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EFFECTS OF HIGH-SALT DIET ON MYOCARDIAL REMODELLING AND THE INTERVENTION OF TELMISARTAN

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Objectives To study the effects of high-salt diet on myocardial remodelling, and investigate the relevant mechanisms of telmisartan on the reverse of myocardial remodelling in Wistar rats.

Methods Twenty-four Wistar rats fed by high-salt diet for 23 weeks which were divided into two groups: high-salt hypertension group (HSH n=12) and high-salt normal blood pressure group (HSN n=12) according to the level of systolic blood pressure (SBP). The rats of telmisartan group (T n=12) were fed high-salt and telmisartan for 23 weeks too. Thirteen age-matched rats fed by normal-salt were used as controls (NS n=13). Myocardial morphology and structural changes were observed by HE staining and Masson staining. The content of superoxide dismutase (SOD) and malondialdehyde (MDA) in blood and left ventricle (LV) were measured by biochemistry and enzymology. Radioimmunoassay and enzyme linked immunosorbent assay (ELISA) were employed to determine the content of tumour necrosis factor- α (TNF- α) and C-reactive protein (CRP). The protein levels of nuclear factor- κ B p65 (NF- κ B p65) were evaluated by western blot.

Results SBP in HSH was higher than other groups. In the high-salt groups, the ratio of left ventricular mass and body mass (LVMI), the myocardial cell diameter (CMD), the fibrosis area of myocardial interstitial (MIFI), the content of CRP, TNF- α (HSH 48.86 ± 8.25 , HSN 56.67 ± 9.67 vs NS 40.89 ± 4.37 ng/g, $p < 0.05$), NF- κ B p65 protein (HSH 87.77 ± 10.3 , HSN 75.18 ± 16.67 vs NS 57.13 ± 10.00), $p < 0.05$) and SOD in the blood were significantly increased, while the level of SOD (HSH 58.34 ± 5.78 , HSN 54.59 ± 6.65 vs NS 68.14 ± 9.98 U/mgprot, $p < 0.05$) in LV decreased. LVMI, CMD and MIFI were negatively correlated with SOD activity in LV respectively, and positively correlated with the protein levels of NF- κ B p65. Telmisartan partly reversed myocardial

remodelling decreased the protein levels of NF- κ B p65 and TNF- α , and increased the SOD activity in LV.

Conclusions The myocardial remodelling caused by high salt diet may be related to decreased SOD activity and inflammatory mechanism. Telmisartan prevents the salt-induced myocardial remodelling at least in part through inhibiting oxidative stress and inflammation.