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Modulation of mucosal immunity in a murine model of food-induced intestinal inflammation.

[Cardoso CR](#), [Teixeira G](#), [Provinciatto PR](#), [Godoi DF](#), [Ferreira BR](#), [Milanezi CM](#), [Ferraz DB](#), [Rossi MA](#), [Cunha FQ](#), [Silva JS](#).

Department of Biochemistry and Immunology, School of Medicine of Ribeirão Preto, University of São Paulo, Ribeirão Preto, SP, Brazil.

BACKGROUND: Hypersensitivity or uncontrolled responses against dietary antigens can lead to inflammatory disorders like food allergy and current models reflect a variety of causes but do not reveal the detailed modulation of gut immunity in response to food antigens after breakdown in mucosal tolerance. **OBJECTIVE:** To develop and characterize a murine model for food-induced intestinal inflammation and to demonstrate the modulation of gut immune response by dietary allergenic antigens. **METHODS:** C57BL/6 mice were sensitized with peanut proteins, challenged with peanut seeds and their sera and gut segments were collected for subsequent analyses. **RESULTS:** Sensitization and challenged with peanut seeds led to alterations in gut architecture with inflammatory response characterized by oedema in lamina propria and cell infiltrate composed mainly by eosinophils, mast cells, phagocytes, natural killer and plasma cells, together with low percentage of $\gamma\delta$ and $CD4^+CD25^+Foxp3^+$ cells in Peyer's patches. These animals also presented high levels of specific IgE and IgG1 in sera and modulation of mucosal immunity was mediated by increased expression of GATA-3, IL-4, IL-13 and TNF- α in contrast to low IFN- γ in the gut. **CONCLUSION:** A murine model for food-induced intestinal inflammation was characterized in which modulation of gut immunity occurs by peanut antigens in consequence of T-helper type 2 (Th2) allergic response and failure of regulatory mechanisms necessary for mucosa homeostasis, resembling food allergy. This work shed some light on the understanding of the pathogenesis of gastrointestinal disorders and intolerance in the gut and supports the development of therapies for food-related enteropathies like food allergy, focusing on gut-specific immune response.