Getting the Lead Out?
The Misuse of Public Nuisance Litigation
By Public Authorities and Private Counsel

By

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The authors note that any similarities between the title of this article and a recently published article by “proud members” of the Rhode Island “trial team,” are purely coincidental. See A. Sprague and F. Fitzpatrick, Getting the Lead Out: How Public Nuisance Law Protects Rhode Island’s Children, 11 Roger Williams U. L. Rev. 603 (2006).
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I.

PREFACE

One of the principal forces behind changes in the common law has been the experience of judges who, because of traditional and allegedly “archaic” rules of law, found themselves unable to achieve justice when faced with “modern” problems. The principal example of such a change was the recognition of strict product liability by common law courts in the latter decades of the twentieth century. In that context, traditional “fault-based” causes of action, such as negligence, inevitably rendered it difficult or impossible for persons injured by defective products to recover compensation – not because it was difficult to prove the existence of a defect, but rather because it was virtually impossible to prove the specific acts or omissions of the manufacturer that produced it.¹

Many courts viewed this situation as an injustice and they developed the doctrine of strict product liability, based either upon consumer expectations of reasonably safe products, or upon broad public policy principles that recognized the social utility of shifting the responsibility for injuries away from the consumer to the manufacturers who were better able to bear them.² Although some – including these authors – have argued that “fault” remains relevant to some types of product liability cases, such as those based upon marketing defects, the general principles of strict product liability are firmly established in most American jurisdictions.³

Despite the availability of these judicially-created “short cuts” in product liability cases, certain types of cases, typically those involving chronic injuries allegedly caused by products produced by unknown manufacturers, continued to frustrate some judges. When confronted with

² Id.
³ Id.
an admittedly injured plaintiff, and an apparently defective product, they were loathe to dismiss claims merely because the identity of the manufacturer could not be established. In various contexts, a few courts developed common law rules that excused the plaintiff from proving the element that, according to prior law, was essential to recovery. Doctrines known as “alternative liability,” “enterprise liability,” and “market share liability” emerged in a few jurisdictions. These doctrines generally allowed an injured party to shift the burden of proof on critical issues to the defendants – and required the defendants to prove that they did not manufacture or supply the product which caused plaintiffs’ injuries. If they could not do so, the defendants were held either jointly and severally liable, or alternatively, liable for a percentage of the damages that corresponded to their “market share.”

Here, as it did when strict product liability originally developed, courts found that public policy favored placing liability on the manufacturers, who were in a position to insure against the losses, rather than upon the injured consumer. Unlike strict product liability, however, these attenuated doctrines were not generally accepted. Instead, only a few jurisdictions adhered to the new principles, and even they carved numerous

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5 Sindell, 607 P.2d at 937.

6 Id.

7 Id. at 936.
exceptions to limit their applicability. As a result, the move toward “absolute liability” halted, and the new doctrines proved to have limited utility.

Within several years, however, a new “assault on the citadel” began. This time, the problem again surfaced in the context of allegedly defective products, such as lead pigments in paint – but the claimants, faced with decades of defeat under traditional common law rules, cleverly decided to change the theory of their cause of action from strict products liability to a less-explored tort known as “public nuisance.” By doing so, they apparently hoped to escape the resistance of common law courts to further expansions of strict liability by encouraging novel expansions of an entirely different tort – one which was, according to traditional common law rulings, completely unsuitable for compensating persons injured by defective products. While professing allegiance to the traditional purposes of public nuisance law, they used the same public policies that fostered the growth of product liability to graft new principles onto the law of public nuisance and – assisted by the same sympathetic reasoning that produced the product liability explosion – new “growth” appeared on the tree of public nuisance law.

With this artifice, strengthened by alliances between public authorities and private counsel, the claims of unnamed and non-consenting injured persons were subsumed in broad

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10 The phrase “assaulting the citadel” was famously coined by Professor Prosser in his landmark article advocating the demise of fault-based causes of action in favor of strict products liability. See Prosser, The Assault on the Citadel, 69 Yale L. J. 1099 (1960).

11 See infra at V.B.
public nuisance claims made by governmental authorities who, up to that point, had remained remarkably silent and, indeed, indifferent, to the plight of their constituents.\textsuperscript{12} With this artifice, jurisdictions continued to ignore the substantial and obvious liability of landlords and property owners, and the possible continuing threat against the occupiers of contaminated properties, only to pursue claims against selected product manufacturers, most of which remain unresolved after years of protracted litigation.\textsuperscript{13}

With this artifice, jurisdictions pursued litigation against selected manufacturers without many of the burdens otherwise applicable to private litigants, such as statutes of limitations and product identification.\textsuperscript{14} Indeed, some courts excused the state from offering any proof that a particular defendant’s product was actually present in any specific location.\textsuperscript{15} To assist in implementing this strategy, state authorities burdened the public purse with obligations to compensate private counsel without the typical safeguards of competitive bidding applicable to most, if not all, other contracts designed to benefit the public interest.\textsuperscript{16} With this sleight of hand, they sought the creation of gigantic “abatement funds” under the guise of equitable relief.\textsuperscript{17} These funds were not sought to compensate property owners for any contamination of their properties, nor were they sought to compensate persons injured by the contamination for medical expenses or impairment, or to remediate or repair any particular structure. Instead, the money was to be accumulated and used only to the extent members of the public came forward voluntarily and sought public assistance.\textsuperscript{18} From that fund, created without any measurable data

\textsuperscript{12} See infra at V.B.2.iii.
\textsuperscript{13} See infra at V.B.3.
\textsuperscript{14} See infra at V.B.3.
\textsuperscript{15} See infra at V.B.
\textsuperscript{16} See infra at V.B.2.iii.
\textsuperscript{17} See infra at V.B.
\textsuperscript{18} See infra at V.B.
regarding the scope of public response, private counsel would be compensated – and presumably compensated extraordinarily well – before the efficacy of the program could be evaluated.  

Despite the wide array of substances and products potentially subject to this expanded cause of action, this article focuses on litigation involving lead, and most particularly, litigation involving lead paint. In order to understand the depth of the errors inherent in these claims, the article provides an exhaustive review of lead generally, including its physical and chemical characteristics, historical uses, regulatory background, and its toxicity. The article then reviews the underpinnings of the tort of “public nuisance” and studies the experience of past and pending cases, ultimately focusing on the controversy surrounding lead paint. As will be seen, such a comprehensive review demonstrates that, far from achieving just and intellectually defensible results, the present litigation is incapable of truly serving the public interest – unless that interest is defined by economically coerced settlements that benefit the public purse (and the purses of private law firms), as opposed to the public health.

II. BACKGROUND PRIMER ON LEAD

A. WHAT IS LEAD?

Lead is a heavy, soft grey metallic element with the symbol Pb (from the Latin plumbum). The English words plumber and plumbing are derived from the Latin word for lead, plumbum. Plumbum is also the source of the chemical symbol for lead, Pb. Lead is one of the basic elements or “building blocks” of nature and is found on the periodic table of chemical elements. Lead is the final stable product from the decay of uranium. It has an atomic number of 82, and a relative atomic mass of 207.19. Some of the physical and atomic properties of elemental lead are listed in Table 1.

19 See infra at V.B.3.
PROPERTIES OF ELEMENTAL LEAD

| Symbol | Pb, derives from plumbum, the Latin name for the metal |
| Synonyms | plomb (French); bloi (German); piombo (Italian); plomo (Spanish); chumbo (Portuguese) |
| Word Origin | From the Anglo-Saxon word for metal, lead |
| Appearance | Bluish white |
| Atomic Number | 82 |
| Atomic Weight | 207.21 |
| Density (20°C) | 11.34 g/cm³ |
| Melting Point | 327.4°C |
| Boiling Point | 1,750°C |
| Oxidation Numbers | +2, +4 |
| Thermal conductivity (near 20°C) | 34.66 W/m K |
| Specific Heat (20°C) | 129.8 J/kg K |
| Coefficient of linear thermal expansion (20°C-100°C mean) | 29.3 x 10⁻⁶/K |

Lead is usually found as an ore in deposits of galena (PbS) which have been worked worldwide for their lead. It is found to a lesser extent in anglesite (PbSO₄), cerussite (PbCO₃) and (Pb₃O₄). Occasionally, lead occurs as a free metal in its native or pure form. Combined with

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other elements, it forms a variety of interesting and beautiful minerals, all of which are heavy due to their lead content.\textsuperscript{23}

Lead is unique among common metals because it has little mechanical strength, virtually no elasticity and it is extremely soft. Indeed, most of its properties are so sensitive to conditions of testing and to minute changes in chemical composition that laboratory test conditions cannot be reproduced exactly in a practical application. As a result, laboratory data often has no practical meaning and it is often difficult to replicate conditions exactly, even in a second laboratory test.\textsuperscript{24}

Perhaps the best known property of lead is its resistance to corrosion in aggressive environments (especially sulfuric acid).\textsuperscript{25} Lead’s ability to provide good service in such situations often gives the erroneous impression that lead is a passive metal. In fact, lead is a very reactive metal and it is this reactivity which enables it to be used in corrosive environments. In air, for example, a close fitting and adherent film of lead carbonate is formed by rapid reaction, first between metallic lead and oxygen to form lead oxide, followed by a second reaction between the lead oxide film and carbon dioxide (which is always present in air) to form a protective film of lead carbonate. Further contact with the metallic lead underneath is then prevented and corrosion ceases.\textsuperscript{26}

\textsuperscript{23} MII Background, \textit{supra} note 22.

\textsuperscript{24} \textit{Properties of Lead}, Lead Development Association International (LDAI), \textit{available at} \url{http://www.ldaint.org/technotes5.htm} (last visited July 20, 2006) [hereinafter LDAI Properties]; for example, tensile tests, identical except for variations in speed of testing, may give ultimate tensile strength (UTS) figures varying from as much as 14 MN/m\textsuperscript{2} (about 2000 psi) to 7 MN/m\textsuperscript{2} (about 1000 psi). Similarly, composition variations between 99.99\% and 99.9999\% purity can result in UTS variations from 14 MN/m\textsuperscript{2} down to 9 MN/m\textsuperscript{2}. \textit{Id.}

\textsuperscript{25} While sensitive to environmental acids, after exposure to environmental sulfuric acid (H\textsubscript{2}SO\textsubscript{4}), metallic Pb becomes impervious to corrosion due to weathering and submersion in water. This effect is due to lead sulfate (PbSO\textsubscript{4}), the relatively insoluble precipitate produced by reaction of Pb with H\textsubscript{2}SO\textsubscript{4}, which forms a protective barrier against further chemical reactions. In: Phillip A. Schweitzer, \textit{Metallic Materials: Physical, Mechanical, And Corrosion Properties}, 695-698 (Marcel Dekker Inc. 2003) (chapter on Lead And Lead Alloys).

\textsuperscript{26} LYDIA Properties, \textit{supra} note 24.
B. SOURCES OF LEAD

1. Natural Sources

Lead is a naturally occurring metal found in the Earth’s crust at about 15–20 parts per million. In comparison to the two most abundant metals in the Earth (aluminum and iron), lead is a relatively uncommon metal. Lead rarely occurs in its elemental state, but rather its +2 oxidation state in various ores throughout the earth. The world’s usable reserves of lead are estimated at $7.1 \times 10^7$ tons, with over one-third located in North America.\(^{27}\)

Lead occurs naturally in low concentrations in all rocks, soils and dusts, usually ranging from 2 to 200 parts per million. The total amount of lead in the earth’s crust is estimated to be $3.1 \times 10^{14}$ tons. Some soils have relatively high concentrations of lead, where the underlying parent rock has significant lead content. Lead contents of waters are generally low, but significant amounts of lead-rich dusts and vapors are carried in the air.\(^{28}\) Common sources of naturally occurring airborne lead include volcanoes, sea-salt spray, biogenic sources, wild forest fires, and wind-borne soil particles in rural areas with background soil concentrations. Natural sources combined contribute an estimated 19,000 metric tons of lead to the air each year.\(^{29}\) In the United States, the levels of lead in the air have been steadily decreasing since the 1980s. This trend is also true worldwide.\(^{30}\)

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\(^{29}\) Jerome O. Nriagu & Jozef M. Pacyna, Quantitative Assessment Of Worldwide Contamination Of Air, Water And Soils By Trace Metals, NATURE (LONDON) 333: 134-39 (May 12, 1988). However, there is significant variability in the lead emissions from 12 volcanoes and forest fires and considerable uncertainty in biogenic and sea-salt emissions of lead. For example, estimates of lead flux from volcanoes range widely from 540 tons/year to 6,000 tons/year. This uncertainty has been attributed to the difficulty in obtaining representative data due to the episodic nature of volcanoes. Jerome O. Nriagu, A Global Assessment Of Natural Sources Of Atmospheric Trace Metals, NATURE (LONDON) 338: 47-49 (1989).

\(^{30}\) Thorton, supra note 28, at 84. Sensitive modern instruments allow detection of trace levels of lead in air. Levels at the South Pole, where anthropogenic influence is very small, are quoted as 0.63ng/m$^3$ air (1ng is one billionth of a gram). For
2. Production of Lead

It is estimated that the identified lead resources worldwide exceed 1.5 billion tons. Much lead is recovered as the primary metal from galena deposits. Because of the way in which minerals are formed, lead is not found in horizontal seams (like coal) but in vertical veins that represent cracks in the rock. The same vein of lead can therefore be found at the surface or deep underground. Veins which occurred near the surface were the easiest to work, but were likely to be hidden by soil and vegetation. Veins could often be seen in places where the solid rock was exposed, such as cliffs and scars, or in stream bottoms. Artificial streams called “prospecting hushes” were used to remove surface soil and vegetation and locate veins. These “hushes” took water from a stream or a dam and directed it across the presumed line of the vein. The largest mining countries are Australia, China and the United States, which between them account for more than 50% of primary production. In the U.S., the majority of lead was produced in mines located in Missouri, Alaska, Idaho and Montana. Significant amounts of lead are being recovered as a by-product or co-product from zinc mines, and silver-copper deposits. From that amount, about 3 million tons of lead are mined each year.

Lead is also imported into the U.S. from a number of countries, both as ore concentrates and as metallic lead. Canada is the most important importer, followed by Mexico, Australia and

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Peru.\textsuperscript{33} A further 3 million tons of lead are produced from secondary sources each year, by recycling scrap lead products such as sheet, pipe and batteries. In fact, in the western world today more lead is produced by recycling than by mining.\textsuperscript{34} For example, although the U.S. is the largest user of lead (1,488,000 tons in 2003), over two-thirds of this lead came from recycling efforts (1,098,000 tons in 2003).\textsuperscript{35} As a result, while the consumption of lead-containing products has steadily increased over the past 40 years, less and less of the lead is coming from mining operations.\textsuperscript{36}

3. A Historical Perspective of the Use of Lead

i. Ancient Usage

Unlike many of the relatively new products at the center of recent litigation storms (MTBE, silicone breast implants, and various pharmaceutical products), lead has a long history. It is one of the oldest metals used by man.\textsuperscript{37} It is abundant, easily mined, very malleable, corrosion resistant, has poor electrical conductivity and a high density. Through these properties, man used lead to overcome many barriers to technological advancement. Yet, the use of lead came with a price – a price that man knowingly paid for thousands of years. It has long been known that toxic effects from excessive exposure to lead can cause madness, blindness, deafness,
kidney failure and death. These and other harmful effects of lead have been noted by physicians and scientists for nearly 3000 years. Nevertheless, lead is still used in products. Many of the ways man uses lead were devised in the industrial age, centuries after humans learned that lead was poisonous.

The use of lead probably preceded the usage of copper and bronze. It has been estimated that people have been using lead for at least 9000 years. A peat bog on a Swiss mountain allowed researchers to construct an atmospheric lead profile going back 14,500 years. Their data suggests that the release of lead to the atmosphere (probably from soil erosion produced when early peoples cleared land for agriculture) increased over natural levels about 6,000 years ago. Tree and cereal pollen records also suggest that the clearing of forested lands and crop raising commenced at this time. The findings also show large fluctuations that coincide with historical events such as the rise and fall of the Roman Empire and onset of the Industrial Revolution in Europe. For example, at about 1000 BC, the ratio between the two lead isotopes studied changed in a manner consistent with what happens with mining activities. This suggests

38 Lead and You, supra note 21.
39 Id.
40 At the ancient Hittite city of Catal Huyuk in Turkey, beads of lead have been uncovered that date back to about 6500 years BC. N.H. Gale, & Z. Stos-Gale, Lead and Silver in the Ancient Aegean. 244 SCIENTIFIC AM., 176-192 (1981) (The authors use isotopic studies to identify the sources of lead and silver used by the Greeks). Lead has been found in a sixth millennium context at Yarum Tepe in Iraq, at the fifth millennium site of Arpachiyeh in Iraq and at the fourth millennium sites of Anau I in Turkestan, Hissar III in Iraq, and Naqada in Egypt. These finds suggest that lead smelting, albeit on a small scale, began at least as early as nine thousand years ago. In the British Museum in London, the oldest artifact of lead is a lead statuette found at the temple of Osiris on the site of Abydos, and purchased in Egypt in 1899. This object is dated on stylistic grounds to the predynastic period of Egypt [circa 3800 BC]. Chris Winder, The DEVELOPMENTAL NEUROTOXICITY OF LEAD, MTP PRESS LTD. (1984). Small lead nuggets have been found in pre-Columbian Peru, Yucatan and Guatemala, but native North Americans made less use of the metal. Emsley, supra note 20, at 229.
41 William Shotyk, of the University of Berne, and his colleagues analyzed changing levels and sources of lead in the atmosphere over the last 14,000 years by studying layers of peat in an ancient bog in Switzerland’s Jura Mountains. He measured not only the amount of lead in various layers of the bog, but also the ratio of the two isotopes, lead-206 and lead-207. See William Shotyk et al., History of Atmospheric Lead Deposition Since 12,370 14C yr BP from a Peat Bog, Jura Mountains, Switzerland, SCIENCE, 281:1635–1640 (Sept. 1998). While peat bogs and ice cores have been used to analyze atmospheric lead levels for small increments of time and back to several thousand years ago, Shotyk and his colleagues present the first complete record to span approximately 14,000 years – nearly the entire time since the last ice age. They were therefore able to contrast the first 8,000 years of natural lead emissions with the contributions that humans made over the years that followed. Id.
42 Id.
that serious lead mining activity commenced about this time, probably by the Phoenicians, and increased even more in the nineteenth century. The latter increase is associated with Australian mining activity because Australian lead ore contains higher levels of the lead-207 isotope.\textsuperscript{43}

Initially, lead was popular because it was easily found, easy to extract from the earth, and because its relatively low melting point (327.5°C) made it useful. Lead is soft, highly malleable (\textit{i.e.}, it can be hammered into shape) and ductile. Thus, given man’s early tools, lead was very suitable for molding and hammering. Lead is also a poor electrical conductor, it is easily cast, rolled and extruded.\textsuperscript{44} Humans took advantage of these properties as they progressed technologically.

The oldest known manmade object containing lead is a statue found in Turkey, dated 6500 BC. Similar artifacts from other civilizations include a carved metal statue from the Osiris Temple in Egypt, believed to date from the First Dynasty about 3400 BC.\textsuperscript{45} Other finds from ancient periods have been principally statuettes and figures. The Greeks mined lead on a large scale from 650 to 350 BC from a large deposit at Laurion near Athens. Although their aim was to extract silver, of which the mine produced 7,000 tons, it also produced more than 2,000,000 tons of lead. It did not take the Greeks long to find ways to use this “by-product” of silver mining. The ancient Greek writer Theophrastus (372-286 BC) first described using lead as a paint pigment.\textsuperscript{46} The Romans were the first civilization to mine and produce lead on a large

\textsuperscript{43} \textit{Id.}


\textsuperscript{46} EMSLEY, supra note 20, at 229. Lead strips were exposed to vinegar fumes (acetic acid vapor), resulting in the formation of a white deposit after about 10 days. This was scraped off to expose fresh metal and the process was repeated. The white deposit was ground into a powder, boiled in water and left to settle to produce white lead. \textit{Id.}
scale. Their mines were located mainly in Spain and Britain. They used it for water pipes, roofing, cookware, coinage, pewter tableware and as a sweetening agent for wine. They used white lead as a paint pigment, and they used red lead as coloring for interior decoration. After the Dark Ages, lead mining resumed and, in addition to traditional uses, humans used lead for pottery glazes, bullets, printing type and for weatherproofing the roofs of large buildings.\footnote{Id. The earliest use of lead for roofing in America may have been at Rosewell, an 18th-century plantation house at Whitemarsh, Virginia. Lead was widely used before the American Revolution for flashings, downspouts and gutters on the best buildings. In the United States, lead never achieved the popularity it had in Europe. Installed on early Federal buildings in Washington, D.C., it failed to perform well. Expansion and contraction due to temperature changes produced fatigue, and on steep roofs the effect of gravity caused creep. In the 20th century, lead found additional application as a coating for both steel and copper. \textit{From Asbestos to Zinc, Roofing for Historic Buildings}, Nat’l Park Serv., available at \url{http://www.cr.nps.gov/hps/TPS/roofingexhibit/metals2.pdf} (last visited July 21, 2006).}

\section*{ii. The Impact of Landmark Studies in the 1960’s}

Although it was common knowledge before the 1960s that exposure to high levels of lead could cause lead poisoning, is was generally believed that the persons most vulnerable to lead poisoning were workers who either mined or smelted lead, or who worked in trades that applied or manufactured lead-based products, such as painters and workers making tetraethyl lead. Although there was some research associated with the dangers of ingesting lead-based paint before the 1960s and there were efforts among regulators to reduce the amount of lead being used in paint, on the whole lead contamination and lead poisoning was not viewed as a national public health crisis by the general public. Things changed, however, in the 1960s following the work done by a scientist named Clair Patterson.

During the 1950s, Dr. Patterson was working on ways to determine the age of rocks (and consequently, the age of the earth) using new lead-based analytical methods developed in conjunction with the Manhattan project (the project that ushered in the atomic bomb) during the

\footnote{See Randolph K. Byers & Elizabeth E. Lord., \textit{Late Effects Of Lead Poisoning On Mental Development}, 66 Am. J. Diseases Child. 471-494 (1943).}
World War II years.\textsuperscript{49} A problem developed, however, when they tested their methods on rocks whose age they knew. The amount of lead found using the new analytical methods was greater than the amount predicted by the formulas upon which the tests were based (\emph{i.e.}, the experimental results did not fit the calculations). In trying to figure out why this happened, Dr. Patterson discovered that his samples were being contaminated with lead coming from multiple sources not only in his laboratory but \textit{elsewhere}, including from clothing and human hair.\textsuperscript{50} After he successfully contained and accounted for the lead contamination, Dr. Patterson began measuring lead in various rocks to ascertain their ages.\textsuperscript{51}

As part of this project he measured lead in ocean sediments – and he made a startling discovery. He found that the rate of lead being deposited into the ocean was about eighty times greater than the amount deposited “naturally.”\textsuperscript{52} From this observation, Dr. Patterson concluded that human activity had severely raised environmental levels of lead. He then determined that

\begin{footnotesize}
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  \item \textsuperscript{49} At that time Dr. Patterson was trying to determine the age of rocks by measuring the amounts and types of uranium and lead in common ordinary igneous rocks using mass spectrometers. \textit{See} George R. Tilton, Biographical Memoirs of Clair Cameron Patterson (1922-1995), Nat’l Academy Sci., \textit{available at} \url{http://newton.nap.edu/html/biomems/cpatterson.html} (last visited Aug. 18, 2006); Interview with Clair C. Patterson, Oral History Project, Calif. Inst. of Tech. Archives, Pasadena, Calif. 14-15 (1997), \textit{available at} \url{http://oralhistories.library.caltech.edu/32/} (last visited Aug. 18, 2006) [hereinafter Patterson Interview].

  \item \textsuperscript{50} Patterson Interview, \textit{supra} note 49, at 17 (stating that it took him years to figure out where the lead was coming from and how to remove that lead contamination). As a consequence of the contamination, Dr. Patterson conducted his experiments in an ultra-clean chamber entered through an airlock in which the air was filtered, the experimenters gowned and masked, and the reagents and water supply purified of any trace of lead. By these measures he established the true concentrations of lead in his samples. Herbert L. Needleman, \textit{The Removal of Lead from Gasoline: Historical and Personal Reflections}, 84 ENV’T L. RES. 20-35, 22 (2000).

  \item \textsuperscript{51} In 1953 Patterson became the first person to accurately determine that age of the earth (4.5 billion - later refined to 4.55 billion years) using a meteorite to measure the isotopic composition of primordial lead. The new age was substantially older than the commonly quoted age of 3.3 billion years, which was based on tenuous modeling of terrestrial lead evolution from galena deposits. Tilton, \textit{supra} note 49. \textit{See also} Clair C. Patterson \textit{et al.}, \textit{Age of the Earth}, 212 Sci. 69-75 (1955); Clair C. Patterson, \textit{The Isotopic Composition of Meteorite, Basaltic and Oceanic Leads, and the Age of the Earth}, in Proceedings of the Conference on Nuclear Processes in Geologic Settings, Williams Bay, Wisconsin 36-40 (Sept. 21-23, 1953).

  \item \textsuperscript{52} Tilton, \textit{supra} note 49. The limitations in the analytical data on which many of the conclusions in Patterson’s 1962 paper were based led him to start new investigations to attack the problem. In 1963 he published a report with Mitsunobu Tatsumoto showing that deep ocean water contained 3 to 10 times less lead than surface water, the reverse of the trend for most elements (\emph{e.g.}, barium). This provided new evidence for disturbance in the balance of the natural geochemical cycle for lead by anthropogenic lead input. \textit{Id.}
\end{itemize}
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the additional amount of lead being placed into the oceans was about equal to the amount of lead being burned in gasoline and emitted into the air.\textsuperscript{53}

In 1965, Dr. Patterson published a controversial paper in which he challenged the prevailing views held by the scientific community regarding the presence of lead in the environment. Prior to Dr. Patterson’s paper, most scientists believed that industrial lead usage increased environmental lead levels over “natural” levels by no more than a factor of two.\textsuperscript{54} Dr. Patterson posited, however, that the prevailing view was the result of poor quality of lead analyses in prehistoric comparison samples. Specifically, he asserted that the “natural” level of lead in the environment was \textit{overestimated} because of the same types of contamination he encountered in his laboratory. To better understand the lead burden being placed into the environment by human usage, Dr. Patterson studied and analyzed the amounts of industrial lead entering the environment from gasoline, solder, paint, and pesticides – and demonstrated that they involved substantial quantities of lead compared to the expected “natural” flux. He then estimated the lead concentration in blood for many Americans to be over 100 times that of the “natural” level, and within about a factor of two of the accepted limits where symptoms of lead poisoning could be expected to occur.\textsuperscript{55}

Dr. Patterson’s paper fundamentally changed the lead debate. The accepted wisdom at the time was that the lead levels observed in the human body were “normal” levels of lead in blood.\textsuperscript{56} Moreover, the literature at the time indicated that the main group of people subject to

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\item \textsuperscript{53} Patterson Interview, \textit{supra} note 49, at 32.
\item \textsuperscript{54} Tilton, \textit{supra} note 49. He maintained that the belief arose from the poor quality of lead analyses in prehistoric comparison samples in which much of the lead reported was actually due to underestimation of blank contamination. \textit{See} Clair C. Patterson, \textit{Contaminated And Natural Environments Of Man}, 11 ARCHIVES ENVTL. HEALTH 344-60 (1965).
\item \textsuperscript{55} Tilton, \textit{supra} note 49; Patterson Interview, \textit{supra} note 49, at 40.
\item \textsuperscript{56} From the 1920s until the 1960s, the leading toxicologist on the health hazards posed by lead was Dr. Robert Kehoe. Dr. Kehoe was hired by General Motors to study the health effects posed by lead. Initially, Dr. Kehoe compared lead concentrations in workers in direct contact with tetraethyl lead to men in the same plant with other assignments (the unexposed
lead-based health hazards were factory workers. Stated simply, at the time Dr. Patterson’s research was published, leading scientists believed that “normal” meant “natural.” Based on Dr. Patterson’s experiences with lead contamination, however, he concluded that “normal” was actually aberrant – a result of lead contamination occurring on a global scale.

As a result of Dr. Patterson’s work and the work of scientists who followed him who suggested that the public was being harmed by exposure to doses of lead in the environment at levels less than the amount known to cause clinical lead poisoning, the government began to address the dangers posed by lead in the environment. Among the steps taken, the government:

- phased out the use of tetraethyl lead in gasoline;
- banned most uses of lead-based paint;
- required most industries to monitor for potential exposure to harmful levels of lead and to take proactive steps to protect workers from harmful exposures;
- determined a health-based limit for the amount of lead in the atmosphere and gave the Environmental Protection Agency responsibility for ensuring that limits were not exceeded by industry or individuals.

control group). Because he found lead in the excreta of the control group he concluded that that lead was naturally present in everyone. Because his work was criticized for using workers from the same factory as his control group, Dr. Kehoe created a new control group consisting of people from a remote farming village outside Mexico City, removed from industry or urban pollution. There he sampled food, utensils and the excreta of the residents. Like the members of his earlier control, the farmers in this remote area also had lead in their excreta. From this he constructed a case that lead in gasoline presented no danger and that the general concern about lead as a health threat was overstated. Needleman, supra note 50, at 20-21.

57 Patterson Interview, supra note 49, at 40.
58 Needleman, supra note 50, at 23. Dr. Kehoe’s “unexposed” subjects from the factory who did not directly handle TEL breathed it, and the food of Kehoe’s “unexposed” Mexican farmers had been cooked in and served from leaded ceramic pots and plates. Id.
60 See infra section IV.A.1.iv.
61 See infra section IV.D.1.
62 See infra section IV.B
63 See infra section IV.A.2.
created regulations specifically designed to address lead emissions for lead smelters and lead recyclers;\(^{64}\)

- promulgated regulations requiring most landlords and home sellers to notify prospective tenants and buyers of known lead paint hazards associated with the property being considered for lease/purchase;\(^{65}\)

- began monitoring products sold in the United States for potential lead hazards and warning the public of dangerous products;\(^{66}\)

- limited the amount of lead that can be used in plumbing fixtures;\(^{67}\)

- commenced monitoring the level of lead in the water in household taps and set action level at which the water system must take corrective actions;\(^{68}\)

- required industrial facilities to annually report the estimated amount of lead that is released into the air, water or disposed as a waste product;\(^{69}\)

- listed lead and lead compounds as a hazardous constituent of waste under RCRA;\(^{70}\)

- required that spills in excess of 10 pounds for most lead compounds and 1 pound for lead arsenate be reported under CERCLA;\(^{71}\) and

- listed numerous lead compounds as hazardous materials subject to special regulations when being transported or shipped across the country.\(^{72}\)

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\(^{64}\) See infra section IV.A.3

\(^{65}\) See infra section IV.C

\(^{66}\) See infra section IV.D.

\(^{67}\) See infra section IV.E.

\(^{68}\) See infra section IV.E.

\(^{69}\) See 40 C.F.R. Part 261. Lead and lead compounds are considered a toxic hazardous waste with a Toxicity Characteristic Leaching Procedure (“TCLP”) threshold of 5.0 mg/L. Waste codes in which listing is based wholly or partly on lead or lead compounds include: F035, F037, F038, K002, K003, K005, K046, K048, K049, K051, K052, K061, K062, K064, K069, K086, K100, P110, U144, U145, and U146. Id.


\(^{71}\) See 49 C.F.R. §172 (2006). Lead and many lead compounds are considered hazardous materials and requirements have been prescribed for shipping papers, package marking, labeling and transport vehicle placarding for their shipment and transportation. See Id.
iii. The Lead Success Story

As a result of these pervasive government interventions, lead is now one of the most highly regulated substances in the U.S. Although many of the programs listed above will be discussed in greater detail in this article, the most significant step taken by the government was phasing out the use of leaded gasoline. This program began in the late 1970s and the government intensified those efforts in 1990. Since 1978, the amount of lead emitted into the air nationally has markedly declined. Airborne concentrations in the U.S. fell an average of 94% between 1983 and 2002 and 57% between 1993 and 2002, and the EPA considers this reduction one of the great successes for public and environmental health.\(^73\) Not surprisingly, this drastic decrease in lead air emissions also caused an extraordinarily decreased in the amount of lead found in humans.

When measuring a person’s level of exposure to lead, most experts analyze the level of lead in a person’s blood. Likewise, of the various groups of people analyzed, most experts first look at the blood lead levels (“BLL”) of children – considered one of the most lead-sensitive groups. Eliminating BLLs above 10 µg/dL in children is one of the national health objectives for 2010.\(^74\) Around 1975-76, when the government began to regulate lead, the average BLL in children under the age of 6 in the U.S. was about 16.5 µg/dL.\(^75\) While this is well below the level of clinical lead poisoning (80 µg/dL) and below the Center for Disease Control’s (“CDC”)

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\(^73\) Criteria Document, *supra* note 44, at E-5 & 2-44 (citation omitted).


current suggested action level (20 μg/dL), it is 65 percent higher than the CDC’s suggested level of concern for over-exposure to lead (10 μg/dL).  

By 1996, when the phase-out of leaded gasoline was complete, the average BLL for children under the age of 6 had dropped to 2.7 μg/dL. This represents a steep decline (from 78.8% to 4.4%) of children who had BLL above 10 μg/dL. The figure below shows the correlation between the phase-out of leaded gasoline and the decrease in BLLs in humans.

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77 Criteria Document, supra note 44, at 4-21. In lead toxicologic and epidemiologic studies, dose-response relationships for nearly all of the major health effects of lead are typically expressed in terms of an index of internal lead dose. Id. at 4-1. During the 1991 to 1994 time period, the geometric mean blood lead concentration of U.S. adults (ages 20 to 49 years), was 2.1 μg/dL. Among adults 70 years of age and older, the blood lead concentration was 3.4 μg/dL. Id. at 4-21.

78 1999-2002 BLLs, supra note 74.

According to the most recent data available (collected during 2001-2002), the average BLL for children in the U.S. continued to decrease to 1.70 μg/dL.\textsuperscript{80} Thus, as a consequence of the governmental intervention, the level of lead in the blood of young children is 90 percent less today than it was 30 years ago. Today, only about 310,000 children aged 1-5 years are considered to be at risk for exposure to harmful lead levels (10 μg/dL). Another critical factor in reducing BLLs in children has been the decline in the number of U.S. homes with lead-based paint, from an estimated 64 million in 1990 to 38 million in 2000.\textsuperscript{81}

\textsuperscript{80} Criteria Document, supra note 44, at 4-22.

\textsuperscript{81} 1999-2002 BLLs, supra note 74. (citing D.E. Jacobs \textit{et al.}, \textit{The Prevalence Of Lead-Based Paint Hazards In US Housing}, 110 ENVTL. HEALTH PERSPECTIVES 599-06. (2002)).
4. **Modern Usage**

Lead has many uses both as a primary metal and as a metal compound. Over the last 50 years and particularly by the mid-1980s, many of the traditional uses of lead declined or were banned as a result of the U.S. lead consumers’ compliance with environmental regulations. Contrary to popular belief, however, the demise of lead-based products has been overstated.Handled with care and knowledge of its toxic potential, lead remains a useful product.

Today, lead’s high resistance to corrosion makes it ideal for weatherproofing buildings and for equipment used in the manufacture of acids. Lead’s high density makes it particularly appropriate as a shield against radiation in the nuclear industry and in hospitals. For these same reasons, lead is also useful for stopping sound waves, and is therefore used to reduce noise from machinery in factories and from engine rooms on ships. One of the most important uses of lead today is in the lead-acid battery which provides power in numerous situations. The most familiar use of lead-acid batteries is for cars and other gasoline-powered vehicles, but such batteries are also used to power electric vehicles and to provide emergency electricity during power failures.

Lead also facilitates high technology. For example, without the use of lead solders and leaded glass, humans could not safely use their computers. No other means of connecting transistors, relays and other electronic components is as reliable. Lead alloy solders enable computers to send electronic data. Lead also plays a vital role in space exploration, energy conservation and telecommunications. Lead-based materials are facilitating the development of hyper-fast computers and high definition TV, as well as cathode ray tubes used in viewing screens for television, computers and radar. NASA’s Space Shuttle uses lead-alloy solder. Lead glazes are used on the Shuttle to encapsulate and protect the latest generation of electronic
microcircuits from atmospheric corrosion.\textsuperscript{82} Specific information about a number of current products that contain lead is discussed below.

i. Batteries

By far the greatest use of lead worldwide is in lead-acid batteries.\textsuperscript{83} Nearly all large rechargeable batteries in common use are the lead-acid type.\textsuperscript{84} There are some Nickel-Cadmium batteries in use, but for most purposes the very high initial expense, and the high expense of disposal, does not justify them.\textsuperscript{85} Lead-acid batteries are used in motor vehicles, electric-powered or hybrid vehicles (golf carts, airport support vehicles and new hybrid cars), and also in some situations such as computer and telecommunication systems, and smaller numbers in other installations including hospitals, which require instant emergency back-up power in case of power failure. Lead is particularly well suited for use in acid batteries because of its conductivity and its resistance to corrosion. Batteries have been the greatest consumer of lead since the 1960s, but their importance has risen dramatically. In 1960, batteries accounted for 28\% of lead use worldwide. By 1999, 74\% of the total amount of lead consumed was for this application.\textsuperscript{86}


\textsuperscript{83} In 2005, 1,280,000 metric tons of lead were used in the making of these products for use in the United States. This represents about 90\% of U.S. lead consumption. \textit{Lead in March 2006}, Mineral Industry Surveys, United States Geological Survey (2006).

\textsuperscript{84} Lead acid batteries were invented in 1859 by Gaston Planté and first demonstrated to the French Academy of Sciences in 1860. They remain the technology of choice for automotive SLI (Starting, Lighting and Ignition) applications because they are robust, tolerant to abuse, tried and tested and because of their low cost. Lead-acid batteries are composed of a lead-dioxide cathode, a sponge metallic lead anode and a sulphuric acid solution electrolyte. The cell voltage is 2 Volts. See \textit{Lead-acid Batteries}, MPower Solutions Ltd, available at \url{http://www.mpoweruk.com/leadacId.htm} (last visited July 26, 2006) (a website containing extensive information about battery technology and applications).

\textsuperscript{85} \textit{Battery Construction Materials}, Northern Arizona Wind & Sun, Inc, available at \url{http://www.windsun.com/Batteries/Battery_FAQ.htm#Major%20Battery%20Types} (last visited July 26, 2006). The acid is typically 30\% Sulfuric acid and 70\% water at full charge. \textit{Id}.

\textsuperscript{86} THORNTON, supra note 28 at 20-21 (citing \textit{Principle Uses of Lead and Zinc}, International Lead Zinc Study Group (2001)). In Western Europe, 57\% of the lead consumed is used in batteries (1999 data, based on total consumption of refined lead, and consumption of lead for batteries, in most of the countries in Western Europe, which account for 95\% of total consumption.). In the U.S., over 80\% of lead is used in batteries (1999 data). \textit{Id}. See also DOE Handbook: Primer on Lead-Acid Storage Batteries, DOE-HDBK-1084-95, U.S. Department of Energy (Sept. 1995).
Historically, the usual alloy used in making batteries was a lead-antimony combination (0.75 - 5% antimony). This alloy was much stronger than pure lead. Minor additions of other elements, such as copper, tin, arsenic and selenium can also be made to improve grain refinement, ease of casting and impart age-hardening characteristics. For starter batteries, the more modern sealed maintenance-free batteries use a lead-calcium-(tin)-based alloy, containing up to 0.1% calcium, and from 0 to 0.5% tin. The tin is added to improve corrosion resistance. These systems are beginning to predominate the marketplace, particularly in the larger, more mature, markets in the U.S. and Europe.\(^\text{87}\)

ii. Ammunition, Shot and Bullets

Lead has been used in the manufacture of bullets, cannon balls and the like for many centuries. Today, these products account for the second largest use of lead in the U.S.\(^\text{88}\) Munitions, expended in hunting and recreational shooting, presently constitute the largest flux of lead into the environment. It is estimated that presently there are about 3.5 million metric tons of spent lead from these sources in the environment and that amount increases by about 55,000 metric tons per year in the U.S. alone.\(^\text{89}\) Although detailed records of the use of lead-bearing munitions in the U.S. date only from the 1920s, it is clear that hunting activities were spreading large quantities of lead throughout the environment by the mid-1800s.\(^\text{90}\)

\(^{87}\) Id. at 22.

\(^{88}\) In 2005, 60,000 metric tons of lead were used in the making of these products. This equates to about 4% of lead consumption. Lead in March 2006, supra note 83.

\(^{89}\) David H. Edwards, Lead Distribution at a Public Shooting Range 1 (2002) (unpublished masters thesis, Virginia Polytechnic Institute) available at http://scholar.lib.vt.edu/theses/available/etd-09142002-113412/unrestricted/thesis.pdf (last visited July 27, 2006). The total amount of lead reported as processed into munitions (excluding military usage) from 1920 through 1997 was 3,540,000 metric tons (3,900,000 short tons). Reasonable extrapolations back to 1900 and through the end of 1999 give total lead usage greater than 4,100,000 metric tons (4,500,000 short tons) of expended munitions during the 20th Century. Although there is some usage of indoor shooting, the vast proportion of the lead has been expended into the environment with no effort at recovery. Accordingly, the release of lead in the form of spent munitions represents the major pathway by which lead is released into the American environment, and those munitions represent, by far, the most rapidly growing reservoir of lead. Id. at 4.

\(^{90}\) Id. at 4.
Ever since the development of the cannon and other firearms, lead has been the metal of choice for the projectiles because it was dense, cheap, was easily shaped, did not damage the barrels, carried well and deformed on impact. Because of its high density, leaden projectiles have greater momentum, and so greater destructive power and longer range, than similar missiles of less dense materials. Consequently, lead in the form of spent munitions has been spread around the world as the result of military activities, hunting and recreational shooting for hundreds of years.91

iii. Lead Sheet and Pipe

The next most important use of lead is for the manufacture of lead sheet and pipe. Lead sheet is widely used in the building, construction and chemical industries. Because of its versatility, man has found many applications for the use of lead. As a result, the worldwide annual demand of lead sheeting and pipe was about 265,000 tons in 1999.92 Lead pipe was once a significant product but is now produced only in very small quantities, exclusively for use by the chemical industry.

In the building and construction industry, lead sheeting is typically used for flashings, gutters, downspouts and conductor heads. Some 85% of lead sheet demand is for these types of applications, with lead’s durability and malleability providing unique properties to weather seal buildings where chimney stacks, windows and abutments are adjacent to roof lines and vertical walls.93 The material is extremely easy to install (assuming basic skill and design techniques are adopted), and once installed it is resistant to wind lift due to its high density.94

91 Id. at 2.
92 THORNTON, supra note 28, at 24.
93 One of the largest applications of lead for waterproofing purposes in recent times was at New York City’s massive World Trade Center Plaza. Approximately 700,000 lbs. of lead sheet were installed in critical areas around the huge center for moisture protection. This enormous use of lead included what is believed to be two record setting applications: (1) the most lead used to flash the base of buildings-about 72,000 lbs and (2) the most lead used in a single installation to line landscaping planters-
In Europe, another important use of lead sheet is for complete roofing systems and for vertical cladding of walls. Though more expensive initially, its long life - over 100 years is quite feasible for correctly installed lead - and low maintenance requirement, can make the long-term costs comparable with, or even lower than, some cheaper alternatives, as other (non-metallic) materials need replacement after about 20 to 50 years.\textsuperscript{95}

Lead sheet is used to a lesser degree for radiation shielding\textsuperscript{96} (27\% of sheet consumption in the U.S.), noise attenuation and soundproofing.\textsuperscript{97} The properties of lead which make it an excellent radiation shielding material are its density, high atomic number, high level of stability, ease of fabrication, high degree of flexibility in application and its availability. For noise reduction inside buildings, layers of lead incorporated into walls can be used as part of a system for sound insulation, particularly where space constraints do not allow thicker walls of cheaper materials. Because of its inherent limpness, or softness, lead cannot be easily set in vibration. It does not resonate or “ring,” hence it cannot radiate sound. The softness and formability of lead allow it to be shaped exactly as required, so an integral sound barrier can be constructed.\textsuperscript{98} Layers of lead sheet can be used to provide watertight linings for tanks and vessels. This application is occasionally used in the building industry, where complete integrity and lack of maintenance are required. However, it is more commonly used by the chemical industry for the

\begin{footnotesize}
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\item 94 THORNTON, supra note 28 at 25.
\item 96 Theoretically, almost any materials can be used for radiation shielding if employed in a thickness sufficient to attenuate the radiation to safe limits. However, due to certain characteristics, lead and concrete are among the most commonly used materials. Radiation Shielding, Lead Industries Association, Inc., available at http://www.leadinfo.com/ARCH/water.html (last visited July 26, 2006).
\item 97 THORNTON, supra note 28, at 26.
\end{itemize}
\end{footnotesize}
lining of tanks and other vessels exposed to corrosive contents such as sulfuric acid, because of its good corrosion resistance compared with other materials.99

iv. Solder

Historically, soldering is the process of heating a lead-tin alloy to its melting point, which then forms a pliable metal paste that binds electrical components or metal pieces together.100 While it does not generally give the same strength as welding or mechanical joining, soldering has the advantages that it is easy to apply, does not require very high temperatures and a completely sealed joint is possible. The lead in the lead-tin alloy is essential to solder performance. While it is possible to solder with other metals, lead carries crucial and unique advantages over the alternatives. Lead-tin alloy melts at the relatively low temperature of 183 degrees Celsius. Other metal alloys must reach at least 206 degrees to melt. Soldering at the higher temperatures can damage computer circuitry components.101

v. Children’s Toys

Just recently, it has been discovered that toys given to children by libraries across the nation as reading prizes this summer may have unacceptable levels of lead.102 The libraries ordered the toys through Highsmith Publications, a Wisconsin-based company that distributes supplies, furniture and equipment to libraries. Highsmith gave the toys to the Collaborative


101 THORNTON, supra note 28, at 33. Solders are used in many applications, but the largest is in the electronics industry, which consumes approximately 60,000 tons of leaded solder worldwide per year. Tin-lead alloys have low melting temperatures - a eutectic (an alloy containing two or more constituents, which has a minimum melting temperature at an exact composition) mixture of 38% lead and 62% tin melts completely at 183°C (which is below the melting temperature of either tin or lead.) This liquid can penetrate minute openings, and is suitable for soldering components easily damaged by heat.

102 Jeri Krentz, After Test Finds Lead In Toys, Libraries Taking Back Prizes; Flexible Dogs And Cats Exceed Level Of Lead Allowed By Federal Rules, CHARLOTTE OBSERVER (Aug. 10, 2006); see also, Courtney Bacalso, Toy Risk Feared, ORANGE COUNTY REGISTER (CALIFORNIA) (Aug. 9, 2006).
Summer Library Program, whose members include about 1,000 libraries in 36 states. According to a Highsmith official, a sample of “Bendable Cat and Dog” tested by the Indiana State Department of Health “indicated the lead content to be in excess of the maximum allowable by current federal regulations.”

vi. Other Applications and Former Applications

There are various other uses for lead in products and processes, some of which continue in effect today, and others which have been partially or wholly banned or discontinued. These are briefly set forth below:

- Lead is used as a coloring element in ceramic glazes used in common tableware, notably in the colors red and yellow.104

- Lead is used in some candles to treat the wick to ensure a longer, more even burn. Some candles that were tested emitted lead levels seven times the rate that could lead to elevated levels of lead in a child.105

- Molten lead is used as a coolant for lead cooled fast reactors.106

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103 See Bacalso, supra note 102. A different batch of the 3 3/4 -inch-long bendable cat and dog toys from an Indiana-based distributor contained 0.24 percent to 0.4 percent lead in its plastic and paint, well past the federal safety limit of 0.06 percent. Id.

104 Michael McCann, Lead Glazes in the Americas, 18 ART HAZARD NEWS 2 (1995). The use of ceramic lead glazes by potters in making utilitarian ware such as eating, drinking, cooking and food storage vessels continues to be a major problem. The problem of lead poisoning from glazed pottery imported from Mexico and other developing countries is well known. It is the main cause of lead poisoning in Mexico, and in April 1995, a child died of lead poisoning from leaded ceramic ware in Mexico. These cases of severe lead poisoning are due to very high levels of lead in the glazes (as much as 75-85%) and poor firing conditions, that result in glazes that leach a lot of lead. Id. Fired glazes can leach lead if the glaze has not been fired high enough to bind the lead with other glaze components, forming lead bisilicate. Any acidic foods or liquids subsequently used with such glazes, e.g., fruit juices, will dissolve unbound lead particles from the glaze surface. In extreme cases this could create a lethal dose of lead. Lead: a once common glaze ingredient, Ceramics Today, available at http://www.ceramicstoday.com/articles/lead.htm (last visited July 27, 2006).

105 Although the U.S. candle-making industry agreed to voluntarily remove lead from candle wicks in the 1970s, a small percentage of candles sold still contained lead-cored wicks. Some of these lead-cored wicks could emit relatively large amounts of lead into the air during burning. According to the U.S. Consumer Product Safety Commission (“CPSC”), these candles place children at risk because they may inhale the vaporized lead or they may mouth objects on which lead has settled or handle such objects and then place their hands in their mouths. CPSC Bans Candles With Lead-Cored Wicks, U.S. CONSUMER PRODUCT SAFETY COMMISSION (2003). Some candles that were tested emitted lead levels in excess of 3,000 micrograms per hour - about seven times the rate that could lead to elevated levels of lead in a child. CPSC estimates that an indoor air lead level of 430 micrograms per hour from burning candles could result in hazardous exposure to children.. Id. Consequently, in 2003, the CPSC banned the manufacture and sale of lead-cored wicks and candles with lead-cored wicks. The federal ban applies to all domestic and imported candles.

