Nitric Oxide Mediates Lung Injury Induced by Ischemia-Reprtfusion in Rats

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摘要

Abstract

Nitric oxide (NO) has been reported to play a role in lung injury (LI) induced by ischemia-reperfusion (I/R). However, controversy exists as to the potential beneficial or detrimental effect of NO. In the present study, an in situ, perfused rat lung model was used to study the possible role of NO in the LI induced by I/R. The filtration coefficient (Kfc), lung weight gain (LWG), protein concentration in the bronchoalveolar lavage (PCBAL), and pulmonary arterial pressure (PAP) were measured to evaluate the degree of pulmonary hypertension and LI. I/R resulted in increased Kfc, LWG, and PCBAL. These changes were exacerbated by inhalation of NO (20-30 ppm) or 4 mM L-arginine, an NO precursor. The permeability increase and LI caused by I/R could be blocked by exposure to 5 mM N omega-nitro-L-arginine methyl ester (L-NAME; a nonspecific NO synthase inhibitor), and this protective effect of L-NAME was reversed with NO inhalation. Inhaled NO prevented the increase in PAP caused by I/R, while L-arginine had no such effect. L-NAME tended to diminish the I/R-induced elevation in PAP, but the suppression was not statistically significant when compared to the values in the I/R group. These results indicate that I/R increases Kfc and promotes alveolar edema by stimulating endogenous NO synthesis. Exogenous NO, either generated from L-arginine or delivered into the airway, is apparently also injurious to the lung following I/R.