

中药活性成分调控三羧酸循环抗动脉粥样硬化的研究进展^Δ

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摘要 动脉粥样硬化(AS)的发生与内皮功能障碍及线粒体代谢异常等存在密切关联。这种异常本质上体现为细胞的代谢重编程,而三羧酸(TCA)循环作为介导该重编程的核心环节,其所引起的中间代谢物(如琥珀酸、 α -酮戊二酸、衣康酸等)稳态失衡,可通过多种机制(如调控氧化应激、炎症及糖脂代谢网络)参与AS的发展进程。中药活性成分具有多靶点、多通路协同调控的特点,在改善代谢紊乱方面显示出独特优势。黄酮类、苷类等多种中药活性成分能够靶向调控TCA循环关键酶及其代谢物,通过纠正异常的代谢重编程网络,减轻氧化应激、炎症及糖脂代谢紊乱对血管内皮的损伤,进而改善代谢紊乱状态并抑制AS进展。

关键词 中药活性成分;动脉粥样硬化;三羧酸循环;炎症反应;氧化应激;糖脂代谢;代谢重编程

Research progress on the regulation of the tricarboxylic acid cycle by bioactive constituents of traditional Chinese medicine in the anti-atherosclerotic process

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ABSTRACT The development of atherosclerosis (AS) is closely associated with endothelial dysfunction and mitochondrial metabolic abnormalities. These alterations are fundamentally characterized by metabolic reprogramming, in which the tricarboxylic acid (TCA) cycle serves as a central regulatory hub. Disruption of the homeostasis of TCA cycle intermediates, including succinate, α -ketoglutarate, and itaconate, contributes to AS progression through multiple mechanisms, such as the modulation of oxidative stress, inflammation, and glucose and lipid metabolic networks. Owing to their multi-target and multi-pathway regulatory properties, bioactive compounds derived from traditional Chinese medicine (TCM) have demonstrated unique advantages in correcting metabolic dysregulation. Flavonoids, glycosides, and other bioactive constituents of TCM can target key enzymes and metabolites involved in the TCA cycle, thereby restoring aberrant metabolic reprogramming, alleviating endothelial injury induced by oxidative stress, inflammation, and glucose and lipid metabolic disorders, ultimately improving metabolic homeostasis and attenuating the progression of AS.

KEYWORDS bioactive constituents of traditional Chinese medicine; atherosclerosis; tricarboxylic acid cycle; inflammation; oxidative stress; glucolipid metabolism; metabolic reprogramming

动脉粥样硬化(atherosclerosis, AS)是由富含脂质的炎性斑块沉积于动脉壁,造成动脉壁增厚、管腔狭窄的一类动脉硬化性血管病。其流行率逐年攀升,且发病率

随患者年龄增长显著上升^[1]。AS的发病机制复杂,除内皮功能障碍、炎症反应及糖代谢紊乱外,代谢重编程已成为当前该领域研究的热点之一。代谢重编程是指细胞为适应微环境应激,对其固有代谢网络(如氧化磷酸化及糖脂代谢等通路)进行动态重塑,以满足生存所需要的能量供应、维持氧化还原稳态及合成生物大分子^[2]。作为代谢重编程的核心环节,线粒体三羧酸(tricarboxylic acid, TCA)循环的失衡在AS的病理演变中发挥了关键作用,因此靶向修复TCA循环功能已成为干预AS的重要策略之一。

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