18β-Glycyrrhetinic acid protectes neonatal rats with hyperoxia exposure through inhibiting ROS/NF-κB/NLRP3 inflammasome

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Abstract

Bronchopulmonary dysplasia (BPD) is a common devastating pulmonary complication in preterm infants. Oxygen supplementation is a lifesaving therapeutic measure used for premature infants with pulmonary insufficiency. However, oxygen toxicity is a significant trigger for BPD, and oxidative stress-induced inflammatory responses, in turn, worsens the oxidative toxicity resulting in lung injury and arresting of lung development. Glycyrrhiza radix is commonly used in the medicine and food industries. 18β-Glycyrrhetinic acid (18β-GA), a primary active ingredient of Glycyrrhiza radix, has a powerful anti-oxidative and anti-inflammatory effects. This study aimed to determine whether 18β-GA has protective effects on neonatal rats with hyperoxia exposure. Newborn Sprague-Dawley rats were kept in either 21% (normoxia) or 80% O2 (hyperoxia) continuously from postnatal day (PN) 1 to 14. 18β-GA was injected intragastrically at 50 or 100 mg/kg body weight once a day from PN 1 to 14. We examined the body weights and alveolar development, and measured ROS level and the markers of pulmonary inflammation. Mature-IL-1β and NF-κB pathway proteins, and the NLRP3 inflammasome, were assessed; concurrently, caspase-1 activity was measured. Our results indicated that hyperoxia resulted in alveolar simplification and decreased bodyweight of neonatal rats. Hyperoxia exposure increased ROS level and pulmonary inflammation, and activated NF-κB and the NLRP3 inflammasome. 18β-GA treatment decreased ROS level, inhibited the activation of NF-κB and the NLRP3 inflammasome, decreased pulmonary inflammation, improved alveolar development, and increased the bodyweight of neonatal rats with hyperoxia exposure. Our study demonstrates that 18β-GA protects neonatal rats with hyperoxia exposure through inhibiting ROS/NF-κB/NLRP3 inflammasome.

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