

# Program for Workshop

As for Oral presentation time, please refer to "Scientific Program" on Annual Meeting's Website.

## W1 T cell response

14:45 ~ 16:05 Room C

Chairpersons: Kazuko Shibuya, Ei Wakamatsu

For the host defense against foreign pathogens by adaptive immunity, T cells are required to be properly activated and differentiated into effector and memory cells. T cell activation is regulated by not only TCR but also various molecules such as co-stimulatory/ co-inhibitory molecules, cytokines, metabolites, nutrients and oxygen concentration. Also, some of these molecules have effects on differentiation of effector and memory T cells through the metabolic reprogramming. Therefore, comprehensive understanding of the mechanisms, by which various molecules are involved in T cell responses in vitro and in vivo, is important. In this session, we would like to discuss the regulation of T cell activation and functional differentiation by cell intrinsic and extrinsic mechanisms. We hope active participations and discussion to deepen understanding of T cell response.

1-C-W1-1-O/P

### ACC1 determines memory potential of individual CD4<sup>+</sup> T cells by regulating de novo fatty acid biosynthesis

Yusuke Endo<sup>1)</sup>, Toshio Kanno<sup>1)</sup>, Takahiro Nakajima<sup>1)</sup>, Toshinori Nakayama<sup>2)</sup>

Laboratory of Medical Omics Research, Kazusa DNA Research Institute, Kisarazu, Japan<sup>1)</sup>, Department of Immunology, Graduate School of Medicine, Chiba University, Chiba, Japan<sup>2)</sup>

1-C-W1-3-O/P

### Glutaminase 1 inhibition reduces glycolysis and ameliorates lupus-like disease in MRL/lpr mice and experimental autoimmune encephalomyelitis

Michihito Kono<sup>1,2)</sup>, Nobuya Yoshida<sup>2)</sup>, Kayaho Maeda<sup>2)</sup>, Abel Suárez-Fueyo<sup>2)</sup>, Vasileios C. Kyttaris<sup>2)</sup>, George C. Tsokos<sup>2)</sup>

Department of Rheumatology, Endocrinology and Nephrology, Faculty of Medicine, Hokkaido University, Sapporo, Japan<sup>1)</sup>, Department of Medicine, Beth Israel Deaconess Medical Center, Harvard Medical School, Boston, USA<sup>2)</sup>

1-C-W1-7-O/P

### The tumor suppressor menin inhibits CD8 T cell senescence by regulating autophagy

Junpei Suzuki<sup>1)</sup>, Amane Konishi<sup>1,2)</sup>, Toshihiro Yorozya<sup>2)</sup>, Msakatsu Yamashita<sup>1)</sup>

Department of Immunology, Ehime university Graduate School of Medicine, Ehime, Japan<sup>1)</sup>, Department of Anesthesia and Perioperative Medicine, Ehime University Graduate School of Medicine, Ehime, Japan<sup>2)</sup>

1-C-W1-9-O/P

### T-bet<sup>hi</sup> memory-phenotype CD4<sup>+</sup> T cells are spontaneously generated in steady state and exert innate Th1-like effector function

Takeshi Kawabe<sup>1,2,3)</sup>, Dragana Jankovic<sup>2)</sup>, Hidehiro Yamane<sup>3,4)</sup>, Jinfang Zhu<sup>3)</sup>, William E. Paul<sup>5)</sup>, Ronald N. Germain<sup>3)</sup>, Alan Sher<sup>2)</sup>

Department of Microbiology and Immunology, Tohoku University Graduate School of Medicine, Sendai, Japan<sup>1)</sup>, Laboratory of Parasitic Diseases, National Institute of Allergy and Infectious Diseases, National Institutes of Health, Bethesda, USA<sup>2)</sup>, Laboratory of Immune System Biology, National Institute of Allergy and Infectious Diseases, National Institutes of Health, Bethesda, USA<sup>3)</sup>, Vaccine Research Center, National Institute of Allergy and Infectious Diseases, National Institutes of Health, Bethesda, USA<sup>4)</sup>, Deceased<sup>5)</sup>

1-C-W1-16-O/P

### The influenza virus replication derived broad protective antibody response

Kosuke Miyauchi<sup>1)</sup>, Taiki Yajima<sup>2)</sup>, Makoto Takeda<sup>3)</sup>, Masato Kubo<sup>1,2)</sup>

Laboratory for Cytokine Regulation, IMS, RIKEN, Yokohama, Japan<sup>1)</sup>, Division of Molecular Pathology, Research Institute for Biomedical Science, Tokyo University of Science, Noda, Japan<sup>2)</sup>, Department of Virology III, National Institute of Infectious Diseases, Tokyo, Japan<sup>3)</sup>