106 One of the designs of the new generation of nuclear reactors uses lead as its primary coolant. See generally Generation IV Nuclear Energy Systems; Lead Cooled Fast Reactor (LFR), Idaho National Laboratory (2005) The LFR system features a fast-spectrum lead or lead/bismuth eutectic liquid metal-cooled reactor and a closed fuel cycle for efficient conversion of fertile uranium and management of actinides. Id.
• Lead is used to absorb the shocks suffered by a building during earthquakes.\textsuperscript{107}

• Lead glass is composed of 12-28% lead. It changes the optical characteristics of the glass and reduces the transmission of radiation.\textsuperscript{108}

• Lead is the traditional base metal of organ pipes, mixed with varying amounts of tin to control the tone of the pipe.\textsuperscript{109}

• Lead is used in high-voltage power cables as sheathing material to prevent water diffusion into insulation.\textsuperscript{110}

• Lead is used for the ballast keel of sailboats and as counterweights for grandfather clocks and elevators.\textsuperscript{111}

• Lead is used as a component of children’s jewelry.\textsuperscript{112}

\textsuperscript{107} \textit{Lead and You}, supra note 21. Lead is used as the core in a invention called base isolators. The isolators reduce the severity of the shaking felt inside the building in the event of a major earthquake by as much as 80%. Base isolators are large rubber blocks laminated with steel, with pure lead columns inside which act as an earthquake shock absorber. During an earthquake the lead deforms and is stretched sideways as the earth shakes. It is able to move sideways to a distance equal to its height - a few hundred millimeters being ample for even the most powerful of earthquakes. The rubber and steel then pulls it back into shape ready for the next shock. \textit{Id.}

\textsuperscript{108} Lead-glass incorporates lead in the form of lead oxide into the glass during manufacture. The lead oxide has a small negative effect on the transmission of visible light through the glass. The lead-glass effectiveness in stopping x-rays is a result of lead’s high atomic number; much higher than the normal constituents in glass. George Chabot, \textit{Lead-Glass Windows Provided in an X-Ray Room are Transparent to Light, But How Are They Made to be Radiopaque?}, Health Physics Society (2004) available at \url{http://hps.org/publicinformation/ate/q4073.html} (last visited July 28, 2006); Phil Rudolph, \textit{Leaded Glass for Shields}, Oak Ridge Associated Universities (2003), available at \url{http://www.orau.org/PTP/collection/Lichtenberg%20figures/leadglasseshield} (last visited July 28, 2006).

\textsuperscript{109} Organ pipes can contain 50% lead or lead-tin alloy. Alan Cowell, \textit{Europe Declares Pipe Organs Health Hazards}, N.Y. TIMES (INT’L) (Mar. 22, 2006). Lead is used in organ pipes because of its malleability and the distinctive sound it produces. Ruth Gledhill, \textit{Requiem For Church Organs}, THE TIMES (Mar. 18, 2006).

\textsuperscript{110} Lead-shielded cables are used in the petrochemical industry, underwater (for example to supply electricity to islands), and for underground high voltage cables. Most countries in Western Europe use between 1 and 3% of their lead for this purpose. Lead is used because it is completely impervious to water; it has very good corrosion resistance in a variety of media, including marine environments; it can be extruded in very long lengths, and also easily jointed by soldering; it is pliable, so it can be coiled and uncoiled, without being damaged, during cable preparation, transport and application; and it can be applied to the cable core at temperatures which do not damage vital cable components. \textit{THORNTON, supra} note 28, at 28.

\textsuperscript{111} Its high weight-to-volume ratio allows it to counterbalance the heeling effect of wind on the sails while at the same time occupying a small volume and thus offering the least underwater resistance. See generally \textit{Lead Ballast}, American Tin & Solder, available at \url{http://www.american-tinandsolder.com/lead-ballast.htm} (last visited July 28, 2006).

\textsuperscript{112} Cheap toy jewelry (rings, necklaces and bracelets) sold in vending machines, at amusement parks and through major retailers have been found to contain high levels of lead. In fact, four toy jewelry importers have recalled 150 million pieces of toy jewelry sold in vending machines nationwide from January 2002 through June 2004, because the U.S. Consumer Product Safety Commission determined that some of this toy jewelry contained dangerous levels of lead. While only about half the recalled jewelry actually contained lead, the industry recalled all of it because it is difficult to distinguish the lead jewelry from the non-lead jewelry. \textit{Lead Warning; Neighborhood Safety Network Program}, U.S. Consumer Product Safety Commission, available at \url{http://www.cpsc.gov/npn/toyjewel.pdf} (last visited July 28, 2006). \textit{See also} Knight Ridder News, \textit{Lead In Children’s Jewelry: Dangerous, Researchers Say}, BILLINGS GAZETTE (Dec. 15, 2004). According to researchers who tested children’s jewelry for lead, most bracelets, rings, necklaces and earrings bought from big chain stores leached enough lead to cause minor neurological damage with just 20 seconds of daily contact. One ring leached lead at 250 times the federal limit of daily exposure.
• Lead is used in some non-Western cosmetics, such as surma and kohl and some types of hair colorants, cosmetics and dyes contain lead acetate.\textsuperscript{113}

• Lead was used as a pigment in lead paint for white as well as yellow and red colors.\textsuperscript{114}

• Tetraethyl lead was used in leaded fuels to reduce engine knocking.\textsuperscript{115}

• Lead was a component in many household putties used to fill cracks in timber, knots and nail holes as well as for sealing glass to window frames.\textsuperscript{116}

III.

LEAD TOXICITY

A. LEAD THE POISON

Historically, lead is one of the most studied toxic substances. As a result we know more about the adverse health effects associated with high level exposures to lead than virtually any other chemical.\textsuperscript{117} It has been well known for many centuries that excessive intake of lead can


\textsuperscript{114} Many compounds of lead are strongly colored and highly durable. Therefore, they have a long history of application in paints, pigments, and even cosmetics. Some lead compounds are added to paints as drying agents. These are very efficient through-dryers in alkyd-based air-drying paints. Typical lead contents are 0.1-0.5% in paints ready for use. The main compounds of interest are: powdered lead metal for heavy-duty corrosion protection; white lead because it can be formulated into paint which is very durable and has good external weathering properties; oxides of lead as additives to glass and PVC; red lead was historically used in paints because of its color and as an anti-corrosive pigment in rust-inhibiting primers used for the protection of steelwork; and calcium plumbate which is very effective on galvanized steel, as it gives good corrosion resistance. \textsuperscript{115} THORNTON, supra note 28, at 40-41.

\textsuperscript{115} In gasoline, the higher the octane rating the smoother the gas burns. A low octane number gasoline can cause “knocking” during combustion, giving poorer performance and causing engine damage. Adding lead compounds to gasoline increases the octane number. The addition of lead to gasoline in the 1920s was hailed as a great success at the time, as it allowed good performance from fuel, without the need for sophisticated refining, which would make the fuel much more expensive. An additional benefit was that metallic lead deposited on the valve seats can act as a lubricant. \textsuperscript{116} THORNTON, supra note 28, at 45. In America and other parts of the world, leaded gasoline has been phased out. However, it is still being used in some countries in eastern Europe, in the newly independent states and the Russian Federation, and in some Asian countries (though in China, the authorities have recently restricted the use of leaded petrol in Beijing). Id.

\textsuperscript{116} Mike van Alphen, \textit{Paint Film Components: National Environmental Health Monographs, National Environmental Health Forum Monographs, General Series No. 2, Public and Environmental Health Service, Australian Department of Human Services 98 (1998).}

\textsuperscript{117} When Mathieu J. B. Orfila wrote the first known book on Toxicology, “Traité des poisons” in 1813, lead poisoning was one of the six major classifications of poisons (as an astringent poison). Mathieu J.B. Orfila, \textit{TRAITÉ DES POISONS}, Paris: Crochard, (1814). This was the first scientific study of the detection and pathological effects of poisons, and it
make people sick and that very high doses are fatal. The health problems caused by lead have been well documented over a wide range of exposures on every continent. Recent advancements in technology make it possible to research lead exposure down to very low levels approaching the limits of detection. We clearly know most of the way that lead gets into the body but the harm lead causes at the low levels of exposure currently under research (blood lead levels of less than 10 μg/dL) is anything but clear.

According to the Agency for Toxic Substances and Disease Registry (“ATSDR”):

Lead serves no useful purpose in the human body, and its presence in the body can lead to toxic effects, regardless of exposure pathway. Lead toxicity can affect every organ system. On a molecular level, proposed mechanisms for toxicity involve fundamental biochemical processes. These include lead’s ability to inhibit or mimic the actions of calcium (which can affect calcium-dependent or related processes) and to interact with proteins (including those with sulfhydryl, amine, phosphate, and carboxyl groups).

- Acute high lead exposure can cause serious physiologic effects, including death or long-term damage to brain function and organ systems.
- Effects of lead exposure vary according to exposure timing and levels, and other factors, and some effects may be latent. 118

The frequency and severity of medical symptoms increase with the concentration of lead in the blood. Clinical symptoms of lead poisoning appear at blood lead levels (“BLLs”) of 80 μg/dL or greater, and symptomatic lead poisoning may appear at BLLs of 50-60 μg/dL,

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particularly in the presence of anemia, although in some individuals symptoms may be so mild that they are overlooked.\(^{119}\)

Common symptoms of acute lead poisoning are loss of appetite, nausea, vomiting, stomach cramps, constipation, difficulty in sleeping, fatigue, moodiness, headache, joint or muscle aches, anemia, and decreased sexual drive. Severe health effects of acute lead exposure include damage to the nervous system, including wrist or foot drop, tremors, and convulsions or seizures.\(^{120}\)

Chronic lead poisoning may result after lead has accumulated in the body over time, mostly in the bone. Long after exposure has ceased, some physiological event such as illness or pregnancy may release this stored lead from the bone and produce adverse health effects such as impaired hemoglobin synthesis, alteration in the central and peripheral nervous systems, hypertension, effects on male and female reproductive systems, and damage to the developing fetus. These health effects may occur at BLLs below 50 µg/dL.\(^{121}\)

Today, most government agencies refer to any child with a BLL above 10 µg/dL as having lead poisoning. While perhaps technically accurate (\textit{i.e.}, \textit{poisoning} is the act of taking in any substance that is injurious to health or dangerous to life\(^{122}\)), this loose use of the term “lead poisoning” ignores the specific level of harm at issue. As stated above, clinical symptoms of lead poisoning appear at BLLs of 80 µg/dL or greater and symptomatic lead poisoning may appear at BLLs of 50-60 µg/dL. Thus, a child with a BLL of 10 µg/dL is not suffering from


\(^{120}\) \textit{Id.}

\(^{121}\) \textit{Id.}

\(^{122}\) \textsc{Stedman’s Medical Dictionary} 1398 (26th ed.1995).
either chronic or acute blood poisoning. Obviously, at 10 µg/dL, government agencies are not concerned with classical examples of lead poisoning. Instead, the are concerned with subclinical manifestations associated with exposure to lead. Specifically, the government agencies are concerned about the possibility that low level exposure to lead may affect a child’s IQ.

B. HISTORICAL KNOWLEDGE OF LEAD POISONING

Lead poisoning (acute and chronic) is one of the oldest known forms of occupational and environmental disease. Knowledge of the dangers associated with lead extends almost as far back as human discovery of the useful properties of this common element. Although both lead poisoning and its connection to lead exposure were known in late Antiquity, this disease was almost completely forgotten in the literature preserved from the Middle Ages. Moreover, when scientists first began investigating and writing about the toxicity of substances, lead was among the first substances analyzed. To put this in perspective, the following quote was written in 1817, almost 200 years ago:

If we were to judge of the interest excited by any medical subject by the number of writings to which it has given birth, we could not but regard the poisoning by lead as the most important to be known of all those that have been treated of, up to the present time.124

Yet, while it is undisputed that people knew that lead was poisonous, that still leaves unresolved the context of what exactly was known or understood about lead as a poison. Was it just poisonous to people who worked in the lead mines, smelters and factories? Or is it poisonous to everyone everywhere? At what levels of exposure is lead poisonous? Is the damage temporary or permanent?

123 Sven Hernberg, Lead Poisoning in a Historical Perspective, AM. J. INDUS. MED. 38:244-54, 244 (2000). This forgetfulness is astonishing, because lead was widely used for industrial, domestic and medicinal purposes. For example, lead acetate (“lead sugar”) was used as a sweetener of wine and ciders, and it caused severe epidemics of poisoning. In some German countries the problem was so severe that death penalty was prescribed, first in 1498 and later in 1577, for those caught mixing lead sugar into wine. Id.

124 This quote is credited to Mathieu J. B. Orfila, considered to be the Father of Toxology, in 1817.
Some people appear to honestly believe that those in industry knew everything there was to know about the hazards of lead long before the medical and research scientists and that they actively suppressed that knowledge. Personally, these authors find it very hard to believe that a few in industry can suppress all the doctors and scientists worldwide who were dealing with people suffering from acute or chronic lead poisoning; especially when doctors have been dealing with lead-poisoned patients for hundreds, if not thousands, of years. One thing cannot be disputed, man’s knowledge regarding the hazards posed by lead has evolved over the millennia. This section tries to provide a brief overview of that evolution.

1. In Antiquity

The toxic effects of chronic lead poisoning have been known for centuries. Long ago people regarded lead as the father of all metals. But, the deity they associated with the substance was Saturn, the ghoulish titan who devoured his own young. The word “sartorine,” in its most specific meaning, applies to an individual whose temperament has become uniformly gloomy, cynical and taciturn as the result of lead intoxication.\textsuperscript{125}

Among the earliest records, whether on papyrus, copper, parchment or cuneiform writing on clay, there are notes that lead miners and individuals who worked with lead developed

\begin{quote}
\begin{quotation}
Hence gout and stone afflict the human race;
Hence lazy jaundice with her saffron face;
Palsy, with shaking head and tottering knees;
And bloated dropsy, the staunch sot’s disease;
Consumption, pale, with keen but hollow eye,
And sharpened feature, shew’d that death was nigh.
The feeble offspring curse their crazy sires,
And, tainted from his birth, the youth expires.
\end{quotation}
\end{quote}

Translated by Humelbergius Secundus (1829). \textit{Id.}

\textsuperscript{125} Jack Lewis, \textit{Lead Poisoning: A Historical Perspective}, EPA Journal, EPA (May 1985), \textit{available at} http://www.epa.gov/history/topics/perspect/lead.htm (last visited Aug. 11, 2006). An anonymous Roman hermit described lead poisoning as follows:
ailments that resulted in their early demise. This was first well documented by the Egyptians who used slaves in their mines and later by the Pre-Greeks, Greeks and Romans.126

2. During the Roman Empire

When discussing the history of lead poisoning as it relates to the Roman Empire, many commentators write as though it is common knowledge that Romans knew all about lead poisoning, yet ignored its consequences because of their fondness of lead products.127 What the Romans actually knew about the health effects of daily exposure to lead is mere speculation and the subject of a lot of debate. Likewise, no one really knows whether lead poisoning was as large a contributor to the decline of the Roman Empire as some people claim.

Generally, historians explain the collapse of the Roman Empire by looking at financial reasons (emperors depleting the imperial coffers), poor leadership or military failure.128 However, some believe that lead poisoning played a prominent role in the decline of the Roman Empire. This idea was first raised in 1965 by S.C. Gilfillan,129 but it became more widespread when Jerome Nriagu, a geochemist, reignited the topic by taking a serious look at the role lead poisoning may have played in the decline of the Romans.130 Dr. Nriagu hypothesized that two Roman food staples (defrutum131 and sapa132) may have contained enough lead acetate to be of danger to those who consumed it regularly.133 In his 1983 article in the New England Journal of Medicine, Jerome O. Nriagu; Saturnine Gout Among Roman Aristocrats: Did Lead Poisoning Contribute to the Fall of the Empire?; NEW ENGL. J. MED. 308(11):660-3, (Mar 17. 1983);


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127 See, e.g., Lewis, supra note 125 (“The Romans were aware that lead could cause serious health problems, even madness and death. However, they were so fond of its diverse uses that they minimized the hazards it posed”).
129 S. Colum Gilfillan, Lead Poisoning and the Fall of Rome, 1 J. OCCUPATIONAL MED., 53-60 (1965).
130 Jerome O. Nriagu; Saturnine Gout Among Roman Aristocrats: Did Lead Poisoning Contribute to the Fall of the Empire?; NEW ENGL. J. MED. 308(11):660-3, (Mar 17. 1983);
131 A mild concentrate of grape juices that was boiled in lead or bronze kettles used in cooking.
132 A stronger concentrate than defrutum used as a souring agent and preservative.
Medicine and in a book later that year, he argued that lead poisoning contributed to the decline of the Roman Empire. According to Dr. Nriagu, the apathy and gluttony that has been associated with the decline of the Roman Empire may have been caused by the lead in food, water and wine. Other commentators disagree, stating that while ancient authorities were aware of lead poisoning, it was not endemic in the Roman Empire nor caused its fall.

Musanus, a Roman writing in the first century A.D., observed that masters were weaker, less healthy and less able to endure labor than the servant class. Those who grew up in the country were stronger than those who grew up in the city. Those who ate plain food were likely to live longer. Using hindsight, some commentators associate descriptions of life during Roman time as examples of people with cases of “gout,” “dropsie” and “colic.” Perhaps these descriptions are as close as anyone got to discovering chronic lead poisoning in the Roman Empire and leaving a record of the hypothesis.

that Roman winemakers insisted on using lead in winemaking. When boiling crushed grapes, Roman vintners used lead pots or lead-lined copper kettles. “For, in the boiling,” wrote Roman winemaker Columella, “brazen vessels throw off copper rust which has a disagreeable flavor.” Lead’s sweet overtones, by contrast, were thought to add complementary flavors to wine and to food as well. As a result, lead enhanced one-fifth of the 450 recipes in the Roman Apician Cookbook, a collection of first through fifth century recipes attributed to gastrophiles associated with Apicius, the famous Roman gourmet. From the Middle Ages on, people put lead acetate or “sugar of lead” into wine and other foods to make them sweeter.


Dan Montgomery, Lead, Fluoride, the Roman Empire and the Decline of Academic Achievement in the United States, available at http://www.sonic.net/kryptox/environ/lead/romans.htm (last visited Sept. 23, 2006); see, e.g., Frederic William Farrar, Seekers after God, The Project Gutenberg eBook. The following was written by Marcellinus, a young and wealthy noble, who starved himself, and then had himself suffocated in a warm bath, merely because he was attacked with a perfectly curable illness:

Numb my hands with palsy,  
Rack my feet with gout,  
Hunch my back and shoulder,  
Let my teeth fall out;  
Still, if Life be granted,  
I prefer the loss;  
Save my life, and give me  
Anguish on the cross.
3. From Hippocrates to Benjamin Franklin

In 370 B.C., the Greek physician Hippocrates described the following symptoms in a man who was a metal worker: appetite loss, colic, pallor, weight loss, fatigue, irritability and nervous spasms. These are the same symptoms that are observed and categorized for lead poisoning today. In the first century A.D., Dioscorides, another Greek physician, noticed that exposure to lead could cause paralysis and delirium in addition to intestinal problems and swelling. References to paralysis in lead-exposed miners increased in Europe in the 1600s, as did reports of colic in wine drinkers. As reports such as these became more widespread, some began taking advantage of lead’s toxicity. Members of the French nobility, for example, called lead “poudre de la succession” - succession powder - a reference to its poisonous potency.

As lead and its compounds gained more and more use in pottery, piping, shipbuilding, window making, the arms industry, pigments and later book printing, chronic lead poisoning became a major health problem in Europe and later in America during the 15th, 16th, 17th, and 18th centuries. However, it was not until the 16th century that lead poisoning reappeared in the medical literature in Paracelsus’ description of what he called “the miner’s disease.” In the beginning of the 18th century it was written that potters who worked with lead became “paralytic, splenetic, lethargic, cachectic, and toothless, so that one rarely sees a potter whose face is not cadaverous and has the color of lead.”

While the problems associated with lead mining and processing date back to antiquity, epidemics of chronic lead poisoning were first recognized in the eighteenth century, usually associated with the use of lead as an adulterant in alcoholic beverages. For example, Sir George

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137 See Lessler, supra note 126, at 79.
138 See Sohn, supra note 133.
139 Hernberg, supra note 123, at 245.
140 Id. (citing B. Ramazzini, Diseases of Workers, 53 (W.C. Wright trans., Hafner Publishing Company 1964 (1713))).
Baker\textsuperscript{141} made an important discovery in 1767 when he figured out that the so-called Devonshire colic, which had plagued the county for a century, was due to contamination of cider with lead. Dr. Baker showed that lead had dissolved from the weights used to crush the apples; hence he could prove that the etiology of the colic was lead poisoning, not stardust or eastern wind, as formerly believed. Severe poisoning was characterized by palsy, encephalopathy, pallor and abdominal cramps, and the case fatality rate was high.\textsuperscript{142} Apple cider was not the only source of lead in the beverages of the day. Similar problems were associated with fortified wines (port and sherry) that England imported from Spain and Portugal after it signed the Treaty of Methuen in 1703. As a result of this trade, there was widespread lead poisoning and a great gout epidemic among the upper classes in England, who imbibed liberally of these beverages.\textsuperscript{143} This problem was first addressed in the United States in 1723 when the Massachusetts Bay Colony passed an act prohibiting the distillation of rum or other strong liquor with leaden heads (covers) or worms (pipes). This was the result of extensive sickness (lead poisoning) caused by the imbibing of distilled rum.\textsuperscript{144}

\textsuperscript{141}Sir George Baker was born on February 8, 1723. He was educated at Eton and at King’s College, Cambridge, where he graduated M.A. in 1749, became a Fellow, and was made an M.D. in 1756. He initially set up practice in Stamford in Lincolnshire; but later moved to London around 1761. He became a Fellow of the College of Physicians, was Censor four times, Harveian orator, elect and, finally, President from 1785-1795. In succession he was appointed physician to the Queen’s household, physician-in-ordinary to the Queen and physician-in-ordinary to King George III. R.M.S. Mcconaghey, \textit{Sir George Baker and the Devonshire Colic,} \textit{MED. HIST.} 11(4): 345–360. (Oct. 1967).

\textsuperscript{142}Hernberg, \textit{supra} note 123, at 245.

\textsuperscript{143}Lessler, \textit{supra} note 126, at 79. Recent atomic absorption analyses of English port bottled during the period 1770 to 1830 showed lead contents of up to 1900 mg Pb/L. \textit{Id.} (citing Nriagu, \textit{supra} note 134).

\textsuperscript{144}Chapter II of the Massachusetts Bay Colony Act of 1723, passed under the charter granted by their Majesties King William and Queen Mary to the inhabitants of the Province of Massachusetts-Bay in New England, reads as follows:

\begin{quote}
An Act for preventing Abuses in distilling of Rum and other strong Liquor with Leaden Heads or Pipes.

Whereas the strong Liquors and Spirits that are distilled thro' leaden Heads or Pipes, are judged on good Grounds to be unwholesome and hurtful; notwithstanding which some Persons to save Charge may be led into making or using such Heads, Worms, or Pipes:

For Remedy and Prevention Whereof: Be it enacted by the Lieutenant Governor, Council and Representatives in General Court assembled, and by the same, That no Person whatsoever, shall make Use of any such Leaden Heads or Worms for the future; and that whosoever shall presume to distill or draw off any
Responding to a request that he describe some of his own encounters with persons using lead, on July 31, 1786, Benjamin Franklin wrote: “The first Thing I remember of this kind, was a general Discourse in Boston when I was a Boy, of a Complaint from North Carolina against New England Rum, that it poison’d their People, giving them the Dry Bellyach, with a Loss of the Use of their Limbs.” Franklin goes on to describe a warning he received not to heat lead type while he worked in a London printing house in 1724. Apparently, two of his co-workers who heated their type had lost the use of their hands after years of holding leaded type close to the fire to dry. Franklin described their misfortunes, along with his own pain that “had sometimes felt as it were in the Bones of my Hand when working over the Types made very hot.” The warning “induc’d me to omit the Practice.” He inquired about the problem, but was told that it was caused by workmen who did not thoroughly wash their hands before eating their meals “so that some of the metalline Particles were taken off by their Bread and eaten with it.” Franklin also discussed health problems associated with inhaling smelting fumes and drinking rainwater that ran off roofs coated with leaded paint that made lead poisoning a serious problem.

4. Modern Day Knowledge

The modern day understanding of lead poisoning began being formed in the early 1800s. It was not until then that B. Ramizzini (an Italian physician) linked the diseases shared by Spirits or strong Liquors through such Leaden Heads or Worms, upon legal Conviction thereof before any of his Majestie's Courts of Record, shall forfeit and pay a sum of One Hundred Pounds.

And Be It Further Enacted By the Authority aforesaid, That no Brazier, Pewterer, or other Artificer whatsoever, shall presume to make any Worm or Head for distilling of coarse and base pewter, or such as hath any Mixture of Lead in it; under the Penalty of One Hundred Pounds.

potters, guilders, glass makers and metal workers with lead poisoning. The anaemia associated with chronic lead poisoning was probably first recognized and described by Laennec in 1831. The first “modern” clinical description of lead poisoning was Tanquerel des Planches’ famous “Traité des maladies de plomb ou saturnines,” which appeared in 1839 and was based on over 1,200 cases. More than 800 of the cases were in painters or workers involved in the manufacture of white or red lead pigments. Probably no one before him had seen so many cases of plumbism and probably no one later, either. Tanquerel des Planches gave a detailed description of the abdominal, neurological and arthritic aspects of lead poisoning, but he erroneously believed that they were different entities.

Later, particularly during the industrialization of Europe, reproductive failures and congenital lead poisoning were described in medical literature. During the first half of the twentieth century, medical literature recognized that the following ailments among the working class women and the wives of men who worked in the lead trades included: sterility, abortion, stillbirth and premature delivery. As a result, a British Royal Commission in 1910 recommended that women be excluded from the lead trades, a recommendation that was enforced in some countries by law.

Today, the issue of most concern is the effect of lead on young children.

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148 See Lessler, supra note 126, at 79.
151 Measuring Lead Exposure In Infants, Children, and Other Sensitive Populations, COMM’N ON LIFE SCIENCES, NAT’L ACADEMY OF SCI. 24 (1993) [hereinafter CLS Study].
152 Hernberg, supra note 123, at 245.
153 CLS Study, supra note 151, at 24 (citing C. Paul, Study Of The Effect Of Slow Lead Intoxication On The Product Of Conception [In French], ARCH. GEN. MED. 15:513 533 (1860)).
154 Id.
i. Childhood Lead Poisoning

Up until the early 1900s, chronic lead poisoning was viewed largely as a disease of adults due to occupational exposure. That changed when an Australian ophthalmologist documented the first known case of childhood lead poisoning.

It has been said that the modern understanding of lead poisoning in children has gone through four stages.\(^{155}\) The first stage occurred when the existence of childhood lead poisoning was first brought to attention in New South Wales (Australia) at the turn of the century.\(^{156}\) It all began in the 1890s when a physician named Turner identified children with symptoms ranging from headache to vomiting and foot- and wrist-drop which he attributed to a leaded water tank.\(^{157}\) Initially, the very existence of childhood lead poisoning was disputed. Physicians in southern Australia were skeptical, because the condition seemed to stop abruptly at the Queensland border. They regarded the condition as a “delusion held by their despised colleagues in the primitive northern state.”\(^{158}\)

Twelve years later, in 1904, another Australian named Gibson determined that the lead poisoning was the result of the children’s ingesting of “white paint” chips from old porches.\(^{159}\) In the warm humid climate, exterior paints weather quickly, and children would soon have their hands coated with powdery leaded material, which was inevitably


\(^{159}\) CLS Study, supra note 151, at 24; see also John L. Gibson, *A Plea for Painted Railing and Painted Walls as the Source of Lead Poisoning Among Queensland Children*, 23 AUSTRALIAN MED. GAZETTE 149-53 (1904). Dr. Gibson had the powdered paint from his own house analyzed and lead carbonate was found. He determined that the paint on wooden Queensland houses dried and powdered in the hot sun. He also ascertained that most of the affected children were nail-biters or thumb-suckers who carried it on sweaty hands to the mouth. Ronald Wood, *Gibson, John Lockhart (1860-1944)*, *AUSTRALIAN DICTIONARY OF BIOGRAPHY* 652-53(Vol. 8, Melbourne University Press 1981). Gibson wrote “I … advance a very strong plea for painted walls and railings as the source of the lead, and for the biting of finger nails and sucking of fingers, as in a majority of cases, the means of conveyance of lead to the patient.” CLS Study, supra note 151, at 24.
carried to their mouths and digestive tracts.\textsuperscript{160} It was not until 1914 that medical literature described the first American case of lead-paint poisoning (in a 5-year-old boy).\textsuperscript{161} Nonetheless, physicians in the United States were initially skeptical of the Australian findings, but by the 1920s were forced to confront the issue because reports of lead poisoning had become more prevalent.\textsuperscript{162}

The second stage began after the existence of childhood lead poisoning was accepted. Most pediatricians believed that children who did not die from lead poisoning suffered no lasting ill effects.\textsuperscript{163} In 1933, a report was published of lead poisoning caused by the burning of wooden battery casings as fuel to cook food in Baltimore homes during the Great Depression.\textsuperscript{164}

The third stage began in the 1940s when observations demonstrated the persistence of serve residua in children who had recovered from acute lead poisoning. While this fact was accepted, most thought that the problems afflicted only those patients who had the most severe symptoms.\textsuperscript{165} More case studies began to appear in the 1950s, as the condition became more widely known in larger cities in the U.S., including New York, Chicago, Philadelphia, Boston, Cincinnati, St. Louis and Cleveland. By 1970, the epidemiology of childhood lead poisoning was well established.\textsuperscript{166}

\textsuperscript{160} CLS Study, supra note 151, at 26.
\textsuperscript{161} H.M. Thomas & K.D. Blackfan. \textit{Recurrent Meningitis Due To Lead In A Child Of Five Years}, 8 Am. J. Dis. Child. 377 380 (1914). They also offered the opinion that children must in some way be peculiarly susceptible to lead. \textit{Id.}
\textsuperscript{162} Tesman, supra note 157, at 2.
\textsuperscript{163} Needleman, \textit{supra} note 155, at 1371 (citing C.F. McKhann & E.C. Vogt, \textit{Lead Poisoning in Children: With Notes on Therapy}, 32 Am. J. Diseases Children 386-92 (1926) (publishing a series of 17 cases of lead poisoning in children)).
\textsuperscript{164} Huntington Williams et al., \textit{Lead Poisoning From The Burning Of Battery Casings}, 100 J. Am. Med. Assoc. 1485 1489 (1933). In the early 1930s, there was an outbreak of lead poisoning among children in an economically depressed area of Baltimore. An intern, Miriam E. Brailey, visited a home there to seek the source of the lead. She had no success until a bystander, Melrose Easter, whose "breath was strong with whisky," said that he thought burning of battery casings was the cause, for "the smell was bad, even made the food taste bad." \textit{Id.}
\textsuperscript{165} Needleman, \textit{supra} note 155, at 1371 (citing R.K. Byers & E.E. Lord, \textit{Late Effects of Lead Poisoning on Mental Development}, 66 J. Am. Med. Assoc. 472-83 (1943)).
\textsuperscript{166} CLS Study, \textit{supra} note 151, at 26.
Although cases of childhood lead poisoning in the U.S. were reported in the first half of the twentieth century, little effort was made to understand the extent of the problem until the 1950s, when caseworkers in a few large cities attempted to identify poisoned children as part of their family nutrition work. Limited results were obtained until 1966, when Chicago was the site of the first mass screening program where many children were identified with elevated BLLs. In 1967, Jane Lin-Fu writing for the Department of Health, Education, and Welfare (now the Department of Health and Human Services), published a paper claiming for the first time that lead poisoning was a serious threat to children’s health causing neurological problems, mental retardation, and even death. Following its publication, efforts to educate the general population regarding the threat of lead poisoning increased dramatically. In 1971 the Surgeon General issued a statement that emphasized the need to shift the focus from identifying poisoned children to prevention. Likewise, in 1971 federally assisted screening of children for lead poisoning began. These screening showed that 20% to 45% of children tested had blood lead levels over 40 μg/dL. However, since the mid-1960s the incidence of lead poisoning has decreased significantly due to mass education and the reduction of lead in both gasoline and food processing (lead solder used in canning).

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168 *Id.*, see also CLS Study, supra note 151, at 26-27.


171 CLS Study, * supra* note 151, at 27.


173 *Id.*
The final stage began in the late 1970s and 1980s when researchers began trying to determine the associations between specific lead blood levels (at the low end of the spectrum) and adverse health effects. While none of the symptoms or manifestations classically associated with lead poisoning are at issue, researchers and regulators still refer to these subclinical manifestations as “childhood lead poisoning.”

C. STATE OF THE SCIENCE

Some commentators would have us believe that evidence of the adverse health effects of lead (all of them) have been known for years. However, that is not true with respect to the adverse health effects caused by low levels of lead in the blood. In 1976, when the D.C. District Court was considering the legality of the regulation phasing down the lead content in gasoline, it made the following observation about the alleged adverse health effects associated with the use of leaded gasoline:

The record in this case is massive – over 10,000 pages. Not surprisingly, evidence may be isolated that supports virtually any inference one might care to draw. Thus we might well have sustained a determination by the Administrator [of the EPA] not to regulate lead additives on health grounds. That does not mean we cannot sustain his determine to so regulate.

1. Early Studies (pre-1975)

Until very recently, lead poisoning was perceived as a potentially fatal illness associated with acutely high exposure to lead that manifested itself as encephalopathy, acute abdominal colic and acute kidney damage. Before the 1950s, the clinical diagnosis, cause and treatment of classical lead poisoning was known to both industry and the medical community. Lead poisoning, together with anemia, were seen in the epidemics of Europe. Initially, the medical community was primarily interested only in the severe cases on lead poisoning. It was not until

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174 Needleman, supra note 155, at 1371 (citing the CLS Study, supra note 151).

175 Ethyl, 541 F.2d at 37.
the late 1960s that methods capable of measuring lead in biologic media (such as blood) began being developed. Their development in the 1970s and 1980s is what allowed scientists to begin researching subclinical manifestations of lead poisoning. In addition to developing methods to measure lead, scientists had to solve problems associated with sample contamination and interlaboratory comparisons.

The problem of sample contamination was first recognized by Dr. Patterson in the 1950s. To resolve this problem, Dr. Patterson created one of the first ultra-clean laboratories. He entered his laboratory through an airlock in which the air was filtered, the researchers gowned and masked, and the reagents and water supply purified of any trace of lead. It was only by taking these precautions that was he able to establish the true concentrations of lead in his samples. Unfortunately, many early researchers did not take the same precautions as Dr. Patterson.

Likewise, before 1970 venous samples of 5–20 μL of blood (usually obtained by external jugular or femoral vein puncture) were required by the older colorimetric procedures to determine a child’s BLL. At that point, a single technician could process only 8 to 10 samples per day. Moreover, in the early 1970s, four to six-fold variations in the results of different laboratories measuring lead from the same blood samples was not uncommon. These highly unreliable results led to confusion as to what blood lead levels were “safe” and “unsafe.” “Fruitless debates in the literature and at conferences led to further confusion because 50 [μg/dL] for one researcher could be the same as 90 to another, and so forth.” But, from this confusion

176 See supra section II.B.3.ii.
177 Needleman, supra note 50, at 22.
178 Tesman, supra note 157, at 3.
179 Hernberg, supra note 123, at 247.
180 Id.
came the realization that analytical methods needed to be controlled and good laboratory practices were a necessity. The situation gradually improved. Today, you rarely see articles accepted for publication in your better journals without documented method controls.\textsuperscript{181}

Since the early 1970s, there have been significant improvements in the technology used to measure lead in blood and in other sources. For example, the introduction of improved instrumentation for the atomic absorption spectrometry and anodic stripping voltammetry made it possible to obtain accurate and precise measures of lead in microsamples of blood. Without these technologies the research of the past 35 years would not have been possible.\textsuperscript{182}

2. Toxic Effects Versus Subclinical Manifestations

Early on, some prominent researchers had a strong belief that their laboratories could analyze blood lead concentrations accurately and better than others. This unfounded belief made some experts categorically state that one could not develop poisoning as long as the blood lead concentration remained below 80 μg/dL.\textsuperscript{183} Dr. Robert Kehoe, the automotive and oil companies’ primary expert on lead in the twentieth century, was a strong advocate of this black-or-white viewpoint.\textsuperscript{184} However, Dr. Kehoe was not alone in believing in this categorical limit.

\textsuperscript{181} \textit{Id.}

\textsuperscript{182} Tesman, \textit{supra} note 157, at 3. To obtain samples of blood lead, two methods are used: the capillary sample and the venous puncture. The capillary sample is obtained from a heel stick, typically used with children under age 1, and a finger stick for older children. The advantage of capillary samples is that this method is a quick, expedient, and simple procedure for trained medical personnel. Capillary samples are easily contaminated, however, because lead can lodge in the grooves of the fingerprint and elevate the amount of lead detected in the sample. To minimize these drawbacks, the CDC has prescribed guidelines for obtaining capillary specimens. And in response to the problems with capillary sampling, the CDC recommends that the “venous blood is the preferred specimen for analysis and should be used for lead measurement whenever practical” \textit{Id.} at 9 (citing Centers for Disease Control, \textit{Preventing Lead Poisoning In Young Children: A Statement By The Centers For Disease Control}. U.S. Department of Health and Human Services 45 (1991)) Current procedure commonly involves testing initially using a finger stick, and then performing confirmatory venous puncture if an elevated lead level is found. \textit{Id.}

\textsuperscript{183} Hernberg, \textit{supra} note 123, at 247.

\textsuperscript{184} \textit{Id.} at 247-48. Dr. Kehoe used to state vigorously that he had never seen a case of lead poisoning with a blood lead level below 80 mg/100 ml before chelation, with the “modest” addition that he had personally seen more lead poisoning than anyone else in the world.
In 1968, a group of European experts chaired by Ronald Lane recommended that Europe should use the 80 μg/dL criteria as a border value for a “safe” exposure to lead.\(^\text{185}\)

With hindsight, some portray the 80 μg/dL standard championed by Dr. Kehoe and industry as foolhardy and as recklessly endangering both workers and the public.\(^\text{186}\) However, when Dr. Kehoe and Dr. Lane recommended their 80 μg/dL standard, they (like most of the world) were fixated on the old clinical concept of lead poisoning, according to which subclinical states did not count and whose very existence was even questioned by many experts.\(^\text{187}\) This “effects parameter” was recommended for the surveillance of lead workers with corrective action being taken before this level was reached. In principle, an action level based on a correctly determined blood lead level of 80 μg/dL probably would have prevented clinical lead poisoning, but it would not prevent the problems and hazards associated with subclinical manifestations of poisoning.\(^\text{188}\)

Research conducted over the last thirty to forty years has greatly enhanced our knowledge on subclinical and nonspecific “manifestations” of lead toxicity and caused a fundamental revision of our concept of lead poisoning. Currently, the blood lead “level of concern” is 10 μg/dL.\(^\text{189}\) Although there have been some recent studies suggesting that adverse health effects exist in children at levels less than 10 μg/dL, the CDC chooses not to lower its level of concern because:

\(^\text{185}\) Id. (citing R.E. Lane et al., Diagnosis Of Inorganic Lead, 4 Brit. Med. J. 501-04 (1968)).

\(^\text{186}\) See, e.g., Kovarik, supra note 75.

\(^\text{187}\) Hernberg, supra note 123, at 248. For example, Lloyd Davies wrote on page 170 in his 1957 textbook The Practice of Industrial Medicine: “A fall in the haemoglobin and red cell count if accompanied by punctate basophilia, is probably the earliest sign of exposure to lead. These changes do not necessarily indicate poisoning.” Id.

\(^\text{188}\) Id. (but noting the problems with the standard during that time period given the analytical inaccuracy of blood lead determinations and the scarcity of laboratories capable of doing any lead analyses at all).

\(^\text{189}\) See infra section IV.F.
there are no known clinical interventions known to reduce BLLs below 10 μg/dL or to reduce the risk of adverse developmental effects;

children cannot be accurately classified as having a BLL above or below a value less than 10 μg/dL because of inherent laboratory inaccuracies; and

any decision to establish a new lower level of concern would be arbitrary and capricious and would provide uncertain benefits because the studies suggest that there is no level below which adverse health effects are not experienced.²⁹⁰

3. Confounding the Causal Association

A lot of current research into the adverse health effects associated with lead is focused on the relationship between low level exposures of lead (less than 10 μg/dL) and its impact on a child’s IQ.²⁹¹ Other researchers are trying to link exposures to lead with future behavior problems, including but not limited to crime.²⁹² These researchers have a difficult task. They are trying to causally connect low level exposures (as compared to high level exposures) of lead to adverse neurological effects (i.e., lower IQs and behavior problems) in very young children and that the adverse effects persist over the person’s lifetime. That’s a very tall order. First, researchers have to have a baseline IQ for the children. Now, we are not talking about children in high school or even middle school. No, we are talking about the IQ of preschoolers – perhaps children as young as 2 years old.²⁹³ Next, they have to show that lead – and only lead – is what


²⁹¹ “Experimental studies, both in rodents and non-human primates, have since documented lead related deficits at low-level exposures and established these to be direct effects of lead.” See, e.g., Bruce Lanphear, Witness testimony to the Cong. Subcomm. on Env’t & Hazardous Materials (Jul. 22, 2004) (emphasis added) (citations omitted) [hereinafter Lanphear testimony]. To support the causal association between lead and IQ, Dr. Lanphear cited his own studies as proof of a “lead-associated reading deficit” in children with BLLs below 5 μg/dL (citing to Bruce P. Lanphear et al., Cognitive Deficits Associated With Blood Lead Levels < 10 Mg/Dl In U.S. Children And Adolescents, 115 PUB. HEALTH REP. 521-29 (2000) and as proving that an increase in a lifetime mean BLL from less than 1 to 10 μg/dL was “associated” with a 7.4 point IQ deficit (citing R.I Canfield et al., Intellectual Impairment In Children With Blood Lead Concentrations Below 10 Micrograms Per Deciliter, 348 N. ENG. J. MED. 1517-26 (2003)).


²⁹³ Testing of infant is especially difficult because they are in a period of rapid developmental change. Also, the way an infant’s cognitive function can be probed is restricted. The lack of continuity between their response modalities and ones that
causes an effect on a person’s IQ or behavior. This is a problem because these researchers are trying to show that a low level exposure to only one substance “directly effects” the brain’s intelligence and a person’s behavior.

It is a problem because (1) we truly do not have an understanding of how the brain works or why some people are intelligent and others are not, and (2) because many variables acting independently or in conjunction with other variables can effect the way the brain functions or a person behaves. This is the-age old nature or nurture question. There are many variables that can effect an individual’s IQ. In a research study, every one of these variables, except the focus of the research (i.e., lead), are known as confounders. In order to make the claim that lead directly effects IQ, in their study the researchers must identify, account for and control all the confounders. Anything less merely means that the researcher has only documented an “association” or an “observation” between what was being looked at (i.e., IQ or behavior) and the object of the study (i.e., exposure to lead). This does not belittle the researchers’ work because “associations” are important in learning. But claims of “associations” are a lot different from claims of “causation.”

When reading these studies or listening to researchers discuss the studies, great care must be taken to determine whether they are claiming that their research shows that lead is causally linked to behavioral problems or IQ deficits or whether they have documented these problems in

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195 When one variable is studied to try to explain another, the relationship between them may be biased by a third variable. The bias, known as ‘confounding,’ is common and must be minimized in research. When present, confounders result in a biased estimate of the effect of exposure on disease. The bias can be negative—resulting in underestimation of the exposure effect—or positive, and can even reverse the apparent direction of effect. It is a concern no matter what the design of the study or what statistic is used to measure the effect of exposure. Roseanne McNamee, Confounding and Confounders, 60 OCCUPATIONAL & ENVN’T MED. 227-234 (2003).
their study group of lead-exposed children. To date, it appears that researchers have been observing “associations” between persons who have been exposed to lead and IQ deficits or behavior problems. However, it is very possible that these problems are not at all linked to lead but to other confounders that are more important in a child’s development than lead exposure. That is the problem with confounders. They are difficult to identify. In large groups, important confounders may not be common to all children (i.e., what effects one child's IQ or behavior may not effect another child). For purposes of research, perhaps the most important problem with confounders is that they are extremely difficult to control.

D. WHO IS AT RISK?

Lead-containing products can be found everywhere in our environment. Lead is commonly found in soil, especially near roadways, older houses, old orchards, mining areas,

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196 The possibility that the adverse health effects associated with increased lead exposure in epidemiologic studies are, in fact, due to risk factors with which increased lead exposure is associated remains the most important impediment to drawing causal inferences. Criteria Document, supra note 44, at 6-263. Some of these confounders are truly independent predictors and can be adjusted for using multiple regression analyses. Under some circumstances, however, lead and the confounder may be so highly related that one cannot be confident that their associations with the outcome have been disentangled by the statistical methods applied. Moreover, the true causal relationships among lead, the confounders, and the outcome might not be sufficiently well understood that the outcome variance shared by lead and the confounders can be characterized appropriately in the analyses. Id. at 6-265.

197 See Lanphear testimony, supra note 191. According to Dr. Lanphear:

Schwartz reported that lead-associated cognitive deficits and hearing loss occur at blood lead levels below 10 mg/dL. In a meta-analysis, the observed decrement was greater for studies with children having blood lead levels below 15 mg/dL compared to those with children having higher blood lead levels. In an analysis of NHANES III, the lead-associated reading deficit increased, from -1.0 point per 1 mg/dL increase in blood lead for the entire sample to -1.7 point per 1 mg/dL increase for the subgroup with blood lead levels below 5 mg/dL. In a prospective study, an increase in lifetime mean blood lead level from < 1 to 10 mg/dL was associated with a 7.4 point IQ deficit. Moreover, consistent with the earlier studies, the lead-associated cognitive deficits associated with each 1 mg/dL increase in blood lead level were greater at blood lead concentrations below 10 mg/dL. Although there are several plausible mechanisms to explain these findings, the specific mechanism is unclear.

Id. (citation omitted). Note that at the end of his testimony discussing IQ deficits, Dr. Lanphear clarifies his “associations” and “observation” regarding humans by stating that “there are several plausible mechanisms to explain these findings.” Id. Each of those several mechanisms are confounders.

198 One noted researcher has admitted that complete confounder control is impossible in real world studies. As a result, in most studies that he reviewed, control of confounders has reduced the magnitude of the lead-IQ effect. But, he notes that it has not obliterated the lead-IQ effect. Herbert L. Needleman & C.A. Gatsonis, Low-level lead exposure and the IQ of children. A meta-analysis of modern studies, 263 J. Am. Med. Assoc. 673-78 (1990).
industrial sites, near power plants, incinerators, landfills and hazardous waste sites.\textsuperscript{199} You and your family can be exposed to lead and lead chemicals from breathing air, drinking water, eating foods, playing with toys, wearing jewelry and swallowing or touching dust or dirt that contains lead. In the past, emissions from vehicles burning leaded gasoline and poorly maintained products (and houses) that were covered with lead-based paint were two of the main sources of exposure. However, now that leaded gasoline has been phased out and lead-based paints were banned from most uses, lead in soils and dusts and poorly maintained lead paint have become the principal sources of exposure in the U.S. The following figure represents one researcher’s view of how lead gets into a child.\textsuperscript{200}

Both children and adults are susceptible to health effects from lead exposure, although the typical exposure pathways and effects are somewhat different. Most governmental agencies believe that children who reside in pre-1978 housing facilities (and especially those in inner cities or those built before 1950) and adults who are occupationally exposed are at greatest risk.

\textsuperscript{199} Toxicological Profile, supra note 118, at 4.

1. **Children**

   i. **Levels of Concern**

   Preschool-age children and fetuses are more vulnerable to the effects of lead than other segments of the population because:

   - their nervous systems have increased susceptibility to the toxic effects of lead;
   - they are more likely to play in dirt and to place their hands in their mouths, thereby increasing the ingestion;
   - the efficiency of lead absorption from the gastrointestinal tract is greater in children than in adults;
   - nutritional deficiencies of iron or calcium (prevalent to children) may facilitate lead absorption and exacerbate the toxic effects of lead.\(^{201}\)

   The good news is that children’s exposure to lead (as indicated by their BLLs) has dramatically declined since the 1970s. In fact, average BLLs for children have dropped at least 80 percent (and probably more than 90 percent) since the 1970s.\(^{202}\) That said, currently Chicago, New York City, Philadelphia, Cleveland and Detroit are the top five cities with the highest number of children with elevated BLLs.\(^{203}\)

   According to the latest information (collected between 1999 and 2002), the CDC estimates that 1.6 percent of pre-school children have elevated BLLs (above 10 μg/dL). Based on that percentage, the CDC believes that 310,000 pre-school children remain at risk for harmful

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\(^{201}\) Fact Sheet, Lead: Questions and Answers, Cntr. For Disease Control and Prevention, Dept. of Health & Human Serv.,

\(^{202}\) Agency for Toxic Substances and Disease Registry (“ASTDR”), Case Studies in Environmental Medicine – Lead Toxicity: Exposure Pathway, Dept. of Health and Human Serv. 7 (Course: SS3059) (Oct. 2000), available at http://www.atsdr.cdc.gov/HEC/CSEM/lead/exposure_pathways.html#paint (last visited Aug. 3, 2006) [hereinafter Lead Toxicity] (citing Screening Young Children For Lead Poisoning: Guidance For State And Local Public Health Officials, Cntr. For Disease Control and Prevention, Dept. of Health & Human Serv (1997)). Given the continuing drastic reduction in BLL for children, and that the CDC found using 1997 numbers that the BLLs had decreased 80% since the 1900s, today it is highly likely that they have decreased by over 90%.

\(^{203}\) Report: Meeting of the CDC Advisory Committee on Childhood Lead Poisoning Prevention, Cntr. For Disease Control and Prevention, Dept. of Health & Human Serv 10 (Mar. 21-22, 2006).
lead levels. Surprisingly, youths ages 6 to 19 years had the lowest prevalence of elevated BLLs (0.2 %), but the CDC qualified this percentage by stating that it was not statistically reliable.

