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CORRIGENDUM

Precision Medicine for Paediatric Severe Asthma: Current Status and Future Direction [Corrigendum]

Ramphul M, Lo DKH, Gaillard EA. *J Asthma Allergy*. 2021;14:525–538.

The authors have advised that Figures 1 and 2 on pages 528 and 533, respectively are in the reverse order. In the original article, Figure 1 appears as Figure 2, and Figure 2 as Figure 1. The Figures with the correct captions are as follows.

The authors apologize for this error.

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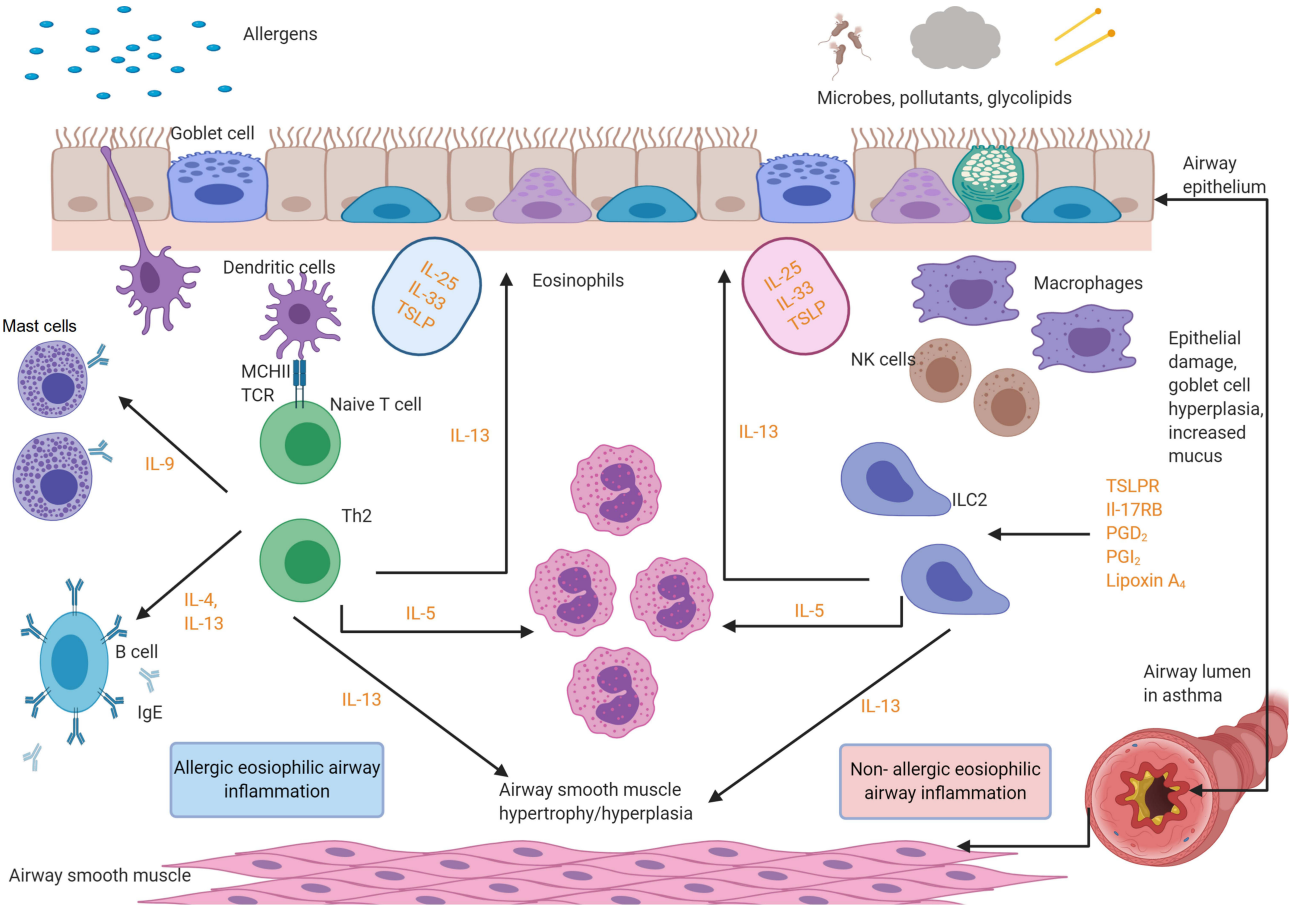

Figure 1 Eosinophilic asthma inflammatory pathways. There are two aetiologies for eosinophilic inflammation in asthma: an allergic pathway triggered by allergens and a non-allergic mechanism triggered by microbes, pollutants and glycolipids. The key mediators in the pathways are depicted below. Eosinophils release cationic proteins,[57] which lead to bronchial epithelial tissue damage, thus causing airways hyper-responsiveness. Eosinophils also lead to airway smooth muscle cell proliferation through increased eosinophils adhesion caused by the release of cationic proteins and the eosinophilic effect on transforming growth factor-β1 and gene coding of wingless/integrase-1 signaling. IL-13 triggers mucus hyper-secretion. Figure created with BioRender.com.