1-C-W1-18-O/P

### Dynamic regulatory network of IFN $\beta$ /co-inhibitory axis in human T cells

Tomokazu Sumida, Matthew Lincoln, David Hafler

Neurology and Immunobiology, Yale School of Medicine, New Haven, United States

1-C-W1-32-O/P

### Indirect suppression of CD4<sup>+</sup> T cell activation by LAG3-mediated trogocytosis of MHC Class II

Ei Wakamatsu, Hiroaki Machiyama, Hiroko Toyota, Masae Furuhashi, Kikumi Hata, Noriko Yanase, Tadashi Yokosuka

Tokyo Medical University, Tokyo, Japan

Cytokines and chemokines are crucial “environmental stimuli” for immune cells, because these molecules govern both innate and adaptive immune responses, maintain tissue homeostasis, repair tissue, and promote inflammation. Increasing knowledge of cytokines and chemokines has resulted in engineering the new antibodies and small molecule inhibitors that exhibit clinical efficacy for various type of autoimmune diseases and allergic diseases e.g. rheumatoid arthritis and asthma, however, it has been still difficult to connect the basic scientific finding and development of novel therapeutic agents. Thus, the aim of this workshop is to discuss the cutting-edge findings of the diverse features of cytokines and chemokines in both health and disease conditions. We hope that this session will be helpful in extending our knowledge of how cytokines and chemokines network together to maintain health.

1-D-W2-1-O/P

**Tetramer-based model of STAT3 activation-inactivation**

Lingyu Wang, Hong Zhao, Junhao Yang, Hiroyuki Kunimoto, Koichi Nakajima

Department of Immunology, Osaka City University, Graduate School of Medicine, Osaka, Japan

1-D-W2-2-O/P

**Crucial involvement of IL-6 in thrombus resolution in DVT model mice**Mizuho Nosaka<sup>1</sup>, Yuko Ishida<sup>1</sup>, Akihiko Kimura<sup>1</sup>, Yumi Kuninaka<sup>1</sup>, Naofumi Mukaida<sup>2</sup>, Toshikazu Kondo<sup>1</sup>Department of Forensic Medicine, Wakayama Medical University, Wakayama, Japan<sup>1</sup>, Division of Molecular Bioregulation, Cancer Research Institute, Kanazawa University, Kanazawa, Japan<sup>2</sup>

1-D-W2-7-O/P

**Attenuation of renal fibrosis by recombinant thrombomodulin**Atsuro Takeshita<sup>1,2</sup>, Yuko Okano<sup>1,2</sup>, Taro Yasuma<sup>1,2</sup>, Akihiro Uchida<sup>2</sup>, Kota Nishihama<sup>2</sup>, Corina N. D'Alessandro-Gabazza<sup>1</sup>, Masaaki Toda<sup>1</sup>, Yutaka Yano<sup>2</sup>, Esteban C. Gabazza<sup>1</sup>Department of Immunology, Mie University Graduate School of Medicine, Tsu, Japan<sup>1</sup>, Diabetes and endocrinology, Mie University Hospital, Tsu, Japan<sup>2</sup>

1-D-W2-12-O/P

**Involvement of LECT2 in the process of intestinal epithelial wound healing**Kanae Minami<sup>1,2</sup>, Osaka Toshifumi<sup>1,2</sup>, Hidehiro Ueshiba<sup>2</sup>, Satoshi Tsuneda<sup>1</sup>, Junji Yagi<sup>2</sup>, Naoko Yanagisawa<sup>2</sup>Department of Life Science and Medical Bioscience, Waseda University, Tokyo, Japan<sup>1</sup>, Department of Microbiology and Immunology, Tokyo Women's Medical University, Tokyo, Japan<sup>2</sup>

1-D-W2-15-O/P

**The mechanism for regulation of type 2 cytokine production from ILC2 by Tristetraprolin**Yuki Hikichi<sup>1,2</sup>, Yasutaka Motomura<sup>1,3</sup>, Kazuyo Moro<sup>1,3</sup>Laboratory for Innate Immune Systems, IMS, RIKEN, Yokohama, Japan<sup>1</sup>, Department of Medical Life Science, Yokohama City University, Yokohama, Japan<sup>2</sup>, Laboratory for Innate Immune Systems, Department of Microbiology and Immunology, Graduate School of Medicine, Osaka University, Suita, Japan<sup>3</sup>