While 310,000 preschool-aged children may seem like a large number, it is substantially lower than earlier estimates. Based on data collected between 1991 to 1994, the CDC reported that 4.4 percent (or 930,000) of pre-school children had elevated BLLs. Finally, back in 1988, the CDC estimated that a whopping 17 percent (or 2,380,600) pre-school children were exposed to lead at levels above 15 μg/dL, 5.2 percent (or 715,500) were exposed to lead at levels above 20 μg/dL and 1.4 percent (or 199,700) were exposed to lead at levels above 25 μg/dL. This drastic reduction in the number of children believed to have elevated levels of blood lead illustrates the great success of the nation’s efforts to reduce and/or eliminate the number of persons (primarily children) exposed to the hazards of lead.

2. Adults

Young children are not the only ones showing a drastic reduction in blood lead levels. According to the CDC’s latest estimates, only 0.7 percent of the population in the U.S. have elevated BLLs (above 10 μg/dL), with the average BLL being 1.6 μg/dL. This is a decrease of 68 percent from the 1991-1994 survey period in the number of persons estimated to have elevated BLLs (above 10 μg/dL), with the average BLL being 1.6 μg/dL.
elevated BLLs coupled with a 30 percent decrease in the BLL itself over less than a 10-year period of time.

Generally, most adults with elevated BLL were occupationally exposed. The CDC has been tracking laboratory-reported adult BLLs since 1994. In 2002, the national rate for BLLs above 25 μg/dL was 8.5 per 100,000 employees. By 2004, this number declined to 7.5 per 100,000 employees.\footnote{Adult Blood Lead Epidemiology and Surveillance–United States, 2003-2004, Centers for Disease Control and Prevention, U.S. Dept. of Health and Human Serv. 55(32) Morbidity and Mortality Weekly Report 876-79 (Aug. 18, 2006), available at http://www.cdc.gov/mmwr/preview/mmwrhtml/mm5532a2.htm (last visited Aug. 24, 2006). With respect to BLLs above 40 μg/dL, there is a similar decline, with the numbers dropping from 1.5 adults per 100,000 employees in 2002 to 1.2 per 100,000 employees in 2004. Id. This represents a 8% decrease over the same three years.} This represents a 9 percent decrease in just three years.

Currently, the top three industries with the most occupationally exposed adults are (1) the manufacturer of storage batteries, (2) painting, paperhanging and decorating, and (3) mining of lead ores.\footnote{Id.} Interestingly, the most recent data suggests that the elevated BLL of 6 percent of adults are associated with non-occupational exposures. Of these non-occupational exposures, 23 percent were associated with shooting firearms, 13 percent from remodeling and renovation activities, 11 percent from hobbies (i.e., casting ceramics or stained glass), 5 percent from retained bullet wounds or gunshot wounds, and 3 percent from pica, ingesting lead-contaminated foods, liquids or folk remedies.\footnote{Id.}

E. SIGNS AND SYMPTOMS OF LEAD EXPOSURE

Frequently, lead exposure appears asymptomatic. With increasing exposure, the severity of symptoms can be expected to increase. The impaired abilities that may be associated with lead exposure in an apparently asymptomatic patient are listed below, as are overt symptoms of lead toxicity associated with various levels of exposure. The impaired abilities may occur at BLLs ranging from 10 to 25 μg/dL, whereas in symptomatic lead intoxication, BLLs generally
range from 35 to 50 µg/dL in children and 40 to 60 µg/dL in adults. Severe toxicity is frequently found in association with BLLs of 70 µg/dL or more in children and 100 µg/dL or more in adults. Some signs and systems associated with being over exposed to lead (according to the CDC), are listed in the following table.\(^{211}\)

<table>
<thead>
<tr>
<th>CONTINUUM OF SIGNS AND SYMPTOMS ASSOCIATED WITH TOXICITY OF ONGOING LEAD EXPOSURE</th>
</tr>
</thead>
<tbody>
<tr>
<td>IMPAIRED ABILITIES (PATIENT MAY APPEAR ASYMPTOMATIC)</td>
</tr>
<tr>
<td>Decreased learning and memory</td>
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<tr>
<td>Decreased verbal ability</td>
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<tr>
<td>Early signs of hyperactivity or ADHD</td>
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<tr>
<th>MILD TOXICITY</th>
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<tbody>
<tr>
<td>Myalgia or paresthesia</td>
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<tr>
<td>Irritability</td>
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<tr>
<td>Occasional abdominal discomfort</td>
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</tbody>
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<tr>
<th>MODERATE TOXICITY</th>
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<tbody>
<tr>
<td>Arthralgia</td>
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<tr>
<td>Difficulty concentrating/ muscular exhaustibility</td>
</tr>
<tr>
<td>Headache</td>
</tr>
<tr>
<td>Vomiting</td>
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<td>Constipation</td>
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<tr>
<th>SEVERE TOXICITY</th>
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<tbody>
<tr>
<td>Paresis or paralysis</td>
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<tr>
<td>Lead line (blue-black) on gingival tissue (gums)</td>
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</table>

\(^{211}\) Lead Toxicity, supra note 202, at 25-26.
F. **Exposure Pathways**

We are exposed to lead through every conceivable exposure pathway. The following

![Diagram of lead exposure pathways](image)

figure represents an attempt by the National Academy of Sciences to diagram the multiple environmental pathways that result in human exposure to lead.²¹²

We may be exposed to lead by eating food or drinking water that contains lead. People who live in areas where there are old houses may be exposed to poorly maintained lead-based paint. Similarly, people who live near busy highways or on old orchard land where lead arsenate

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pesticides were used in the past may be exposed to higher levels of lead. Children may ingest lead by eating soil, by breathing lead dust and by chewing on surfaces containing lead-based paint. People may also be exposed to lead when they work in jobs where lead is used or have hobbies in which lead is used, such as making stained glass.\textsuperscript{213}

Skin contact with dust and dirt containing lead occurs every day. Recent data have shown that inexpensive cosmetic jewelry pieces sold to the general public may contain high levels of lead which may be transferred to the skin through routine handling.\textsuperscript{214} In the home, you or your children may be exposed to lead if you take some types of home remedy medicines that contain lead compounds. Lead compounds are in some non-Western cosmetics, such as surma and kohl. Some types of hair colorants, cosmetics and dyes contain lead acetate.\textsuperscript{215}

1. Air

In the past, transportation sources, particularly automotive sources, were the major contributor to air emissions of lead. Today, industrial processes, especially metal processing, are the major sources of lead emissions to the atmosphere with the highest lead concentrations found around smelters and battery manufacturers.\textsuperscript{216}

Airborne concentrations of lead in the U.S. have fallen dramatically over the last 30 years due largely to the phase-out of leaded gasoline additives. Major declines over several orders of magnitude have been observed not only in urban areas, but also in rural regions and remote

\begin{footnotes}
\footnote{213}{Toxicological Profile, supra note 118, at 4.}
\footnote{214}{Id.}
\footnote{215}{Id.}
\end{footnotes}
According to the latest information available, the ambient level of lead in the air is between 0.0 and 0.1 μg/m³ as shown in the following graph.218

![Graph of Lead Air Quality, 1983–2002](image)

According to the Toxic Release Inventories (“TRI”) data, in 2002, a total of 288,082 pounds of lead were released to air from 4,297 reporting facilities and 1,175,868 pounds of lead compounds were released to air from 4,312 reporting facilities.219 The emissions of lead and lead compounds to the atmosphere reported to TRI has declined from 2.8 million pounds in 1988 to 1.3 million pounds in 1997 when new industries were added to TRI reporting requirements. In 2000, before the reporting thresholds were drastically reduced, air emissions were 1.5 million

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217 Criteria Document, supra note 44, at 3-3. Today there are four long-term networks in the United States that provide data on ambient air concentrations of lead, all funded in whole or in part by the EPA. Id. at 3-4.


219 Toxicological Profile, supra note 118, at 281 (noting that TRI data should be used with caution because only certain types of facilities are required to report releases to the EPA – it does not include employers with fewer than 10 employees or if they use less than 100 pounds of lead or lead compounds in a calendar year).
pounds. In 2004, a total of 1.35 million pounds of lead and lead compounds were released into the air from industrial facilities.\textsuperscript{220}

2. Soil and Dust

Lead-contaminated soil can be directly ingested through hand-to-mouth behavior common in children, indirectly ingested through contaminated food, or inhaled when breathing air containing re-suspended soil particles. Soil ingestion, as reported by parents, peaks during the second year of life and diminishes thereafter.\textsuperscript{221} For children, dust ingested via hand-to-mouth activity may be one of the most important exposure pathways.

Outside of the home, the major sources of lead in urban soils are automotive traffic from the days of leaded gasoline and poorly maintained exterior lead-based paint.\textsuperscript{222} Soil concentrations decrease both with depth and distance from roadways. The concentration of soil lead varies significantly throughout urban areas depending on proximity to stationary sources and roadways and on wind speed and direction. Extensive lead studies in Baltimore, New Orleans, and cities throughout Minnesota found the highest concentrations of lead in the central sections of each city (where traffic and population density are greatest) while the lowest concentrations were found in the outskirts of these cities and in smaller cities.\textsuperscript{223} In all of these


\textsuperscript{221} Criteria Document, supra note 44, at 3-18 (citing Bruce P. Lanphear et al., Environmental Lead Exposure During Early Childhood, 140 J. PEDIATRICS 40-47 (2002)).

\textsuperscript{222} Id. at 3-16-17. In one study of 831 homes in the U.S., 24\% of housing units that had deteriorated, exterior, lead-based paint had bare soil lead levels in excess of 1200 ppm. Id. (citing D.E. Jacobs et al., The Prevalence of Lead-Based Paint Hazards in U.S. Housing, 110 ENVT. HEALTH PERSPECTIVES A599-606 (2002)). For housing units without deteriorating paint, just 4\% of homes had soil lead levels greater than 1200 ppm. In one study of several urban areas, there was little correlation between soil lead and the age of nearby houses, which suggests that lead-based paint may not be as significant of a source as automotive lead under some conditions. Id. (citing H.W. Mielke, Lead Dust Contaminated USA Communities: Comparison Of Louisiana And Minnesota, 2 APPL. GEOCHEM. SUPPL. 257-261 (1993)).

\textsuperscript{223} Id. at 3-17.
studies, the age of housing did not seem to be a major factor, which suggests that the impacts of lead-based paint may be dominated by historic emissions of leaded gasoline additives.\textsuperscript{224}

Inside the home, the lead in the dust can come from multiple sources. One researcher classified the sources of lead in house-dust in London by particle size. He determined that the predominant source of lead in the large 64 to 1000 μm size range was lead-based paint. Smaller sized dust particles could come from paint, road dust and garden soil.\textsuperscript{225}

3. **Drinking Water**

The United States Center for Disease Control estimates that lead in drinking water contributes between 10 and 20 percent of the total lead exposure in young children.\textsuperscript{226} Lead gets into drinking water primarily as a result of corrosion from lead pipes, lead-based solder, or brass or bronze fixtures within a residence.\textsuperscript{227} The concentration of lead in drinking water can vary greatly, depending on the corrosivity of the water, the type and age of the plumbing materials used in the house, and the length of time the water stands in the pipes. The highest levels of lead occur when very corrosive water is motionless in lead or lead-soldered copper pipe for long periods of time.\textsuperscript{228}

\textsuperscript{224} Id.

\textsuperscript{225} Id. at 3-26 (citing A. Hunt \textit{et al.}, \textit{Apportioning The Sources Of Lead In House Dusts In The London Borough Of Richmond, England}, 138 SCI. TOTAL. ENV’T. 183-206 (1993)).

\textsuperscript{226} Fact Sheet: Lead and Drinking Water from Private Wells, Ctr. for Disease Control and Preventions, U.S. Dept. of Health & Human Serv. (2003), \textit{available at} http://www.cdc.gov/ncidod/dpd/healthywater/ factsheets/pdf/lead.pdf (last visited Aug. 22, 2006). This is known as the first flush or first draw phenomenon. It is generally observed in the morning after water has stayed in the pipe through the night. An estimated 47\% of total leached lead was observed in the first 500 mL of water after prolonged stagnation, with 60 to 75\% of total lead leached appeared in the first 125 mL of water after prolonged stagnation. For cold water, the peak lead concentrations occurred in the first or second 25 mL sample and decreased exponentially with time thereafter. For hot water the peak lead concentration occurred in the second or third 25 mL sample before decreasing exponentially. Criteria Document, \textit{supra} note 44, at 3-36 to 3-37 (citations omitted).

\textsuperscript{227} Criteria Document, \textit{supra} note 44, at 3-33. Very little lead in drinking water comes from the municipal water supply. Tap water analyses for a public school, apartments and free-standing houses indicate that the indoor plumbing is a greater source of lead in drinking water than the utility, even for residences and schools serviced by lead-pipe water mains. \textit{Id.}

The type of plumbing system used in a house can often be determined by its age. Through the early 1900s, it was common for lead pipes to be used for interior plumbing in certain areas. Before the 1940s, lead pipe was often used to connect houses to the local water mains. Beginning in the 1930s, copper and galvanized steel pipes began being used in most residential homes. In the U.S., copper piping with lead-based solder has largely replaced pure lead piping.\(^{229}\) In houses with copper piping and lead-based solder, brass fixtures may contribute as much as 50% of lead in drinking water. Before the Safe Drinking Water Act\(^ {230}\) was amended in 1986, the primary type of solder used in the U.S. was 50–50 tin-lead solder (50% tin, 50% lead). Although new or repaired pipes may not use solder containing more than 0.2% lead, 50-50 solder still exists in many older structures.\(^ {231}\)

Researchers have determined that about 10.2% of homes had lead levels greater that 15 μg/l in the first portion of water drawn from the tap ("first-draw"). They also found that in 83% of cases, the level could be reduced to less then 15 μg/l simply by running the tap for one full minute.\(^ {232}\) For example, in the District of Columbia, in an analysis of 96 residences, a 95% reduction in lead was achieved when water was flushed for 10 minutes. These findings strongly suggest that childhood tap water lead exposure, especially in homes without lead service lines, can usually be reduced greatly simply by flushing the water for a specified length of time prior to

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\(^{229}\) Criteria Document, \textit{supra} note 44, at 3-33. A survey of 94 water companies nationwide in 1988 revealed that copper pipe was present in 73% of homes, galvanized pipe was present in 13% of homes, a mixture of galvanized and copper was present in 11% of homes and plastic pipes were present in 2% of homes. \textit{Id.} at 3-33 to 3-35.

\(^{230}\) 42 U.S.C.\$300f, \textit{et seq.}

\(^{231}\) Criteria Document, \textit{supra} note 44, at 3-35.

use, e.g., one minute.\textsuperscript{233} Lead-brass water meters are another source of lead-contaminated water in residential homes. Research has shown that these meters, traditionally manufactured using alloys containing 5\% to 7\% brass, discharge substantial amounts of lead into household water. The long-term concentrations of lead discharged from these meter ranged from less than 5 \(\mu g/l\) to 40–50 \(\mu g/l\) depending on the corrosivity of the public water supply.\textsuperscript{234}

4. Food

Lead-contaminated food and beverages are a major source of lead exposure for everyone in the U.S. About 5 percent of children ingest enough lead from food and beverages to be at risk for adverse health effects. Lead enters the food chain from soil, deposition from the air, containers or dishes and from food processing equipment. Some studies of lead ingestion from food showed that North Americans ingest an estimated 50 \(\mu g\) of lead each day through the food they eat and the beverages they drink.\textsuperscript{235} In 1987, the global average daily intake is about 80 \(\mu g/day\) from food and 40 \(\mu g/day\) from drinking water, according to estimates made by the UN Environment Program.\textsuperscript{236} However, more recent data from the FDA indicates that lead intake from food decreased between the early 1980s and mid-1990s. For example, the dietary lead intake by infants aged 6-11 months had dropped to a range of 0.8 to 5.7 \(\mu g/day\) in the mid-1990s from 16.7 \(\mu g/day\) in the early 1980s. For 2 year old children, the dietary intake of lead dropped to a range of 2.4 to 10.1 from 23.0 \(\mu g/day\) during the same time period. For older children and adults, the dietary intake of lead dropped to a range of 4 to 19 \(\mu g/day\) from 28.7 to 41.3 \(\mu g/day\)

\begin{itemize}
  \item \textsuperscript{234} Id. (citation omitted).
  \item \textsuperscript{235} Criteria Document, \textit{supra} note 44, at 3-41 to 3-42 (citing A.R. FLEGAL \textit{ET AL.}, \textit{LEAD CONTAMINATION IN FOOD in FOOD CONTAMINATION FROM ENVIRONMENTAL SOURCES} 85-120 (J.O. Nriagu, & M.S. Simmons eds, John Wiley & Sons, Inc. 1990)).
  \item \textsuperscript{236} Id. at 3-42 (citing D.R. Juberg \textit{et al.}, \textit{Position Paper Of The American Council On Science And Health: Lead And Human Health}, \textit{38 ECOTOXICOLOGY & ENVT’L SAFETY} 162-80 (1997)).
\end{itemize}
during the same time period. The EPA attributes this reduction in the dietary intake of lead directly to the decrease in airborne emissions of lead from automotive gasoline as well as the reduction in the use of Pb solder in cans.

Since the elimination of lead solder in U.S. canned food and the pesticide lead arsenate, the primary source of lead in U.S. food is atmospheric deposition. Overall, smog and haze account for an estimated 40% of lead in food, while the bulk of the remainder is derived from harvesting, transporting, processing, packaging, or preparing the food. Lead contamination in poultry and livestock is also primarily atmospheric in origin. Lead settles from the atmosphere onto forage, feed or onto soil that is directly ingested by animals.

As a result of contamination during processing and transportation, lead concentrations in food have been reported to increase by a factor of 2 to 12 between harvest and consumption. A food production facility in Turkey was shown to contaminate pasta with lead, as indicated by lead concentrations in the semolina being 14.2 to 36.5 ng/g compared with the finished pasta product where concentrations ranged from 107.1 to 147.6 ng/g. An increase between raw and finished cocoa products has also been observed (from an average value ≤0.5 ng/g to average

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237 Id. (citations omitted). Overall, recent studies conducted in the United States indicate that daily dietary lead intake ranges from about 1 to 10 μg/day. Some researchers have estimated the contribution of lead from sources other than food in the diet. For example, one researcher estimated a daily lead intake of 8.4 μg/day in children based on a diet survey, but when they estimated exposure due to the handling of food by children (including lead from the floor and house dust), the daily lead intake from ingestion was estimated at 29.2 μg/day. Id.

238 Id. at 3-41.

239 Use of lead arsenate pesticides began in the United States in 1892 and continued for 100 years, right through the mounting furor over leaded gas and paint, until the EPA canceled their registration in 1993. Deborah K. Rich, Not Easy To Get The Lead Out Of Our Lives: Recent Evidence Suggests Rules Don’t Go Far Enough To Protect Children, F-6 SAN FRANCISCO CHRON. (Aug. 19, 2006). Lead arsenate was employed extensively on apple orchards but was also used for control of agricultural pests in vegetable fields and other fruit orchards. Golf courses and turf farms also received applications of lead arsenate on a regular basis. Lead arsenate was generally applied at a rate of several pounds per acre. Findings and Recommendations for the Remediation of Historic Pesticide Contamination; Final Report, New Jersey Historic Pesticide Contamination Task Force 7 (Mar. 1999).

240 Criteria Document, supra note 44, at 3-48 (citation omitted).
values between 11.9 and 69.8 ng/g). In that case, contamination seems to have occurred during shipping and/or processing.\footnote{241}{Id. (citations omitted).}

Many non-Western folk remedies used to treat diarrhea or other ailments may contain high levels of lead. Examples of these include: Alarcon, Ghasard, Alkohl, Greta, Azarcon, Liga, Bali Goli, Pay-loo-ah, Coral and Rueda.\footnote{242}{Toxicological Profile, supra note 118, at 315.} In 1997, there was an adult case of lead poisoning attributed to an Asian remedy for menstrual cramps known as Koo Sar.\footnote{243}{Adult Lead Poisoning from an Asian Remedy for Menstrual Cramps – Connecticut, 1997, Morbidity and Mortality Weekly Report, Centers for Disease Control and Prevention (Jan. 29, 1999). On February 19, 1997, a 33-year-old Cambodian woman, her husband, and their two children were screened at a free lead-screening event sponsored by a nursing school community health promotion center. All were tested for lead, but only the woman had an elevated level of lead in her blood (44 µg/dL, and later confirmed at 42 µg/dL). The woman reported no symptoms associated with lead poisoning (e.g., muscle pains or weakness, headaches, or loss of appetite). The CDC requested samples of any medicines, teas, or cosmetics that the woman had used that might have been the source of the lead and the Koo Sar pills were found to contain lead. Id.}

The pills contained lead at levels as high as 12 ppm. The source of the lead was thought to be in the red dye used to color the pills.\footnote{244}{Id.} Lead is a common adulterant found in samples of Asian traditional remedies available at health food stores and Asian groceries in Florida, New York and New Jersey. Sixty percent of the remedies tested would give a daily dose of lead in excess of 300 mg when taken according to labeling instructions.\footnote{245}{Toxicological Profile, supra note 118, at 315.} Similarly, lead poisoning has been caused by ingestion of a Chinese herbal medicine to which metallic lead was added to increase its weight and sales price. Likewise, during 2000–2003, 12 cases of lead poisoning among adults were reported in five states due to the use of ayurveda medications obtained from ayurvedic physicians. Ayurveda is a traditional form of medicine practiced in India and other South Asian countries; the medications used often contain herbs, minerals, metals, or animal products and are made in standardized and non-standardized formulations.\footnote{246}{Id. at 315-16.}
Lead contamination may also come from the use of lead crystal. Lead may leach from lead crystal decanters and glasses into the liquids they contain. Port wine that contained an initial concentration of 89 μg/L lead was stored for 4 months in crystal decanters containing up to 32% lead oxide. At the end of 4 months, lead concentrations in the port were 5,331 μg/L, 3,061 μg/L and 2,162 μg/L in decanters containing 32%, 32%, and 24% lead oxide, respectively. Of more concern, lead was also found to leach from lead crystal wine glasses within minutes. Mean lead concentrations in wine contained in 12 glasses rose from 33 μg/L initially to 68 μg/L, 81 μg/L, 92 μg/L and 99 μg/L after 1, 2, 3 and 4 hours, respectively.

G. BILOGIC FATE

Lead exposure is the amount of lead (from whatever source) that is presented to an organism; dose is the amount that is absorbed by the organism. Various factors—such as blood flow and capillary permeability, which transport lead into an organ or tissue as well as a number of active binding and receptor sites—determine the path of lead through the body and can influence the biologically effective dose. For example, lead-laden dust could be retained in the lungs, removed from the lungs by protective mechanisms and ingested, stored in bone, or eliminated from the body via the kidneys. Toxicity can be observed in the kidneys, blood, nervous system, or other organs and tissues. At any step after exposure, biologic markers of exposure to lead can be detected. Lead is primarily absorbed through two primary routes: gastrointestinal and inhalation. Whether lead is inhaled or ingested, most of the lead that is

247 Id. at 316 (citation omitted).
248 Id. (citation omitted).
249 CLS Study, supra note 151, at 144.
250 Toxicological Profile, supra note 118, at 144. Dermal absorption of lead compounds is generally considered to be much less than absorption by inhalation or oral routes of exposure. See also, CLS Study, supra note 151, at 144.
251 Criteria Document, supra note 44, at 4-5. Inhaled tetraalkyl lead vapor is nearly completely absorbed following deposition in the respiratory tract. A study of the dispensation of a single exposure to vapors of radioactive tetraethyl lead showed that 37% of it was initially deposited in the respiratory tract, of which approximately 20% was exhaled in the subsequent
absorbed into the body is excreted either by the kidney (in urine) or through biliary clearance (ultimately, in the feces). The amount of lead excreted and the timing of excretion depend on a number of factors, including age and nutrition.

Studies have shown that adults excrete the majority (50%-60%) of an absorbed fraction of lead (when in a steady-state condition with regard to lead intake/output) on a short-term (several weeks) basis, and the vast majority of absorbed lead over time. Ultimately, while adults retain only about 1% of absorbed lead, children up to about age 2 retain about one-third of the total amount of absorbed lead.252

Lead absorption in children is affected by nutritional iron and calcium status. Children who are iron-deficient have higher blood lead concentrations which suggests that iron deficiency may result in higher lead absorption or, possibly, other changes in lead biokinetics that contribute to altered blood lead concentrations.253 The same is true for calcium where an inverse relationship has been observed between dietary calcium intake and blood lead concentration in both children and adults. Consequently, children who are calcium-deficient may absorb more lead than calcium-replete children.254

H. DISTRIBUTION IN THE BODY

1. Lead in Blood

Because absorbed lead enters plasma and undergoes rapid removal to various body compartments, blood lead concentration is the most widely used and considered to be the most

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252 Lead Toxicity, supra note 202, at 13; see also Criteria Document, supra note 44, at 4-5 to 4-6; Toxicological Profile, supra note 118, at 137.

253 Criteria Document, supra note 44, at 4-7 (noting that in studies of rats, iron deficiency increases the gastrointestinal absorption of lead, possibly by enhancing binding of lead to iron binding proteins in the intestine).

254 Id.
reliable biomarker for general clinical use and public health surveillance. Currently, blood lead measurement is the screening test of choice to identify children with elevated levels of lead in their blood.\textsuperscript{255} In lead toxicologic and epidemiologic studies, dose-response relationships for nearly all of the major health effects of lead are typically expressed in terms of an index of internal lead dose. Blood lead concentration is extensively used in epidemiologic studies as an index of exposure and body burden due mainly to the feasibility of incorporating its measurement into human studies relative to other potential dose indicators (\textit{e.g.}, lead in kidney, plasma, urine, or bone). Blood lead is determined by both the recent exposure history of the individual, as well as the long-term exposure history that leads to accumulation in bone.

Although the blood generally carries only a small fraction of the total lead body burden, it serves as the initial receptacle of absorbed lead and distributes lead throughout the body, making it available to other tissues (or for excretion).\textsuperscript{256} The half-life of lead in adult human blood has been estimated to be from 28 to 36 days. Approximately 99\% of the lead in blood is associated with red blood cells; the remaining 1\% is found in blood plasma. It is blood plasma, however, which transfers lead between the blood compartment and the soft and mineralizing tissues and which therefore may be more biologically significant. In addition, the higher the lead concentration in the blood, the higher the percentage partitioned to plasma.\textsuperscript{257}

As stated previously, blood lead concentrations in the U.S. general population have decreased over the past three decades as regulations regarding lead paint, leaded fuels and lead-containing plumbing materials have decreased exposure. Data from the most recent survey years (2001-2002) show the following blood lead concentrations:

\textsuperscript{255} Toxicological Profile, supra note 118, at 144.
\textsuperscript{256} Lead Toxicity, supra note 202, at 14; Toxicological Profile, supra note 118, at 146-47.
\textsuperscript{257} Id.
• for babies >1 year of age 1.45 μg/dL;
• for children ages 1 to 5 1.70 μg/dL;
• for children ages 6-11 1.25 μg/dL;
• for children ages 12-19 0.94 μg/dL;
• for adults over age 20 1.56 μg/dL;
  o for males over age 20 1.78 μg/dL;
  o for females over age 20 1.19 μg/dL.258

To put these numbers in perspective, in 1974-75, the average blood lead concentration in the U.S. was 16.5. Today, if a child has a blood lead level between 10 and 14 μg/dL, it should trigger community-wide childhood lead poisoning prevention activities.259

2. Lead in Soft Tissues

The blood distributes lead to various organs and tissues.260 Most of the lead in soft tissue is in the liver. Relative amounts of lead in soft tissues expressed as a percent of total soft tissue lead were: liver, 33%; skeletal muscle, 18%; skin, 16%; dense connective tissue, 11%; fat, 6.4%; kidney, 4%; lung, 4%; aorta, 2%; and brain, 2% (other tissues were <1%). The highest soft tissue concentrations in adults also occur in liver and kidney cortex.261

3. Lead in Our Bones and Teeth

In human adults, approximately 94% of the total body burden of lead is found in the bones. In contrast, bone lead accounts for 73% of the body burden in children.262 Lead

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258 Criteria Document, supra note 44, at 4-22.
259 Toxicological Profile, supra note 118, at 204.
260 Lead Toxicity, supra note 202, at 14. Animal studies indicate that the liver, lungs and kidneys have the greatest soft-tissue lead concentrations immediately after acute exposure regardless of pathway (i.e., inhalation, oral, dermal and intravenous routes). Id. (citation omitted).
261 Toxicological Profile, supra note 118, at 149 (citations omitted).
262 Id. at 147; Criteria Document, supra note 7, at 4-13 (citation omitted).
concentrations in bone increase with age throughout the lifetime, indicative of a relatively slow turnover of lead in adult bone. This large pool of lead in adult bone can serve to maintain blood lead levels long after exposure has ended. It can also serve as a source of lead transfer to the fetus when maternal bone is resorbed for the production of the fetal skeleton.263

Lead in mineralizing tissues is not uniformly distributed; however, it tends to accumulate in bone regions undergoing the most active calcification at the time of exposure. During infancy and childhood, bone calcification is most active in trabecular bone; whereas, in adulthood, calcification occurs at sites of remodeling in cortical and trabecular bone. This suggests that lead accumulation will occur predominantly in trabecular bone during childhood, but in both cortical and trabecular bone in adulthood.264

I. PHYSIOLOGIC EFFECTS

1. Neurological Effect

We are only now beginning to have an appreciation of how the nervous system is affected by lead. Because the past 10 years have been a period of very rapid change in understanding the toxicity of lead, much of what has been written is now outdated. At lower exposures, effects on the peripheral nervous system can occur. These include behavioral changes, visual-motor deficiencies and cognitive motor function difficulties (coordination, speech and memory can be affected). These effects can occur at blood levels of 30 µg/dL in children.

The nervous system is the most sensitive target of lead exposure. Young children (and fetuses) are especially vulnerable to the neurologic effects of lead because:

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263 Criteria Document, supra note 44, at 4-14 (citations omitted).
264 Id. (citation omitted).
they have a higher intake of lead as a proportion of the body weight as compared to adults;
they often place objects in their mouths, possibly resulting in lead laden dust and soil being ingested;
they have higher physiological uptake rates of lead as compared to adults;
their brains and nervous systems are still developing and the blood-brain barrier is incomplete.\textsuperscript{265}

Scientists first began using animal studies to observe the effects of lead in developing rats in the mid-1960s.\textsuperscript{266} Since then there has been a continuing research focus on defining the extent lead affects the central nervous system (“CNS”). While significant research advances have been made in animal studies over the last four decades, relating these findings to neurotoxicity in children has been challenging and difficult.\textsuperscript{267}

At very high levels of lead exposure (creating BLLs of 100 to 120 \( \mu g/dL \) in adults and 80 to 100 \( \mu g/dL \) in children), serious neurotoxic effects occur including acute lead poisoning that can progress to convulsions, coma and sudden death. Less severe lead exposures (creating BLLs of 40 to 60 \( \mu g/dL \)) produce both central and peripheral nerve dysfunction including slowed nerve conduction velocity and overt signs and symptoms of neurotoxicity. Decrements in IQ have been observed in children with BLLs of 30 to 50 \( \mu g/dL \), with some studies showing effects at lower BLLs. Neurobehavioral effects are observed in rats and monkeys at levels below 20

\begin{footnotesize}
\begin{itemize}
\item \textsuperscript{265} Shilu Tong \textit{et al.}, Bulletin; Environmental Lead Exposures: A Public Health Problem of Global Dimensions, Ref. No. 0686, World Health Org. 1069 (2000); Lead Toxicity, \textit{supra} note 202, at 16.
\item \textsuperscript{266} Criteria Document, \textit{supra} note 44, at 5-18 (citing A. Pentschew & F. Garro, \textit{Lead Encephalo-Myelopathy of the Suckling Rat and its Implications on the Porphyrinopathic Nervous Diseases, With Special Reference to the Permeability Disorders of the Nervous System’s Capillaries}, 6 \textit{Acta Neuropathol} 266-278 (1966)).
\item \textsuperscript{267} \textit{Id.}
\end{itemize}
\end{footnotesize}
µg/dL. Nonetheless, it is because of these observed effects that the level of concern related to exposure to lead had decreased from 60 µg/dL to 10 µg/dL.

Before the mid-1960s, a level above 60 µg/dL was considered toxic. In the 1960s, blood lead levels greater than 60 µg/dL concerned medical care providers. Beginning in the 1970s, our understanding of childhood lead poisoning changed. As investigators have used more sensitive measures and better study designs to study lead’s effect on the central nervous system, the generally recognized level for lead toxicity progressively shifted downward. By 1978, CDC’s defined level of toxicity had declined 50% to 30 µg/dL. In 1985, this level was lowered again to 25 µg/dL. The 1985 statement made clear that adverse effects were recognized as occurring blood lead concentrations below 25 µg/dL (so the chosen criteria represented the best compromise between health protection and practical matters related to limitations in screening methods at that time). Finally, in 1991, the CDC reduced the level of concern to 10 µg/dL. Recently, the CDC reviewed the appropriateness of its 10 µg/dL level of concern for lead and chose not to lower it further.

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268 Id. at 5-18 to 5-19.
271 Id. CDC’s criteria for identifying lead toxicity underwent a number of changes. The 1975 and the 1978 revisions of CDC guidelines used 30 µg/dL or above and different definitions of high erythrocyte protoporphyrin (EP) concentrations to produce several risk categories. CLS Study, supra note 151, at 27.
272 Roper, supra note 270. (acknowledging that CDC set the 25 µg/dL level of concern for blood lead although it was acknowledged that adverse effects occur below that level).
273 CLS Study, supra note 151, at 28.
274 Id. In this report the CDC proclaimed that “it is not possible to select a single number to define lead poisoning for the various purposes of all of these groups.” It acknowledged that epidemiologic studies have identified harmful effects of lead in children at blood lead levels at least as low as 10 µg/dL and recognized that 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 µg/dL to be evaluated definitively. It further recognized that no threshold has been identified for the harmful effects of lead. Id.
275 See supra, Section III.C.2, for CDC’s reason for leaving the lead level of concern unchanged.
i. Children

Because their brains are still developing, it is generally believed that children suffer neurologic effects at much lower exposure levels due to their greater absorption and retention of lead.\(^{276}\) Therefore, scientists have been trying since the mid-1960s to understand how lead effects the central nervous system at sub-chronic levels of exposure. While significant research advances have been made in animal studies over the last four decades, there are problems with extrapolating the results of these animal studies and applying them to children.\(^{277}\) For example, animal studies show that alterations in neurobehavioral function can persist long after lead exposure has stopped and BLLs have returned to normal, but scientists have been unable to duplicate these results in human studies. In its 2006 Lead Criteria Document, the EPA notes that “[h]uman studies provide little information on the persistence of effects that lead may have on the brain.”\(^{278}\) There can be multiple reasons why scientists are having such a difficult time studying the effects of low level exposures to lead on the brain. Some believe the principal problem is lead’s multiple toxic mechanisms of action in brain tissue, which encompass variable, overlapping, and, at times, opposing dose-effect relationships.\(^{279}\) The problem could also be a result of factors that confound the studies. Or, the reason why the scientist have been unable to duplicate the results of the animal studies in man is simply because “Man is not a big mouse.”

(a) Some Studies Associate Lead Exposure to Neurological Problems

There is a body of evidence that “associates” decrement in intelligence quotient (IQ) performance and other neuropsychologic defects with lead exposure. Some studies have found,

\(^{276}\) Criteria Document, supra note 44, at 5-36.

\(^{277}\) Id. at 5-18. The EPA delicately addresses this problem by noting that “[w]hile significant research advances have been made in animal studies over the last four decades, relating these findings to neurotoxicity in children has been challenging and difficult.” Id.

\(^{278}\) Id.

\(^{279}\) Id.
for example, that for every 10 µg/dL increase in BLL, children’s IQ dropped by four to seven points. There is also some evidence that the probability of ADHD, hearing impairment and vision problems in children increases with increasing BLLs. Some scientists believe that these effects begin at low, more widespread BLLs (at or below 10 µg/dL in some cases). There is also a belief among some researchers that some of the neurologic effects of lead in children may persist into adulthood. One study, for example, correlated lead exposure with lower class standing (classroom performance); greater absenteeism; more reading disabilities; and deficits in vocabulary, fine motor skills, reaction time, and hand-eye coordination in young adults more than 10 years after childhood exposure. The problem with these beliefs, however, is that it may not be possible to detect these sorts of problems during a clinical examination. While these studies are important and help increase our understanding of the medical science, the majority of studies, if not all of them, have fundamental weaknesses and limitations as a result of confounders and/or other problems.

(b) Confounders

Depending on the subjects being examined for effects associated with exposure to lead, numerous factors can lead to confounding of the relationship being considered. Deciding which of the potential confounding factors are important largely depends on the health outcome of interest and the study population. It is difficult to distinguish between neurobehavioral effects

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280 See Lead Toxicity, supra note 202, at 16 (citations omitted); see also Bruce P. Lanphear et al., Low-Level Environmental Lead Exposure And Children's Intellectual Function: An International Pooled Analysis, 113 Envt’l Health Perspectives 894-899 (2005).

281 See e.g., D. M. Fergusson et al., Early Dentine Lead Levels And Subsequent Cognitive And Behavioural Development, 34 J. CHILD PSYCHOLOGY & PSYCHIATRY & ALLIED DISCIPLINES 215-227 (1993). However, the EPA notes that there is no compelling evidence that lead exposure is directly related to ADHD. Criteria Document, supra note 44, at 6-45.

282 Criteria Document, supra note 44, at 6-50 to 6-51 (citations omitted).

283 Id. at 6-254 to 6-255.


285 Lead Toxicity, supra note 202, at 16.
due to lead and effects owing to the many social, economic, urban-ecological, nutritional, and other medical factors that are known to have important effects on neurobehavioral development. It is highly probable that the concentration-response relationship and even the neurobehavioral lesion associated with childhood lead exposure may vary as a function of these confounders.\textsuperscript{286}

Of all the confounders, one of the most complex and most important appears to be the socioeconomic status of the child ("SES"). Moreover, the most significant SES confounder with respect to a child’s IQ is their mother’s IQ. The exact mechanisms of the impact of SES on lead’s neurotoxic effects on the CNS are unknown. Individually or collectively, factors such as poverty, pre-1960 housing in segregated communities, ethnicity, and nutritional deficiencies can contribute to neurological problems.\textsuperscript{287} One noted researcher has admitted that complete confounder control is impossible in real world studies. As a result, in most studies reviewed, control of confounders has reduced the magnitude of the lead-IQ effect. But, he notes that it has not obliterated the lead-IQ effect.\textsuperscript{288} This reduction was even noted in Dr. Lanphear’s pooled analysis of seven prospective studies\textsuperscript{289} where the lead affect on IQ has been characterized as

\textsuperscript{286} Criteria Document, supra note 44, at 6-10 to 6-11. Some potential confounding factors in children include: neurological and developmental deficiencies, include: socioeconomic status (SES); nutritional status; quality of home environment (e.g., HOME score); parental education; parental IQ; and birth weight, as a few examples. \textit{Id.} at 6-7.

\textsuperscript{287} \textit{Id.} at E-9, 4-21, 4-80, 5-71 and 5-102 to 5-103. Historically, the SES confounders were measured rather crudely in most studies with such indices as the Hollingshead Four-Factor Index of Social Position that incorporates education and income of both parents. In an effort to better control for confounding by SES more recent lead studies have incorporated more direct assessments such as the HOME scale, parental intelligence, parental attitude assessments, and measures of parental substance abuse and psychopathology. \textit{Id.} at 6-73 to 6-74.

\textsuperscript{288} Herbert L. Needleman & C.A. Gatsonis, \textit{Low-level lead exposure and the IQ of children. A meta-analysis of modern studies,} 263 J. Am. Med. Assoc. 673-78 (1990). It is often the case that following adjustment for factors such as social class, parental neurocognitive function, and child rearing environment using covariates such as parental education, income, and occupation, parental IQ, and HOME scores, the lead coefficients are substantially reduced in size and statistical significance. Criteria Document, supra note 44, at 6-75. In the Port Pirie study, (I. S. Tong, & Y. Lu, \textit{Identification Of Confounders In The Assessment Of The Relationship Between Lead Exposure And Child Development.} 11 ANN. EPIDEMIOLOGY 38-45 (2001)) the authors observed that adjustment for four factors (i.e., quality of home environment, SES, maternal intelligence, and parental smoking behavior) reduced the magnitude of the estimated association between Pb and IQ by 40% and inclusion of additional factors resulted in another 10% reduction. \textit{Id.} at 6-265.

\textsuperscript{289} See supra note 280.
modest after an attempt was made to account for the confounders.\textsuperscript{290} It has even been noted that blood lead typically accounts for only 1 to 4\% of the variance in child IQ scores, compared to the 40\% or more by social and parenting factors.\textsuperscript{291}

In their 2006 Criteria Document on Lead, the EPA recognizes the profound influence SES factors have in potentially skewing the results of neurological studies because it is an effect modifier.\textsuperscript{292} The EPA also acknowledged that SES confounders have been found to exacerbate the causal relationship between lead and the neurological parameter being studied.\textsuperscript{293} It further acknowledges that in the analyses of data regarding lead effect on child development is the lack of critical consideration of which potential confounder in a particular model “owns” the variance in neurodevelopmental performance. For example, in the case of social class, it is assumed that if an effect of lead is reduced to nonsignificance following adjustment for some measure of SES standing, the assumption is that all of the variance belongs to the confounder.\textsuperscript{294}

Although EPA recognizes the problems raised by confounders, it still questions the possibility that lead nonetheless does have a causal effect on the brain at low doses (BLLs at or below 10 µg/dL).\textsuperscript{295} In support of this possibility it looks to experimental animal studies and

\textsuperscript{290} The crude coefficient for concurrent lead and childhood IQ score was −4.66 (95\% CI: −5.72, −3.60), while the coefficient adjusted for study site, quality of the home environment (HOME score), birth weight, maternal IQ, and maternal education was −2.70 (95\% CI: −3.74, −1.66). When expressed as the percentage of variance accounted for in a health outcome, the contributions of lead have been characterized as modest in magnitude. Criteria Document, supra note 44, at 6-265.

\textsuperscript{291} Id. at 6-265 to 6-266 (citing K. Koller et al., Recent Developments In Low-Level Lead Exposure And Intellectual Impairment In Children, 112 ENVT’L HEALTH PERSPECTIVES 987-994 (2004)).

\textsuperscript{292} Id. at 6-75. Effect modification occurs when the magnitude of an association between an exposure (lead) and an outcome (neurobehavior) varies across strata of some other factor. Id. The EPA recognizes that the inability to measure and control confounders is a concern and could result in a finding that the lead affect could be reduced further, perhaps all the way to the null, if better, more comprehensive methods of measurement were applied. Id. at 6-266.

\textsuperscript{293} Id. (citing D.C. Bellinger, Effect Modification In Epidemiologic Studies Of Low-Level Neurotoxicant Exposures And Health Outcomes, 22 NEUROTOXICOLOGY & TERATOLOGY 133-140 (2000)).

\textsuperscript{294} Id.

\textsuperscript{295} The EPA wonders whether the methods used to adjust for such factors may be excessively conservative insofar as they attribute to a factor all of the outcome variance that it shares with lead, despite the likelihood that the true relationships among lead, social factors, and outcome are unlikely to be as simple as this model assumes (i.e., some factors might, in part, be markers of lead exposure opportunities). For example, both lead biomarker levels and lower cognitive function in children are
proclaims as fact that animals “are not compromised by the possibility of confounding by such factors as social class and correlated environmental factors.”296 Unfortunately, the EPA does not (and perhaps cannot) explain why animals cannot be affected by confounders such as social class or parental intelligence. Instead, the EPA simply states that “[t]he enormous experimental animal literature that proves that lead at low levels causes neurobehavioral deficits and provides insights into mechanisms must be considered when drawing causal inferences.”297 Whether or not animals are affected by SES type confounders is debatable. What is not debatable is that man is not a big mouse.

(c) Man is Not a Big Mouse

Even among mammals, there are many biologic and biochemical differences. Indeed, there are substantial differences in responses, even among apparently similar species of rodents, such as rats and mice, and even within subspecies of the same species. These variations are not uncommon, but if extrapolation between animal species is a delicate exercise, extrapolations from animals to humans raise even greater questions.298 Different species also have different metabolic processes and products, and absorption and metabolism may differ radically from

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296 Id. at 6-75.
297 Id. (citations omitted).
298 See CASARETT & DOULL, TOXICOLOGY: THE BASIC SCIENCE OF POISONS 26 (Curtis Klaassen et al., ed., 5th ed. 1996). For example, the following results are obviously bewildering:

- differing findings regarding carcinogenicity between rats and mice similarly exposed to 1,3-butadiene, and variations between sexes among the same species;
- production of nasal carcinomas uniquely in rats after inhalation exposure to formaldehyde;
- the development of kidney tumors associated with exposure to 2,3,5-trimethylpentane and d-limonene uniquely in male rats;
- liver tumors production in some rodent species from “peroxisomal pro-liberators” such as the antilipidemic drug clofibrate and the common solvent trichloroethylene; and
- the high resistance of mice to the carcinogenic effects of the fungal toxin aflatoxin B1 in the liver, while low doses produced significant excesses of liver tumors in rats.
species to species. Consequently, chemical agents may be toxically selective. Selective toxicity means that a chemical can produce an injury to one living system without harming another even though the two living systems may co-exist.\textsuperscript{299} Therefore, we need to know if a substance produces the same metabolite in animals and humans because only through a thorough understanding of these difference can the relevant animal data to human response be verified. If the response is not the same in both the animal and man, then a toxic finding in the animal bioassay may have little relevance to what might occur in a human. Under such circumstances, the bioassay would have been conducted in what is now termed an “inappropriate animal model.” For example, we know that butadiene metabolizes differently in rodents, monkeys, and humans. Thus, reliance on butadiene studies of monkeys and rodents are based on inappropriate animal models and the data is unhelpful for understanding toxicity to humans. “In other words, Man is not a big mouse.”\textsuperscript{300}

(d) Summary

This is an extremely complicated subject that can only be discussed in this format using broad brush stokes. The studies looking at low level exposure to lead (BLLs below 10 µg/dL) are at best suspect. First, identifying and adequately controlling confounders represents a fundamental weakness and limitation in most of these studies.\textsuperscript{301} Second, repeatability has been

\textsuperscript{299} Id. at 25 (noting that large differences in carcinogenic response between experimental animal species are not unusual).

\textsuperscript{300} See Robert Scott, Of Mice and Men: Extrapolation of Animal Data to Humans (Def. Res. Inst. April 30, 1998). Much of the information related in this section is condensed and paraphrased from Mr. Scott’s excellent article.

\textsuperscript{301} See e.g., Bruce P. Lanphear et al., Cognitive Deficits Associated With Blood Lead Concentrations < 10 Mg/Dl In U.S. Children And Adolescents, 115 PUBLIC HEALTH REP. 521-529 (2000) (noting that while a large number of potential confounding factors were controlled in these analyses, no data on maternal IQ or direct observations of caretaking quality in the home were available); see also, J. Walkowiak et al., Cognitive And Sensorimotor Functions In 6-Year-Old Children In Relation To Lead And Mercury Levels: Adjustment For Intelligence And Contrast Sensitivity In Computerized Testing, 20 NEUROTOXICOLOGY & TERATOLOGY 511-521 (1998) (Although the authors concluded that these findings roughly correspond with those of other studies that find lead effects exposure on measures of intelligence at BLL <10 µg/dL, they also cautioned that some important covariates and potential confounding variables were not measured, including parental IQ and home environment (e.g., HOME score)); see also, Katarzyna Kordas, et al., Blood Lead, Anemia, And Short Stature Are Independently Associated With Cognitive Performance In Mexican School Children, 134 J. NUTRITION 363-371 (2004) and Katarzyna Kordas et al.,
a problem. While there are some studies that claim to show a statistically significant neurological defect associated with lead, there are similar studies looking at the same exact issue that find no statistically significant evidence of the association.\(^\text{302}\) Third, some studies claims that the problem is worse at lower BLLs than at higher BLLs.\(^\text{303}\) That counter-intuitive results makes the study itself suspect. Nonetheless, some researchers discounts it by stating “that there are a number of examples of nonlinear or supralinear dose-effect relationships in toxicology. [Therefore, it] is conceivable that the initial neurodevelopmental lesions at lower Pb levels may be disrupting very different biological mechanisms than the more severe effects of high exposures that result in symptomatic poisoning or frank mental retardation.”\(^\text{304}\) Finally, there are methodology problems associated with both the method used to determine a child’s BLL and the the ability of neurobehavioral tests to detect damage to the central nervous system.\(^\text{305}\) As a result of the complexities associated with studying neurological affects as a consequence of low level exposure to lead and the multiple problems associated with confounders, there is no consensus among scientists relating to the behavioral affects of lead exposure under 10 μg/dL.

\(^{302}\) See e.g., I. Al-Saleh et al., Relationships Between Blood Lead Concentrations, Intelligence, And Academic Achievement Of Saudi Arabian Schoolgirls, 204 INT’L J. HYGIENE & ENV’T’L HEALTH 165-174 (2001) (After adjusting for various factors, including family income and parental education, a significant inverse relationship was observed between BLL and Beery-VMI. However, unlike various other studies, most notably the seven study pooled analysis by Lanphear et al. (2005), that observed larger effects at lower blood Pb levels, a significant association between blood Pb and Beery-VMI was not observed when data were restricted to those with blood Pb levels <10 μg/dL).

