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Lung alert

Lysophosphatidic acid is an important mediator of fibroblast recruitment in IPF

Lysophosphatidic acid (LPA) and its G protein-coupled receptor (LPA1) play key pathogenic roles in idiopathic pulmonary fibrosis (IPF). Previous research has highlighted the importance of fibroblast chemoattractant activity in the lungs in patients with IPF. New findings suggest the LPA-LPA1 pathway has a pivotal role in mediating fibroblast migration and vascular leakage in IPF. The end result is the aberrant healing process that characterises this fibrotic condition.

Using an experimental bleomycin-induced lung injury mouse model, the investigators showed that LPA levels were high in bronchoalveolar lavage samples compared with unexposed controls. They also showed that LPA1 knockout mice were protected from fibrosis after bleomycin challenge with reduced fibroblast accumulation and vascular leakage.

Similar findings were reproduced in human subjects; nine patients with IPF had high LPA levels in bronchoalveolar lavage samples compared with seven healthy controls. Increased fibroblast chemotactic activity in these samples was inhibited by a specific LPA1 antagonist, Ki16425, suggesting that fibroblast migration is mediated by the LPA-LPA1 pathway.

These experimental and clinical findings suggest that the LPA-LPA1 pathway is crucial in fibroblast recruitment and vascular leakage in IPF. It provides a novel therapeutic target in this generally refractory disease. Only a small number of subjects were studied, so the relative importance of this pathway needs to be confirmed. As fibroblast proliferation persists in bleomycin-challenged LPA1 knockout mice, the evidence suggests that IPF is mediated by other pathogenic factors.

► Tager AM, LaCamera P, Shea BS, *et al*. The lysophosphatidic acid receptor LPA1 links pulmonary fibrosis to lung injury by mediating fibroblast recruitment and vascular leak. *Nat Med* 2008;**14**:45–54.

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