1-D-W2-18-O/P

**Control of chemoattractant receptor signaling by the COMMD3/8 complex**Akiko Nakai<sup>1</sup>, Jun Fujimoto<sup>1,2</sup>, Kazuhiro Suzuki<sup>1,3</sup>Laboratory of Immune Response Dynamics, WPI Immunology Frontier Research Center, Osaka University, Osaka, Japan<sup>1</sup>, Department of Respiratory Medicine and Clinical Immunology, Graduate School of Medicine, Osaka University, Osaka, Japan<sup>2</sup>, Department of Molecular Immunology, Research Institute for Microbial Diseases, Osaka University, Osaka, Japan<sup>3</sup>

1-D-W2-20-O/P

**Physiological significance of soluble RANKL in the immune systems**Tatsuo Asano<sup>1</sup>, Kazuo Okamoto<sup>2</sup>, Masanori Tsutsumi<sup>1</sup>, Ryunosuke Muro<sup>1</sup>, Takeshi Nitta<sup>1</sup>, Hiroshi Takayanagi<sup>1</sup>Department of Immunology, The University of Tokyo, Tokyo, Japan<sup>1</sup>, Department of Osteoimmunology, The University of Tokyo, Tokyo, Japan<sup>2</sup>

1-D-W2-22-O/P

**SREBF1 EXPRESSION CORRELATES TO CXCL10 EXPRESSION OPPOSITELY BETWEEN INFRAPATELLAR FAT PAD AND SUBCUTANEOUS TISSUES FROM RHEUMATOID ARTHRITIS AND OSTEOARTHRITIS PATIENTS**Shuhe Ma<sup>1</sup>, Kosaku Murakami<sup>1</sup>, Motomu Hashimoto<sup>2</sup>, Masao Tanaka<sup>2</sup>, Hiromu Ito<sup>3</sup>, Tsuneyo Mimori<sup>1</sup>Dept. of Rheumatology and Clinical Immunology, Kyoto University Graduate School of Medicine, Kyoto, Japan<sup>1</sup>, Advanced Medicine of Rheumatic Disease, Kyoto University Graduate School of Medicine, Kyoto, Japan<sup>2</sup>, Dept. of Orthopedic Surgery, Kyoto University Graduate School of Medicine, Kyoto, Japan<sup>3</sup>

Breaking immunological self-tolerance results in autoimmune diseases. That is the reason why multiple mechanisms are involved in maintaining immunological self-tolerance, i.e., central tolerance and peripheral tolerance. Here in this workshop of oral presentations, we will discuss the major player of peripheral tolerance, regulatory T (Treg) cells, and other mechanisms such as controlling antigen presentation and co-stimulatory molecules. We will learn how Treg cells are developed and controlled in thymus and periphery, and how MHC or co-stimulatory molecules control immune suppression. We look forward to active discussion in oral presentations as well as in poster presentations. [Each presentation is expected to finish within 8 min (6 min talk+2 min discussion).]

1-E-W3-1-O/P

### A crucial role of the conserved non-coding sequences Foxp3-CNS0 and -CNS3 in the lineage specification of thymic Foxp3+ regulatory T cells

Ryoji Kawakami<sup>1,2)</sup>, Yohko Kitagawa<sup>1,2)</sup>, Keiko Yasuda<sup>1,2)</sup>, Norihisa Mikami<sup>1)</sup>, Kelvin Y Chen<sup>1)</sup>, Naganari Ohkura<sup>1)</sup>, Hitomi Watanabe<sup>3)</sup>, Gen Kondoh<sup>3)</sup>, Keiji Hirota<sup>1,3)</sup>, Shimon Sakaguchi<sup>1,2)</sup>

Immunology Frontier Research Center, Laboratory of Experimental Immunology, Osaka university, Osaka, Japan<sup>1)</sup>, Institute for Frontier Life and Medical Science, Laboratory of Experimental Immunology, Kyoto university, Kyoto, Japan<sup>2)</sup>, Institute for Frontier Life and Medical Sciences, Laboratory of Integrative Biological Science, Kyoto university, Kyoto, Japan<sup>3)</sup>

1-E-W3-2-O/P

### Foxp3 promotes T cell receptor-dependent effector differentiation and tissue accumulation of Treg cells through functional cooperation with BATF

Ryuichi Murakami<sup>1)</sup>, Wataru Ise<sup>2)</sup>, Tomohiro Kurosaki<sup>2)</sup>, Shohei Hori<sup>1)</sup>

Graduate School of Pharmaceutical Sciences, The University of Tokyo, Tokyo, Japan<sup>1)</sup>, WPI Immunology Frontier Research Center, Osaka University, Osaka, Japan<sup>2)</sup>