\(^{303}\) Criteria Document, supra note 44, at 6-70 (A common observation among some of these low blood-Pb level studies is that of nonlinear dose-response relationships between neurodevelopmental outcomes and blood Pb concentrations); see also R.L. Canfield et al., Intellectual Impairment In Children With Blood Lead Concentrations Below 10 Mg Per Deciliter, 348 NEW ENG. J. MED. 1517-1526 (2003) (the Rochester Study) (In this study, the relationship between children’s IQ score and BLLs was found to be nonlinear. A semiparametric analysis indicated a decline of IQ of 7.4 points for a lifetime average blood Pb concentration of up to 10 μg/dL, whereas for levels between 10 to 30 μg/dL a more gradual decrease of only about 2.5 IQ points was estimated. Consequently, the authors concluded that the most important aspect of their findings was that effects associated with BLLs of <10 μg/dL observed in previous cross-sectional studies were confirmed by this rigorous prospective longitudinal investigation).

\(^{304}\) Id. (citing K. N. Dietrich, et al., Early Exposure To Lead And Juvenile Delinquency, 23 NEUROTOXICOLOGY & TERATOLOGY 511-518 (2001)).

\(^{305}\) Id. at 6-71 to 6-73 (citations omitted).
The truth is that little is truly known about children’s neurobehavioral functioning at BLLs below 10 μg/dL. The problem is that there are just too many confounders potentially affecting a child’s IQ or his behavior, including, but not limited to parental intelligence, quality of the home environment, a multitude of social economic factors and exposures to other toxins. Even where children from earlier studies have been followed and assessed, who can truly state as fact that the exposure to lead is the “cause” of their neurological deficiencies or problems.306

ii. Adults

Lead-exposed adults may experience many of the same neurologic symptoms that are experienced by children; however, the thresholds tends to be higher. Lead encephalopathy may occur at extremely high BLLs (e.g., 460 μg/dL), while precursors of encephalopathy (such as dullness, irritability, poor attention span, muscular tremor, loss of memory and hallucination) may occur at lower BLLs.307

Researchers have documented less severe neurologic and behavioral effects in lead-exposed workers with BLLs ranging from 40 to 120 μg/dL. These effects include: malaise, forgetfulness, irritability, lethargy, impaired concentration, depression and mood changes, increased nervousness; headache; fatigue; impotence; decreased libido; dizziness; weakness; and paresthesia, as well as diminished reaction time, visual motor performance, hand dexterity, IQ scores, and cognitive performance. Likewise, there is also some evidence that lead exposure may affect adults’ postural balance and peripheral nerve function. Slowed nerve conduction and

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306 Criteria Document, supra note 44, at 6-73 (“The major challenge to observational studies examining the impact of Pb on parameters of child development has been the assessment and control for confounding factors. By definition, a confounder is associated with both the exposure and the outcome and thusly has the potential to influence the association between the exposure and the outcome.”).

307 Lead Toxicity, supra note 202, at 17 (citation omitted).
forearm extensor weakness (wrist drop), as late signs of lead intoxication, are more classic signs in workers chronically exposed to high lead levels.  

2. Renal (Kidney-Related) Effect

One of the functions of the kidney is to filter substances for elimination and to allow for reabsorption of certain nutrients back into the body. Lead interferes with these functions by altering the metabolism of the kidney. The body can then be affected by the reduction of nutrient reabsorption. The lowest level at which lead has an adverse effect on the kidney remains unknown. The renal effects are most often associated with workers who were subjected to acute high-dose exposures and high-to-moderate chronic exposures (BLLs above 60 µg/dL). Most often, once the exposure is stopped, the kidney can function normally again. Currently, there are no early and sensitive indicators (e.g., biomarkers) considered predictive or indicative of renal damage from lead. However, certain urinary biomarkers show elevations with current exposures, even at BLLs less than 60 µg/dL. Some renal disease or decrement in renal function may be caused by latent effects of lead exposure that occurred years earlier. In children, acute lead-induced renal effects appear reversible, with recovery usually occurring within 2 months of treatment.

3. Other Effects

Other areas where researchers are investing lead-based effects are hematologic, endocrine, cardiovascular (hypertension) and reproductive.

Studies have shown that lead inhibits the body’s ability to make hemoglobin (this is what makes blood cells red) by interfering with several enzymatic steps in the heme pathway. The EPA estimates that the threshold BLL for a decrease in hemoglobin is 50 µg/dL for

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308 Id. (citations omitted).
309 Id. (citations omitted).
occupationally exposed adults and approximately 40 µg/dL for children, although other studies have indicated a lower threshold (e.g., 25 µg/dL) for children. This is what causes anemia among persons with lead poisoning.\textsuperscript{310}

Studies have also shown that lead affects the endocrine system by impeding the body’s ability to digest and use vitamin D. In general, these adverse effects seem to be restricted to children with chronically high BLLs (most significantly in children with BLLs above 62 µg/dL) and chronic nutritional deficiency, especially with regard to calcium, phosphorus and vitamin D.\textsuperscript{311}

Hypertension is a complex condition with many causes and risk factors, including older age, increased weight, poor diet and exercise habits, and excess alcohol intake. Lead exposure is but one factor of many that may contribute to the onset and development of hypertension. Although low-level lead exposures (BLLs below 30 µg/dL) show only a low magnitude of association with hypertension, studies show that greater exposures (primarily occupational) increase the risk for hypertensive heart disease and cerebrovascular disease as latent effects. It is estimated that, on a population mean basis, systolic blood pressure may rise 1-2 mm with each doubling of blood lead, and that blood lead can account for a 1 to 2% variance in blood pressure.\textsuperscript{312}

Finally, some studies in humans suggest that ongoing occupational exposures may decrease sperm count totals and increase abnormal sperm frequencies. Effects may begin at BLLs of 40 µg/dL. Long-term lead exposure (independent of current lead exposure levels) also may diminish sperm concentrations, total sperm counts and total sperm motility. It is unclear

\textsuperscript{310} Id. at 19
\textsuperscript{311} Id. at 19-20.
\textsuperscript{312} Id. at 20-21.
how long reproductive effects may last in humans after lead exposure ceases. With respect to
fertility, to date the results are contradictory and there is at present no body of evidence to
address this question. Whether and how lead exposure may affect female fertility remains an
even more open question. Similarly, increased frequency of miscarriages and stillbirths among
women working in the lead trades was reported as early as the turn of the century. Although the
data concerning exposure levels are incomplete, these effects were probably a result of far
greater exposures than are currently found in lead industries. Thus, there appears to be an
association between higher occupational lead exposure levels and adverse pregnancy outcomes.
This association becomes equivocal when looking at women exposed to lower environmental
levels of lead.  

J. LEAD AND CANCER

All governmental agencies that reviewed the medical literature as it relates to lead and
lead compounds have concluded that while there is evidence that inorganic lead is carcinogenic
to rats and mice, there is not enough evidence to conclude that lead is carcinogenic to humans.
Despite the lack of evidence of human carcinogenicity, agencies such as the EPA conservatively
concluded that human carcinogenicity remains a possibility based on animal studies. Each of the
three main agencies’ (the EPA, NTP and IARC) evaluation of lead is discussed below.

1. The EPA

In its 1986 Lead Air Quality Control Document and its 1990 supplement, the EPA
concluded that there was not enough direct evidence to conclude that lead is a human carcinogen.

313 Id. at 21.
Prot. Agency, Office of Health & Envtl. Assessment, Envtl. Criteria and Assessment Office (1990); see also, Lead and
However, because lead may be carcinogenic to laboratory animals (particularly the rat), it was prudent to consider lead to be a possible human carcinogen.\textsuperscript{316}

According to the EPA, epidemiological studies have provided ambiguous results. While some studies reported that adult occupational exposure to lead increases the risk of lung, kidney, brain, stomach and liver cancer,\textsuperscript{317} the EPA recognized the problem of drawing a strong conclusion from these reports because in addition to being exposed to lead, the study participants were also exposed to other known carcinogens such as arsenic, cadmium and hexavalent chromium. Thus, it is difficult to determine if the excess cancers observed were due to exposure to lead, one of these other carcinogens, or some combination of the various chemicals. Furthermore, other reports indicate that occupational or environmental exposure to lead does not alter cancer risk.\textsuperscript{318}

With respect to children, there have been two studies of children whose fathers worked in lead-related industries such as welding, painting and auto repair. Although these studies reported nonetheless classifying lead as a type B2 carcinogen [probable human carcinogen] based on animal studies [10 rat and 1 mouse] that show statistically significant increases in renal tumors with dietary and subcutaneous exposure to several soluble lead salts that is reproducible in multiple rat strains with some evidence of multiple tumor).

\textsuperscript{316} Criteria Document, \textit{supra} note 44, at 5-125 (noting that animal cell culture studies were considered to be supportive of these observations, but also indicated that lead was not particularly potent).


\textsuperscript{318} \textit{Id.} (citing P. Cocco et al., \textit{Mortality Of Lead Smelter Workers With The Glucose-6-Phosphate Dehydrogenase-Deficient Phenotype}, 5 \textit{CANCER EPIDEMIOLOGY BIOMARKERS & PREVENTION} 223-25 (1996); D. Fanning, \textit{A Mortality Study Of Lead Workers, 1926-1985}, 43 \textit{ARCHIVES ENVT. HEALTH} 247-51 (1988); A. Jemal et al., \textit{The Association Of Blood Lead Level And Cancer Mortality Among Whites In The United States}, 110 \textit{ENVTL. HEALTH PERSPECTIVES} 110: 325-29 (2002)).
elevated childhood tumors (Wilm’s tumor and acute nonlymphocytic leukemia), they suffered
from the same confounding problems encountered in the adult population studies (in addition to
lead, their fathers were also exposed to arsenic, cadmium and hexavalent chromium), making it
difficult to draw any definitive conclusions. Moreover, a report from the printing industry in
Norway found no link between paternal exposure and childhood cancers and, perhaps, even
found a possible reduction in the incidence of childhood cancers with paternal lead exposure.

2. The National Toxicology Program

In 2005, the National Toxicology Program (“NTP”) of the National Institute of
Environmental Health Sciences (“NIEHS”), a part of the Department of Health and Human
Services, voted to include lead and lead compounds as being “reasonably anticipated to be
human carcinogens” based on limited evidence from studies in humans and sufficient

319 Id. (citing J.D. Buckley et al., Occupational Exposures Of Parents Of Children With Acute Nonlymphocytic
Leukemia: A Report From The Children’s Cancer Study Group, 49 CANCER RES. 4030-37 (1989); A.F. Olshan et al., Wilms’
Tumor And Paternal Occupation, 50 CANCER RES. 3212-17 (1990)).

320 Id. (citing P. Kristensen & A. Andersen, A Cohort Study On Cancer Incidence In Offspring Of Male Printing
Workers, 3 EPIDEMIOLOGY 6-10 (1992)).

321 The Public Service Act of 1978 requires NTP to prepare a report listing substances that are either “known” or
“reasonably anticipated” to cause cancer in humans, and to which a significant number of people in the United States are
exposed. NTP is a research agency funded by several regulatory and research agencies, including the EPA, the FDA and the
NIOSH.

322 Substance Profiles: Lead (CAS No. 7439-92-1) and Lead Compounds, Eleventh Report on Carcinogens, U.S.
report, the NTP RoC lead review committee noted that more than 20 published cohort and case-control studies consistently found
that exposure to lead or lead compounds is associated with increased cancer risk, particularly for lung and stomach cancer, but
that the increase in risk was small (a meta-analysis over nine cohort studies reported relative risks of ~1.3 for both sites). Review
Summary of the National Institute of Environmental Health Sciences (NIEHS/NTP) RoC Review Committee (RG1): Lead And
RoC lead review committee later concluded that most studies reviewed had shortcomings including poor exposure assessment,
failure to control for confounders (both co-exposure to other occupational exposure and lifestyle exposures) and an absence of
dose-response relationships. Additionally, the two available meta-analyses have reported an excess risk for lead exposure and
lung cancer but the authors of the latest meta-analysis stated that the lung cancer findings were potentially confounded.
Nonetheless, some committee members believe that a causal relationship between lung cancer and lead exposure has been
established. These committee members rely on a few studies that had a good exposure assessment (using biomarkers) and/or
controlled for confounders that reported an association between lead exposure and lung cancer. The majority of the committee
members, however, felt the epidemiological evidence was limited and that there was no supporting mechanism data to support
the human studies. Review Summary NTP Executive Committee Working Group for the Report on Carcinogens (RG2), Lead And
evidence from studies in experimental animals. While it was not until 2005 that lead and all of its compounds were reviewed and listed by the NTP, the compounds lead phosphate and lead acetate have been listed since the Second Annual Report on Carcinogens as reasonably anticipated to be human carcinogens.

3. The World Health Organization

The World Health Organization’s (“WHO”) International Agency for Research on Cancer (“IARC”) is currently in the process of re-evaluating the potential carcinogenic hazard exposure to lead posed to humans. According to IARC Working Group’s report on its lead evaluation, it has concluded that there is limited evidence for the carcinogenicity to humans of exposure to inorganic lead compounds. Moreover, they concluded that the available epidemiological data provided inadequate evidence for carcinogenicity to humans. Nonetheless, based on animal studies, IARC has concluded that inorganic lead compounds are probably carcinogenic to humans (Group 2A), but that organic lead compounds are not classifiable as to their carcinogenicity to humans (Group 3).

Over the past 30 years, several IARC monograph evaluations of lead compounds have been undertaken. In fact, lead salts was examined in IARC’s first monograph (published in

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323 NTP RoC, supra note 322. The NTP RoC lead review committee stated that carcinogenicity has been observed for insoluble (lead phosphate), soluble (lead acetate and lead subacetate) and organic (tetraethyl lead) lead compounds. It noted that lead has been shown to be carcinogenic in two species of rodents (rats and mice), is effective by multiple routes of exposure (oral, parenteral, transplacental), and has been associated with malignant tumor development in multiple sites (kidney, brain, hematopoietic system). It also stated that lead exposure can also increase the incidence or accelerate the appearance of tumors induced by organic chemical carcinogens in various systems. Id.

324 NTP RoC, supra note 322.


327 Id.
In that monograph, IARC was only able to evaluate animal studies and one epidemiological study of the relationships between exposure to lead and the occurrence of cancer. Based on its review, IARC found no evidence to suggest that exposure to them causes cancer of any site in man but noted that high levels of lead acetate was found to be carcinogenic in rats and mice, and that lead subacetate and lead phosphate are carcinogenic in the rat.\footnote{Vol. 1: Some Inorganic Substances, Chlorinated Hydrocarbons, Aromatic Amines, N-Nitroso Compounds and Natural Products, IARC Monographs on the Evaluation of Carcinogenic Risks to Humans, Int’l Agency for Res. on Cancer, World Health Org. 40 (1972).}

When IARC re-examined lead and its compounds in 1980, there were three epidemiological studies of workers who were exposed to either lead and inorganic lead compounds or to tetraethyl lead and several case control studies investigating the possibility of a causal link between paternal occupation and childhood cancer.\footnote{Vol. 23: Some Metals and Metallic Compounds, IARC Monographs on the Evaluation of Carcinogenic Risks to Humans, Int’l Agency for Res. on Cancer, World Health Org. 325 (1980).} IARC found the experimental and epidemiological data either unavailable or inadequate. Thus, it was unable to conclude that lead was a human carcinogen. But, IARC did find that lead subacetate is carcinogenic to mice and rats and that lead acetate and lead phosphate are carcinogenic to rats. Therefore, they believed that it was reasonable, for practical purposes, to regard lead compounds as if they presented a carcinogenic risk to humans.

IARC supplemented its lead monograph in 1987, looking at updates of the same studies it evaluated in 1980, along with two new studies; one of a U.S. lead smelter and the other of a Swedish copper smelter. Based on its evaluation of the new studies and previous animal studies, IARC concluded that lead and inorganic lead compounds are possibly carcinogenic to humans.

\footnote{IARC discounted the study linking lead-related occupations with the occurrence of Wilms’ tumour because of the “disputable appropriateness of the occupation subcategories used.” Id.}
(Group 2B), but that Organolead compounds are not classifiable as to their carcinogenicity to humans (Group 3).  

IV.

THE OVERLAPPING REGULATION OF LEAD

The dangers of being overexposed to lead have been known since antiquity. \(^{331}\) In the U.S., beginning in the 1970s, Congress reacted to the dangers of lead by creating an overlapping regulatory scheme. Lead is now a highly regulated metal. It is regulated when it is mined, smelted and molded into products. Many lead-containing products are regulated. Even abatement and disposal of lead-containing products is regulated. It is regulated by various environmental laws limiting the amount of lead released into the soil, water and air. It is regulated by occupational health laws that limit the amount of lead workers can be exposed to during a work day. It is regulated by consumer protection laws that limit or ban the use of lead in products used by consumers, including children. There are even laws that require homeowners and lessors to give notice to prospective buyers and renters that a dwelling contains lead paint. Similarly, there are state laws requiring property owners to properly maintain lead-painted walls and/or abate the lead-based paint on walls if it becomes deteriorated. This section discusses some of those regulations.

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\(^{331}\) Even in antiquity, the toxicity of lead was known and those affected were often described as suffering from ‘saturnism’, ‘plumbism’ and ‘colic’. In 370 BC, Hippocrates recognized lead as a cause of the symptoms in a man who worked extracting metals and who suffered a severe attack of colic (stomach upset). During the time of Augustus (63 BC–AD 14), Vitruvius, the architect, wrote, “Water from clay pipes is much more wholesome than that which is conducted through lead pipes, because lead is found to be harmful for the reason that white lead [lead acetate] is derived from it, and this is said to be hurtful to the human system.” Vitruvius also remarked on the pernicious effects of water found near lead mines and its effect on the body. Lead and You, supra note 21. Even Benjamin Franklin described the pernicious effects of lead in tinkers, typesetters and painters. Herbert L. Needleman, Low Level Lead Exposure: A Continuing Problem, 19:3 PEDIATRIC ANNUALS 208, 208 (Mar. 1990).
A. **THE CLEAN AIR ACT – LIMITING AIR EMISSIONS OF LEAD**

By virtually any measure used, the air we breathe in the U.S. today is cleaner than at any time since air quality monitoring started in 1970. This success includes a remarkable reduction in airborne lead emissions.

By 1970, one of the most troubling air pollutants was lead. That, however, quickly changed. From 1982 to 2002 lead air emissions decreased by 93% and the average air quality concentration of lead decreased by 94% from 1983 to 2002. Total lead emissions into the air decreased from about 220,000 tons in 1970 to less than 4,000 in 1999. Major declines over several orders of magnitude have been observed not only in urban areas, but also in rural regions and remote locations.

The principal statutory authorities for controlling air pollution are found in the Clean Air Act. The Clean Air Act of 1970 (the “CAA”) was the first comprehensive, medium-based, federal regulatory program established to regulate air pollution. It provides a comprehensive program for controlling air pollution, using both federal and state regulation. Through the Clean Air Act – Limiting Air Emissions of Lead

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335 42 U.S.C. §§7401-7671q (1995 & Supp. 1999). The structure of the Clean Air Act dates from the 1970 amendments to the previous statute, supplemented by further important amendments in 1977 and 1990. See Pub No. 91-604, 84 Stat. 1676; Clean Air Act Amendments of 1977, Pub. L. No. 95-95, 91 Stat. 685. See generally *General Motors Corp. v. United States*, 110 S. Ct. 2528, 2530 (1990); *Chevron U.S.A. Inc. v. NRDC, Inc.*, 467 U.S. 837, 845-849 (1984); *Train v. NRDC, Inc.*, 421 U.S. 60, 63-67 (1975). In passing the Act, Congress found, among other things, “that the growth in the amount and complexity of air pollution brought about by urbanization, industrial development, and the increasing use of motor vehicles, has resulted in mounting dangers to the public health and welfare, including injury to agricultural crops and livestock, damage to and the deterioration of property, and hazards to air and ground transportation. . . .” 42 U.S.C. §7401(a)(2). Congress declared that the purpose of the Clean Air Act was “to protect and enhance the quality of the Nation’s air resources so as to promote the public health and welfare and the productive capacity of the population. . . .” 42 U.S.C. §7401(b)(1). Air quality remains one of the most important public policy issues in the nation today, some 35 years later, particularly in view of the substantial growth of our urban areas and the significant increase in the number of cars on the road.
Air Act, the government has phased out leaded gasoline and regulates the amount of emissions coming from lead smelters and refiners.

1. The Rise and Fall of Tetraethyl Lead

The greatest impact of lead on human and environmental health was an American contribution: invention and use of alkyllead (tetraethyl lead) in the 1920s as a gasoline antiknock additive. This invention ultimately resulted in the uncontrolled dispersion of millions of tons of lead throughout the world, most intensively in the cities and megacities of the Americas. By the 1970s, more than 250,000 tons of lead per year was being used in the production of tetraethyl lead in the U.S. (380,000 tons per year worldwide). According to the EPA, this accounted for about 90% of all airborne lead. Additionally, lead concentrations in the air over America’s largest cities was 2,000 times greater than lead concentrations in the air over the mid-Pacific.

Although leaded gasoline is no longer around, the millions of tons of lead emitted into the air continues to be a major source of lead in the environment. It is most predominately found in the soil and dust in urban areas and along the country’s highways and byways. Unfortunately, this lead-laden soil and dust does not necessarily stay outside. It is often tracked into homes, schools, apartments and offices where we and our children live and work. Ultimately, the use of tetraethyl lead created almost as many problems as it solved.

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337 CLS Study, supra note 151, at 42.

338 See Ethyl Corp. v. Environmental Protection Agency, 541 F.2d 1, 9, n.7 & 38 (D.C. Cir. 1976) (en banc), cert denied, 96 S.Ct. 2662, 2663 (1976) (citation omitted) (noting that only Ethyl Corporation contested this estimate, but could not claim it was unreasonable).

339 Id. at 38 (listing uncontested facts); see also Environmental Health Criteria 3: Lead, International Programme On Chemical Safety, World Health Organization (1977).
i. The Discovery and Early Warnings About Lead Poisoning

The discovery of the anti-knock effect of tetraethyl lead in gasoline is among the most celebrated achievements of automotive engineering in the 20th century. It is often portrayed as the result of genius, luck and a great deal of hard work. Leaded gasoline allowed an increase in engine power and efficiency by raising fuel anti-knock quality – what is today called the “octane rating.”

The dangers posed by tetraethyl lead were realized almost immediately. First in 1923, when Thomas Midgeley (one of its discoverers) spent a long winter battling lead poisoning. He and three other lab employees had experienced “digestive derangements, subnormal body temperatures and reduced blood pressure” from handling tetraethyl lead. Apparently, they did not realize just how dangerous the substance was in its concentrated liquid state.

Throughout its development in the early 1920s, people were questioning the wisdom of using tetraethyl lead. To some scientists of the day, tetraethyl lead was viewed as a potential public health problem even when the additive was diluted 1000-to-one in gasoline. Thomas

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341 Id. (citing a letter from T. Midgley to Dr. R.L. Allen dated Sept. 9, 1922). Midgley was exposed routinely to tetraethyl lead but had also been caught in at least two laboratory explosions. In July, 1922, when Kettering and Midgley gave a demonstration of tetraethyl lead production to visiting du Pont engineers, the process “got entirely out of hand, and spewed all over the place, and we had to get out,” said Willis F. Harrington of du Pont. On another occasion in 1922, Midgley lost control of the process and fragments of lead embedded in his eyes. According to a note to his doctor, he used mercury as an eyewash to dissolve it. Midgley wrote openly about the problem. He declined speaking offers at three American Chemical Society regional panels in January, 1923 by noting: “After about a year’s work in organic lead I find that my lungs have been affected and that it is necessary to drop all work and get a large supply of fresh air.” Id. (citation omitted).

342 Id. Throughout 1922, Midgley had received alarming letters from four of the world’s leading experts in the field: Wilson of MIT, Reid Hunt of Harvard, Yandell Henderson of Yale and Charles Kraus of Potsdam in Germany. Kraus had worked on tetraethyl lead for many years and called it “a creeping and malicious poison” that had killed a senior scientist at his university. Hunt had informed Henderson about the work at G.M. because the Yale researcher was considered America’s leading expert on automotive exhaust. Id. Another warning came from a lab director in the Public Health Service (P.H.S.) who had heard about tetraethyl lead and wrote an October, 1922 memo to the Assistant Surgeon General warning of a “serious menace to public health.” Several other memos traded hands and in November, Surgeon General Hugh Cumming wrote to Pierre S. du Pont about the public health question. The Surgeon General’s letter was referred to Thomas Midgley, who responded on December
Midgley, however, believed otherwise. While in Miami recovering from lead poisoning, he wrote to an oil industry engineer that poisoning of the public was “almost impossible, as no one will repeatedly get their hands covered in gasoline containing tetraethyl lead - it stings and burns.... The exhaust does not contain enough lead to worry about, but no one knows what legislation might come into existence fostered by competition and fanatical health cranks.”

Nonetheless, enough concerns were raised that the U.S. Public Health Service contacted the U.S. Bureau of Mines to perform a health study of Ethyl gasoline. The Bureau concluded that the danger of the public breathing lead in the exhaust of automobiles was “seemingly remote” based on observations of animals exposed to leaded gasoline exhaust.

ii. The Public Controversy Over the Dangers of Leaded Gasoline

It was not long after manufacturing efforts got under way in 1923 and 1924, that 17 workers involved in making tetraethyl lead died of lead poisoning and 149 other workers were hospitalized. While the lead poisoning deaths of the first workers had not attracted attention, a medical examiner summoned to the second group of work-related deaths called for an investigation into the mysterious gas that was driving workers crazy. The deaths quickly became front page news across the nation. Meanwhile, New Jersey authorities banned leaded

30, 1922 that the problem “has been given very serious consideration... although no actual experimental data has been taken.”  
Id. (citations omitted). Despite his own condition, Midgley was nonchalant about the dangers of tetraethyl lead. In a December 2, 1922 letter to A.W. Browne at Cornell, who had been contracted for some analytical work, Midgley said that tetraethyl lead was irritating to the skin and should not be breathed or taken in the mouth. He added: “It would not surprise me if in the course of using tetraethyl lead for a year that some of your men would experience a slight case of painter’s colic. This is nothing to worry about as several of our boys have it.”  
Id. (citations omitted).

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Id. The actual tests began in the fall of 1923 with a small Delco motor provided by G.M.. Various animals were exposed to Ethyl gasoline exhaust from the motor. One dog exposed to the fumes gave birth to five puppies in the test chamber “without harm of any kind,” Boyd later wrote. The dogs were called the “Ethyl Gas Hounds.”  
Id.

345  
Id. Workers at one of Ethyl’s facilities who were aware of the effects of tetraethyl lead called the factory the “House of Butterflies” for the hallucinations they experienced.  
Id.

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Id. Yandell Henderson of Yale told reporters that the mystery gas was “one of the most dangerous things in the country today,” and was being produced without regard for public health. The New York Times said on October 31, 1924: “In all the history of chemistry, no case like this is recorded. Laboratory workers, of course, have been killed before now, but in each instance the number has been small, and usually they have died while experimenting with known explosives or seeking to find
gasoline; then state legislatures in New York, Pennsylvania and others in New England condemned the new additive and forced gasoline dealers to take it off the market.

This controversy pitted respected public health experts arguing for a permanent nationwide ban on leaded gasoline against the top automotive engineers in the country who believed that the criticism of leaded gasoline was anti-scientific. “As there is no measurable risk to the public in its proper use as a fuel, the chemists see no reason why its manufacture should be abandoned,” said the New York Times. “That is the scientific view of the matter, as opposed to the sentimental, and it seems rather cold-blooded, but it is entirely reasonable.”

On the other hand, public health experts and specialists in lead poisoning found it hard to believe that anyone could reasonably advocate the sure, slow public poison from the use of lead in gasoline.

On May 20, 1925, a well attended public inquiry was held to hear arguments about tetraethyl lead. At the hearing GM and Standard Oil defended tetraethyl lead while academics talked about the seriousness of lead poisoning, beseeching industry to find an alternative anti-knock agent. The Surgeon General ended the conference by announcing that a committee of experts would be appointed to look into the safety of tetraethyl lead.

Unfortunately, the Public Health Service was not in a position to exercise federal authority over an industrial hazard. It had no enabling legislation and there were no regulatory precedents. In the absence of authority, the committee of experts would had to have found striking evidence of serious immediate harm to justify unprecedented action. Instead, in the fall
of 1925, the Public Health Service issued a study that found that drivers and garage workers exposed to leaded gasoline showed some “stippling” damage to red blood cells, but no evidence of outright lead poisoning.\textsuperscript{350} It concluded that there were “no good grounds for prohibiting the use of Ethyl gasoline.” Prophetically, however, the committee cautioned:

It remains possible that, if the use of leaded gasoline becomes widespread, conditions may arise very different from those studied by us which would render its use more of a hazard than would appear to be the case from this investigation. Longer experience may show that even such slight storage of lead as was observed in these [Bureau of Mines] studies may lead eventually to recognizable lead poisoning or to chronic degenerative diseases of a less obvious character. In view of such possibilities the committee feels that the investigation begun under their direction must not be allowed to lapse.... With the experience obtained and the exact methods now available, it should be possible to follow closely the outcome of a more extended use of this fuel and to determine whether or not it may constitute a menace to the health of the general public after prolonged use or under conditions not now foreseen.... The vast increase in the number of automobiles throughout the country makes the study of all such questions a matter of real importance from the standpoint of public health.\textsuperscript{351}

However, the investigation did lapse. Other major events such as the Great Depression and World War II dominated the public consciousness. As America became more dependent on cars, more leaded gasoline was consumed and more lead emissions were being released into the air.

iii. Leaded Gasoline is Accepted

In 1927 the Surgeon General set a voluntary standard of 3 cubic centimeters per gallon (3 cm\textsuperscript{3}/g) for the oil industry to follow in mixing tetraethyl lead with gasoline. At about the same time, industry was making giant strides toward instituting safer working conditions in oil refineries, thereby protecting individual laborers working with or around tetraethyl lead.\textsuperscript{352}

\textsuperscript{350} Id.

\textsuperscript{351} Id. (citing The Use of Tetraethyl Lead Gasoline in its Relation to Public Health, Public Health Bulletin No. 163, U.S. Pub. Health Service, Treasury Dept. (Washington: GPO, 1926)). See also, Lewis, supra note 336.

\textsuperscript{352} See Lewis, supra note 336.
Three decades later, the Surgeon General raised the lead standard to 4 cm$^3$/g (equivalent of 4.23 grams per gallon). This voluntary standard once again represented the outside range of industry practice. Nevertheless, the Surgeon General concluded in 1958 that a loosening of the voluntary standard posed no threat to the health of the average American: “During the past 11 years, during which the greatest expansion of tetraethyl lead has occurred, there has been no sign that the average individual in the U.S. has sustained any measurable increase in the concentration of lead in his blood or in the daily output of lead in his urine.”

The actual industry average during the 1950s and the 1960s hovered in the vicinity of 2.4 grams per total gallon. The Department of Health, Education and Welfare (“HEW”), which was home to the Surgeon General starting with the Kennedy Administration, had authority over lead emissions under the Clean Air Act of 1963. The criteria mandated by this statute were still in the draft stage when the Act was reauthorized in 1970 and a new agency called the Environmental Protection Agency came into existence.

iv. The Regulations Phasing-out Leaded Gasoline

In 1966, Senator Edward Muskie was Chairman of the Senate Subcommittee on Air and Water Pollution. While presiding over hearings on the Clean Air Act he gave considerable attention to the status of lead in the air and in gasoline. At those hearings the Surgeon General testified about evidence of increased lead blood levels, that children and pregnant women might be particularly susceptible to lead injury and that some studies suggested an association between lead exposure and the occurrence of mental retardation among children. In contrast, the representative for the manufacturers of tetraethyl lead told the Senate subcommittee that lead

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353 Id.
354 Lewis, supra note 336.
355 Needleman, supra note 50, at 24.
was a natural element of the earth, that there was no evidence that lead levels in the air had increased since 1937 and that lead was not harmful at lower levels (less than what causes lead poisoning).\footnote{Id.} Dr. Clair Patterson also testified at the hearing providing his perspective.\footnote{See supra section II.B.3.ii.}

Senator Muskie’s aggressive inquiry marked a shift away from complacency towards lead. New questions were raised about the health hazards posed by lead and gone was the notion that lead poisoning was an all-or-nothing phenomenon.\footnote{Needleman, \textit{supra} note 50, at 26.}

In January 1971, the EPA’s first Administrator, William D. Ruckelshaus, declared that “an extensive body of information exists which indicates that the addition of alkyl lead to gasoline ... results in lead particles that pose a threat to public health.”\footnote{Lewis, \textit{supra} note 336 (emphasizing that scientific evidence capable of documenting this conclusion did not exist in previous decades and stating that only recently have scientists been able to prove that low-level lead exposure resulting from automobile emissions is harmful to human health in general, but especially to the health of children and pregnant women).}

On November 28, 1973, the EPA took the position that lead from automobile exhaust was posing a direct threat to public health. Pursuant to CAA requirements, that conclusion left the EPA with no option but to control the use of lead as a fuel additive known to “endanger the public health or welfare.”\footnote{Id. Section 211(c)(1)(A) of the CAA authorizes the EPA to “regulate gasoline additives whose emission products will endanger the public health or welfare. ...” 42 U.S.C. §1857f-(6)(c)(1)(A). \textit{See also Ethyl Corp. v. Environmental Protection Agency}, 541 F.2d 1, 9n.2 (D.C. Cir. 1976) (en banc) (noting that “it is beyond question that the fuel additive Congress had in mind was lead”) (citations omitted).}

One of the reasons cited by the EPA for regulating leaded gasoline was that urban children are particularly threatened by lead additives in that they are prone to ingest lead emissions that have fallen to the ground and mixed with dust.\footnote{Id. at 43 (noting that in most circumstances, lead from exhaust and not lead paint or lead from stationary sources is the primary source of lead in urban dust and dirt).} Compared to the usual range of dust lead concentrations of 2 to 200 parts per million (“ppm”), dust lead concentrations in cities average 1,636 ppm and 2,413 ppm respectively for residential and commercial sites. Even in

\footnotesize{\textit{Ethyl}, 541 F.2d at 43 (noting that in most circumstances, lead from exhaust and not lead paint or lead from stationary sources is the primary source of lead in urban dust and dirt).}
city parks, where young children are expected to play, lead concentrations ranged from 194 ppm to 3,357 ppm. According to the EPA, lead-laden dust is a serious problem for children with “pica.” Pica is a psychological disorder characterized as an excessive habit of eating material (such as dust and dirt) unsuitable as food. Pica afflicts about 50 percent of both middle-and lower-class children. Thus, the lead laden dust eaten by children with pica “can make a significant contribution to ... total lead intake and thereby contribute to the occurrence of lead poisoning, especially in urban areas.” Work done in the 1970s found that children living within 100 feet of heavily traveled roadways had significantly higher blood level than those living beyond that distance. When only children living near highways were considered, the study showed a direct relationship between blood lead levels and traffic density (i.e., the more heavily traveled the roadway, the higher the blood lead level).

The very next month, in December 1973, the EPA issued regulations calling for a gradual reduction in the lead content in all grades of gasoline. The restrictions were scheduled to be implemented starting on January 1, 1975, and to extend over a five-year period. The average lead content of the total gasoline pool of each refinery was to be reduced from the level of approximately 2.0 grams per total gallon that prevailed in 1973 to a maximum of 0.5 grams per total gallon after January 1, 1979. These regulations were challenged by the manufacturers of

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362 Id. at 45 (citing the National Academy of Sciences Committee on Biologic Effects of Atmospheric Pollutants, Airborne Lead in Perspective 30 (1972)) (noting that the lead concentration in New York City’s Central Park zoo was 2,630 ppm and on Philadelphia’s school playgrounds it was 674 ppm). According to the Ethyl Court, The tetraethyl lead manufacturers did not contest the fact that high lead dust levels come from leaded gasoline because their own studies showed that lead content in soil decreases with distance from highways. Id. (citing ter Haar & Aronow, New Information on Lead in Dirt and Dust as Related to the Childhood Lead Problem, presented at the EPA NIEHS Conference on Low Level Lead Toxicity, Raleigh, N.C., (Oct. 1, 1973)).

363 Id. (emphasis in original).

364 Id. at 64 (citing Margulis et al., Residential Location, Ambient air Lead Pollution and Childhood Lead Poisoning, Archives Envtl. Health __).

365 See 40 C.F.R. §80 (1975). See also, Ethyl, 541 F.2d at 7n.2. In Ethyl, the D.C. Circuit addressed a challenge to the EPA’s decision to regulate the lead content of gasoline under section 211(c)(1)(A) of the CAA. See Id. at 6–7.

tetraethyl lead and by representatives of petroleum refiners. Initially, the D.C. Court of Appeals set aside the regulation holding that they were arbitrary and capricious, but they were eventually upheld.\textsuperscript{367} Litigation postponed implementation of this phasedown until 1977.

Additionally, the 1970 Clean Air Act called for the first tailpipe emissions standards. The pollutants controlled are carbon monoxide (CO), volatile organic compounds (VOC), and oxides of nitrogen (NO\textsubscript{x}). The new standards went into effect in 1975 with a NO\textsubscript{x} standard for cars and light-duty trucks of 3.1 grams per mile (gpm).\textsuperscript{368} In order to meet the air pollution emission standards set by Congress in §202 of the CAA, the automobile companies developed the catalytic converter.\textsuperscript{369} Leaded gasoline, however, fouls the system because the lead settles upon and coats the catalysts, making them useless.\textsuperscript{370} In order to allow the catalytic converter to be effectively used on cars, the EPA ordered the marketing of lead-free gasoline pursuant to authority granted by §211(c)(1)(B).\textsuperscript{371} The development of the catalytic converter ushered in the era of unleaded gasoline.

As a result of the required use of catalytic converters on new cars and the phase-down program, by 1988, the total lead usage in gasoline had been reduced to less than 1\% of the amount of lead used in the peak year of 1970.\textsuperscript{372} In 1990, Congress amended the CAA to ban the use of gasoline containing lead or lead additives as fuel in motor vehicles. On February 2, 1996, the EPA incorporated the statutory ban in a direct final rule which defined unleaded gasoline as

\textsuperscript{367} Ethyl, 541 F.2d 1.
\textsuperscript{369} Section 202(b)(1)(A) & (B).
\textsuperscript{371} Section 211(c)(1)(B) gives EPA authority to regulate gasoline additives whose emissions products “will impair to a significant degree the performance of any emission control devise or system which is in general use. . . .” 42 U.S.C. §1857f-6c(1)(B).

gasoline containing trace amounts of lead up to 0.05 gram per gallon. Although Congress banned the use of leaded gasoline as fuel in motor vehicles, it did not restrict other potential uses of gasoline containing lead or lead additives. As a result, gasoline produced with lead additives continues to be made and marketed for use as fuels in aircraft, race cars, and non-road engines such as farm equipment engines and marine engines. However, tetraethyl lead has not been produced in the U.S. since March 1991. Moreover, since January 1, 1996, all gasoline sold for motor vehicles has been unleaded.

2. National Ambient Air Quality Standards

The CAA also required the creation of national ambient air quality standards for six air pollutants. Known as “criteria pollutants,” these six pollutants are: CO, lead, ozone, sulfur dioxide, nitrogen dioxide, and particulate matter. Air quality control regions throughout the country are rated based on whether they meet national air quality standards for each of these designated pollutants. Regions that do not meet the standards for any of the criteria pollutants are designated as “non-attainment areas.” As of March 2, 2006, only two counties in the U.S. (one in Missouri and one in Montana) with a combined population of 4,000 persons are deemed lead non-attainment areas. Lead industrial facilities are located in each of these two counties.

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373 Id. The definition still allowed trace amounts of lead but expressly prohibited the use of any lead additive in the production of unleaded gasoline. The term “lead additive” was defined to include pure lead as well as lead compounds. Id.

374 Id.

375 40 C.F.R. §80.22 (banning the use of lead additives in motor fuels after December 31, 1995).

376 42 U.S.C. §7409 (1995); 40 C.F.R. §§50.2(a) and 50.4-50.12 (1999). Under Section 108(a) of the Act, the EPA issues air quality criteria documents reviewing scientific evidence about the possible effects of certain listed air pollutants that endanger the public health or welfare. 42 U.S.C. 7408(a). Pursuant to Section 109(b) of the Act, the EPA must, for each such pollutant, promulgate “primary” national ambient air quality standards (NAAQS) “to protect the public health” and “secondary” NAAQS “to protect the public welfare from any known or anticipated adverse effects associated with the presence of such air pollutant in the ambient air.” 42 U.S.C. 7409(b).

377 40 C.F.R. §§51.852 and 50.4-50.12.

378 42 U.S.C. §§7407(d) and 7501(2).

379 EPA Lead Nonattainment Area Summary (Jul. 31, 2006), available at http://www.epa.gov/oar/oaaqs/greenbk/lnsum.html (last visited July 31, 2006). The two counties classified as non-attainment areas are (1) the East Helena Area (Lewis and Clark Co.) in Montana and (2) within the city limits of Herculaneum (Jefferson
Pursuant to 42 U.S.C. §7409, the United States’ Environmental Protection Agency (the “EPA”) has the responsibility of promulgating regulations relating to air quality standards. The national ambient air quality standards (“NAAQS”) are to be “based on such criteria and allowing an adequate margin of safety” to such an extent as is necessary “to protect the public health.” The air quality criteria are supposed to “accurately reflect the latest scientific knowledge useful in monitoring the kind and extent of all identifiable effects on public health or welfare which can be expected from the presence of such air pollutant in the ambient air.”

As to lead, Congress spoke as if the CAA required quick action. But, instead of treating lead as a criteria air pollutant, the EPA decided to regulate the lead content of the leaded gasoline used by pre-1975 vehicles. The Natural Resources Defense Council (“NRDC”) sued the EPA to force it to treat lead as a criteria air pollutant claiming that since the EPA had previously acknowledged that lead posed a danger to human health and welfare that this classification had earned lead a spot on the NAAQS’s list. The circuit court agreed with the NRDC and the Second Circuit affirmed.

In 1978, the EPA promulgated NAAQS for lead. That standard is 1.5 µg/m³. Shortly thereafter, the standards were unsuccessfully challenged in court. In 1986, the EPA revised

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384 See Natural Resources Defense Council v. Train, 545 F.2d 320 (2d Cir. 1976). The EPA took the position that whether it lists a pollutant, and thus the extent to which it protects health from that pollutant, is within its discretion. The Second Circuit rejected this argument, in part on the basis that “the deliberate inclusion of a specific timetable for the attainment of ambient air quality standards incorporated by Congress in §§108-110 would become an exercise in futility if the Administrator could avoid listing pollutants.” Id. at 327.
385 “National primary and secondary ambient air quality standards for lead and its compounds, measured as elemental lead by a reference method based on Appendix G to this part, or by an equivalent method, are: 1.5 micrograms per cubic meter,
the Air Quality Criteria Document ("CD") for lead. The CD was supplemented by the EPA in 1990. Also in 1990, the EPA’s Office Air Quality and Planning Standards issued a staff paper; however, the EPA did not publish a revised NAAQS for lead and the standard has remained the same.

On May 27, 2004, a suit was filed in Missouri seeking a declaratory judgment that the Administrator of the EPA was in violation of the requirement that the EPA review, and if necessary, revise the NAAQS for lead every five years. The plaintiffs requested that the court issue a mandatory injunction ordering defendant to review, and if appropriate, revise the NAAQS for lead. The EPA admitted that it had not performed the mandatory 5-year review for the lead NAAQS and on September 14, 2005, the court ordered the EPA to:

- complete the initial draft of the Criteria Document no later than December 1, 2005;
- finalize the Criteria Document no later than October 1, 2006;
- prepare the initial draft of the Staff Paper no later than January 1, 2007;
- finalize the Staff Paper no later than November 1, 2007;
- sign a notice of proposed rulemaking no later than May 1, 2008 for publication in the Federal Register;
- provide an 60-day open public comment period;
- sign the notice of final rulemaking concerning any revisions to the NAAQS on or before September 1, 2008 for publication in the Federal Register;


The EPA is currently in the process of performing a multimedia risk assessment for lead.

The success of previous efforts to reduce air emissions of lead is one of the main problems confronting the EPA in deciding what to do about the lead NAAQS. While the current NAAQS standard is 1.5 µg/m³, the current levels of lead in the air is less than 0.1 µg/m³ as shown in the following graph.³⁸⁹

On July 12, 2006, the Battery Council International (“BCI”) filed an unprecedented request for the EPA to delete lead as one of the six key national air pollutants regulated under the Clean Air Act’s health-based standards.³⁹⁰ BCI made two arguments for deleting lead as a criteria pollutant. First, as discussed above, lead emissions have been cut so much that the metal no longer needs to be regulated stringently. Secondly, the strict rules governing lead emissions


from battery makers and secondary smelters – two of the few remaining sources of lead air
emissions – are stringent enough that the EPA no longer considers the plants major contributors
to lead pollution. Activists responding to the request call it a remarkably irresponsible move
because lead is widely recognized as a health hazard, especially to children.

3. Control of Smelter Emissions

Primary lead smelting is the process of recovering lead from lead-bearing ore. Lead
smelting involves three processes – sintering, reduction and refining – each with its own
characteristic emissions. Primary lead smelting in the U.S. emitted was about 565 metric tons of
lead in 2000 (about 14.7% of total lead emissions in the U.S. that year), but only about 58.9
metric tons in 2002 (about 4% of total lead emissions in the U.S. that year). Secondary lead
smelters reclaim scrap lead. Both the principal input to and the principal major product market
of secondary smelters are lead-acid batteries. Secondary lead production in the U.S. emitted
about 422 metric tons of lead in 2000 (about 11.0% of total lead emissions in the U.S. that year),
but only about 4.3 metric tons of lead in 2002 (less then 1.0% of total lead emissions in the
U.S. that year). Although recycling of lead-acid batteries with minimal emissions may be
possible, the EPA still considers secondary smelters and battery recycling facilities are still one
of the most significant stationary sources of airborne lead emissions.

391 Id. BCI claims that the dramatic decrease in air concentrations of lead since the phase-out of the metal from
gasoline begun in the late 1970s justifies its removal from the criteria pollutants. They claim that continued inclusion of lead as a
criteria pollutant is no longer consistent with Section 108 of the Clean Air Act, which governs the criteria air pollutants regulated
by EPA.

392 Id.


395 See supra note 393.

396 Id. at 2-20 to 2-21.
To address emissions of lead components and other toxic pollutants from both primary lead smelters and secondary lead smelters, the EPA has also promulgated regulations under section 112(d) of the CAA. Consequently, the smelters are required to obtain “the maximum degree of reduction in emissions” that are “achievable.” Thus, the EPA promulgated section 112(d) standards for secondary lead smelters on June 23, 1995, and revised them on June 13, 1997, followed by promulgation of section 112(d) standards for primary lead smelters on June 4, 1999. The final rule establishes a “plant wide” emission limit of 500 grams of lead per megagram of lead produced from the aggregation of emissions discharged from eight identified sources that emit hazardous air pollutants. It also requires primary smelters to develop and operate according to a Standard Operating Procedure (SOP) Manual for the control of fugitive dust sources and for the operation and maintenance of control devices. Each smelter’s SOP Manual will be reviewed and subject to approval by the EPA Administrator or designated authority.

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397 See 42 U.S.C. §7412); 40 CFR Subparts X and TTT. Under the CAA Amendments of 1990, the EPA is required to regulate sources listed toxic air pollutants. On July 16, 1992, the EPA published a list of industry groups (known as source categories) that emit one or more of these air toxins. For listed categories of major sources, the CAA requires EPA to develop standards that require the application of stringent air pollution reduction measures known as maximum achievable control technology (MACT). The EPA’s list of source categories to be regulated includes primary lead smelting. The EPA considered and addressed all public comments in formulating this final rule.


400 See 64 Fed. Reg. 30,194 (June 4, 1999).

401 Id.; see also EPA Fact Sheet: Final Air Toxics Rule For Primary Lead Smelters (July 1, 1999) available at http://www.epa.gov/ttncaaa1/t3/fact_sheets/prleadfs.pdf (last visited July 28, 2006).
B. OCCUPATIONAL SAFETY AND HEALTH ACT – PROTECTING WORKERS FROM LEAD EXPOSURES

1. The Lead Standard for General Industry

The Occupational Safety & Health Administration ("OSHA") is the federal agency charged with overseeing the safety and health concerns of American workers. The agency has developed specific workplace standards and policies businesses to make sure these standards are being met. In fulfillment of its duties, OSHA issued its Lead permanent occupational safety and health standard on November 21, 1978. The standard applies to employment in all industries except construction and agriculture. With respect to lead:

- Employers must assure that no employee is exposed to lead at concentrations greater than fifty micrograms per cubic meter of air (50 µg/m³) averaged over an 8-hour period.

- Where any employee is exposed to lead above the permissible exposure limit for more than 30 days per year, the employer shall implement engineering and work practice controls (including administrative controls) to reduce and maintain employee exposure to lead... Wherever the engineering and work practice controls which can be instituted are not sufficient to reduce employee exposure to or below the permissible exposure limit, the employer shall nonetheless use them to reduce exposures to the lowest feasible level and shall supplement them by the use of respiratory protection.

- Where any employee is exposed to lead above the permissible exposure limit, but for 30 days or less per year, the employer shall implement engineering controls to reduce exposures to 200 µg/m³, but thereafter may implement any combination of engineering, work practice (including administrative controls), and respiratory

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402 OSHA was created to oversee and implement the Occupational Safety & Health Act of 1970, Pub L. 91-596, 84 STAT. 1590 (Dec. 29, 1970) [hereinafter OSH Act]. The primary purpose of the OSH Act is to assure, so far as possible, safe and healthful working conditions for every working man and woman.

403 See OSHA §6, 29 USC §655. “... the Secretary shall, as soon as practicable ..., by rule promulgate as an occupational safety or health standard any national consensus standard, and any established Federal standard, unless he determines that the promulgation of such a standard would not result in improved safety or health for specifically designated employees. In the event of conflict among any such standards, the Secretary shall promulgate the standard which assures the greatest protection of the safety or health of the affected employees.” Id.

404 43 FED. REG. 52952 (Nov. 14, 1978).

