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Benefits of the active agents from Yupingfeng for pulmonary fibrosis

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Pulmonary fibrosis (PF) is a chronic inflammatory, debilitating and often lethal lung disease with unclear aetiology. The imbalance of extracellular matrix (ECM) secretion and metabolism in the lung tissue is considered to be a key reason for the loss of lung function and respiratory failure [1,2]. Till now, limited effective therapies have been developed to reverse PF or halt it to lung failure.

Yupingfeng, a classical complex prescription of traditional Chinese medicines (TCM), composes of Astragali Radix, Atractylodis Macrocephalae Rhizoma and Saposhnikoviae Radix in a dry weight ratio of 3:1:1. It has been early reported to improve respiratory tract diseases such as viral infections and chronic bronchitis [3,4]. Impressively, recent studies have shown that total glucosides of *Yupingfeng* (YPF-G) could effectively attenuate bleomycin-induced alveolitis and PF with decreased levels of ECM protein including laminin, hyaluronic acid and type I collagen, as well as reduced the over-expression of TGF-β1 and high-mobility group box 1[5,6]. Moreover, Cui *et al.* also demonstrated that YPF-G reversed the process of epithelial–mesenchymal transition, which is critical origin of ECM-producing cells [6]. Meanwhile, total polysaccharides of *Yupingfeng* was reported to attenuate TGF-β1 mediated synthesis of Col-I on bleomycin-induced rats [7].

These data suggest the benefits of the active agents from *Yupingfeng* for PF. However, further efforts are still needed to clearly illuminate the mechanisms and effective components of *Yupingfeng* extracts on PF before the potential application in clinical. Moreover, other compositions other than glucosides and polysaccharides may also

contribute to the anti-fibrotic role. Meaningfully, it would provide new perspectives for PF therapy if further investigations were conducted to clarify whether the effects of *Yupingfeng* extracts are superior to the single composition (such as astragaloside IV) or *Yupingfeng San*.

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