

## Editorial



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### \*Correspondence to

**Yasuhiro Gon**

Division of Respiratory Medicine, Department of Internal Medicine, Nihon University School of Medicine 30-1 Oyaguchi Kmicho, Itabashi-ku, Tokyo 173-8610, Japan.

Tel: +81-3-3972-8111

Fax: +81-3-3972-2893

Email: gon.yasuhiro@nihon-u.ac.jp

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### ORCID iDs

Yasuhiro Gon

<https://orcid.org/0000-0003-0514-3054>

# Preface of the Proceedings of the 34rd Workshop on Eosinophils in Allergy and Related Diseases 2020 (WEA 2020)

**Yasuhiro Gon** \*

Division of Respiratory Medicine, Department of Internal Medicine, Nihon University School of Medicine, Tokyo, Japan

The 34th Workshop on Eosinophils in Allergy and Related Diseases 2020 (WEA 2020) was held on November 3, 2020 as the second wave of coronavirus disease 2019 (COVID-19) was subsiding amidst the onslaught of COVID-19 that had plunged the world into chaos.

This workshop has been held annually for 34 years. It was launched in 1988 by Prof. Sohei Makino (Dokkyo University, Tochigi, Japan) as the chairperson, who was succeeded by Prof. Takeshi Fukuda (Dokkyo University), and it is presently held under the leadership of Prof. Makoto Nagata (Saitama Medical University, Saitama, Japan).

Although the workshop initially focused on the basic biology of eosinophils, recent publications have discussed various aspects of allergies and related diseases more broadly.

This is the second time since 2019 that the Editor-in-Chief of *Asian Pacific Allergy* (AP Allergy) has announced to publish the proceedings of WEA in AP Allergy [1]. This has been conducted by Prof. Yoon-Seok Chang (Seoul National University, Seoul, Korea) with the support of Prof. Ruby Pawankar of the Asia Pacific Association of Allergy, Asthma and Clinical Immunology (APAAACI) (Nippon Medical School). We would like to express our deepest gratitude to APAAACI for their cooperation, and we sincerely hope for the development of both parties.

For 34 years, the WEA has been continued without interruption. However, this year, due to COVID-19, we had to postpone the workshop to November (from June) and conduct it as a webinar.

WEA 2020 was held under the supervision of Prof. Yasuhiro Gon (Nihon University School of Medicine, Tokyo, Japan). There were 25 oral presentations, and 3 educational seminars conducted by researchers representing Japan in each field.

The proceedings of WEA2020 include a review article, original articles, and case reports. All of which have been peer-reviewed and accepted through the regular review process of AP Allergy. The review entitled 'Eosinophil-mediated inflammation in the absence of eosinophilia' was contributed by Prof. Ueki et al [2]. In the original articles, Kobayashi et al. [3] reported that mepolizumab decreased the levels of serum galectin-10 and eosinophil cationic protein in asthma. Miyauchi et al. [4] reported the effect of Japanese cedar pollen on the effector functions of eosinophils. Koyama et al [5]. reported the inhibitory effect of dexamethasone on murine Th9 cell-mediated nasal eosinophilic inflammation. Ito et al.

[6] reported that L-type amino acid transporter 1 inhibitors suppressed Th2 cell-mediated bronchial hyperresponsiveness independent of eosinophil accumulation, and Uchida et al. [7] reported the use of rush immunotherapy in patients with asthma using dust mite allergens. Furthermore, Kouzaki et al. [8]. reported that interleukin-35 elicited an anti-inflammatory response in the pathogenesis of Japanese cedar pollinosis.

Takeda et al. [9] reported a case of chronic eosinophilic pneumonia involving eosinophil extracellular traps. Furthermore, Sano et al. [10] reported a case of eosinophilic sialadenitis in a patient with severe eosinophilic asthma. Moreover, Nishie et al. [11] reported the case of an adult with food allergy and severe asthma who was successfully treated using omalizumab, and Masaki et al. [12] reported a case with eosinophilic annular erythema showing eosinophil cytolytic ETosis successfully treated with benralizumab.

We hope that this issue will be of significance to the readers of AP Allergy. We believe that WEA will continue to provide opportunities for exchanging research information between basic and clinical researchers and play an important role in further understanding the pathogenesis of allergic diseases.

WEA 2021 is scheduled to be conducted. We expect an increase in the number of presentations and participants, and especially welcome international delegates. For details, please check the website at <http://www.sec-information.net/eosinophils/data/announce.html>.

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