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论著

PPARγ 激动剂对糖尿病大鼠载脂蛋白M 分泌和表达的影响

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摘要： 目的:研究高选择性过氧化物酶增殖体激活型受体γ(PPARγ) 激动剂罗格列酮(rosiglitazone,RSG) 对糖尿病大鼠血清载脂蛋白M(apoM) 水平和肝、肾、脂肪组织apoM mRNA 表达的影响。方法:雄性SD 大鼠分为对照组(Con 组,*n*=7)、高脂组(HF 组,*n*=8)、糖尿病组(DM 组,*n*=7) 和糖尿病RSG 干预组(RSG 组,*n*=7)4 组。实验模型建立前采血检测各组大鼠空腹血糖(FBG)、空腹胰岛素(FINS)、三酰甘油(TG)、总胆固醇(TC)。糖尿病大鼠模型的建立参照周氏等方法,给予高脂高糖饲料喂养、腹腔注射链脲佐菌素。应用RSG 对糖尿病大鼠干预治疗8 周。全部动物于实验第15 周结束时集中处死,搜集血标本和肝、肾、脂肪组织标本。检测各组大鼠的血FBG,FINS,TG,TC 浓度。ELISA 检测各组大鼠的血apoM 水平,并与血FBG,FINS,TG,TC 水平进行相关分析。RT-PCR 测定各组大鼠肝、肾、脂肪组织apoM mRNA 的表达。结果:与Con 组和HF 组比较,DM 组大鼠血清apoM 水平明显降低 (*P*<0.05),RSG 组大鼠的血清apoM 水平较DM 组明显升高 (*P*<0.05)。各组大鼠以肝组织apoM mRNA 的表达最高,肾组织次之,脂肪组织的表达最低。与Con 组比较,HF 组、DM 组和RSG 组的大鼠肝、肾、脂肪组织apoM mRNA 的表达明显减少(*P*<0.05),RSG 组大鼠肝、肾、脂肪组织apoM mRNA 的表达较DM 组均有明显增加(*P*<0.05)。大鼠血清apoM 水平与血清TG (*r*=-0.466,*P*=0.011),TC (*r*=-0.568,*P*=0.001),FBG (*r*=-0.371,*P*<0.001),FINS (*r*=-0.768,*P*=0.048) 水平均呈显著负相关。结论:ApoM 在血糖和血脂的代谢中起着重要作用并受PPARγ 激动剂调节的影响。

关键词： 载脂蛋白M PPARγ 糖尿病 SD大鼠

Reduced expression and secretion of apolipoprotein M in fat-fed, streptozotocin-diabetic rats is partially reversed by an artificial ligand of PPARγ Neurol Scand, 2010,121(5): 338-341.4. Leonard B, Maes M. Mechanisticex planations how

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Abstract: Objective: To investigate the effect of administration of rosiglitazone, an artificial ligand of PPARγ,on the expression and secretion of apolipoprotein (apoM) in fat-fed, streptozotocin-treated rats, ananimal model for type 2-like diabetes. Methods: Healthy male SD rats were divided into 4 groups: a control group (*n*=7), a high-fat chow group (HF group, *n*=8), a diabetes mellitus group (DM group, *n*=7), and a diabetes mellitus group with rosiglitazone intervention group (RSG group, *n*=7). Fasting blood glucose (FBG), fasting insulin (FINS), triglyceride (TG) and total cholesterol (TC) were measured at the beginning of the study. The diabetic rats model was established by feeding high fat chow and intraperitonealb injection of streprozotocin. Then the randomly selected treatment group was given rosiglitazone by daily gavage for 8 weeks. All the rats were killed at the fifteenth week, at which time blood and tissues (liver, kidney, adipose) were collected and prepared. The levels of FBG, FINS, TG and TC were assayed. The level of apoM in serum was measured by enzyme-linked immunosorbent assay (ELISA). Reverse transcription polymerase chain reaction (RT-PCR) was used to determine apoM mRNA expression in liver, kidney, and adipose tissues. Results: Compared with either control group or HF group, serum apoM concentration in the DM group was reduced significantly (*P*<0.05); compared with the DM group, however, serum apoM concentrations in RSG group were increased (*P*<0.05). The expression of apoM mRNA in liver was highest, in kidney medium, and in adipose tissue extremely low (*P*<0.05). ApoM mRNA expression in liver and kidney was decreased in both DM and HF groups compared to control group (*P*<0.05). But, as with serum apoM concentration, apoM mRNA in the liver, kidney and adipose tissues of the RSG group were all increased markedly (*P*<0.05). The level of serum apoM in SD rats correlated negatively with TG (*r*=-0.466, *P*=0.011), TC (*r*=-0.568, *P*= 0.001), FBS (*r*=-0.371,P<0.001), and FINS(*r*=-0.768, *P*= 0.048).Conclusion: These results suggest that apoM may participate in the glucose and lipid metabolism by the regulation of PPARγ.

Keywords: apolipoprotein M PPARγ diabetic mellitus SD rats

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