

**IN THE UNITED STATES DISTRICT COURT
FOR THE DISTRICT OF NEW JERSEY**

NEWARK EDUCATION)	
WORKERS CAUCUS et al.,)	
)	
Plaintiffs,)	2:18-cv-11025
)	
v.)	Judge Katharine S. Hayden
)	Magistrate Judge Cathy L. Waldor
CITY OF NEWARK, et al.,)	
)	
Defendants.)	
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Declaration of Jeffrey K. Griffiths, M.D., M.P.H.&T.M.

I, Jeffrey K. Griffiths, do hereby affirm and state:

Introduction and Qualifications

1. I am a board-certified physician, having worked nationally and internationally on drinking water contamination, environmental health and water-related health issues for more than thirty years. At present, I am Professor of Public Health and Medicine at Tufts University, where I have held a series of positions rising to Professor since 1988. At Tufts University, I hold joint appointments in Civil and Environmental Engineering at the School of Engineering and the Friedman School of Nutrition Science and Policy. I was also Director of the U.S. Agency for International Development Feed the Future Nutrition Lab for Africa. I previously served as the Director of Public Health programs at Tufts University from 2000 to 2005.

2. From November 1, 2009 to September 30, 2012, I served as Chair of the U.S. Environmental Protection Agency's Science Advisory Board's Drinking Water Committee. From November 1, 2006 to September 30, 2012, I was a member of the U.S. Environmental Protection Agency's Science Advisory Board. I also served on the National Drinking Water Advisory Council from 1998 to 2006. In these capacities, I served as a scientific advisor to the U.S. Environmental Protection Agency's drinking water program and reviewed scientific literature pertinent to the impact of drinking water lead

exposures on human health. I chaired the Science Advisory Board's meetings, and report entitled Evaluation of the Effectiveness of Partial Lead Service Line Replacements, completed in September 2011. I also was a member of the National Research Council's Committee on Drinking Water Contaminants, a Division of the National Academies of Sciences, and helped to develop and write the 2001 Report, "Classifying Drinking Water Contaminants for Regulatory Consideration."

3. I graduated from Harvard College in 1977. In 1982, I received my medical degree from Albert Einstein College of Medicine as well as my Masters in Public Health and Tropical Medicine from the Tulane University School of Public Health and Tropical Medicine in 1988, where I was the Valedictorian and elected to Delta Omega Public Health honor society. I subsequently completed my two residencies in Internal Medicine and in Pediatrics at Yale-New Haven Hospital and undertook further post-doctoral scientific and medical training at the Harvard School of Public Health, and the Tufts-New England Medical Center.

4. I have authored more than 130 scientific research papers, including papers on the impact of drinking water contamination on the health of exposed populations and on maternal health, often based on statistical analysis, primary research and data collection.

5. In addition to my work with populations exposed to contaminated drinking water in the United States, I have worked and continue to work in Africa and South America on multiple projects related to drinking water contamination and environmental health. I lead multi-disciplinary teams addressing the public health policy implications of complex health problems.

6. I have extensively studied, researched and written about the effects of drinking water contamination on human health. Through my research, clinical practice, teaching, professional memberships, educational training, and my knowledge of scientific literature, I have become a recognized expert, both within the United States and internationally, on drinking water contamination and associated health impacts.

7. A more complete description of my educational and work experience, as well as a list of my publications, is appended as Exhibit A to this declaration.

8. The information set forth in this declaration is based upon my education, personal knowledge, and experience as well as my review of the literature and the documents attached as Exhibits [A-S].

Blood Lead Levels and Total Body Lead Burden

9. Lead is well-established as a powerful neurotoxin with severe and irreparable effects on the human body, even at low levels. Digestive exposure to lead through drinking water is a known route of lead exposure. As drinking water lead concentrations increase, water becomes an increasingly important source of lead exposure.¹ Drinking water may be the predominant source of exposure where water lead levels are high.

