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The potential role of advanced glycation end products in food allergy pathogenesis

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Abstract

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BACKGROUND: Food allergy(FA) prevalence has dramatically increased in the last two decades. Among dietary factors, it has been hypothesized that advanced glycation endproducts(AGEs), present at high level in junk food, could be involved in FA pathogenesis. AGEs are a heterogeneous group of compounds deriving from sugars(sweets and beverages), autoclaved/processed foods, microwaved foods, more roasted/barbecued meat. To evaluate the AGEs levels in FA children compared with healthy controls and subjects with respiratory allergy.
Methods: We evaluated paediatric patients with challenge-proven FA, children with respiratory allergy(RA) and age and sex-matched healthy controls. Subcutaneous AGEs levels were evaluated through the AGE reader. Food-frequency questionnaires were evaluated in all study subjects.
In vitro studies were performed on human enterocytes(Caco-2 cells) stimulated with 200 mg/ml of BSA-AGE for 24and48 hours to evaluate effects on gut barrier function: mucin2(mucus production), transpithelial electrical resistance(TEER), ZO-1, occludin expression(intestinal permeability). The direct effects elicited on peripheral blood mononuclear cells (PBMCs) after the treatment with 200 mg/ml of BSA-AGE for 48hours, 4and 7days of treatment were also evaluated.
RESULTS: 115 subjects were evaluated and subdivided into 3 groups: group 1 patients with FA (n=31); group 2 patients with RA (n=18), group 3 healthy controls (n=66). The consumption of food containing AGEs was higher in subjects with FA compared to RA children and healthy controls ($p<0.05$). FA and RA children presented significant higher subcutaneous AGEs levels compared to healthy controls ($p<0.05$). Linear regression analysis confirmed a significant positive correlation between subcutaneous levels of AGEs and consumption of food containing AGEs. Human enterocytes exposed to BSA-AGE treatment showed a reduction of TEER, of Muc2 and tight junction proteins (Occludin and ZO-1). Moreover, the treatment with BSA-AGE on human PBMCs stimulates pro-inflammatory cytokines TNF-α and Th2 cytokines(IL-5 and IL-13)production , but it was unable to modulate IL-10 production. Finally, after7days of treatment with BSA-AGE, we found a low percentage of proliferating CD4+T.
CONCLUSIONS: Current hypotheses and models of FA do not adequately explain the dramatic increase observed in the last years.

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