1-E-W3-3-O/P

### DNAM-1 suppresses Foxp3 stability of regulatory T cells in a TIGIT dependent manner

Kazuki Sato<sup>1,2,5)</sup>, Yumi Yamashita-Kanemaru<sup>1,2)</sup>, Fumie Abe<sup>1,2)</sup>, Yuho Nakamura<sup>1,3)</sup>, Rikito Murata<sup>1,4)</sup>, Mamoru Ito<sup>6)</sup>, Akira Shibuya<sup>1,2,5)</sup>, Kazuko Shibuya<sup>1,5)</sup>

Department of Immunology, Faculty of Medicine, University of Tsukuba, Ibaraki, Japan<sup>1)</sup>, Life Science Center for survival dynamics, Tsukuba Advanced Research Alliance (TARA), University of Tsukuba, Ibaraki, Japan<sup>2)</sup>, Graduate School of Comprehensive Human Sciences, University of Tsukuba, Ibaraki, Japan<sup>3)</sup>, Ph.D. Program in Human Biology, University of Tsukuba, Ibaraki, Japan<sup>4)</sup>, R&D Center for Innovative Drug Discovery, University of Tsukuba, Ibaraki, Japan<sup>5)</sup>, Central Institute for Experimental Animals, Kanagawa, Japan<sup>6)</sup>

1-E-W3-4-O/P

### Functional characterization of regulatory T (Treg) cells in UVB-irradiated skin

Hiroaki Shime<sup>1)</sup>, Mizuyu Odanaka<sup>1)</sup>, Masaki Imai<sup>1)</sup>, Naganari Ohkura<sup>2,3)</sup>, Shimon Sakaguchi<sup>2)</sup>, Akimichi Morita<sup>4)</sup>, Sayuri Yamazaki<sup>1)</sup>

Department of Immunology, Nagoya City University Graduate School of Medical Sciences, Nagoya, Japan<sup>1)</sup>, Department of Experimental Immunology, World Premier International Research Center Initiative, Immunology Frontier Research Center, Osaka University, Osaka, Japan<sup>2)</sup>, Department of Frontier Research in Tumor Immunology, Center of Medical Innovation and Translational Research, Graduate School of Medicine, Osaka University, Osaka, Japan<sup>3)</sup>, Department of Geriatric and Environmental Dermatology, Nagoya City University Graduate School of Medical Sciences, Nagoya, Japan<sup>4)</sup>

1-E-W3-5-O/P

### Single-cell transcriptomic atlas of thymic Treg development

Yoshiaki Yasumizu<sup>1)</sup>, Naganari Ohkura<sup>1,2)</sup>, Yamami Nakamura<sup>1)</sup>, Atsushi Tanaka<sup>1,2)</sup>, Shimon Sakaguchi<sup>1)</sup>

Experimental Immunology, Immunology Frontier Research Center, Osaka University, Osaka, Japan<sup>1)</sup>, Frontier Research in Tumor Immunology, Faculty of Medicine, Osaka University, Osaka, Japan<sup>2)</sup>

1-E-W3-6-O/P

### Antigen-specific Foxp3<sup>+</sup>CD4<sup>+</sup> Tregs suppress T cell response to cognate antigen but not suppress T cell response to non-cognate antigen in vivo

Yoshihiro Oya<sup>1,3)</sup>, Ryutaro Matsumura<sup>1)</sup>, Hiroshi Nakajima<sup>2)</sup>, Ethan Shevach<sup>3)</sup>

Department of Rheumatology, Allergy & Clinical Immunology, Laboratory of Autoimmune diseases, National Hospital Organization Chibahigashi National Hospital, Chiba, Japan<sup>1)</sup>, Department of Allergy and Clinical Immunology, Graduate School of Medicine, Chiba University, Chiba, Japan<sup>2)</sup>, Cellular Immunology Section, Laboratory of Immunology, National Institute of Allergy and Infectious Diseases, National Institutes of Health, MD, U.S.A<sup>3)</sup>

1-E-W3-7-O/P

**Harnessing immunity by controlling the stability of antigen-specific regulatory T cells via manipulation of the functional avidity of self-dominant peptide that regulates the kinetics of TCR signaling**

Youwei Lin<sup>1,2)</sup>, Chandirasegaran Massilamany<sup>3)</sup>, Jayagopala Reddy<sup>3)</sup>, Takashi Yamamura<sup>1)</sup>

Department of Immunology, National Institute of Neuroscience, National Center of Neurology and Psychiatry, Tokyo, Japan<sup>1)</sup>, Department of Neurology, National Center Hospital, National Center of Neurology and Psychiatry, Tokyo, Japan<sup>2)</sup>, School of Veterinary Medicine and Biomedical Sciences, University of Nebraska-Lincoln, Lincoln, USA<sup>3)</sup>

