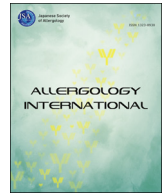




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Editorial

Food allergy: Current perspectives



In *Allergy International* (AI) Vol. 65 Issue 4, we offer a set of review articles entitled “Food allergy: Current perspectives” as well as original articles and letters to the editor. This issue will provide you with a comprehensive review of the latest concepts in food allergy.

Food allergy is currently a hot topic in the field of allergology. The number of publications dealing with food allergies reached 100 per year in 1971, based on a Pub Med search, then 500 per year in the beginning of 21st century, and more than 1200 per year in 2014. As Hugh Sampson comments in his review article,¹ no one would have imagined such a phenomenon in the 1980s, just 30 years ago. He sketches the intriguing historical background of food allergies over the past several thousand years. He further describes the recent past and the present state of food allergies, as a living witness. Finally, as a leading researcher in his field, he gives us his thoughts on the future of food allergies.

We invited George Du Toit, together with Gideon Lack, both leading researchers into the prevention of food allergy, to contribute to this issue. They have written a comprehensive review article entitled “Prevention of food allergy: Early dietary interventions”.² They have published several important original articles in consecutive issues of *the New England Journal of Medicine*, in 2015 and 2016.^{3–5} Although in the past, the prevention of food allergy focused on allergen avoidance, recent findings from interventional studies have prompted a shift in mind-set from avoidance to early introduction of potentially allergenic foods. In addition to their own work, they review many hypotheses for the possible prevention of food allergy.

Thirdly, Magnus Borres writes a comprehensive review of recent advances in component-resolved diagnosis in food allergy.⁶ Molecular allergology is a breakthrough science that enables the quantification of IgE antibodies against individual allergen protein components at the molecular level. Identifying whether the sensitization is primary (species-specific) or due to cross-reactivity to proteins with similar protein structures helps the clinician to judge the risk of allergic reaction.

Finally, Aaron K. Kobornick together with Wesley Burks co-author a review of active treatment for food allergy, including oral, sublingual, and epicutaneous immunotherapy for IgE-mediated food allergy.⁷ They describe for the readers of *Allergy International* the current consensus regarding efficacy and safety of those active treatments for food allergies.

Among the contributors to this issue, Kohno *et al.* report the characterization of a hypoallergenic wheat lacking ω -5 gliadin.⁸ Wheat-dependent exercise-induced anaphylaxis (WDEIA) is wheat allergy induced by exercise after the ingestion of wheat products.⁹ One solution for WDEIA patients is to limit their intake of wheat products, but that significantly lowers their quality of life. Since ω -5 gliadin is a major allergen in WDEIA,¹⁰ reduction or deficiency of ω -5 gliadin in wheat may decrease allergenicity. Kohno *et al.* searched for candidate wheat lines among the stocks in the National Bioresource Project (NBRP)-WHEAT organized by Japanese scientists and found one, 1BS-18, which has a short deficiency on chromosome 1B of Chinese Spring. It is of note that they cultivated this line for two years to get a sufficient amount of the grain to use in their research. Gluten from 1BS-18 showed low allergenicity in their *in vitro* analysis and hyposensitization ability in guinea pigs. This study shows the possibility that a wheat line with a deletion in the ω -5 gliadin gene can decrease the allergenicity of wheat, which may provide a feasible way to prevent the onset of wheat allergies, including WDEIA. This has the practical advantage that the 1BS-18 line is not a genetically modified crop, so it can be cultivated without any limitation.

Kanai *et al.* show the effects of prostaglandin (PG)D₂ on VEGF release in nasal polyp fibroblasts.¹¹ VEGF plays an important role in the pathogenesis of chronic rhinosinusitis with nasal polyps (CRSwNP) by inducing the proliferation of NP epithelial cells.⁵ VEGF is produced mainly by NP fibroblasts in response to various stimuli—hypoxia, TNF- α , LPS, and rhinovirus.^{12–14} PGE₂, one of the major PGs in NP, was recently shown to induce VEGF release in NP fibroblasts.¹⁵ Kanai *et al.* show in this article that in addition to PGE₂, PGD₂ can promote VEGF production in NP fibroblasts. It is known that PGD₂ has two receptors: the D-prostanoid (DP) receptor and a chemoattractant receptor-homologous molecule expressed on the Th2 cell (CRTH2) receptor. They show that PDG₂ induces VEGF production via the DP receptor, not via the CRTH2 receptor. Addition of a CRTH2 receptor-selective antagonist conversely enhanced PGD₂-induced VEGF release. This result provides a new insight into the involvement of lipid mediators in the pathogenesis of CRSwNP.

