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Title :	Effect of γAtorvastatin on Paraquat-induced pulmonary fibrosis: Evidence for PPAR
Authors :	Hassan Malekinejad, Masoud Mehrabi, Mona Khoramjouy, Araz Ostadi, Sima Ahsan
Address :	Department of Pharmacology & Toxicology, Faculty of Veterinary Medicine, Urmia University, Urmia, Iran.
Abstract :	Introduction: The similarity of Praquat (PQ)-induced fibrosis to the pulmonary fibrosis in human exposed to herbicides has been reported. The receptors in theγpresent study carried out to discover the role of PPAR PQ-induced fibrosis in the lungs. Materials & Methods: Forty two male Wistar rats (180-200 g) were exposed either against saline as control group or PQ (3.5 mg/kg) as test groups for 14 consecutive days. The animals in test groups received saline (PQ), pioglitazone (PGT, 10 mg/kg), Atorvastatin (STN, 10mg/kg), PGT+ STN, PGT+ GW9662 (0.6 mg/kg) and STN+ GW9662 (0.6 mg.kg). The rate of lipid peroxidation and total thiol molecules in the lungs were determined. 1 at mRNA level was examined using RT-PCR technique.βThe expression of TGF- Results: Atorvastatin and PGT were able to recover the PQ-reduced level of total thiol and the GW9662 administration resulted in antagonizing the protective effect of both compounds. Although both PGT and STN were able to reduce the malondialdehyde content of the lungs, GW9662 however could reverse only STN effect. Co-administration of PGT and STN could not exert a synergistic effect on the PQ-induced biochemical changes. STN and not PGT could significantly (P< 1 up-regulation and GW9662 only wasβ0.05) regulate the PQ-induced TGF- able to antagonize the STN effect. Conclusions: Our findings suggest that the PQ-induced biochemical and molecular alterations could be more effectively reversed by STN rather than PGT. Moreover, STN-induced protective effects on absolute antagonistγfibrotic biomarkers have been antagonized by PPAR receptors.γsuggesting that STN improves the PQ-induced fibrosis via PPAR
Keywords :	, Pulmonary Fibrosisγ1, PPAR βAtorvastatin, TGF-