

LECTURE SERIES THE IMMUNE ALLIANCE

a German-Japanese Cooperation
in Immunology

セミナーシリーズ 免疫同盟

免疫学における日独協力

Dr. Motoko Y. Kimura, Chiba, Japan

CD69 biology and pathology

Dr. Vigo Heissmeyer, Munich, Germany

Post-transcriptional regulation of TCR repertoire diversity

Wednesday, June 19, 2024

9 am in Germany (GMT+2) / 4 pm in Japan (GMT+9), via zoom

Organizers:

Christina Zielinski (Leibniz-HKI, Jena, Germany)

Osamu Takeuchi (Kyoto University, Japan)



Register via

www.dgfi.org/immune-alliance



Speakers

Prof. Dr. Motoko Y. Kimura

Graduate School of Medicine, Chiba University, Japan

Motoko Y. Kimura received her Ph.D. from Chiba University Medical School in 2002. After working as an assistant professor for four years, she joined Dr. Alfred Singer's Laboratory at the National Institutes of Health (NIH) in the United States. After returning to Japan in 2014, she joined Dr. Toshi Nakayama's laboratory at Chiba University and then she started her own laboratory in 2021 at Chiba University. Her research interest is how T cells discriminate self and non-self to respond against nonself-antigens such as pathogens to mount appropriate immune response, and respond to self-components to maintain tissue and immune homeostasis.

Prof. Dr. Vigo Heissmeyer

Institute for Immunology, Biomedical Center, LMU Ludwig-Maximilians-Universität München, Germany

Vigo Heissmeyer received his PhD from the Free University of Berlin in 1999. He was a postdoctoral fellow first at the Max-Delbrück Center for Molecular Medicine, Berlin, Germany and then at Harvard Medical School, Boston, USA. He started his independent lab in 2005 at the Helmholtz Centrum in Munich. In 2012 he became tenured Professor at the Institute for Immunology of the Ludwig-Maximilians-University of Munich as well as head of the Research Unit Molecular Immune Regulation at the Helmholtz Centrum in Munich. His group studies post-transcriptional gene regulation in T cells. With a focus on RNA-binding proteins the lab investigates how these trans-acting factors prevent autoimmunity or control anti-tumor responses.