

Forensic Policy Considerations of Childhood Lead Toxicity

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Childhood lead toxicity remains a nationwide problem, despite significant progress in reducing exposure sources in the United States since the 1970s. Neurodevelopmental sequelae of lead exposures often persist into adulthood, with limited reversibility. These include long-lasting deficits in intelligence, impulse control, and emotional and behavioral regulation, which can increase one's propensity for aggression and criminality. Yet, despite the strong association between lead poisoning and criminality, there is little agreement thus far among U.S. jurisdictions about how to apply such knowledge to criminal legal proceedings. Here, we explore forensic considerations of childhood lead toxicity for evaluation, sentencing, and staff support in the criminal justice system. We cite both clinical science and case law in navigating this complex intersection of forensic psychiatry, public health, and the environment. We aim to provide preliminary guidance on how forensic psychiatrists can approach and refer to lead toxicity in their medicolegal work. This article may hopefully inspire further forensic research on the topic and the eventual creation of standardized, lead-related guidelines for criminal legal proceedings.

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Many U.S. physicians picture childhood lead poisoning as a problem of the past. Although Americans' lead exposure has declined by over 95 percent since the 1970s, it remains at 10 to 100 times as high as the lead burden of people who lived in preindustrial times.¹ National bans in the past few decades on residential lead paint and leaded gasoline for motor vehicles have produced considerable societal benefits, yet childhood exposures still occur. These chronic, environmental exposures can induce long-lasting neurodevelopmental, cardiovascular, renal, and other organ-related damages.²⁻⁴ Moreover, high-risk, acute sources can emerge even in developed countries like the United States, as shown by the release of inhabitable, air-based lead in the destructive Los Angeles wildfires in January 2025.⁵

A May 2024 federal report highlighted multiple sources of childhood lead exposure that the federal government has made renewed efforts since 2018 to reduce.⁶ Examples of described remaining sources included older, deteriorating homes with chipping

lead paint, millions of extant lead pipelines that can leach into municipal water supplies, soil deposits of lead, and industrial air emissions. These disproportionately affect lower socioeconomic communities of color nationwide.^{7,8} Environmental sources of lead continue to merit government-sanctioned, targeted efforts at removal because of their prolonged decay rate and prevalence. Of note, soil-based lead's half-life is estimated to be over 700 years long.⁹

We acknowledge societal progress made over the past few decades in reducing U.S. childhood lead poisoning but also recognize it as still problematic, and even devastating, for many young Americans. We aim in this article to highlight not only the phenomenon's pathophysiology and neurodevelopmental implications but also its significance in the criminal justice system. To this end, we explore the existing body of forensic-relevant case law on lead exposure and toxicity, as found through the LexisNexis database. We wish to increase forensic psychiatrists' awareness of legal case precedent pertaining to lead toxicity-related criminality, sentencing implications and challenges, and potential helpful community interventions.

Public Health and Pathophysiology

Researchers now understand there is no safe threshold for blood lead level in the human body.^{9,10} Likewise,

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there is no known natural, human physiological role for lead.¹¹ Prolonged low-level exposures, which are often subtle and unrecognized, can still exert insidious and life-long damage beginning at an early age. Higher concentrations of lead exposure induce further harms in a dose-dependent manner. Moreover, children are more sensitive to exposures as they physiologically absorb four to five times as much lead per unit of ingestion as adults do.¹² From a psychiatric perspective, neurodevelopmental effects include decreased intelligence, impulse control, attention span, and educational completion as well as increased antisocial behavior and aggression.^{8,9,13–16} These have profound implications for forensic psychiatry in both the juvenile and adult criminal justice systems.

A large body of historical public health statistics align with such cognitive effects. Many studies support the hypothesis that U.S. criminality rates have directly correlated with societal lead exposure amounts.^{7,8,17,18} With an approximately 20-year latency period reflecting children's growth into young adults, U.S. violent crime rates appeared to increase following the rise of lead paint and leaded gasoline use throughout the early and mid-twentieth century.¹⁹ Likewise, violent crime rates declined significantly within two decades after associated bans in the later 20th century.^{18,19} Although lead toxicity cannot explain all of U.S. society's fluctuations in criminality over the past century, it is one tangible contributor. A meta-analysis suggests environmental lead reductions may explain between seven and 28 percent of the recent decrease in U.S. homicides.²⁰