1-E-W3-8-O/P

**Restriction of PD-1 function by *cis*-PD-L1/CD80 interactions is required for optimal T cell respons**

Daisuke Sugiura, Takumi Maruhashi, Il-mi Okazaki, Kenji Shimizu, Takeo K. Maeda, Taku Okazaki

Division of Immune Regulation, Institute of Advanced Medical Sciences, Tokushima University, Tokushima, Japan

1-E-W3-9-O/P

**CD74 isoform p41 regulates soluble MHC class II production**

Masako Kohyama<sup>1,2)</sup>, Sumiko Matsuoka<sup>1)</sup>, Testuya Shishido<sup>1)</sup>, Hisashi Arase<sup>1,2)</sup>

Department of Immunochemistry, Research Institute for Microbial Diseases, Osaka University, Osaka, Japan<sup>1)</sup>, Laboratory of Immunochemistry, WPI Immunology Frontier Research Center, Osaka University, Osaka, Japan<sup>2)</sup>

1-E-W3-10-O/P

**Ten-eleven translocation (Tet) in B cells prevent autoimmunity**

Shinya Tanaka<sup>1)</sup>, Wataru Ise<sup>2)</sup>, Tomohiro Kurosaki<sup>2)</sup>, Yoshihiro Baba<sup>1)</sup>

Division of Immunology and Genome Biology, Department of Molecular Genetics, Medical Institute of Bioregulation, Kyushu University, Fukuoka, Japan<sup>1)</sup>, Laboratory of Lymphocyte Differentiation, WPI Immunology Frontier Research Center, Osaka University, Osaka, Japan<sup>2)</sup>

Mouse models of human immunological disorders have contributed greatly to the understanding of the human immune system. This approach culminated in a spectacular success of checkpoint inhibitors in cancer therapies. However, there still remain many unanswered questions in the etiology and pathology of human immunological disorders. Recent development of comprehensive and unbiased genome analysis, including genome-wide association study and whole genome/exome sequencing provided important new information to tackle complex human disorders. In this context, the scope of this workshop is to share up-to date information and to cover the latest topics in immunology research using human subjects, human cells, and humanized mouse models. We hope that the presentation will contribute to the better understanding of the molecular mechanisms involved in the control of human immunological disorders, including cancer, autoimmunity, and allergy.

1-F-W4-1-O/P

#### **A metagenome-wide association study of gut microbiome revealed novel etiology of rheumatoid arthritis in the Japanese population**

Toshihiro Kishikawa, Yuichi Maeda, Takuro Nii, Toru Hirano, Masashi Narazaki, Yukihiko Saeki, Atsushi Kumanogoh, Kiyoshi Takeda, Yukinori Okada

Osaka University Graduate School of Medicine, Suita, Japan

1-F-W4-2-O/P

#### **A genome-wide association study of Stevens-Johnson syndrome based on whole genome sequencing approach**

Yuki Hitomi<sup>1,2)</sup>, Mayumi Ueta<sup>3)</sup>

Department of Microbiology, Hoshi University School of Pharmacy and Pharmaceutical Sciences, Tokyo, Japan<sup>1)</sup>, Department of Human Genetics, Graduate School of Medicine, The University of Tokyo, Tokyo, Japan<sup>2)</sup>, Department of Ophthalmology, Kyoto Prefectural University of Medicine, Kyoto, Japan<sup>3)</sup>

1-F-W4-3-O/P

#### **Type I IFN-mediated effective priming of functional HIV-1-specific CD8<sup>+</sup> T cells from naïve T cells**

Nozomi Kuse<sup>1,2)</sup>, Xiaoming Sun<sup>2)</sup>, Anna Lissina<sup>3)</sup>, Takuya Yamamoto<sup>4)</sup>, Victor Appay<sup>3)</sup>, Masafumi Takiguchi<sup>1,2)</sup>

Division of International Collaboration Research, Department of Frontier Research, Joint Research Center for Human Retrovirus Infection, Kumamoto University, Tokyo, Japan<sup>1)</sup>, Center for AIDS Research, Kumamoto University, Kumamoto, Japan<sup>2)</sup>, INSERM/Université Pierre et Marie Curie-Paris6, Paris, France<sup>3)</sup>, Laboratory of Immunosenescence, National Institutes of Biomedical Innovation, Health and Nutrition, Osaka, Japan<sup>4)</sup>

1-F-W4-4-O/P

#### **Immune cell profiling of rheumatoid arthritis identified gene networks and BCR repertoire diversity correlated with treatment resistance**

