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Contribution of childhood lead exposure to psychopathology in the US population over the past 75 years

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Background: More than half of the current US population was exposed to adverse lead levels in childhood as a result of lead's past use in gasoline. The total contribution of childhood lead exposures to US-population mental health and personality has yet to be evaluated. **Methods:** We combined serial, cross-sectional blood–lead level (BLL) data from National Health and Nutrition Examination Surveys (NHANES) with historic leaded-gasoline data to estimate US childhood BLLs from 1940 to 2015 and calculate population mental-health symptom elevations from known lead-psychopathology associations. We utilized five outcomes: (1) General Psychopathology "points", reflecting an individual's liability to overall mental disorder, scaled to match IQ scores ($M = 100$, $SD = 15$); (2) Symptoms of Internalizing disorders (anxiety and depression) and Attention-deficit/Hyperactivity Disorder (AD/HD), both *z*-scored ($M = 0$, $SD = 1$); and (3) Differences in the personality traits of Neuroticism and Conscientiousness ($M = 0$, $SD = 1$). **Results:** Assuming that published lead-psychopathology associations are causal and not purely correlational: We estimate that by 2015, the US population had gained 602-million General Psychopathology factor points because of exposure arising from leaded gasoline, reflecting a 0.13-standard-deviation increase in overall liability to mental illness in the population and an estimated 151 million excess mental disorders attributable to lead exposure. Investigation of specific disorder-domain symptoms identified a 0.64-standard-deviation increase in population-level Internalizing symptoms and a 0.42-standard-deviation increase in AD/HD symptoms. Population-level Neuroticism increased by 0.14 standard deviations and Conscientiousness decreased by 0.20 standard deviations. Lead-associated mental health and personality differences were most pronounced for cohorts born from 1966 through 1986 (Generation X). **Conclusions:** A significant burden of mental illness symptomatology and disadvantageous personality differences can be attributed to US children's exposure to lead over the past 75 years. Lead's potential contribution to psychiatry, medicine, and children's health may be larger than previously assumed.
Keywords: Lead; psychopathology; personality; cohort; population burden.

Introduction

Lead is a potent neurotoxicant believed to be harmful at any level of exposure. Further, likely much more than half of the US population were exposed to neurotoxic levels of lead in early childhood. While lead exposure is still routine in many communities in the United States and around the world, lead exposures peaked from 1960 through 1990 in most industrialized countries due to the peak use of lead in gasoline (phased out in United States by 1996). Recent estimates of legacy-lead exposure owing to lead's past use in gasoline indicate that 170 million Americans were exposed as young children (ages 0–5 years) to lead levels greater than current (3.5 μg/dL) and previous (5 μg/dL) reference values for clinical follow-up (McFarland, Hauer, & Reuben, 2022). Nearly one-third of these children (60 million) were exposed to 3–6 times the previous reference value.

These levels of lead exposure can damage the brain, heart, kidneys, liver, and bones, as determined through hundreds of human observational studies and causal mechanistic animal-model studies (ATSDR, 2020). Despite this evidence, the full contribution of legacy lead exposure to trends in population health and behavior over the past century has yet to be fully evaluated.

Lead exposure in early life can be particularly disruptive to brain development, resulting in lowered cognitive ability, fine motor skills, and emotional regulation capacity (Bellinger, 2008; Bouchard et al., 2009; Braun, Kahn, Froehlich, Auinger, & Lanphear, 2006; McFarland et al., 2022; Reuben et al., 2019; Winter & Sampson, 2017). While the contribution of legacy lead exposure to US population brain health has yet to be fully estimated, one study recently determined that legacy lead exposures cost the US population over 800 million IQ points (McFarland et al., 2022), a meaningful measure of general cognitive capacity and population brain integrity.