Medical science supports a distinct explanatory mechanism for lead exposure's long-term effects on cognition and behavioral regulation. Lead potently competes with and physiologically mimics calcium in the body.² In this way, it is toxic to all human organs. From a skeletal perspective, such competition with calcium means absorbed lead partly deposits into human bones, which intermittently release the element into the bloodstream over time.²¹ Regarding the human nervous system, lead dysregulates the usual neurotransmitter functions of calcium. The brain is the human organ most sensitive to lead toxicity.¹⁰

Lead enters the circulatory system typically following ingestion or inhalation. From there, it readily crosses the blood-brain barrier.² Lead inappropriately activates or interferes with typically calcium-mediated processes in the developing brain, especially the calmodulin-dependent and brain-derived neurotrophic

factor-signaling pathways.^{22,23} This can adversely restrict a range of associated processes crucial in childhood brain development, including synapse formation, axonal dendritic growth, and neuroplasticity.²⁴ Lead also disrupts the neurotransmitter releases of dopamine and gamma-aminobutyric acid (GABA).²⁴ It triggers cellular excitotoxicity by reducing glutamine synthetase activity, increasing glutamate accumulation, and harming mitochondrial activity, contributing to neuronal apoptosis.²⁵ Consequently, lead delays myelin formation and accumulation, stunting long-term myelination and causing long-term damage to the developing brain.²⁶

Lowered brain structural integrity and cerebral volumes, including of both gray and white matter, have been observed on magnetic resonance imaging (MRI) in adults with childhood histories of lead toxicity.^{27,28} Notably, gray matter appears especially affected in medial portions of the prefrontal cortex, including in the anterior cingulate cortex and ventrolateral prefrontal cortex.²⁸ These regions are directly involved in mood regulation, decision-making, and executive functioning. Areas influencing impulse control, including the inferior frontal gyrus and supplemental motor area, also demonstrate gray matter reductions.^{29,30} Conversely, lead-related neuroanatomical damage to such areas collectively correlates with worsened emotional regulation, inattention, impulsivity, and executive functioning deficits. These deficits are directly associated with increased aggression and decreased self-control. These two features, particularly when combined with opportunity, are believed to contribute to higher crime rates, including rates of violent crime.²⁰

Additionally, childhood lead toxicity has been well studied as contributing to long-term decreases in intelligence. Brain imaging research supports lead-related decreases in subcortical brain volume, especially in parts of the corpus callosum, as contributing to such cognitive loss.³¹ Curiously, similar callosal volume decreases have been observed in youth with neurodevelopmental deficits arising from prenatal exposures to alcohol or cigarette smoking.³¹ When accumulated to a functionally impairing extent, such exposure-induced neurocognitive deficits can raise questions as to criminal defendants' ability to fully understand the nature and consequences of their actions.

Forensic Literature Search

Despite the strong association between lead exposure and crime, jurisdictions throughout the United

States have little experience or agreement in their application of this knowledge in criminal legal proceedings.³² The current body of law on lead toxicity focuses largely on environmental regulation, tort law, and other civil matters. A legal search of all state and federal criminal jurisdictions in the LexisNexis database was performed using the search terms “lead exposure” and “lead toxicity.” The search included all cases in the “Criminal Law and Procedure” database, with no exclusions based on date of publication. This LexisNexis search generated 33 cases. On further review, 29 cases were excluded as noncriminal; these were based on various civil and family law theories, including corporate negligence, products liability, fraudulent medical billing, workers’ compensation, and a due process right to bodily integrity. One case was criminal but dealt with lead exposure of the victim rather than the defendant. Of the remaining three cases, two were unpublished opinions, and the lone published criminal case made only passing mention of lead exposure as one of many factors considered by a lower court.³³ Thus far, published cases involving lead toxicity in the criminal justice system are quite rare, offering little guidance on forensic approaches to a well established medical condition that, per recent biomedical literature, correlates with higher propensity for future criminal behaviors.

