and intrarenal RAS activity are related to regulating blood pressure in infants with FGR. Sodium intake has been recently reported to remain elevated in pediatric population[9]; therefore, in providing dietary guidance or intervention for children born with a risk of FGR-associated hypertension, the relationship of amount of salt in the diet and the intrarenal RAS should be considered.

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Reply

To the Editor:

We were pleased to receive the comments by Arisaka et al regarding our recent review of fetal growth restriction (FGR) and programmed hypertension. We agree that rapid weight gain, development of obesity, and high sodium intake during the first year of life and throughout childhood are likely major factors that further promote hypertension development in children with FGR in an additive or even multiplicative fashion, mediated in part through renin–angiotensin system (RAS) alterations (**Figure**).[1-6] The adverse health outcomes of FGR combined with rapid postnatal growth (mismatch hypotheses) have been reported.[7,8] Therefore, preventive and therapeutic strategies centered on early-life nutrition to target the RAS may be beneficial in mitigating several perinatal programming mechanisms.

Introduction of solid foods during infancy increases salt intake. A high percentage of 12- to 24-month-old children exceed "adequate levels" of salt intake.[9] In school-age children, increased processed food consumption increases salt intake. In infants, reducing salt intake by one-half yielded a 2.5-mm Hg reduction in systolic blood pressure (BP).[9] In young adults, reduced salt intake can induce relatively rapid and sustained[10] decreased BP. In a long-term study, neonates on a low-salt diet for the first 6 months of life had 2.1-mm Hg lower systolic BP compared with controls on a normal salt diet, and at the 15-year follow-up they had 3.6-mm Hg lower systolic BP.[11] This indicates that salt intake in early life can induce sustained changes in BP, an important intervention

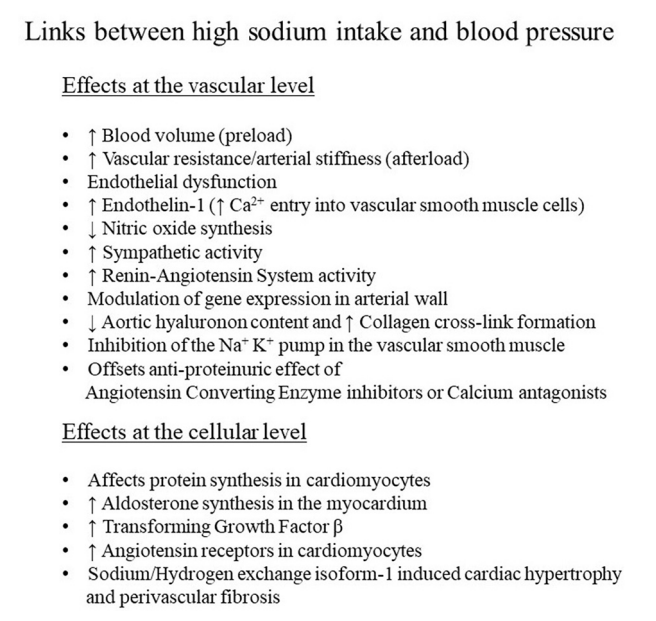

Figure. Vascular and cellular effects of salt.

as BP tracks from childhood well into the third and fourth decades of life.[12] We are currently investigating how perinatal programming factors (eg, FGR), the RAS, and nutrition (eg, early-life growth and salt intake) interact with the development of hypertension over the life course in our Prenatal Events-Postnatal Consequences birth cohort.[13]

Unfortunately, little is known about how to target preventive and therapeutic nutritional strategies centered on salt intake in this high-risk pediatric population. For now, we must follow current guidelines for the general pediatric population.[14] Intrarenal RAS or its role in programmed hypertension in humans remains to be defined. High-quality clinical trials and observational studies will better define these strategies and delineate these mechanisms across the life course.

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Racial disparities in Kawasaki disease are the effect not the cause

To the Editor:

The report by Padilla et al should be applauded for investigating racial disparity in children with Kawasaki disease, yet the methodology and conclusions must be further examined.[1] When racial or ethnic disparities are identified, it is imperative that we scrutinize what race or ethnicity is serving a proxy for in the observed association, avoiding the pitfall of using race as a proxy for genetics.

The authors cite that socioeconomic status has been associated with worse outcomes in Kawasaki disease and appropriately question whether the known over-representation of black children in this group could explain the disparities.[2] Obesity research highlights how socioeconomic status and race both impact health making it important to factor both in analyses, which was not performed in this study.[3,4]