405 29 C.F.R. §1910.1025(c)(1).

controls to reduce and maintain employee exposure to lead to or below 50 µg/m$^3$.\textsuperscript{407}

The lead standard was challenged by both industry and labor, with all cases transferred to the U.S. Court of Appeals for the District of Columbia. On August 15, 1980, the Court of Appeals affirmed most aspects of the regulation covering worker exposure to airborne lead.\textsuperscript{408} The court also upheld OSHA’s findings of feasibility for 10 industries: primary lead production, secondary lead production, can manufacturing, lead acid battery manufacturing, paints and coatings manufacturing, ink manufacturing, wallpaper manufacturing, electronics, printing, and grey-iron foundries. However, the court further found that OSHA had failed to present adequate evidence of feasibility for 38 lead industries.\textsuperscript{409} In December 1981, after re-categorizing and adding other industries to the list, OSHA asked the court to remand the record for further consideration as to nine industries: brass and bronze ingot manufacturing/production, collection and processing of scrap (including independent battery breaking), lead chemicals, lead chromate pigments, leaded steel, nonferrous foundries, secondary copper smelting, shipbuilding and ship repairing, and stevedoring.\textsuperscript{410} OSHA requested that the record for these nine be remanded again to the Agency for further consideration of economic and technological feasibility. In March 1987, the court remanded the record to OSHA for these industries.

\textsuperscript{407} 29 C.F.R. §1910.1025(e)(1)(ii).
\textsuperscript{409} Id. The Court remanded the record to OSHA for reconsideration of the technological and economic feasibility of paragraph (e)(1) and stayed enforcement of paragraph (e)(1) for those industries. Nonetheless, the Court held that the 38 industries were required to meet the PEL by a combination of engineering controls, work practices, and respiratory protection.
\textsuperscript{410} 46 FED. REG. 60758 (Dec. 11, 1981). In accordance with a remand order of the Court of Appeals for the District of Columbia in United Steelworkers v. Marshall, OSHA conducted additional rulemaking proceedings to determine whether the requirement of §1910.1025(e)(1) that employers implement engineering or work practice controls to meet the PEL of 50µg/m$^3$, was feasible for certain industries, including nonferrous foundries. OSHA subsequently reaffirmed its original findings that compliance with this exposure level was feasible and issued a supplemental statement of reasons in support of that conclusion. 54 FED. REG. 29,142 (1989).
On March 8, 1990, in response to OSHA’s statement of reasons regarding the feasibility of paragraph (e)(1), the U.S. Court of Appeals for the D.C. Circuit lifted the judicial stay for all remand industries (39 of them) except the six that contested OSHA’s feasibility findings. These industries were given two and one-half years from the date the stay was lifted, until September 8, 1992, to comply with the permissible exposure limits (“PEL”) by means of engineering and work practice controls. On July 19, 1991, the D.C. Court of Appeals affirmed OSHA’s findings of technological and economic feasibility for all industries except the finding of economic feasibility for brass and bronze ingot manufacturing. Accordingly, the court lifted the judicial stay for the other five industries. On June 27, 1995, OSHA and the final industry segment reached a settlement agreement. Since then OSHA has been regulating worker exposure to lead in all industry segments in the U.S.

2. The Lead Standard for Construction Workers

While the general industry lead standard does not apply to construction workers, they are not without a standard. Beginning on May 4, 1993, the exposure of construction workers to lead in the construction industry will be controlled by a standard similar to that which applies in the general industry. This standard will be based on the same permissible exposure limit (PEL) of 50 µg/m³ established for the general industry. The standard will also include engineering and work practice controls to reduce exposure to lead, such as ventilation and personal protective equipment. In addition, the standard will require training and medical surveillance for employees who are exposed to lead.

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411 See 95 FED. REG. 25067 (Oct. 10-1995). The 39 industries for which the stay was lifted are: agricultural pesticides; aluminum smelting; ammunition manufacturing; artificial pearl processing; book binding; brick manufacturing; cable coating; cutlery; diamond processing; electroplating; explosives manufacturing; gasoline additive manufacturing; glass manufacturing; jewelry manufacturing; lamp manufacturing; lead burning; lead chromate pigments; leather manufacturing; machining; miscellaneous lead products; nickel smelting; pipe galvanizing; plastics and rubber manufacturing; plumbing; pottery and ceramics; primary and secondary smelting of gold, silver, and platinum; primary copper smelting; sheet metal manufacturing; shipbuilding and ship repair; solder manufacturing; soldering; spray painting; steel manufacturing (excluding leaded steel manufacturing); stevedoring; terne metal; textiles; telecommunications; tin rolling and plating; and zinc smelting. Id.

412 These industries are: nonferrous foundries; secondary copper smelting; brass and bronze ingot manufacturing; collection and processing of scrap (including independent battery breaking); leaded steel manufacturing; and lead chemicals manufacturing. Id.

413 Id.

414 American Iron & Steel Inst. v. OSHA, 939 F.2d 975, 980 (D.C. Cir. 1991) (holding that “[a] standard is economically feasible if the costs it imposes do not ‘threaten massive dislocation to, or imperil the existence of, the industry.’”) [quoting United Steelworkers of Am. v. OSHA, 647 F.2d 1189, 1265 (D.C. Cir. 1980)]. Secondary copper smelters, lead chemical manufacturing, and large nonferrous foundries were allowed five years from July 19, 1991, the date of the Court’s decision, to implement engineering and work practice controls to achieve the PEL of 50 µg/m³. Small nonferrous foundries were allowed five years from that date to achieve an airborne lead concentration of 75 µg/m³. 95 FED. REG. 25067 (Oct. 10-1995).

415 95 FED. REG. 25067 (Oct. 10-1995).

416 Because Congress did not anticipate publication of OSHA’s proposed comprehensive lead standard for the construction industry before late spring of 1993 or publication of a final standard before 1996 (House Report on H.R. 5730, pp.
lead is regulated by a lead standard appropriately titled: Safety and Health Regulations for Construction - Lead. This standard applies to all construction work excluded from coverage in the general industry standard for lead.

Construction work is defined as work involving construction, alteration and/or repair, including painting and decorating. Construction work covered under the Lead in Construction Standard includes any repair or renovation activities. Specifically, construction includes but is not limited to the following:

- demolition or salvage of structures where lead or materials containing lead are present;
- construction activities that involve removal or encapsulation of materials containing lead;
- new construction, alteration, repair, or renovation of structures, or significant portions thereof, that contain lead or materials containing lead (e.g., steel structure renovation and repair);
- installation during construction of products containing lead;

14-15), Congress in October 1992, passed Sections 1031 and 1032 of Title X of the Housing and Community Development Act of 1992 (“the Act,” Pub. L. 102-550, signed by the President on October 28, 1992, 106 Stat. 3924). In those sections, Congress included worker protection provisions expressly requiring that: (1) no later than 180 days after enactment (April 26, 1993), the Secretary of Labor must issue an interim final lead standard covering the construction industry; (2) the standard must be as protective as the worker protection guidelines for identification and abatement of lead-based paint in public and Indian housing issued by the Department of Housing and Urban Development (Revised Chapter 8, “HUD Guidelines,” 55 FED. REG. 38973 [Aug. 1991]; (3) the interim final standard is to take effect upon “issuance,” except that the standard may include a reasonable delay in the effective date; (4) the standard will have the effect on the OSHA standard and will apply until a final standard becomes effective under Section 6 of the OSHA; (5) the Secretary of Labor in developing this standard must consult and coordinate with the Environmental Protection Agency (EPA) to achieve maximum enforcement of the Toxic Substances Control Act (TSCA) and OSHA while minimizing duplication. See generally 58 FED. REG. 26590 (May 4, 1993); infra section IV.C.2.iv.

58 FED. REG. 26590 (May 4, 1993) (promulgated at 29 C.F.R. §1926.62). The 1978 general industry lead standard excluded the construction industry from its coverage. OSHA in the preamble explained that it had exempted the industry because of insufficient information in the record to resolve issues raised about the applicability of the standard to conditions in the construction industry. Id. Subsequently, OSHA's exemption of the construction industry was challenged in litigation involving the lead standard for general industry. In response to that challenge, the court upheld OSHA's decision. Although the court declared that “OSHA would be shirking its statutory responsibilities if it made no effort to protect workers in the construction industry from lead exposure...,” the court accepted OSHA's assurances at the time “that it will take reasonably prompt steps to fashion this protection,” and indicated that “[s]o long as it does so, OSHA has met its duty.” “Nothing in the Act,” the court said, “prevents the agency from exercising discretion in delaying specific standards according to the unique problems of specific industries....” United Steelworkers of America v. Marshall, 647 F.2d 1189, 1310 (D.C. Cir. 1980).

See supra section IV.B.1.

See 29 CFR §1910.12(b).
• lead contamination/emergency cleanup during construction;
• transportation, disposal, storage, or containment of lead or materials containing lead on the site or location at which construction activities are performed; and
• maintenance operations associated with the construction activities described in items 1 through 6.  420

These activities do not include routine cleaning and repainting (for example, minor surface preparation and repainting of rental apartments between tenants or at scheduled intervals) where there is insignificant damage, wear, or corrosion of existing lead-containing paint and coating or substrates. According to OSHA, all construction work excluded from coverage in the general industry standard (§1910.1025(a)(2)) is covered by this interim final rule. Thus, there should be no gaps and no overlaps between the two standards. 421

Like the general industry standard, employers must assure that no employee is exposed to lead at concentrations greater than 50 micrograms per cubic meter of air (50 µg/m$^3$) averaged over an 8-hour period. 422 Moreover, each employer must initially assess the worksite to determine if any employee may be exposed to lead at or above the action level. 423 Until the employee has made the initial assessment, he is to assume the presence of lead and implement measures to protect his employees. 424

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420 29 C.F.R. §1926.62(a).
421 58 FED. REG. 26590 (May 4, 1993).
422 29 C.F.R. §1926.62(c)(1).
423 29 C.F.R. §1926.62(d)(1)(i).
424 29 C.F.R. §1926.62(d)(2). Some specific tasks covered by this requirement are: manual demolition of structures (e.g., dry wall), manual scraping, manual sanding, heat gun applications, and power tool cleaning with dust collection systems, spray painting with lead paint, using lead containing mortar, lead burning, abrasive blasting, welding, cutting and torch burning. Id.
C. EPA AND HUD EFFORTS TO REGULATE HOUSING PAINTED WITH LEAD-BASED PAINT

1. Lead-Based Paint Poisoning Prevention Act of 1971

   i. Eliminating the Use of Lead-Based Paint from Federal-Assisted Housing and Cookware

   In 1971, Congress passed the Lead-Based Paint Poisoning Prevention Act (the “LPPPA”). Through the LPPPA, Congress banned the use of lead-based paint in residential structures constructed or rehabilitated by the federal government. The LPPPA also prohibited the application of lead-based paint to any cooking utensil, drinking utensil, or eating utensil manufactured and distributed after January 13, 1971. Additionally, the Consumer Product Safety Commission was required to impose conditions to prohibit the application of lead-based paint to toys and furniture. It also contained grant provisions for states to “detect and treat incidents of lead-based paint poisoning” and “to develop and carry out programs to eliminate the hazards of lead-based paint poisoning.” In addition, the Secretary of Housing and Urban Development (HUD) was required to investigate the “nature and extent of the problem of lead-based paint in the United States.”

   In 1973, Congress amended the LPPPA to require, inter alia, the Secretary of HUD to “implement procedures to eliminate the hazards of lead-based paint poisoning in all federally owned properties prior to the sale of such properties when their use is intended for residential

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426 42 U.S.C. §4831(b). Lead-based paint was defined as paint containing 1% lead by weight in the non-volatile content of the paint or in the dried film of the paint. Pub. L. No. 91-695, §501(3), 84 Stat. 2078, 2080 (1971). The definition of lead-based paint is currently paint or other surface coatings that contain lead in excess of 1.0 milligrams per centimeter squared or 0.5 percent by weight or (A) in the case of paint or other surface coatings on target housing, such lower level as may be established by the Secretary of Housing and Urban Development, as defined in section 4822(c) of title 42, or (B) in the case of any other paint or surface coatings, such other level as may be established by the Administrator. 42 U.S.C. §4851b(14) (1994); 15 U.S.C. §2681(9) (1994)); 15 U.S.C. §2681(9) (Toxic Substances Control Act Section 401(9)) (2006).
428 42 U.S.C. §4831(c).
habitation.” The 1973 Amendments also required the Secretary of HUD “to eliminate as far as practicable the hazards of lead-based paint poisoning with respect to any existing housing which may present such hazards and which is covered by an application for mortgage insurance or housing assistance payments under a program administered by the Secretary.” These amendments demonstrate Congress’ intent to eliminate (as far as practicable) lead-based paint hazards in housing covered by mortgage insurance and in housing receiving assistance payments, and its intent to eliminate lead-based paint hazards in housing owned by federal agencies when it was sold. Congress’ practice of imposing different lead-based paint requirements on federally owned housing and on federally assisted housing began in 1973 and continues to the present.

Congress again amended the LPPPA in 1988, requiring the inspection of all intact and non-intact interior and exterior painted surfaces of applicable housing for lead-based paint using an approved x-ray fluorescence analyzer or comparable approved sampling or testing technique.

The amendment also required the results of the inspection be “provided to any potential purchaser or tenant of the housing.”

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430 Id.

431 Zimmerman, supra note 429 at 175-76 & n.58. In 1988, Congress directed HUD to provide tenants and purchasers of federally assisted housing with a brochure describing the hazards associated with lead-based paint. Tenants of federally owned housing were not required to receive the same brochure. See Housing and Community Development Act of 1987, Pub. L. No. 100-242, §566, 101 Stat. 1815, 1945 (1988). In 1992, under Title X, Congress required that lead-based paint risk assessments be performed on federally assisted housing. 42 U.S.C. §4822(a) (1996). In addition, Congress required interim lead-based paint controls be implemented at federally assisted housing. Id. Interim controls are “a set of measures designed to reduce temporarily human exposure or likely exposure to lead-based paint hazards.” 42 U.S.C. §4851b(13) (1996). Congress has not required risk assessments or interim controls for all federally owned housing.

432 42 U.S.C. §4822(c). Zimmerman, supra note 429 at 176. Zimmerman notes the existence of confusion because §4822(c) applies to “housing subject to this section” and §4822 establishes requirements for both federally assisted housing (e.g., eliminate as far as practicable lead-based paint hazards in federally assisted housing) and federally owned housing (i.e., abate lead-based paint hazards in federally owned housing prior to sale). Arguably, since both federally assisted and federally owned housing are subject to §4822 requirements, both types of housing are subject to the inspection and disclosure requirements of §4822(c). Id.
2. Residential Lead-Based Paint Hazard Reduction Act of 1992

When Congress passed the Residential Lead-Based Paint Hazard Reduction Act,\(^{433}\) it concluded that “despite the enactment of laws in the early 1970s requiring the Federal Government to eliminate as far as practicable lead-based paint hazards in federally owned, assisted, and insured housing, the Federal response to this national crisis remains severely limited.”\(^{434}\) Congress also stated that “the Federal Government must take a leadership role in building the infrastructure – including an informed public, State and local delivery systems, certified inspectors, contractors, and laboratories, trained workers, and available financing and insurance – necessary to ensure that the national goal of eliminating lead-based paint hazards in housing can be achieved as expeditiously as possible.”\(^{435}\)

i. Amending the Toxic Substances Control Act to Define Lead Hazards, Set Remediation Contractor Training and Accreditation Requirements, and Establish Testing Protocols and Certifications for Laboratories

Subtitle B of Title X of this Act, entitled the “Lead Exposure Reduction Act,” added a new Title IV to the Toxic Substances Control Act (“TSCA”).\(^{436}\) It applies to (1) federal agencies that own residential property, (2) property owners that receive assistance through federal housing programs (e.g., state and city public housing authorities, owners of multi-family rental properties that receive project-based assistance, owners of rental properties that lease units under HUD’s tenant-based assistance program), and (3) property owners (e.g., owner occupants, rental property owners, public housing authorities, and Federal agencies) who, if they know about the

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\(^{434}\) Id. at §1002(7); 42 U.S.C. §4851(7) (Congressional findings).

\(^{435}\) Id. at §1002(8); 42 U.S.C. §4851(8).

\(^{436}\) Id. at §1021; amending 15 U.S.C. §§2601-2692 (subchapter IV – Lead Exposure Reduction).
identified hazards would have to disclose them pursuant to EPA/HUD joint regulations promulgated under Title X, §1018.  

Title IV was written to accelerate federal efforts to reduce risks to young children who daily are exposed to lead-based paint in their homes. In addition, it is expected to stimulate development of lead inspection and hazard abatement services in the private sector, while ensuring that the services provided and any products employed are reliable and effective in reducing risk. To these ends, Title IV directs the EPA to

- promulgate definitions of lead-contaminated dust, lead-contaminated soil and lead-based paint hazards;
- ensure that people engaged in detection and control of lead hazards are properly trained and that contractors are certified;
- publish requirements for the accreditation of training programs for workers;
- develop criteria to evaluate the effectiveness of commercial products used to detect or reduce risks associated with lead-based paint;
- establish protocols, criteria, and minimum performance standards for laboratory analysis of lead in paint films, soil and dust;
- establish a program to certify laboratories as qualified to test substances for lead content; and
- publish and distribute to the public a list of certified or accredited environmental sampling laboratories.

Title IV explicitly applies these requirements to federal facilities and activities that may create a lead hazard. Moreover, Title X was shifting the focus of public and private sector decision makers from the mere presence of lead-based paint to the presence of lead hazards.

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439 Id.
The Act also mandated a study by the Centers for Disease Prevention and Control ("CDC") and the National Institute for Environmental Health Sciences to determine the sources of lead exposure to children who have elevated lead levels in their bodies. The National Institute for Occupational Safety and Health is directed to study ways of reducing occupational exposure to lead during abatement activities.440

ii. EPA’s Lead Rule and its Omnibus Definition of a Lead-Based Paint Hazard

Title X directed the EPA to conduct a study of lead hazards due to renovation and remodeling activities that may incidentally disturb lead-based paint. It also requires the EPA to promulgate guidelines for the renovation and remodeling of buildings or other structures when these activities might create a hazard.441 Moreover, when Congress defined the term “lead-based paint hazard” in this Act, it defined it much more broadly than just paint that contains lead. According to Congress, a “lead-based paint hazard” is any condition that causes an exposure to lead-containing paint, dust or soil (regardless of the source) which, due to their condition and location, would adversely affect human health.442

On January 5, 2001, the EPA issued its “Lead Rule.”443 As could be expected, the EPA followed Congress’ lead and defined the term “lead-based paint hazard” as being “lead-based paint and all residential lead-containing dusts and soils regardless of the source of the lead,

440 Schierow, supra note 438.
441 Schierow, supra note 438.
442 15 U.S.C. §§2681(10). “The term ‘lead-based paint hazard’ means any condition that causes exposure to lead from lead-contaminated dust, lead-contaminated soil, lead-contaminated paint that is deteriorated or present in accessible surfaces, friction surfaces, or impact surfaces that would result in adverse human health effects as established by the Administrator under this subchapter.” Id.
443 Lead; Identification of Dangerous Levels of Lead, 66 Fed. Reg. 1206 (Jan. 5, 2001) (final rule). In addition to establishing lead hazard levels, this final rule amended dust and soil sampling requirements and amended state program authorization requirements. Id. The EPA promulgated these regulations in accordance with 15 U.S.C. §2683; TSCA §403 (which were added to TSCA via Title X).
which, due to their condition and location, would result in adverse human health effects.”

Shortly thereafter, three trade associations representing the multi-family rental housing industry, challenged the Lead Rule’s “regardless of source interpretation” as being contrary to Congress’ intent in enacting Title X and as being arbitrary and capricious. The court noted the “regardless of source” language in the statutory definition of lead-based paint hazard and agreed that the EPA’s broad definition was not prohibited. The EPA then quantified when a hazard exists from (1) paint-lead, (2) dust-lead and (3) soil-lead. The EPA defined these terms as follows:

(a) Paint-lead hazard. A paint-lead hazard is any of the following:

(1) Any lead-based paint on a friction surface that is subject to abrasion and where the lead dust levels on the nearest horizontal surface underneath the friction surface (e.g., the window sill or floor) are equal to or greater than the dust-lead hazard levels identified in paragraph (b) of this section.

(2) Any damaged or otherwise deteriorated lead-based paint on an impact surface that is caused by impact from a related building component (such as a door knob that knocks into a wall or a door that knocks against its door frame).

(3) Any chewable lead-based painted surface on which there is evidence of teeth marks.

(4) Any other deteriorated lead-based paint in any residential building or child-occupied facility or on the exterior of any residential building or child-occupied facility.

(b) Dust-lead hazard. A dust-lead hazard is surface dust in a residential dwelling or child-occupied facility that contains a mass-per-area concentration of lead equal to or exceeding 40 mg/ft² on floors or 250 mg/ft² on interior window sills based on wipe samples.

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446 Id.
447 Id. (codified at 40 C.F.R. §745.65(a)).
448 Id. (codified at 40 C.F.R. §745.65(b)).
(c) Soil-lead hazard. A soil-lead hazard is bare soil on residential real property or on the property of a child-occupied facility that contains total lead equal to or exceeding 400 parts per million (mg/g) in a play area or average of 1,200 parts per million of bare soil in the rest of the yard based on soil samples.  

iii. HUD’s Mandatory Inspections for Lead-Based Paint  

Title X amended the LPPPA to require HUD to establish procedures mandating an inspection for the presence of lead-based paint before a federally-funded renovation is likely to disturb painted surfaces. It also required HUD to establish procedures mandating the inspection and abatement of lead-based paint hazards in all federally owned target housing constructed before 1960, and an inspection for lead-based paint and lead-based paint hazards in all federally owned target housing constructed between 1960 and 1978.  

iv. Amending OSHA to Protect Construction Worker  

As stated above, it was Subtitle C of Title X of this Act, entitled the “Worker Protection Act,” that required OSHA to prepare a lead standard for workers in the construction industry by adding a new Title IV to the Toxic Substances Control Act (“TSCA”).  

3. EPA/HUD’s Residential Lead-Based Paint Disclosure Rule  

On March 6, 1996, the EPA and HUD jointly established regulations requiring sellers, lessors and brokers to provide purchasers and tenants with information about lead-based paint hazards (as defined by the EPA’s Lead Rule) prior to the sale or lease of residential properties. The rule applies only to certain “target housing” (including condos and co-ops) constructed

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449 Id. (codified at 40 C.F.R. §745.65(c)).
451 See supra section IV.B.2 & note 416.
452 Id. at §1021; amending 15 U.S.C. §§2601-2692 (subchapter IV – Lead Exposure Reduction).
before 1978 when the Consumer Product Safety Commission banned the use of lead-based paint. The rule exempts “zero-bedroom dwellings” such as lofts and studio apartments and housing for the elderly and the handicapped, provided that such housing will not be occupied by children under the age of six.

The Lead Disclosure Rule’s purpose is to ensure that parents are informed about potential hazards from lead-based paint, and what they might do to protect themselves and their children. It affects an estimated 9 million renters and 3 million homebuyers each year. The Rule has four main requirements that must be met before a contract for sale or lease is ratified. Specifically, the Rule requires:

- sellers and lessors (landlords) to provide the purchaser or lessee with the EPA, CPSC and HUD jointly-developed pamphlet entitled Protect Your Family From Lead in Your Home or an equivalent pamphlet that the EPA has approved for use in a particular State;

- sellers and lessors to disclose known lead-based paint and lead-based paint hazards to purchasers or lessees (renters);

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455 Id. “Zero-bedroom dwelling means any residential dwelling in which the living areas are not separated from the sleeping area. The term includes efficiencies, studio apartment, dormitory or single room occupancy housing, military barracks, and rentals of individual rooms in residential dwellings. . . .” 24 C.F.R. §35.110. Altera, supra note 433 at n.38.


457 While the disclosure rules generally apply to all transactions to sell or lease target housing, including subleases, they do not apply to (1) sales of target housing at foreclosure; (2) leases of target housing found to be lead-based paint free by a certified inspector; (3) short-term leases of 100 days or less, where no lease renewal or extension can occur; and (4) renewals of existing leases in target housing in which the lessor has previously disclosed all required information and where the lessor does not have any new information about the presence of lead-based paint and/or lead-based paint hazards. Altera, supra note 433 at 111-12.

458 Copies of the required federal pamphlet, entitled “Protect Your Family From Lead in Your Home,” may be ordered by calling 1-800-424-LEAD or found on the EPA’s lead web site at: http://www.epa.gov/lead (last visited Aug. 2, 2006). This pamphlet concerns minimizing lead-based paint dangers to children and identifying and controlling lead-based paint hazards. States are permitted to develop their own lead hazard information pamphlets subject to the final authorization and approval of the EPA. John B. Shumway, HUD Enforcement Of Lead-Based Paint Rules And Other Lead-Based Paint Activities, 12 J. Affordable Hous. & Cmty Dev. L. 366, 368-69 (2003).
• sellers and lessors to provide the purchaser or lessee with relevant records or reports concerning lead-based paint and lead-based paint hazards in the housing; and

• sellers to give purchasers a 10-day period to conduct an inspection or risk assessment before the purchaser is obligated under a contract to purchase target housing.\textsuperscript{459}

The Rule’s primary focus is on disclosure of known hazards. It does not require that the seller or landlord conduct or finance a lead inspection, nor does it require that a building owner or landlord test for lead-based paint or remove lead paint discovered during an inspection. Although the above requirements must be satisfied before the parties become obligated under the contract, non-compliance with the Rule’s requirement does not invalidate sale and leasing contracts.\textsuperscript{460}

A seller, lessor, or agent who fails to give the proper information is subject to civil and criminal penalties of up to $30,000 per occurrence and one year in jail, as well as triple damages and attorneys’ fees in a private civil suit.\textsuperscript{461} Agents are not liable for hazards which were not made known to them by their principals.\textsuperscript{462} Since the Lead Disclosure Rule took effect, HUD – in cooperation with the U.S. Department of Justice (“DOJ”), the EPA, and state and local health and housing agencies – has initiated numerous cases against housing authorities, sellers and landlords for violating the Rule.\textsuperscript{463} Persons and entities found violating this Rule have had to abate housing units, fund child health improvement projects, and pay significant fines.\textsuperscript{464}

\textsuperscript{459} \textit{Shumway, supra} note 458 at 369. Sellers, landlords and agents are required to document their compliance with the rule by attaching a disclosure form to the sales contract or lease containing signature lines certifying that lead-based paint information was provided and received. \textit{Id.}

\textsuperscript{460} \textit{EPA Fact Sheet, supra} note 454.


\textsuperscript{462} 40 C.F.R. §745.115(2)(b).

\textsuperscript{463} \textit{See, e.g., EPA, Salem, Mass. Housing Authority Faces Fines for Failure to Disclose Lead Paint Hazards to Tenant}, News Release (July 6, 2006) (the EPA filed an administrative complaint claiming that the Salem Housing Authority violated several federal Lead Disclosure Rule requirements at 14 of its residential units, by failing to disclose information about
4. **HUD’s “Lead-Safe Housing Rule”**

On September 15, 1999, HUD issued its final lead-based paint regulation for federally owned residential property and housing receiving federal assistance. The Rule far exceeds disclosure requirements and includes testing and treatment of lead-based paint hazards. In brief, the Rule completely revises HUD’s regulations on the testing and treatment of lead-based paint hazards in federally assisted and owned housing, and it codifies all of HUD’s lead-based paint regulations in one part of the Code of Federal Regulations to make it easier to find and apply HUD policy. In its Rule, HUD set hazard reduction requirements more strongly emphasizing the reduction of lead in dust.

**D. CONSUMER PRODUCT SAFETY COMMISSION (“CPSC”) AND FOOD AND DRUG ADMINISTRATION (“FDA”) EFFORTS TO REGULATE AND/OR BAN LEAD-CONTAINING PRODUCTS**

1. **The Lead Paint Ban**

   i. **A Primer on Paint**

   People have been using lead-based paint since before the Roman Empire. By the 12th century, the painting trade was a widely recognized profession in England. About this time a guild (The Worshipful Company) was established in London. In addition to training painters and

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464 See generally id.

465 Shumway, supra note 458 at 372.


standardizing quality, the guild also kept a close rein on available jobs, since the best projects came directly through it. At this point in history, paint making involved the painters mixing dry pigments with oils such as linseed oil.

Advancements in the production of paint came about in the early 1700s when paint mills in England and America were making finely powdered pigments ground with a granite ball. The first recorded paint mill in America was reportedly established in Boston in 1700 by Thomas Child. Painters would blend pigments with a solvent on their own. Thus, each painter created his own unique brand of paint. It was not until the mid-to-late 1860s that paint manufacturers began making pre-mixed paints for customers. By the mid-1880s, mechanization was making the manufacturing process accessible to a larger and less specialized group of entrepreneurs. More importantly, the weight of prepared paint makes it expensive to transport, so a decentralized structure of small paint factories began springing up in population and industrial centers across the nation. These small independent paint mills dominated the industry until the mid-1900s.

Contrary to popular belief, paint is not a fungible product because, as shown above, there have been many manufacturers of the lead pigment from its introduction in America until the mid-1950s (300 or more years). Moreover, until the late 1800s and early 1900s, most painters mixed up their own paint to meet their own unique and diverse needs. This diversity in paint

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470 Ester Brody, supra note 468.

471 Id.

472 With respect to inorganic pigments: black lead sulfate was first introduced between 1855 and 1866; chrome green was first used in 1825; chrome orange, red and yellow were discovered in 1809; lead sulfate (white lead) goes back to at least Roman times; mineral yellow was patented in 1871; and red lead also dates back to Roman times. Mike van Alphen, PAINT FILM COMPONENTS: NATIONAL ENVIRONMENTAL HEALTH MONOGRAPHS, National Environmental Health Forum Monographs, General
formulations is driven by the need for a range of colors, the wide range of surfaces to which paint is applied, a diversity of paint uses and differing exposure (weathering) conditions. There are many paint properties such as covering or obscuring power, drying time, paint-film-hardness, film flexibility, color permanence, water resistance, UV resistance, ease of application, control of paint layer thickness, rate of chalking, mold and fire resistance, among others, that are able to be modified by varying the properties and proportions of major components and minor additives.\footnote{Paint Monograph, supra note 473, at 9 (citing F.N. Vanderwalker, The Mixing of Colors and Paints (Frederick J. Drake & Co. 1944)).} The commercial pressures to differentiate products in the marketplace, and the many possible ways to prepare paints resulted in a wide variety of accessory compounds in pigment materials. Likewise, variations in the origin and amount of processing of minerals and chemical raw materials also had a strong influence on accessory compounds in a bulk pigment product as well as an influence on trace elements in bulk pigment samples and trace elements within pigment particles.

Of all the lead-based paints, white lead was once clearly the foundation of the paint industry. A 1944 book about mixing paint contained a listing of 246 paint color recipes, 135 of which included white lead as an ingredient, and an additional 38 that did not contain white lead but did list other lead pigments such as lead chromates.\footnote{Id. at 9.} This just illustrates the uniqueness and variability of paint.

(a) White Lead Paint

The use of white lead in paint goes back to at least Roman times. However, in the 1620s, the Dutch greatly increased availability of white lead and lowered its cost with the invention of
the Stack Process. Generally, all white lead paints included chalk in their undercoats, reserving purer white lead for finish coats.\textsuperscript{475}

White lead or basic lead carbonate is an intimate mixture of lead carbonate, lead hydroxide and sometimes lead oxide, and has an “ideal” composition $\text{PbCO}_3 \cdot 2\text{Pb(OH)}_2$.\textsuperscript{476} White lead can be formulated into paint which is very easy to work with. The product is very durable and has good external weathering properties. This was one of the major reasons for using white paint although its use was largely phased out in the 1950s. The only remaining and legally permitted uses are for certain historic buildings and in artists’ colors for restoration work.\textsuperscript{477}

(b) White Basic Sulfate of Lead

One problem with white lead was its tendency to produce dirty brown stain on its surface in sulfur polluted atmospheres.\textsuperscript{478} It also reacted to other industrial atmospheric pollution by yellowing. To combat this problem manufacturers created lead sulfate pigment to be used as a primer and in building finishes. It was first introduced between 1855 and 1866. The leaded zinc oxide form was introduced in 1896.\textsuperscript{479} Basic sulfate of lead was considered to be an excellent paint when mixed with other pigments such as zinc oxide or carbonate of lead. It was considered especially suitable for surfaces that are subjected to conditions such as are to be found in sea-

\textsuperscript{475} The History of Paint, available at \url{http://www.brendasemanick.com/art/historyofpaint.htm} (last visited Sept. 15, 2006).

\textsuperscript{476} Applications of Lead, Lead Development Association International 40 (LDAI), available at \url{http://www.ldaint.org/factbook/chapter3.pdf} (last visited Sept. 15, 2006). White lead is usually the basic carbonate lead and is, or has been known as: Berlin White, Blanc de Plomb, Ceruse White, Corroded White Lead, Cremnitz White, Cremser White, Dutch White Lead, Flake White (also a Bi pigment), Kremser White, London White, Minium Album, Psmithium, Silver White, Slate White, Foundation White, Nottingham White and Snowflake White. A pigment mixture known as reduced white lead also goes by the following names: Dutch White, Hamburg White and Venetian White. Reduced white lead is a mixture of barium sulfate with increasing white lead in the ratios 80:20, 65:35 and 50:50, respectively. Paint Monograph, \textit{supra} note 473, at 27.

\textsuperscript{477} Applications of Lead, \textit{supra} note 476, at 40.

\textsuperscript{478} Paint Monograph, \textit{supra} note 473, at 31. The names given to lead sulfate pigments include: Freeman White, Basic Lead Sulphate, Milk White, Mulhouse White, Bartlett White Lead, Sublimed White Lead and Freeman’s lead, Silver lead and Purex white. \textit{Id.}

\textsuperscript{479} \textit{Id.} at 124.
coast locations. While it was not as good a white color as high-grade basic carbonate white lead, it is claimed to be more stable in color when used in exterior paints in cities where it is subject to sulfurous gases.\textsuperscript{480}

\textbf{(c) Red Lead Paint}

Red lead (Pb\textsubscript{3}O\textsubscript{4}) is prepared by the oxidation of lead monoxide. The advantage of red lead paint was its anti-corrosiveness, good mechanical strength, water resistance and adhesion to steel surfaces. However, red lead paint had a tendency to quickly thicken, requiring constant mixing. Ready-mixed red lead paints from the 1940s to 1960s contained pigment having greater than 93\% Pb\textsubscript{3}O\textsubscript{4} or greater than some 84.3\% Pb as Pb\textsubscript{3}O\textsubscript{4} with the remaining material being other lead oxides. The greater the proportion of Pb\textsubscript{3}O\textsubscript{4}, the more red the color. Lower grades were likely to be yellow-orange in color.\textsuperscript{481}

Red lead primers were often reserved for “highly exposed” surfaces such as windowsills and doorway sills. Areas having exposed woodwork and that become cracked, prior to repainting, are commonly “spot primed” with red lead primer for example.\textsuperscript{482}

\textbf{(d) Titanium Dioxide: Lead’s Replacement}

Although titanium dioxide was introduced shortly after 1918, it was not used widely because of its high cost.\textsuperscript{483} The first titanium dioxide pigment was a composite of 30\% titanium dioxide (anatase crystal structure) and 70\% barium sulfate. Titanium dioxide pigment, like other

\textsuperscript{480} Id. at 31.
\textsuperscript{481} Id. at 32-33.
\textsuperscript{482} Id. at 33.
\textsuperscript{483} Id. at 49-50. DuPont was the largest producer of lithopone in the United States in 1930, producing 33,000 tons in 1914 and 200,000 tons in 1928. The first U.S. commercial production of titanium white began in 1918 by Titanium Products. In 1920, National Lead purchased an interest in Titanium Products and acquired the remaining interest in the company by the 1930s. DuPont soon entered the TiO\textsubscript{2} business but could not sell TiO\textsubscript{2} as a pigment without infringing patents held by the US National Lead Company, so it manufactured titanated lithopone. With the depressed price of zinc in the 1920s, titanated lithopone was cost competitive relative to otherwise superior TiO\textsubscript{2} products. At that time U.S. National Lead and Commercial Pigments Corporation controlled the anatase TiO\textsubscript{2} pigment patents. DuPont and US National Lead came to an agreement in 1933 relating to TiO\textsubscript{2} manufacture and US National Lead and British lead industry and other interests joined together in 1932. Id.
modern pigment particles, consists of a pigment core coated with a thin layer of material designed to improve the pigment characteristics. According to IARC, by 1945 titanium dioxide represented 80% of white pigment on the market. Concomitantly, the use of white lead in paints fell during 1900-1945 from nearly 100% to less than 10%.\textsuperscript{484} In 1924, the Bureau of Standards issued proposed master specifications for a lead-free exterior white titanium and zinc paint. Tests comparing titanium dioxide to white lead demonstrated the former to be a technologically competitive pigment. Moreover, by 1929, titanium pigments had assumed an important role in European countries that had legislatively restricted the use of white lead in paints.\textsuperscript{485}

\textbf{ii. Early Paint Regulations}

\textbf{(a) Europe}

One of the early conventions directed towards the control of lead-based paint was the International Labour Office (“ILO”) White Lead (Painting) Convention of 1921 (No. 13). Twenty-seven countries had ratified the 1921 ILO convention which prohibited the use of white lead or sulfate of lead in “internal” painting.\textsuperscript{486} The regulation of the use of toxic pigments and dye in the manufacture of objects of daily use, in Germany, dates back to 1887.

\textbf{(b) Great Britain}

In an effort to control occupational dust exposures, Great Britain began regulating the methods of manufacturing lead pigments between the 1920s and 1940s. The UK, Lead Paints (Protection Against Poisoning) Act of 1926 restricted lead in paints as follows:

For lead paints to be applied by spraying, the Act lays down a maximum figure (5 percent) for the lead compounds soluble in 0.25% hydrochloric acid, which is the

\textsuperscript{484} Id. at 44 (citing IARC, Some Organic Solvents, Resin Monomers and Related Compounds, Pigments and Occupational Exposures in Paint Manufacture and Painting. IARC Monographs, Volume 47 World Health Organization (1989)).

\textsuperscript{485} Id.

\textsuperscript{486} Id. at 12 (citations omitted).
concentration in normal digestive juices. This soluble-lead figure is calculated as lead monoxide (PbO) and is expressed as a percentage on the pigment.\textsuperscript{487}

One interpretation of the above is that an extraction of a maximum of 5% lead (calculated in terms of the PbO compound at 92.85% Pb) using a 0.25% HCl digestion of paint was permitted. This equates to paint returning an apparent analysis of 4.65 wt.% Pb by the weak HCl digestion method. In the early 1960s, the British Paint Manufacturers’ Executive Committee recommended to its members that paints containing more than 1.5% toxic materials on the dry film from January 1963 onwards should be labeled as unsuitable for surfaces likely to be chewed by children.\textsuperscript{488}

(e) Australia

The Queensland Health Act (“QHA”) of 1937 prohibited lead, arsenic or antimony in toys, wallpaper, decorative paper, paper serviettes and food wrapping paper amongst other things. Section 127 of the QHA restricted the soluble lead concentration of paints to 5% and prohibited the use of paints containing lead on roofs. The QHA appears to be very similar to the 1926 UK legislation. Section 127 of the Act does not appear to address the use of lead paint on interior surfaces. However, structures already painted with “soluble lead” could have attracted prosecution. Where paints in excess of 5% soluble lead were discovered, the State had the power to enforce the removal of that paint. Section 128 of the Act required the labeling of the name or names of ingredients and the quantity or percentage of each in the paint. It appears that this legislation was not designed to limit the amount of lead in paint (except roof paint), but to reduce the proportion of carbonate of lead to 5% (calculated as PbO), or 4.65 wt % lead soluble in weak hydrochloric acid.\textsuperscript{489} The use of white lead in paint was not banned until 1956.\textsuperscript{490}

\textsuperscript{487} Id. (citing British Standard 282: 1963).
\textsuperscript{488} Id. at 12-13 (citations omitted).
\textsuperscript{489} Id. at 14 (citations omitted).
iii. Z66.1: The Voluntary Standard

As a result of the discovery and subsequent use of titanium dioxide as a paint pigment, the use of white lead steadily decreased as the use of titanium dioxide pigments steadily increased, particularly from the early 1950s to the early 1960s. Today, HUD classifies homes into three categories: those built before 1960, those built between 1960 and 1977 and those built after 1978. HUD classifies houses by these categories because of voluntary industry action taken in 1955 and federal regulations passed in 1977.

Before 1955, most of the white house paint used in the U.S. could have contained up to 50% lead. In 1955, paint industry representatives, working with the American Academy of Pediatrics and a host of other organizations participated in an American Standards Association Committee on Hazards to Children, and established the “Z66.1 Standard.” This standard required that any paint sold for interior use should contain less than one percent lead by weight in its dried film.

490 Id. The main provision of Australia’s 1956 paint law were:

(1) The manufacture, sale and use of paint containing basic lead carbonate (white lead) was banned in its entirety.

(2) Paint containing lead of any description could not be used on the roof of a house or other building or structure; any exterior portion of any house or other building whatsoever; any fence or gates whatsoever; any interior portion whatsoever of a house; or any household furniture.

(3) Paint containing not more than 5% soluble lead chromate may be used on certain places but shall not be used on any exterior portion of a building which is accessible to children under fourteen years of age.

Id.

491 Paint Monograph, supra note 473, at 10.


iv. The Federal Ban

On September 1, 1977, the CPSC issued a final ban on lead-containing paint and on toys and furniture coated with such paint. Before this action was taken, the maximum level of lead allowed in consumer paints had been 0.5 percent. CPSC has lowered this amount to 0.06 percent, a level conforming with the maximum permissible under the Lead-Based Paint Poisoning Prevention Act. This ban became effective in 1978. In 1997, it was estimated that between 83 and 86% of all homes built before 1978 in the U.S. have lead-based paint in them.

2. Children’s Jewelry

Not surprisingly, lead paint is what most people think of when asked how children get lead into their bodies. But believe it or not, even today, a lot of jewelry made for children, marketed to children and sold to children contains high concentrations of lead. It was not until February 3, 2005, that the CPSC developed an enforcement policy to reduce the potential for health risks from lead in children’s metal jewelry. Enforcement is one thing, but getting the cheap jewelry off the market is another. Recalls of children’s jewelry are seemingly becoming a

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494 16 C.F.R. §1303.1 (declaring that that paint and similar surface-coating materials for consumer use that contain lead or lead compounds and in which the lead content [calculated as lead metal] is in excess of 0.06 percent of the weight of the total nonvolatile content of the paint or the weight of the dried paint film [which paint and similar surface-coating materials are referred to hereafter as ‘‘lead-containing paint’’] are banned hazardous products under sections 8 and 9 of the Consumer Product Safety Act [CPSA], 15 U.S.C. 2057, 2058). Specifically, the CPSC declared the following products as banned hazardous products: (1) toys and other articles intended for use by children that bear “lead-containing paint;” (2) furniture articles for consumer use that bear “lead-containing paint.” Id.


496 CPSC Announces Final Ban On Lead-Containing Paint, Release No. 77-096 (Sept. 2, 1977). Exempted from the new ban are mirrors backed with lead-containing paint which are part of articles of furniture, as well as artists’ paints and related materials. Also exempted, provided they bear specified cautionary labeling, are certain agricultural and industrial coatings, touch-up coatings for appliances and lawn and garden equipment, graphic arts coatings, and certain coatings for powered model aircraft. Id.

497 See Lead Toxicity, supra note 202.

common occurrence these day. In the last couple of years over a hundred million pieces of cheap and free children’s jewelry has been recalled because of high lead concentrations. And these are just the products we know about. There is no telling how much of this jewelry has been sold. More importantly, there is no telling how many children stuck this jewelry in their mouths and unknowingly ingested high concentrations of lead.

i. The Recalls

CPSC’s Jewelry Policy was developed after it learned about a 4-year-old child who died from lead poisoning after swallowing a necklace distributed by Reebok International, Ltd. (“Reebok”) that contained high concentrations of lead. The products at issue in this case were 8-inch-long metal bracelets and charms that have the name “Reebok” engraved on the side of the charm. The jewelry was manufactured in China and were given by Reebok to customers as a free gift with the purchase of various styles of children’s footwear at major shoe stores nationwide from May 2004 through March 2006.\textsuperscript{499} As a result of this incident, Reebok recalled about 300,000 silver-colored, heart-shaped charm bracelets beginning in March 2006.\textsuperscript{500} Reebok is not the only company whose “free gifts” contained high levels of lead. As of May 2006, Twentieth Century Fox was having to recall over 700,000 silver-colored metal charms that were included as a free giveaway in certain Shirley Temple movie DVDs.\textsuperscript{501} Also in March, 2006, the CPSC recalled over half a million pieces being made for and sold by the Dollar Tree Store and its affiliates. The Jewelry recalled were Mood Necklaces and Rings, Glow-in-the Dark Necklaces and Rings, and UV Necklaces and Rings.\textsuperscript{502}

\begin{flushleft}
\textsuperscript{500} \textit{Id.}
\textsuperscript{501} CPSC, \textit{Metal Charms Sold with Twentieth Century Fox DVDs Recalled for Toxic Lead Hazard}, New Release No. 06-156 (May 5, 2006).
\textsuperscript{502} The rings are silver in color, adjustable, and have one of a variety of designs with a toy “gem” in the center. The necklaces have a black string with silver colored clasps and a silver charm with a “gem” in the center. The following are printed
\end{flushleft}
Unfortunately, parents cannot avoid the problem just by avoiding cheap jewelry or discarding the free giveaways. On May 10, 2006, Oregon announced the recall of a charm necklace emphasizing cheerleading that was sold at department stores nationwide from September 2005 through April 2006 for about $95. Likewise, on April 4, 2006, the CPSC recalled 180,000 “American Girl” children’s jewelry sets due to high levels of lead in the product. The jewelry sets include American Girl necklaces, bracelets, earrings and hair accessories. This jewelry was made in China and sold for $8 to $12. There are many other examples as agencies and consumer groups test products for lead content.

While the above recalls are the latest, they are not the largest. On July 8, 2004, the CPSC announced the voluntary recall of 150 million pieces of toy jewelry sold in vending machines across America. Although CPSC believe only half (75 million pieces of children’s jewelry) contain “dangerous levels of lead,” industry is recalling all of the jewelry because it is difficult to distinguish the lead jewelry from the non-lead jewelry. The toy jewelry was sold nationwide in vending machines located in shopping malls, discount stores, department stores and grocery

503 Oregon, Lead Poisoning Prevention, Juicy Couture Children’s Jewelry Recalled for Lead Poisoning Hazard (May 10, 2006). The charms included a yellow painted metal shirt with “Viva La Juicy” printed on the front, a cheerleader’s megaphone with the word “Juicy,” a green heart with “Juicy Couture,” a black dog, the letter “J,” and a purple flower. Id.


stores for between $0.25 and $0.75. The jewelry came in various styles of rings, necklaces and bracelets. All the jewelry was manufactured in India.  

ii. CPSC’s Jewelry Policy

As a result of incidents involving children’s jewelry, the CPSC collected and tested many items and found that a lot of them contained high levels of accessible lead. Under the Federal Hazardous Substances Act (“FHSA”), articles of metal jewelry are deemed “hazardous substances” if they contain toxic quantities of lead sufficient to cause substantial illness as a result of reasonably foreseeable handling or use, including reasonably foreseeable ingestion by children. If such jewelry is intended for use by children and its toxic lead content is accessible by a child, then it constitutes a “banned hazardous substance” under the FHSA.

Under this policy, CPSC staff will conduct a screening test to determine the lead content of each type of component in a piece of jewelry. For example, a necklace may consist of a chain, a clasp, a pendant and one or more types of beads. If the lead content of each component is less than or equal to 600 parts per million (ppm), the staff will not pursue a recall or other corrective action. If the lead content of any component exceeds 600 ppm, the CPSC will conduct further testing to determine if the jewelry has an amount of accessible lead less than or equal to 175 micrograms (µg). If it has more they 175 µg of accessible lead (an amount the CPSC believes could result in elevated blood lead levels in children), the CPSC may order a recall depending the age of the children who are most likely to wear the jewelry, the level of accessible lead, the

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507 Id. The rings are gold- or silver-colored with different designs and paint finishes with various shaped center stones. The necklaces have black cord or rope or gold- or silver-colored chains. The necklaces have pendants, crosses or various geometrical designs or shapes, and can include gemstones. The various styles of bracelets include charm bracelets, bracelets with medallion links and bracelets with faux stones. Id.


size and shape of the jewelry components, the probable routes of exposure and other factors. According to the CPSC, the benchmark for lead in children’s jewelry is 600 ppm.\textsuperscript{511}

3. Baby Cribs and Accessories

Children’s jewelry is not the only product being sold for use by children that contain lead-based paint. As a result of testing, the CPSC has found that some baby cribs and accessories being sold in the U.S. contain hazardous levels of lead paint.

In 1985, Danara International, Ltd., a New Jersey company, voluntarily recalled two styles of baby crib exercisers because of excess levels of lead in the paint on the products. Danara sold about 100,000 of these crib exercisers between 1981 and 1985 for approximately $8.00 each through stores nationwide. Two crib exercisers are involved: a Winnie the Pooh exerciser that had an image of Pooh suspended from a wooden cross bar along with wooden beads and plastic rings, and a Smurf exerciser similar to the Winnie the Pooh exerciser except that a blue plastic Smurf is suspended from the wooden cross bar. The paint on both crib exercisers contained 2 percent lead.\textsuperscript{512}

In 1992, the Little Tikes Company had to recall 16,300 crib toys known as “Little Tikes Crib Centers” because of hazardous levels of lead paint in the red stripes on the candy cane roller. The Little Tikes Crib Center was intended to be mounted on an inside crib rail. The crib center has nine large bright features including the red and white cylindrical candy cane roller. Surprisingly, the label on the carton in which the crib center was sold stated that the product was

\textsuperscript{511} CPSC, Interim Enforcement Policy for Children’s Metal Jewelry Containing Lead (2005).