Saeko Yamada<sup>1)</sup>, Yasuo Nagafuchi<sup>1)</sup>, Mineto Ota<sup>1,2)</sup>, Yusuke Takeshima<sup>1,2)</sup>, Hiroaki Hatano<sup>1)</sup>, Yukiko Iwasaki<sup>1)</sup>, Shuji Sumitomo<sup>1)</sup>, Tomohisa Okamura<sup>1,2)</sup>, Hirofumi Shoda<sup>1)</sup>, Kazuhiko Yamamoto<sup>3)</sup>, Keishi Fujio<sup>1)</sup>

Department of Allergy and Rheumatology, Graduate School of Medicine, the University of Tokyo, Tokyo, Japan<sup>1)</sup>, Department of Functional Genomics and Immunological Diseases, Graduate School of Medicine, the University of Tokyo, Tokyo, Japan<sup>2)</sup>, Laboratory for Autoimmune Diseases, Center for Integrative Medical Sciences, RIKEN, Tokyo, Japan<sup>3)</sup>

1-F-W4-5-O/P

#### **Common TCR $\beta$ Repertoire was found in both decidual and peripheral CD8<sup>+</sup>T cells in normal term pregnancy**

Keiko Morita<sup>1)</sup>, Eiji Kobayashi<sup>2)</sup>, Sayaka Tsuda<sup>1)</sup>, Kiyomi Shitaoka<sup>2)</sup>, Tatsuhiko Ozawa<sup>2)</sup>, Hiroshi Hamana<sup>2)</sup>, Shigeru Saito<sup>1)</sup>, Hiroyuki Kishi<sup>2)</sup>

Department of Obstetrics and Gynecology, University of Toyama, Toyama, Japan<sup>1)</sup>, Dept. Immun., Grad. Sch. Med. & Pharm. Sci., University of Toyama, Toyama, Japan<sup>2)</sup>

1-F-W4-6-O/P

#### **Creation of HLA class I and class II Tg NOD/SCID/Il2rgKO (NSG) mice for studying interaction between human immunity and leukemia**

Ari Itoh-Nakadai<sup>1)</sup>, Yuho Najima<sup>1,4)</sup>, Rintaro Ono<sup>1)</sup>, Masashi Matsuda<sup>2)</sup>, Kaori Sato<sup>1)</sup>, Mariko Murasawa-Tomizawa<sup>1)</sup>, Osamu Ohara<sup>3)</sup>, Haruhiko Koseki<sup>2)</sup>, Fumihiko Ishikawa<sup>1)</sup>

Laboratory for Human Disease models, Center for Integrative Medical Sciences, RIKEN, Kanagawa, Japan<sup>1)</sup>, Laboratory for Developmental Genetics, Center for Integrative Medical Sciences RIKEN, RIKEN, Kanagawa, Japan<sup>2)</sup>, Laboratory for Integrative Genomics, Center for Integrative Medical Sciences RIKEN, RIKEN, Kanagawa, Japan<sup>3)</sup>, Hematology Division, Komagome Hospital, Tokyo metropolitan cancer and infectious diseases center, Tokyo, Japan<sup>4)</sup>

### **Novel proteasome-related autoinflammation and immunodeficiency disease caused by a distinct heterozygous missense mutation in the *PSMB9* gene**

Nobuo Kanazawa<sup>1)</sup>, Noriko Kinjo<sup>2)</sup>, Satoru Hamada<sup>2)</sup>, Tsunehiro Mizushima<sup>3)</sup>, Akira Kinoshita<sup>4)</sup>, Koh-Ichiro Yoshiura<sup>4)</sup>, Jun Hamazaki<sup>5)</sup>, Shigeo Murata<sup>5)</sup>, Hidenori Ohnishi<sup>6)</sup>, Takashi Orimo<sup>7)</sup>, Hiroaki Hemmi<sup>7,8)</sup>, Tsuneyasu Kaisho<sup>7)</sup>

Department of Dermatology, Wakayama Medical University, Wakayama, Japan<sup>1)</sup>, Department of Child Health and Welfare (Pediatrics), University of the Ryukyus Graduate School of Medicine, Okinawa, Japan<sup>2)</sup>, Picobiology Institute, University of Hyogo Graduate School of Life Science, Hyogo, Japan<sup>3)</sup>, Department of Human Genetics, Nagasaki University Atomic Bomb Disease Institute, Nagasaki, Japan<sup>4)</sup>, Laboratory of Protein Metabolism, University of Tokyo Graduate School of Pharmaceutical Sciences, Tokyo, Japan<sup>5)</sup>, Department of Pediatrics, Gifu University Graduate School of Medicine, Gifu, Japan<sup>6)</sup>, Department of Immunology, Wakayama Medical University Institute of Advanced Medicine, Wakayama, Japan<sup>7)</sup>, Faculty of Veterinary Medicine, Okayama University of Science, Imabari, Japan<sup>8)</sup>

