Staphylococcus aureus and its IgE-inducing enterotoxins in asthma: current knowledge

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Late-onset non-atopic, often severe asthma is not well understood. There is increasing evidence that bacteria and their proteins, specifically S. aureus and its superantigens and serine protease-like proteins may represent the triggers we are looking for. http://bit.ly/386oP4G


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ABSTRACT While immunoglobulin (Ig) E is a prominent biomarker for early-onset, its levels are often elevated in non-allergic late-onset asthma. However, the pattern of IgE expression in the latter is mostly polyclonal, with specific IgE low or below detection level albeit with an increased total IgE. In late-onset severe asthma patients, specific IgE to Staphylococcal enterotoxins (Se-IgE) can frequently be detected in serum, and has been associated with asthma, with severe asthma defined by hospitalisations, oral steroid use and decrease in lung function. Recently, Se-IgE was demonstrated to even predict the development into severe asthma with exacerbations over the next decade. Staphylococcus aureus manipulates the airway mucosal immunology at various levels via its proteins, including superantigens, serine-protease-like proteins (Spl), or protein A (SpA) and possibly others. Release of IL-33 from respiratory epithelium and activation of innate lymphoid cells (ILCs) via its receptor ST2, type 2 cytokine release from those ILCs and T helper (Th) 2 cells, mast cell degranulation, massive local B-cell activation and IgE formation, and finally eosinophil attraction with consequent release of extracellular traps, adding to the epithelial damage and contributing to disease persistence via formation of Charcot–Leyden crystals are the most prominent hallmarks of the manipulation of the mucosal immunity by S. aureus. In summary, S. aureus claims a prominent role in the orchestration of severe airway inflammation and in current and future disease severity. In this review, we discuss current knowledge in this field and outline the needs for future research to fully understand the impact of S. aureus and its proteins on asthma.

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