# Demystifying morphomolecular alterations of vasculature in interstitial lung diseases

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**Cite this article as:** Yanagihara T, Jones KD. Demystifying morphomolecular alterations of vasculature in interstitial lung diseases. *Eur Respir J* 2020; 55: 1902446 [https://doi.org/10.1183/13993003.02446-2019].



Interstitial lung diseases (ILDs) encompass a complex group of hundreds of lung disorders that affect lung tissue with variable morphologies and clinical presentations. The most extensively studied type of ILD is idiopathic pulmonary fibrosis (IPF), which is characterised by progressive pulmonary fibrosis, a decline in lung function, and high mortality with a histological pattern of usual interstitial pneumonia (UIP). A proportion of patients with other types of ILD also develop a progressive fibrosing phenotype, including idiopathic nonspecific interstitial pneumonia (NSIP), as well as restrictive allograft syndrome (RAS) and idiopathic pleuroparenchymal fibroelastosis (iPPFE) with a histological pattern of alveolar fibroelastosis (AFE). RAS is a novel form of chronic lung allograft dysfunction first described in 2011 [1]. iPPFE was newly designated as a rare entity of idiopathic interstitial pneumonia in 2013 [2]. However, the pathogenesis of both RAS and iPPFE remains largely unknown [1, 3, 4].