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Anti-acid medication as a risk factor for food allergy.

Pali-Schöll I, Jensen-Jarolim E.

IPA, Center of Pathophysiology, Infectiology and Immunology, Medical University Vienna, Vienna, Austria.

Abstract

An important feature for oral allergens is their digestion-resistance during gastrointestinal transit. For some oral allergens, digestion stability is an innate feature, whereas digestion-labile antigens may only persist in times of impairment of the digestive system. In this review, we collect evidence from mouse and human studies that besides the inherent molecular characteristics of a food protein, the stomach function is decisive for the allergenic potential. Gastric acid levels determine the activation of gastric pepsin and also the release of pancreatic enzymes. When anti-ulcer drugs inhibit or neutralize gastric acid, they allow persistence of intact food allergens and protein-bound oral drugs with enhanced capacity to sensitize and elicit allergic reactions via the oral route. Mouse studies further suggest that maternal food allergy arising from co-application of a food protein with anti-acid drugs results in a Th2-biased immune response in the offspring. Especially, anti-ulcer drugs containing aluminum compounds act as Th2 adjuvants. Proton pump inhibitors act on proton secretion but also on expression of the morphogen Sonic hedgehog, which has been related to the development of atrophic gastritis. On the other hand, atrophic gastritis and resulting hypoacidity have previously been correlated with enhanced sensitization risk to food allergens in elderly patients. In summary, impairment of gastric function is a documented risk factor for sensitization against oral proteins and drugs.

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