

Allergic rhinitis, asthma, and atopic dermatitis

ALLERGIC DISEASES

are among the fastest growing chronic conditions in Australia¹



Around
1 in 7
Australians have an
ALLERGIC DISEASE²



PREVALENCE IN AUSTRALIANS:

- Allergic rhinitis affects nearly 1 in 4²
- Just under 1.6 million suffer from atopic dermatitis³
- Around 11% have asthma⁴



It is predicted that by 2050 the prevalence of
ALLERGIC DISEASES
in Australia will increase by
70%⁵



NUTRITIONAL INHIBITION OF THE ALLERGIC RESPONSE⁶⁻³⁰

SENSITISATION

An allergen is taken up by antigen-presenting cells, e.g. dendritic cells, and presented to naive T cells which then differentiate into Th2 cells. This results in the generation of an allergen-specific humoral response that is predominated by the production of IgE antibodies.

These IgE antibodies attach to mast cells and basophils thereby sensitising them to subsequent exposure. This process of binding of IgE to the receptors is hallmark of sensitisation.

EARLY PHASE RESPONSE: TYPE 1 ALLERGIC REACTION

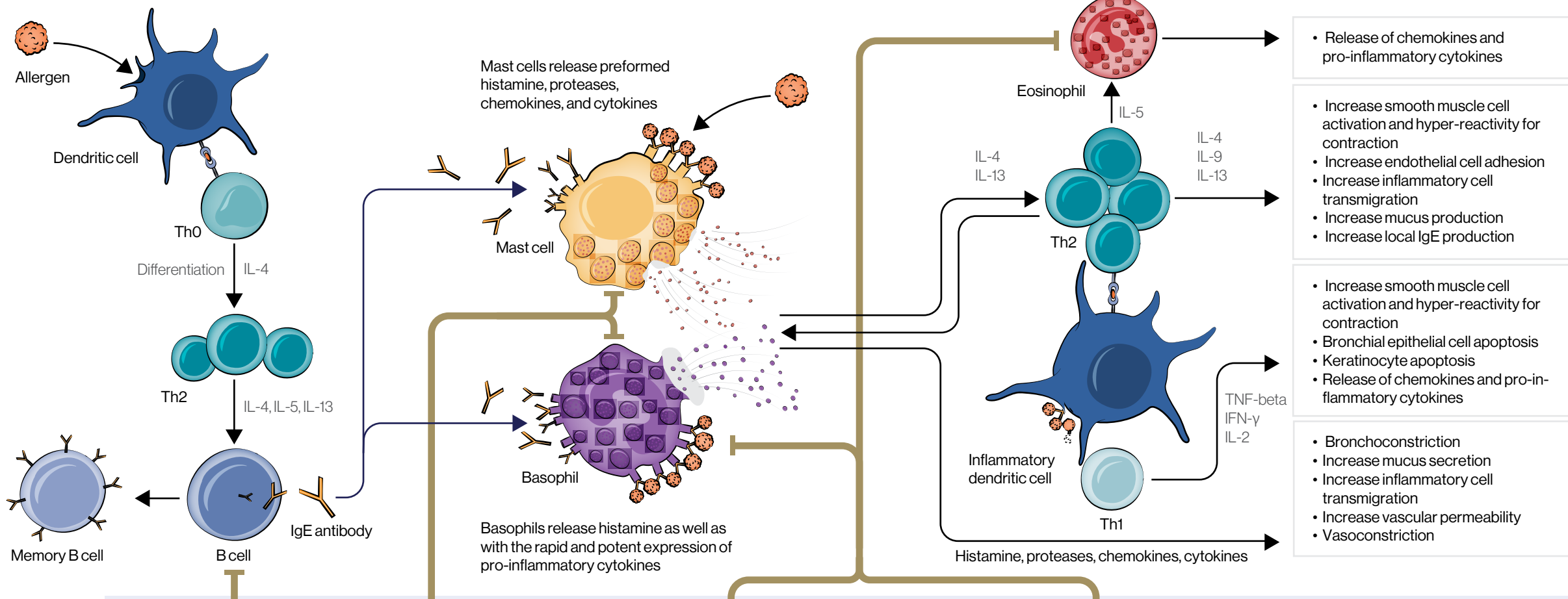
On subsequent re-exposure to the allergen, the allergic response is triggered: the allergen cross-links with the IgE on the surfaces of the mast cells or basophils, causing the cell to rapidly degranulate and release inflammatory mediators.

Much of the early phase allergic reaction can be attributed to the direct effects of histamine on surrounding tissues, e.g. swelling, itching, sneezing in allergic rhinitis, airway constriction in asthma, or pruritis in atopic dermatitis.

LATE PHASE RESPONSE: ALLERGIC INFLAMMATION

Chemokines released by mast cells and basophils promote the recruitment of inflammatory cells that contribute to the late allergic response, which is characterised by an influx of eosinophils and Th2 cells. There is now evidence that Th1-cell responses might also be responsible for some of the pathogenic features of allergy, and the model of unbalanced Th1/Th2 may be an oversimplification of the process.

Late phase responses are characterised by: an oedematous, red and slightly indurated swelling in the skin; sustained blockage in the airways; and further wheezing in the airways.



QUERCETIN and RUTIN reduce IgE antibody release by B cells	QUERCETIN, VITAMIN C, HESPERIDIN stabilise mast cells and inhibit histamine release	QUERCETIN, RUTIN, VITAMIN C inhibits the recruitment and activation of eosinophils and neutrophils	VITAMIN C and VITAMIN B6 cofactors of the DAO enzyme for histamine metabolism and breakdown	QUERCETIN, VITAMIN C, and HESPERIDIN regulate Th1/Th2 levels QUERCETIN, VITAMIN C, HESPERIDIN, RUTIN, and BROMELAINS modulate cytokine/chemokine production and release	PROTEASES decrease inflammatory cytokines and T cell response QUERCETIN increases thioredoxin levels
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