did those rats fed a normal diet. It has also been shown that endothelial xanthine oxidase activity is higher than normal in the hypertensive rat model.[34]

Enhanced production of ROS contributes to the dysregulation of physiological processes, which leads to structural and functional alterations in hypertension.[35] Two characteristic alterations of the vascular wall in hypertension are endothelial dysfunction and vascular smooth muscle cell (VSMC) hypertrophy. Enhanced production of ROS causes a loss of NO bioavailability, which impairs endothelial function, causing (among other things) decreased endothelium-dependent vasodilation.[36] Among these ROS, $\cdot O_2^-$ is critically involved in the breakdown of NO.[37]

Somers et al.[38] showed that enhanced vascular $\cdot O_2^-$ production was associated with impaired endothelium-dependent relaxation in DOCA-salt rats, a hypertension model characterized by low plasma renin activity. Recently, Wu et al.[39] reported that the enhanced $\cdot O_2^-$ production present in the aorta of DOCA-salt-induced hypertensive rats was associated with increased NADH oxidase activity. It seems that this increased oxidase activity was independent of the rise in blood pressure. It has been suggested that increased vascular angiotensin II release as a consequence of nephrectomy is the origin of the increased NADH oxidase activity in these rats.

This is the first study to evaluate the amount of SOD enzymes in the heart of DOCA-salt-induced hypertensive rats. The clinical significance of evaluation of these antioxidant enzymes in rat heart is based on the fact that hypertension frequently causes left ventricular hypertrophy as 1 aspect of common target organ damage, and these hypertrophic hearts are more susceptible to ischemic injury.[40-42] Elevation of SOD may suggest a way to better protect hypertrophic hearts from ischemic injury. However, our previous study showed significant elevation of SOD-mRNA in hearts of SHRs.[43] It is possible that different mechanisms of hypertension may have variable effects on SOD gene expression. These findings further suggest that hypertension is associated with complex mechanisms. Whether this phenomenon implies that hearts of SHRs are more resistant to ischemic injury than these of DOCA-salt-induced hypertensive rats needs further study.

In conclusion, this is the first study to evaluate the SOD gene in the heart of DOCA-salt-induced hypertensive rats. The similar SOD gene expression between DOCA-salt-induced hypertensive rats and normotensive WKY rats suggests that SOD gene expression is not well correlated to changes in blood pressure under such conditions.

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