

论著

E2F1蛋白在高氧致慢性肺疾病早产鼠肺组织的动态表达及意义

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摘要:

目的:通过检测E2F1蛋白在高氧致慢性肺疾病早产鼠肺组织中的动态表达,初步探讨E2F1蛋白与慢性肺疾病肺间质纤维化发生发展的关系。方法:剖宫术取出孕21 d Wistar 大鼠作为早产鼠,生后12 h 随机分为高氧组和对照组,高氧组持续暴露于90% 氧气中,空气组置于同一室内常压空气中。分别于暴露3, 7, 14 d 时,每组取动物10 只,留取其肺组织标本。应用HE 染色观察不同时间点其肺组织病理改变,在光镜下进行肺组织纤维化评分,并采用免疫组织化学法及Western 印迹检测不同时间点肺组织E2F1 蛋白的表达。结果:早产鼠高氧暴露3 d 后未出现纤维化改变,7 d 后出现少许纤维化改变,14 d 后纤维化改变明显;E2F1 在高氧暴露3 d 在肺组织中E1F1 蛋白表达虽较同时点空气组有所升高,但差异无统计学意义($P>0.05$),高氧暴露7 d 及14 d E2F1 蛋白表达明显高于同时点空气组($P<0.05$, $P<0.01$)。结论:高氧导致早产鼠肺组织E2F1 表达持续性增高,其异常表达可能是导致肺成纤维细胞过度增殖,最终发生肺间质纤维化的重要原因。

关键词: 高氧 早产儿 慢性肺疾病 肺间质纤维化 E2F1 蛋白

Dynamic expression of E2F1 in lung of premature rats with hyperoxia-induced chronic lung disease and its significance

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Abstract:

Objective: To determine the dynamic expression of E2F1 in lung of premature rats with hyperoxia-induced chronic lung disease and the relation between E2F1 and pulmonary fibrosis. Methods: Premature Wistar rats at 21 days gestation were randomly and equally divided into a hyperoxia group and a room air group. The hyperoxia group was continuously exposed to hyperoxia (90%) while the air group in room air. Lung tissues in the 2 groups were obtained at 3, 7 and 14 days after exposing to either room air or hyperoxia. The changes of pulmonary histopathology at different time points were observed by hematoxylin and eosin staining; the severity of pulmonary fibrosis was evaluated; and the expression of E2F1 in lung tissue was detected by immunohistochemistry and Western blot. Results: After 3 days of hyperoxia, no significant interstitial fibrosis was observed; while after 7 days in the hyperoxia group, interstitial fibrosis was observed. These changes became more obvious after 14 days of prolonged hyperoxia exposure. No significant difference in the expressions of E2F1 protein was found between the hyperoxia group and the room air group 3 days postnatally ($P>0.05$). The expression of E2F1 in the hyperoxia group significantly increased 7 days and 14 days postnatally ($P<0.05$, $P<0.01$). Conclusion: Abnormality of E2F1 expression is involved in the pathological process of the proliferation of lung fibroblasts in hyperoxia-induced chronic lung disease neonatal rats, and it plays an important role in lung fibrosis.

Keywords: hyperoxia premature infant chronic lung disease interstitial fibrosis E2F1

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