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Title: Involvement of oxidative and nitrosative stress in the development of proteolytic pulmonary emphysema

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Body: Our aim was to investigate the participation of oxidative stress in elastase-induced pulmonary emphysema. C57BL/6 mice were submitted to pancreatic porcine elastase (PPE) instillation (0.05U or 0.5U) per mouse (i.t.) to induce pulmonary emphysema. A separated group of mice were treated with aminoguanidine 1% (AMG). Lungs were collected on days 7, 14 and 21 after PPE instillation. Control group was sham-injected. We performed BAL, biochemical analyses of oxidative stress, and lung stereology and morphometry. Emphysema was histologically characterized at 21 days after 0.5 U of PPE, presenting increased alveolar linear intercept and volume density of airspaces in comparison with the control group. TNF-α was elevated at 7 and 14 days after PPE 0.5 U, concomitant with reduction in the IL-10 levels at the same time-points. Myeloperoxidase was elevated in all groups treated with 0.5 U of PPE. A contribution of oxidative stress at early stage of emphysema was observed with increased levels of nitrite, malondialdehyde and superoxide dismutase activity at 7 days after PPE 0.5 U. Glutathione peroxidase activity was increased in all groups treated with 0.5 U of PPE. With iNOS inhibition by AMG 1%, emphysema was attenuated. Furthermore, the proteolytic stimulus by PPE enhanced expression of nitrotyrosine and iNOS, while the group PPE+AMG showed low expression of iNOS and nitrotyrosine. PPE stimulus also induced eNOS expression, but AMG reduced it. Our results suggest a pathway of oxidative and nitrosative stress by nitric oxide production via iNOS expression in pulmonary emphysema.