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论著

气道给rhSOD对胎粪诱导肺损伤中NF-κB及MIP-1α表达的影响

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摘要 目的：观察早期经气道给予重组人超氧化物歧化酶（rhSOD）对胎粪诱导大鼠肺NF-κB和炎症因子MIP-1α表达的影响，以探讨其在胎粪诱导肺损伤中的作用及其机制。 方法： 24只雄性SD大鼠，随机分为：（1）对照组（control）,经气管插管注入生理盐水1 mL/kg；（2）胎粪+生理盐水处理组（Mec/saline）；（3）胎粪+ rhSOD治疗组(Mec/rhSOD)。后两者先由气管插管注入20%新生儿胎粪生理盐水混悬液1 mL/kg建立急性肺损伤模型，再分别经气管插管注入生理盐水1 mL/kg或rhSOD 20 g·L-1·kg-1。24 h后取材， RT-PCR法测定肺组织MIP-1α mRNA、Western blotting法测定NF-κB蛋白表达改变，同时行支气管肺泡灌洗液（BAL）细胞计数。 结果： Mec/saline组大鼠BAL细胞计数、肺组织MIP-1α mRNA和NF-κB蛋白表达均明显高于control组［(4.68±1.40)×109 cells/L vs (0.53±0.19)×109 cells/L, 3.60±0.75 vs 1.56±0.33，0.72±0.31 vs 0.23±0.21］，（均P<0.01）； Mec/rhSOD组大鼠BAL细胞计数、肺组织MIP-1α mRNA和NF-κB蛋白表达分别为(3.13±0.77)×109cells/L、2.20±0.39和0.44±0.21，均显著低于Mec/saline组（均P<0.01），但仍显著高于control组（均P<0.01）。 结论： 早期经气道给rhSOD可能通过抑制肺MIP-1α和NF-κB表达而减轻胎粪诱导的肺炎症反应。

关键词 超氧化物歧化酶； 胎粪； 肺损伤； NF-κB； 巨噬细胞炎性蛋白质1

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Effect of rhSOD on pulmonary nuclear factor-kappa B and MIP-1α expression in meconium-induced acute lung injury

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Abstract

<FONT face=Verdana>AIM: To evaluate the role and mechanisms of recombinant human superoxide dismutase (rhSOD) in meconium-induced acute lung injury (ALI) by evaluating pulmonary MIP-1α and NF-κB expression. METHODS: 24 health male Sprage-Dawley rats were randomized to 3 groups (8, each group), followed by intratracheal (IT) administration with (1) saline at 1 mL/kg (control group); (2) 20% human newborn meconium suspension at 1 mL/kg, followed by saline at 1 mL/kg (Mec/saline group); (3) 20% human newborn meconium suspension at 1mL/kg, followed by rhSOD at 20 mg/kg (Mec/rhSOD group). The animal was killed 24 h after treatment. The measurements included the bronchoalveolar lavage (BAL) cell count, RT-PCR analysis of pulmonary MIP-1α mRNA expression, Western blotting analysis of pulmonary NF-κB expression. RESULTS: Meconium-induced ALI was characterized by increased BAL cell count, increased expressions of pulmonary MIP-1α mRNA and NF-κB protein ［(4.68±1.40)×109 cells/L vs (0.53±0.19)×109 cells/L, 3.60±0.75 vs 1.56±0.33, 0.72±0.31 vs 0.23±0.12, respectively in control rats, all P<0.01］. IT administration of rhSOD early in the ALI rat significantly decreased meconium-induced BAL cell count ［(3.13±0.77)×109 cells/L vs (4.68±1.40)×109 cells/L in Mec/saline rats, P<0.01］, inhibited the expression of pulmonary MIP-1α mRNA (2.20±0.39 vs 3.60±0.75, in Mec/saline rats, P<0.01) and NF-κB protein (0.44±0.21 vs 0.72±0.31 in Mec/saline rats, P<0.05). CONCLUSION: The early IT administration of rhSOD in ALI rat following meconium aspiration protects lung from inflammatory injury through inhibiting meconium-induced pulmonary MIP-1α mRNA and NF-κB protein expression.</FONT>

Key words Superoxide dismutase Meconium Lung injury NF-kappa B Macrophage inflammatory

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