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Commentary

The Flint Water Crisis Shows the Need for Long-Term Commitment to Health Care

by Steven A. Kolmes

The Flint, Michigan, drinking-water crisis (“The Paradox of Water and the Flint Crisis,” Bhawani Venkataraman, *Environment*, January/February 2018) is poised to enter the national consciousness and conscience of the United States, in much the same way that that contamination by a fatal cocktail of mixed industrial chemicals did for the Love Canal neighborhood in Niagara Falls, New York, in the late 1970s, or the dioxin contamination of what is now the ghost town of Times Beach, Missouri, did in the early 1980s (see Matthew C. Nisbet’s article “Communicating Climate Change: Why Frames Matter for Public Engagement” and Alan McGowan’s accompanying editorial on framing environmental issues in the March/April 2009 issue of *Environment*). However, the contamination of the Flint, Michigan, drinking water by lead, a developmental neurotoxin, and trihalomethanes, which are carcinogens, differs fundamentally by being carried out, as Venkataraman thoroughly describes, by a public agency upon which the trust of the population ought to have been able to reside. Her article chronicles the crisis, with its causes and implications, in a thoughtful way to which I would like to add one additional element, that of extended time. The crucial significance of a long scale of time in any ethical reflection about the Flint tragedy is alluded to when Venkataraman writes, “More than a year had passed since the switch—a switch that was intended to save money—resulted in the

residents of Flint paying dearly and, due to the lead poisoning of the children, potentially intergenerationally,” upon which I next expand because now enough is known to say that epigenetic intergenerational injustice can exist, and that it calls for a specific social response in the name of restorative justice for the most vulnerable (Steven A. Kolmes, *Environment*, May/June 2016; David R. Boyd, *Environment*, July/August 2012).

We have come to understand recently that while DNA sequences of genes are very stable across generations, heritable changes in gene activity occur despite the DNA sequence being unaltered. Such changes in genetic activity are due to epigenetic processes; environmental toxins such as lead have epigenetic impacts. Lead exposure is virtually always a result of human activities, and the exposure of children to lead has special and serious ethical implications.¹ The new recognition of heritable epigenetic damage projects ethical concerns into a new dimension by extending them across time.² DNA methylation is one form of epigenetic change caused by environmental toxins; it involves attachment of a carbon with three hydrogens bonded to it, a methyl group, to a specific location on the ring structure of cytosine (a major DNA component), and it changes DNA activity.

The Centers for Disease Control and Prevention says, “No safe blood lead level in children has been identified. Lead exposure can affect nearly every system in the body.”³ This conclusion can be reached on just the basis of the immediate harm to children from lead exposure, which many studies have demonstrated causes developmental and cognitive problems, as well as worse pregnancy outcomes. But from an intergenerational perspective, lead exposure like that seen in the children of Flint also produces heritable epigenetic change. As levels of lead

measured in a mother's kneecap increase, there is an accompanying increase in DNA methylation in umbilical cord blood.⁴ Mothers in Detroit with high blood lead levels around the time of birth also had altered DNA methylation at more than 500 genetic locations in their newborn's blood. Researchers went on to test the hypothesis that lead exposure caused epigenetic changes in the grandchildren of lead-exposed pregnant women. Lead exposure during pregnancy changed the DNA methylation of the initial infants' fetal germ cells, because researchers subsequently measured altered DNA methylation in the next generation's (the grandchildren's) neonatal dried blood spots collected at the time of birth.⁵ The significance later in life of this epigenetic change in blood collected at the time of the grandchildren's birth remains to be determined, since such epigenetic patterns may reverse within the first 3–5 years of life,⁶ but by that point much neural development will have become irreversible. In terms of mechanisms by which epigenetic changes due to lead exposure might damage human health, increased lead exposure in adults results in epigenetic changes in ALAD genes that result in less ALAD gene activity. Normal ALAD gene activity is needed in turn to produce an enzyme that goes on to be crucial to hemoglobin production for red blood cells.⁷ Red blood cells with less hemoglobin carry less oxygen to all the organs of the body, producing widespread health impacts.

Why is it worth considering this new and longer dimension of time in terms of lead exposure's impacts? It suggests that not only does the bureaucratic system, with the dangerous infrastructure for delivering drinking water in Flint, need to be remedied, but a multigenerational commitment to providing health monitoring and public health interventions for impacted children is called for, which is the first recognition of a situation that requires a three-generation

response in terms of health care.⁸ The damage of the tragedy of Flint needs to be fully recognized for what it is, and for a time scale that perhaps will prove to be an important component in the future of more and more circumstances requiring restorative justice, maybe including not just the children of Flint, but also those of Hoosick Falls, NY, Appalachia, the tribal nations, service people on military bases, and farm communities in the Central Valley (Bhawani Venkataraman, *Environment*, January/February 2018).

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1. R. A. Butkus and S. A. Kolmes, “Children in Jeopardy: Anthropogenic Toxins and Childhood Exposure,” *Journal of Catholic Social Thought* 7 (2010): 83–114.
 2. R. A. Butkus, and S. A. Kolmes, “Integral Ecology, Epigenetics and the Common Good, Reflections on *Laudato Si* and Flint, Michigan.” *Journal of Catholic Social Thought* 14 (2017): 291–320.
 3. Centers for Disease Control and Prevention, “Lead,” <http://www.cdc.gov/nceh/lead> (accessed January 25, 2018).
 4. J. R. Pilsner, H. Hu, A. Ettinger, B. N. Sánchez, R. O. Wright, D. Cantonwine, A. Lazarus, H. Lamadrid-Figueroa, A. Mercado-García, M. M. Téllez-Rojo, and M. Hernández-Avila, “Influence of Prenatal Lead Exposure on Genomic Methylation of Cord Blood DNA,” *Environmental Health Perspectives* 117 (2009): 1466–71.
 5. A. Sen, N. Heredia, M. Senut, S. Land, K. Hollocher, X. Lu, M. O. Dereski, and D. M. Tuden, “Multigenerational Epigenetic Inheritance in Humans: DNA Methylation Changes Associated

With Maternal Exposure to Lead,” *Nature, Science Reports* 5 (2015):14466.

doi:10.1038/srep1446; M. Senut, P. Cingolani, A. Sen, A. Kruger, A. Shaik, H. Hirsch, S. T.

Suhr, and D. Ruden, “Epigenetics of Early-Life Lead Exposure,” *Epigenomics* 4 (2012): 665–74.

6. Ibid.

7. C. Li, M. Xu, S. Wang, X. Yang, S. Zhou, J. Zhang, Q. Liu, and Y. Sun, “Lead Exposure Suppressed *ALAD* Transcription by Increasing Methylation Level of the Promoter CpG Islands,”

Toxicology Letters 203 (2011): 48–53.

8 Butkus and Kolmes, 2017