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Murine eosinophilic and neutrophilic chronic rhinosinusitis models reveal phenotype-specific steroid responses

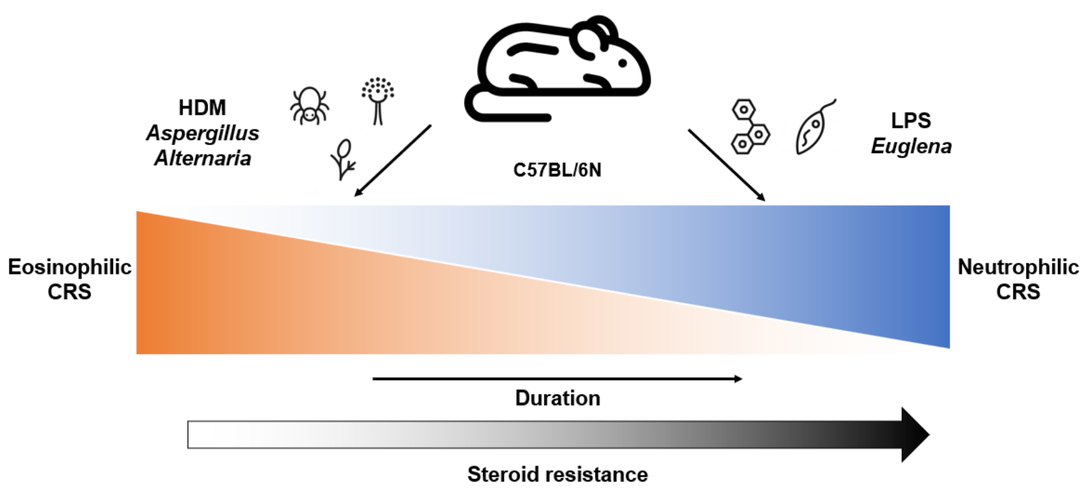
Dachan Kim[1], Miran Kang[1], Hyung-Ju Cho[1,2,3]
[1]Department of Otorhinolaryngology, Yonsei University College of Medicine, Seoul, Korea
[2]The Airway Mucus Institute, Yonsei University College of Medicine, Seoul, Korea
[3]Department of Otorhinolaryngology, Graduate School of Medical Science, Brain Korea 21 Project, Yonsei University College of Medicine, Seoul, Korea

Background

Chronic rhinosinusitis (CRS) comprises heterogeneous eosinophilic (type-2) and neutrophilic (type-1/3) endotypes, yet existing murine models rarely distinguish them or compare their therapeutic responsiveness.