Another way to conceptualize lead's contribution to population brain-health is through elevations in mental disorders and disorder symptoms. While the epidemiological evidence is observational and not causal, childhood lead exposure has been robustly associated with increased symptoms of a variety of

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mental disorders spanning internalizing conditions, where distress/dysregulation is inwardly expressed (e.g., depression and anxiety) (Roy et al., 2009; Searle et al., 2014; Winter & Sampson, 2017), externalizing conditions, where distress is outwardly expressed (e.g., conduct disorder, hyperactivity) (Bouchard et al., 2009; Donzelli et al., 2019; Goodlad, Marcus, & Fulton, 2013; Marcus, Fulton, & Clarke, 2010), and the higher-order factor of General Psychopathology (the so-called '*p*' or *psychopathology* factor) (Reuben et al., 2019), which indexes an individual's overall liability to mental illness across diagnostic categories and across the lifespan (Caspi & Moffitt, 2018). Finally, lead has also been associated with disadvantageous alterations in personality that reflect greater propensities to negative emotionality (Neuroticism) and lower capacities for effortful control of impulses and pursuit of goal-directed behavior (Conscientiousness) (Reuben et al., 2019; Schwaba et al., 2021)—personality differences that are associated with lower levels of health, wealth, and happiness across the lifespan (Friedman, 2019; Moffitt et al., 2011). The full extent to which early life lead exposure has affected the psychological health of the US population is unknown.

Here, this gap is addressed, in part, by combining published estimates of the legacy-lead exposure of the US population attributable to leaded gasoline, a primary historical source of lead exposure, with published associations of lead exposure with continuous, non-dichotomous mental health outcomes that reveal dose–response relationships across the distribution of lead exposures *and* full dimensionality, severity, and comorbidity of mental illness symptomatology. Specifically, this study combines these data to: (1) estimate lead contributions to the broadest previously-evaluated measure of lead and mental illness symptomatology, the continuous General Psychopathology factor (Reuben et al., 2019), spanning all major Diagnostic and Statistical Manual of Mental Disorders (DSM-5) categories of mental disorder; (2) test lead contributions to two narrower psychopathology domains previously-evaluated continuously with respect to lead, Internalizing symptoms (indexing anxiety and depression) and AD/HD symptoms (indexing inattention, impulsivity, and hyperactivity) (Winter & Sampson, 2017); and (3) test lead contributions to the non-clinical, personality domains that influence an individual's capacity to lead a successful life through emotional and behavioral control, the lead-linked personality traits of Neuroticism and Consciousness (Schwaba et al., 2021). Taking as an assumption that published lead-psychopathology associations are causal, our results demonstrate the contribution of early-life lead exposure to the psychological health of the US population over the past century.

Data

Lead exposure

This study uses previously published estimates of lead exposure experienced by the US population in early life (ages 0–5 years) attributable to leaded gasoline, created by McFarland et al. (2022). These data contain estimates of early life blood-lead levels (BLLs) for each five-year age group from ages 0–4 to 85+ in the US population in 2015. McFarland et al. (2022) used data from the US Census, leaded gasoline consumption, and cross-sectional BLL data from the National Health; and Nutrition Examination Survey (NHANES), a nationally representative sample of US children aged 1–5 from 1976 to 2015. The NHANES and leaded gasoline consumption data were used to estimate BLLs for 1940 to 2015 birth cohorts. They estimated the number and proportion of current adults that fell within seven BLL categories as children (<4.99; 5–0.9.99; 10–14.99; 15–19.99; 20–24.99; 25–29.99; and ≥30 μg/dL), by year and birth cohort.

Outcome measures

Outcome measures included the broadest, dimensional metrics of psychopathology and personality previously published in relation to lead exposure along the full distribution of non-occupational lead-levels.

General Psychopathology indexes an individual's overall liability to mental illness, an approach that takes into account the dimensional nature of mental disorder, disorder symptom severity, and the high concurrent and sequential comorbidity seen across all psychiatric disorders (Caspi et al., 2020; Caspi & Moffitt, 2018). Childhood lead exposure has been robustly associated with elevations in General Psychopathology across adulthood (Reuben et al., 2019) in the New Zealand-based Dunedin Study, which utilized multiple waves of diagnostic clinical interviews over four decades. Utilizing Dunedin Study coefficients, this study estimates how lead exposures could have changed the distribution of psychopathology in the American population across the past 8 decades. The psychopathology curve is normally distributed (typically scaled in studies to match the IQ scale; $M = 100$, $SD = 15$), allowing us to estimate changes to all sections of the curve. This includes those at the highest levels who experience severe mental illness requiring continual treatment and/or hospitalization, as well as those at lower levels of psychopathology, who may have been unlikely to develop a mental disorder in the absence of lead exposure. Appendix S1 provides more information on how this measure was constructed.