Criminal Responsibility

Society’s interest in holding an individual accountable for a crime is based largely on the concept of culpability, which considers ambiguous concepts of free will as well as more quantifiable medical concepts of cognitive functioning. States incorporate these concepts into criminal legal systems in various ways, including insanity defenses and *mens rea* requirements.³⁴ Depending on the jurisdiction, states may consider many different factors in determining culpability, including elements of cognitive incapacity, moral incapacity, and inability to conform behavior.³⁴ Several landmark cases over the past few decades, including the decisions in *Atkins v. Virginia*,³⁵ *Miller v. Alabama*,³⁶ and *Moore v. Texas*,³⁷ suggest increasing judicial openness to consideration of neuroscientific data when evaluating criminal responsibility. In *Atkins*, the Supreme Court found the execution of intellectually disabled convicts to be in violation of the Eighth Amendment prohibition on cruel and unusual punishment.³⁵ Writing for the majority, Justice Stevens based

this holding in part on conclusions that adults with intellectual disability have reduced culpability and societal interests in retribution and deterrence were not served by the execution of intellectually disabled adults.³⁵ Although the holding in *Atkins* was limited to capital cases, the opinion suggests that courts may find evidence of the sequelae of lead toxicity to be relevant in determination of guilt.

When considering the theoretical aims of criminal law, the justification for punishing individuals with severe impairments from lead toxicity is diminished. In theory, a society’s interest in retribution should correspond to the level of culpability of the accused, which may be decreased by factors such as intellectual deficits and impulsivity.^{36,37} Deterrence may not be a reasonable justification in cases of involuntary toxin exposures, particularly as lead exposure is most toxic in early childhood, when individuals have the least control over their physical environment. Society may benefit from sequestering certain people with histories of lead exposure, but considerations of dangerousness must be balanced against individual due process rights. Finally, rehabilitation is a noble goal for individuals with disorders of cognitive development, but existing correctional system resources may not be suitable for this population’s specific needs.

Lead exposure involves the unintentional and insidious ingestion of a toxic substance during important developmental windows, with specific and permanent cognitive effects. Courts have allowed, and at times been persuaded by, evidence of toxic exposures,^{38,39} but not all toxic exposures are treated the same.³⁹ As with lead toxicity, for instance, there is little legal precedent for the use of fetal alcohol spectrum disorder in criminal responsibility arguments, although the cognitive outcomes suggest application as evidence of diminished capacity.³⁸ A severe, long-lasting effect from a time-limited exposure to an environmental toxin with well established effects may be more well received as a defense than chronic exposures with vague or impermanent effects.³⁵ External factors, such as neighborhood environments and nutritional deficiencies, may be less persuasive in criminal courts than internal factors, such as malignancy or schizophrenia, in part because of weaker causal linking to the crime.³⁴

One of the earliest examples of toxic exposure used in an affirmative defense was *U.S. v. Frank*.³⁹ In this case, defendant Terrance Frank argued that he was temporarily insane because of uranium exposure on the Navajo reservation where he grew up,³⁹

leading to his having subsequent impairing symptoms of depression and mental retardation. Mr. Frank's not guilty by reason of insanity defense was not successful, as he was convicted of second-degree murder and related charges.³⁹ The verdict was likely influenced by complicating factors, including Mr. Frank's acute alcohol intoxication at the time of the crime.^{39,40} Based on his brain injury, however, jurors may have found that he was not able to form the requisite level of intent required for a first-degree murder conviction.^{39,40}

In *People v. Bradt*, an unpublished California case, Arent Bradt argued that he was unable to distinguish between neutral and threatening behavior because of a combination of childhood lead exposure, head injuries, trauma, and psychosis.⁴¹ Rather than an insanity defense, Mr. Bradt made an imperfect self-defense argument that would permit conviction for manslaughter but negate the *mens rea* requirement for a murder conviction. In an outcome similar to *Frank*, the jury acquitted Mr. Bradt of first-degree murder but ultimately rejected his imperfect self-defense argument and convicted him of second-degree murder.⁴¹ With so many factors that could affect mental state, it is impossible to know how much Mr. Bradt's history of lead exposure influenced the verdict.

Depending on local laws, lead exposure with severe negative outcomes, such as intellectual disability, could be offered as evidence in consideration of criminal responsibility. There is some precedent for introducing neurodevelopmental and neurocognitive disorders through insanity defenses in some states, although successful acquittal by reason of insanity is rare for autism or intellectual disability without a comorbid psychiatric disorder.⁴² A few jurisdictions explicitly exclude developmental disorders or impulse control disorders without psychiatric comorbidity from consideration in insanity defense criteria.⁴² In some cases, an insanity defense based on neurodevelopmental disorder alone may be irrelevant, as a defendant with impairment significant enough to meet criteria for an insanity defense may be found incompetent to stand trial and not restorable.⁴² Although evidence for use in an insanity defense is limited, based on the few related cases available, evidence of specific effects of lead toxicity could contribute to a successful negation of criminal intent.

