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论著

氧自由基对3T3-L1脂肪细胞表达PAI-1的调节及其可能机制

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摘要 目的： 研究脂肪细胞中氧自由基（ROS）对纤溶酶原激活物抑制物-1（PAI-1）表达的调节，并探讨其机制。方法：培养3T3-L1细胞，并诱导其分化成为脂肪细胞，以MTT比色法检测细胞的活性。分别以定量PCR、多重免疫分析及夹心ELISA法检测PAI-1 mRNA和蛋白表达的水平，并采用多重磷酸化蛋白分析系统检测细胞内多种信号分子的蛋白磷酸化水平。结果：H2O2可剂量依赖性地增加PAI-1的产生。并且激活了3T3-L1脂肪细胞中多种信号转导通路，包括ERK1/2、JNK、Akt、p70 S6K及JAK/STAT，其中Akt、JAK/STAT及ERK1/2的活化可能参与到H2O2对于PAI-1的调节过程中。结论： H2O2可能通过磷酸化激活Akt、JAK/STAT及ERK1/2，上调脂肪细胞PAI-1的表达。

关键词 自由基； 脂细胞； 纤溶酶原激活物抑制物1

分类号 R33

Regulatory effects of reactive oxygen species on the production of PAI-1 in 3T3-L1 adipocytes and its related possible mechanisms

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Abstract

<FONT face=Verdana>AIM: To investigate the regulatory effects of reactive oxygen species (ROS) on the production of plasminogen activator inhibitor 1 (PAI-1), and try to determine the signaling cascades involved in it. METHODS: 3T3-L1 cells were cultured and differentiated into mature adipocytes. Cell viability was measured by MTT. The PAI-1 mRNA expression levels were evaluated by quantitative real-time PCR. Quantification of the PAI-1 protein levels secreted into conditioned medium was performed by multiplex immunoassay and sandwich ELISA. The phosphorylation status of protein kinases was determined by Bio-Plex phosphoprotein assays. RESULTS: In 3T3-L1 adipocytes, H2O2 significantly augmented the expression of PAI-1. Also, H2O2 activated several signaling pathways including ERK1/2, JNK, Akt, p70 S6K and JAK/STAT. Verified by protein kinase inhibitors, Akt, JAK/STAT and ERK1/2 may participate in the H2O2-induced increase in PAI-1. CONCLUSION: H2O2 markedly up-regulates the production of PAI-1 in 3T3-L1 adipocytes via some intracellular signaling pathways such as Akt, JAK/STAT and ERK1/2. </FONT>

Key words Free radicals Adipocytes Plasminogen activator inhibitor 1

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