Internalizing Symptoms index internally directed experiences of psychiatric distress related to anxiety and depression symptoms, including unpleasant

physiological arousal, excessive worry, withdrawal, and low mood. Childhood lead exposure has been robustly associated with Internalizing Symptoms by adolescence (Winter & Sampson, 2017) in the US-based Project on Human Development in Chicago Neighborhoods study, which utilized parent reports on the Achenbach System of Empirically Based Assessment (ASEBA) Child Behavior Checklist (CBCL) 'anxiety or depression' scale. The scale asks parents to report whether their child has experienced problems within the past 6 months such as: sudden changes in mood or feelings; feels or complains that no one loves him or her; feels worthless or inferior; is too fearful or anxious; and acts unhappy, sad, or depressed. The association of child lead exposure and internalizing symptoms have been replicated in other samples in other countries (Bouchard et al., 2009).

AD/HD Symptoms index executive function difficulties that manifest as problems related to inattention, impulsivity, and hyperactivity. Estimates were once again obtained from Winter and Sampson (2017), which utilized the ASEBA CBCL Attention Deficit/Hyperactivity Problems scale. The scale asks parents to report on whether their child experiences problems (rated as often (2), sometimes (1), or not (0) true). Items tap into whether adolescents: can't concentrate; can't pay attention for long; can't sit still, are restless, or hyperactive; act confused or seem to be in a fog; and are impulsive or act without thinking. The association of child lead exposure and AD/HD symptoms has been extensively replicated in other samples in multiple countries and summarized in two separate meta-analyses (Donzelli et al., 2019; Goodlad et al., 2013).

Neuroticism and *Conscientiousness*. Neuroticism indexes an individual's tendency towards negative emotionality and Conscientiousness indexes an individual's tendency to be organized, controlled, and capable of successfully pursuing goal-directed behavior across different settings. Personality estimates derive once again from Reuben et al. (2019), which reported robust associations of childhood lead exposure with adult Neuroticism and Conscientiousness as measured across adulthood via self and informant reports at multiple study waves on the 25-item version of the Big Five Personality Inventory, which also measures extraversion, openness to experience, and agreeableness. These findings have been replicated at the nation-level in the United States and Europe (Schwaba et al., 2021).

Analytical strategy

The first analytic step was to estimate the proportion of Americans exposed to lead from 1940–2015. Representative estimates were taken by NHANES from 1976–2015. In order to obtain estimates from 1940–1975 we combined observed BLL estimates from 1976 to 1993 NHANES data with yearly leaded gasoline consumption information obtained from the now defunct US Bureau of Mines which monitored the amount of lead-based gasoline consumed in the United States each year from 1933 to 1993. We regressed each of the seven BLL categories on leaded gasoline consumption. For each of these regressions, we estimated a linear model, a quadratic model, and a cubic model. The regression with the best model fit was chosen and predicted BLL values were obtained for the years under question. See McFarland et al. (2022) for more information. BLL estimates are shown by five-year birth cohort designations in Table S1.

Lead-associated changes in psychological symptoms were calculated in the following manner. First, midpoints for the 5 μg/dL-unit BLL categories from McFarland et al. (2022) were utilized (e.g., 7.5 μg/dL for the 5–9.9 μg/dL category). Second, regression coefficients for the association of lead exposure in 5 μg/dL-unit-increments and our outcomes of interest were selected from Reuben et al. (2019) where the General psychopathology coefficient = 1.34; Neuroticism = 0.10; Conscientiousness = −0.14, and Winter and Sampson (2017), where the internalizing problems coefficient = 0.45, and AD/HD symptoms = 0.30. Third, the regression coefficients were multiplied by each midpoint and the previous values were summed to generate the number of psychological symptom 'points' lost or gained for each lead exposure category. Fourth, the symptom points were multiplied from each lead category by the number of people who were exposed to that level of lead exposure in early life. This generates the total psychological points gained or lost for each level of lead exposure. These numbers were also summed to generate the total number of points from all levels of lead exposure. Fifth, this total number of points was divided by the size of the population to give a measure of the average number of points lost or gained in the population per person. Sixth, 95% confidence intervals were calculated by utilizing the confidence intervals reported in Reuben et al. (2019) and Winter and Sampson (2017).

Because BLLs changed considerably between 1940 and 2015, calculations were repeated for each five-year birth cohort starting in 1940 for each outcome of interest. A ridge plot was used to visualize the trends in mental health symptoms. These calculations were performed, along with margins of errors for each estimate in Excel (reported in Table S2).

