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Title: Pulmonary function, oxidative stress and inflammatory markers in LPS-induced acute lung injury: Differential effects of atorvastatin, pravastatin and simvastatin

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Body: The present study was designed to determine what statin could attenuate acute lung injury (ALI) induced by lipopolysaccharide (LPS) in C57BL/6 mice. Young male mice (± 23 g) were divided into 5 groups (n=6 each): injected with LPS i.p. (10 mg/kg), LPS plus atorvastatin (10 mg/kg/day; LPS+A group) or pravastatin (5 mg/kg/day; LPS+P group) or simvastatin (20 mg/kg/day; LPS+S group). Control group received saline (i.p.). In a separated group of mice (n=5) the sum of pulmonary resistive and viscoelastic pressures (DeltaPtot) and static elastance (E(st)) were measured. One day later (24 h), the animals were sacrificed, BAL performed and lungs were removed for histopathological analysis and homogenized for biochemical analyses. The amount of leukocytes was lower in LPS+P ($p<0.01$) and LPS+S ($p<0.05$). Cytokine levels of MCP-1 was lower in LPS+P ($p<0.01$) while IL-6 was lower in LPS+P ($p<0.01$) and LPS+S ($p <0.05$). Redox markers (superoxide dismutase and catalase) were lower in LPS+A ($p<0.01$). Lipid peroxidation (malondialdehyde and hydroperoxides) were lower in all treated groups ($p<0.05$). Myeloperoxidase was lower in LPS+P ($p<0.01$). DeltaPtot and E(st) were significantly higher in the LPS group than in the other groups. Our results suggest that atorvastatin and pravastatin, but not simvastatin, exhibits anti-inflammatory and antioxidant actions in LPS-induced ALI.