Mitigation

States or fact finders who reject toxic exposures as a defense to a crime may still be open to consideration

of the effects of toxicity in determining a sentence. In *Atkins v. Virginia*, Justice Stevens discussed the limitations of individuals with intellectual disability that reduce governmental interest in punishment, including cognitive impairments and impulsive tendencies.³⁵ Justice Stevens concluded, "Their deficiencies do not warrant an exemption from criminal sanctions, but they do diminish their personal culpability" (Ref. 35, p 305). In line with this reasoning, introduction of lead toxicity records as mitigating evidence could add value in cases where defendants have some level of impairment, but not rising to the level required under a state's insanity defense.

In capital cases, defendants are afforded wide latitude in the evidence they may offer, particularly in the sentencing phase. Neurological evidence, such as imaging and cognitive testing, is introduced in 25 percent of death penalty cases.⁴³ In *Caro v. Woodford*, the Ninth Circuit Court of Appeals considered a toxic exposure as mitigating evidence in a capital case.⁴⁴ After being convicted of murder, kidnapping, and assault and sentenced to death, Fernando Eros Caro was granted an evidentiary hearing on claim of ineffective assistance of counsel. Evaluating medical experts, including a neurologist, neuropsychiatrist, and toxicologist, testified to evidence of brain injury from Mr. Caro's extensive exposure to neurotoxic pesticides during his childhood. Symptoms ranged from memory loss, depression, and autonomic abnormalities to inappropriately present snout and suck reflexes. These signs were deemed well established by literature as consistent with long-term sequelae of pesticide toxicity. Despite apparent signs of Mr. Caro's adequate intelligence, such as his intelligence quotient (IQ), high school grades, and performance in the Marines, all three experts noted that none of these signs were inconsistent with frontal lobe injury resulting from pesticide exposures and physical trauma. The court concluded that physiological defects had contributed to Mr. Caro's "impulse discontrol" and "irrational aggressiveness." After determining that earlier failure to present such testimony had prejudiced the prior jury, the appellate court vacated Mr. Caro's death sentence and ordered a new sentencing hearing.

In *Dickerson v. Stirling*, the South Carolina District Court considered an appeal based in part on failure of counsel to adequately explore and present evidence of lead toxicity. William Dickerson was sentenced to death based on convictions for murder, kidnapping, and criminal sexual conduct.⁴⁵ The appellate court

found that the defense had in fact explored Mr. Dickerson's childhood lead exposure, noting that all but one record of exposure had been destroyed. Mr. Dickerson then refused to participate in neuropsychological testing to assess his cognitive condition and support a lead exposure argument. Despite these setbacks, the defense relied on social history to present evidence of "extremely high" lead levels in Mr. Dickerson's childhood home, the effects of lead toxicity during childhood, and the possibility that Mr. Dickerson's abnormal electrocardiogram (EKG) was related to lead exposure. As one forensic psychologist for the defense put it, lead toxicity was one of "a catastrophically cumulative assortment of risk factors" that contributed to the crime (Ref. 45, p 44). Ultimately, the appellate court determined that, in light of a large volume of aggravating evidence, the exclusion of additional evidence regarding lead exposure would have been unlikely to change Mr. Dickerson's sentence.

The introduction of evidence of toxic lead exposure in noncapital cases has even less precedent. In the unpublished case of *State v. Olbert*, the trial court considered lead exposure as one of a "constellation of factors" contributing to a juvenile defendant's criminal behavior.⁴⁶ On appeal, the Superior Court of New Jersey noted that the defense "identified no published opinion from any jurisdiction in the United States that has treated lead poisoning as a mitigating factor in sentencing" (Ref. 46, p 16). The court declined to commit to a requirement for lower courts to consider lead poisoning evidence in sentencing, but the inclusion of lead exposure evidence in this case was reasonable.

Even when permitted by a court, presenting evidence of the effects of lead exposure carries risk. Certain permanent deficits attributed to lead toxicity, such as increased impulsivity and aggression, could be viewed as aggravating rather than mitigating factors. Courts have wide discretion in interpreting psychiatric and neurological evidence, and they are not required to find even credible evidence of impairment to be mitigating.