### **Immunophenotyping by CyTOF can reveal impaired activation of monocytes or CD4<sup>+</sup> T cells by anti-IL-6 autoantibody**

Takayoshi Morita<sup>1)</sup>, Yusuke Manabe<sup>2)</sup>, Hachiro Konaka<sup>2)</sup>, Yasuhiro Kato<sup>3)</sup>, Akiko Kajihara<sup>2)</sup>, Masashi Narazaki<sup>3)</sup>, Atsushi Kumanogoh<sup>1,2)</sup>

Laboratory of Immunopathology, World Premier International Immunology Frontier Research Center, Osaka University, Suita, Japan<sup>1)</sup>, Department of Respiratory Medicine and Clinical Immunology, Graduate School of Medicine, Osaka University, Suita, Japan<sup>2)</sup>, Department of Advanced Clinical and Translational Immunology, Graduate School of Medicine, Suita, Japan<sup>3)</sup>

Great progress in studies about innate immunity has revealed a variety of pattern recognition receptors (PRRs) that sense microbe/pathogen-associated molecular patterns (MAMPs or PAMPs) to massively induce innate cytokine responses during microbial infection, leading to acute inflammation as a host-protective response. In addition, damage-related stimuli such as tissue injury, ischemia, or stress induce the secretion of so-called danger-associated molecular patterns (DAMPs), which triggers sterile inflammation. In addition, these inflammatory responses should be properly resolved for both tissue repair and restoration of homeostasis. Persistent inflammation or unresolved inflammation may cause pathogenic conditions. In this regard, ligands for these PRRs have not been fully identified yet, and it remains to be completely understood how PRR-mediated signalings and resolution of inflammation are regulated. In this session, eight selected papers will provide key insights into these issues in the innate immune system.

1-G-W5-1-O/P

**ZNF598 delivers a ubiquitin-like modifier FAT10 to RIG-I and attenuates the innate immune response against viral infection**

Guanming Wang, Takahisa Kouwaki, Hiroyuki Oshiumi  
Department of Immunology, Kumamoto University, Kumamoto, Japan

1-G-W5-11-O/P

**Cytidine deaminase enables Toll-like receptor 8 activation by cytidine or its analogs**

Katsuhiro Furusho<sup>1)</sup>, Takuma Shibata<sup>2)</sup>, Ryota Sato<sup>2)</sup>, Ryutaro Fukui<sup>2)</sup>, Yuji Motoi<sup>2)</sup>, Shin-ichiroh Saitoh<sup>2)</sup>, Kensuke Miyake<sup>2)</sup>  
National Center for Geriatrics and Gerontology, Aichi, Japan<sup>1)</sup>, Division of Innate Immunity, Department of Microbiology and Immunology, The Institute of Medical Science, The University of Tokyo, Tokyo, Japan<sup>2)</sup>

1-G-W5-15-O/P

**Identification of novel lipophilic ligands of Siglec5 and 14 that modulate innate immune responses**

Yasunobu Miyake, Rie Suematsu, Hiroki Yoshida  
Saga University, Faculty of Medicine, Saga, Japan

1-G-W5-19-O/P

**Unwinding of stem-loops by UPF1 licenses Regnase-1 to degrade inflammatory mRNAs**

Takashi Mino, Osamu Takeuchi  
Graduate School of Medicine, Kyoto University, Kyoto, Japan

1-G-W5-21-O/P

**Unique role of an oligopeptide transporter SLC15A3 in the lung inflammation and resolution**

Toshihiko Kobayashi, Dat Nguyen-Tien, Noriko Toyama-Sorimachi  
Department of Molecular Immunology and Inflammation, National Center for Global Health and Medicine, Tokyo, Japan

1-G-W5-6-O/P

**DJ-1 plays a pivotal role in the induction of sterile inflammation after ischemic stroke**

Koutarou Nakamura<sup>1,2)</sup>, Takashi Shichita<sup>2)</sup>  
Department of Computational Biology and Medical Sciences, Graduate School of Frontier Sciences, The University of Tokyo, Tokyo, Japan<sup>1)</sup>, Stroke Renaissance Project, Tokyo Metropolitan Institute of Medical Science, Tokyo, Japan<sup>2)</sup>

