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23281 | Decoding host-environment interactions in progressive pulmonary fibrosis: insights from hypersensitivity pneumonitis

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Background & Aim: Interstitial lung diseases (ILDs) comprise a heterogeneous group of parenchymal lung disorders characterized by diffuse infiltration of immune effector cells, fibroblasts, myofibroblasts, and extracellular matrix deposition at various pulmonary compartments. These conditions can progress to end-stage fibrosis, respiratory failure, and eventually, death. In 2020, we initiated the first national ILD registry and biobank under the FIBRALUNG project, which has enrolled over 950 cases to date, with over 40% of the cases being fibrotic ILDs. The most represented fibrotic ILD groups are Hypersensitivity Pneumonitis (HP) (46%), Idiopathic Pulmonary Fibrosis (IPF) (20%), and unclassifiable ILD (8%). HP is the leading cause of pulmonary fibrosis, nearly doubling the number of IPF cases, which contrasts with numbers from other countries. This underscores the importance of investigating non-IPF progressive pulmonary fibrosis within our setting. **Methods:** Longitudinal patient follow-up and biological sample collection were performed allowing patient stratification according to progression criteria. **Results:** Our work so far has highlighted a potential role of CCL2-CCR2 axis in fibrotic HP disease progression. Elevated serum CCL2 strongly associated with disease progression and acute exacerbations, with baseline levels above 1080 pg/mL predicting one-year progression/mortality. To complement these findings, we are conducting blood transcriptome analyses across different HP patient groups to identify progression-specific signatures. Simultaneously, lung microbiome profiling is underway to explore its role in fibrotic progression. **Conclusions:** These integrative approaches aim to uncover novel biomarkers and mechanistic pathways, paving the way for tailored therapeutic interventions.

Keywords: Pulmonary fibroses, hypersensitivity pneumonitis, biomarkers, CCL2.