Abstract 3

Protective effect of dexamethasone against paraquat-triggered toxicity in A549 cells through inhibiting inflammation, apoptosis and TGF-β1/Smad3 pathway

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Dexamethasone is a glucocorticoid that is used for the treatment of interstitial pneumonia and pulmonary fibrosis as it possesses anti-inflammatory and anti-fibrosis properties. In the current study, A549 cells were exposed to paraquat, dexamethasone, or both of them, to investigate the potential effect of dexamethasone against paraquat triggered poisoning in A549 cells. The inflammatory response was evaluated by measuring tumor necrosis factor-α, interleukin-1β, and interleukin-6 while the degree of fibrosis was assessed by detecting collagen I and fibronectin using enzyme-linked immunosorbent assay. Western blotting was used to assess the protein expression of apoptotic proteins as well as transforming growth factor-β1, Smad 3 and phospho-Smad 3. DNA ladder assay was performed to estimate DNA damage in different groups of the alveolar epithelial cells. Dexamethasone protected against paraquat-induced inflammatory response as shown by the significantly reduced levels of the pro-inflammatory cytokines and it also alleviated paraquat-provoked fibrosis as it substantially diminished collagen I and fibronectin levels. Moreover, dexamethasone remarkably decreased the relative expression levels of transforming growth factor-β1 and phospho-Smad3 that were upregulated upon PQ treatment. Dexamethasone also protected against paraquat-induced genotoxicity and apoptosis. In conclusion, dexamethasone may protect against paraquat-induced inflammation, fibrosis, genotoxicity, and apoptosis via modulating TGF-β1/Smad 3 signaling pathway.

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