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论著

代谢综合征大鼠肠系膜脂肪组织中肾素-血管紧张素系统的变化

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摘要 摘要：目的 探讨代谢综合征(MS)大鼠肠系膜脂肪组织中肾素-血管紧张素系统(RAS)变化及拮抗血管紧张素Ⅱ(AngⅡ)对脂肪细胞成脂作用的影响。方法 30只8周龄健康雄性Wistar大鼠随机分为MS组和正常对照组, 分别给予高脂饲料和普通饲料喂养24周, 造成MS模型后, 取出肠系膜脂肪组织, 应用RT-PCR和Western blot法检测脂肪组织中mRNA和蛋白质表达。同时, 将前脂肪细胞(3T3-L1)进行诱导分化, 油红O染色观察脂滴形成情况, 比率荧光倒置显微镜检测脂肪细胞内钙水平（\[Ca2+\]i）。给予AngⅡ刺激, 并观察血管紧张素Ⅱ受体阻断剂(ARB)坎地沙坦或血管紧张素转换酶抑制剂(ACEI)巯甲丙脯酸对脂滴形成及细胞内钙水平（\[Ca2+\]i）的作用。 结果 MS大鼠肠系膜脂肪组织的血管紧张素原(AGT)、血管紧张素转换酶(ACE)和血管紧张素Ⅱ受体亚型1(AT1R)表达均显著高于正常对照组(P＜0.05, P＜0.01); 未诱导前脂肪细胞和经AngⅡ处理的成熟脂肪细胞未见明显脂滴形成, 给予ACEI和ARB的成熟脂肪细胞有明显的脂滴形成; AngⅡ可致前脂肪细胞内钙水平（\[Ca2+\]i）显著增加(P＜0.01), 巯甲丙脯酸和坎地沙坦可阻断其效应, 而对成熟脂肪细胞, AngⅡ介导的细胞内钙水平(\[Ca2+\]i)升高受到抑制, 但坎地沙坦能恢复AngⅡ的效应,巯甲丙脯酸与AngⅡ组比较细胞内钙水平(\[Ca2+\]i)差异无显著性。结论 代谢综合征大鼠肠系膜脂肪组织中RAS系统处于激活状态, 拮抗RAS能恢复脂肪细胞的基本功能。

关键词 肾素-血管紧张素系统 血管紧张素Ⅱ 3T3-L1前脂肪细胞 脂肪组织 细胞内钙

分类号

Renin-Angiotensin System in Mesenteric Adipose Tissues in Rats with Metabolic Syndrome

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Abstract ABSTRACT:Objective To investigate the renin-angiotensin system (RAS) in mesenteric adipose ti- ssues and effect of angiotensin Ⅱ on adipocyte differentiation. Methods Thirty normal 8-week-old male Wis- tar rats were divided into groups on normal diet and high-fat diet. The rats on high-fat diet for 24 weeks deve- loped the metabolic syndrome respectively. The mRNA and protein expression of mesenteric adipose tissue were measured by reverse transcription-polymerase chain reaction(RT-PCR) and Western blot. Lipid drop in 3T3-L1 preadipocytes and mature adipocytes were observed using oil-red O staining. The fluorescence microscope was used to detect cytosolic-free calcium in 3T3-L1 predipocytes and mature adipocytes. Results The expressions of angiotensinogen, angiotensin converting enzyme, angiotensin Ⅱ receptor type 1 in mesenteric adipose tissue were significantly increased in rats with metabolic syndrome compared with those in rats on normal diet (P＜0.05,P＜0.01＝. After administration of angiotensin Ⅱ, no lipid droplet in 3T3-L1 preadipocytes and adipocytes were observed, however, intensive lipid droplet in adipocyte was found after administration of captopril and candesartan. Angiotensin Ⅱ increased the intracellular-free calcium concentration in preadipocytes(P＜0.01), which was blocked by captopril and candesartan; in contrast, angiotensin Ⅱ effect was blunt in mature adipocyte. Captopril and candesartan partially recovered the angiotensin Ⅱ-mediated increase of cytosolic-free calcium. Conclusion RAS in the mesenteric adipose tissues is active in rats with metabolic syndrome, and antagonization of RAS can recover the lipogenesis of adipocyte.

Key words renin-angiotensin system angiotensin Ⅱ 3T3-L1 preadipocyte adipose tussue intracellular Ca2+ level

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