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无患子皂苷对自发性高血压大鼠主动脉血管内皮功能调节作用的实验研究

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中文摘要:目的:通过研究无患子皂苷对自发性高血压大鼠(spontaneously hypertensive rats, SHR)离体主动脉血管对于不同血管收缩剂和舒张剂反应性以及对血管内皮活性物质血清含量的影响。考察无患子皂苷对SHR内皮功能的调节作用.为进一步研究其作用 及机制提供实验依据。 方法: 取16周龄5HR 40只随机均分为5组.即SHR模型组、 附性对照组(卡托普利,27 mg·kg<sup>-1</sup>),无患子皂 苷低、中、 高(27.54,108 mg·kg<sup>-1</sup>)剂量组.另取8只健康WKY大鼠作为正常对照组.按剂量连续给费8周.检测指标如下:① 对胸主动 118.、  $\mathbf{r}$  、  $\mathbf{r}$  ( $\mathbf{r}$  ) の  $\mathbf{r}$  、  $\mathbf{r}$  、  $\mathbf{r}$  の  $\mathbf{r}$  の  $\mathbf{r}$  、  $\mathbf$ 清TXB<sub>2</sub>:ET-1含量增加,而NO,6-KPG $_{1a}$ 含量減少;无患子皂苷各剂量组可抑制主动脉对KCl,PE,Ang II 的收缩反应,增强对Ach的内皮 依赖性舒张效应,降低血清TXB<sub>2</sub>,ET-1含量,增加NO,6-KPG<sub>1 $\alpha$ </sub>含量,上述指标与SHR对照组有显著性差异(P<0.05或P<0.01)。 结论: 无患子皂苷对SHR内皮功能具有一定的保护作用,其机制与恢复血管内皮活性物质TXB2,ET-1/NO,6-KPG $_{1\alpha}$ 平衡状态有关。

