第558回 難研セミナー
第131回 難治疾患共同研究拠点セミナー

下記により難研セミナーを開催しますので、多数御来聴下さい。

記

日　時：　平成29年３月３０日（木）17:00～18:00

場　所：　Ｍ＆Ｄタワー21階　セミナー室

演　者：Dr. Philipp Yu

(Yu Philipps-Universität Marburg, Germany)

演　題：IgE Regulation : Then and Now

要　旨：Allergy is a common disease in all industrialized countries. Its symptoms can range from mild hayfever to life-threatning anaphylactic shock. A deregulated immunoglobulin E (IgE) response together with a Th-2 biased T cell response is at the core of the allergic immune response. However, basic research has still to gain a better understanding of fundamental regulations of the IgE B cell response, before better immunotherapy is available for patients. Here, I will discuss advances in genetic murine models for IgE and its low affinity receptor on B cells (CD23). In particular we will discuss recent findings that IgE expression on B cells results not in memory IgE^+ B cell generation, but rather in plasma cell formation or early cell death by apoptosis. This mechanism has been detected by Japanese scientists and could be confirmed by a novel IgE knock in mouse model we generated. In addition we will present recent data on CD23 function, which suggest that IgE is indeed regulated by a mechanism that modulates B cell receptor signaling.

連絡先：免疫疾患・安達貴弘　（内線　4591　）

共催：生体情報薬理学

The 558th Medical Research Institute Seminar
The 131th Joint Usage/Research Program of Medical Research Institute Seminar

Date: 2017.03.30

Venue: MD Tower 21th floor seminar room

Lecturer: Dr. Philipp Yu (Philipps-Universität Marburg, Germany)

Title: IgE Regulation : Then and Now

Summary: Allergy is a common disease in all industrialized countries. Its symptoms can range from mild hayfever to life-threatning anaphylactic shock. A deregulated immunoglobulin E (IgE) response together with aTh-2 biased T cell response is at the core of the allergic immune response. However, basic research has still to gain a better understanding of fundamental regulations of the IgE B cell response, before better immunotherapy is available for patients. Here, I will discuss advances in genetic murine models for IgE and its low affinity receptor on B cells (CD23). In particular we will discuss recent findings that IgE expression on B cells results not in memory IgE^+ B cell generation, but rather in plasma cell formation or early cell death by apoptosis. This mechanism has been detected by Japanese scientists and could be confirmed by a novel IgE knock in mouse model we generated. In addition we will present recent data on CD23 function, which suggest that IgE is indeed regulated by a mechanism that modulates B cell receptor signaling. These experiments are extended by a collaboration with Dr. Takahiro Adachi, Tokyo Medical and Dental University, who will analyze Ca^{2+} mobilization in vivo. Taken together our data shed a new light on the various levels of IgE regulation.

Organizers: Department of Immunology, Takahiro Adachi (ext. 4591)

Co-organizer: Bio-informational Pharmacology