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The epithelial gatekeeper against food allergy.

Yu LC.

Graduate Institute of Physiology, National Taiwan University College of Medicine, Taipei, Taiwan. lchyu@ntu.edu.tw

Abstract

The rapid rise of allergic disorders in developed countries has been attributed to the hygiene hypothesis, implicating that increased environmental sanitation in early childhood may be associated with higher incidence of hypersensitivity. Intestinal epithelial barriers play a crucial role in the maintenance of gut homeostasis by limiting penetration of luminal bacteria and dietary allergens, yet allowing antigen sampling via the follicle-associated epithelium for generation of tolerance. However, this intricate balance is upset in allergic intestines, whereby luminal proteins with antigenic properties gain access to the subepithelial compartment and stimulate mast cell degranulation. Recent studies demonstrated that food allergens were protected from lysosomal degradation, and were transported in large quantities across the epithelium by binding to cell surface IgE/CD23 (FcepsilonRII) that prevented the antigenic protein from lysosomal degradation in enterocytes. IL-4 (a Th2-type cytokine) not only increased production of IgE from B cells, but also upregulated the expression of CD23 on intestinal epithelial cells. Further studies indicated that CD23 was responsible for the bidirectional transport of IgE across epithelium. The presence of IgE/CD23 opens a gate for intact dietary allergens to transcytose across the epithelial cells, and thus foments the mast cell-dependent anaphylactic responses. The understanding of the molecular mechanism responsible for epithelial barrier defects may be helpful in designing novel therapies to treat food allergy and other allergic diseases.

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