

Testimony of Bruce P. Lanphear, MD, MPH

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I am physician with board certification in public health and preventive medicine from Tulane University. I also received post-doctoral training in community pediatric research at the University of Rochester in New York. I have conducted research on the sources of lead exposure and the health impacts of lead poisoning for over 25 years. I have conducted studies or served as an advisor on how to reduce lead contamination in Chicago, Detroit, St Louis, Herculaneum, Rochester, Los Angeles, Santa Clara County, and many other communities across the United States. I have served on numerous science advisory committees of the US EPA, the Centers for Disease Control, the National Toxicology Program of the National Institutes of Health, and the American Academy of Pediatrics. I was a member of the work group that advised the CDC to reduce their action level to 3.5 µg/dL and I am currently a member of the US EPA's science advisory panel for the national air lead standard.

Lead is cumulative poison. We are all exposed to lead in our drinking water and food, lead in our homes and airborne lead. Children – especially low-income children from minority communities – are more heavily exposed to lead: lead-contaminated paint and house dust from older, poorly maintained rental housing; lead-contaminated soil in smelter communities; lead contaminated water from

lead-service lines and lead from aircraft emissions. Blood lead levels have declined by 95% since the 1970s, but they are still 10- to 100-times higher than our pre-industrial ancestors (Flegal, 1992).

Airborne lead remains an important source of lead exposure in the United States. Jennifer Richmond-Bryant and other US EPA scientists found that children's blood lead concentrations rose sharply at airborne lead concentrations (TSP) below $0.15 \mu\text{g}/\text{m}^3$ and then decelerated at concentrations $> 1.0 \mu\text{g}/\text{m}^3$ (Richmond-Bryant, 2014). Richmond-Bryant's study indicates that, for a given exposure, children's blood lead concentrations would decline considerably more by lowering airborne lead concentrations $< 0.15 \mu\text{g}/\text{m}^3$ – the current EPA air lead standard – than by lowering airborne lead concentrations $> 1.0 \mu\text{g}/\text{m}^3$. This study indicates that the existing air lead standard fails to protect the public – including children – from lead exposure.

The major sources of airborne lead in the United States are piston-engine aircraft, lead battery recycling operations, and incinerators. The EPA estimated that over 450 tons of lead were emitted by piston-engine aircraft every year; 70% of all lead emissions (EPA, 2017).

Leaded aviation fuel is an important source of lead exposure for communities who live near general airports (Miranda, 2007; Zahran, 2017; Zahran, 2021). In a study of 448 airports and over 1 million children in Michigan, Sammy Zahran found that children who lived near a general airport had significantly higher blood lead levels after accounting for age of housing stock and

industrial sources (Zahran, 2017). Compared with children who resided > 4 km from an airport, children who lived < 1 km, 1–2 km, and 2–3 km were 25.2%, 16.5%, and 9.1% more likely to have a blood lead > 5 µg/dL, respectively. The increase in blood lead concentration was larger for children who lived downwind from the airport, especially toddlers. Children who lived near airports were more likely to live in households receiving public assistance (Zahran, 2017).

In 2021, Sammy Zahran was invited to conduct a study of childhood lead exposure at Reid-Hillview airport in Santa Clara County, California. Zahran and his team used blood lead tests of 17,000 children collected by the California Department of Public Health from January 1, 2011 to December 31, 2020. Zahran found that 2% of toddlers who lived > 0.5 miles from the airport had a blood lead > 3.5 µg/dL. In contrast, 5.7% of toddlers who lived within 0.5 miles of the airport had a blood lead > 3.5 µg/dL and 10.5% of toddlers who lived within 0.5 miles of the airport and were downwind of the airport had a blood lead > 3.5 µg/dL during heavy traffic (Zahran, 2021). This study indicates that the existing standard fails to protect children from lead toxicity.

The US EPA estimated that sixteen million Americans – including three million children – live within a kilometer of a general airport.

