題名:Transforming growth factor upegulation is independent of angiotensin in paraquat induced lung fibrosis

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摘要:Transforming growth factor-beta1 (TGF-beta1) contributes to the fibrosis of injured organs. Angiotensin II (Ang II) is an inducer of TGF-beta1 in cells of the heart and kidneys, and the regulation of TGF-beta1 by Ang II has not yet been confirmed in lung tissue. We evaluated the role of TGF-beta1 and its relationship with Ang II in paraquat-induced lung fibrosis. Adult male Sprague-Dawley rats were treated intraperitoneally with paraquat (20mg/kg) or saline in the control group. On days 1, 3, 7, and 21 after paraquat treatment, TGF-beta1 and collagen gene expressions, TGF-beta1 protein, angiotensin-converting enzyme (ACE) activity, Ang II, and hydroxyproline contents were measured in lung tissue. Lung TGF-beta1 mRNA expression progressively increased and reached a peak on day 7 after paraquat treatment. Increases in TGF-beta1 mRNA expression and TGF-beta1 levels preceded the onset of increased collagen I mRNA expression and hydroxyproline contents. c-myc mRNA expressions were inversely correlated with TGF-beta1 protein levels in paraquat-treated lungs. Lung ACE activity decreased after paraquat administration and the decrement was maximal on day 7. Lung Ang II concentrations immediately decreased after paraquat administration and the values were not related to TGF-beta1 levels. We conclude that TGF-beta1 is upregulated and contribute to the paraquat-induced lung fibrosis and this effect is independent of the renin-angiotensin system.