Proper Salt Intake During Pregnancy
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Humoral and hemodynamic changes occur during pregnancy, which may cause cardiovascular events that could result in maternal and fetal death in some cases. These changes are partly influenced by lifestyle factors, such as salt intake. However, there is little evidence to indicate the proper level of salt intake during pregnancy.

In this issue of the Journal, Inoue et al examined the association between salt intake estimated from early morning urinary sodium excretion and home blood pressure (BP) for 7 consecutive days in pregnant women. The results of this observational study do not suggest any association between these 2 parameters. Thus, the authors conclude that higher salt intake was not a predictor of pregnancy-induced hypertension, although pregnancy-induced hypertension developed in a small number of subjects from among the heterogeneous group of pregnant women, the majority of whom were normotensive. Also, there is methodological limitation in the estimation of salt intake from early morning urine samples, as mentioned by the authors. In addition, salt intake varies from day to day, so sampling over 3 or more consecutive days may be preferable to estimating the usual salt intake of the study subjects, which is often difficult to perform in practice. Because of these methodological limitations, the range of salt intake in this study (5.4 g/day and 13.0 g/day at 30th gestational week) might be underestimated compared with usual intake, resulting in a range of salt intake in the study subjects that might be too narrow to evaluate any relationship between salt intake and BP. The multiple linear regression analysis in this study did not include all of the complicated factors in the influence of salt on BP; for example, potassium intake, which suppresses the pressor effects of high salt intake, was not included. Because of these limitations of this observational study, it cannot be completely concluded that high salt intake does not contribute to pregnancy-induced hypertension.

Rather the results of this study suggest that the slightly higher salt intake (~7–8 g/day) than that of the target level of salt restriction for essential hypertensive patients indicated in Japanese Society of Hypertension’s guidelines for the management of hypertension in 2014 (JSH 2014; <6 g/day) was not harmful in the pregnant women, although this viewpoint should be confirmed by further studies. Otherwise, the major question concerning salt intake in pregnancy is how much salt is adequate during pregnancy. It is well known that volume expansion is a hallmark of the circulatory state in normal pregnancy; plasma volume begins to increase from the 6th gestational week and on average 50% of its increment is observed during the 25–30th gestational week. The results of the present study showed that estimated salt intake was slightly but significantly increased during pregnancy. Similarly, Gennari-Moser et al reported that urinary sodium excretion corrected for creatinine shifted toward a higher level in pregnant normotensive women compared with young healthy females. Thus, pregnancy may lead to increased salt intake to maintain the expanded plasma volume.

Despite the volume expansion in pregnancy, BP is rather decreased during the 1st trimester and then returns to the same or slightly higher level as before pregnancy. When the ratios of systolic and diastolic BP to urinary sodium/creatinine ratio are calculated, they are lower in pregnant women at any gestational week compared with non-pregnant females, suggesting that the salt sensitivity of BP is blunted during pregnancy. This viewpoint is strengthened by an intervention study conducted by the authors; salt loading caused a decrease in mean BP in pregnant women during the 1st trimester, although BP increased mean BP in non-pregnant women and the changes in mean BP were statistically different between the 2 groups. Thus, moderate salt loading may be beneficial in order to maintain not only the hemodynamic changes but also appropriate BP levels during pregnancy.

BP will incrementally decrease under the influence of vasodilators and natriuretic humoral factors such as progesterone in pregnant women. On the other hand, the renin-angiotensin (RA) system, which increases BP by vasoconstriction and sodium retention, is greatly stimulated during pregnancy, probably because of the increase in renin induced by estrogen. Interestingly, it has been reported that, in a pregnant woman with essential hypertension, salt loading decreased BP, which might be caused by suppression of the RA system through moderate salt loading-induced volume expansion. Pregnancy-induced hypertension is considered to have volume depletion (a similar humoral and hemodynamic state is observed with pheochromocytoma and renin-producing tumors); as a result, it could be hypothesized that BP rises with salt restriction, because of the further decrease in plasma volume, which stimulates the RA system.

The viewpoint that moderate salt loading may be beneficial in pregnancy-induced hypertension is comparable with the recent intervention trial to evaluate the effect of salt restriction to prevent pregnancy-induced hypertension and/or pre-eclampsia. For example, Knuist et al examined the effect of restricting dietary salt intake during pregnancy for the prevention of
pre-eclampsia in women with pregnancy-induced hypertension, who had 1 or more of the following: 2 diastolic BP recordings >85 mmHg, weight gain >1 kg/week for 3 successive weeks, or ‘excessive’ edema. The study subjects were divided into 2 groups, who were advised to either reduce salt intake to 3.0 g/day or to continue with their usual diet. Urinary sodium excretion as converted to salt intake was 4.9 g/day in the low salt intake group and 7.3 g/day in the usual salt intake group. The incidence of pre-eclampsia was not different between the 2 groups of pregnant women (RR 0.96; 95% CI 0.37–2.51). Also, a systematic review in 2005 concluded that low salt intake in pregnancy is unlikely to prevent pre-eclampsia. However, there are only a few available studies that have evaluated the appropriate level of salt intake in pregnancy.

In conclusion, the correct salt intake for pregnant women, which may prevent pregnancy-induced hypertension, is not well known and further studies, such as larger sample observational and/or interventional studies, in which salt intake is accurately controlled, are required to clarify this question, including its mechanism. In addition, the effect of salt intake on the infants, which is evaluated in this study, is another important and unresolved question.

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