17th International Conference on
European Immunology

April 17-18, 2023

Rome, Italy

J Clin Cell Immunol 2023, Volume 14

Contribution of elastin peptides to the regulation of innate lymphoid cells during chronic obstructive pulmonary disease

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Chronic Obstructive Pulmonary Disease (COPD) is characterized by chronic bronchitis associated with emphysema that generates Elastin-derived Peptides (EP) impacting many immune populations. Innate Lymphoid Cells (ILC) plays a role in many pathologies including COPD. However, the mechanisms regulating their functionalities, especially linked to EP, are not yet known. Moreover, while ILC are predominantly resident, recruitment from other sites is possible under inflammatory conditions and their origin in COPD remains to be elucidated. Our goal is to understand the mechanisms of ILC regulation and orientation induced by EP in COPD. Our results demonstrated that the proportion of ILC2 was increased and the proportion of ILC3 was decreased in the blood of COPD patients. In addition, we observed a significant increase in the expression of cytokines associated with the different types of ILC in COPD patients indicating an increase in activation. Moreover, ILC cultured with serum from COPD patients with emphysema or with supernatants of EP-stimulated macrophages showed an increase in the proportion of ILC2. In a mouse model of emphysema, we observed a decrease in the number of lung ILC2 concomitant with an increase in the mediastinal draining lymph nodes 7 days after instillation, suggesting a role of EP in the regulation of proliferation and/or migration of ILC. The human phenotypic and functional studies indicate an orientation of ILC towards an ILC2 profile in COPD patients as well as a general activation of ILC represented by an increase in their cytokine secretion capacity. Moreover, *in vitro* culture experiments suggest a role of EP, via their impact on macrophage, in the regulation of ILC during COPD. Finally, in vivo studies performed in mice suggest a role of EP on the migration and/or proliferation of ILC in the context of COPD.