Lead particles found in aircraft emissions are smaller than those found in automobiles emissions. Exhaust particles in piston-engine aircraft emissions are “irregular particles measuring 13 nanometers with a 4 nm microcrystal of lead dibromide surrounded by a halo of hydrocarbons”. In contrast, exhaust particles

from automobile burning leaded fuel averaged 35 nm in diameter and contained five to ten 4 nm beads of lead” (Griffith, 2021). The size of lead particles from aircraft emissions are smaller than TSP and may be transported directly to the brain via the olfactory nerve.

Small particles of lead, which are readily absorbed, may be transported directly to the brain via divalent metal transporters (DMT) found in the olfactory nerve (Thomason 2007). Zeliha Kayaalti found that people with the CC genotype of DMT 1 had significantly higher blood lead concentrations than those with AA and CA genotypes ($p = 0.036$) (Kayaalti, 2015). Lead that is in the vapor form could also be readily inhaled and transported directly to the brain.

Thus, measuring lead concentrations in total suspended particles – EPA's current standard – is unlikely to be an adequate indicator of lead exposure. Moreover, up to 20% of lead in aircraft emissions is in the vapor phase (also known as alkyl or organic lead) that can be readily inhaled or dermally absorbed (EPA, 2013).

Dozens of studies show that exceedingly low levels of lead adversely impact children's cognitive abilities (Canfield, 2003; Lanphear, 2005; Desrochers-Couture, 2018). In a study of 58,000 Chicago school children, Ann Evens found that a 5 $\mu\text{g}/\text{dL}$ increase in blood lead concentration was associated with a 32% increased risk of reading failure ($\text{RR} = 1.32$, $95\% \text{CI} = 1.26, 1.39$) on standardized tests in 3rd grade children. Evens estimated that 13% of reading failures in Chicago school children were attributable to blood lead concentrations of 5 to 9 vs. 0 to 4 $\mu\text{g}/\text{dL}$.

Lead especially impacts children who are already struggling with reading. Using quantile regression, Sheryl Magzamen found in over 1,000 Milwaukee children that lead exposure led to an 18-point decrease in reading scores (95% CI: 48.7, 3.3) for children with the poorer reading abilities (Magzaman, 2015). In the NIH-funded ABCD cohort, a national study of over 9,000 US children, Andrew Marshall found that low-income children living in neighborhoods at high-risk for lead poisoning had diminished brain volume (Marshall, 2020).

Lead-associated IQ deficits extend beyond childhood. Aaron Reuben found that children (n=565) who higher blood lead concentrations at 11-years of age had further decrements in intellectual abilities by 38 years of age. After adjusting for childhood IQ score, their mothers' IQ score, and socioeconomic background, each 5 $\mu\text{g}/\text{dL}$ higher concentration of blood lead measured in childhood was associated with an additional 1.6-point lower full-scale IQ score (95% CI: -2.5 to -0.74) (Reuben, 2017). Reuben also found that children with higher blood lead concentrations were less likely to attain the same social standing as their parents (Reuben, 2017).

Lead increases the risk of children developing ADHD (Nigg, 2008; Froehlich, 2009). In a national study of 8- to 15-year-old children, Tanya Froehlich found that the fraction of children with ADHD increased from 5% to 13% as blood lead concentrations increased from $< 0.7 \mu\text{g}/\text{dL}$ to $>1.3 \mu\text{g}/\text{dL}$. Froehlich estimated that one in five cases of ADHD – representing 600,000 children with ADHD - was attributable to lead exposure (Froehlich, 2009).

Lead is a risk factor for preeclampsia, a disorder of severe hypertension in pregnant women. In a meta-analysis – a study of all high-quality studies – Arthur Poropat found that higher concentrations of lead in the blood of pregnant women was a risk factor for pre-eclampsia (Poropat, 2017). For every 1 $\mu\text{g}/\text{dL}$ increase in blood lead in pregnant women, the risk of pre-eclampsia rose by 1.6%.