10. The human body accumulates lead over a lifetime, primarily in bones.² Blood serves as the initial receptacle of lead absorbed from drinking water. However, blood carries only a small fraction of the body's total lead burden. The half-life of lead in human blood is estimated to be about 28 days to 36 days; the lead is thereafter distributed throughout the body, to

¹ M.L. Miranda et al., *Changes in Blood Lead Levels Associated with Use of Chloramines in Water Treatment Systems*, 115 *Envtl. Health Persp.* 2, 221-5 (2007) (attached as Ex. B); M. Edwards et al., *Elevated Blood Lead in Young Children Due to Lead-Contaminated Drinking Water: Washington, DC, 2001-2004*, 43 *Envtl. Science & Tech.* 5, 1618-1623 (2009) (attached as Ex. C); R. Renner, *Out of Plumb: When Water Treatment Causes Lead Contamination*, 117 *Envtl. Health Persp.* 12, A542-A547 (2007).

² U.S. Dep't of Health & Hum. Servs., Agency for Toxic Substances and Disease Registry, *Lead Toxicity* (June 12, 2017), <https://www.atsdr.cdc.gov/csem/csem.asp?csem=34&po=9> (attached as Ex. D).

mineralizing and soft tissues and to the skeletal system.³ It typically requires slightly over one year for children with blood levels greater than 10 micrograms per deciliter to decline to under 10 micrograms per deciliter.⁴

11. Atoms of lead (Pb) are nearly identical in size and charge as atoms of calcium (Ca). When humans are exposed to lead, the lead can harmfully replace calcium in many metabolic activities. For adults, bones and teeth will carry about 94 percent of the body's total lead burden; for children, the lead burden accumulated in bones and teeth is about 73 percent.⁵ Lead is found in three main compartments in humans: bones, blood, and soft tissues such as the brain.⁶ Under certain circumstances, including advanced age, kidney disease, menopause, physiologic stress and pregnancy, the lead will leave the bones and reenter the body's blood and soft tissue organs.⁷ Lead deposited in bones will persist in the body for decades.⁸

³ T.B. Griffin et al., *Clinical Studies on Men Continuously Exposed to Airborne Particulate Lead*, in *Lead 221-40* (Georg Thieme ed., 1975).

⁴ T. Dignam et al., *Reduction of Elevated Blood Lead Levels in Children in North Carolina and Vermont, 1996-1999*, 116 *Envtl. Health Persp.* 7, 981-5 (2008).

⁵ U.S. Dep't of Health & Hum. Servs., *supra* note 2.

⁶ *Id.*

⁷ *Id.*

⁸ J. H. Graziano, *Validity of Lead Exposure Markers in Diagnosis and Surveillance*, 40 *Clinical Chem.* 7, 1387-90 (1994).

12. Blood lead levels, therefore, tell a significant but incomplete part of the story about a person's overall lead exposure and the total amount of lead persisting in the body. In a majority of cases, toxic blood lead levels reflect a combination of relatively recent exposures and a smaller amount of prior endogenous exposures. Blood lead concentrations mainly reflect a person's exposure history over the previous few months and are a temporal snapshot of these recent exposures. To assess long-term exposure to lead, samples of bone can be assessed by researchers for lead but obtaining pieces of bone from children would be invasive and costly, as well as impractical and unpopular. Thus, nationally and internationally, blood lead levels are used to estimate recent exposure to lead in children.

Newark Children's Blood Lead Levels and Associated Harm

13. Notwithstanding the limitations of using blood lead levels to gauge cumulative lead exposure identified above, children's reported blood lead levels in Newark are strikingly high and indicate high recent exposure.