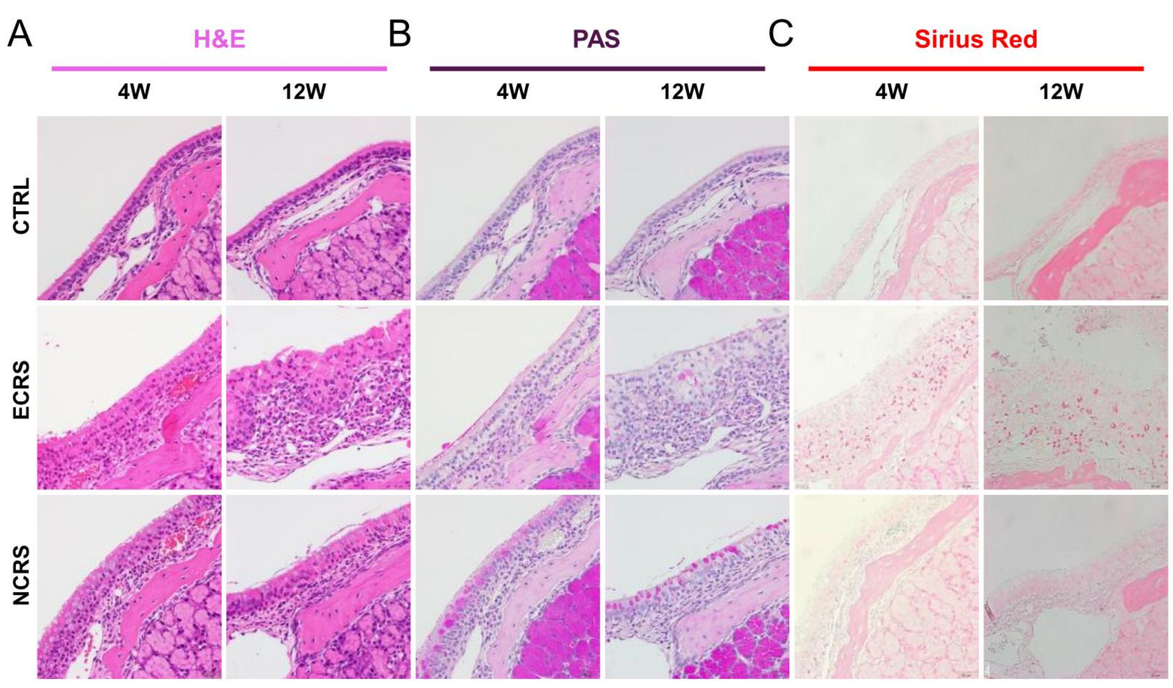
Materials and Methods

Seven-week-old female C57BL/6N mice received intranasal instillations three times weekly for 4 or 12 weeks with either (i) a clinically relevant airborne allergen cocktail (house dust mite [HDM], Aspergillus fumigatus, Alternaria alternata, and Staphylococcus aureus protease) to induce eosinophilic CRS (ECRS) or (ii) an innate stimulus mixture (lipopolysaccharide, β-1,3-glucan, and S. aureus protease) to induce neutrophilic CRS (NCRS). A parallel cohort received weekly dexamethasone administration (2 mg/kg). Endpoints included flow cytometry, enzyme-linked immunosorbent assays, quantitative PCR, histology, and immunofluorescence assays.



CRS, chronic rhinosinusitis; ECRS, eosinophilic CRS; NCRS, neutrophilic CRS; HDM, house dust mite; LPS, lipopolysaccharide

Results



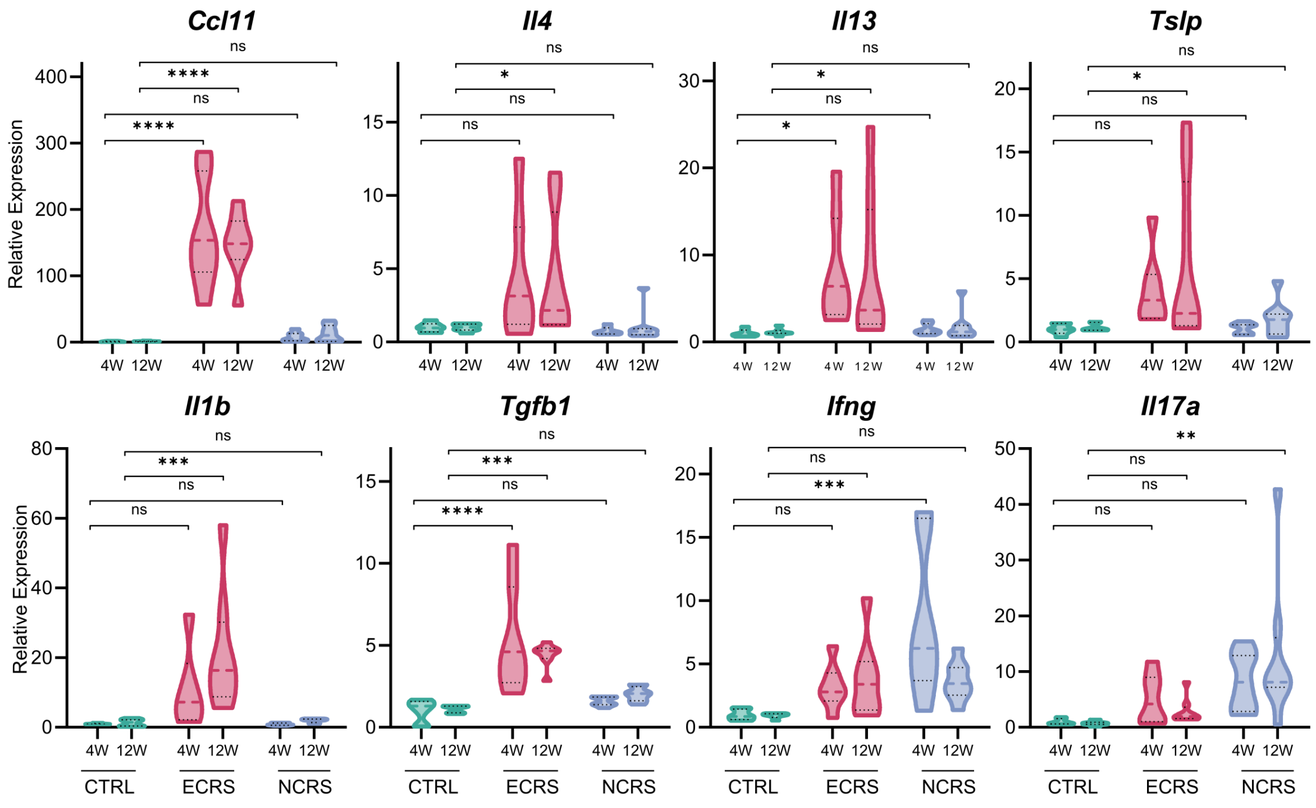


Figure 2. Nasal cytokine gene expression distinguishes Th2- and Th1/Th17-skewed endotypes.

