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Validation and shortcomings of the most common mouse model of chronic rhinosinusitis with nasal polyps

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BACKGROUND: Chronic rhinosinusitis (CRS) is a highly prevalent airway disease worldwide. Whereas eosinophilic CRS with nasal polyps (eCRSwNP) represents its most severe phenotype, pathogenic mechanisms remain poorly understood despite a wide spectrum of in vitro and in vivo experimental models. A mouse model of experimental ovalbumin (OVA)-induced airway allergy with coadministration of Staphylococcus aureus enterotoxin B (SEB) has been widely used to study eosinophilic eCRSwNP. This study revisits the features of this model and its suitability for studying eCRS.

METHODOLOGY: We implemented the most used eCRSwNP mouse model based on OVA+SEB intranasal challenges. Readouts including inflammatory features by (immuno)histology of the sinonasal epithelium (NP formation, eosinophils, epithelial and basement membrane thickness, fibrosis, goblet cells, Charcot-Leyden crystals (CLC)-like, tight junctions) and IgE production by enzyme-linked immunosorbent assay (ELISA), were compared to features of the corresponding human disease.

RESULTS: The OVA+SEB model induced eosinophilic inflammation of upper and lower airways, with epithelial and basement membrane thickening, goblet cell hyperplasia and subepithelial fibrosis in the sinuses, along increased IgE production. Except local IgE in nasal lavage (NL), which was only increased in OVA+SEB group, all other features did not differ between OVA and OVA+SEB groups. Macro- or microscopic NP were not detected.

CONCLUSIONS: With the notable exception of local IgE production, the addition of SEB did not induce additional inflammatory or structural change in the sinuses from mice exposed to and challenged with OVA. This model might represent a model for severe upper airway allergy rather than a specific model of human eCRSwNP.

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