題名:Cyclosporine; an Immunosuppressant ; attenuates phorbol-induced lung injury in rats.

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上傳時間:2009-08-24T03:31:53Z

摘要:White cell activation in the lung plays a critical role to induce lung injury and lymphocytes in the thoracic duct system may also participate. We evaluated the effect of cyclosporine on phorbol myristate acetate (PMA)-induced lung injury. MATERIALS AND METHODS: We used an in situ isolated, blood perfused rat lung model to measure pulmonary arterial pressure (PAP) and lung weight gain (LWG; g) for 50 minutes after a bolus injection of PMA (0.05 microg/mL). Oxygen radical release was estimated by an LKB 1251 luminometer and by nitric oxide (NO) release as measured by an ENO-20 NO analyzer. RESULTS: In the group exposed to PMA alone, the mean PAP increased from 16.53 +/- 1.28 to 43.33 +/- 3.40 mm Hg (P < .001), and lung weight increased by 4.35 +/- 0.67 g during the 50-minute perfusion after PMA challenge (P < .001). In vitro measurement showed that PMA induced a significant increase in oxygen radical release (P < .001). PMA attenuated NO release (P < .001) into the perfusion system. Pretreatment with cyclosporine (3 mg/kg) for 3 days prevented the increases in both PAP (P < .01) and LWG (P < .001). NO release was maintained in cyclosporine-pretreated rats. Cyclosporine also showed dose-dependent attenuation of oxygen radical release by PMA-activated white blood cells. CONCLUSION: The mechanisms responsible for the protective effect of cyclosporine on the lung injury induced by phorbol may be related to an attenuation of oxygen radical production with maintenance of NO release.