Editorial

Preface to the Proceedings of the Workshop on Eosinophils in Allergy and Related Diseases 2014

We are pleased to announce that Allergology International (AI) is publishing the proceedings of the 28th Workshop on Eosinophils in Allergy and Related Diseases. This Workshop was first established by Prof. Sohei Makino of Dokkyo University in 1988 and has since been held annually under the strong leadership of Prof. Makino, Prof. Takeshi Fukuda—also of Dokkyo University—and Prof. Makoto Nagata of Saitama Medical University.

The 2014 Workshop was held in Tokyo on October 4, 2014. It was supervised by me (K. Matsumoto; National Research Institute for Child Health and Development) and comprised 28 oral presentations, including one special lecture and one luncheon lecture.

This Proceedings issue contains one Review Article representing the special lecture, 8 Original Articles and 5 Letters to the Editor. All of the papers were peer-reviewed and accepted through the AI review process.

In the Review Article, Prof. Kenji Izuhara and his colleagues described the history, current understanding and future prospects of periostin biology in relation to allergic inflammation and tissue fibrosis. I would like to note that serum periostin has two characteristics as a biomarker for bronchial asthma: it is both a surrogate biomarker of type 2 immune responses and a biomarker reflecting tissue remodeling and fibrosis.

In one Original Article, Nunomura et al. evaluated the role of liver X receptors (LXRs) in cytokine production by murine mast cells. They found that activation of LXRs by a synthetic LXR ligand, GW3965, attenuated antigen- or lipopolysaccharide (LPS)-induced production of pro-inflammatory cytokines, such as IL-1α and IL-1β, by the mast cells, and that LXRβ played an important role in that attenuation.

Uchimizu et al. evaluated the concentrations of specific granular proteins and the cytokine/chemokine profile in middle ear effusion samples from patients with eosinophilic otitis media (EOM) and patients with secretory otitis media (SOM). They found that the concentrations of both eosinophil and neutrophil granular proteins were significantly higher in EOM than in SOM, and that they correlated significantly with eosinophil-recruiting chemokine and with IL-6, respectively. These findings suggest that not only eosinophils but also neutrophils are involved in the pathogenesis of middle ear inflammation in EOM.

Ogawa et al. evaluated the role of IL-23 in two distinct antigen-induced airway inflammation models (intraperitoneal injection followed by intranasal exposure) in IL-23p19-deficient mice. They found that endogenous IL-23 production at the site of OVA sensitization facilitated type-2 immune responses, whereas house dust mite (HDM) induced IL-23 production and subsequent IL-17A synthesis in the airways, which in turn suppressed allergic inflammation.

Kobayashi et al. evaluated the effects of stimuli via P2 or P2Y2 receptors on adhesion to ICAM-1, superoxide generation and eosinophil-derived neurotoxin (EDN) release in eosinophils. They found that formoterol, a long-acting β2 agonist, suppressed adhesion to ICAM-1, production of IL-5 and LTD4, IP-10-induced eosinophil adhesion to ICAM-1, superoxide generation and EDN release in eosinophils.

Noguchi et al. evaluated the effects of a long-acting β2 agonist on eosinophil functions induced by mediators associated with acute exacerbation, such as cysteinyl leukotrienes (cysLTs) or IP-10. They found that formoterol, a long-acting β2 agonist, suppressed adhesion to ICAM-1, production of IL-5 and LTD4, IP-10-induced O2− generation and EDN release in eosinophils.

Shintani et al. evaluated the mechanisms of how corticosteroid enhances airway epithelial barrier integrity. Using small interfering RNAs (siRNAs) for signal transduction molecules, they found that the nuclear factor erythroid 2-related factor 2 (Nrf2)-aldehyde oxidase 1 (AOX1) pathway plays important roles in corticosteroid-enhanced airway epithelial barrier integrity, and suggested that this pathway may be a potential therapeutic target for bronchial asthma.

Kato et al. evaluated the cytokine profiles and eosinophil activation during virus-induced acute asthma exacerbations and found that serum eosinophil cationic protein (ECP) and IL-5 production correlated significantly with age, whereas serum IP-10 showed an inverse correlation with age. These results suggest that acute asthma exacerbations in young children are prone to be related with viral infection, whereas those in older children are more type 2 inflammation-related.

The 5 Letters to the Editor dealt with cutting-edge topics in both basic and clinical allergy. The results collectively covered a wide academic scope and will significantly enhance our insight into the
biological properties and/or clinical importance of eosinophils and other inflammatory cells such as mast cells and basophils, as well as many cytokines and chemokines.

We sincerely believe that the Workshop and this supplemental issue will attract significant interest from not only the members of Japanese Society of Allergology but also many other physicians and researchers around the world. For organizing the Workshop, we greatly appreciate Prof. Nagata for his excellent management, and Drs. Soma and Nakagome, and Ms. Aoyama, all of Saitama Medical University, for devoting much time and effort to the Workshop. We would also like to thank Ms. Toshiko Takeda of the Editorial Office of the Japanese Society of Allergology for her excellent handling of this supplemental issue.

Personally speaking, I am writing this Editorial while on a flight back to Tokyo from Chicago, where the 9th Biennial Symposium of the International Eosinophil Society (IES) was held on July 14–18, 2015 (the final program is available at http://www.eosinophil-society.org/symposia/2015-biennial-symposium-scientific-program). Presented at that symposium were 4 prenyary lectures, 11 special lectures, 15 cutting-edge lectures, 21 oral presentations and 82 poster abstracts dealing with various aspects of eosinophils and eosinophil-related diseases. More than 200 attendees from over 30 countries held extremely hot discussions that often exceeded the allotted presentation times!

I would like to introduce a few of the topics discussed at the symposium. From a historical point of view, eosinophils were first thought to play a favorable role in allergic diseases by degrading chemical mediators,1 but the situation was completely reversed in the early 1980s by the finding that eosinophil-specific granule proteins directly damage the bronchial epithelium.2 Even today, the number of eosinophils in the sputum is the best clinical marker for optimizing treatment of patients with bronchial asthma.3 Administration of anti-IL-5 monoclonal antibody (mAb) to mild asthmatic patients strikingly reduced peripheral eosinophil counts, but it showed no effects on the lung function or late-phase reactions.4 Those findings dampened eosinophil research. Honestly speaking, eosinophil researchers, including me, were considered to be dream-seekers in those days, even though several well-designed studies revealed the critical roles of eosinophils in the pathogenesis of airway remodeling and other pathological conditions.5,6

Recent studies yet again up-ended the general understanding of the roles of eosinophils. Administration of anti-IL-5 mAB to severe eosinophil-predominant steroid-refractory asthmatic patients showed about 50% reduction of asthma exacerbation.7 A subsequent study using another clone of anti-IL-5 mAb affirmed that finding.8 Thus, I strongly believe that eosinophils are involved in exacerbation in certain types of asthma patients. Of course, further investigations will be essential to fully elucidate the roles of eosinophils in human diseases. Finally, I found that my personal impression of current eosinophil biology is perfectly summed up by a famous quote from Sir Winston Churchill, made on November 10, 1942: “Now this is not the end. It is not even the beginning of the end. But it is, perhaps, the end of the beginning”.

I ask that you all keep in mind that the 2015 Workshop will be held on October 24, 2015, in Tokyo under the supervision of Prof. Koichiro Asano of Tokai University. We hope for even greater numbers of presentations and attendees on that date. Please check the website for more details at http://ns1.sec-information.net/eosinophils/data/announce.html. I am looking forward to seeing you there.

Conflict of interest
The author has no conflict of interest to declare.

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