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The promising role of cannabinoid receptors 2 activation by AM1241on induced lung fibrosis in rats

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Activation of cannabinoid receptor type 2 has been shown to have anti-fibrosis action in some organs such as in liver, skin and heart. However, whether activating cannabinoid receptor type 2 could inhibit pulmonary fibrosis still obscure. The key markers in the pathogenesis of lung fibrosis are, pulmonary fibroblasts and TGF- β 1. The aim of the current work was to investigate the stimulating role of cannabinoid receptor 2 agonist AM1241 in *in vivo* model of pulmonary fibrosis. Lung fibrosis was induced in rat model using bleomycin (0.05 IU) instilled intratracheally once in rats, then rats were treated either with vehicle 0.1 ml of 1:2 DEMSO: PBS or AM1241 (3mg/kg DMSO then

diluted in PBS by ratio 1:2) once daily for 14 days. The use of AM1241 markedly decreased the pulmonary fibrosis by reducing the level of the profibrotic cytokines TGF- $\beta1$ as well as reducing the levels of collagen I and α -SMA. The oxidative stress marker thiobarbituric acid reactive substance showed marked decrease. The histopathological examination of lung revealed that the use of AM1241 could suppress collage deposition and inflammatory reaction as well as decreased fibrosis scoring. Our results indicated that activating cannabinoid receptor type 2 could be a novel significant strategy for curing pulmonary fibrosis.

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