\textsuperscript{512} Danara Baby Crib Exercisers Recalled Because of Lead Hazard, Release No. 85-063 (Dec. 5, 1985); see also, Musical Crib Mobile Recalled, Release No. 87-033 (June 4, 1987) (recall of 364 musical mobiles because the red paint on the clear plastic rings of the toy contains approximately 8% lead).
made in the U.S. The Little Tikes Crib Center was sold nationwide at a variety of retail stores from November 1991 through January 1992, at an estimated price of $20.00 each.513

As recently as November 2005, the CPSC announced the recall of 335 full size cribs sold by the Delta Enterprise Corp. of New York City. The cribs were marketed Lov’s “Europa” Natural Color Cribs. They were manufactured in Indonesia for Delta. The cribs were sold exclusively at Toys R Us nationwide from July 2004 through August 2005 for about $200. The cribs were made of wood and are natural in color. The crib’s paint contains high levels of lead.514

4. Lead in Candy and in the Candy Wrappers
   i. The Candy Wrappers

The CPSC is looking into several brands of candy manufactured in Mexico that are wrapped in wrappers containing lead or bearing lead-containing ink. It analyzed several samples of the wrappers and found various levels of lead in or on the outside of many of the wrappers. CPSC is concerned about the risk of lead poisoning to consumers, in particular, young children, who handle candy with wrappers that contain or bear lead. Consequently, the CPSC sent letters in July 2004 to Mexican candy producers and to U.S. candy importers warning them of the dangerous wrappers.515

513   The Little Tikes Company Recalls Little Tikes Crib Center Due To Lead Paint Hazard, Release No. 92-094 (June 16, 1992).


ii. The Candy

In 1995, the Food and Drug Administration ("FDA") issued a letter\(^{516}\) advising manufacturers, importers, and distributors of imported candy that levels of lead found in some imported candy products indicate that lead contamination of the candy not attributable to the use of lead-based printing inks on packaging materials may have occurred. At that time, the FDA did not know whether the lead came from the use of food ingredients, from food processing equipment, from utensils used in food plants, or from some other source. Furthermore, at that time, the FDA did not have a standard for the maximum permissible level of lead in candy. Nonetheless, it believed that it had the authority to take regulatory action against candy with lead levels that exceeded 0.5 parts per million (ppm).\(^{517}\) Since that letter was issued in 1995, the specification for lead in sucrose has been reduced from 0.5 ppm to 0.1 ppm. Likewise, the FDA also learned that certain ingredients often used in candy products imported into the U.S. may be a source of avoidable lead exposure. These ingredients include chili powder and tamarind, which are used in candies marketed to the U.S. Hispanic population. Additionally, the FDA is concerned that certain manufacturing processes or conditions are being used that contribute to elevated lead levels in candy: such as the drying of raw ingredients in areas with high levels of airborne lead, storing ingredients in improperly glazed ceramic vessels that may leach high levels of lead into the product, and grinding ingredients under conditions that may result in lead contamination.\(^{518}\)

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\(^{517}\) FDA, Letter to Manufacturers, Importers, and Distributors of Imported Candy (Mar. 25, 2004) available at http://www.cfsan.fda.gov/%7Edms/pbltr.html (last visited Aug. 7, 2006). The 0.5 ppm guideline was, at that time, equivalent to the Food Chemicals Codex (FCC) specification for lead in sucrose (sugar), the main ingredient in candy. The FCC is a compendium published by the Food and Nutrition Board of the Institute of Medicine, National Academy of Sciences, which contains food-grade specifications for food ingredients; in most cases, these specifications are eventually incorporated into relevant FDA regulations. Id.

\(^{518}\) Id.
As a result, in December 2005, the FDA issued non-enforceable draft guidelines recommending that lead levels in candy products likely to be consumed frequently by small children not exceed 0.1 ppm because such levels are achievable under good manufacturing practices and would not pose a significant risk to small children for adverse effects.\(^{519}\)

5. Crayons and Sidewalk Chalk

In 1994, the CPSC announced a recall of certain color crayons made in China after testing them for lead. CPSC found that the yellow and orange crayons contained hazardous amounts of lead. If a child were to eat or chew on these crayons, lead poisoning could occur.\(^{520}\)

The following were the crayons at issue:

- **“12 Jumbo Crayons,”** Concord Enterprises, Los Angeles, CA. Sold approximately 430 cases in 18 States.

- **“Safe 48 Non-Toxic I’m a Toys “R” Us Kid! Crayons,”** Toys “R” Us, Paramus, NJ. Sold 100,000 packages in 1990-91 and 2,000 packages in 1993. The crayons were packaged in plastic cylindrical containers with a crayon sharpener in the lid and a paper label featuring Geoffrey the Giraffe and reading in part: “SAFE 48 NON-TOXIC I’M A TOYS “R” US KID! CRAYONS *** manufactured by BSA Assoc., N.Y., N.Y. 10010 for Geoffrey Inc. *** Made in China.” Individual crayons bear a paper label reading in part “RAINBOW CRAYONS *** BSA *** Made in China.”

- **“12 Crayons, Glory” and “18 Crayons That Paint, Conforms ASTM D-4236,”** Glory Stationery Manufacturing Company Limited, Los Angeles, CA.

- **“64 Crayons, School Quality, No. 8064,”** A.J. Cohen Distributors, Hauppauge, NY. Sold 226,008 units in Eastern United States.

- **“64 Crayons, #CR 64-64 CT,”** Baum Imports, New York, NY. Sold 80,400 boxes in metropolitan New York City area.

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\(^{520}\) CPSC, CPSC And Concord Enterprises Announce Recall Of Certain Crayons Because Of Lead Poisoning Hazard, (News Release No. 94-049) (Mar. 22, 1994). Parents should buy only crayons and other children’s art materials that have this label: “Conforms to ASTM D-4236,” or similar words. This label means that the crayons and other art materials have been reviewed by a toxicologist for chronic hazards and are labeled appropriately. Id.

“8 Crayons, No 5 CL 850,” Dynamic Division of Agora International, St. Albans, NY. Sold 120,000 boxes nationwide.


“64 Crayons, Kidz Biz,” Bargain Wholesale, Los Angeles, CA. Sold 101,184 boxes nationwide.

“64 Crayons, SKU#51-02600,” Universal International, Minneapolis, MN. Firm imported 295,000 boxes.


According to the CPSC, all of these crayons contain lead. The first three (Concord, Toys “R” Us, and Glory) contained enough lead to present a lead poisoning hazard to young children who ate or chewed on the crayons. The other eight brands did not contain enough lead to increase the blood lead level above the threshold level for lead poisoning (assuming the child only chewed on small pieces of lead-containing crayons over a span of time). But, those other brands of crayons add to the overall “lead load” to children who may have eaten them. If a child ate an entire lead-containing crayon over the same span of time, the lead poisoning threshold for these crayons would have been exceeded.\footnote{Id.}

As recently as 2004, Target and Toys “R” Us had to recall sidewalk chalk they sold because it contained high levels of lead.\footnote{CPSC, CPSC, Target Corporation Announce Recall of Multicolored Sidewalk Chalk, (News Release No. 04-032) (Nov. 13, 2003); CPSC, CPSC, Toys “R” Us, Inc. Announce Recall of Solid-colored and Multi-colored Sidewalk Chalk (News Release No. 04-038) (Nov. 24, 2003).} The Wisconsin Department of Health tested Target’s chalk and identified the lead in the chalk. The sidewalk chalk is packaged in plastic that is
molded to five sticks of chalk and a cardboard backing that is labeled “Double Dipp’n Fun.” Each stick of chalk is triangular shaped and multicolored, with three colors layered together (green, red, yellow, or blue). It was manufactured in China and sold at Target stores from March 2003 to July 2003 for about $1 per package. Toys “R” Us’ sidewalk chalk is packaged in a clear-plastic backpack-type carrying case with these words on the label: “Chalk To Go...Totally Me!” It came in 24 pieces and had different colors and shapes. It was also manufactured in China and was sold nationwide from March 2003 to November 2003 for about $4.99 per package.524

6. Candle Wicks

Until April 2003, candle manufacturers were allowed to use lead as the core of their wicks. A CPSC investigation found that despite a voluntary industry agreement in the 1970s to remove lead from candle wicks, a small percentage of candles still contained lead-cored wicks. CPSC found that some lead-cored wicks could emit relatively large amounts of lead into the air during burning, placing children who inhaled the vaporized lead at risk. Some of the candles tested by CPSC emitted lead levels in excess of 3,000 micrograms per hour - about seven times the rate that could lead to elevated levels of lead in a child. CPSC estimates that an indoor air lead level of 430 micrograms per hour from burning candles could result in hazardous exposure to children.525

524 Id.

525 CPSC, CPSC Bans Candles With Lead-Cored Wicks (News Release No. 03-105) (Apr. 7, 2003). The metal core is used to provide structural rigidity to the wick, i.e., to keep the wick straight during candle production, and to provide an upright wick during burning. CPSC, Metal-Cored Candle Wicks Containing Lead and Candles With Such Wicks; Notice of Proposed Rulemaking, 67 FED. REG. 20062 (Apr. 24, 2002); see also, CPSC, Metal-Cored Candle Wicks Containing Lead and Candles With Such Wicks; Final Rule, 68 FED. REG. 19142-48 (Apr. 18, 2003).
E. DRINKING WATER REGULATION

In 1991, the EPA estimated that 14 to 20% of the total U.S. lead exposure was from drinking water.526 Studies have shown that about 16% of the lead in the first liter of water drawn after the tap has sat idle for at least a six-hour dwell time originated from the faucet fixture.527 The Safe Drinking Water Act (“SDWA”), the main federal law that ensures the quality of drinking water in the U.S., was promulgated by Congress in 1974 and amended in 1986 and 1996. 528 It established a federal program to monitor and increase the safety of the nation’s drinking water supply. It authorizes the EPA to set and implement health-based standards to protect against both naturally occurring and man-made contaminants in drinking water. From a regulatory perspective, lead-contaminated water poses a formidable challenge because finished water (i.e., water leaving a water treatment plant) seldom contains detectable lead. Instead, lead contamination nearly always results from contact with building plumbing systems, which traditionally has been beyond the regulatory authority of the EPA and the states.529

1. The Federal Lead Ban

The first major initiative to control lead in drinking water was the 1986 “Federal Lead Ban.” This was a set of amendments to the Safe Drinking Water Act, signed into law in December 1986 with a state-level enforcement deadline of July 1, 1988.530 The Federal Lead Ban

526 Maas, supra note 232, at 317 (citing the Lead Copper Rule, Env’tl Protection Agency, Drinking Water Regulations: Maximum Contaminant Level Goals And National Drinking Water Regulations For Lead And Copper, 53 FED REG. 110 (1991)).

527 Id. at 318 (citing S.C. Patch, et al., Lead Leaching From Faucet Fixtures Under Residential Conditions, 61 J ENVN’T HEALTH 18-21 (1998)).


529 Maas, supra note 232, at 317.

530 Safe Drinking Water Act Amendments of 1986, Pub. L. No. 99-339 (Dec. 8, 1986). Before this law went into effect, lead in drinking water had been regulated under the 1975 National Interim Primary Drinking Water Regulations. These earlier regulations were inadequate because they mandated lead monitoring at a distribution system’s entrance point rather than at the consumer’s tap. This monitoring did not take into account the lead that entered drinking water through corrosion of materials in the distribution system. Maas, supra note 232, at 317. See also, Ala. Code §§22-37-3, 22-37-6 (1988) (Alabama Lead Ban Act).
Ban prohibited any person from using in any public water system or residential home any pipe or plumbing fitting or fixture that was not lead-free after June 19, 1986. This law was amended in 1996 to prohibit any person from introducing into commerce after August 6, 1998, any pipe or any pipe or plumbing fitting or fixture that is not lead free, except for a pipe that is used in manufacturing or industrial processing. The law also required development of a voluntary standard to limit the leaching of lead into the drinking water for devices that are intended by the manufacturer to dispense water for human ingestion. This was accomplished when the EPA recognized NSF International’s consensus Standard 61, Section 9, as the standard for lead-free plumbing, fittings and fixtures.

2. The Lead Contamination Control Act

In 1988, Congress passed the Lead Contamination Control Act which, among other things, declared that drinking water coolers with lead-lined tanks were considered to be “imminently hazardous consumer products.” In part, this Act was designed to assist schools in

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531 Section 1417 (a)(1)(A) of the SDWA states that “[n]o person may use any pipe, any pipe or plumbing fitting or fixture, any solder, or any flux, after June 19, 1986, in the installation or repair of (i) any public water system or (ii) any plumbing in a residential or nonresidential facility providing water for human consumption that is not lead free.” “Lead free” means that solders and flux may not contain more than 0.2 percent lead; pipes, pipe fittings, and well pumps may not contain more than 8.0 percent lead; and plumbing fitting and fixtures must meet standards established under §1417(e). §1417(d) (defining lead free).

532 SWDA §1417(a)(3). The EPA has interpreted this Section to mean that no one, including the manufacturer or distributor, may distribute any pipe or plumbing fitting or fixture that is not lead free after the August 6, 1998 deadline. Because the statute makes no distinction between wholesale or retail sales of these materials, the EPA believes this provision covers not only initial offering of products for sale but sale or distribution from an inventory of products referred to in §1417(a)(3) after August 6, 1998, except for manufacturing or industrial processes. Fact Sheet: Lead & Cooper Rule, Commonly Asked Questions: Section 1417 of the Safe Drinking Water Act and the NSF Standard, U.S. Env'l. Prot. Agency (2006).

533 62 FED. REG. 44684-85 (August 22, 1997). NSF International’s consensus Standard 61, Section 9 is a health effects-based performance standard, limiting the leaching of lead into the drinking water for devices that are intended by the manufacturer to dispense water for human ingestion. Id.


implementing measures to test for and reduce lead contamination in drinking water from water coolers and other sources.\footnote{536} The Act required:

- EPA to publish a guidance document and testing protocol to assist schools in determining the source and extent of lead contamination in their drinking water;
- EPA to identify and publish a list of brands and models of water coolers that contained lead, including those with lead-lined tanks;
- imposed civil and criminal penalties on the manufacture and sale of lead-containing water coolers; and
- directed the Consumer Product Safety Commission (CPSC) to issue an order requiring water cooler manufacturers and importers to repair, replace, or provide refunds for water coolers containing lead-lined tanks.

While the Act encouraged local authorities to test drinking water for lead hazards at childcare facilities and schools and authorized the EPA to provide financial assistance for such testing, it did not give the EPA authority to enforce the Act’s provisions. Moreover, it did not require states and local authorities to test drinking water for lead and it did not appropriate funds to allow the EPA to assist with testing. As a result, states and local governments conducted only limited testing for lead hazards in drinking water at childcare facilities and schools.\footnote{537} The Act requires schools and daycares to report all lead-testing data for drinking water to school staff, students and parents and remediate all sources of lead producing lead contamination above the federal limit of 20 parts per billion (ppb).\footnote{538} However, because the rules are unenforceable, many schools do nothing. For example, lead tests for Seattle public schools show that more than 80% of its schools have serious lead contamination in one or more drinking fountains. In some

\footnote{536}{“The Administrator shall publish a guidance document and a testing protocol to assist schools in determining the source and degree of lead contamination in school drinking water supplies and in remedying such contamination.” Id. at §300j-24(b).}


\footnote{538}{Mark S. Cooper, \textit{Get The Lead Out Of Schools’ Water}, Seattle Post-Intelligencer (July 2, 2004).}
schools, virtually every drinking fountain in the school was above the EPA limit of 20 ppb. One Seattle school—Alternative School No. 2 (Decatur Elementary) — has a drinking fountain with a lead level of 1,600 ppb.\textsuperscript{539}

3. **The Lead and Copper Rule**

The Lead and Copper Rule, which became effective in December 1992, requires treatment when lead and/or copper in drinking water exceeds certain levels.\textsuperscript{540} It requires every public water utility to determine the severity of lead-contaminated water by monitoring lead level at customer taps - generally every six months, annually, or triennially, depending on the levels of lead observed in drinking water.\textsuperscript{541} If more than 10% of the residences have first-draw lead levels (i.e., following a standing time in the plumbing system of at least six hours) exceeding a concentration of 15 μg/l (EPA’s non-health-based “action level,” expected to be exceeded by about 25% of systems), the public water supplier is required to: (1) provide warning notices to all customers encouraging the owners to test their water for lead; (2) determine what treatment modifications could be implemented to reduce lead levels at the tap; and (3) implement these corrosion-optimization methods and monitor at-the-tap lead levels to determine the effectiveness of the treatment modifications.\textsuperscript{542}

Furthermore, the Rule requires states to report specific results of monitoring to the EPA for systems serving populations greater than 3,300 people beginning in 2002. As of 2004, the EPA found that 22 of the 714 systems for which it had data exceeded the lead action level during

\textsuperscript{539} Id.

\textsuperscript{540} Envt’l Protection Agency, Drinking Water Regulations: Maximum Contaminant Level Goals And National Drinking Water Regulations For Lead And Copper, 53 Fed Reg. 110 (1991) (mandating some creative and nontraditional approaches to achieving a projected 50% reduction in U.S. drinking water lead exposure by the end of the 1990s).


\textsuperscript{542} Maas, supra note 232, at 317.
one or more monitoring periods since 2000. While some cities, such as Seattle and Norfolk, Virginia, have been able to dramatically reduce average residential lead levels, an extensive EPA-funded study of more than 1,000 public water supply systems found that residential tap water lead levels were only slightly more likely to decrease following corrosion control than to remain constant or even increase. Even more disturbing was the finding that the use of zinc orthophosphate (the most popular corrosion inhibitor employed at the time) was almost equally likely to be associated with either a decrease or an increase in average residential lead levels.

Even with all of these regulation, lead-contaminated drinking water remains a very significant issue. In late 2003, water samples in the District of Columbia showed lead levels above the EPA’s 15 ppb action level in many homes and buildings in the city. As a result, D.C. is having to replace lead service lines and provide water filters to homes with lead service lines. It has also added phosphoric acid to the water to reduce corrosivity. Believe it or not, experts looking into the problem found that the chloramines the city began using in 2001 to disinfect its water increased the corrosivity of the water, therefore allowing high levels of lead to leach out of lead service lines. This is a very significant situation because at least 400 other public water suppliers have switched to chloramine disinfection since 2000, and thus the problem experienced by D.C. may prove to be widespread.

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544 Maas, supra note 232, at 318 (citing B.L. Ramaley, Monitoring And Control Experience Under The Lead And Copper Rule, 85 J AM WATER WORKS ASSOC: 64-69 (1993)).


546 Id. Annual expenditures under the Act rose from $6.5 million in fiscal year 1972 to $11.25 million in fiscal year 1980. The money supported up to 62 screening programs in 25 states. Id.
F.  **THE CDCS EFFORTS TO ELIMINATE CHILDHOOD LEAD POISONING**

The Lead-Based Paint Poisoning Prevention Act of 1971 authorized a Categorical Grants Program to help communities carry out screening and treatment programs. The goal of this program was to identify children with high blood lead concentrations and to attempt to abate their environmental sources of lead. Initially, it was administered by the Bureau of Community Environmental Management of the Department of Health, Education and Welfare. Later, it became administered by the Centers for Disease Control and Prevention.\(^{547}\) Over 2.7 million children were screened from July 1, 1972, to June 30, 1979. Of these 2.7 million children, 183,452, or 7%, tested positive by the prevailing criteria. In 1981, over 500,000 children were screened with 18,000 being case “defined” as lead poisoning.

The Lead Contamination Control Act of 1988 authorized the Centers for Disease Control and Prevention (“CDC”) to initiate program efforts to eliminate childhood lead poisoning in the U.S.\(^{548}\) As a result of this Act, the CDC Childhood Lead Poisoning Prevention Branch was created, with primary responsibility for (1) developing programs and policies to prevent childhood lead poisoning, (2) educating both the public and public healthcare providers about childhood lead poisoning, (3) providing funding to state and local health departments to (i) determine the extent of childhood lead poisoning by screening children for elevated blood lead levels, (ii) help to ensure that lead-poisoned infants and children receive medical and environmental follow-up, and (iii) develop neighborhood-based efforts to prevent childhood lead

\(^{547}\) CLS Study, *supra* note 151, at 24.

\(^{548}\) Although no money was appropriated for fiscal 1989, $4 million was appropriated for fiscal 1990, about $8 million for fiscal 1991, and $21.3 million for fiscal 1992. *Id.* at 28.
poisoning, and (4) supporting research to determine the effectiveness of prevention efforts at federal, state and local levels.\textsuperscript{549}

Since its inception in 1990, the CDC childhood lead poisoning prevention effort has, among other accomplishments:

- funded nearly 60 childhood lead poisoning prevention programs to develop, implement, and evaluate lead poisoning prevention activities;
- provided technical assistance to support the development of state and local lead screening plans;
- supported the formation of collaborative relationships between CDC’s funded partners and other lead poisoning prevention organizations and agencies (e.g., community-based, nonprofit, and housing groups);
- developed the Childhood Blood Lead Surveillance System through which 46 states currently report data to CDC;
- expanded public health laboratory capacity in states to analyze blood and environmental samples and to ensure quality, timely and accurate analysis of results; and
- published targeted screening and case management guidelines which provide health departments and healthcare providers with standards to identify and manage children with elevated blood lead levels.

In November 2000, the CDC unveiled its “Healthy People 2010” initiative.\textsuperscript{550} Among the many goals set by the CDC is the elimination of elevated blood lead levels in children.\textsuperscript{551} In 2003, the CDC directed its childhood lead poisoning prevention program grantees to develop a

\textsuperscript{549} Fact Sheet, \textit{About the Childhood Lead Poisoning Prevention Program (CLPPP)}, Cntr. For Disease Control and Prevention, Dept. of Health & Human Serv., \textit{available at} http://www.cdc.gov/nceh/lead/about/program.htm (last visited Aug. 28, 2006).

\textsuperscript{550} Healthy People 2010: Understanding and Improving Health, Cntr. For Disease Control and Prevention, Dept. of Health & Human Serv (2000), \textit{available at} http://www.healthypeople.gov/Document/HTML/Volume1/08_Editorial.htm (last visited Sept. 1, 2006). Healthy People 2010 is a comprehensive, nationwide health promotion and disease prevention agenda designed to serve as a roadmap for improving the health of all people in the United States during the first decade of the 21st century. It includes 467 objectives in 28 focus areas, with each objective was developed a target to be achieved by the year 2010. \textit{Id.} at 1-2.

\textsuperscript{551} \textit{Id.} at §8-11.
plan to eliminate statewide (and therefore, national) childhood lead poisoning by 2010\textsuperscript{552}. To achieve this goal, the CDC awarded $31.7 million in 2003 to states’ departments of health to develop and implement lead poisoning prevention efforts.\textsuperscript{553} This activity became a program requirement for the CDC Childhood Lead Poisoning Prevention Program. To be eligible for grants, the CDC requires that each state’s elimination plan

- Establish an advisory workgroup to publish and implement the statewide childhood lead poisoning elimination plan. This group monitors the plan, leverages resources and enhances cooperative efforts toward the plan’s goal.

- Include, among other items, a detailed assessment of the lead poisoning problem in the jurisdiction, clearly stated goals and objectives and a plan to annually evaluate progress toward elimination.\textsuperscript{554}

1. State Efforts to Eliminate Childhood Lead Poisoning

As part of the plan, states set their elimination goal. For example, some states set as their goal a statewide lead poisoning rate (defined as children with elevated blood lead levels [“BLLs”] in excess of 10 μg/dL) of no more than a set percentage. Michigan, for example, set as its goal a statewide lead poisoning rate of no more than 0.1 percent by 2010. In 2004 (latest numbers available), 1.7 percent of the children tested in Michigan showed elevated BLLs.\textsuperscript{555} Other states, such as Indiana, defined elimination as a reduction in the actual number of elevated BLLs found in the state. For Indiana, the goal is to have fewer than 200 children with BLLs

\textsuperscript{552} The CDC requires that states (or jurisdictions) define “elimination” using the best available data and through solid analysis of the state’s infrastructure. See Rhode Island’s Plan to Eliminate Childhood Lead Poisoning by 2010, Rhode Island Dept. of Health 19 (July 30, 2004).


\textsuperscript{555} See Tyson, supra note 553.
above 10 μg/dL by 2010. As of March 16, 2006, Indiana had 548 children with elevated BLLs. This however, represents only 1.3 percent of the children. Of more concern, the state found that 20% of the children initially showing an elevated BLL were false positives. Other jurisdictions, such as New York City, simply state their goal as having no cases of elevated BLLs by 2010. Yet, states such as Rhode Island, define their goal on a community basis. Rhode Island has defined its elimination goal as:

To decrease the proportion of new cases [incidence] of lead poisoning (defined as blood lead level of 10 μg/dL or more) in children under six years of age to less than 5% in all RI communities without decreasing the availability of lead safe and affordable subsidized housing.

As a measure of its progress toward this goal, Rhode Island looks at the number of communities with an incidence of lead poisoning of less than 5 percent among children under the age of six. In 2005, Rhode Island’s incident rate ranged from 0.0 percent to 4.5 percent. Thus, in 2005, Rhode Island met and exceeded its main goal of elimination. After Rhode Island achieved its goal in 2005, it changed the goal to provide for a declining target ceiling of new incident cases, statewide, for each year from 2006 to 2010. Specifically, it wants the number of new cases of lead-poisoned children to decrease by 100 cases each year (i.e., from 621 at the end of 2005 to


559 Rhode Island’s Plan to Eliminate Childhood Lead Poisoning by 2010, Rhode Island Dept. of Health 19 (July 30, 2004).

560 Id. at 18. Rhode Island’s average incident rate was 2.0% with over 60% of reporting communities having an incident rate of less than 1.0%. Id.
Presently, in Rhode Island the number of children under the age of six who have a BLL in excess of 10 μg/dL at any given point in time has been steadily declining.\textsuperscript{562} In fact, all of the graphs presented by Rhode Island show a continuing downward trend in the number of elevated BLLs.\textsuperscript{563} Clearly, contrary to the state’s arguments to the judge and jury, Rhode Island, like many other states, is continuing to make great strides in their efforts to eliminate childhood lead poisoning.

G. STATE EFFORTS TO DEAL WITH OLD LEAD-BASED PAINT

Generally, there are two ways of addressing the problem of lead-based paint in older housing stock: the “lead-free” approach and the “lead-safe” approach. Under the lead-free approach, property owners are required to remove all lead-based paint from the home or building. This approach is expensive and requires diligence during abatement because if the dust and debris created while removing the old paint is not properly controlled, there is a risk that children may ingest it. Conversely, under the lead-safe approach, the old paint is left in place, but the property owner is required to maintain the painted surfaces in a manner that assures the old paint remain intact.

Since 1970, more than 20 states have enacted statutes or administrative regulations requiring landlords to maintain their properties in a manner that avoids the hazards caused by

\textsuperscript{561} Childhood Lead Poisoning In Rhode Island: The Numbers 2006 Edition, Rhode Island Dept. of Health 5 (July 2006), available at http://www.health.ri.gov/lead/databook/2006_Databook.pdf (last visited Sept. 1, 2006). Specifically, Rhode Island’s goal is to reduce the number of new cases of lead poisoned children (BLL less than 10 μg/dL) in the state from 621 at the end of 2005 to 520 at the end of 2006; 420 at the end of 2007; 320 at the end of 2008; 220 at the end of 2009; and 120 at the end of 2010.

\textsuperscript{562} Id. at 20. Over the last 10 years in Rhode Island, the number of children under the age of six who have had elevated BLL has dramatically declined from 17.7% in 1996 to 3% in 2005. Id.

\textsuperscript{563} Id. The prevalence of childhood lead poisoning fell from 5% in 2004 to 3% in 2005. This represents a 40% decline in one year.
poorly maintained lead-based painted surfaces that are peeling and chipping. All of these states, including Rhode Island, choose the lead-safe approach over the lead free approach.

V.

**LEAD PAINT LITIGATION**

Generally, lead paint cases arose from persons who had elevated levels of lead in their blood who lived in older homes once painted with lead-based paint. Lead paint, when poorly maintained and allowed to chip or flake, can be a health hazard for small children because the ingestion of lead paint chips can cause lead poisoning. These cases have been brought by parents, activists and lawyers seeking relief because children suffer from lead poisoning, have elevated levels of lead in their blood, or are merely in danger of being exposed to high concentration of lead because they live in older homes.

The following scenario is typical. Consider the case of a 3 or 4 year-old girl living in the downtown area of a major city. She has lived in six or seven different old houses since her family moved to the city, and all of those houses carried a risk for lead in their paint. The houses changed hands over the years, so it is unlikely that the persons who owned the structures in the 1950s (when lead pigment was still used in paint) own them now. Moreover, the houses have probably been painted dozens of times and the paint used could have been made by a dozen or more companies. Who is to blame for this situation? If the situation is multiplied by the

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thousands of older homes in the city and thousands of children living in those homes, the vastness and complexity of the issue is apparent.

Traditionally, the landlords or owners of the homes are responsible for the upkeep and maintenance of their properties.\textsuperscript{565} Upkeep of rental properties is just one issue; however, disclosure of lead hazards is another. Beginning September, 1996, landlords were required, by federal law, to provide lead-paint disclosure information to all present and future tenants. Likewise, homeowners (and potentially their real estate agents) were required by the same federal law to disclose to prospective buyers any knowledge they had of lead-based paints in their home, and include a federal form about lead-based paints with the sales contract.\textsuperscript{566} Litigation against the product manufacturers began in the mid-1980s when Ralph Nader formed an alliance of contingency fee lawyers and filed strict products liability lawsuits.\textsuperscript{567} This section discusses the types of lawsuits that have been brought in lead paint cases, available causes of action and discusses new trends by governmental entities to try to force others to remove old lead paint from dilapidated housing.

A. TRADITIONAL LITIGATION

1. Against Landlords

In these types of claims the plaintiffs allege that the landlord negligently failed to properly maintain the home or failed to provide the warnings required by the Residential Lead-

\textsuperscript{565} See Restatement (Second) of Torts, §358, p. 243 (1965); Restatement of Law 2d, Property, §17.6: “A landlord should be subject to liability for physical harm caused to the tenant and others upon the leased property with the consent of the tenant or his subtenants by a dangerous condition existing before or arising after the tenant has taken possession, if he has failed to exercise reasonable care to repair the condition and the existence of the condition is in violation of: (1) an implied warranty of habitability; or (2) a duty created by statute or administrative regulation.” Id. See also, Young v. Garwacki, 402 N.E.2d 1045, 1049 (Mass. 1980); Thompson v. Crownover, 381 S.E.2d 283, 284-85 (Ga. 1989); Pines v. Perssion, 111 N.W.2d 409 (Wis. 1961).

\textsuperscript{566} See supra section IV.C.3.

\textsuperscript{567} Until the 1980s, most litigation over lead poisoning from ill-maintained lead paint was aimed at individual landlords and property owners who allowed their properties to fall into disrepair. Michael B. Sena, Sorting Out the Complexities of Lead-Paint Poisoning Cases, 4 J. Affordable Housing & Community Dev. L. 169, 177 (1995). The first lawsuit against a group of former lead paint manufacturers was filed in 1987 on behalf of five Massachusetts children. See Martha R. Mahoney, Four Million Children at Risk: Lead Paint Poisoning Victims and the Law, 9 Stan. Envtl. L.J. 46, 60 (1990).
Based Paint Hazard Reduction Act. According to the statutes, these requirements went into effect on October 28, 1995.\textsuperscript{568} However, in the Federal Register and Code of Federal Regulations, HUD and the EPA set December 6, 1996 as the effective date of the regulations. It was not very long until a plaintiff claimed that she was not warned about lead hazards when her lease began during this gap and the defendant claimed he had no duty to warn because the regulations were not yet effective.\textsuperscript{569} Noting that the statutory deadlines were directives to the agencies (as compared to directly imposing duties on sellers and lessors), the court held that the Act’s implementing regulations, rather than the Act itself, created duty to make disclosures.\textsuperscript{570} Thus, the duty to warn became effective on December 6, 1996.

Few courts have considered the questions of what qualifies as a violation, what disclosure is required under the statute and what consequences flow from violation of the statute.\textsuperscript{571} The most thoughtful and complete opinion on the subject to date is \textit{Smith v. Caldwell Banker Real Estate Service}.\textsuperscript{572} In that case, the court considered whether substantial compliance with the statute was adequate (it was not), when the relevant disclosures must be made (they must be made before the purchaser is obligated on the contract), what is meant by “knowing” violation (acts done with awareness and not through ignorance, mistake or accident-applies), and whether

\textsuperscript{568} See 42 U.S.C. §4852d(a)(1) & (d) (regulations to be promulgated no later than 2 years after October 28, 1992, regulations become effective 3 years after October 28, 1992).

\textsuperscript{569} See \textit{Sweet v. Sheahan}, 235 F.3d 80 (2d Cir. 2000).

\textsuperscript{570} \textit{Id.} at 87 (holding that although the statute unambiguously states that the regulations are to take effect on October 28, 1995, that provision was rendered unenforceable by the failure of HUD and EPA to promulgate final regulations before that date).

\textsuperscript{571} Specifically, 42 U.S.C. §4852d.

\textsuperscript{572} 122 F. Supp 2d 267 (D. Conn. 2000). In this case, the plaintiff purchasers were verbally advised that lead was present in the home and were provided with a copy of the EPA lead hazard handbook prior to signing the contract for sale. \textit{Id.} at 271. However, they were not provided with a copy of a prior lead paint report until the closing and never signed the lead-based paint disclosure form. The signed contract in \textit{Smith} also included a provision incorporating the lead disclosure form into the lease. Specifically, the provision stated that the parties agreed as a precondition to the validity of the agreement that each party received, signed and annexed a lead disclosure and acknowledgment form as required by law. \textit{Id.} at 272.
the purchaser or tenant has the right to rescind if the seller or landlord fails to comply with the Act’s requirements (no right to rescind).  

Individuals are not the only parties suing over disclosure requirements. In 2003, the EPA began an investigation into a Massachusetts landlord and subsequently brought a civil action for his failure to provide information about the presence of lead-based paint present in housing built before 1978. In January 2006, the landlord and his company settled the lawsuit by agreeing to pay a $17,800 fine and undertake lead abatement projects.

2. Against Product Manufacturers

i. Failure to Identify the Manufacture of the Paint

One of the biggest hurdles plaintiffs have when suing paint or pigment manufacturers is their inability to identify with any specificity the manufacturer of the lead paint that they claim caused their injuries. Therefore, plaintiffs often cast a wide net by suing all known paint and pigment manufacturers claiming that they marketed most of the lead paint pigments used in lead-based paints that were sold in the U.S. between the early part of the twentieth century and the early 1970s, when lead paint pigment was outlawed for residential purposes. But with just a few exceptions, products liability law requires plaintiffs to identify the manufacturer of the allegedly defective product they claim caused their injury in order to prove proximate causation.

573 Id. See also, Keegan v. Kivitz, 2005 WL 2036919 (N.D. Calif. 2005).
575 See, e.g., Jefferson v. Lead Industries Association, Inc., 106 F.3d 1245, 1250 (5th Cir. 1997); see also, Santiago v. Sherwin Williams Co., 3 F.3d 546, 547 (1st Cir. 1993) (noting that “[p]laintiff could not and cannot identify either which, if any, of the defendants are the source of the lead she ingested”); Lewis v. Lead Industries Assn., Inc., 793 N.E.2d 869 (Ill. 2003) (holding that “[b]y failing to identify the defendant that supplied the lead pigment used in the paint to which their children were exposed, the plaintiffs failed to satisfy the causation element of a claim ... for lead poisoning”); Santiago v. Sherwin Williams Co., 3 F.3d 546 (1st Cir. 1993) (plaintiff could not identify which, if any, of the defendants were the source of the lead she ingested or when the alleged injury-causing paint may have been applied to the walls and woodwork of her childhood home).
Consequently, when plaintiffs are unable to make this showing, they often have their case dismissed on summary judgment grounds or later at trial.  

ii. Alternative Theories of Liability

Because plaintiffs could not satisfy the basic standards of products liability law (proving product defect, proximate cause and product identification), they often asked courts to relieve them of that burden by applying some form of various industry-wide theories of liability such as: market-share liability, enterprise liability, alternative liability or civil conspiracy. Courts have generally, but not always, found these theories insufficient to establish causation. Of these theories, most plaintiffs have unsuccessfully asked courts to impose some form of market-share liability. Recently, the Wisconsin Supreme Court held that in spite of the plaintiff’s inability to identify the “precise producer of the white lead carbonate pigment he

See, e.g., Jefferson, 106 F.3d at 1253 (noting that plaintiff's obligation to identify the manufacturer of the allegedly defective product is inherent in the Louisiana Products Liability Act requirements); see also, Cofield v. Lead Indus. Ass’n, No. MJG-99-3277, 2000 WL 34292681 (D. Md. Aug. 17, 2000); Philadelphia v. Lead Indus. Ass’n, 994 F.2d 112, 114, 121-22 (3d Cir. 1993) (stating that the city’s claims for negligence and strict liability were time barred because they accrued in 1976 when Congress enacted federal law regarding lead abatement in federally funded public housing, and the complaint was filed 14 years later); Hymowitz v. Eli Lilly & Co., 539 N.E.2d 1069, 1073 (N.Y. 1989) (explaining that “[i]n a products liability action, identification of the exact defendant whose product injured the plaintiff is, of course, generally required”); Santiago, 3 F.3d at 550-52 (1st Cir. 1993) (holding that under Massachusetts law, plaintiff could not recover under either market share theory of liability or concert in action theory).

See infra section V.A.2.ii(a).

Under enterprise liability there is an “industry-wide standard that is the cause of injury, and each defendant that participates in perpetuating and using the inadequate standard has contributed to and is liable for the plaintiff’s injury.” Thomas v. Mallett, 685 N.W.2d 791, 793 (Wis. Ct. App. 2004). Like market-share liability, this doctrine is used only in a very limited set of circumstances: a small number of manufacturers produced the injury-causing product; virtually all are named defendants; they had joint knowledge of the risks inherent in the product and joint capacity to reduce the risks; and each delegated the responsibility to establish safety standards to a trade association, which failed to reduce the risk. See Philadelphia, 994 F.2d at 126 (declining to adopt enterprise and market-share liability and to extend alternative liability into the field of toxic torts; “[m]arket share liability compromises fairness to defendants who must incur oftentimes staggering litigation costs as they are forced to defend all claims involving their product irrespective of their market share”).

Alternative liability shifts the burden to defendants to exculpate themselves or be held “jointly and severally liable for the plaintiff’s injury.” Philadelphia, 994 F.2d at 127-28.

See Lewis v. Lead Industries Assn., Inc., 2006 WL 701981 (Ill. App. 1st Dist. Feb. 1, 2006). To prevail on a theory of civil conspiracy, a plaintiff must plead and prove: (1) the existence of an agreement between two or more persons; (2) to participate in an unlawful act, or a lawful act in an unlawful manner; (3) an injury caused by an unlawful overt act performed by one of the parties; and (4) that the overt act was done pursuant to and in furtherance of a common scheme. Id. at *4. If the plaintiffs can prove that the defendants were the sole suppliers of the lead pigments which caused the injuries to their children, then it would be of little consequence that they could not identify whose lead caused the injury because civil conspiracy theory has the effect of extending liability for a tortious act beyond the active tortfeasor to individuals who have not acted but have only planned, assisted, or encouraged the act. Id. (citing McClure v. Owens Corning Fiberglass Corp., 188 Ill.2d 102, 133, 645 N.E.2d 888 (1994)).
ingested at his prior residences,” he could still maintain an action against lead pigment manufacturers under a “risk-contribution theory.” Market-share liability and Wisconsin’s risk-contribution theory are discussed in more detail below.

(a) Market-Share Liability

Market-share liability developed in the 1980s as a way to hold defendants liable in litigation against manufacturers of diethylstilbestrol (DES), a drug that caused vaginal and cervical cancer in daughters of women who took it to prevent miscarriage. Under this doctrine, it is unnecessary for a plaintiff to identify the manufacturer of the product that caused the injury. Instead, defendants are held liable for their “market share,” but no more than that amount, even if other defendants are insolvent. Market-share liability is limited to a narrow set of cases meeting particular characteristics: fungible products; a “signature injury” caused by the product; no other medical or environmental factors that could have caused or materially contributed to the harm; enough market-share data to support a reasonable apportionment of liability; a market that is limited in time; the inability of the plaintiff to identify the manufacturer through no fault of her own; and a long latency period between the time the product was used and the time the harm occurred. Courts considering lead paint cases have all rejected market-share liability.

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581 Thomas ex rel. Gramling v. Mallett, 701 N.W.2d 523, 533 (Wis. 2005).
583 Id. at 937.
584 Id. at 936-37.
585 See, e.g., Brenner v. Am. Cyanamid Co., 699 N.Y.S.2d 848, 850 (App. Div. 1999) (explaining that while New York recognizes market-share liability in DES cases, lead litigation did not meet the standards that support dropping the proximate cause requirement in products liability law); Skipworth v. Lead Indus. Ass’n, 665 A.2d 1288, 1291-92 (Pa. Super. Ct. 1995) (“As we find that application of market share liability to lead paint cases would grotesquely distort liability, we decline to apply it in this case.”); Spring Branch Indep. Sch. Dist. v. NL Indus., Inc., No. 01-02-01006-CV, 2004 WL 1404036, at *4 (Tex. App. June 24, 2004) (rejecting market-share liability and stating that plaintiff school district’s position “disregards the bedrock principle of Texas law that a plaintiff must identify the manufacturer of the product that allegedly injured it”).
iii. Risk- Contribution Theory – Absolute Liability Comes to Wisconsin

In 2005, in Thomas ex rel. Gramling v. Mallett, the Wisconsin Supreme Court reversed a summary judgment motion allowing a childhood lead paint claim to go forward to trial against lead-pigment manufacturers despite the plaintiff’s inability to identify which manufacturers caused his injury.\(^{586}\) It reversed the summary judgment by creating a new “risk-contribution theory” that is applicable in cases against the lead paint industry in Wisconsin. The dissent in this case described this as a type of absolute liability by creating an irrebuttable presumption of causation.\(^{587}\)

The plaintiff in this case lived in three different Milwaukee homes during the early 1990s and allegedly sustained lead poisoning by ingesting lead from paint chips, flakes and dust in the homes. During litigation, he received settlements from two of his three landlords and pursued claims against seven lead-paint pigment manufacturers—conceding, however, that he could not causally link any specific manufacturer to his injury. Consequently, the trial court dismissed his negligence and strict liability claims against the product manufacturers.\(^{588}\) The Wisconsin Supreme Court reversed, becoming the first court in the nation to allow such a case to go forward by eliminating the causation requirement in lead-paint cases in favor of a form of collective liability based on mere participation in the lead-pigment industry.

As applied to the lead-paint industry, Wisconsin’s new risk contribution theory very likely will function as a form of absolute liability. In lead-paint cases (as contrasted to Wisconsin’s use of market-share liability in DES cases) the opportunity for the defendant

\(^{586}\) Thomas ex rel. Gramling v. Mallett, 701 N.W.2d 523 (Wis. 2005).

\(^{587}\) Id. at 576.

\(^{588}\) In affirming summary judgment to the defendants, the Circuit Court declined to apply market-share liability because under Wisconsin law it is imposed only when the plaintiff has no other remedy. Here, the plaintiff has settled with two of his landlords. Thomas ex rel. Gramling v. Mallett, 685 N.W.2d 791, 795 (Wis. Ct. App. 2004) (distinguishing this case from Collins v. Eli Lilly Co., 342 N.W.2d 37 (1984)).
manufacturers to exculpate themselves is almost nonexistent.\textsuperscript{589} The majority in \textit{Thomas} made it clear that the relevant time period for lead-paint risk contribution liability is not the time period of the plaintiff’s exposure but the entire time period each house with lead paint existed. In \textit{Thomas}, the lead paint present in the three houses where the plaintiff lived could have been applied at any time between 1900 and 1978.\textsuperscript{590} Apportioning risk contribution liability among manufacturers of lead pigment based on market share and relative culpability over a 78-year period of time is nearly impossible as a purely factual matter. As one commentator has noted:

This is, then, a form of collective tort liability untethered to any actual responsibility for the specific harm asserted, imposed by the judiciary as a matter of loss-distribution policy in response to an admittedly serious public health problem. As Justice Wilcox observed in his dissent, “[t]he end result of the majority opinion is that the defendants, lead pigment manufacturers, can be held liable for a product they may or may not have produced, which may or may not have caused the plaintiff’s injuries, based on conduct that may have occurred over 100 years ago when some of the defendants were not even part of the relevant market.”\textsuperscript{591}

This decision has reverberated throughout Wisconsin’s business community which fears that the decision will have drastic, perhaps staggering, consequences for Wisconsin industry and commerce in all manner of product-involved materials.\textsuperscript{592}

In response to the \textit{Thomas} decision, the Wisconsin State Senate introduced Senate Bill 402, which would have eliminated the newly expanded risk contribution theory in Wisconsin and restored a plaintiff’s burden to prove causation against particular defendants. On January 6,\textsuperscript{592}

\textsuperscript{589} In DES cases each drug company had (at least in theory) a meaningful opportunity to defend against liability by proving it did not produce or market the drug either where the plaintiff lived or during the specific nine-month period she was exposed. \textit{See also}, Diane S. Sykes, \textit{Reflections on the Wisconsin Supreme Court}, Hallows Lecture, Marquette University Law School 11 (March 7, 2006), \textit{available at} http://204.11.208.101/s3/site/images/alumni/HallowsLecture.pdf (last visited Aug. 10, 2006).

\textsuperscript{590} \textit{Thomas ex rel. Gramling v. Mallett}, 701 N.W.2d at 562-63.

\textsuperscript{591} \textit{See} Sykes supra note 589, at 11.

\textsuperscript{592} \textit{See} Wisconsin Manufacturers & Commerce, Priority Issues (2006), \textit{available at} http://www.wmc.org/governmentaffairs/display.cfm?ID=1155 (last visited Aug. 10, 2006). “[T]he Thomas decision represents a radical departure from longstanding and fundamental tort law requirements of proof.” “Consequently, under the Thomas ruling, a completely innocent manufacturer could be forced to pay damages for harm caused by some other manufacture’s similar product. It is an expensive ‘guilty even if you’re innocent’ standard that is not recognized in other states.” \textit{Id.}
2006, Wisconsin Governor Jim Doyle vetoed the bill claiming that signing it into law would “close the doors of justice” to “poisoned children.”

B. THE NEW WAVE: THE COMMON LAW OF PUBLIC NUISANCE

Recently, some state attorneys general and personal injury lawyers have been trying to convert the tort of public nuisance into a cutting edge legal theory. They are attempting to move public nuisance law far outside its traditional boundaries by using it to sue product manufacturers in an effort to circumvent the well-defined structure of products liability law.

The tort of public nuisance has developed over nine centuries of English and American common law. Its essence is to allow governments to use the tort system to stop quasi-criminal conduct that, while not illegal, is unreasonable given the circumstances and could cause injury to someone exercising a common, societal right. The traditional public nuisance involves blocking a public roadway or, in recent times, dumping sewage into a public river or blasting a stereo when people are picnicking in a public park. Under public nuisance theory, the government may seek an injunction to stop the activity causing the public nuisance or force the party to abate the public nuisance itself. Public nuisance theory also specifies the types of parties who may sue and which remedies each type of party may seek. Namely, the government may only seek injunction or abatement, not monetary damages. Individuals who have sustained a particular injury, such as harm to one’s personal property from the public nuisance, can use the tort to seek

593 “Nuisance has been described as an ‘impenetrable jungle.’” W. Page Keeton et al., PROSSER & KEETON, LAW OF TORTS (5th ed. 1984) §86, p. 616. “[Nuisance] 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.” Id.

594 See Victor E. Schwartz and Phil Goldberg, THE LAW OF PUBLIC NUISANCE: MAINTAINING RATIONAL BOUNDARIES ON A RATIONAL TORT, 45 WASHBURN L. J. 541-83 (2006) [hereinafter Schwartz]. For the sake of brevity, and because of the authors’ great respect for Messrs. Schwartz and Goldberg’s outstanding scholarship, much of the information related in this section is condensed and paraphrased from their superb article.

595 RESTATEMENT (SECOND) OF TORTS §821A cmt. b (1979). The term frequently is used in several different senses. In popular speech it often has a very loose connotation of anything harmful, annoying, offensive or inconvenient, as when it is said that a man makes a nuisance of himself by bothering others. Occasionally this careless usage has crept into a court opinion. If the term is to have any definite legal significance, these cases must be completely disregarded. Id.
compensatory damages. Public nuisance theory was not developed to allow private citizens the power to stop or abate conduct, to allow government to grow its coffers, to spread the risk of an enterprise, or to punish defendants.

The current effort to expand public nuisance theory to provide sanctions against manufacturers of lawful products is disconcerting because it attempts to fundamentally change the entire character of public nuisance doctrine, as well as undermine products liability law. Not surprisingly, the targets involve “unpopular” products, such as asbestos, guns, tobacco, lead paint, the gasoline additive methyl tertiary butyl ether (MTBE), and others. Most courts have rejected these suits, stating that public nuisance theory has always targeted how properties or products are used, not manufactured. Nevertheless, a few courts have broken from traditional public nuisance theory and have allowed these cases to proceed. Whether those rulings remain an aberration, thereby following the pattern of previous divergences from traditional public nuisance theory, has yet to be determined.

1. The Development of Public Nuisance Law

   a. English Common Law

   Public nuisance goes back to twelfth-century English common law. At that time, it was a tort-based crime for infringing on the rights of the Crown. In the fourteenth century, English courts extended the principle of public nuisance beyond the rights of the Crown to include rights

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596 Schwartz, supra note 596 at 542. Unlike government plaintiffs, private individuals cannot seek injunction or abatement. Other members of the general public, even if inconvenienced by the public nuisance, cannot use the tort at all.