Lead is a risk factor for preterm birth (Taylor, 2014; Li, 2017; Vigehe, 2011). In a pregnancy and birth cohort study in Bristol, England, pregnant women with a blood lead $> 5 \mu\text{g}/\text{dL}$ ($> 50 \text{ ppb}$) were 1.9-fold more likely to give birth preterm (Taylor, 2014). In the China-Anhui Birth Cohort Study with a mean blood lead of 1.5 $\mu\text{g}/\text{dL}$, Jun Li found that the risk of PTB was elevated in those with moderate (1.18-1.79 $\mu\text{g}/\text{dL}$; OR=2.33, 95% CI:1.49, 3.65) and high ($\geq 1.61 \mu\text{g}/\text{dL}$; OR=3.09, 95% CI: 2.01, 4.76) serum lead concentrations compared with women who had lower exposure ($<1.18 \mu\text{g}/\text{dL}$) (Li, 2017). In an Iranian cohort of 348 pregnant women with a geometric mean blood lead of 3.5 $\mu\text{g}/\text{dL}$, Mohsen Vigehe found using logistic regression that higher blood lead concentrations measured during early pregnancy were associated with a higher risk of preterm birth (OR=1.41, 95% CI: 1.08, 1.84) (Vigehe, 2011).

In a natural history study, Linda Bui examined the impact of short-term lead exposure on birth outcomes in 147,000 women following NASCAR's decision to eliminate leaded gasoline (Bui, 2021). After leaded fuel was no longer used, the probability of preterm births declined by 2.7%, and small for gestational age declined by 4.1% (Bui, 2021). The authors concluded that the EPA's National

Ambient Air Quality Lead Standard, which is based on a 3-month moving average, failed to protect against risks from short-term exposures.

Exceedingly small amounts of lead can delay conception. In a study of 501 couples, Germaine Buck-Louis found that women who had male partners with higher blood lead levels took 15% longer to conceive; the difference in blood lead concentrations among men with diminished fertility was only 0.24 µg/dl (Buck Louis, 2012).

In 2013, the US EPA concluded that lead is a causal risk factor for coronary heart disease, the leading cause of death worldwide (US EPA, 2013). In laboratory studies, chronic lead exposure causes HTN and enhances atherosclerosis by inactivating NO, increasing H₂O₂ formation, inhibiting endothelial repair, impairing angiogenesis, and promoting thrombosis (US EPA, 2013; Vaziri, 2008).

Studies published over the past decade confirm that lead is a leading, if largely overlooked risk factor for coronary heart disease (McElvenny, 2015; Aoki, 2016; Chowdhury, 2018; Lanphear, 2018; Wang, 2019). Fifteen prospective cohort studies conducted in Europe (4) and the United States (11) examined blood lead concentrations and cardiovascular mortality; all found that lead was a risk factor for CVD mortality (Navas-Acien, 2021). In a meta-analysis in the British Medical Journal, Rajiv Chowdhury and his team examined over 90,000 people in eight studies. Comparing the lowest tercile with the highest tercile, Chowdhury found that blood lead concentration was a risk factor for coronary heart disease (RR = 1.85, 95% CI: 1.27, 2.69) (Chowdhury, 2018).

The global burden of cardiovascular disease attributed to lead only includes high blood pressure and the risk for CHD deaths at blood lead > 5 ug/dL (Shaffer, 2019). But lead is also a risk factor for atherosclerosis (Vaziri, 2008) and no apparent threshold exists for lead-induced coronary heart disease (Navas-Acien, 2021; Lanphear, 2018). Using a national study in the United States, Lanphear found that lead was the leading risk factor for deaths from coronary heart disease, accounting for 185,000 deaths every year (Lanphear, 2018).

Collectively, these studies indicate that low-level lead exposure is a major risk factor for cognitive deficits, ADHD, PTB, preeclampsia, and coronary heart disease in Americans. They also indicate that lead exposure from aircraft emissions is a major source of lead exposure for nearby communities, especially low-income communities. The recent report on the Reid-Hillview airport has demonstrated that lead exposure from aircraft emissions is an urgent public health problem. Santa Clara County has demonstrated that we can solve it.

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