Results

Table 1 shows the cumulative estimated changes in American psychological symptoms from 1940 to 2015 attributable to early life lead exposure over the past 75 years arising primarily but not exclusively from lead use in gasoline. As the table depicts, the US's experience with leaded gasoline has

Table 1 Mental Health Losses as of 2015 due to Lead Exposure in Early Life

	Pts. gained/lost[a]	95% Confidence interval	Pts. gained/lost per person[b]	95% Confidence interval	Standard deviation
Mental disorder					
General Psychopathology	602,585,856	52,079, 884 to 1,153,091,829	1.8921	0.1635 to 3.6206	15.0000
Internalizing Symptoms	202,360,922	175,415,443 to 229,306,400	0.6354	0.5508 to 0.7200	1.0000
AD/HD symptoms[c]	134,907,281	107,925,825 to 161,888,738	0.4236	0.3389 to 0.5083	1.0000
Personality					
Neuroticism	44,969,094	7,904,438 to 82,033,749	0.1412	0.0248 to 0.2576	1.0000
Conscientiousness	−62,956,731	−111,351,004 to −14,562,459	−0.1977	−0.3496 to −0.0457	1.0000

[a]This column represents the total number of mental health symptom "points" gained in the full population.
[b]This column represents the average loss in mental health per person in the population.
[c]AD/HD stands for Attention-deficit/Hyperactivity Disorder.

contributed to significantly worse mental health in the population.

By 2015, the US population had gained 602 million General Psychopathology (*p*-factor) points as a result of childhood lead exposure, reflecting a 0.13 standard deviation increase in overall liability to mental illness in the population and a gain of approximately 1.9 *p*-factor points per person. As a new mental disorder diagnosis has been observed for every 3 *p*-factor points an individual demonstrates above a critical minimum (80 *p*-factor points, or the 16th percentile) (Caspi et al., 2020), this elevation in General Psychopathology equates to approximately 151 million excess mental disorders in the US population that are attributable to early life lead exposure.

By 2015, the US population had also gained 202 million internalizing symptom-points and 135 million AD/HD symptom-points, reflecting a 0.64- and 0.42-standard-deviation increase in these symptoms in the population.

By 2015, the US population had also gained 45 million Neuroticism points and lost 63 million Conscientiousness points, reflecting a 0.14-standard-deviation increase in Neuroticism and a 0.20-standard-deviation decrease in Conscientiousness in the population.

These estimates depict averages across all years between 1940 and 2015. There are, however, strong cohort differences in exposure (McFarland et al., 2022). Figure 1 shows a sideways U-shaped distribution displaying a marked relationship between mental-health symptoms and lead exposure by birth cohort. Panel A shows that General Psychopathology increased in the population for most birth cohorts, but those born around 1940 and 2015 had the lowest lead exposures and, consequently, lowest lead-link mental illness. This pattern extends to all outcomes. Conversely, the most lead-exposed cohorts, those born between 1966 and 1970, 1971 and 1975, or 1976 and 1980, experienced the largest increase in mental illness symptomology. For instance, those born between 1966 and 1970 (Generation X) likely experienced a lead-linked 1.75-standard-deviation increase in internalizing symptoms. The largest associations between birth cohort and mental health standard-deviation changes occurred for this 1966–1970 cohort and were: 0.35 standard-deviation increase in General Psychopathology, a 1.75 standard-deviation increase in Internalizing Symptoms, a 1.17 standard-deviation increase in AD/HD symptoms, a 0.39 standard-deviation increase in Neuroticism, and 0.54 standard-deviation decrease in Conscientiousness.

Finally, because our estimates of the psychiatric burden of childhood lead exposures in the United States from 1940 to 2015 are based primarily on lead exposure arising from leaded gasoline for the pre-NHANES years of 1940–1975, the addition of other potential sources of lead exposure would necessarily increase our final psychiatric estimates. For example, additional estimation of lead exposures due to ingested lead paint (Table S3), the leading source of *clinical lead poisonings* (BLLs >60–80 μg/dL) prior to the leaded-paint ban of 1978, increases the estimated total number of US adults with childhood lead levels ≥30 μg/dL by approximately 581,000, from 4.7 million to 5.3 million (Warren, 2000). Our primary psychiatric findings should therefore be considered likely floors for lead-attributable psychiatric disease rather than ceilings.

