中国药理学与毒理学杂志 2009 23 (2): 111-115 ISSN: 1000-3002 CN: 11-1115/R

论著

# 新生大鼠高氧性肺损伤肺组织内源性谷氨酸释放的变化

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收稿日期 2008-6-18 修回日期 网络版发布日期 2009-4-9 接受日期 2009-2-27

摘要 目的 探讨谷氨酸在新生大鼠高氧性肺损伤中的作用。方法 SD新生大鼠，出生后12 h内随机分为空气对照组和高氧组。高氧组维持氧浓度≥95%，分别在1，3和7 d后每组处死5只大鼠，取肺脏，测定肺组织湿重/干重（W/D）比值，HE染色观察肺组织病理变化；另取小鼠，进行支气管肺泡灌洗，制备支气管肺泡灌洗液（BALF），用血细胞计数板进行白细胞计数，全自动生化分析仪测定乳酸脱氢酶（LDH）活性，Lowry法检测总蛋白含量，高效液相色谱法检测谷氨酸含量。结果 与空气对照组比较，持续高氧暴露1 d新生大鼠肺组织W/D比值无明显变化，暴露3和7 d W/D比值明显增加。HE染色可见，持续高氧暴露3 d肺泡腔内少量炎症细胞渗出，暴露7 d肺泡内红细胞和炎症细胞进一步增多，肺组织结构紊乱，肺泡数量减少。持续高氧暴露1 d新生大鼠BALF中LDH活性明显增加，白细胞计数和总蛋白含量无明显变化，暴露3和7 d BALF中LDH活性、总蛋白含量和白细胞计数均高于空气对照组。持续高氧暴露1和3 d BALF中谷氨酸含量亦明显高于空气对照组。结论 高浓度氧可引起新生大鼠急性肺损伤，诱导肺组织内源性谷氨酸的释放，提示谷氨酸在高氧性肺损伤中发挥重要作用。

关键词 高氧症 急性肺损伤 谷氨酸盐类

分类号 R963

# Changes of endogenous glutamate release in lungs of newborn rats with hyperoxia-induced lung injury

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**Abstract**

**AIM** To demonstrate the role of intrinsic glutamate (Glu) in hyperoxia-induced lung injury. **METHODS** SD newborn rats, in 12 h after birth, were randomly divided into 2 groups: air control and hyperoxia (95% oxygen) groups. After 1, 3 and 7 d of continuous exposure to high concentration oxygen, the lungs of 5 rats in each group were removed. Then the wet-to-dry weight ratio (W/D) was measured and histopathological changes were observed with HE staining. The bronchoalveolar lavage fluid (BALF) of other rats in each groups was prepared and the leucocyte numbers, total protein (TP) concentrations and lactate dehydrogenase (LDH) activity in BALF was determined with hemocytometer, Lowry method and automatic biochemical analyzer, respectively. In addition, the level of Glu in BALF was determined by using high performance liquid chromatograph. **RESULTS** There was no difference in W/D between the air control and hyperoxia groups 1 d after hyperoxia exposure. After 3 and 7 d, W/D in hyperoxia groups were much higher than that of the air control group. HE staining showed that a few inflammatory cells appeared in some alveolar space in the hyperoxia group on 3 d. On 7 d, leukopedesis and red blood cells increased in the alveolar space, and there were fewer alveolai compared with control group. On 1 d, LDH activity in hyperoxia group was significantly higher than that in control group. The TP contents and leucocyte count had no obvious changes between the 2 groups. On 3 and 7 d, LDH activity, TP content and leucocyte count in hyperoxia groups were much higher than that of control group. In addition, the glutamate level in BALF on 1 and 3 d of hyperoxia exposure was significantly higher than that in control group. **CONCLUSION** Hyperoxia can induce acute lung injury and intrinsic Glu release in lungs of newborn rats, which show that Glu may play an important role in hyperoxia-induced lung injury.

**Key words** hyperoxia acute lung injury glutamates

DOI: 10.3867/j.issn.1000-3002.2009.02.005

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