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Maternal High Salt Intake Contributes to Enhancement of Salt Sensitivity of Offspring
– Influence of Maternal Dietary Salt on Vasodilation in Offspring –

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Summary

High salt intake is an important environmental factor that causes hypertension. High salt intake during pregnancy is thought to affect the blood pressure (BP) of offspring; however, it is unknown whether maternal dietary salt alters cardiovascular function, which is involved in the regulation of BP, in offspring. We previously showed that excessive salt intake causes elevated BP and impaired vasodilation in response to nitric oxide (NO) in spontaneously hypertensive rats (SHR). Therefore, the present study was designed to assess whether prenatal high salt intake affects BP and cardiovascular function in the SHR offspring.

SHR were exposed to either a high-salt maternal diet (4% NaCl) or a control maternal diet (0.3% NaCl) in utero and during the suckling period. After weaning, male offspring were given a diet containing 0.3% or 4% NaCl for 8 weeks. Compared to the offspring of the control diet-fed dams, the adult offspring of the high-salt diet-fed dams had slightly decreased systolic BP, slightly decreased nitroprusside-induced vasodilation but unchanged acetylcholine-induced vasodilation in isolated aortas, and reduced left ventricular contractile and diastolic function. Postnatal high salt intake resulted in unchanged systolic BP, increased acetylcholine-induced vasodilation but unchanged nitroprusside-induced vasodilation, and elevated cardiac function in adult offspring of high-salt diet-fed dams.

The findings of our study indicate that maternal high salt intake causes cardiac dysfunction along with impaired vasodilation in response to NO, resulting in a decrease in the BP of adult offspring independent of postnatal salt intake. Moreover, postnatal high salt intake enhances NO-dependent vasodilation even though cardiac function markedly increases in the offspring exposed to prenatal salt intake. This enhanced vasodilation might be a compensatory action that explains the unchanged BP in adult offspring with postnatal high salt intake. Thus, maternal dietary salt intake disturbs cardiovascular function in offspring later in life.