Correspondence

Potential role of advanced glycation end products in the TH2 adjuvant effect of peanut protein

To the Editor:

Ruiter et al showed that peanut protein induces a TH2 response by eliciting retinaldehyde dehydrogenase production in myeloid antigen-presenting cells. Noting that such an effect can also be produced by pathogen-associated molecular patterns, Ruiter et al excluded the possibility that the effect was produced by endotoxin or aflatoxin. However, they did not exclude the possible role of advanced glycation end products (AGEs).

AGEs are created during the browning of foods, including during the roasting of peanuts, and they contribute to peanuts’ allergenicity. AGEs activate the same receptor (receptor for advanced glycation end products [RAGE]) as does the damage-associated molecular pattern HMGB1. Although some of the experiments reported by Ruiter et al used raw peanut extract, key studies showing gene expression and enzyme production used an extract made from roasted peanuts.

In addition to their deleterious effects in conditions ranging from asthma, atherosclerosis, and diabetes to aging and Parkinson disease, AGEs have been hypothesized to be a root cause of food allergy.

An argument against the role of AGEs here is that they work via the stimulation of Toll-like receptor (TLR) 4, whereas in the study by Ruiter et al signaling of the antigen-presenting cells occurred largely via TLR1 and TLR2. However, Ruiter et al found that blocking TLR1/TLR2 reduced enzyme expression by 70%. Was the other 30% due to AGEs?

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Disclosure of potential conflict of interest: J. D. Miller is the owner and President of Mission: Allergy, Inc.

REFERENCES


https://doi.org/10.1016/j.jaci.2021.08.033