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综述

烟酸受体GPR109A介导的烟酸作用机制研究进展

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摘要 烟酸作为调脂药物应用于临床已经50多年, 相对高剂量的烟酸具有广泛的调脂作用。越来越多的证据表明，烟酸单用或联合降低低密度脂蛋白胆固醇（LDL-C）药物使用能够延缓动脉粥样硬化（AS）的进程和降低发生心血管事件的风险；但它的作用机制一直不是很明确。2003年，三个研究小组同时发现了烟酸受体, 使人们对烟酸的作用机制有了进一步认识。烟酸受体GPR109A是一种Gi蛋白偶联受体，主要表达于白色、棕色脂肪组织、脾和免疫细胞。研究表明，烟酸受体主要介导以下作用：① 烟酸作用于脂肪细胞的烟酸受体，抑制脂肪组织甘油三酯（TG）的水解，降低血浆游离脂肪酸（FFA）；② 烟酸作用于皮肤角质细胞和朗格汉细胞的烟酸受体诱导前列腺素分泌，引起皮肤血管舒张，引发潮红；③ 激活烟酸受体还可增加脂联素分泌，诱导中性粒细胞凋亡，上调过氧化物酶体增殖激活受体γ表达。

关键词 动脉粥样硬化 烟酸受体 潮红 G蛋白偶联受体

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Progess in mechanism of nicotinic acid mediated by nicotinic acid receptor GPR109A

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Abstract

Nicotinic acid (niacin) has been used for the treatment of lipid disorders for more than fifty years and relatively high doses of nicotinic acid induces profound change in the lipid and lipoprotein profile. There is increasing evidence that nicotinic acid alone or in addition to LDL cholesterol -lowering drugs can improve the progression of atherosclerosis and reduce the risk of cardiovascular events; but the mechanisms underlying the pharmacological effects of nicotinic acid have been unclear for decades. The discovery of nicotinic acid receptors in 2003 by 3 research groups has allowed for better understanding of the machanisms of nicotinic acid. Nicotinic acid receptor GPR109A is an Gi-coupled receptor,highly expressed in white and brown adipose tissue,spleen and immune cells.New findings indicated that nicotinic acid receptor induced the following: ① in adipose, nicotinic acid agonized the receptor inhibiting triglyceride hydrolysis to lower plasma free fatty acids (FFA) level; ② nicotinic acid provoked the receptor of keratinocytes and Langerhans cells of skin to produce prostaglandin,dilating vascellum of skin,initiating flushing; ③ the activation of nicotinic acid receptor enhanced adiponectin secretion, induced neutrophils apoptosis, and upregulated the expression of PPARgamma.

Key words atherosclerosis nicotinic acid receptor flushing G protein- coupled receptor

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