Challenges in Evaluation

According to the American Academy of Psychiatry and the Law (AAPL) Practice Guideline for the Forensic Assessment, evaluators must collect a thorough medical history and assess for co-occurring medical conditions that may "contribute to criminal behavior and help the evaluator to understand it"

(Ref. 47, p S17). In assessing medical contributors to an individual's criminal behavior, psychiatrists should consider toxic exposures, including lead toxicity.

Uncovering evidence to support the use of lead poisoning as a factor in sentencing can present a real challenge. Few adults retain their childhood medical records, and pediatrics practices are not required to store records indefinitely. Preserving and accessing this type of evidence may become easier over time, as electronic medical records replace paper records and patients have greater access to their medical records under modern federal laws. Records of family members or other children raised in the same apartment building, neighborhood, or water supply catchment may be useful clinically, but they may not meet the evidentiary standards for relevance or probative value.⁴⁸ Lead toxicity is a diagnosis based on objective lab values. Given lead's substantial absorption rate into blood from dietary and inhaled sources, the most commonly utilized diagnostic biomarker of lead exposure is blood lead (BPb), primarily from erythrocytes.² There is not, however, an exact correlation between a transient BPb level noted in childhood and the level of cognitive or behavioral change later in life for a specific individual. Lead also does not accumulate in bones in children to the extent that it does in adults.⁴⁹ Moreover, adult bone lead measurements may not be reflective of childhood lead exposures.⁵⁰ Evidence of lead exposure may also be excluded as cumulative, as in many cases, lead exposure coincides with many other criminogenic risk factors. For example, poverty correlates with both lead toxicity and criminal activity.^{51,52} Children with nutritional deficiencies or nontoxin-related developmental disorders are at higher risk for exposure, related in part to their higher rates of pica.⁵³ Evidence of lead toxicity may be overshadowed by factors such as substance use and serious mental illness.

As in any forensic evaluation, malingering must be considered in cases of reported lead toxicity. Even with a confirmed elevated blood level in childhood medical records, evaluators must assess for consistency between reported functional deficits and real-life functioning.⁴² In cases of discrepancy, evaluators may consider administration of measures of cognitive symptom exaggeration.⁴² Moreover, the majority of medical evidence underlying the forensic implications of lead toxicity is based on correlation. In reports and testimony, forensic psychiatrists must avoid overgeneralizing conclusions from medical literature in individual cases.

Accommodations for Lead Toxicity Sequelae

Professionals in the criminal justice system, including attorneys, judges, probation officers, and correctional officers, can learn to work with individuals with cognitive delays related to lead exposure. This may include the use of simple language, concrete verbiage, and cross-questioning as well as avoiding compound questions.⁴² Additionally, explaining to correctional system staff the neurodevelopmental toxicity-related context of these inmates may help staff better understand and sensitively work with them.

It may be beneficial to offer educational and vocational supports appropriate for the cognitive capacity of inmates with a history of lead toxicity. As with individuals with neurodevelopmental disabilities in general, inmates affected by early-life lead poisoning may have difficulty understanding, focusing on, and completing vocational coursework that most other inmates can manage. For those with lead-associated neurodevelopmental deficits who could someday reintegrate into the community, suitable community supports can help motivate and facilitate their challenging transition back. Legal precedent supports the rights of inmates with disabilities, presumably including lead toxicity-related cognitive deficits, to receive reasonable accommodations to help them succeed in educational and vocational programs available to fellow inmates.⁵⁴

Conclusion

In recent decades, government efforts to contain toxic lead exposure have been mostly successful. Yet, despite these successes, lead poisoning remains a public health hazard that disproportionately affects vulnerable groups. As psychiatrists advocate for prevention efforts in the criminal justice system, environmental factors should not be forgotten or left out. As more case law emerges, forensic psychiatrists can further develop guidelines for evaluating justice-involved individuals with lead-induced cognitive symptoms.

In the forensic setting, experts should carefully consider toxic lead exposure when presented with specific cognitive findings. Forensic experts should be aware of the challenges of presenting lead toxicity as a mitigating factor, including loose causal linking, comorbidity with other risk factors for crime, and the possibility of lead exposure sequelae comprising an aggravating, rather than mitigating, factor. Cognitive and behavioral sequelae of lead toxicity present an underexamined yet pivotal intersection

among forensic psychiatry, public health, and the environment.

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