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Excessively Low Salt Diet Damages the Heart through Activation of Cardiac (Pro) Renin Receptor, Renin-Angiotensin-Aldosterone, and Sympatho-Adrenal Systems in Spontaneously Hypertensive Rats

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Summary

A high salt intake causes hypertension and leads to cardiovascular disease. Therefore, a low salt diet is now recommended to prevent hypertension and cardiovascular disease. However, it is still unknown whether an excessively low salt diet is beneficial or harmful for the heart. Wistar Kyoto rats (WKYs) and spontaneously hypertensive rats (SHRs) received normal salt chow (0.9 % salt diet) and excessively low salt chow (0.01% salt diet referred to as saltless diet) for 8 weeks from 8 to 16 weeks of age. The effects of the excessively low salt diet on the cardiac (pro) renin receptor, renin-angiotensin-aldosterone, and sympatho-adrenal systems were investigated.

The excessively low salt diet did not affect the systolic blood pressure but significantly increased the heart rate both in WKYs and SHRs. The excessively low salt diet significantly elevated plasma renin activity, plasma angiotensin I, II and aldosterone concentrations, and plasma noradrenaline and adrenaline concentrations both in WKYs and SHRs. Cardiac expressions of renin, prorenin, (P)RR, angiotensinogen, and angiotensin II AT1 receptor and phosphorylated (p)-ERK1/2, p-HSP27, p-38MAPK, and TGF-ß1 were significantly enhanced by the excessively low salt diet in both WKYs and SHRs. The excessively low salt diet accelerated cardiac interstitial and perivascular fibrosis and increased the cardiomyocyte size and interventricular septum thickness in WKYs and SHRs but the extent was greater in SHRs.

In conclusion, an excessively low salt diet damages the heart through activation of plasma renin-angiotensin-aldosterone and sympatho-adrenal systems and activation of cardiac (P)RR and angiotensin II AT1 receptor and their downstream signals both in WKYs and SHRs.