Hikari Koyama, MD,

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In the present study, we attempted to study the airway

in asthma. Statins may cause possible immune alteration that promotes allergic diseases such as asthma.

Conclusions: Statin drugs may worsen asthma control in patients with AERD compared to that in patients with ATA. FABP1 was observed in epithelial, eosinophils, macrophages, and the smooth-muscle cells of blood vessels in the polyps.

Conclusions: Our results indicate that alterations in 15 proteins, including FABP1, may be related to the development of AERD.

ASTHMA

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The Correlation of Cholesterol Lowering Statin Drugs and Worsening Asthma Control in Mild Persistent Asthmatics

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Background: To show that pharmacological agent statins use, adversely alter the immunomodulatory activities that promote the worsening of the clinical course of allergic diseases such as asthma.

Methods: Two groups of 20 asthmatics patients each were compared from baseline values. Twenty patients with extrinsic asthma (group A) were prescribed statins for their lowering of their cholesterol necessity, and 20 patients (group B) were controls who did not receive statins. Group A and group B were designed to compare FEV1, exacerbation asthma rates, beta agonists use, nocturnal awakenings, and daytime symptoms from baseline values.

Results: Statins treated asthmatic patients group A had significant worsening of FEV1 at 3 months, 6 months and 12 months, to almost no change in control asthmatic patients group B. Statins treatment patients group A were associated with more frequent use of rescue medication (albuterol inhaler), increased nocturnal awakenings, and increased daytime asthma symptoms, compared to group B.

Conclusions: Statin drugs may worsen asthma control in mild persistent asthmatics. Statins may cause possible immune alteration that promotes allergic diseases such as asthma.

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Studies on the Relationship between Airway Inflammatory Responses in Patients With Asthma or Not-yet Onset Asthma and Air Pollution

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Background: Substantial data have demonstrated that air pollution is associated with cardiopulmonary mortality and morbidity in the world. Among a variety of pollutants, particulate components, particularly PM2.5, are especially suggested to be harmful to our lung health. Diesel exhaust particles (DEPs) are the major component of PM2.5, and therefore the relationship between PM2.5 or PM10 and airway inflammatory responses of asthmatic and people of not-yet asthma onset is important to be investigated. Recent findings suggested that susceptibility to DEPs is dependent upon certain genetic variations of anti-oxidative stress enzymes such as GSTP1, which is largely regulated by a transcription factor Nrf2. By preliminary experiments, we found that exhaled breath condensates (EBC) are safely and repeatedly obtained from both disease and health persons, and that several biomarkers including growth factors, cytokines and oxidant stress markers could be measured.

Methods: In the present study, we attempted to study the airway inflammatory/fibrogenic responses from patients with asthma, and further, those from people who have suggestive, but not yet definite symptoms of asthma. Participants are asked to present exhaled breath condensates (EBC) by

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