Abbreviations: IgE, immunoglobulins E; IL, interleukins; ILC2, type 2 innate lymphoid cells; TCR, T-cell receptors; NK, natural killer T cell; TSLPR, thymic stromal lymphopoietin receptor; PG, prostaglandin; ECP, eosinophil cationic protein; EPX, eosinophil protein X; EPO, eosinophil peroxidase; MBP, major basic protein.

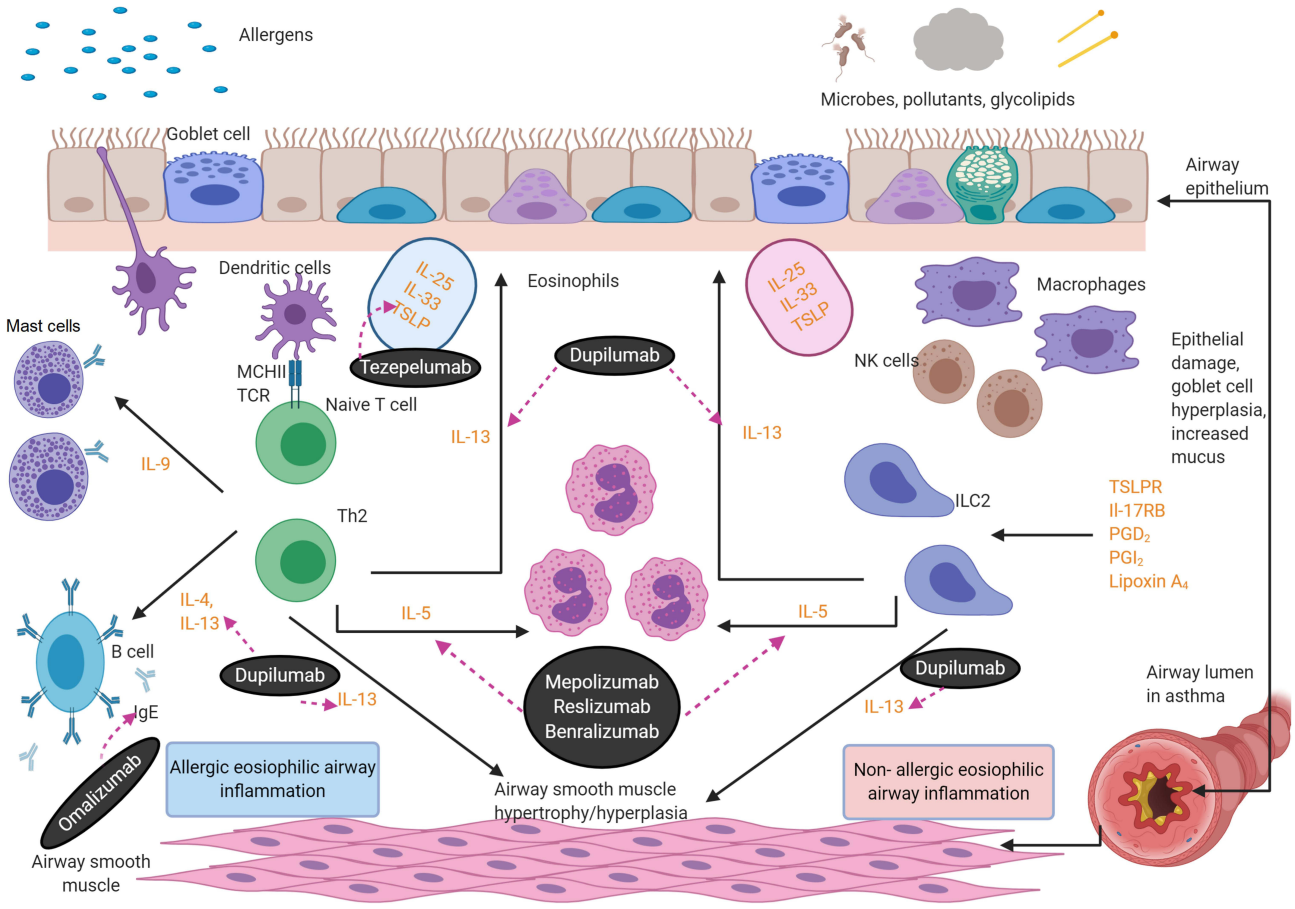

Figure 2 Licensed mediator cascade of biologics used in eosinophilic asthma. The figure shows the targets for the licensed biologics. Omalizumab is an IgE-blocker. Mepolizumab, reslizumab and benralizumab are anti IL-5 agents. Dupilumab is an Anti-IL-4/anti-IL-13 agent. Figure created with BioRender.com.

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