[1]李青,徐树红,郑兴惠,等.神经肽P物质对大鼠高氧肺损伤的保护作用[J].第三军医大学学报,2014,36(06):578-582.
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Title: Protective effect of substance P on hyperoxic lung injury in rats

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关键词: 高氧肺损伤; 神经肽P物质; p38MAPK

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摘要: 目的 观察p38MAPK信号途径在高氧肺损伤中的表达，探讨神经肽P物质(substance P，SP)在高氧肺损伤中的保护作用及机制。 方法 将32只早产Sprague-Dawley大鼠按随机数字表法分配给代母鼠并分为4组，每组8只：正常对照组、空气加SP组、高氧加9 g/L盐水组、高氧加SP组。空气加SP组和高氧加SP组动物每天予腹腔注射SP 1×10^{-6} mol·L^{-1}·kg^{-1}，正常对照组和高氧加9 g/L盐水组腹腔注射同等体积的9 g/L盐水。实验7 d后，观察肺组织病理改变；测定肺湿/干质量值(W/D)及肺组织中SP的含量；检测PCNA观察肺组织细胞的凋亡情况；采用免疫组化法检测p38在肺组织中的分布；蛋白免疫印迹法检测p38MAPK蛋白含量。 结果 与正常对照组相比，高氧各组均有不同程度的肺损伤，但SP干预组肺损伤较高氧加9 g/L盐水组有所减轻。肺W/D在第3、7天高氧肺损伤组明显高于正常对照组，高氧加SP组比高氧加9 g/L盐水组肺W/D在第7天显著降低。空气加SP组和高氧加SP组肺组织中的SP含量与正常对照组相比显著升高。免疫组化显示，高氧加9 g/L盐水组p38阳性表达较正常对照组明显增强；SP干预后p38MAPK阳性细胞较高氧加9 g/L 盐水组明显减少。蛋白免疫印迹法显示，高氧加9 g/L盐水组p38MAPK含量明显高于正常对照组，干预后高氧加SP组p38MAPK含量低于高氧加9 g/L盐水组。各组肺W/D值、PCNA组织分布表达与p38MAPK蛋白含量变化趋势一致。 结论 高氧应激可激活损伤肺组织p38MAPK活性，SP对高氧肺损伤保护作用机制可能是通过下调高氧诱导的p38MAPK的激活实现的。

Abstract: Objective To investigate the expression of p38MAPK in hyperoxic lung injury (HLI), and explore the protective effect of substance P (SP) on HLI and its mechanism. Methods Thirty-two SD rats were divided randomly into 4 groups with 8 rats in each group: a room-air plus saline group, a room-air plus SP group, a hyperoxia injury plus saline group, and a hyperoxia injury plus SP group. Rats were injected with SP (1×10^{-6} mol·L^{-1}·kg^{-1}·d^{-1}) intraperitoneally in the room-air plus SP group and hyperoxia injury plus SP group. The rats were sacrificed after 7 d of experiment. Lung pathology was examined with light microscopy. Lung wet/dry (W/D) ratio and the content of SP and PCNA in lung were evaluated. The distribution and quantity of p38MAPK protein were detected by immunohistochemistry and Western blotting, respectively. Results The pathological changes were observed in the lungs of all the hyperoxia injury groups, while the lung pathological pictures in the hyperoxia injury plus SP group was improved significantly as compared to those in the hyperoxia injury plus saline group. Lung W/D in all the hyperoxia injury groups was significantly higher than that in the room-air plus saline group at day 3 or 7. Lung W/D in the hyperoxia injury and SP group was decreased significantly as compared to that in the hyperoxia injury plus saline group at day 7. The content of SP in the hyperoxia injury and SP group and

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room-air plus SP group were increased significantly as compared to that in the room-air plus saline group. P38MAPK-positive cells increased in the hyperoxia injury plus saline group as compared to those in the room-air plus saline group. In the hyperoxia injury and SP group, the positive cells remarkably decreased as compared to those of the hyperoxia injury plus saline group. P38MAPK content was higher in the hyperoxia injury plus saline group than in the room-air plus saline group, and p38MAPK expression in the hyperoxia injury plus SP group decreased significantly as compared to that in the hyperoxia injury plus saline group. Changes in W/D ratio and PCNA expression were comparable with those of p38MAPK protein quantity. Conclusion Hyperoxia activates p38MAPK signaling pathway. SP may exert a protective effect on HLI through attenuation of hyperoxia-induced p38MAPK activation.

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