597 See, e.g., RESTATEMENT (SECOND) OF TORTS §402A cmt. g (1979) (stating that there is no liability to a manufacturer when the product is delivered in a safe condition); RESTATEMENT (THIRD) OF TORTS: PRODS. LIAB. §2 cmt. d (1998) (rejecting category liability).

common to the public,\textsuperscript{599} such as “the right to safely walk along public highways, to breathe unpolluted air, to be undisturbed by large gatherings of disorderly people and to be free from the spreading of infectious diseases.”\textsuperscript{600}

It was not until 1535 that an English court first allowed individuals to sue and recover damages under the doctrine. The case involved the blocking of a highway and set the precedent that an individual who had suffered “particular damages” could file a public nuisance suit to recover those damages.\textsuperscript{601} The individual’s injury must have been different in kind, not simply more severe than the injury to the public as a whole, and the individual could not sue for injunction and abatement because those actions were reserved solely for the Crown.\textsuperscript{602}

\textbf{ii. Public Nuisance in the United States}

Historically, American public nuisance cases involved non-trespassing invasions of the public use and enjoyment of land. In the eighteenth and early nineteenth centuries, most public nuisance cases involved the obstruction of public highways and waterways, though some involved using property in ways that conflicted with public morals or social welfare.\textsuperscript{603} With the onset of the Industrial Revolution in the 1840s came the first test of public nuisance theory

\begin{itemize}
\item \textsuperscript{599} \textit{RESTATEMENT (SECOND) OF TORTS} §821B cmt. a (1979).
\item \textsuperscript{600} Joseph W. Cleary, \textit{Municipalities Versus Gun Manufacturers: Why Public Nuisance Claims Just Do Not Work}, 31 U. BALT. L. REV. 273, 277 (2002). Although the Crown primarily used public nuisance against those who interfered with a public right of way or operated “noisome trades,” the apparent flexibility of public nuisance led to its use against numerous activities such as the following: “digging up a wall of a church,” helping a “homicidal maniac” to escape, being a common scold, keeping a tiger in a pen next to a highway, leaving a mutilated corpse on a doorstep, selling rotten meat, embezzling public funds, keeping treasure trove, and subdividing houses which “become hurtful to the place by overpestering it with poor.” Abrams & Washington, \textit{supra} note 598.
\item \textsuperscript{601} William A. McRae Jr., \textit{The Development of Nuisance in the Early Common Law}, 1 U. FLA. L. REV. 27, 36 (1948) (“Though this view eventually prevailed, it was not accepted without dissent, the dissent being that a public offence should not give rise to a private right”).
\item \textsuperscript{602} F.H. Newark, \textit{The Boundaries of Nuisance}, 65 L.Q. REV. 480, 483 (1949). Justice Fitzherbert, one of the judges hearing the case, illustrated the difference between injury-in-kind and injury-in-degree through the example of a person riding on a public highway at night and coming across a man-made ditch. If the rider were delayed or inconvenienced—no matter by how much or what the consequences—his injury would be the same as that sustained by anyone else. But, if his cart were harmed by falling into the ditch, he would have sustained damage that was different in kind from the general public. William L. Prosser, \textit{Private Action for Public Nuisance}, 52 VA. L. REV. 997, 1005 (1966).
\item \textsuperscript{603} Donald G. Gifford, \textit{Public Nuisance as a Mass Products Liability Tort}, 71 U. CIN. L. REV. 741, 800-01 (2003). These cases often involved gambling halls, taverns, or prostitution houses. \textit{Id.} at 801.
\end{itemize}
boundaries in the U.S. In the absence of significant regulation, public nuisance became a substitute for governments that “could not anticipate and explicitly prohibit or regulate through legislation all the particular activities that might injure or annoy the general public.”

After the New Deal movement in the 1930s, with the expansion of “comprehensive statutory and regulatory schemes” determining acceptable societal behaviors, public nuisance theory was not necessary to define societal boundaries and largely faded from American jurisprudence. In fact, when the first Restatement of Torts was published in 1939, it did not even include a reference to the tort of public nuisance.

When the Restatement (Second) of Torts was finished, it contained public nuisance, thereby codifying 900 years of history. During the debate on public nuisance, some in the environmental community sought a relaxation of the strict binds of public nuisance law. The first issue involved the type of conduct required in public nuisance theory. Traditionally, public nuisance was considered quasi-criminal in nature. Environmentalists, however, unsuccessfully fought this quasi-criminal standard because they wanted to use public nuisance theory to combat pollution that was not subject to criminal sanctions, and in fact, was often

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604 Id. at 804. For example, water pollution suits against companies for industrial run-off often succeeded because polluting a river was akin to the obstruction of a public waterway. By contrast, claims filed against railroads for noise and air pollution affecting the communities near the tracks often failed. “Where the operation of the railroad was pursuant to a legislative charter or license and the operation of the railroad was in accordance with the expectations of the legislature,” there was no conflict with a public right. Id. at 802-03.

605 Id. at 805-06 (“A principal reason was . . . the development of comprehensive statutory and regulatory schemes that . . . substituted other means of regulation for many former targets of public nuisance prosecutions”).

606 Id. at 806. According to some accounts, nuisance law was assigned to the Restatement of Property during the first Restatement because of its grounding in property. The drafters of the Restatement of Property only focused on private nuisance, so that when nuisance was transferred to torts, public nuisance was not included. “In fact, the tort lawyers and tort professors at work on the RESTATEMENT (FIRST) OF TORTS treated nuisance as though it were solely an issue of interference with private property rights, that is, an invasion of interests in the private use of land.” Louise A. Halper, Untangling the Nuisance Knot, 26 B.C. ENVTL. AFF. L. REV. 89, 120-21 (1998).

607 Schwartz, supra note 596 at 547. As Dean Prosser wrote in 1966, “[a] public or ‘common’ nuisance is always a crime . . . a species of catch-all low-grade criminal offense, consisting of an interference with the rights of the community at large, which may include anything from the blocking of a highway to a gaming-house or indecent exposure.” Id. (citing Prosser, supra note 16, at 997, 999). But see Gifford, supra note 603, at 781 (disagreeing with Prosser and suggesting that public nuisances were not always criminal actions); Halper, supra note 606, at 118 (paraphrasing Judge Benjamin Cardozo as stating that “where a use is not in itself unlawful or hazardous, negligence is the appropriate liability standard for the injuries attributable to that use”).
permitted by federal, state, or local regulatory regimes or zoning regulations. As a compromise, the Restatement suggested that conduct need only be an “unreasonable interference” with a public right. However, the Restatement clarified that when a defendant’s conduct at issue “does not come within one of the traditional categories of the common law crime of public nuisance or is not prohibited by a legislative act, the court is acting without an established and recognized standard.” In addition, environmentalists wanted individuals and organizations to have standing to bring private attorney general-type actions to enjoin or abate a public nuisance. Environmentalists also sought to expand standing in private compensatory suits to include anyone affected by the public nuisance, not just those who suffered an injury different-in-kind from the general public. There efforts failed, and the Restatement maintained the difference-in-kind requirement.

Undeterred, in the early 1970s, environmental activists attempted to gain acceptance for these new theories by filing a purported public nuisance class action against scores of companies alleged to have contributed to air pollution in Los Angeles, California, seeking injunctive relief

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609 *Restatement (Second) Of Torts* §821B cmt. e (1979). The Restatement provides the following factors to determine whether an activity unreasonably interferes with a common right:

(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* at §821B(2).

610 *Id.*, §821B cmt. e

611 To accommodate that concern, the Restatement suggested that individuals could have standing if they were suing “as a representative of the general public, as a citizen in a citizen’s action or as a member of a class in a class action.” *Id.*, §821C(2)(c).
in addition to compensatory and punitive damages. The California court rejected the lawsuit, denying class certification and reasoning that public nuisance theory is ill-suited for this type of litigation. The Court held similarly in the well-publicized case of Alaska Native Class v. Exxon Corp., which stemmed from the environmental damage caused by Exxon’s oil spill off the coast of Alaska. In that case, the plaintiffs (private citizens whose lives were upended by the spill because they could no longer fish in the area waters) filed a public nuisance claim to collect cultural damages associated with the loss of their “subsistence way of life.” Although the court sympathized with the impact the spill had on the local community, it held that the plaintiffs failed to prove any particular injury – a core requirement in public nuisance theory for a private cause of action.

Courts have, however, on occasion, broken the bounds of public nuisance law to meet a desired end. For example, in the famous Love Canal cases in the mid-1980s, the court allowed a public nuisance action to proceed against a company that had not engaged in the act of pollution and never owned or controlled the land where the pollution took place. While the court

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612 Diamond v. General Motors Corp., 97 Cal. Rptr. 639, 639 (Ct. App. 1971) (seeking an injunction against 293 named corporations and municipalities, as well as 1,000 unnamed defendants, for air pollution).

613 Id. at 642-46. The court noted that regulating activities that are not criminal in nature, such as manufacturing a lawful product, is the province of the legislature, not the judiciary. As the court also noted, the “plaintiff is simply asking the court to do what the elected representatives of the people have not done: adopt stricter standards over the discharge of air contaminants in this county, and enforce them with the contempt power of the court.” Id. at 645. The court also recognized that the power of injunctive relief in public nuisance cases is only appropriate when wielded by the government, which is accountable to the public as a whole and can assess the societal value of the competing activities. In this case, as the court pointed out, “[t]he immediate effect of . . . an injunction would be to halt the supply of goods and services essential to the life and comfort of the persons whom plaintiff seeks to represent.” Id. at 644. Finally, the court found that massive public nuisance class actions are not appropriate because the plaintiffs would have to claim that each member of the class sustained particular damages from the pollution in order to seek compensatory and punitive damages for the class. The court stated that “[r]equire[ing] plaintiff to state separately the seven million causes of action, and to plead factually the damage as to each, would in and of itself constitute a practical bar to this action.” Id. at 643.

614 104 F.3d 1196 (9th Cir. 1997).

615 Id. at 1197. Noting that “[w]hile the oil spill may have affected Alaskan Natives more severely than other members of the public, the right to [their culture and lifestyle] is shared by all Alaskans.” Id. at 1198.

acknowledged that deciding who should pay for the clean up “is essentially a political question
to be decided in the legislative arena,” it “[n]onetheless” allowed the public nuisance claims to
proceed, with the surprising and open-ended observation that “[s]omeone must pay to correct the
problem.” Likewise, in 1982, the Supreme Court of Hawaii in Akau v. Olohana Corp.
replaced the traditional “particular injury” rule with an “injury in fact” test for claims brought by
private parties. Under this ruling, anyone injured or inconvenienced by a public nuisance,
regardless of the degree or kind of injury, could file a class action for injunctive relief and
abatement. The Hawaii ruling still stands alone almost 20 years later as the only state court
decision expressly to abandon the traditional special injury rule. Other courts have rejected
this “end-justifies-the-means” approach to public nuisance displayed by the New York lower
court in Love Canal and the Supreme Court of Hawaii in Akau.

2. Elements of a Public Nuisance

i. Type of Injury

The tort of public nuisance requires proof that the injury is to “a right common to the
general public.” According to the Restatement (Second) of Torts, “[a] public right is one
common to all members of the general public. It is collective in nature and not like the

617 Id. at 977.
618 652 P.2d 1130, 1134 (Haw. 1982) (allowing a class action against a private company for interfering with public
trails).
619 Id. at 1133-34. The rationale the court provided for creating such a “liberal standing” requirement was based “not
in nuisance,” but in the increasing use of relaxed standing trends in other types of cases, such as taxpayer suits for improper
expenditure of public funds, challenges to administrative decisions and claims of harm to public trust property. Id.
620 See Antolini, supra note 608, at 786.
621 See, e.g., U.S. Steel Corp. v. Save Sand Key, Inc., 303 So. 2d 9, 13 ( Fla. 1974) (stating that a nonprofit citizens' group
could not seek to enjoin a steel corporation from using portions of the soft sand beach area of Sand Key), see also, Antolini, supra note 608, at 776-86.
622 Hydro-Mfg., Inc. v. Kayser-Roth Corp., 640 A.2d 950, 958 (R.I. 1994) (emphasis added) (citations omitted). This
notion of a specific type of injury for a specific cause of action is not uncommon in American law. For example, to bring a suit
in antitrust law, a plaintiff “must prove antitrust injury, which is to say injury of the type the antitrust laws were intended to
prevent and that flows from that which makes defendants’ acts unlawful.” Cargill, Inc. v. Monfort of Colo., Inc., 479 U.S. 104,
individual right that everyone has not to be assaulted or defamed or defrauded or negligently injured.\textsuperscript{623} Not all interferences with public rights are public nuisances. The nuisance must also produce a common injury, or be dangerous or injurious to the general public.\textsuperscript{624}

The key inquiry is whether a person exercising a common right would be injured if she came into contact with the offending conduct. Consider the quintessential public nuisance claim against a party for blocking a public road. Blocking a public road interferes with the public right to drive on that road. Thus, a government could seek an injunction to stop the blockage even if no one actually encountered the blockage. Conversely, if the party blocked the entrance to someone’s home, a commercial shopping plaza, or a church, no public right would be violated, and the state would not have a public nuisance claim even if the blockage caused an injury.\textsuperscript{625}

Some personal injury lawyers have attempted to confuse public nuisance injury with an injury sufficient to bring a private nuisance claim, which is a wholly separate and distinct tort from public nuisance claim. Unlike public nuisance cases, private nuisance claims are always between private parties and always center on conflicting uses of private land. The injury is the intentional invasion of a person’s property that interferes with that person’s use of the property. Courts, in reaching an equitable resolution, weigh the harm caused by the defendant’s conduct against its social utility. There is no public right or collective harm involved.\textsuperscript{626}

\textsuperscript{623} \textit{Restatement (Second) Of Torts} §821B cmt. g (1979) (“[T]he pollution of a stream that merely deprives fifty or a hundred lower riparian owners of the use of the water for purposes connected with their land does not for that reason alone become a public nuisance. If, however, the pollution prevents the use of a public bathing beach or kills the fish in a navigable stream and so deprives all members of the community of the right to fish, it becomes a public nuisance.”).

\textsuperscript{624} 58 Am. Jur. 2d Nuisances §39 (2002).

\textsuperscript{625} Schwartz, \textit{supra} note 596, at 562-63. As in this example, a public nuisance injury traditionally involved the misuse of real property. While land use is still the foundation of public nuisance law, some courts have stretched public nuisance to include other types of conduct that conflict with a public right, such as a common right to “the health, safety, peace, comfort or convenience of the general community.” Either way, as most courts have held, this communal-based injury is wholly distinguishable from personal injuries that give rise to product-based suits. \textit{Id.} at 563 (citations omitted).

\textsuperscript{626} Schwartz, \textit{supra} note 596 at 564. As Dean Prosser succinctly observed, the two “are quite unrelated except in the vague general way that each of them causes inconvenience to someone” and the two share a “common name.” \textit{Id.} (citing Prosser, \textit{supra} note 602, at 999.)
ii. Type of Conduct

(a) Common Law

Historically, the level of offense required in public nuisance claims has been comparable to quasi-criminal activity.\(^627\) As part of the compromise reached during its drafting, the Restatement (Second) of Torts suggests lowering the standard to any “unreasonable interference” with a public right. The factors for determining unreasonableness would be: whether the interference is significant; whether there is a statute proscribing or prohibiting the conduct; and whether the harm is of a continuing, long-lasting nature and the defendant knows its conduct has a “significant effect” on this ongoing harm.\(^628\)

Generally speaking, courts have encountered four categories of conduct in common law public nuisance claims: (i) unlawful, intentional acts; (ii) lawful conduct involving conflicting uses of property; (iii) lawful conduct, not involving the use of land that leads to unintended consequences; and (iv) otherwise tortious conduct. In examining the conduct, “the role of ‘creator’ of a nuisance, upon whom liability for nuisance-caused injury is imposed, is one to which manufacturers and sellers seem totally alien.”\(^629\) Traditionally, public nuisance law has not supported recovery simply because the “manufacture and sale of a product [was] later discovered to cause injury.”\(^630\)

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\(^{627}\) Public nuisance is “a species of catch-all criminal offense[s].” Keeton, supra note 593, at 618. When the manufacturer or seller is engaged in lawful activity, particularly a well-regulated activity, an injunction may violate the commerce clause of the U.S. Constitution. Schwartz, supra note 596, at 564n.150.


\(^{630}\) Id.
(b) Statutes and Regulations

Through legislation, regulation, or ordinance, state and local governments often define specific activities as being a public nuisance. When a field of conduct is well regulated, conduct that might be categorized as unreasonable under common law may become non-tortious. In these instances, courts accept that the legislative or regulatory body has determined that such conduct is acceptable to society and is therefore not unreasonable.

(c) Control

If public nuisance injury and conduct exist, the “paramount” issue becomes whether the element of control can be satisfied with respect to each defendant. As a Rhode Island court observed, “[i]f the defendants exercised no control over the instrumentality, then a remedy directed against them is of little use.”

Historically, when public nuisance cases involved property, the party who controlled the public nuisance was the party who owned or operated the property at the time of abatement. This is because the “inability to allege that the defendants ha[ve] a legal right to abate the nuisance is fatal to [a] nuisance claim.” Thus, as a general rule, former landowners, even ones who created the nuisance, would not be liable for abatement because they could no longer gain

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631 The only means of pursuing a public nuisance claim for such lawful conduct would be to show that “the law regulating the defendant’s enterprise is invalid.” Chicago, 821 N.E.2d at 1124.

632 For example, if an environmental statute provides for a maximum amount of pollution that a factory can emit, then a company would not be subject to liability under public nuisance theory for emissions that are within those limits. Schwartz, supra note 596 at 566-67.


634 Manchester, 637 F. Supp. at 656.

635 Detroit Bd. of Educ. v. Celotex Corp., 493 N.W. 513, 521-22 n.8 (Mich. Ct. App. 1992) (stating that “[L]iability of a possessor of land is not based upon responsibility for the creation of the harmful condition, but upon the fact that he has exclusive control over the land and the things done upon it and should have the responsibility of taking reasonable measures to remedy conditions on it that are a source of harm to others.”).

636 Manchester, 637 F. Supp. at 656.
access to the land. The exception to this rule occurs in the environmental world where statutes require the polluter to pay for their equitable share of the clean-up. Under any method of assessing control, there is no doubt that product manufacturers no longer have control over a product after it is sold.

Courts have applied this bedrock rule to preclude the liability of manufacturers of products, including lead pigment, in public nuisance lawsuits. In City of Chicago v. Am. Cyanamid Co., for example, the Illinois Court of Appeals held:

These public policy concerns dictate that legal cause cannot be established with respect to defendants in the present case that produced a legal product decades ago that was used by third parties who applied the product to surfaces in Chicago . . . . We therefore hold that the conduct of defendants in promoting and lawfully selling lead-containing pigments decades ago, which was subsequently used lawfully by others, cannot be a legal cause of plaintiff’s complained-of injury, where the hazard only exists because Chicago landowners continue to violate laws that require them to remove deteriorated paint.

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637 See, e.g., Maisenbach v. Buckner, 272 N.E.2d 851, 854 (Ill. App. Ct. 1971) (“Where a landowner clearly has no right to control the property after he sells it to another, he likewise can have no duty to third persons injured in connection with the property after the sale.”); RESTATEMENT (SECOND) OF TORTS §839 cmt. d (1979) (“[A] vendee or lessee of land upon which a harmful physical condition exists may be liable under the rule here stated for failing to abate it after he takes possession, even though it was created by his vendor, lessor or other person and even though he had no part in its creation.”).


639 823 N.E.2d 126, 139 (Ill. Ct. App. 2005), app. den. 833 N.E.2d 1 (Ill. 2005); see also, Young v. Bryco Arms, 821 N.E.2d 1078, 1091 (Ill. 2004) (“[D]efendants’ conduct is not a legal cause of the alleged nuisance because the claimed harm is the aggregate result of numerous unforeseeable intervening criminal acts by third parties not under defendants’ control”); District of Columbia v. Beretta, U.S.A., Corp., 872 A.2d 633, 650 (D.C. Ct. App. 2005), cert. denied, 126 S.Ct. 399 (2005) (“[T]he alleged public nuisance is not so foreseeable to the dealer defendants that their conduct can be deemed a legal cause of a nuisance that is the result of the aggregate of the criminal acts of many individuals over whom they have no control”) (emphasis in the original) (internal quotations omitted); cf. Martinez v. Pacific Bell, 225 Cal. App. 3d 1557, 1565 (Cal. Ct. App. 1990) (finding that robbery was intervening cause to alleged nuisance that purportedly drew criminal activity to area).
(d) Proximate Causation

Finally, the defendant’s conduct must have proximately caused the public nuisance.\(^{640}\) The proximate cause analysis in public nuisance theory is the same as with claims for traditional negligence. A plaintiff must show that the conduct that created the public nuisance was foreseeable and satisfies the doctrine of remoteness.\(^{641}\) Thus, the injury to the plaintiff must be the type of injury that a reasonable person would see as a likely result of his/her conduct.

In determining proximate cause, acts of third parties are intervening events that may cut off proximate cause.\(^{642}\) Courts distinguish between tortious acts, such as creating a public nuisance, and benign acts, such as furnishing a condition upon which a tortfeasor acts. Generally, a party is not liable unless it “increase[s] an unreasonable risk of harm” caused by the intervening event.\(^{643}\) This is a key reason why public nuisance theory has failed in most gun and lead paint claims.\(^{644}\)


\(^{641}\) Under the doctrine of remoteness, plaintiffs alleging “harm flowing merely from the misfortunes visited upon a third person by the defendant’s acts [are] generally said to stand at too remote a distance to recover.” Holmes v. Sec. Investor Prot. Corp., 503 U.S. 258, 268-69 (1992). “Remoteness is an aspect of the proximate cause analysis, in that an injury that is too remote from its causal agent fails to satisfy tort law’s proximate cause requirement . . . .” Steamfitters Local Union No. 420 Welfare Fund v. Philip Morris, Inc., 171 F.3d 912, 921 (3d Cir. 1999).

\(^{642}\) John L. Diamond, CASES AND MATERIALS ON TORTS 256 (2001) (“An intervening force is one which joins with the defendant’s conduct to cause the injury. Such a force, whether it be human, animal, mechanical, or natural is considered intervening because it occurs after the defendant’s conduct. An intervening force will only act to cut off proximate cause if it is characterized as superseding. . . . [W]hile courts are quick to find negligence of a third party foreseeable and hence not superseding, criminal acts are often characterized as extraordinary unforeseeable and hence superseding.”).

\(^{643}\) Keeton, supra note 593, at 305.

\(^{644}\) For example, in Chicago v. Beretta, the court stated that there is no proximate cause if the tortfeasor could have created the nuisance without the conduct of the manufacturer. Thus, if the creator of the nuisance could have purchased the product from another vendor, the retailer or manufacturer could not be deemed to have proximately caused the nuisance. 821 N.E.2d at 1127 (defining proximate cause in a public nuisance case as “whether the injury is of a type that a reasonable person would see as a likely result of his conduct”). In addition, the defendant would have to foresee which individual sales or behaviors would lead to the creation of the particular nuisance at issue.
iii. Government as Plaintiff

Traditionally, when a governmental entity serves as the plaintiff and is suing in its role as the sovereign, the only appropriate remedies available are an injunction or abatement. Litigation in which a government uses public nuisance theory against product manufacturers for the costs of police protection or other government programs raises two key problems. First, it violates well-established public nuisance remedies by seeking monetary compensation. “Even when it acts in the name of public health, the state is not the party who has suffered the special damages being sought.” Second, the public services doctrine prevents the costs associated with the performance of governmental functions from being recoverable in tort. The costs of police protection, government abatement programs and other similar services are borne by the public as a whole and cannot be assessed against an individual tortfeasor.

(a) Profitability or Public Interest: The States’ Use of Contingency-Fee Private Counsel

Since the “success” of the tobacco litigation, many states have considered entering into contingency fee contracts with private plaintiff law firms. There are obvious incentives for doing so. Private counsel are not salaried government employees, but private plaintiffs’ lawyers whose standard compensation method is the contingency fee. In private litigation, the plaintiff lawyers sometimes advance expenses associated with maintaining and prosecuting the lawsuit, subject to later reimbursement by the client. If such an agreement is reached with a sovereign state, the state might pay nothing for their services unless they win at trial or obtain a lucrative settlement, and reimbursement of expenses could be delayed substantially. The fee contemplated in this type of arrangement, subject to the approval of the court, perhaps could be deemed a

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645 See Restatement (Second) Of Torts §821B cmt. a (1979) (explaining that public nuisance law started solely as an action by the state—through the King’s sheriff, the equivalent of the modern state attorney general—to stop a private party from invading a public right).

646 Schwartz, supra note 596, at 570.
proper, reasonable and customary contingency fee. If approved, the agreement is a “win-win” situation for the state! Or is it? Ultimately, using private counsel raises many ethical issues such as hiring friends and campaign contributors (political cronyism) or whether the state and the “assisting” private counsel are pursuing the lawsuit in the public interest or for profitability.

Private counsel hired by states to pursue litigation, are often referred to as “Special Assistants.” Although these Special Assistants are hired by the states, they are not considered state employees. State attorneys general routinely hire outside counsel for help in prosecuting cases, but these outside attorneys are normally paid by the hour. Contingent fee deals with outside counsel, however, introduce a profit motive to the state-sponsored litigation. The use of

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647 Contingency deals raise the question of whether State attorneys general are pursuing the public interest or merely rewarding campaign donors with lucrative business. For example, in 2001, West Virginia Attorney General Darrell McGraw used four outside law firms – three based outside of the state – to sue Purdue Pharmaceuticals, the maker of OxyContin. The lawsuit was brought on behalf of the state’s workers’ compensation fund and other state agencies. But when Purdue settled the case for $10 million, a third of the money went to the lawyers instead of the state. The one in-state law firm used by Mr. McGraw turned out to be a major contributor to his re-election campaign. Editorial, Rhode Island Rhapsody, WALL. ST. J. A10 (Aug. 16, 2006). In 1996, then-Attorney General Carla Stovall of Kansas hired her former law partners at Entz & Chanany to serve as local counsel in the State’s tobacco lawsuit. See Hearing on H.B. 2893, before the Kansas House Taxation Comm., Feb. 14, 2000, at 16 (testimony of Carla Stovall, Attorney General of Kansas), available at http://kslegislature.org/committeeminutes/2000/house/HsTax2-14-00b.pdf (last visited Sept. 10, 2006). Attorney General Stovall testified that she asked her former law firm to take the case “as a favor” in part due to their “personal loyalty.” Id. at 17. In addition to accepting the case against big tobacco, Entz & Chanany performed other “favors” for General Stovall during her campaign. They housed Ms. Stovall’s Attorney General campaign. Id. at 16. They also contributed money to her campaign effort. See John L. Peterson, Payment for Law Firm Draws Fire; Hearing Continues In Case Involving Tobacco Litigation, B3 KANSAS CITY STAR, (Feb. 17, 2000). In Texas, then-Attorney General Dan Morales hired contingency fee lawyers to file his state’s tobacco litigation in 1996. Four of the five hired firms together had contributed nearly $150,000 in campaign contributions to Morales from 1990 to 1995. See Robert A. Levy, The Great Tobacco Robbery: Hired Guns Corral Contingent Fee Bonanza, LEGAL TIMES, 27 (Feb. 1, 1999). After hiring the firms, Morales reportedly asked them to make an additional political contribution of $250,000. See Miriam Rozen & Brenda Sapino Jeffreys, Why Did Dan Morales Exchange Good Judgment for the Good Life?, TEX. LAW 1 (Oct. 27, 2003). The eight-year Attorney General, former state representative and prosecutor was ultimately sentenced to four years in federal prison for attempting to funnel millions of dollars worth of legal fees to a long-time friend who did little work on the case. See John Moritz, Morales Gets 4 Years in Prison, FT. WORTH STAR TELEGRAM, 1A (Nov. 1, 2003). In Connecticut, Attorney General Richard Blumenthal selected four firms to represent the state in the tobacco litigation from a list of sixteen firms that expressed interest in that representation. As reported in the local media, the three Connecticut-based firms selected included: (1) General Blumenthal’s own former law firm, Silver, Golub & Teitell, (2) Emmet & Glander in Stamford, whose name partner, Kathryn Emmet, is married to partner David Golub of Silver, Golub & Teitell; and (3) Carmody & Torrance of Waterbury, whose managing partner, James K. Robertson, served as personal counsel and counselor to Governor John Rowland. See Thomas Scheffey, Winning the $65 Million Gamble, CONN. L. TRIB. 1 (Dec. 6, 1999). Other firms that wanted to be considered for the litigation publicly stated they did not have a fair chance at the contract. For example, Robert Reardon of New London, a former president of the Connecticut Trial Lawyers Association, reportedly could not even get in the door for a meeting. See id.


contingent fees in the hiring of outside counsel emerged as a trend beginning in the mid-1990s with lawsuits against “Big Tobacco.” In addition to generating hundreds of millions in fees for the private lawyers involved, tobacco litigation pioneered a new model for state-sponsored litigation that combines the prosecutorial power of the government with private lawyers aggressively pursuing litigation that has the potential to generate hundreds of millions – or billions – of dollars in contingent fees.⁶⁵⁰

(1) What is in the Public Interest?

According to the United States Supreme Court, a government attorney’s duty is not necessarily to prevail, or to achieve the maximum recovery in a particular case; rather, “the Government wins its point when justice is done in its courts.”⁶⁵¹ A government attorney “is the representative not of an ordinary party to a controversy, but of a sovereignty whose obligation to govern impartially is as compelling as its obligation to govern at all,” and therefore the government attorney is required to use the power of the sovereign to promote justice for all citizens.⁶⁵²

Unlike “special assistants,” attorneys general and assistant attorneys general take an oath to “faithfully and impartially discharge the duties of the office of the Attorney General” and to “support the Constitution and laws of this state, and the Constitution of the United States, so help me God.”⁶⁵³ They are paid in full through the annual appropriation adopted by the general

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⁶⁵² *Berger v. United States*, 295 U.S. 78, 88 (1935); see also, *State v. Powers*, 526 A.2d 489, 494 (R.I. 1987) (“the primary duty of a prosecutor is to achieve justice, not to convict”). It is beyond dispute that this solemn duty applies “with equal force to the government’s civil lawyers.” *Freeport-McMoran Oil & Gas Co. v. Federal Energy Regulatory Comm’n*, 962 F.2d 45, 47 (D.C. Cir. 1992) (Mikva, C.J.). Thus, it has long been recognized that a government lawyer in a civil proceeding should be held to a higher standard than a private lawyer, and that in civil proceedings “government lawyers have ‘the responsibility to seek justice,’ and ‘should refrain from instituting or continuing litigation that is obviously unfair.’” (citation omitted).

assembly to ensure that their loyalty is to the people of the State. Finally, state employee are forbidden from using their positions for financial gain or advantage. This means that they are not allowed to have a financial interest, direct or indirect, in the areas they are administrating because it creates a conflict of interest “which is in substantial conflict with the proper discharge of his or her duties or employment in the public.” Unlike state employees working for attorneys general, special assistants are not bound by these principles. In fact, the very nature of a contingency fee seems contrary to the conflict-of-interest prohibitions applicable to most elected attorneys general.

In the Rhode Island case, the State hired two law firm as its special assistants. According to their contingency fee contract, the firms were to receive 16 2/3 percent of any monies recovered by the State. Additionally, the State granted its special assistants “full authority and responsibility for all case management, trial strategy, and other decisions necessary or incident to the necessary prosecution of the claims.” Thus, the State gave its special assistants full rein to prosecute the suit in any manner they chose, including the choice of who to sue and who not to sue.

Because of the profit motive, contingency fee agreements encourage lawsuits against “deep pocket” industries. The lawsuits are often brought against out-of-state industries or those that are viewed as unpopular by the general public, making it difficult for such defendants to

655 See, e.g., R.I. Gen. Laws §36-14-1 (It is the policy of the state of Rhode Island that public officials and employees must adhere to the highest standards of ethical conduct, respect the public trust and the rights of all persons, be open, accountable, responsive, avoid the appearance of impropriety, and not use their position for private gain or advantage); Rhode Island law also requires that the Attorney General and his or her employees may not “use their position for private gain or advantage,” are forbidden to “have any interest, financial or otherwise, direct or indirect . . . which is in substantial conflict with the proper discharge of his or her duties or employment in the public interest.” See R.I. Gen. Laws §36-14-5(a). A substantial conflict of interest exists when the Attorney General “has reason to believe or expect that he or she . . . will derive a direct monetary gain . . . by reason of his or her official activity.” R.I. Gen. Laws §36-14-7(a).
656 See Rhode Island v. Lead Indus. Assoc., No. 2004-63-M.P. (PC 99-5526) (R.I. June 2, 2006) (Opinion in which the Rhode Island Supreme Court held that the question of whether the use by the State to hire private contingent feed counsel is constitutional was not ripe because the district court had yet to render a final judgment in the underlying case).
receive a fair trial. This is particularly true when “special assistants” can cloak themselves with the state’s authority and prosecute its cases unfettered by any of the constraints otherwise applicable to true government attorneys. Although the result is private litigation that undeniably furthers the private interests of the private attorneys pursuing it, this new species of private litigation is backed by the state’s moral authority and seal of approval.\textsuperscript{657}

(2) Contingency Fee and Profit Motivation

Contingency fee agreements were meant to increase access to courts for individuals without the resources to pay an hourly attorney fee. They provide access to the legal system regardless of a person’s means.\textsuperscript{658} Contingency fees allow an individual to assert a claim that he otherwise would not be able to afford if he had to pay an attorney’s hourly fee. Thus, it enables a poor man with a meritorious cause of action to obtain competent counsel.\textsuperscript{659}

States do not have problems with being able to afford competent counsel. All of them have their own attorneys that work just for them under the direction of the state’s attorney general. Contingent fee agreements were not meant for state governments. Governments are wealthy because they have the power to tax and condemn. Governments also control access to the legal system. By using contingency fee agreements, state attorneys general are able to avoid asking the state legislature for appropriations to file the lawsuit\textsuperscript{660} and in the process they create the illusion that the lawsuit is being pursued at no cost to the taxpayers. But, these contracts also

\textsuperscript{657} See Chamber of Commerce, supra note 648 at 24.


\textsuperscript{659} Although widely accepted today, contingency fee agreements are not always allowed. For example, contingency fees remain prohibited in criminal defense cases. See Brickman, supra note 658, at 40-41. They are prohibited in criminal cases because they have the potential to create mis-incentives that threaten to corrupt justice. For instance, if a lawyer’s recovery is based on his or her client’s acquittal, the incentive is to win at any cost, possibly by suborning perjury. See id. Likewise, contingency fee agreements create improper incentives when they encourage use of the state’s police power to obtain the highest monetary award at any broader cost to society.

\textsuperscript{660} See, e.g., City & County of San Francisco v. Philip Morris, 957 F. Supp. 1130, at 1136, n.3 (N.D. Cal. 1997) (finding unconvincing “plaintiff’s argument that, as a matter of public policy, a contingent fee arrangement is necessary . . . to make it feasible for the financially strapped government entities to match resources with the wealthy [corporate] defendants”).
create the potential for outrageous windfalls or even outright corruption for political supporters of the officials who negotiated the contracts.\textsuperscript{661}

Furthermore, windfalls gained by the “special assistants” can seriously injure the morale of talented government attorneys. For example, in Washington State, after Steve Berman of the private Seattle law firm of Chandler, Franklin & O’Bryan Ferguson was awarded a lucrative amount for leading Washington State’s lawsuit against the tobacco companies, the State’s antitrust chief, Jon Ferguson, announced that he was leaving his post to join the Chandler, Franklin law firm to work on a class action lawsuit against the tobacco industry. When asked why he was leaving his post to go work for the firm that handled the State’s case, Mr. Ferguson succinctly explained: “Steve Berman got $50 million and I got a plaque.”\textsuperscript{662}

(3) Circumventing State Legislatures: Regulation through Litigation

Another problem with state attorneys generals using contingency fee agreements is the “end-run” the lawyers make around the state’s legislative process. With respect to the lead paint litigation, Motley Rice set its sights on manufacturers of lead pigment. The State of Rhode Island became its accomplice by allowing the private law firm to bring suit instead of resolving

\textsuperscript{661} Bill Pryor, Curbing the Abuses of Government Lawsuits Against Industries, Speech Before the American Legislative Exchange Council 8 (Aug. 11, 1999). One has to look no further than the litigation against big tobacco to find example of the type of outrageous windfall, these special assistants seek. For example, Kansas Attorney General Stovall’s former firm, Entz & Chanay, reportedly received $27 million in legal fees for its “favor” of serving as local counsel in the State’s tobacco lawsuit. See John L. Peterson, Attorneys for Kansas Collect $55 Million In Tobacco Case, Stovall’s Ex-Firm Expects $27 Million, KANSAS CITY STAR B1 (Feb. 1, 2000). Entz & Chanay was paid the equivalent of $2,700 per hour for simply acting as local counsel in the State’s case. The tobacco settlement awarded the lawyers (hired by then-Texas Attorney General Dan Morales) fifteen percent of the State’s $15.3 billion recovery – about $2.3 billion, which ultimately was increased by an arbitration panel adjudicating the fee dispute to $3.3 billion. See Bruce Hight, Lawyers Give up Tobacco Fight, AUSTIN AM.-STATESMAN, A1 (Nov. 20, 1999). That amounted to $105,022 per hour, assuming the lawyers worked eight hours per day, seven days per week, for eighteen months. See Sheila R. Cherry, Litigation Lotto, Insight on the News, (Apr. 3, 2000), available at \url{http://www.insightmag.com/news/2000/04/03/CoverStory/Litigation.Lotto208397.shtml} (subscription required). In Maryland, Attorney General J. Joseph Curran, Jr. entered into a contingency fee agreement with personal injury attorney (and Baltimore Orioles owner) Peter Angelos. Angelos demanded the full 25% share of the state’s $4.4 billion of the national settlement, as provided in his 1996 contract, and refused to submit his claim to arbitration. See David Nitkin & Scott Shane, Angelos to Get $150 Million for Tobacco Lawsuit, BALTIMORE SUN, 1A (Mar. 23, 2002). This would have entitled Angelos to more than $1 billion, the equivalent of $30,000 per hour. See Scott Shane, Angelos Says Panel Can’t be Impartial, BALTIMORE SUN, 1B (Nov. 30, 2001). After a three-year legal battle, Angelos settled with the state for $150 million. See David Nitkin & Scott Shane, supra.

\textsuperscript{662} For the Record, WASH. POST. F35 (Feb. 14, 2000).
the issue through legislation. Did Rhode Island’s Attorney General bring the suit to further
the will of the state or merely as a possible means to raise new revenue?

In Rhode Island, what is authorized by law cannot be a public nuisance. In 1991, Rhode Island adopted regulations to deal with the lead paint problem when its General Assembly enacted the Lead Poisoning Prevention Act (“LPPA”). The LPPA and the regulations promulgated by the Rhode Island Department of Health (“RIDOH”) require property owners to maintain painted surfaces so that old lead paint remains covered or intact. Moreover, under Rhode Island law, intact lead-based paint is not deemed to be a hazardous material. Rhode Island’s legislature considered and rejected the approach promoted by the state attorney general (i.e., the complete removal of all existing layers of lead-based paint, even if located under layers of intact non-lead paint). It rejected the Attorney General’s proposed solution because it determined that complete removal would be enormously expensive, would provide no greater public health benefits on balance than the “lead safe” approach that was actually adopted, and it would create additional hazards resulting from the dust and debris generated by the lead paint removal efforts.

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663 State v. Barnes, 40 A. 374, 375 (R.I. 1898); see also, Richmond Realty, Inc. Realty v. Town of Richmond, 644 A.2d 831, 831 (R.I. 1994) (where defendant had complied with all applicable Department of Environmental Management requirements in constructing a new drainage system “its actions were authorized by law and cannot constitute a public nuisance”); Nugent v. Vallone, 161 A.2d 802, 806 (R.I. 1960) (“Exercise of the right to do that which the law authorizes cannot be a public nuisance.”); RESTATEMENT (SECOND) OF TORTS of §821B.

664 See Lead Poisoning Prevention Act, DOH 14000-013-6.1 (lead safe standards) and DOH 14000 013-4.4(3) (intact lead paint not on a friction surface is lead safe).

665 See Rules & Regulations for Lead Poisoning Prevention, R23-24.6-PB, §§6.1, 11.1, 12.1 (Nov. 2001). The LPPA directed RIDOH to “promulgate regulations for acceptable environmental lead levels in dwellings . . . including standards for lead on painted surfaces. . . .” R.I. Gen. Laws §23-24.6-5(c). The LPPA also provided that all rules and regulations promulgated by RIDOH “shall provide for the use of ‘lead safe’ reduction as the preferred method where possible to meet the requirements of this chapter. . . .” R.I. Gen. Laws §23-24.6-26 (emphasis added). RIDOH’s regulations under the LPPA require removal of old lead paint only where it is a “hazard.” Old lead paint that is covered by later layers of non-lead paint and is intact is not a hazard and need not be removed. See Rules & Regulations for Lead Poisoning Prevention, R23-24.6-PB, §§6.1 (setting standards for lead in paint), 11.1 (applicability and scope of lead hazard reduction rules), 12.1 (identifying approved lead hazard reduction techniques for painted surfaces) (Nov. 2001).

However, in March 2003, the Rhode Island court hearing the lead paint case ruled that because the State Attorney General was bringing a “common law” claim, it was not bound or constrained by the statutory and regulatory definition of lead poisoning or what constitutes an elevated BLL that the State’s legislative assembly enacted as the law or the State’s regulatory agencies promulgated as the regulation of the State.\(^{667}\)

Obviously, the state attorney general’s goal is at odds with the will of the people as represented and expressed by the legislation passed and the bases for that legislation. Clearly, the benefit to the public becomes suspect at best, as compared to the profit motive of the attorney general and his “special assistants.” As Robert B. Reich, Secretary of Labor in the Clinton Administration, has sagely observed, “[t]he strategy may work, but at the cost of making our frail democracy even weaker. . . . This is faux legislation, which sacrifices democracy to the discretion of administration officials operating in secrecy.”\(^{668}\) Professor Schwartz also eloquently recognizes the dangers of disenfranchising the public in this manner, stating that, “[i]f no rational brakes are applied to the attorney general-personal injury attorney alliance, public health and safety questions may no longer be debated and settled by elected officials beholden to the will of the people. Instead, personal injury lawyers, motivated by profit, joined by selective attorneys general and judges who want to make, not interpret, the law, will fill that role.”\(^{669}\)

\(^{667}\) Hearing Transcript, August 31, 2006, at 189 (“You also said in March of 2003 that the Court did not find that the plaintiffs in this common law claim is required to adopt the statutory and regulatory definition of lead poisoning or of elevated blood levels.”) (“But what you said is the Department of Health can define lead poisoning any way they want in their rules and regulations, but that’s got no bearing on how it’s defined here.”).

\(^{668}\) Robert B. Reich, Don’t Democrats Believe in Democracy?, \textit{WALL ST. J.} A22 (Jan. 12, 2000).

iv. “Private” Public Nuisance Actions

Individuals have traditionally had standing to bring public nuisance actions against wrongdoers for just compensation. The prerequisite for such a cause of action is that the basic elements of public nuisance theory must be satisfied: public injury, unreasonable conduct, defendant control and proximate cause. However, a private individual could not seek an injunction or abatement.

Consider the following illustration: a landowner pollutes the ground and local river in a way that creates a public nuisance. The government will have a cause of action under public nuisance theory to stop the polluting activity and force the responsible party to abate the condition. A neighbor whose well water was polluted by the contamination, thereby sustaining a particular injury from the public nuisance, could seek compensation for her own damages. Conversely, a person who does not have a particular injury could not sue under a public nuisance theory. Thus, under this example, members of the general public, who may no longer swim or fish in the river, would not have standing to sue.

3. The Use of Public Nuisance Lawsuits Against Product Manufacturers

During the past 20 years, personal injury lawyers have increasingly sought to expand the boundaries of public nuisance law as it applies to product manufacturers. They argued that, even if the offending product is lawfully manufactured, distributed, and sold, by their nature it interferes with the public’s right to health or safety. The most prominent public nuisance claims have been against makers of products that could pose danger if used or stored incorrectly, such as asbestos, lead pigment and paint, firearms and MTBE. Similarly, State attorneys general have

670 The Restatement (Second) of Torts explains that allowing such a private cause of action is legally justified because it would be unreasonable for a party to engage in the conduct without also paying for the harm done by that conduct. Restatement (Second) of Torts §821B cmt. i (1979).

671 The key issue in these suits is determining whether the alleged injury is different in kind or just different in degree—the same, but more severe—than that of the general public.
also brought public nuisance claims against manufacturers of tobacco products. In many of these actions, the plaintiffs sought abatement, compensation and punitive damages.

Public nuisance claims are very enticing because if it is successfully plead they act as a “super tort.” Like product liability claims, public nuisance offers strict liability. But, by filing claims under public nuisance theory, these lawyers seek to avoid a number of products liability requirements, such as defect, the statute of limitation and the rule against recovery for purely economic loss. If plaintiffs can avoid these requirements, their chances of recovery are greatly increased. While most courts have rejected these new claims, others have been willing to accept them, leaving this chapter in public nuisance theory with an uncertain ending. Lead paint litigation is just the latest arena in which lawyers have sought to expand public nuisance law. This sections describes some of the earlier forums in which modern lawyers brought public nuisance claims.

i. Asbestos Litigation

In the 1980s and 1990s, lawyers for several municipalities and school districts seeking to recover the costs of removing asbestos from their buildings asserted public nuisance claims against manufacturers of asbestos-containing products. For the first time, plaintiffs alleged that the product itself constituted a public nuisance, not that the product was used to create a public nuisance. Further, instead of being filed by third parties, these suits were filed by the consumers themselves.

As the court stated in *Detroit Bd. of Educ. v. Celotex Corp.*, however, “manufacturers, sellers, or installers of defective products may not be held liable on a nuisance theory for injuries caused by [a product] defect,” and “all courts that have considered the question have rejected

\[672\] Schwartz, *supra* note 596, at 552 (citations omitted).
nuisance as a theory of recovery for asbestos contamination.\textsuperscript{673} Most courts have agreed that the creation of a product is not the same as the creation of a nuisance and that the facts in products liability cases do not fit the elements of public nuisance theory.\textsuperscript{674} For example, some courts observed that the element of “control” could not be satisfied because “a nuisance claim may only be alleged against one who is in control of the nuisance creating instrumentality.”\textsuperscript{675} In these cases, even if asbestos were considered a nuisance, “[t]he ‘nuisance’ creating property . . . was in possession and control of the plaintiff from the time it purchased the asbestos-containing products.”\textsuperscript{676}

Courts were also troubled by the practical implications of the suits on products liability law. They recognized that the plaintiffs’ theory would “give rise to a cause of action . . . regardless of the defendant’s degree of culpability or of the availability of other traditional tort law theories of recovery.”\textsuperscript{677} They also recognized that the existence of asbestos cannot be considered a “continuing nuisance for which the statute of limitations [does] not bar recovery.”\textsuperscript{678} Thus, even though the offending product caused serious harm to many people and there was widespread sympathy for school boards, courts maintained the common law boundaries of the public nuisance tort in the context of asbestos litigation.

\textsuperscript{676} Mercer, 1986 WL 12447, at *6.
\textsuperscript{678} Detroit Bd. of Educ., 493 N.W.2d at 520.
ii. Tobacco Litigation

Everyone is generally familiar with the tobacco litigation of the 1990s in which state attorneys general sought reimbursement of state expenditures for Medicaid and other medical programs for smokers, if for no other reason than the sheer size of the awards and resulting attorneys’ fees. But, what is often overlooked in the tobacco story is the fact that many of the lawsuits included claims for public nuisance.679 “By using public nuisance and other equitable theories of recovery, the state attempted both to avoid the need to prove specific causation of any individual’s illness and to eliminate defenses based upon a smoker’s own conduct, such as contributory negligence and assumption of risk.”680

The only court to rule on a public nuisance claim was a federal district court in *Texas v. American Tobacco Co.*681 The allegations in that case were that the defendants “intentionally interfered with the public’s right to be free from unwarranted injury, disease, and sickness and have caused damage to the public health, the public safety, and the general welfare of the citizens.”682 The court dismissed this claim, stating that it was not within the traditional bounds of public nuisance theory: “The overly broad definition of the elements of public nuisance urged by the State is simply not found in Texas case law and the Court is unwilling to accept the State’s invitation to expand a claim for public nuisance.”683

679 Schwartz, *supra* note 596, at 554. Not every lawsuit included public nuisance claims; however, some did, marking the first time that public nuisance was used in mass actions in products litigation. *Id.*

680 Gifford, *supra* note 603, at 759.


682 *Id.* at 972.

683 *Id.* at 973. In 1998, the state attorneys general settled all of their claims with the manufacturers of tobacco products in what was called the Master Settlement Agreement (MSA). Even though some of the lawsuits covered by the settlement contained public nuisance claims, nothing in the MSA indicated that the sale, distribution and promotion of tobacco products constituted a public nuisance. Nonetheless, the use of public nuisance theory quickly became a misleading aspect of the state attorney general tobacco litigation legend. Schwartz, *supra* note 596, at 554-55.
iii. Firearms Litigation

The use of public nuisance theory in firearms litigation was a direct outgrowth of the tobacco litigation. In an attempt to adapt public nuisance to firearm litigation, the public nuisance would not be “in the manufacture of guns [or] in the existence or sale of guns,” but in the marketing and distribution practices and policies of the manufacturers. In other words, gun manufacturers created an unreasonable threat to public safety by following distribution practices that permit criminals to acquire guns. Specifically, the plaintiffs alleged that the manufacturers facilitated the illegal secondary market for firearms, thereby interfering with the public health of the community. Most courts have rejected this extension of public nuisance.

An Indiana Court that allowed the case to proceed, acknowledged that it was acting without precedent. It also defined an “interference with a public right” to include any “lawful

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684 Professor David Kairys, who taught at the Beasley School of Law at Temple University, worked with some cities to file public nuisance claims against gun manufacturers. Kairys stated that although the tobacco public nuisance claims had “legal problems” and “never [won] in court,” he believed they were a “vehicle for settlement” and, therefore, a model for the gun suits. This observation appears to be ipse dixit; no facts have been found to support it. See David Kairys, The Origin and Development of the Governmental Handgun Cases, 32 CONN. L. REV. 1163, 1167 (2000).

