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Environmental Chemicals and Kidney Disease

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About the Study

We are grateful for the opportunity to comment on our recent study that evaluated glyphosate exposure in young children and its potential adverse effect on the kidney [1].

The assessment of exposure to environmental chemicals in clinical practice and research is generally indirect and relies upon measurement of the molecule of interest in serum, urine, stool, hair, or nail samples. Investigators rarely have access to detailed information about diet, place of residence, geospatial environment, and lifestyle features that reflect the actual intake of the chemical under study [2]. For example, in our study, the detection of glyphosate in the urine of infants suggests absorption from breast milk. However, we were unable to prove this in our pilot study. These methods are susceptible to systematic errors based on the turnover and half-life of the molecule, variations in body composition and distribution of the molecule, and other medical conditions that impact on health and alter clearance of the molecule

The later factor is especially noteworthy for chemicals that are handled by the kidney. The kidney can eliminate compounds via glomerular filtration or tubular secretion and both pathways can be impaired in patients with chronic kidney disease (CKD). As kidney function declines, it is likely that serum levels will rise and urinary excretion will decline. This may lead to erroneous conclusions about the level of exposure and the impact of the molecule, a phenomenon called reverse causation [3]. Moreover, even the details of how to report urinary levels of environmental is open to discussion. Some investigators favor reporting the concentration of the pollutant and controlling for urine creatinine concentration to account for variations in urinary concentrating capacity that declines with advancing CKD. In contrast, other normalize the urinary concentration of chemicals of interest to urinary creatinine concentration [3]. There are no simple solutions to these problems. But they highlight the need to read the fine print in the Methods sections of research papers when evaluating reports of environmental chemicals and discussion of potential harms.

Once a relation between a chemical exposure and a health outcome is noted, environmental studies and reports of adverse consequences of specific chemicals are often criticized as associations only without clear-cut evidence of direct causation. This is especially true in cross sectional studies such as our studies linking exposure to bisphenol A and phthalates with increasing levels of low-level albuminuria in healthy American children enrolled in the NHANES cohort studies [4,5]. However, the concern applies even in longitudinal studies because of the presence of multiple known and unknown confounding factors. It is important to acknowledge that this is an almost unavoidable feature of clinical environmental health research. It is impractical and unethical to conduct controlled studies of graded exposure to potentially toxic substances in healthy volunteers and patients. Short-term studies may be feasible under select circumstances. Alternative approaches to demonstrate causation are required. In this regard, it will be important to identify potential biomarkers of target organ injury or activation of known injury pathways that can provide a more precise link between exposure and organ damage. Application of biomarker driven research may be used to test whether an intervention to reduce exposure is having a beneficial effect and would parallel the use of biomarkers to guide precision medicine initiatives [6]. These indices could be incorporated into large-scale studies of strategies to determine the benefits of reducing specific environmental chemical exposures.

Our study of glyphosate exposure in young children was notable for a failure to detect any evidence of kidney injury in relationship to the amount of the pesticide in the urine. This negative conclusion needs to be tempered by two considerations. Life is long and lack of an adverse signal in infancy childhood does not preclude long-term consequences as a result of low-grade exposure over the course of a lifetime. In addition, healthy people and patients live in a complex environment and are likely to exposed to a wide of chemicals in an ever changing variety of formats and settings. Analytic methods are being developed to evaluate the impact of mixtures of chemical exposures serially over time. These protocols need to account for variations in the level of exposure and the varying impact of the chemical over the lifespan and progressive course of organ injury (manuscript under review). This work is challenging but will provide a more comprehensive and realistic picture of what is actually happening to children and adults and enable more effective exposure reductions approaches to be designed and implemented.

To date, most of our studies of the effects of short-lived and persistent pollutant have demonstrated mild-to-moderate effects on glomerular filtration rate or albuminuria. Even children and adolescents with CKD do not appear to be overly susceptible to injury after exposure to bisphenols and phthalates even they do increase oxidative stress. Only bisphenol A had an adverse effect after 5 years of observation. Neither class of chemicals reduced GFR or increased proteinuria. However, there is evidence of subclinical injury based on increased urinary excretion of biomarkers of renal tubular injury, namely neutrophil gelatinase associate lipocalin (NGAL) and kidney-injury molecule-1 (KIM-1) (manuscript under review). The risk of environmental chemical injury may vary in different populations. For example in our pilot study of children with CD living in Africa we demonstrated significant adverse effects of polyfluoroalkylated substances on GFR despite levels of exposure that were substantially less than in healthy children living in the United States [7]. These findings argue in favor of the need for maintained awareness to environmental chemicals and routine surveillance using novel biomarkers that can detect injury while it is still preclinical and potentially reversible. Modifications in diet, food preparation, medication use, and lifestyle choices can be implemented to modulate exposure and minimize the risk of subtle ongoing injury and progression to organ failure over time.

It is important to recognize that exposure to environmental chemicals and hazards like air and water pollution are beyond the control of individuals. Factors like food manufacturing and packaging, the location of factories, and processes incorporated into the full range of industries can only be changed by adopting thoughtful regulatory standards and meaningful balancing of risks and benefits of individual chemicals. This underscores the need for ongoing thoughtful conversation between educated citizens, pediatricians, internists, geriatricians, environmental health specialists, and government authorities to develop standards for chemical use that promote health and safe economic growth.

References

1. Trasande L, Aldana SI, Trachtman H, Kannan K, Morrison D, et al. (2020) Glyphosate exposures and kidney injury biomarkers in infants and young children. Environ Pollut 256: 113334.
2. Kataria A, Trasande L, Trachtman H (2015) The effects of environmental chemicals on renal function. Nat Rev Nephrol 11: 610-625.
3. Jacobson MH, Liu M, Wu Y, Furth S, Warady B, et al. (2020) Oxidant stress and renal function among children with chronic kidney disease: A repeated measures study. Sci Rep 10: 3129.
4. Trasande L, Attina TM, Trachtman H (2013) Bisphenol A exposure is associated with low-grade urinary albumin excretion in children of the United States. Kidney Int 83: 741-748.
5. Trasande L, Sathyanarayana S, Trachtman H (2014) Dietary phthalates and low-grade albuminuria in US children and adolescents. Clin J Am Soc Nephrol 9: 100-109.
6. Mariani LH, Pendergraft WF 3rd, Kretzler M (2016) Defining glomerular disease in mechanistic terms: Implementing an integrative biology approach in nephrology. Clin J Am Soc Nephrol 11: 2054-2060.
7. Sood S, Ojo AO, Adu D, Kannan K, Ghassabian A, et al. (2019) H3Africa kidney disease research network investigators: Association between perfluoroalkyl substance exposure and renal function in children with CKD enrolled in H3Africa kidney disease research network. Kidney Int Rep 4: 1641-1645.