14. As the New Jersey Health Department recently noted, "Approximately 4,800 New Jersey children were identified with elevated blood lead levels (at or above 5 micrograms per deciliter) in 2016. About 13% of

those children live in Newark, yet the city comprises only 3.8 percent of the state's children in that age group.”⁹ Indeed, of Newark children screened in 2016, nearly a quarter of children under 6 years of age had blood lead levels of 3 to 4 micrograms per deciliter.¹⁰ In 2016, 5.3 percent of screened Newark children aged between 6 and 26 months tested at or above 5 micrograms per deciliter for lead in blood.¹¹ Further, 4.7 percent of children under age 6 tested at or above 5 micrograms per deciliter.¹² The percentage of Newark children at ages 6 to 26 months with blood lead levels at or above 5 micrograms per deciliter (5.3 percent) was more than twice as high as the percentage of children with elevated blood lead levels in the state of New Jersey overall (2.4 percent).¹³

⁹ N.J. Dep't of Health, *Murphy Administration Committed to Reduce Childhood Lead Exposure* (Mar. 6, 2018), <https://www.nj.gov/health/news/2018/approved/20180306a.shtml> (attached as Ex. E).

¹⁰ Advocates for Children N.J., *Childhood Lead Exposure in Newark 7* (2018), https://acnj.org/downloads/2018_03_27_newark_kids_count_childhood_lead_exposure.pdf (attached as Ex. F).

¹¹ N.J. Dep't of Health, *Childhood Lead Exposure in New Jersey: Annual Report 16* (2016), https://www.state.nj.us/health/childhoodlead/documents/reports/childhood_lead2016.pdf.

¹² *Id.* at 20.

¹³ *Id.* at 14, 16.

15. A substantial number of published, publicly available, and peer-reviewed studies document harm at these blood lead levels. Blood lead levels at 3 to 4 micrograms per deciliter are associated with significant health harms. Evidence of neurodegenerative, cardiovascular, renal, and reproductive effects at blood lead levels as low as 1 to 2 micrograms per deciliter have been summarized in the literature.¹⁴ For children presenting with blood lead levels at 3 to 4 micrograms per deciliter, associated health harms include reduction of intelligence quotient and attention-related behaviors.¹⁵

¹⁴ See, e.g., S.C. Cho et al., *Effect of Environmental Exposure to Lead And Tobacco Smoke on Inattentive and Hyperactive Symptoms and Neurocognitive Performance in Children*, 51 J. Child Psychol. Psychiatry, 1050-57 (2010); J.J. Fadrowski et al., *Blood Lead Level and Kidney Function in U.S. Adolescents; The Third National Health and Nutrition Examination Survey*, 170 Archives of Internal Med. 1, 75-82 (2010); J.T. Nigg et al., *Confirmation and Extension of Association of Blood Lead with Attention-Deficit/Hyperactivity Disorder (ADHD) and ADHD Symptom Domains at Population-Typical Exposure Levels*, 51 J. Child Psychol. Psychiatry 1, 58–65 (2010); M.L. Miranda et al., *The Relationship between Early Childhood Blood Lead Levels and Performance on End-of-Grade Tests*, 115 Env'tl. Health Persp. 8, 1242–1247 (2007) (attached as Ex. G).

¹⁵ See, e.g., R. Nicolescu et al., *Environmental Exposure to Lead, But Not Other Neurotoxic Metals, Relates to Core Elements of ADHD in Romanian Children: Performance and Questionnaire Data*, Env'tl. Res. 110, 476-483 (2010); J.T. Nigg et al., *63 Low Blood Lead Levels Associated with Clinically Diagnosed Attention-Deficit/Hyperactivity Disorder and Mediated by Weak Cognitive Control*, 63 Biol. Psychiatry 3, 325–341 (2010); B.P. Lanphear et al., *Cognitive Deficits Associated with Blood Lead Concentrations <10 ug/dL in US Children and Adolescents*, 115 Pub. Health Rep., 521-9 (2000) (attached as Ex. H).

