综述

解偶联蛋白在脑缺血缺氧性损伤中的作用

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摘要

线粒体解偶联蛋白(uncoupling proteins，UCPs)是线粒体载体蛋白家族的一个亚族，位于线粒体内膜，可以将线粒体内膜外的质子转运回基质，降低线粒体的跨膜质子电动势，形成质子漏，使氧化磷酸化解偶联，ATP生成减少。目前共发现有5个成员，UCP1仅存在于棕色脂肪组织，主要参与机体非震颤产热；UCP2，UCP4和UCP5(又名脑线粒体膜载体蛋白1)在脑组织中表达较多。近年来UCPs在脑缺血缺氧性损伤中的病理生理作用研究较多，但也颇多争议。有人认为UCPs通过解偶联作用加重能量衰竭，进一步促进脑缺血缺氧损伤；也有人认为UCPs通过调节线粒体膜电位，抑制活性氧簇生成，维持线粒体内钙稳态，从而起到对神经系统的保护作用。

关键词： 解偶联蛋白；线粒体；脑缺血缺氧性损伤

Role of uncoupling proteins in the hypoxic-ischemic brain damage

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Abstract

Mitochondrial uncoupling proteins (UCPs) are a subfamily of the mitochondrial transporter family, which locate in the inner mitochondrial membrane. UCPs play a role in transferring proton from membrane space to matrix, reduction of proton gradient and uncoupling the oxidative phosphorylation, which ultimately leads to the decreases of ATP levels

in cell. There are 5 members (from UCP1 to UCP5) in total in UCPs family. UCP1 is expressed exclusively in the brown adipose tissue (BAT) and is responsible for non-shivering thermogenesis. UCP2, UCP4 and UCP5 (brain mitochondrial carrier protein 1, BMCP1) were found to be expressed in the brain at high level. Recently, the important roles of UCPs in the pathophysiological process of the hypoxia and ischemia brain diseases have emerged, but these roles are also controversial. Some people think that uncoupling process by UCPs aggravates the energy failure and accelerates the progress of the disease, while others think that UCPs could regulate the mitochondrial membrane potential (MMP), reduce ROS generation and maintain the calcium homeostasis, and thus protect the central nerve system.

Keywords: uncoupling proteins | mitochondrial | hypoxic-ischemic brain damage

全文

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引用

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