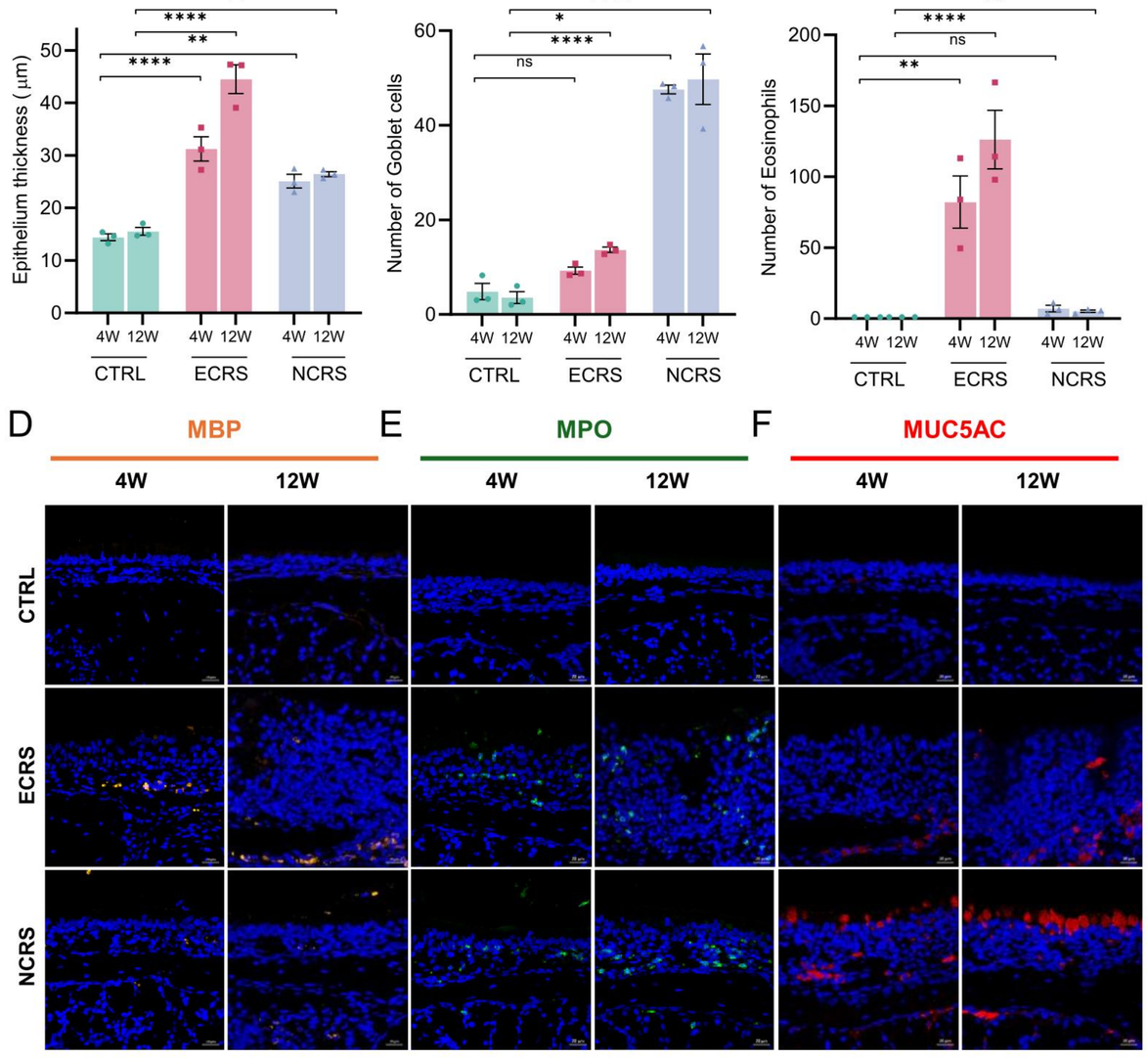


Figure 1. Histological and immunofluorescent assessment of the nasal mucosa.

