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Dihydroartemisinin Alleviates Oxidative Stress in Bleomycin-induced Pulmonary Fibrosis

Dong-xia Yang[a, #], Jun Qiu [b, #], Hui-Hui Zhou[c, #], Yan Yu[a], Dong-li Zhou[d], Yan Xu[e], Ming-zhe Zhu[e], Xing-ping Ge[e], Jing-Min Li[a], Chang-jun Lv[a], Hong-Qin Zhang[a, *], Wen-dan Yuan[a, *]

[a] *College of Basic Medicine, Binzhou Medical University, Yantai, Shandong 264003, China.*

[b] *Department of Blood Purification Center, The Affiliated Yantai Yuhuangding Hospital of Qingdao Unviersity,Yantai, Shandong 264000,China.*

[c] *Department of pathology Center, The Affiliated Yantai Yuhuangding Hospital of Qingdao Unviersity,Yantai, Shandong 264000,China.*

[d] *Shandong Laiyang health school, Laiyang, Shandong 265200,China.*

[e] *Pediatric Nephro Department, The Affiliated Hospital of Binzhou Medical University, Yantai, Shandong 264000, China.*

ABSTRACT

Aims：Dihydroartemisinin has been shown to inhibit the development of pulmonary fibrosis in rats, but its mechanism has yet to be elucidated. This study aimed to determine the mechanisms of dihydroartemisinin in bleomycin-induced pulmonary fibrosis in a rat model.

Main metheds：Morphological changes and collagen deposition were analyzed via hematoxylin-eosin staining and Masson staining and the expression of biotic-factor-related oxidative stress in lung tissues was assayed with standard assay kits. The expressions of α-SMA, E-cadherin, and Nrf2/HO-1 were detected by western blot and RT-PCR, and the cell morphology and proliferation of cultured type II alveolar epithelial cells (AECs) were assessed via microscopy and immunocytochemical assay.

Key findings：Dihydroartemisinin treatment significantly decreased the level of oxidative stress and collagen synthesis and inhibited AECs differentiation in bleomycin-induced pulmonary fibrosis compared to the control group (P<0.001).

Significance：Our results indicated that dihydroartemisinin might decrease oxidative damage to attenuate lung injury and fibrosis.

Keywords: Dihydroartemisinin; Oxidative Stress; Bleomycin; Pulmonary Fibrosis.

1. Introduction

Idiopathic pulmonary fibrosis (IPF) is a chronic lung disease with a median survival of less than three to five years from the time of diagnosis[1]. The etiology of pulmonary fibrosis is complex. Its formation is affected by virus, fungus, environmental pollution and toxic substances, but the pathological changes are basically the same, and they all show the presence of interstitial fibrosis and the infiltration of inflammatory cells in the alveolar cavity.

[#] These authors contributed equally to this work. [*] Correspondence should be addressed to Wendan Yuan or Hong-Qin Zhang（email: 981713509@qq.com）.