中文关键词:无患子皂苷 自发性高血压 内皮功能

## Research of sapindus saponins on endothelial function in spontaneously hypertensive rats

Abstract: Objective: To investigate the regulation on endothelial function of sapindus saponins in spontaneously hypertensive rats by studying the reactivity on different vasoconstrictor and dilator, and the content of the active substances. Method: Forty 16-week-old spontaneously hypertensive rats were randomly divided into five groups, one with placebo as model group, one with captopril tablets (27 mg  $\cdot$  kg $^{-1}$ ) as positive control, one with low-dose sapindus saponins (27 mg  $\cdot$  kg $^{-1}$ ), one with medium-dose (54 mg  $\cdot$  kg $^{-1}$ ), one with high-dose (108 mg  $\cdot$  kg $^{-1}$ ). And another eight healthy Wistar-Kyoto strain(WKY) rats were used as the normal group. The animals were treated for eight weeks, and the indicators to be detected were as follows: ① the response of thoracic aorta on different vasoconstrictors Ang  $\parallel$  (1 × mg) and the strain of th  $10^9 - 1 \times 10^5 \text{mol} \cdot \text{L}^1), \text{PE}(1 \times 10^8 \cdot 1 \times 10^4 \text{ mol} \cdot \text{L}^1), \text{KCI}(20 \cdot 120 \text{ moml} \cdot \text{L}^1); \text{ @the endothelium-dependent or non-endothelium-dependent vasodilation response of horacic aorta on Ach(1 \times 10^{10} \cdot 1 \times 10^5 \text{ mol} \cdot \text{L}^1) \text{ or SNP} (1 \times 10^8 \cdot 1 \times 10^3 \text{ mol} \cdot \text{L}^1); \text{ @the content of NO,6-KPG}_{1a}; \text{ET-1} \text{ and TXB}_2 \text{ in serum was determined by Elisa.} \text{Result: In SHR model group, the response of thoracic aorta on Ang } [1, 1, 1] \text{ or SNP} (1 \times 10^8 \cdot 1 \times 10^8 \text{ mol} \cdot \text{L}^1); \text{ or SNP} (1 \times 10^8 \cdot 1 \times 10^8 \text{ mol} \cdot \text{L}^1); \text{ or SNP} (1 \times 10^8 \cdot 1 \times 10^8 \text{ mol} \cdot \text{L}^1); \text{ or SNP} (1 \times 10^8 \cdot 1 \times 10^8 \text{ mol} \cdot \text{L}^1); \text{ or SNP} (1 \times 10^8 \cdot 1 \times 10^8 \text{ mol} \cdot \text{L}^1); \text{ or SNP} (1 \times 10^8 \cdot 1 \times 10^8 \text{ mol} \cdot \text{L}^1); \text{ or SNP} (1 \times 10^8 \cdot 1 \times 10^8 \text{ mol} \cdot \text{L}^1); \text{ or SNP} (1 \times 10^8 \cdot 1 \times 10^8 \text{ mol} \cdot \text{L}^1); \text{ or SNP} (1 \times 10^8 \cdot 1 \times 10^8 \text{ mol} \cdot \text{L}^1); \text{ or SNP} (1 \times 10^8 \cdot 1 \times 10^8 \text{ mol} \cdot \text{L}^1); \text{ or SNP} (1 \times 10^8 \cdot 1 \times 10^8 \text{ mol} \cdot \text{L}^1); \text{ or SNP} (1 \times 10^8 \cdot 1 \times 10^8 \text{ mol} \cdot \text{L}^1); \text{ or SNP} (1 \times 10^8 \cdot 1 \times 10^8 \text{ mol} \cdot \text{L}^1); \text{ or SNP} (1 \times 10^8 \cdot 1 \times 10^8 \text{ mol} \cdot \text{L}^1); \text{ or SNP} (1 \times 10^8 \cdot 1 \times 10^8 \text{ mol} \cdot \text{L}^1); \text{ or SNP} (1 \times 10^8 \cdot 1 \times 10^8 \text{ mol} \cdot \text{L}^1); \text{ or SNP} (1 \times 10^8 \cdot 1 \times 10^8 \text{ mol} \cdot \text{L}^1); \text{ or SNP} (1 \times 10^8 \cdot 1 \times 10^8 \text{ mol} \cdot \text{L}^1); \text{ or SNP} (1 \times 10^8 \cdot 1 \times 10^8 \text{ mol} \cdot \text{L}^1); \text{ or SNP} (1 \times 10^8 \cdot 1 \times 10^8 \text{ mol} \cdot \text{L}^1); \text{ or SNP} (1 \times 10^8 \cdot 1 \times 10^8 \text{ mol} \cdot \text{L}^1); \text{ or SNP} (1 \times 10^8 \cdot 1 \times 10^8 \text{ mol} \cdot \text{L}^1); \text{ or SNP} (1 \times 10^8 \cdot 1 \times 10^8 \text{ mol} \cdot \text{L}^1); \text{ or SNP} (1 \times 10^8 \cdot 1 \times 10^8 \text{ mol} \cdot \text{L}^1); \text{ or SNP} (1 \times 10^8 \cdot 1 \times 10^8 \text{ mol} \cdot \text{L}^1); \text{ or SNP} (1 \times 10^8 \cdot 1 \times 10^8 \text{ mol} \cdot \text{L}^1); \text{ or SNP} (1 \times 10^8 \times 10^8 \text{ mol} \cdot \text{L}^1); \text{ or SNP} (1 \times 10^8 \times 10^8 \text{ mol} \cdot \text{L}^1); \text{ or SNP} (1 \times 10^8 \times 10^8 \text{ mol} \cdot \text{L}^1); \text{ or SNP} (1 \times 10^8 \text{ mol} \cdot \text{L}^1); \text{ or SNP} (1 \times 10^8 \text{ mol} \cdot \text{L}^1); \text{ or SNP}$ PE and KCI was increased, the endothelium-dependent vasodilation on Ach was reduced, but the effects on SNP was not obvious, the content of ET-1 and TXB<sub>2</sub> was increased, and the content of NO and 6-KPG<sub>Ia</sub> was reduced, Vs the normal control group, there were significant differences (P < 0.05 or P < 0.01); in the treatment groups, the response of thoracic aorta on Ang [], PE and KCl was reduced, the endothelium-dependent vasodilation of horacic aorta on Ach was inproved, the content of ET-1 and TXB<sub>2</sub> was reduced, and the content of NO and 6-KPG<sub>Ia</sub> was increased, Vs the SHR model group, there were significant differences (P < 0.05 or P < 0.01). Conclusion: Our findings  $_{1d}$  and  $_{1d}$   $_{1d}$ 

keywords:sapindus saponins spontaneously hypertension endothelial function

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