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Sheng Baoliang,Xu Gang,Chen Dewei,et al.T-AOC，MDA，SOD，CAT and IL-6 levels in rat pulmonary edema induced by hypobaric hypoxia[J].J Third Mil Med Univ,2012,34(23):2364-2367.

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大鼠实验性高原肺水肿中T-AOC、MDA、SOD、CA

《第三军医大学学报》[ISSN:1000-5404/CN:51-1095/R] 卷: 34 期数: 2012年第23期 页码: 2364-2367 栏目: 论著 出版日期: 2012-12-15

Title: T-AOC，MDA，SOD，CAT and IL-6 levels in rat pulmonary edema induced by hypobaric hypoxia

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关键词: 缺氧; 高原肺水肿; 氧化应激

Keywords: hypoxia; high-altitude pulmonary edema; oxidative stress

分类号: R362；R364.4；R563.9

DOI: -

文献标识码: A

摘要: 目的　观察氧化应激反应在急性缺氧致大鼠高原肺水肿发生中的变化，并探讨其病理生理学意义。　方法　成年雄性SD大鼠72只，体质量（200 ±20）g，按随机数字表法分为平原对照（C）组、模拟急性高原缺氧（H）6、12、24、48和72 h组（*n*=12）。H组动物置于减压舱内模拟海拔6 000 m高原暴露相应时间；分别在平原和模拟高原环境取材，检测肺组织湿干质量比值、总抗氧化能力（total-antioxidant capacity，T-AOC）、丙二醛（malondialdehyde，MDA）、超氧化物歧化酶（superoxide dismutase，SOD）、过氧化氢酶（catalase，CAT）、血浆及肺组织IL-6。　结果　与平原对照组比较：缺氧各组大鼠肺组织湿干质量比值均显著增加（*P*<0.05，*P*<0.01）；MDA随缺氧时间延长逐渐增加，缺氧72 h达到最高（*P*<0.01）；SOD在缺氧12、24 h显著降低（*P*<0.05），在缺氧48、72 h进一步降低（*P*<0.01）；T-AOC、CAT随缺氧时间延长在24、48、72 h显著降低（*P*<0.05）；血浆IL-6在缺氧24、48、72 h组显著增加（*P*<0.01），肺组织IL-6在缺氧48（*P*<0.01）、72 h（*P*<0.05）组显著增加。　结论　急性缺氧诱导大鼠高原肺水肿的发生和氧化应激有关，机体抗氧化能力降低、自由基增加是HAPE发生的重要机制。

Abstract: Objective　To observe the changes of oxidative stress in the occurrence of pulmonary edema in rats induced by hypoxia and to investigate the pathophysiological significance.　Methods　A total of 72 adult male SD

rats weighting 200±20 g were randomly divided into control group (C), and simulated altitude acute hypoxia groups (H). The hypoxia group rats were exposed to a simulated altitude of 6 000 m for 6, 12, 24, 48 and 72 h respectively. The lung wet/dry (W/D) ratio, total-antioxidant capacity (T-AOC), malondialdehyde (MDA) content, activity of superoxide dismutase (SOD) and catalase (CAT) were measured. Lung and plasma IL-6 were measured by ELISA. Results The W/D ratio was increased in hypoxia groups than in control group (H6, H12 and H24 vs C, $P<0.01$, H48 and H72 *vs* C, $P<0.05$). MDA content was gradually increased with the prolongation of hypoxia time, and reached the highest in 72 h after hypoxic exposure （$P<0.01$）. SOD content was significantly deceased in 12 and 24 h after hypoxic exposure（$P<0.05$） and further reduced in 48 and 72 h（$P<0.01$）. Contents of T-AOC and CAT were significantly reduced with the prolongation of hypoxia time in 24, 48 and 72 h（$P<0.05$）. Plasma IL-6 was significantly increased in hypoxic 24, 48 and 72 h groups （$P<0.01$）, while that in lung tissue was significantly increased in hypoxic 48 and 72 h groups compared with the control group（H48 *vs* C, $P<0.01$; H72 *vs* C, $P<0.05$）. Conclusion Acute hypoxia-induced high altitude pulmonary edema is related to oxidative stress. Decrease of antioxidant capacity and increase of free radicals contribute to the occurrence of high altitude pulmonary edema.

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备注/Memo: -

更新日期/Last Update: 2012-12-04