Fish allergy is particularly problematic in coastal countries such as Japan.¹⁶ It is of note that in Asian countries, fish allergy is more common than allergies to nuts, peanuts, or wheat. It is known that parvalbumin, a sarcoplasmic Ca²⁺-binding protein, is a major fish allergen and that anti-parvalbumin IgE is reported to be detected in all patients by some researchers and in 2/3 of patients by other.^{17,18} Type I collagen is another major fish allergen; 50% of Japanese patients with fish allergy show positive for anti-type I

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collagen IgE.¹⁹ Kobayashi *et al.* analyze in this article the thermostability of type I collagen in IgE reactivity.²⁰ Although collagen from Pacific mackerel was degraded by heating (140 °C × 10 min or 100 °C × 320 min), IgE reactivities detected in heated meat did not change during heating, but no reactivities were observed in unheated meat. IgE in pooled serum of the patients with fish allergy reacted with all of the investigated heated meat of bony fish but not of cartilaginous fish, whose collagens are classified as type I. This result suggests that heating is unlikely to reduce IgE reactivity to type I collagen found in fish.

We offer our appreciation to all the authors for their contributions to the present issue of *Allergology International*.

Conflict of interest

The authors have no conflict of interest to declare.

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References

1. Sampson HA. Food allergy: past, present and future. *Allergol Int* 2016;**65**:363–9.
2. Du Toit G, Foong RM, Lack G. Prevention of food allergy – early dietary interventions. *Allergol Int* 2016;**65**:370–7.
3. Du Toit G, Roberts G, Sayre PH, Bahnson HT, Radulovic S, Santos AF, et al. Randomized trial of peanut consumption in infants at risk for peanut allergy. *N Engl J Med* 2015;**372**:803–13.
4. Du Toit G, Sayre PH, Roberts G, Sever ML, Lawson K, Bahnson HT, et al. Effect of avoidance on peanut allergy after early peanut consumption. *N Engl J Med* 2016;**374**:1435–43.
5. Perkin MR, Logan K, Tseng A, Raji B, Ayis S, Peacock J, et al. Randomized trial of introduction of allergenic foods in breast-fed infants. *N Engl J Med* 2016;**374**:1733–43.
6. Borres MP, Maruyama N, Sato S, Ebisawa M. Recent advances in component resolved diagnosis in food allergy. *Allergol Int* 2016;**65**:378–87.
7. Kobernick AK, Burks AW. Active treatment for food allergy. *Allergol Int* 2016;**65**:388–95.
8. Kohno K, Takahashi H, Endo TR, Matsuo H, Shiwa K, Morita E. Characterization of a hypoallergenic wheat line lacking ω-5 gliadin. *Allergol Int* 2016;**65**:400–5.
9. Morita E, Kunie K, Matsuo H. Food-dependent exercise-induced anaphylaxis. *J Dermatol Sci* 2007;**47**:109–17.
10. Lee HS, Myers A, Kim J. Vascular endothelial growth factor drives autocrine epithelial cell proliferation and survival in chronic rhinosinusitis with nasal polyps. *Am J Respir Crit Care Med* 2009;**180**:1056–67.
11. Kanai K, Okano M, Fujiwara T, Kariya S, Haruna T, Omichi R, et al. Effect of prostaglandin D2 on VEGF release by nasal polyp fibroblasts. *Allergol Int* 2016;**65**:414–9.
12. Matsuno S, Sun D, Ohori J, Nishimoto K, Fukuiwa T, Ushikai M, et al. Inhibition of vascular endothelial growth factor by macrolides in cultured fibroblasts from nasal polyps. *Laryngoscope* 2005;**115**:1953–6.
13. Wang JH, Kwon HJ, Jang YJ. Rhinovirus upregulates matrix metalloproteinase-2, matrix metalloproteinase-9, and vascular endothelial growth factor expression in nasal polyp fibroblasts. *Laryngoscope* 2009;**119**:1834–8.
14. Cho JS, Kang JH, Park IH, Lee HM. Steroids inhibit vascular endothelial growth factor expression via TLR4/Akt/NF-κB pathway in chronic rhinosinusitis with nasal polyp. *Exp Biol Med* 2014;**239**:913–21.
15. Perez-Novo CA, Watelet JB, Claeys C, Van Cauwenberge P, Bachert C. Prostaglandin, leukotriene, and lipoxin balance in chronic rhinosinusitis with and without nasal polyposis. *J Allergy Clin Immunol* 2005;**115**:1189–96.
16. Boye JL. Food allergies in developing and emerging economies: need for comprehensive data on prevalence rates. *Clin Transl Allergy* 2012;**2**:25.
17. Bugajska-Schretter A, Elfman L, Fuchs T, Kapiotis S, Rumpold H, Valenta R, et al. Parvalbumin, a cross-reactive fish allergen, contains IgE-binding epitopes sensitive to periodate treatment and Ca²⁺ depletion. *J Allergy Clin Immunol* 1998;**101**:67–74.
18. Hamada Y, Nagashima Y, Shiomi K, Shimojo N, Kohno Y, Shibata R, et al. Reactivity of IgE in fish-allergic patients to fish muscle collagen. *Allergol Int* 2003;**52**:139–47.
19. Kobayashi Y, Akiyama H, Hige J, Kubota H, Chikazawa S, Satoh T, et al. Fish collagen is an important panallergen in the Japanese population. *Allergy* 2016;**71**:720–3.
20. Kobayashi Y, Kuriyama T, Nakagawara R, Aihara M, Hamada-Sato N. Allergy to fish collagen: thermostability of collagen and IgE reactivity of patients' sera with extracts of 11 species of bony and cartilaginous fish. *Allergol Int* 2016;**65**:450–8.

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