Discussion

This nationwide estimation of the psychological consequences of early life lead exposure for the US population over the past 75 years identified significant downward shifts in population mental health due to the past addition of lead to gasoline. Estimates show that the US population gained 602 million General Psychopathology points, reflecting the likely addition of 151 million excess mental disorders that would not have occurred were it not for the addition of lead to gasoline, along with an approximately half-a-standard deviation increase in specific internalizing and AD/HD-related symptoms across the population.

The use of mental illness 'points' and standard deviation increases as outcome measures can be difficult to interpret. Specific numbers of mental

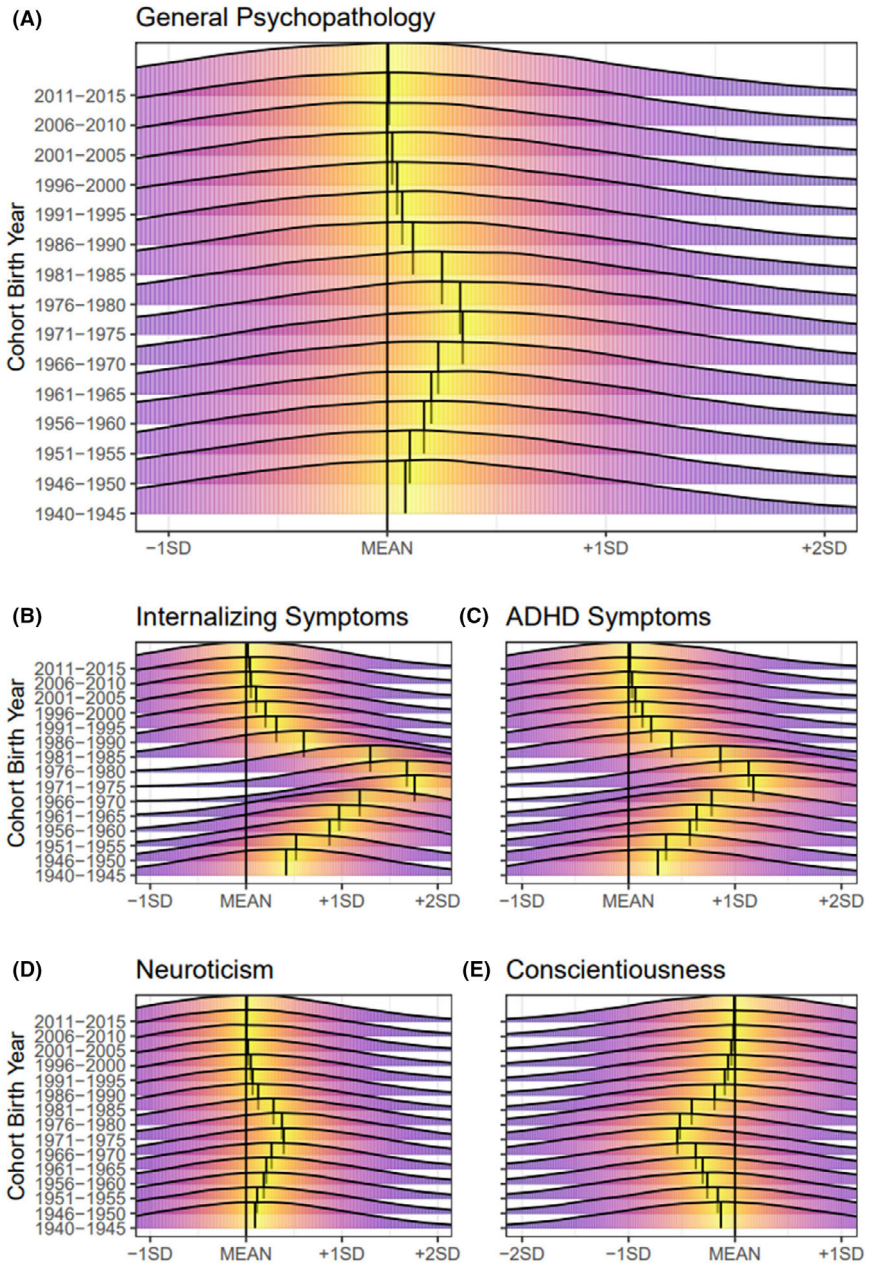

Figure 1 Estimated elevations in US population mental illness symptomatology and personality difficulties attributable to early life lead exposure over the past 75 years, by birth cohort. Ridge plots visualize the additional mental illness symptomatology (panels A–C) and personality difficulties (panels D and E) gained by US population birth cohorts as a result of adverse childhood lead exposures over the past 75 years. Panels A–C present elevations in General Psychopathology, Internalizing, and AD/HD symptoms, respectively. Panels D and E present alterations in personality characteristics that are disadvantageous, including increased Neuroticism and decreased Conscientiousness. Elevations are depicted as estimated changes from the mean under conditions of no lead exposure, in standard deviation units