16. Research has also shown increased systolic blood pressure or risk of hypertension among environmental cohorts with average blood lead concentrations as low as 3 to 5 micrograms per deciliter.¹⁶ A review of the epidemiological literature concluded that lead contributes to nephrotoxicity (kidney toxicity) at blood lead levels below 5 micrograms per deciliter; this is particularly true in susceptible populations, such as those with hypertension, diabetes and chronic kidney disease.¹⁷

17. The impacts of blood lead levels at 5 micrograms per deciliter on societal cognitive resources and intellectual deficits are well-documented. Children who have blood lead concentrations over 5 micrograms per deciliter experience, on average, a lead-associated IQ deficit of 6.1 points.¹⁸ These blood lead levels have been known to result in long-lasting, permanent damage to

¹⁶ D. Martin et al., *Association of Blood Lead and Tibia Lead with Blood Pressure and Hypertension in a Community Sample of Older Adults*, 163 *Am. J. Epidemiol.*, 467-478 (2006); S. Vupputuri et al., *Blood Lead Level Is Associated With Elevated Blood Pressure in Blacks*, 41 *Hypertension*, 463–468 (2003).

¹⁷ E.B. Ekong et al., *Lead-Related Nephrotoxicity: A Review of The Epidemiologic Evidence*, 70 *Kidney Int.*, 2074–2084 (2006) (attached as Ex. I).

¹⁸ *Am. Acad. of Pediatrics, Prevention of Childhood Lead Toxicity*, 138 *Pediatrics* 1, 4 (2016) (attached as Ex. J).

children's developing brains.¹⁹ Lead exposure also leads to attention deficit hyperactivity disorder and may lead to antisocial behavior.²⁰

Newark's Water Lead Levels and Associated Blood Lead Level Increase

18. There is substantial and compelling scientific evidence indicating a probable link between Newark's high blood lead levels and Newark's high drinking water lead levels. A large number of studies of exposure to lead through drinking water have reported strong associations with increased blood lead levels.

19. I have reviewed the water lead level data reported on the New Jersey Drinking Water Watch System for the Newark Department of Water and Sewer Utilities. I observed that Newark's 90th percentile lead levels exceeded 26.7 ppb in two consecutive monitoring periods in 2017. Scores of homes have tested above 30 ppb in the past eighteen months; several homes

¹⁹ M.O. Min et al., *Cognitive Development and Low-Level Lead Exposure in Poly-Drug Exposed Children*, 31 *Neurotoxicol. Teratol.* 4, 225-231 (2009); L.M. Chiodo, *Blood Lead Levels and Specific Attention Effects in Young Children*, 2 *Neurotoxicol. Teratol.*, 538-546 (2007).

²⁰ David C. Bellinger, *Very Low Lead Exposures and Children's Neurodevelopment*, 20 *Current Op. Pediatrics* 2, 172-177 (2008) (attached as Ex. K).

have tested above 50 ppb and some homes exceeded 100 ppb. The 90th percentile lead level as of October 11, 2018, for the 2018 monitoring period still in progress, was at 42.9 ppb. To my knowledge, this most recent 90th percentile lead level is among the highest reported by a large water system in recent years.

20. The severity of the lead-contamination in Newark drinking water is such that Newark water lead levels are almost certain to contribute to significant blood lead level increase among Newark children. This view is supported by the scientific literature: in a dose-response relationship for children aged between 1 to 5 years, for example, every 1 ppb increase in water lead levels will result in a corresponding blood lead level increase of 35 percent after 150 days of exposure.²¹

21. A robust study of the association between water lead level increase and blood lead levels similarly showed that the incidence of blood lead levels

²¹ G. Ngueta et al., *Use of a Cumulative Exposure Index to Estimate the Impact of Tap Water Lead Concentration on Blood Lead Levels in 1-to 5-Year-Old Children (Montréal, Canada)*, 124 *Envtl. Health Persp.* 3 (2016) (attached as Ex. L). For modelling, Ngueta et al. consider a 50 percent gastrointestinal absorption rate; they assume that children consume on a daily basis 80 percent of stagnant water and 20 percent of flushed water. They also find that “a child’s blood lead level will increase by an amount 1.10 times greater than a corresponding increase in the cumulative lead concentration ingested via drinking water.”

