Eva M Carmona Porquera et al., J Clin Cell Immunol 2018, Volume 9
DOI: 10.4172/2155-9899-C3-054

Activation of B-cells, *via* pattern recognition receptors, contributes to the pro-inflammatory and pro-fibrotic milieu in patients with pulmonary fibrosis

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B-cell activation is increasingly linked to numerous fibrotic lung diseases and it is well known that they form aggregates in the lung of many of these patients. Activation of B-cells by pattern recognition receptors (PRRs) drives the release of inflammatory cytokines, chemokines and metalloproteases important in the pathophysiology of pulmonary fibrosis. The specific mechanisms of B-cell activation, in patients with idiopathic pulmonary fibrosis (IPF), a deadly disease with very little therapeutic options, are poorly understood. Herein, we have demonstrated that B-cell activation of PRRs by microbial antigens such as - glucan and CpG contribute to the pool of the inflammatory and pro-fibrotic milieu seen in IPF patients. Stimulation of the PRRs, TLR9 and Dectin-1, resulted in activation of mTOR-dependent and independent pathways, respectively. While rapamycin, a well-known mTOR inhibitor, was able to decrease TLR9 mediated signaling, it had no effect on Dectin-1 mediated pathways. The use of the anti-fibrotic agent, nintedanib abrogated B-cell activation and decreased the pro-fibrotic milieu by interfering with mTOR- dependent and -independent pathways.

Biography

Eva M Carmona Porquera is a Physician-Scientist focused on understanding the role of infection and chronic inflammation in the development of interstitial lung diseases (ILD). Her research to date has focused on understanding the immunomodulatory effects of fungal beta-glucans (BG) in innate immune cells. Particularly, she had characterized the role of BG on dendritic cells and their participation in T-cell polarization (Th1 and Th17).

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