health disorders attributable to lead would be more parsimonious and actionable metrics. Certainly, good Odds Ratios exist to describe the relationship between lead exposure at different levels and the odds of experiencing an individual specific mental disorder (for example, the OR of Schizophrenia for individuals with early-life BLL ≥15 μg/dL compared to <15 μg/dL = 1.92 [95% CI, 1.05–3.52]) (Opler et al., 2004) and the OR of AD/HD for individuals with early-life BLL >2.0 μg/dL compared to <0.7 μg/dL = 4.1 [95% CI, 1.2–14.0] (Braun et al., 2006). However, while easy to interpret, these measures do not lend themselves to population estimations, as true prevalence rates at different levels of lead exposure (i.e., more and less exposed cases) do not exist. Applying this case–control approach to the US population does, however, identify approximately 89 million adults over the past century who had twofold greater risk of schizophrenia than their less exposed peers owing to lead exposure, and approximately 170 million adults who had fourfold greater risk of AD/HD than their peers.

Mental illness symptomology elevations attributable to lead in the United States varied considerably

by birth cohort. The most pronounced elevations were for individuals born between 1966 through 1986, peaking in 1966–1970 (Generation X) with the peak use of lead in gasoline from the mid-1960s through the mid-1970s. For these, and temporally neighboring cohorts, lead-attributable elevations in psychopathology likely manifest with observable psychiatric and societal/cultural consequences, such as increased demand for psychiatric care or increased juvenile delinquency. Indeed, a recent meta-analytic analysis of the so-called Lead-Crime Hypothesis has indicated that 'the abatement of lead pollution may be responsible for 7%–28% of the fall in homicide in the US' from the 1990s through the 2010s (Higney, Hanley, & Moro, 2022).

Leaded gasoline was used extensively around the world across the 20th century (Thomas, Socolow, Fanelli, & Spiro, 1999), until August 30, 2021, when the final leaded-gasoline ban went into effect, in Algeria (Rukikaire, 2022). As leaded-gasoline use is robustly associated with population blood-lead levels (Angrand, Collins, Landrigan, & Thomas, 2022; Lacerda, Pestana, dos Santos Vergilio, & de Rezende, 2023), the US psychological experience with lead is likely mirrored by elevations in psychiatric disease across most other countries. This hypothesis warrants investigation. While lead has been removed from gasoline it still nevertheless appears in many products, such as automotive batteries, contaminated foods and soils, water supply pipes and commercial paints, industrial production systems, and small-piston aircraft fuel. The United Nations Children's Fund estimates that 800 million children worldwide are currently exposed to high levels of lead, largely owing to under-regulation of lead-emitting industries in developing countries (Rees & Fuller, 2020), with likely consequences for global mental health over the next century.

Recently, some clinicians have advocated for 'lead poisoning' to be recognized as a cause of intellectual disabilities within the neurodevelopmental disorder classification of the DSM owing to early life lead exposure's known negative implications for brain development (Neuwirth & Emenike, 2024; Neuwirth, Lopez, Schneider, & Markowitz, 2020). Such a move could facilitate greater awareness of the harms of lead exposure, particularly among pediatricians (Neuwirth et al., 2020; Reuben et al., 2024), increase screening rates, and potentially unlock additional resources for research and intervention that, as with psycho-educational services, are driven by DSM diagnoses. Our exposure estimates indicate that a significant portion of the US population would have been eligible for such a diagnosis in the past, while our disorder estimates indicate that they would have potentially benefited from some forms of psychological/behavioral intervention. Our findings reinforce the benefit of child lead screening, which is not always widely available in the United States (Neuwirth, 2018; Reuben et al., 2024).

Finally, our estimates may provide new context to the so-called 'Lucifer Curves' (Nevin, 2016), which hypothesize that extensive lead exposures in early childhood over the past century resulted in several social 'pathologies' including increased rates of violent crime and homicides and to abiding racial disparities in educational outcomes, arrests, and incarcerations. Our analysis suggests psychiatric symptoms and mental disorders might be reasonably added to this list.

