, suitable personal protective clothing, housekeeping activities, and personal hygiene facilities (showers, changing rooms, and handwashing facilities) were either not provided or were inadequate to withstand the level of contaminants to which the plaintiff was being exposed. For example, even if properly fitted, a mask with the wrong filter will not protect a worker from the contaminants within the workspace confined by tarps, tents, or plastic containment mechanisms.

Occupations and trades associated with lead exposure include construction work, manufacturing (pigment, lead battery, chemical, etc.), gas companies and stations, work associated with lead (mining, smelting, refining, soldering), and others. It is critical that employers follow normal industry standards, as well as Occupational Safety Health Administration (OSHA) regulations to minimize lead exposure.

Occupational standards are regulated by the 1978 Occupational Safety Health Administration Lead Standard. OSHA guidelines set the permissible exposure limit (PEL) of lead in the workplace, the frequency and extent of medical monitoring, and other employer responsibilities. Workers exposed to air concentrations at or above the action level of 30 µg/m³ for more than 30 days per year require periodic determination of BLLs. If a BLL is found to be greater than 40 µg/dL, the worker must be notified in writing and provided with a medical examination. If a worker’s one-time blood lead level reaches 60 µg/dL (or averages 50 µg/dL or more on three or more tests), the employer must remove the employee from the source of exposure, with maintenance of seniority and pay, until the employee’s blood lead level falls below 40 µg/dL.
In adults, levels above 80 mcg/dl are considered serious and likely to cause permanent health damage (extremely dangerous). Levels between 40 and 80 mcg/dl may cause serious health damage; levels between 25 and 40 mcg/dl suggest regular exposure is occurring; and levels of 10 to 25 mcg/dl suggest some lead exposure.\textsuperscript{11}

Commonly claimed physical injuries include joint pain in the arms, elbows, and knees; abdominal pain; body cramps; sleep disorders; impotence (erectile dysfunction); and memory deficits. Adult plaintiffs also often allege they have suffered cognitive brain impairment (as a result of toxic encephalopathy to the brain caused by lead exposure); depression (a condition secondary to lead poisoning); and peripheral neuropathy (a condition common in cases of lead poisoning). Treatment for adults with EBLLs includes a complete blood count test and a free erythrocyte protoporphyrin (FEP) test, which is a useful test in determining the extent of lead poisoning. If the test results are sufficiently high and the symptoms warrant it, workers may be treated with ethylenediaminetetraacetic acid (EDTA), a compound which coagulates heavy metals in the body, including lead. This is known as chelation therapy and is generally not used unless the BLL is greater than 80 mcg/dl.

\textsuperscript{11} State of New York, Department of Health, Lead Exposure in Adults: A Guide for Health Care Providers (March 2009).