

Adaptive Immunity in Interstitial Lung Diseases and Pulmonary Hypertension

Peter Heukels, Rotterdam, 23.11.2021

1. Only by integrating biological information with patient-reported and patient-collected information, will we be able to realize truly personalized treatment. (This thesis)
2. The increased frequency of IgA⁺ memory B cells, the presence of IgA autoantibodies and increased expression of Bruton's tyrosine kinase all support a role of B cells in the pathogenesis of idiopathic pulmonary fibrosis. (This thesis)
3. Targeting B cell receptor signaling in idiopathic pulmonary fibrosis may contribute to the therapeutic effect of nintedanib. (This thesis)
4. Patients with idiopathic pulmonary arterial hypertension show increased B cell receptor signaling and increased circulating follicular T helper-17 polarization, indicating that adaptive immune activation may contribute to vascular remodeling. (This thesis)
5. Increased levels of serum C-X-C motif chemokine ligands 13 and 9 distinguish patients with sarcoidosis-associated pulmonary hypertension from patients with idiopathic pulmonary hypertension or patients with sarcoidosis. (This thesis)
6. We no longer consider pulmonary fibrosis to be idiopathic as advances in our understanding of disease pathogenesis of idiopathic pulmonary fibrosis progresses. (Richeldi R, Collard H, et al, Lancet 2017)
7. Clinical drug trials in idiopathic pulmonary fibrosis may benefit from oncologists, following their vast experience in designing trials of new treatments. (Vancheri C, European Respiratory Review 2013)
8. More data are needed on novel agents with immunomodulatory properties, as current PAH therapies are essentially focused on decreasing pulmonary vascular resistance rather than vascular remodeling. (Huertas A, Perros F, Circulation 2014)
9. An important goal in sarcoidosis research is the identification of disease-triggering antigens, but for now improving disease management with more personalized treatments will offer benefit to patients. (Grunewald J, Grutters J, Nature Reviews 2019)
10. Understanding the mechanisms underlying COVID-19-mediated lung fibrosis may help developing new strategies for treating patients with Long-COVID as well as patients with pulmonary fibrosis due to other causes. (Alison J, Chitra J, Immunological Reviews 2021)
11. Science is a wonderful thing if one does not have to earn one's living at it. (Einstein A, Letter to E. Holzapfel 1951)