685 Kairys, supra note 684, at 1173.

686 See, e.g., Ganim v. Smith & Wesson Corp., 780 A.2d 98, 115 (Conn. 2001) (“The plaintiffs alleged that the existence of the nuisance is a proximate cause of injuries and damages suffered by [the city], namely, that the presence of illegal guns in the city causes costs of enforcing the law, arming the police force, treating the victims of handgun crimes, implementing social service programs, and improving the social and economic climate of [the city].”); Gary v. Smith & Wesson Corp., 801 N.E.2d 1222, 1231 (Ind. 2003) (stating that the city alleged that the “manufacturers, distributors, and dealers knowingly participate in a distribution system that unnecessarily and sometimes even intentionally provides guns to criminals, juveniles, and others who may not lawfully purchase them”); Cincinnati v. Beretta U.S.A. Corp, 768 N.E.2d 1136, 1141 (Ohio 2002) (stating the city alleged that the defendants “know, or reasonably should know, that their conduct will cause handguns to be used and possessed illegally and that such conduct produces an ongoing nuisance that has a detrimental effect upon the public health, safety, and welfare of the residents”).


688 Gary v. Smith & Wesson Corp., 801 N.E.2d 1222, 1231 (Ind. 2003) (acknowledging that under Indiana law, courts have recognized public nuisance claims only when the claims involve land use or illegal activities).
activity conducted in such a manner that it imposes costs on others." The court provided that “[i]f the marketplace values the product sufficiently to accept that cost, the manufacturer can price it into the product.” The court also allowed the city to sue for damages in addition to injunctive relief and abatement.

The majority view holds, however, that a public right must be implicated, stating that the “right to be free from the threat that members of the public may commit crimes against individuals” was a personal, not a public, right. It also holds that balancing the harm and utility of the sale and marketing of guns is a policy question better suited for the legislature, not the courts, particularly because these activities already are well regulated. Other courts have denied these claims because the defendants lacked the requisite control over the source of the alleged public nuisance.

Today, public nuisance suits against gun manufacturers have been restricted, if not precluded, by the enactment of the Protection of Unlawful Commerce in Arms Act of 2005. This Act precludes tort actions against firearm manufacturers in federal or state courts based on criminals’ unlawful uses of their products.

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689  Id. at 1233-34 (defending this position by stating that “there is no injustice in requiring the activity to tailor itself to accept the costs imposed on others or cease generating them”); see also Cincinnati, 768 N.E.2d at 1142 (stating that “a public-nuisance action can be maintained for injuries caused by a product if the facts establish that the design, manufacturing, marketing, or sale of the product unreasonably interferes with a right common to the general public”).

690  Gary, 801 N.E.2d at 1234 & 1240.

691  See, e.g., Chicago, 821 N.E.2d at 1114-16 (“We are also reluctant to recognize a public right so broad and undefined that the presence of any potentially dangerous instrumentality in the community could be deemed to threaten it.”); Camden County Bd. of Chosen Freeholders v. Beretta U.S.A. Corp., 273 F.3d 536, 539 (3d Cir. 2001) (stating that, under New Jersey law, “the scope of nuisance claims has been limited to interference connected with real property or infringement of public rights”).

692  Chicago, 821 N.E.2d at 1121 (“We are reluctant to interfere in the lawmaking process in the manner suggested by plaintiffs, especially when the product at issue is already so heavily regulated by both the state and federal governments.”).

693  See, e.g., Camden, 273 F.3d at 539.

iv. Lead Paint Litigation

The plaintiffs bar has been suing lead pigment manufacturers for negligence and products liability since the late 1980s in an attempt to place the responsibility for poorly maintained lead paint on these companies. Some were brought on behalf of individual plaintiffs or classes of plaintiffs alleging exposure to lead paint in their homes. Other were brought by governmental entities seeking reimbursement for childhood lead exposure programs and/or funding for lead paint abatement. A handful of the cases even involved adult painters blaming a myriad of health problems from hypertension to kidney disease on long-ago exposure to lead paint.

All lead-paint cases against lead pigment manufacturers are based on the alleged subtle neurological and psychological impairments in children that post-1970 epidemiological reports suggest are associated with elevated BLLs. Ever since these studies were first published, federal and state governments have enacted laws and regulations banning or limiting the use of lead in many products. While these regulations greatly curtailed new releases of lead into the environment, they do nothing to abate all the lead placed into the environment during the last 100 to 400 years. Usually, lead-based paint only become a concern when landlords and homeowners allow the paint to deteriorate in a manner that allows the lead in the paint to potentially be ingested by young children.

Many states and municipalities have enacted regulations that require landlords to maintain existing lead paint in a manner that does not create a hazard. Moreover, landlords have control over these affected premises that are in the best position to prevent the hazard simply by keeping the property properly maintained. Thus, children who are injured by poorly maintained property can, and often do, sue their landlords. However, these cases are often fact specific and driven by individual issues of causation. Thus, entrepreneurial plaintiffs’ lawyers have a difficult time turning them into a lucrative “mass tort.” To get around problems associated with
individual causation issues, these attorneys bring public nuisance claims, aggregating claims and seeking costs for screening, monitoring and abatement. Perhaps most important, former lead pigment manufacturers have deeper pockets than landlords.  

Public nuisance was not used by plaintiffs in lead paint litigation until 1999 when lawyers from the law firm Motley Rice convinced the Attorney General of Rhode Island to partner with them, on a contingency fee basis, in commencing a government public nuisance action against the former lead companies. The alleged public nuisance was the mere presence of lead paint in homes and buildings. Armed with the power of the sovereign, Mr. Motley sought the costs of removing lead paint from every building in Rhode Island that contained it. He even boasted that he would “bring the entire lead paint industry to its knees.” Since filing that case in 1999, the plaintiffs’ bar has partnered with public entities to bring public nuisance claims on behalf of several states, counties and municipalities.

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696 State v. Lead Indus. Ass’n, Inc., No. 99-5226, Retainer Agreement, App. 4 to Pet. For Cert. ¶ 1 (R.I. filed Mar. 2, 2004). See also Scott A. Smith, Turning Lead into Asbestos and Tobacco: Litigation Alchemy Gone Wrong, 71 DEF. COUNS. J. 119, 119 (2004) (stating that “lead paint and pigment defendants had never lost or settled a case” since 1987); Editorial, Rhode Island Rhapsody, WALL STREET J. A10 (Aug. 16, 2006). State attorneys general regularly hire outside counsel for help in prosecuting cases, but these outside attorneys are typically paid by the hour. By contrast, contingency-fee deals introduce a profit motive that has no place in state-sponsored litigation. Mr. Lynch didn’t originate this practice, which has become increasingly common. This trend emerged in the mid-1990s with lawsuits against Big Tobacco. Not only did the settlements from state tobacco litigation generate hundreds of millions in fees for the private lawyers involved, it also pioneered a new model for state-sponsored litigation that combines the prosecutorial power of the government with private lawyers aggressively pursuing litigation that has the potential to generate hundreds of millions – or billions – of dollars in contingent fees. Id. (citing former Virginia Attorney General Jerry Kilgore).


698 Mark Currider, Tobacco Fees Give Plaintiffs’ Lawyers New Muscle for Other Litigation, DALLAS MORNING NEWS, Oct. 31, 1999; Michael Freedman, Turning Lead into Gold, FORBES, May 14, 2001, at 122 (explaining that Mr. Motley targeted the former lead companies as his “next big-game hunt,” found victims, and “demonized” the industry because they were a “fat target”).

699 Public nuisance suits have been filed in Santa Clara County, California; Chicago, Illinois; St. Louis, Missouri; and Milwaukee, Wisconsin. Schwartz, supra note 596, at 559n.112.
In response to these suits, defendants have responded with some general defenses. First, they have no control over the instrumentality (the building or home) causing the nuisance.\footnote{See supra section V.B.2.ii(c).} The lead pigment at issue was legally made and sold anywhere from 40 to 300 years ago. The companies relinquished control over lead pigment decades ago. They were not the parties that applied it to the buildings. Moreover, they have no legal right or ability to ensure that property owners maintain lead paint in a safe condition. Only the current landlords have control of the paint and they have the legal responsibility to maintain their properties and address any lead paint hazards that may develop. Finally, no public right is being interfered with.\footnote{See supra section V.B.2.i.} As stated earlier, a public right is a right common to all people, such as the right to use a public street or a waterway. At issue in these cases is the right to be free of lead paint in private residences, places that are inaccessible to the public at large. It is not the general public that is allegedly injured, but specific individuals or classes of individuals.

(a) The Wisconsin Solution

In April 2001, the city of Milwaukee filed a lawsuit against Mautz Paint (now part of Sherwin-Williams) and NL Industries seeking $85 million from the two companies to cover the cost of abating harmful lead paint in roughly 41,000 houses in Milwaukee neighborhoods.\footnote{Tom Held, City’s Lawsuit Against Lead Paint Makers Can Go To Trial, MILWAUKEE J. SENTINEL (Nov. 10, 2004).} The companies were also sued for false advertising for allegedly failing to warn the public of the dangers of lead-based paint.\footnote{City’s Lead Paint Lawsuit Dismissed, BUS. J. OF MILWAUKEE (July 29, 2003).} In October 2001, the court dismissed the false advertising claim and a conspiracy claim.\footnote{Tom Held, Judge Dismisses Lawsuit Against Lead Paint Companies, MILWAUKEE J. SENTINEL ON-LINE (July 30, 2003).} On September 26, 2003, the trial court dismissed the remaining parts of Milwaukee’s case because the City could not prove that the defendants’ conduct or products...
were a substantial factor in causing the injury. The court held that the City had not met its burden of proving that, at a minimum, the NL Industries’ pigment or lead paint or Mautz’s lead paint is present on windows in the target area properties and that their conduct somehow caused the paint to become a hazard to children.\(^{705}\) The City appealed the court decision and on November 9, 2004, a Wisconsin Court of Appeals reversed the trial court and reinstated the case. The appeals court held that whether the defendants’ product and conduct contributed to the harm was a question of fact for the jury to decide.\(^{706}\) The defendants petitioned the Wisconsin Supreme Court to reverse the opinion of the Court of Appeals, but the Supreme Court dismissed the petition on August 1, 2005. The case is now pending in the trial court.\(^{707}\)

Though the Wisconsin Supreme Court’s *Thomas* decision\(^{708}\) is not directly applicable to the City of Milwaukee’s case against two pigment companies, in light of the court’s decision in that case, Mautz Paint and NL Industries dropped their request to the Wisconsin Supreme Court to stop the City’s lawsuit. The City’s case against the pigment manufacturers will now proceed to trial, which should begin sometime in 2006.\(^{709}\)

(b) California Litigation

In March 2000, officials in Oakland and San Francisco joined other California counties and municipalities in a lawsuit against eight U.S. paint and pigment manufacturers, the Lead

\(^{705}\) Memorandum from Rudolph M. Konrad, Deputy City Attorney, City of Milwaukee to Jennifer Gonda, Legislative Fiscal Manager-Sr., City of Milwaukee, Regarding Assembly Bill 778 Relating Actions Against Manufacturers, Distributors, Sellers, And Promoters Of Products (Nov. 5, 2005), available at [http://66.102.7.104/search?q=cache:Vo9ulzdon8J:legistar.milwaukee.gov/Attachments/766aca86-56d9-4d4b-aea5-9ce37159b0d2.DOC+Mautz+Paint+%26+milwaukee+%26+nl+%26+lawsuit&hl=en&gl=us&ct=clnk&cd=12](last visited Aug. 10, 2006).

\(^{706}\) Id.

\(^{707}\) Id.

\(^{708}\) See supra section V.A.2.iii (discussing the Mallet decision).

Industries Association, and “up to 50 fictitiously named companies.” Their complaint, filed in Santa Clara County Superior Court, argues that the defendants engaged in a concerted campaign against government regulations by opposing warnings, attacking the credibility of public health workers and using public relations campaigns to mislead consumers. After the trial court dismissed the plaintiffs’ claims of public nuisance, strict liability, negligence and fraud in 2003, they appealed to the California 6th District Court of Appeals.

In reversing the district court, the appellate court held that the lawsuit’s public nuisance cause of action was not premised on a defect in a product or a failure to warn but on affirmative conduct that assisted in the creation of a hazardous condition. Thus, the defendants’ liability is based on their affirmative promotion of lead paint for interior use, not merely the manufacture and distribution of lead paint. As for the claims of negligence and strict liability, the court said, the economic loss alone, without physical injury, does not amount to the type of damage that will cause a negligence or strict liability cause of action to prevail. The appeals panel said that even though the plaintiffs failed to plead the existence of any physical injury to their buildings, the trial court was wrong to grant the defendants summary judgment. The trial court should have allowed the plaintiffs to rectify their error. The fraud claim should be reinstated, the appeals panel said, because the defendants concealed evidence that low levels of lead exposure could be hazardous. The claim was not barred by the state’s three-year limitations period because the plaintiffs were not aware of the dangers of low-level lead exposure until scientific studies were published in 1998. Since the complaint was filed in 2000, the plaintiffs filed their action within

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the three-year window, the appeals panel said. Therefore, their claims were not barred. On June 31, 2006, the California Supreme Court refused to review the appellate court’s decision. As a result, the case was remanded for further proceedings.

(c) The Showdown in Rhode Island

In Rhode Island, the court sent the case to the jury twice. In 2002, a jury deadlocked four to two against the state’s public nuisance claim. In the second trial, the court made a number of significant jury charge rulings that significantly affected the verdict.

First, it defined a public nuisance injury simply as “the cumulative presence of lead pigment in paints and coatings in [or] on buildings in the state of Rhode Island.” This definition suggests that an injury to a significant number of individuals is the same as an injury to the community as a whole. This logic is faulty. It is clear from case law that “harm to individual members of the public” (no matter how many) is not the same as harm “to the public generally.” As one court explained, “[t]he test is not the number of persons annoyed, but the

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712 Id. The government entities sued the paint manufacturers on behalf of the public in March 2000, long after the industry had pulled lead paint from the market, but only two years after scientific studies showed that even low-level exposure could cause serious physical harm.


716 In its jury instructions, the Court altered the language in comment g of §821B of the Restatement (“A public right is one common to all members of the general public”). Instead of following the Restatement, the Court instructed the jury that: A right common to the general public is a right or an interest that belongs to the community-at-large. It is a right that is collective in nature. A public right is a right collective in nature and not like an individual right that everyone has not to be assaulted, defamed, or defrauded, or negligently injured.

Jury Instructions, at 11. This language ignores the Restatement sentence which states that a public right is common “to all members of the general public.” The Court refused the defendant’s requested instruction that conduct “does not become a public nuisance merely because it interferes with the use and enjoyment of land by a large number of persons.” RESTATEMENT (SECOND) TORTS §821B, cmt. g. In fact, the Court instructed the jury contrary to comment g that: “When you consider the reasonableness of the interference, you may consider a number of factors including . . . the numbers of the community who may be affected by it . . . .” Jury Instructions, at 12.

possibility of annoyance to the public by the invasion of its rights. A public nuisance is one that injures the citizens generally who may be so circumstanced as to come within its influence."

Second, it stated that the jury should find “unreasonable interference” so long as the children “ought not to have to bear” the injury of lead poisoning; there was no requirement to find that the defendants engaged in any unreasonable conduct. This suggests that it “is a public right to be free from the threat that some individuals may use an otherwise legal product (be it a gun, liquor, a car, a cell phone, or some other instrumentality) in a manner that may create a risk of harm to another.”

Most courts have disagreed with this idea, however, concluding that there is no public right to be free from the threat that someone may use a legal product in a way that creates risk of harm to another. In dismissing a public nuisance claim against gun manufacturers in New Jersey, the United States Court of Appeals for the Third Circuit wrote, “[i]f defective products are not a public nuisance as a matter of law, then the non-defective, lawful products at issue in this case cannot be a nuisance without straining the law to absurdity.”

Third, it instructed the jury that it “need not find that lead pigment manufactured by the Defendants, or any of them, is present in particular properties in Rhode Island to conclude that Defendants, or one or more of them, are liable” to Rhode Island under public nuisance theory.

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718 Higgins v. Conn. Light & Power Co., 30 A.2d 388, 391 (Conn. 1943) (internal quotation marks omitted).
719 Jury Instructions, Rhode Island v. Atlantic Richfield Co., C.A. No. 99-5226, *12 (R.I. Super. Ct. Feb. 13, 2006) (“When you consider the unreasonableness of the interference, you may consider a number of factors including the nature of the harm, the numbers of community who may be affected by it, the extent of the harm, the permanence of the injuries and the potential for likely future injuries or harm.”).
720 Schwartz, supra note 596 at 564. A public nuisance requires interference with a public right. See, e.g., Hydro-Manufacturing Inc. v. Kayser-Roth Corp., 640 A.2d 950, 957-58 (R.I. 1994); Citizens for Preservation of Waterman Lake v. Davis, 420 A.2d 53, 59 (R.I. 1980). “Conduct does not become a public nuisance merely because it interferes with the use and enjoyment of land by a large number of persons. There must be some interference with a public right. A public right is one common to all members of the general public.” RESTATEMENT (SECOND) OF TORTS §821B comment g.
721 Chicago, 821 N.E.2d at 1114-15.
723 Supra note 719, at *14.
In fact, the court did not even require that any defendant “sold lead pigment in Rhode Island.” 724 Given the court’s expansive definitions and jury instructions, it is of little surprise that the jury found against the defendants. 725

(1) Post-Verdict Issues

Following the trial, a controversy arose – and still rages – regarding the meaning of the jury’s findings and how they might be effectuated. Instead of proposing a detailed plan of action, or even a form of judgment, plaintiffs argued that the judge should appoint “special masters” to determine the manner in which the jury’s findings might be implemented. Although their complaint sought the funding of various specific programs, including (i) a public education campaign regarding the continuing dangers posed by lead, (ii) lead poisoning detection and preventative screening programs, (iii) treatment of lead-related health issues, (iv) detection and remediation of lead containing paint from all pre-1978 homes in the state, the State seemed to argue that some sort of equitable relief was now appropriate. 726 Defendants strongly objected to this apparent shift of position, noting that the complaint upon which the case was tried was cast in claims for damages, not injunctive relief. 727 The court has not yet resolved this critical issue.

Defendants also asked for a new trial. They believe that the trial resulted in a mosaic of serious issues, many of which, standing alone, constitute reversible error. When viewed as a whole, however, the defendants believe that multiple errors fatally undercut the one thing that courts try to give all parties – a fair trial. Specifically, the defendants argue that the trial violated

724 Id.

725 See Peter B. Lord, Three Companies Found Liable in Lead-Paint Nuisance Suit, PROVIDENCE J. (Feb. 23, 2006). Post-verdict interviews have indicated that the jury was initially deadlocked four to two in favor of the defense, but that the court’s definitions in the jury instructions lead them to find for liability. See also, Peter Krouse, Verdict Raises Risk for Paint Companies, PLAIN DEALER, Apr. 2, 2006 (including interviews with jurors stating that some members of the jury did not want to find for liability, but the jury instructions, according to one juror, “didn’t give the paint companies much of a window to crawl through”).


727 Id.
fundamental notions of justice and due process, procedurally, substantively and constitutionally. The following illustrates some of the issues that the defendants claim represent reversible error in the first lead paint case in which the jury found the defendant guilty of creating a public nuisance.

(i) **Proximate Cause**

By adopting the State’s “cumulative presence” definition of what constitutes a public nuisance, the court relieved the State of its burden to prove that the defendants did anything wrong. Consequently, the State was not required to prove that any child actually had been injured by lead-containing pigment, or that any home in Rhode Island actually contained lead pigment manufactured by any of the defendants. Instead, the court allowed the State to introduce general evidence that many children had elevated blood lead levels, that lead-containing paint probably contributed to those blood lead levels, and that the defendants historically made lead paint. Once the jury found that a defendant historically made lead paint, it was assumed that the defendant contributed to the nuisance and deemed that the defendant caused any harm flowing from the nuisance. However, proof of the existence of a nuisance or of general harm is not proof that a specific defendant caused the harm being complained about. 728

(ii) **Defendants Never Controlled the Nuisance**

The defendants complain that the court wrongly relieved the State of its burden to prove that any of them physically controlled the circumstances which produced the nuisance. Historically, Rhode Island courts have required plaintiffs to prove that a defendant controlled the

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728 Proof of causation is a fundamental requirement of not only public nuisance, but of all tort law. See, e.g., Clift v. Vose Hardware, Inc., 848 A.2d 1130, 1132 (R.I. 2004); Citizens for Preservation of Waterman Lake v. Davis, 420 A.2d 53, 59-60 (R.I. 1980); see also, W. Page Keeton et al., PROSSER AND KEETON ON THE LAW OF TORTS 269 (5th ed. 1984) (“A mere possibility of . . . causation is not enough; and when the matter remains one of pure speculation or conjecture, or the probabilities are at best evenly balanced, it becomes the duty of the court to direct a verdict for the defendant” (footnotes omitted)).
instrumentality causing the nuisance when the damage occurred. Furthermore, Rhode Island law has consistently held that once a product enters the stream of commerce, the manufacturer loses control over the use (or misuse) of the product and cannot be held liable for any nuisance flowing from the product. By not requiring the State to prove that a defendant controlled the circumstances that produced the nuisance (the poorly maintained properties), the court made the defendants insurers of how purchasers used and maintained their product. To the extent that plaintiffs now argue that defendants should be required to abate the “cumulative presence” of lead paint in Rhode Island, it is unclear how such an order could be enforced when defendants lack control of all of the premises where the substance can be found.

(iii) Removing the Role of the Property Owner and Alternative Sources of Lead from the Trial

Defendants believe that the court improperly removed the role of property owners from the jury’s consideration when the court instructed the jury that it need only find that the defendants were a substantial contributing factor to the public nuisance and that the public nuisance was a substantial contributing factor to the alleged harms. This instruction short-

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729 See, e.g., City of Manchester v. National Gypsum Co., 637 F. Supp. 646, 656 (D.R.I. 1986) (applying New Hampshire law) (“[L]iability for damage caused by a nuisance turns on whether the defendants were in control over the instrumentality alleged to constitute the nuisance, either through ownership or otherwise.”); see also, Friends of Sakonnet v. Dutra, 738 F. Supp. 623, 633-34 (D.R.I. 1990) (applying Rhode Island law) (“[T]he paramount question is whether the defendant was in control of the instrumentality alleged to have created the nuisance when the damage occurred.”) (emphasis added); Friends of Sakonnet v. Dutra, 749 F. Supp. 381, 395 (D.R.I. 1990) (court emphasized that liability for public nuisance “depends primarily on the question of control and duty to maintain” and thus, “[o]ne who controls a nuisance is liable for damages caused by that nuisance.”).

730 See City of Manchester, 637 F. Supp. at 646 (holding manufacturers of asbestos cannot be held liable on nuisance because the manufacturers did not have control over product after sale); RESTATEMENT (SECOND) OF TORTS §834 cmt. d. See also, supra, section V.B.2.i(c) (discussing the control element of a public nuisance action).


732 The court instructed that “liability for a public nuisance arises when the defendant’s acts set in motion a force or chain of events which proximately cause the public nuisance.” Jury Instructions, at 14 (emphasis added).
circuited the jury’s consideration of property owners in the context of proximate cause or as an intervening cause because the State did not have to prove a chain of causation between defendants and the harms they were alleged to have caused. If defendants’ conduct created the condition (the presence of lead pigments), but subsequent acts of third parties (poorly maintained properties) caused the State’s injuries (elevated blood lead levels in children under six), the defendants should not have been liable for the State’s injuries.

According to the defendants, they cannot be held liable for paint that has not been properly maintained because Rhode Island law provides manufacturers immunity from all lawsuits arising out of injuries caused by alterations of their products. Moreover, the property owners’ failure to correct a known, dangerous condition (the deteriorated paint), as a matter of law, supersedes and eliminates the defendants’ liability:

[W]hen a second actor has become aware of the existence of a potential danger caused by the negligence of a first actor and the second actor acts negligently with regard to the dangerous condition, thereby bringing about an accident with injurious consequences to others, the first actor is relieved of liability. This is so because the condition created by the first actor is merely a circumstance and not the proximate cause of the accident.

Thus, the defendants’ claim the court erred by expressly instructing the jury that liability should be imposed for furnishing the underlying condition of liability without regard to the superseding role of the property owners who negligently failed to maintain their properties.

Likewise, the defendants complain that the judge instructed the jury that because the State’s public nuisance claim concerns only lead pigment contained in paints and coatings in or

733 See R.I. Gen. Laws §9-1-32; see also, La Plante v. Am. Honda Co., 27 F.3d 731, 736 (1st Cir. 1994) (Section 9-1-32 prohibits liability where consumer failed to maintain product).

734 Pantalone v. Advanced Energy Delivery Sys., Inc., 694 A.2d 1213, 1215 (R.I. 1997) (emphasis added) (citing Walsh v. Israel Couture Post No. 2274 V.F.W., 542 A.2d 1094 (R.I. 1988)); see also, Drazen v. Otis Elevator Co., 189 A.2d 693, 695 (R.I. 1963) (“Accordingly, where the second actor, after having become aware of the existence of a potential danger created by the negligence of the first actor, acts negligently in respect of the dangerous situation and thereby brings about an accident with injurious consequences to others, the first actor is relieved of liability, because the condition created by him was merely a circumstance and not the proximate cause of the accident.”).
on buildings throughout the state, the jury should not take into account lead from other sources in determining whether such public nuisance exists. They complain because the injury at the heart of the public nuisance is “lead poisoning” and lead poisoning is caused by exposure to lead – any source of lead. Thus, the court’s instruction implicitly instructs the jury to ignore the fact that lead poisoning can be a consequence of lead in the water systems of the children’s homes or schools,735 the lead in the soil outside of their homes (courtesy of leaded gasoline) that is also tracked into their homes,736 the food they eat,737 or putting the toys they play with in their mouths or in the case of sidewalk chalk, their chalk-dusted fingers in their mouths.738 According to the defendants, this was in essence, a directed verdict by the Court on a disputed factual issue.

(iv) Guilt by Association

The State promised the court that it would prove the existence of a conspiracy between the defendants and the Lead Industries Association (“LIA”) – the lead trade association. The State claimed that it would prove that the LIA was the defendants’ agent. Although the State presented a lot of evidence about the LIA’s legislative lobbying activities in other states, it presented no evidence of a conspiracy at trial. It was only after the LIA information had been conditionally admitted that the State conceded that it had no further evidence, and the court finally ruled that there was no conspiracy. Nonetheless, the defendants complain that the court allowed the genie out of the bottle and the court made no attempt to put it back in the bottle when it refused to instruct the jury to disregard the evidence.

The defendants believe that the court should never have allowed the state to introduce this evidence because it is a form of speech that both the Federal and Rhode Island Constitutions

735 See supra section IV.E, infra section III.F.3.
736 See supra section IV.A.1.iv, infra section III.F.2.
737 See supra section IV.D.4, infra section III.F.4.
738 See supra sections II.B.4.v, IV.D.2, and IV.D.5.
expressly protect. Under a line of cases known as the Noerr-Pennington doctrine, the U.S. Supreme Court has recognized that “opposing legislation is a way of participating in the legislative process just as proposing legislation is” and is equally protected by the First Amendment. Therefore, the State’s introduction of the LIA’s lobbying efforts against lead-related legislation in other states was constitutionally prohibited. Opposition to regulations and legislation is exactly the kind of petitioning activity that the Constitution protects. The introduction of lobbying efforts is prohibited even if the legislative activities were part of the alleged nuisance harm itself. Thus, there is no reason the court should have allowed the State to introduce and use the LIA’s constitutionally protected lobbying efforts as a basis for convincing the jury to impose liability.

Similarly, the defendants’ complained that the court allowed the State to introduce into evidence the fact that defendants were ever members of the LIA. It is well established that the free association right guaranteed by the First Amendment prohibits the imposition of liability for a defendant’s membership in, or association with, a trade organization. “Joining organizations that participate in public debate, making contributions to them, and attending their meetings are

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741 See, e.g., Sessions Tank Liners, Inc. v. Joor Mfg., Inc., 17 F.3d 295, 300 (9th Cir. 1994) (prohibiting plaintiff from arguing that defendant’s legislative and petitioning activities were the cause of a harm: “Proof of causation would entail deconstructing the decision-making process to ascertain what factors prompted the various governmental bodies to erect the anticompetitive barriers at issue. This inquiry runs afoul of the principles guiding the Parker [v. Brown, 317 U.S. 341 (1942)] and Noerr decision.”). “The right of freedom of thought protected by the First Amendment against state action includes both the right to speak freely and the right to refrain from speaking at all.” Wooley v. Maynard, 430 U.S. 705, 714 (1977).

742 See Thomas v. Collins, 323 U.S. 516, 530-31 (1945) (freedom of association applies to organizations engaged in economic activity, as it does to individuals and political organizations).
activities that enjoy substantial First Amendment protection.”

Courts should preserve and protect this associational right, not penalize parties by imputing “guilt by association.”

(v) The “Plateau” that Never Was

During the trial, the State and its experts relied on 2004 data to argue that Rhode Island’s lead-poisoning prevention programs had reached the limits of their effectiveness, that too many children still had elevated blood lead levels, and that elevated blood lead levels had “plateaued.”

After the verdict was returned, the defendants complained about this argument for a fundamental reason – it was simply untrue. The truth is that there were 621 elevated blood lead levels in Rhode Island for all of 2005 (compared to 1,167 elevated blood lead levels in 2004), a drop of 47% from the previous year.

The State knew these facts by not later than January 31, 2006 (during the trial) when Rhode Island’s Department of Health prepared a draft report

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744 In re Asbestos School Litig., 46 F.3d at 1294; see also, Abood v. Detroit Board of Education, 431 U.S. 209, 233 (1977) (holding the First Amendment protects “the freedom of an individual to associate for the purpose of advancing beliefs and ideas”).

745 Contrary to well-established First Amendment jurisprudence, the court allowed the State to cast guilt on all defendants for merely belonging to and associating with the LIA. For example, it argued that:

What the State is saying is that if you don’t agree with your industry organization, get out or speak out or do something different. Don’t continue paying your dues. Don’t keep funding the programs, don’t keep going to the meetings, don’t keep serving on the board of directors, don’t keep serving on committees. . . . Not one, not a single one of these defendants said stop it. Not a single one of them – not a single one of them quit the LIA to protest their conduct.

See Trial Transcript, February 10, 2006, at 49; see also, Trial Transcript, November 11, 2005, at 36; Trial Transcript, December 8, 2005, at 56-71.

746 Morning Trial Transcript, November 1, 2005, 23-24, 65-68 (“Ladies and Gentlemen, we’ve reached a plateau. We’ve gone as far as the secondary measures of enforcement and the screening program can take us.”); see also, Trial Transcript, February 29, 2006, at 80. (“We also know that in 2004 more than 1,100 Rhode Island children – there’s actually 1,167 children, real children with real families, who tested positive for lead poisoning…. We know that Rhode Island has made great strides but that today too many children still have lead poisoning.” “But if you’ll remember what Dr. Shannon told you, he was asked if lead poisoning and the treatment of lead poisoning was a public health success story, and he said yes because the numbers have come down. But he said there’s been a plateau recently and that it is still a public health menace.”).

documenting the 2005 numbers. Yet, after learning that the new 2005 data directly contradicted its theme of the “plateauing” of declining lead levels, the State still allowed its “special assistants” to continue claiming that a plateau existed. According to the Defendants, this misrepresentation of facts is sufficient grounds for granting a new trial.

To compound the problem, neither the State nor its “special assistants” disclosed this relevant and critical information regarding the effectiveness of Rhode Island’s existing lead poisoning prevention program to the defendants after it became aware of the new data. The choice was made even though there was a discovery request seeking that very information. Defendants argue that the state breached its duty of candor to the Court and its Rule 26(e) duty to supplement its discovery responses. They claim that this undisclosed information was relevant to the heart of the issue in this trial – whether a public nuisance exists in Rhode Island – and was crucial to Defendants’ case.

To rebut this claim, the State and its special assistants claim that they were not obligated to supplement discovery because the Court ended discovery on May 30, 2005. Therefore, the State argues that the Defendants were obligated to go to the judge to seek an order requiring the

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748 Eliminating Lead Poisoning in Rhode Island, Draft Report (January 31, 2006) (noting that there were only 621 new cases of lead poisoning at the end of 2005). This draft report was provided as part of the Report Of The Interagency Council On Environmental Lead that was submitted to Rhode Island’s governor on March 17, 2006.

749 See February 29, 2006 Trial Excerpt, supra note 746; see also Trial Transcript, February 9, 2006, at 172 (“Ladies and gentlemen, we’ve reached a plateau. We’ve gone as far as the secondary measures of enforcement and the screening program can take us. And the only ones who should do more, I submit - that would be the first time for the defendants…”); Trial Transcript, February 9, 2006, at 81 (“Just because it’s a public health success story doesn’t mean that we leave the rest of the kids who have been unaffected behind. It’s not getting worse, but it’s not getting better.”).

750 Rule 26(e)(2)(b) states: “A party is under a duty seasonably to amend a prior response if the party obtains information upon the basis of which … (b) The party knows that the response, though correct when made, is no longer true or complete, and the circumstances are such that a failure to amend the response is in substance a knowing concealment.”

751 During the trial, on January 10, 2006, defendants asked counsel for the State to: “Please provide us with the most current version of the [LESS] database [Rhode Island’s Lead Poisoning database]. We understand that this version will include blood lead data through the end of 2005. As the State is scheduled to conclude its case shortly, please forward this….” Hearing Transcript, August 31, 2006, at 171. The State’s response was “… not only is this an overly burdensome request to comply with while in the midst of trial but discovery has been closed for over eight months.” Id at 171-72.
State to supplement its discovery. Moreover, the State claims that the Defendants waived their right to complain that the State did not supplement discovery because they did not seek a court order complying the State to supplement its discovery. The State argues that it would not be fair to inject the 2005 numbers into the case because it “totally changes the playing field of numbers.” Thus, according to the State, their duty to supplement ended once the trial began. Also, their arguments throughout trial were consistent with the facts as they existed before the trial started and the 2005 numbers were finalized.

The State also argues that its decision to deny providing the final 2005 numbers is not relevant to the case because: (1) the defense rested its case-in-chief without putting on any evidence, thus the defendants decided the jury did not need any additional information, and (2) the court previously ruled that because the State’s Attorney General brought a common law claim, it did not have to adopt the statutory definition of lead poisoning or what constitutes an elevated BLL that the State’s legislative assembly enacted as the law of the Rhode Island.

But, by refusing to supplement discovery and provide the defendants with the year-end 2005 data, the State denied the defendants an opportunity to show the jury, with the State’s own

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752 According to the State, if defendants wanted “additional discovery,” beyond May 30, 2005, they were to work it out by agreement if possible, or show the court good cause for the discovery and ask it to order the discovery. Hearing Transcript, August 31, 2006, at 189.

753 Id. at 245.

754 Id. at 246 ("On January 10 we sent an e-mail saying, 'Time out, we're in the middle of trial, not supplementing discovery anymore. We have been limited to the 2004 numbers. If we go and give the additional 2005 numbers, it totally changes the playing field of numbers. We don't agree. We are not going to give it to you.'").

755 Id. at 201 ("Now, in that time period as well, going chronologically, the defendants asked the State what – we were going to rely on data post 2004. Any of the 2005 data for trial. And we told them no. And that's what we did, Your Honor. We closed it at 2004 because that was the last full year of data that was available before the trial started. And in October, couple of months – couple of weeks before the trial started, we produced them – to the defendants the LESS database with the first three quarters of the 2005 data. We're still a couple of weeks pre-trial. We are post the May 30 cutoff. But by agreement we provided it. And we provided it, Your Honor, because we were pre-trial. Trial hadn't started yet, so they could have the information.").

756 Id. at 247.

757 Id. at 207-08.

758 Id. at 189 ("But what you said is the Department of Health can define lead poisoning any way they want in their rules and regulations, but that's got no bearing on how it's defined here.").
numbers, that there was no “plateau,” and it denied the jury the opportunity to weigh the credibility of the State’s claims using the best and most current information available.

While the State correctly notes that the court had a discovery cutoff date and a requirement to seek a court order for additional discovery if the State refused their requests, nothing in the rules “freezes” the truth. The 2005 data contradicted the 2004 data on which the State was relying to make its case. Nothing in the rules prohibited the State from telling the court, the jury and the defendants the truth. Even if the State is “technically” correct, this issue vividly illustrates the problem with states hiring private counsel as “special assistants” to prosecute these types of cases pursuant to contingent fee agreements. Indeed, it exposes the conflict between public service and private interests that undercuts the fundamental duties owed by those representing the sovereign, and leads to the inevitable inquiry of whether such alliances should ever be permitted.

The situation raises fundamental questions. Did the “profit motive” underlying the aggressive efforts of the “special assistants” cause the State’s counsel to forget the admonition of the United States Supreme Court that a government attorney’s duty is not necessarily to prevail, or to achieve the maximum recovery, in a particular case; rather, “the Government wins its point when justice is done in its courts?”

Did the rush to claim the contingent fee bounty blur the truth that a government attorney “is the representative not of an ordinary party to a controversy, but of a sovereignty whose obligation to govern impartially is as compelling as its obligation to govern at all,” and therefore the government attorney is required to use the power of the sovereign to promote justice for all citizens?

Even if the participating counsel deny these

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Berger v. United States, 295 U.S. 78, 88 (1935); see also, State v. Powers, 526 A.2d 489, 494 (R.I. 1987) (“the primary duty of a prosecutor is to achieve justice, not to convict”). It is beyond dispute that this solemn duty applies “with equal force to the government’s civil lawyers.”  
Freeport-McMoran Oil & Gas Co. v. Federal Energy Regulatory Comm’n, 962 F.2d
inquiries, does the situation nevertheless present an unacceptable “appearance of impropriety” – especially when representation of the public interest is concerned? These questions are undeniably worth asking – and if they are worth asking, they surely deserve better answers than procedural gamesmanship.

(2) Rhode Island’s Aftermath: Whose Settlement is it Anyway?

Before the start of the second trial, Patrick Lynch, Rhode Island’s Attorney General, announced that he settled the State’s claims against the DuPont Co. Interestingly, however, it appears that the settlement may not be a “settlement.” Both Lynch and DuPont say the deal was not a legal settlement but simply an agreement. Because it is not a settlement, DuPont is not giving money to the state. In return for dropping DuPont from its lawsuit, DuPont agreed to donate $12.5 million to charity. Moreover, because it was not a “settlement,” Lynch’s private law firms had to agree to waive their customary attorney fees.

Specifically, the settlement requires DuPont to donate $9 million to Children’s Health Forum, $1 million to Brown University and $2.5 Million to the Dana-Farber/Brigham and Women’s Cancer Center in Boston. Lynch’s office described the DuPont deal as a major victory for the state because at the time of the agreement, it was unclear whether Rhode Island would ever see a penny from the lawsuit that already had one trial end with a hung jury. At first blush, this settlement appears to be a reasonable deal for Rhode Island. However, as the old saying goes, “the Devil is in the details” – and those details have been coming to light in 2006.

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45, 47 (D.C. Cir. 1992) (Mikva, C.J.). Thus, it has long been recognized that a government lawyer in a civil proceeding should be held to a higher standard than a private lawyer, and that in civil proceeding “government lawyers have ‘the responsibility to seek justice,’ and ‘should refrain from instituting or continuing litigation that is obviously unfair.’” (citation omitted).

(i) **The Children’s Health Forum**

At the time of the settlement, Attorney General Patrick Lynch described Children’s Health Forum (“CHF”) as a national nonprofit organization focused on preventing childhood exposure to lead. He failed to mention, however, that (1) the Washington-based Children’s Health Forum was founded in 2002 by a lawyer hired by DuPont to work on lead poisoning issues; (2) it has received most of its funding from DuPont; and (3) most of its board members have ties to DuPont.\(^{762}\) Furthermore, CHF leases its office space from the Dewey Square Group, a high-powered Washington lobbying and public-affairs firm that DuPont uses as its consultant on “communications” issues, including lead paint. If that was not enough, CHF’s executive director, Olivia Morgan, is a partner in the Dewey Square Group.\(^{763}\)

Lynch’s spokesman claims that the attorney general did not know the group had a relationship with DuPont when he struck the deal, and DuPont is silent about whether it ever informed the attorney general about its relationship with CHF. While Lynch may have been ignorant about DuPont’s relationship with CHF, his chief of staff, Leonard Lopes, who sat in on talks with DuPont, was aware that there was a relationship between the two.\(^{764}\)

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\(^{762}\) Id. According to DuPont, CHF was founded in 2002 by Dr. Benjamin Hooks, a former head of the NAACP, after DuPont hired him to help the company address childhood lead poisoning. To assist him, Dr. Hooks enlisted the help of Kurt Schmoke, a former mayor of Baltimore, and former U.S. Secretary of Housing and Development Jack Kemp. Both have been consultants for DuPont. After receiving start-up money from DuPont, CHF was incorporated as a lobbying group on June 25, 2002. On December 10, 2003, CHF was incorporated as a nonprofit charity, making contributions tax-deductible. The following year, it received only one significant contribution — a $2 million donation from DuPont. When the state’s deal with DuPont was announced, four of CHF’s five board members had business ties to DuPont: Hooks, Schmoke, Kemp and Olivia Morgan, the group’s executive director who also works for lobbying firm Dewey Square Group, another DuPont consultant. The fifth board member, Antonio Villaraigosa, now mayor of Los Angeles, who has since left the board. Since then, CHF has added two other board members who DuPont said have no business ties to the company – former HUD Secretary Henry Cisneros and Booker T. Jones, founder of a job training group. Id.


\(^{764}\) See Smith, supra note 761. Attorney General Patrick Lynch also failed to disclose to the public that he accepted campaign donations from people with ties to DuPont, including a donation he accepted from its chief negotiator at the same time the deal was being discussed. According to Lynch, he did nothing wrong. Id. Others disagree. According to Robert Arruda, president of Operation Clean Government, a nonpartisan public watchdog in Rhode Island, the donation issue “does not pass the smell test, as far as I’m concerned.” He and others like him believe there is an aura of conflict of interest surrounding the deal. Id.
Thus, $9 million of the $12.5 million “agreement” is being controlled by a Washington D.C. based charitable group with extremely close ties to DuPont. Interestingly, there is no written agreement stating how CHF is to spend the DuPont donation. While CHF is supposed to dole the monies out to groups in Rhode Island, who apparently will seek it through an advisory commission set up by Lynch, CHF could arguably spend the money in any manner it chooses.

(ii) The International Mesothelioma Program at Brigham and Women’s Hospital

Although DuPont was unwilling to allow any money to be used as attorney’s fees, it was willing to donate an equivalent amount ($2.5 million) to charity and asked Lynch to identify which charity he wanted to receive the money. Instead of deciding which Rhode Island charity should benefit from the $2.5 million DuPont gift, Lynch asked Jack McConnell (Motley Rice’s lead lawyer in the lead-paint case) if he had a favorite charity. Mr. McConnell identified the International Mesothelioma Program at Brigham and Women’s Hospital in Boston, Massachusetts. Lynch honored Motley Rice’s request and told DuPont to make the gift to that program. As a result, the money is not going to a Rhode Island hospital, it is going to a Boston hospital. Moreover, mesothelioma is not related to any lead-based health hazard. Mesothelioma is a deadly cancer of the tissue surrounding the lungs that is caused by exposure to asbestos. Hence, millions of dollars generated by resolution of claims against a major defendant in a Rhode Island public nuisance case involving lead are going to a Massachusetts program that addresses asbestos-related illnesses. How such a diversion serves the public interest or benefits the public health of Rhode Island citizens is an unfathomable mystery.

Mike Stanton, Questions Persist Over Lynch’s Deal With DuPont: Political Opponent J. William W. Harsch Asks Why $2.5 Million Is Going To A Boston Hospital For A Cause Not Associated With Lead Poisoning, PROVIDENCE J., 2 (Aug. 9, 2006).
Motley Rice identified that charity because when the law firm joined the executive advisory board of the International Mesothelioma Program, it made a $3 million pledge to the program; a pledge that could be funded with monies raised from other sources, as opposed to a check written by the law firm or its lawyers. Thus, while Motley Rice agreed to waive its attorney’s fees, it saw no problem with using equivalent monies to fund the majority of the firm’s financial obligation to the mesothelioma program. Motley Rice, however, is not the only law firm wanting monies to go to this program. It turns out that another law firm Lynch hired to serve as co-counsel on this case – Thornton & Naumes – also sits on the board of the mesothelioma program and also has a $3-million pledge to the same program. Neil Leifer, a Thornton & Naumes lawyer who worked on the lead case, said it “seems reasonable” that his firm should also receive a credit toward its $3 million pledge to Brigham and Women’s.

Both Motley Rice and Thornton & Naumes attempt to justify the monies being used to settle their pledges because they both waived their legal fees associated with the Rhode Island case. Donald A. Migliori, an attorney with Motley Rice, told the press that: “[w]e’re not ashamed – this money isn’t going to pay our legal fees. Our law firm’s work in asbestos litigation over the years has enabled us to finance the lead-paint litigation for the past nine years.” Neil Leifer echoed a similar sentiment when he said it “seems reasonable” that his firm’s share of the waived legal fee should be credited toward the $3 million that it has pledged to Brigham and Women’s. “I’m not sure why it would be inappropriate.” Some people, however, see things a bit differently. Leonard Decof, one of the state’s original lawyers in the

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766 Id. According to Brigham and Women’s annual report, one of the requirements for have a seat on the executive advisory board of the International Mesothelioma Program is a pledge of $3 million. Id.
767 Id.
768 Id.
769 Id.
lead-paint case, argues that $2.5 million is a “de facto” legal fee, and that he is therefore entitled to a portion of this money for his past services.  

At this time, it is not certain whether any of the $2.5 million DuPont gift will be credited toward Motley Rice’s $3-million pledge. A spokesman for Brigham and Women’s said hospital officials have had no conversations with Motley Rice about whether the $2.5 million from DuPont will be credited toward the law firm’s pledge. According to DuPont’s spokeswoman, the company was not aware of Motley Rice’s ties to the mesothelioma program, but simply agreed to donate the $2.5 million to Brigham and Women’s as the charity designated by Lynch. DuPont has released a statement saying that it “has instructed the hospital that its payment should not be credited to any pledge or obligation of Mr. McConnell, his law firm, or any other entity.”

VI.

CONCLUSION

As this article has shown, nothing accomplished (or accomplishable) in any pending or resolved public nuisance case against lead paint manufacturers can reasonably, rationally or constitutionally “get the lead out” of our national environment. Indeed, the litigation and its surrounding hype has served to distract and delay public authorities from honestly evaluating confronting the true public health situation in their communities. This travesty is dangerously close to realization in some U.S. courts, particularly in Rhode Island.

In Rhode Island, the immediate public interest in protecting residents from lead contamination was ignored for years by indifferent public authorities, and even now nothing is being done to coerce landlords and property owners to remediate their contaminated residences. Meanwhile, the manufacturer defendants’ claims against those same persons for contribution are
severed and abated from the main action, which now proceeds against the manufacturers only. Moreover, all of the parties potentially responsible for lead contamination in Rhode Island are not before the court. Conspicuously absent are a legion of other manufacturers of lead-based paint. Even more surprisingly, none of the manufacturers and suppliers of tetraethyl lead, the greatest source of lead contamination in American history, are named as defendants. Indeed, the State and its assisting private counsel now insist that the defendants should be ordered to abate the “cumulative presence” of lead paint within Rhode Island to the point of eliminating any incidence of lead in children’s blood – notwithstanding the impact of alternative sources of lead not attributable to lead paint. Such an argument places the defendants in the impossible position not only of eliminating situations they did not create, but also of abating problems over which they have no control.

Even a casual reading of comments by plaintiffs’ counsel from the Rhode Island litigation reveals that they have distorted the law of product liability by grafting its principles into the law of public nuisance, a cause of action that was never designed to include those concepts.\footnote{See generally A. Sprague and F. Fitzpatrick, Getting the Lead Out: How Public Nuisance Law Protects Rhode Island’s Children, 11 Roger Williams U. L. Rev. 603 (2006).} By ignoring the presence of alternative sources of lead in the environment, the responsibility of landlords and property owners, and by ignoring the overwhelming contribution of inexplicably absent parties, while focusing narrowly on a few persons never shown to have contributed to contamination in a single household, they inexplicably blend product liability – a private remedy intended to serve the interests of individual parties – and public nuisance – a public remedy intended to serve the interests of the general population. Of course, this conflation is mirrored by the equally unjust fusion of the advocates responsible for advancing those views – elected public attorneys charged with protecting public health – and selected
private counsel who seek massive fees without political accountability to the persons they are charged to protect. This distortion stretches the otherwise salutary goals of product liability beyond any reasonable utility, and transforms the law of public nuisance into an oppressive tool designed to transfer huge amounts of wealth without any findings of personal involvement or responsibility.

This oppressive and distorted cause of action not only deprives defendants of a reasonable opportunity to be heard – a hallmark of procedural due process – but also disenfranchises citizens by distributing significant portions of recoveries to private law firms selected without meaningful public involvement. As a result, both the legislative branch of government – the branch responsible for appropriating money for public projects – and the executive branch – the branch responsible for ensuring those funds are properly applied for the public good – are circumvented by the judiciary – the branch least capable of investigating and resolving broad issues of public policy. Most significantly, these vast expenditures of time and resources against remote manufacturers delays remediation by those most immediately responsible, namely, the landlords and property owners who continue to expose residents to risks with impunity. Although these problems were created by overly zealous common law courts, those same courts, including the Supreme Court of the United States, have the power to solve them. Their failure to do so should surely motivate legislative intervention, but until that occurs, parties must insist upon the primacy of traditional nuisance law over the oppressive expansion of those principles, especially where the expansion serves narrow private interests, as opposed to the public interest generally.
VII.

THE AUTHORS