[1]王敏,吴晓华,李真.瘦素增强脂蛋白酯酶和肝脂酶表达加速胰岛素抵抗肝细胞模型内脂肪代谢[J].第三军医大学学报,2014,36(10):1059-1063.
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分享到:

《第三军医大学学报》[ISSN:1000-5404/CN:51-1095/R] 卷: 36 期数: 2014年第10期 页码: 1059-1063 栏目: 论著 出版日期: 2014-05-30

Title: Leptin accelerates lipid metabolism by increasing lipoprotein lipase and hepatic lipase expression in insulin-resistant liver cell model

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关键词: 胰岛素抵抗; 瘦素; 脂蛋白酯酶; 肝脂酶

Keywords: insulin resistance; leptin; hepatic lipase; lipoprotein lipase

分类号: R587.1; R589.2; R977.6

文献标志码: A

摘要: 目的 在体外模型中探讨胰岛素抵抗情况下，瘦素(leptin)对脂代谢的影响及途径。 方法 用1 μmol/L胰岛素诱导LO2(人正常肝细胞) 细胞，构建肝细胞胰岛素抵抗模型，并用0、50、100、250 μg/L瘦素以及2 μmol/L瘦素拮抗剂 (leptin-A) 分别处理细胞。实时定量PCR检测细胞中肝脂酶(hepatic lipase, HL) 和脂蛋白酯酶(lipoprotein lipase, LPL) mRNA表达；蛋白质免疫印迹法检测细胞中HL和LPL蛋白表达；油红O染色观测细胞内油脂的堆积程度。 结果 ①高浓度胰岛素处理LO2细胞后，处理组细胞对一定浓度胰岛素的响应比对照组有显著下降，其代谢葡萄糖效率显著下降，细胞产生胰岛素抵抗。②胰岛素抵抗细胞中HL和LPL mRNA和蛋白水平明显低于对照组细胞。瘦素处理胰岛素抵抗细胞24 h后，HL和LPL mRNA和蛋白水平有明显回升，细胞内油脂堆积明显减少。③瘦素拮抗剂能明显抑制瘦素诱导的HL和LPL mRNA和蛋白的上调，导致细胞内油脂明显堆积。 结论 肝细胞胰岛素抵抗模型中瘦素通过增强HL、LPL mRNA和蛋白水平的表达加速细胞内脂肪的代谢。

Abstract: Objective To determine the relationship between leptin and the intracellular fat metabolism in insulin-resistant liver cell models. Methods Insulin-resistant liver cell models (LO2-I) were generated from LO2 cells with long-term treatment of 1 μmol/L insulin. The LO2-I cells were then treated with leptin (0, 50, 100, and 250 μg/L) and/or 2 μmol/L leptin antagonist (leptin-A). The

transcription of hepatic lipase (HL) and lipoprotein lipase (LPL) genes was determined by real-time PCR. The protein expression of HL and LPL was evaluated by Western blotting. Oil red-O staining was used to observe intracellular lipid accumulation in LO2-I cells. Results LO2 cells with long-term treatment of 1 μmol/L insulin showed a significant decrease in insulin treatment response, indicating insulin resistance. LO2-I cells had an expression decrease of HL and LPL on both mRNA and protein levels, as compared to LO2 cells. The mRNA and protein levels of HL and LPL were increased significantly after being treated with leptin for 24 h in the LO2-I cells. Meanwhile, the intracellular accumulation of lipid was also significantly reduced. Leptin antagonist inhibited the leptin-induced HL and LPL expression up-regulation and caused obvious intracellular lipid accumulation. Conclusion Leptin accelerates lipid metabolism by increasing LPL and HL expression at both mRNA and protein levels in gestational diabetes liver cell model.

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