over 10 micrograms per deciliter for children aged 1.3 years or under in Washington, DC, increased more than 4 times from 2001 to 2003, when the city's drinking water lead levels rose rapidly during that time-period.²² The incidence of elevated blood lead levels was highly correlated to 90th percentile water lead levels from 2000 to 2007 for children aged 1.3 years or under.²³

22. The researchers noted that their findings of a link between elevated lead in drinking water and higher blood lead in Washington, DC “is consistent with decades of research linking elevated [water lead levels] to higher [blood lead levels] and [elevated blood levels]. Studies in France, Scotland and Germany correlated [water lead levels] to adult [blood lead levels], even for adults drinking water after corrosion control markedly reduced water lead levels. Lanphear has also noted a correlation between [blood lead levels] and higher [water lead levels] in a U.S. city in which no system-wide problem with [water lead levels] was occurring.”²⁴

23. A compelling array of additional scientific studies have confirmed a positive correlation between drinking water lead levels and blood lead levels

²² M. Edwards et al., *Elevated Blood Lead in Young Children Due to Lead-Contaminated Drinking Water: Washington, DC, 2001–2004*, 43 *Envtl. Science & Tech* 5, 1618-1623 (2009) (attached as Ex. M).

²³ *Id.*

²⁴ Edwards et al., *supra* note 22 (*citations omitted*).

in the exposed population.²⁵ The overwhelming weight of the scientific evidence indicates that lead exposure through drinking water is a statistically significant contributor to blood lead levels and that children and pregnant women are among those populations at greatest risk of harm.

24. This direct relationship between drinking water lead levels and blood lead levels was again demonstrated in Flint, where a statistically robust study showed a striking surge in the percentage of Flint children under 5 years of age – an increase from 2.4 percent of children to 4.9 percent of children – with elevated blood lead levels over 5 micrograms per deciliter when considering identical seasons before and after the city’s drinking water lead level increase in 2014.²⁶ There was no statistically significant increase in

²⁵ See, e.g., M. Hanna-Attisha et al., *Elevated Blood Lead Levels in Children Associated With the Flint Drinking Water Crisis: A Spatial Analysis of Risk and Public Health Response*, 106 Am. J. Public Health 2 (2016): 283-290 (attached as Ex. N); Patrick Levallois et al., *The Impact of Drinking Water, Indoor Dust and Paint on Blood Lead Levels of Children Aged 1–5 Years in Montréal (Québec, Canada)*, 24 J. Exposure Science & Env’tl. Epidemiology 2, 185-91 (2014); Marc Edwards, *Fetal Death and Reduced Birth Rates Associated with Exposure to Lead Contaminated Drinking Water*, 48 Env’tl. Science & Tech. 1, 739-46 (2014) (attached as Ex. O); Simoni Triantafyllidou et al., *Lead (Pb) in Tap Water and in Blood: Implications for Lead Exposure in the United States*, 42 Critical Reviews in Env’tl. Science & Tech. 13, 1297-1352 (2012) (attached as Ex. P); Bruce P. Lanphear et al., *Env’tl. Lead Exposure During Early Childhood*, 140 J. Pediatrics, 40-47 (2002) (attached as Ex. Q).

²⁶ Hanna-Attisha, *supra* note 25.

elevated blood lead levels in areas outside Flint and no other environmental confounders during the same time period.