Limitations

Causation was not established here and the specific internal validity of our estimates rests on the validity of estimates from our two key studies (Reuben et al., 2019; Winter & Sampson, 2017), although their findings are replicated in many cohorts and settings described earlier in this report. Notably, Reuben et al. utilized a sample where lead exposure did not vary by socioeconomic status in childhood, and Winter and Sampson utilized several analytical models meant to capture causal effects via instrumental variables analysis. However, neither of these studies establishes a causal relationship, and therefore this study does not report causal estimates either, although causal language is used at times to best reflect the predominant weight of causal evidence from both observational and mechanistic studies (ATSDR, 2020). Further, our reliance on these two cohorts would have resulted in estimation errors to the extent to which their reported continuous dose–response associations do not generalize to the full US population, either because one (Reuben et al.) is a New Zealand-born cohort or because the other (Winter & Sampson) is a predominantly urban US cohort. Future studies should replicate and extend our findings as more studies with continuous assessment of lead and psychiatric symptoms become available. Our estimates, which utilized a linear dose–response association curve, would be underestimates if the true lead-psychiatric curve follows more supra-linear functions.

We set the lowest lead exposure category as ≤5 μg/dL although the current scientific consensus is that there is no safe level of lead exposure (ATSDR, 2020). Moreover, lead-IQ associations tend to follow a supra-linear pattern (Lanphear et al., 2019), such that low-level exposures appear to be more harmful per lead microgram than higher levels for reasons that are not yet fully understood. It is likely that large samples with fine-grained outcome measurements may detect psychiatric and neurocognitive endpoints or symptoms at very low lead-levels (e.g., below the current reference value, 3.5 μg/dL). However, there is not yet sufficient published evidence on these levels that we could draw upon for the current empirical evaluation. This is an important limitation for the field and direction for future research.

Finally, our pre-NHANES population BLL estimates for the years 1940–1975 are based on leaded-gasoline consumption levels and do not necessarily capture likely lead exposures from other sources. During this period exposures would also have arisen from lead in pipes, contaminated food, solder, soil, and airborne dust from lead-emitting industries, waste incineration, and lead processing operations. As noted earlier, our estimates should be considered likely floors for lead-attributable psychiatric disease rather than ceilings.

Conclusions

A century of leaded-gasoline use in the United States created vast exposure to adverse lead levels across the population. Only recently have estimates of this widespread exposure been produced; the implications for public health are under-characterized but likely significant.

Here, we estimate manifold consequences of lead exposure in the form of diminished US population mental health—consequences that likely accompanied the previously reported loss of cognitive health (IQ points) attributable to lead exposure (McFarland et al., 2022). Large swaths of the population likely experienced elevated lead-linked mental illness symptomatology and altered personality, with significant implications for national well-being, innovation, economic productivity, need for and use of psychiatric services, and the prevalence of physical comorbidities, all of which bear individual investigation and estimation.

In summary, a significant burden of mental illness symptomatology and disadvantageous personality differences can be attributable to US children's exposure to lead over the past century. The contribution of legacy lead exposures to population health and disease may be much larger than previously assumed.

Supporting information

Additional supporting information may be found online in the Supporting Information section at the end of the article:

Appendix S1. Further details on the psychiatric outcome measures.

Table S1. United States population estimates of blood lead levels. Above the former CDC level of concern (>5 μg/dL) in Early Life by Age in 2015.

Table S2. Average mental health losses linked to lead exposure by cohort with margins of error.

Table S3. Reported and estimated cases of child lead poisoning in US Cities, from 1940 to 1975.

Acknowledgement

The authors have declared that they have no competing or potential conflicts of interest.

Data availability

Data utilized in this publication are available as supplementary material in McFarland et al. (2022) (*PNAS*) and are described in the Methods section.

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Key points

- More than half the current US population were exposed to adverse levels of lead, a neurotoxicant, in childhood.
- Childhood lead exposure disrupts brain development and has been associated with elevated mental illness symptomatology later in life. The total contribution of childhood lead exposure to US population mental health has yet to be determined.
- By 2015, the US population had gained a 0.13-standard-deviation increase in overall liability to mental illness as a result of early-life lead exposures over the past 75 years, resulting in an estimated 151-million excess psychiatric disorders attributable to lead.
- Childhood lead exposure has likely made a significant, underappreciated contribution to psychiatric disease in the United States over the past century.

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