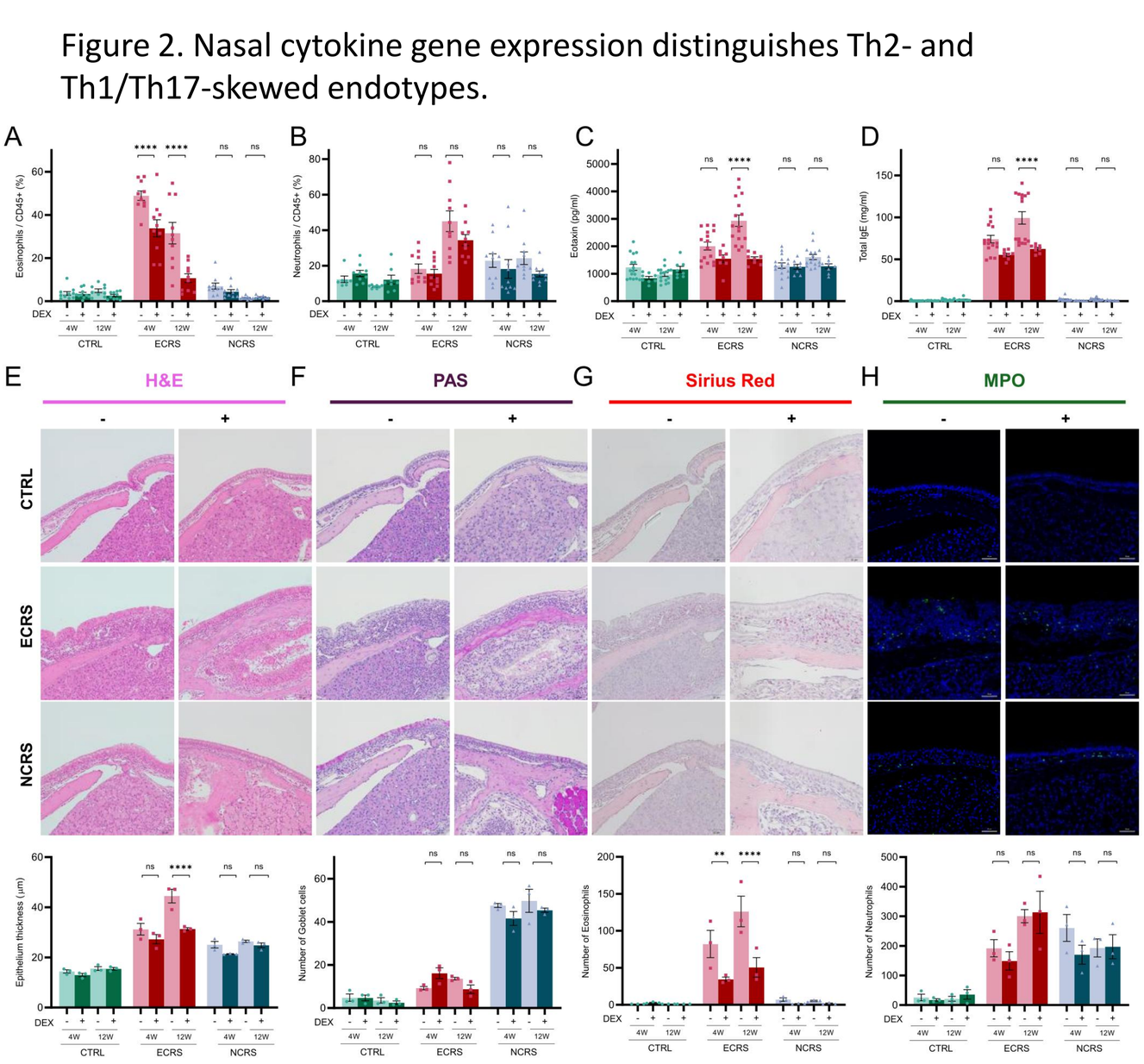


Figure 3. Dexamethasone selectively suppresses eosinophil-predominant inflammation.

Conclusion

By adjusting the stimulus type and duration, we established tunable murine models that recapitulate pure ECRS, mixed CRS, and steroid-resistant NCRS within a single genetic background. These paired models provide a versatile platform for dissecting endotype-specific mechanisms and evaluating tailored interventions, highlighting the potential importance of early, phenotype-directed CRS therapy.