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## EFFECTS OF HIGH SALT DIET ON ARTERIAL REMODELLING AND THE INTERVENTION OF TELMISARTAN IN WISTAR RATS

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**Objectives** To investigate the effects of dietary salt on aorta and mesenteric artery remodelling in Wistar Rats and explore the possible mechanism of salt-induced arterial remodelling and AngiotensinII receptor blockers telmisartan intervention.

**Methods** 60 Wistar rats were randomly divided into normal control group, high salt (8%) model group and high salt+telmisartan group. The tail artery pressure was determined every 2 weeks. After 24 weeks, high salt model group was divided into model hypertension (MH) group and model normal pressure (MN) group. The structural changes and proliferation in the media of aorta and mesenteric arteries were observed by HE staining, Masson staining and immunohistochemical staining. The activities and mRNA levels of Na<sup>+</sup> pump and Ca<sup>2+</sup> pump in aortic media were determined by enzyme colorimetry and real-time PCR respectively.

**Results** Compared with control group, the blood pressure was significantly increased in MH Group, Media thickness (MT), lumen diameter (LD), ratio of media to lumen (MT/LD), the collagen volume fraction and PCNA positive expressive percentage of arteries in high-salt group were increased (p<0.05), the activities of Na<sup>+</sup>-K<sup>+</sup>-ATPase and Ca<sup>2+</sup>-ATPase in MH group were decreased (p<0.05). The mRNA expression of Na<sup>+</sup>-K<sup>+</sup>-ATPase  $\alpha_1$  subunit in

MH and MN groups was decreased (P<0.05), and PMCA1 expression raised in MH group, Correlation analysis showed that two ATPase activities and vascular remodelling indicators have a negative correlation (p<0.05). Compared with high-salt group, blood pressure, media thickness, ratio of media to lumen, the collagen volume and PCNA positive expressive percentage were lower in telmisartan group (p<0.05).

**Conclusions** High-salt diet could lead to arterial remodelling directly or indirectly (elevated blood pressure), The decreased ion pump activity and abnormal gene expression may be one of the mechanisms of high-salt induced arterial remodelling. Telmisartan may inhibit the proliferation of vascular smooth muscle and collagen accumulation, and prevent salt-induced hypertension and arterial remodelling.