25. In neighborhoods in Flint with the highest lead in drinking water levels, the blood lead levels increased even more, from 4.0 percent prior to the water change that caused a spike in lead levels to 10.6 percent after the tap water lead levels increased.²⁷

26. Another study conducted on children between 0.5 to 2 years of age reported that drinking water lead level variations corresponding to an average concentration increase of 6.55 ppb for homes served by lead service lines, and an average concentration increase of 0.3 ppb for homes without lead service lines, increased the probability of children who drink tap water experiencing blood lead levels above 5 micrograms per deciliter, from less than 5 percent to about 20 percent.²⁸

27. In a study of urban children, drinking water lead level increases from background levels to 15 ppb were associated with an increase of 13.7 percent in the percentage of children 12 to 31 months of age testing with blood

²⁷ Hanna-Attisha, *supra* note 25 at 285.

²⁸ Gerard Ngueta et al., *Exposure of Young Children to Household Water Lead in The Montreal Area (Canada): The Potential Influence of Winter-To-Summer Changes in Water Lead Levels on Children's Blood Lead Concentration*, 73 *Envtl. Int.*, 57-65 (2014) (attached as Ex. R).

lead concentrations above 10 micrograms per deciliter.²⁹ Researchers have also reported that the mean blood lead levels in mothers and children on a housing estate with lead water pipes were over twice as high as those on an adjacent estate using copper pipes.³⁰ The removal of the lead water pipes produced a drop of approximately 50 percent in mean blood lead levels, reducing them to levels comparable to those on the copper piped estate.³¹

28. Young children and infants are among the most lead-sensitive population groups and among those at greatest risk from exposure to lead through ingestion of lead-contaminated drinking water. Infants and young children are believed to absorb about 50 percent of ingested water-soluble lead.³²

29. Formula-fed infants consume high volumes of water through reconstituted formula milk and are therefore particularly vulnerable to lead-

²⁹ Bruce P. Lanphear et al., *Environmental Exposures to Lead and Urban Children's Blood Lead Levels*, 76 *Envtl. Res.* 2, 120-130 (1998) (attached as Ex. S).

³⁰ P.C. Elwood et al., *Blood-Lead Concentrations in Mothers and Their Children*, 33 *The Lancet*, 1363–64 (1978).

³¹ H.F. Thomas et al., *Relationship of Blood Lead in Women and Children to Domestic Water Lead*, 282 *Nature*, 712-3 (1979).

³² U.S. Dep't of Health & Hum. Servs., Agency for Toxic Substances and Disease Registry (2007), *Toxicological Profile for Lead*, <http://www.atsdr.cdc.gov/toxprofiles/tp13.pdf>.

contaminated drinking water. Researchers have reported that blood-lead levels of infants who were fed formula with 70 ppb lead had spiked to an average of 14.4 micrograms per deciliter within a few months.³³ When the formula contained 10 ppb lead, the children's blood lead tested at an average of 7.2 micrograms per deciliter.³⁴

30. Lead in drinking water has also been implicated in increased fetal deaths and reduced birth weights; in Washington, D.C., for example, fetal deaths substantially increased during the city's multi-year spike in water lead levels from 2000 to 2004.³⁵

31. There is an established scientific consensus that increased drinking water lead levels contribute to elevated blood lead levels and that even low levels of lead in blood cause significant harm to human health. This substantial body of scientific evidence indicates that Newark drinking water lead levels are causing serious harm to Newark residents and particularly to children.

³³ Rebecca Renner, *Exposure on Tap: Drinking Water as an Overlooked Source of Lead*, 118 *Envtl. Health Persp.* 2, A68-A74 (2010).

³⁴ *Id.*

³⁵ Marc Edwards, *Fetal Death and Reduced Birth Rates Associated with Exposure to Lead-Contaminated Drinking Water*, 48 *Envtl. Sci. & Tech.* 1, 739-46 (2014).

32. Mitigating exposure to lead in drinking water is necessary to prevent further harm to Newark residents.

I declare under penalty of perjury that the foregoing is true and correct.


Jeffrey K. Griffiths, M.D., M.P.H.&T.M.


Date