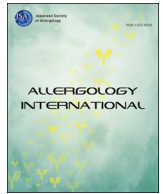




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Editorial

Japanese guidelines for allergic diseases 2017



In *Allergology International* (AI) Vol. 66, Issue 2, we offer a set of review articles covering the Japanese Guidelines for Allergic Diseases 2017, the updated English version based on JAGL2016, as well as original articles and letters to the editor. We believe that this issue will be of great help to both clinical and basic investigators working in this field.

The discovery of immunoglobulin E (IgE) by Kimishige and Teruko Ishizaka in 1966 opened a new era in the fields of allergy and immunology. Even before this historical discovery, allergic diseases had been assumed or hypothesized to share the same pathway to develop various symptoms specific for each allergic disease. In fact, IgE has been found to play a pivotal role in allergic diseases such as asthma both in adults and children, allergic rhinitis, atopic dermatitis, and food allergy. We allergists have noted that we often see patients having more than two allergic diseases at the same time or appearing with one allergic disease and then with others as time passes during childhood. Through the progress in the investigation of allergic diseases clinically as well as basically, therapeutic strategies based on the concept of evidence-based medicine have been published as guidelines for various allergic diseases. In Japan, Sohei Makino and selected members of Japanese Society of Allergology (JSA) published the first guidelines for allergic diseases in 1993. After the first issue, guidelines were drawn up independently for each allergic disease until 2007, when Sankei Nishima and the members of JSA's guideline committee wrote up separate guidelines for each allergic disease together and published them as JAGL. Since then, the committee has been trying to revise them every three years; this is the third revision following the one in 2013 (JAGL2013). The Guidelines have been building a solid reputation, dealing with most of the common allergic conditions. The Guidelines offer useful information to clinicians who see patients having more than two allergic diseases and require their diagnosis and treatment at the same time, without having to move from one specialist to another. This new version, called JAGL2016, was produced in the memorial year, i.e., the fiftieth anniversary of the discovery of IgE. It contains the newest information and recommendations for diagnosis and management of allergy based on the new guidelines for each allergic condition. As you will note, each condition shares the same basic pathways and pathogenesis, resulting in similar therapeutic approaches. This English version of JAGL2016 will add useful information to global guidelines for their updating, and will be able to enable people around the world to understand how we Japanese specialists treat allergic patients successfully.^{1–7} We also hope to receive comments on our diagnostic and therapeutic strategies as we disseminate our views

more widely. Our goal is to find treatments that can cure allergy so that no one is bothered by allergic diseases throughout life.

Innate lymphoid cells (ILCs) are recently identified innate immune cells lacking T cell antigen receptors. It has been reported that all ILCs—ILC1, ILC2, and ILC3—are correlated with asthma.^{8–10} However, how each population of ILCs in peripheral blood is correlated with asthma phenotypes still remains unclear. Among the contributors to this issue, Ishimori *et al.* demonstrate a pioneering approach to this question, using samples from 75 adults with mild to severe asthma.¹¹ The frequencies of total or activated (CD69-positive) ILCs are invariant among levels of asthma severity. However, activated ILC1s and ILC2s, but not ILC3s, are associated with impaired lung functions, which may suggest the involvement of these cells in airflow limitation in asthma. These results may shed light on the significance of ILCs in the pathogenesis of asthma and may help us to stratify asthma phenotypes.

IL-35, a newly identified cytokine, belongs to the IL-12 family.¹² IL-35 is a heterodimer composed of Epstein–Barr virus-induced gene 3 (EBI3) and the p35 subunit of IL-12. IL-35, mainly produced by Tregs, plays anti-inflammatory or immune-suppressive roles. It has been reported that IL-35 together with Tregs is downregulated in peripheral blood cells in asthma patients¹³ and that the deficiency of EBI3 causes enhanced airway inflammation in mice.¹⁴ Suzuki *et al.* investigate, in this issue, the effects of IL-35 on allergic rhinitis model mice.¹⁵ Intranasal administration of IL-35 into mice significantly inhibited upper-airway inflammation—sneezes, nasal rubbing movements, and type 2 inflammation—whereas it increased IL-10 production and the expansion of Treg. These results suggest the anti-inflammatory roles and the therapeutic potentials of IL-35 on allergic rhinitis.

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Conflict of interest

The authors have no conflict of interest to declare.

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