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Invited Perspective: Toxic Metals and Hypertensive Disorders of Pregnancy

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The worldwide prevalence of gestational hypertension and preeclampsia is estimated at 10% and 2%–8%, respectively.[1] In the United States alone, the incidence of preeclampsia—a leading cause of maternal mortality—increased by 25% between 1987 and 2004.[2] Despite the prevalence and severity of these conditions, definitive causes remain elusive, hindering risk reduction interventions.[3] There has been increasing attention to the role of environmental chemicals, including toxic metals, in the development of gestational hypertension and preeclampsia (together termed hypertensive disorders of pregnancy).[4,5] The recent study by Borghese et al. significantly contributes to the growing literature establishing associations between toxic metal exposure and these disorders.[6]

The authors examined mixture effects, highlighted modification of toxic metal effects by essential metals, evaluated exposure windows of susceptibility, measured various species of toxic metals (e.g., arsenic), and assessed confounding by seafood consumption and air pollution, all within one of the largest study populations to address this topic. They found an increased risk of preeclampsia with elevated third-trimester blood lead levels. They also observed an increased risk of preeclampsia and gestational hypertension with elevated first-trimester blood arsenic concentrations. These data underscore arsenic and lead as perinatal toxicants that remain an urgent public health concern. Lead has been previously found to increase the risk of preeclampsia[7]; however, there is more mixed evidence with regard to arsenic's contribution to hypertensive disorders of pregnancy.[4] The findings by Borghese et al. expand upon prior work that has also documented other metals of concern, including cadmium, as potential etiologic factors underlying hypertensive disorders of pregnancy.[4,8]

Although toxic metals have been studied for hundreds of years, these chemicals have received relatively less research attention than newer, engineered chemicals in relation to hypertensive disorders of pregnancy—which is unfortunate given their omnipresence. Exposure to toxic metals, such as arsenic and lead, predominately occurs via contaminated drinking water,[9,10] geogenic and industrial sources,[11–13] and contaminated food sources.[14,15] Despite the established toxicity of lead and governmental efforts to reduce exposure,[16] measured biomarker levels remain concerningly high among reproductive-age women around the world.[17–21] In fact, >500,000 pregnant women in the United States were predicted to have blood lead levels >5 μg/dL between 2011 and 2017.[19] This is particularly salient when considering that the median lead levels in the study by Borghese et al. were orders of magnitude lower (0.52–0.64 μg/dL).[6]

Arsenic also continues to be a contaminant of concern, particularly in federally unregulated private well water, but also in public community water systems. In the United States, concentrations hundreds of times over the maximum contaminant level (MCL) set by the U.S. Environmental Protection Agency (EPA; 10 μg/L) have been reported in private well water.[22] Although public community water systems are regulated by the Safe Drinking Water Act,[23] evidence shows that arsenic remains a problem in these systems as well, with exceedances especially likely in the Southwestern United States, in communities that are smaller or predominantly Hispanic and systems that rely on groundwater.[24] However, placing the findings of blood arsenic from this study in the broader public health context is slightly more challenging than with lead given that there were inconsistent findings across the different arsenic biomarkers evaluated. In addition, the half-life of blood arsenic is several hours (thereby reflecting recent exposure that may or may not be chronic) and there are no specific public health guidelines on arsenic exposure for pregnant women, as exist for lead.[25] Thus, more research is needed to validate the findings on arsenic from the study by Borghese et al.[6] and to more fully grasp the clinical and public health implications.

Currently, it is not standard prenatal clinical care to test for maternal body concentrations of toxic metals or assess for potential exposure sources, although movement in this direction is endorsed by the American College of Obstetricians and Gynecologists and the International Federation of Gynecology and Obstetrics.[26–28] With studies such as Borghese et al. bolstering the evidence that these toxicants contribute to hypertensive disorders of pregnancy,[6] the foundation is strong for motivating change. Cultural shifts, such as clinicians asking patients about their drinking water sources and providing information on effective, low-cost water testing and filters,[29,30] could improve outcomes for women at high risk of exposure. Clinics could incorporate biomonitoring of arsenic, lead, cadmium, and mercury, among other metals, and offer interventions as needed. Moving forward, health insurance companies should consider environmental health as preventative care, including covering the costs of biomonitoring and water/air filters. Achieving these changes may require evidence from clinical trials that evaluate the impact of such interventions on perinatal outcomes. Of course, action at the patient–provider level must be coupled with continued pressure to improve and tighten environmental regulations to reduce water, food, and air contamination in the first place.[31,32] In fact, tighter federal regulations have been documented to reduce body burdens and disease incidence for both lead and arsenic.[10,33] For example, the U.S. EPA's more stringent MCL for arsenic implemented in 2006 reduced urinary arsenic levels by an average of 17% among public community water system users, which was predicted to reduce bladder and lung cancer incidence by 200–900 cases per year.[10]

