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Immunoregulatory role of secretory leukocyte protease inhibitor in allergic asthma

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Background

Asthma is a complex and multi-factorial inflammatory disease [1]. It is one of the most common chronic diseases among children and adolescents [2]. Secretory leukocyte protease inhibitor (SLPI) has shown higher levels in asthmatic patients and its function as an anti-inflammatory protein has been documented in respiratory diseases [3,4]. However, its role in the immunomodulation of the response during allergic asthma has not yet been fully elucidated. The aim of this study was to evaluate the role of SLPI in the development of phenotypes associated with allergic asthma, and the effect of resiquimod treatment on the SLPI and the possible mechanisms of action involved in the disease.

Materials and methods

The importance of SLPI was assessed by evaluating airway resistance and inflammatory parameters in SLPI transgenic and knock-out mice using an ovalbumin (OVA)-induced model of acute allergic asthma and treatment with resiquimod.

Results

Allergic SLPI transgenic mice showed a significant decrease in airway resistance compared to wild-type mice (6.3 ± 1.1 vs. 8.0 ± 2.1 cm H_20 × s/ml, $p < 0.001$), the same effect was observed with inflammatory cell infiltration, eosinophil percentage (24 ± 1.1% vs. 29 ± 2.3%, $p < 0.001$), goblet cells (6 ± 1.4 vs. 36 ± 4.0%, $p < 0.001$) in the lungs and IgE levels (2014.1 ± 309.2 vs. 4173.2 ± 685.6 ng/ml, $p < 0.001$) in plasma. Allergic SLPI knock-out mice displayed significantly higher values compared to wild-type mice. They include lung resistance (8.6 ± 2.7 vs. 6.6 ± 0.5 cm H_20*s/ml, $p < 0.001$), inflammatory cell influx, eosinophils (36.0 ± 2.7 vs. 29.0 ± 1.5%, $p < 0.001$), goblet cells (40 ± 4.1 vs. 30 ± 1.4%, $p < 0.001$), cytokine levels in the lungs ($p < 0.05$) and plasma IgE levels (3598 ± 204.7 vs. 2763 ± 220.3 ng/ml, $p < 0.001$). Expression of SLPI decreased inflammation in the lungs, plasma IgE levels, and lung resistance, whereas the ablation of SLPI has the opposite effect. Treatment with resiquimod improved airway resistance and inflammation of the lungs in SLPI knock-out and wild type, demonstrating that its effect is independent of the expression of SLPI.

Conclusions

SLPI plays an immunoregulatory role in the respiratory tract by reducing the inflammatory process and by improving lung physiology in a murine model of acute allergic asthma.

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