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综述

血管紧张素转换酶2在SARS病理途径中的作用

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摘要 人类严重急性呼吸综合征(SARS)相关冠状病毒（SARS-CoV）感染导致的严重急性呼吸系统病变，其临床肺部病理损害特征与急性肺损伤和急性呼吸窘迫病变相似。SARS-CoV可以结合人血管紧张素转换酶（ACE）2，二者结合效率与病毒感染复制能力相关。ACE2与ACE1共同调控肾素 血管紧张素系统，二者的功能平衡维持肺的正常功能。SARS-CoV感染时，其棘突蛋白与ACE2结合，下调人体ACE2水平，肺内ACE2和ACE1功能失衡，血管紧张素Ⅱ过度激活AT_1受体，导致肺部毛细血管通透性增加，随之出现肺水肿和急性肺损伤。ACE2是SARS病理途径中的关键因子，在SARS临床治疗和SARS药物研制中有重要意义。

关键词 血管紧张素转换酶2 严重急性呼吸综合征 急性肺损伤

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Role of angiotensin converting enzyme 2 in pathogenesis of severe acute respiratory syndrome

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Abstract

Infection of human severe acute respiratory syndrome-associated coronavirus(SARS-CoV) can lead to serious lung injury symptoms which resemble the acute lung injury(ALI) and acute respiratory distress syndrome(ARDS). Human angiotensin converting enzyme(ACE) 2 has been recognized as the specific binding receptor of SARS-CoV spike protein(S protein), and the efficiency of their binding is relative to the replication and infection ability of SARS-CoV. The balance between ACE1 and ACE2 is crucial for the normal function of lung, in which the stability of functional ACE2 can attenuate the severity of ALI and ARDS. In infection of SARS-CoV, S protein of SARS-CoV binds to the human ACE2 and down-regulates the ACE2 level. Then it follows the unbalance between ACE2 and ACE1, which leads AT_1 receptor to be over stimulated by increasing angiotensin Ⅱ level. The pulmonary edema and symptom of ALI in SARS patients can be due to the leaky pulmonary blood vessels through over-stimulation of AT_1 receptor. Therefore, ACE2 is a key molecule involved in the development and progression of SARS.

Key words angiotensin converting enzyme 2 severe acute respiratory syndrome acute lung injury

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