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One particularly striking finding in the study by Borghese et al. is that the toxicity of blood arsenic was reduced at higher blood manganese concentrations.[6] Previous studies have also demonstrated the capacity of essential metals and other dietary factors to reduce the effects or body burden of toxic metals.[8,34–37] These studies suggest that nutritional factors that act on common toxicity pathways could be used in clinical practice. For example, appropriate supplementation with manganese may improve outcomes in patients at high risk for metals exposure, a hypothesis worthy of further investigation. Future epidemiologic research on this topic should be encouraged to stratify by essential metals to test whether this specific finding is repeated in different populations. In addition, animal model experimentation would likely be required to evaluate safety of manganese supplementation, given its toxicity at higher doses, before proceeding to trial therapeutic use in pregnant populations. However, the use of essential metal supplementation is feasible, as evidenced by the fact that calcium supplementation has been shown to lower lead body burden in mouse models and among pregnant and lactating women in human studies.[38,39] In turn, calcium supplementation is known to reduce the risk of preeclampsia; to our knowledge, the potential mediating role of the reduction in blood lead levels in this relationship has not been investigated.[37,40]

In addition to offering an avenue of potential clinical intervention for risk reduction, the antagonistic effect of manganese on arsenic toxicity furthers the toxicologic evidence that oxidative stress, particularly within the placenta, plays a critical role in the pathogenesis of hypertensive disorders of pregnancy.[41] Manganese is a component of superoxidase dismutase, an antioxidant enzyme.[42] Pathways related to oxidative stress and inflammation may play a role in poor placentation, one of the hallmarks of preeclampsia.[43–45] The activation of these pathways by toxicants such as arsenic may be attenuated by the antioxidant capacities of chemicals such as manganese. Interestingly, data support environmentally responsive epigenetic control of these key pathways as part of the complex biological underpinnings of preeclampsia, offering another avenue for therapeutic strategies to be investigated.[43,44,46,47] Continued epidemiologic studies along with *in vivo* and *in vitro* research into the mechanisms of environmentally induced hypertensive disorders of pregnancy may lead to further insights for risk-reducing interventions.

Last, the findings in the study by Borghese et al.[6] take on added importance when considering the appalling racial disparities in maternal and infant mortality in the United States, where Black women are more likely to develop preeclampsia than their White counterparts.[48,49] Although disparities are less extreme in Canada, where this study was conducted, they still persist.[50] These disparities are likely in part driven by several forms of environmental injustice that result in women of color having greater exposure to harmful chemicals, including toxic metals.[51,52] For example, municipal underbounding leaves periurban communities of color more likely to rely on unregulated private well water.[53,54] Cultural and commercial pressure to attain White beauty standards often pushes women of color to use toxic skin and hair care products.[55,56] Further, Superfund sites and other contaminating sources are disproportionately likely to adjoin communities of color.[13,57] Thus, mounting evidence of toxic metals' impact on adverse perinatal health outcomes behooves us to confront environmental racism to tackle maternal–child health disparities.

Taken together, the evidence raises three critical points to consider for improving perinatal environmental health. First, we must remain vigilant in focusing on toxic metals as chemicals of concern for perinatal health. Second, it is imperative that clinical care of pregnant patients include an assessment of environmental health history, perhaps moving toward biomonitoring of toxic metals and, ultimately, the implementation of exposure-reducing interventions. Finally, to translate these findings into improved perinatal health, we must advocate for solution-oriented changes, such as subsidizing and distributing water filters to families at high risk of exposure, ensuring community water systems comply with federal regulations, expanding clinical trials of nutritional interventions, and tackling environmental racism to promote clean drinking water for all.

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