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敲除解偶联蛋白3促进高盐介导的血管内皮功能损害

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Title: Uncoupling protein 3 ablation exacerbates high salt intake-induced vascular endothelium dysfunction in mice

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关键词: 解偶联蛋白3; 血管内皮功能; 高盐饮食; 氧化应激

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摘要: 目的 探讨解偶联蛋白3（uncoupling protein 3, UCP3）敲除对高盐介导的血管内皮功能损害的影响及机制。 方法 雄性UCP3敲除（UCP3 KO, C57BL/6J背景)及相应的野生型小鼠（wild type, WT）各20只，分普盐饮食组（normal salt, NS：饮食中含0.5%食盐）和高盐饮食干预组（High Salt, HS: 饮食中含8%食盐），每组10只小鼠；饮食干预24周后：①二氢乙锭(dihydroethidium, DHE) 染色荧光显微镜观察各组小鼠肠系膜动脉中超氧阴离子水平；②微血管张力检测仪观察肠系膜动脉血管功能，包括内皮依赖性舒张功能和非内皮依赖性舒张功能；③测定鼠尾血压观察各组小鼠干预前后的收缩压与舒张压水平。④取小鼠的胸主动脉，蛋白印迹法（Western blot）观察UCP3蛋白和p-eNOS的表达。 结果 与普盐饮食WT小鼠比较，高盐饮食WT小鼠的血管超氧阴离子水平显著升高（$P<0.01$）；而UCP3敲除则显著增加高盐环境下超氧阴离子的水平（$P<0.05$），与高盐WT小鼠比较；高盐环境使血管内皮依赖性舒张功能受损，而高盐干预的UCP3敲除小鼠与WT小鼠比较，内皮功能的损害更为显著($P<0.05$)；Western blot结果提示，高盐饮食显著增加WT小鼠胸主动脉组织中的UCP3

表达水平，降低p-eNOS水平。　　结论　　UCP3敲除后使小鼠抗氧化应激能力下降，在高盐状态下，使血管功能的损害加重，血压升高。

Abstract: Objective To determine the effects of uncoupling protein 3 (UCP3) knock-out in the vascular endothelium dysfunction in mice induced by high salt intake. Methods Male UCP3 knock-out (UCP3 KO, C57BL/6J background) and the matched wild type (WT) mice were divided into 2 groups, the normal salt intake (NS) group and high salt intake (HS) group, 10 mice in each group. The former group was given normal diet containing 0.5% salt, while the other group was fed with the diet containing 8% salt, both for 24 weeks. Mesenteric arteries were obtained and stained with dihydroethidium (DHE), and the level of superoxide anion was observed under fluorescence microscope. Vascular function, including the endothelium-dependent relaxation and endothelium-independent relaxation was measured by wire myography. Blood pressure and tail blood pressure (both systolic and diastolic blood pressures) was detected. The expression of UCP3, and phosphorylated eNOS (p-eNOS) in mice aorta were detected by Western blotting. Results Superoxide anion was much higher in mesenteric arteries from WT and UCP3 KO mice from the high salt diet group than from the normal salt diet group (P<0.01). While, high salt-fed UCP3 KO mice had markedly higher content of superoxide anion than their corresponding WT littermates (P<0.05). Endothelium-dependent relaxation was obviously impaired in mesenteric arteries from both WT and UCP3 KO mice after high salt diet feeding compared with mice having normal diet (P<0.01). Moreover, the impairment was more severe in high salt-fed UCP3 KO mice than their corresponding WT littermates (P<0.05). High salt diet feeding resulted in significantly enhanced expression of UCP3 and obviously decreased expression of p-eNOS in the WT mice aorta. Conclusion UCP3 ablation shows impaired anti-oxidative stress activity in mice, and high salt diet then further exacerbates the impairment of vascular endothelium function and leads to higher blood pressure.

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