

題名:Nitric Oxide Mediates Acute Lung Injury Caused by Fat Embolism
in Isolated Rat's Lungs

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摘要:BACKGROUND: The involvement of nitric oxide (NO) in acute lung injury (ALI) induced by fat embolism (FE) has not been investigated. The present study elucidated the role of NO in ALI because of FE. METHODS: FE was produced by introduction of fatty acid (corn oil micelles) into the isolated rat's lungs. Nonselective NO synthase (NOS) and selective inducible NOS (iNOS) inhibitors, N-nitro-L-arginine methyl ester (L-NAME) and L-N(1-iminoethyl)-lysine (L-Nil) as well as NO donors, sodium nitroprusside (SNP), and S-nitroso-N-acetylpenicillamine (SNAP) at a dose of 10 mol/L were given 60 minutes before FE. There were six groups of isolated lungs randomly assigned to receive vehicle (physiologic saline solution), FE, FE with pretreatment of L-NAME, L-Nil, SNP, or SNAP. Each group was observed for 4 hours. RESULTS: FE significantly increased the lung weight changes, pulmonary arterial pressure, and microvascular permeability. The concentration of nitrate or nitrite, methyl guanidine, tumor necrosis factor- α , and interleukin-1 β was significantly elevated after FE. Histopathologic examination revealed lung edema with multiple fatty droplets in lung tissue. Pretreatment with L-NAME or L-Nil attenuated, whereas SNP or SNAP exacerbated most of the FE-induced changes. Addition of NO donors (SNP or SNAP) into the isolated lungs did not produce significant changes in the lungs, suggesting that NO donation alone without FE does not exerts harmful effect. CONCLUSIONS: Our results suggest that NO production through the iNOS isoform plays a detrimental role in the FE-induced ALI. Free radical and

proinflammatory cytokines may also be involved in the pathogenesis of ALI because of FE.