1-G-W5-13-O/P

**Comprehensive analysis of macrophages in the pathogenesis of diabetic nephropathy in db/db mice**

Seigo Ito<sup>1,2)</sup>, Takuya Ishikiriya<sup>1)</sup>, Kazuki Koiwai<sup>1)</sup>, Hiroyuki Nakashima<sup>1)</sup>, Masahiro Nakashima<sup>1)</sup>, Manabu Kinoshita<sup>1)</sup>, Shuhji Seki<sup>1)</sup>  
Department of Immunology and Microbiology, National Defense Medical College, Tokorozawa, Japan<sup>1)</sup>, Department of Nephrology and Endocrinology, National Defense Medical College, Tokorozawa, Japan<sup>2)</sup>

1-G-W5-22-O/P

**Immunologic potential of herbal medicines as vaccine adjuvant**

Kou Hioki<sup>1,2)</sup>, Etsushi Kuroda<sup>2,3)</sup>, Ken J. Ishii<sup>1,2)</sup>  
Division of Vaccine Science, Department of Microbiology and Immunology, The Institute of Medical Science, The University of Tokyo, Tokyo, Japan<sup>1)</sup>, Center for Vaccine and Adjuvant Research (CVAR), National Institutes of Biomedical Innovation, Health and Nutrition (NIBIOHN), Osaka, Japan<sup>2)</sup>, Department of Immunology, Hyogo College Of Medicine, Hyogo, Japan<sup>3)</sup>



The bone marrow microenvironment is crucial for hematopoietic stem cell maintenance and immune cell development, and also contributes to the maintenance of memory T cells. On the other hand, the thymus is specialized for T lymphopoiesis, and the lymph nodes and spleen provide the platform for initiating immune responses. Various types of stromal cells with specific roles regulate the function of each lymphoid organ. In addition, recent studies have revealed that these functional processes can be modulated by several stimuli such as infection and inflammation. In this session, we will discuss the functional characterization of stromal cells in lymphoid organs and the mechanisms that regulate their in vivo dynamics in response to various stimuli.

1-H-W6-3-O/P

**Essential roles of PCGF1 in controlling hematopoietic cell fates**Tomokatsu Ikawa<sup>1)</sup>, Junichiro Takano<sup>2)</sup>, Haruhiko Koseki<sup>2)</sup>Division of Immunobiology, Research Institute for Biomedical Sciences, Tokyo University of Science, Noda, Japan<sup>1)</sup>, Laboratory for Developmental Genetics, Center for Integrative Medical Sciences, RIKEN, Yokohama, Japan<sup>2)</sup>

1-H-W6-8-O/P

**Sensing of the microbiota by NOD1 in mesenchymal stromal cells regulates murine hematopoiesis**Chiaki Iwamura<sup>1,2)</sup>, Alan Sher<sup>2)</sup>, Toshinori Nakayama<sup>1)</sup>, Dragana Jankovic<sup>2)</sup>Chiba University Graduate school of Medicine, Chiba, Japan<sup>1)</sup>, NIAID, Laboratory of Parasitic diseases, National Institute of Health, Bethesda, USA<sup>2)</sup>

1-H-W6-9-O/P

**Gut microbiota orchestrates early hematopoiesis upon gut inflammation**Yoshikazu Hayashi<sup>1)</sup>, Shinji Fukuda<sup>2)</sup>, Hitoshi Takizawa<sup>1)</sup>International Research Center for Medical Sciences, Kumamoto University, Kumamoto, Japan<sup>1)</sup>, Institute for Advanced Biosciences, Keio University, Yamagata, Japan<sup>2)</sup>

1-H-W6-11-O/P

**Bone marrow endothelial cells induce rapid activation of memory CD8<sup>+</sup> T cell by cross-presentation of blood-borne antigens**Takeshi Ito<sup>1,2)</sup>, Nagahiro Minato<sup>3)</sup>, Yoko Hamazaki<sup>1,2)</sup>Center for iPS Cell Research and Application (CiRA), Kyoto University, Kyoto, Japan<sup>1)</sup>, Laboratory of Immunobiology, Graduate School of Medicine, Kyoto University, Kyoto, Japan<sup>2)</sup>, Medical Innovation Center, Graduate School of Medicine, Kyoto University, Kyoto, Japan<sup>3)</sup>

1-H-W6-12-O/P

**Identification and characterization of thymic medullary fibroblasts**

Takeshi Nitta, Masanori Tsutsumi, Ema Suzuki, Ryunosuke Muro, Hiroshi Takayanagi

Department of Immunology, Graduate School of Medicine and Faculty of Medicine, The University of Tokyo, Tokyo, Japan

1-H-W6-15-O/P

**Estrogens suppress T cell production by a TEC-dependent mechanism**

Shinya Abe, Aki Ejima, Akihiro Shimba, Mizuki Mori, Guangwei Cui, Takuma Asahi, Daichi Takami, Takahiro Hara, Shizue Tani-ichi, Koichi Ikuta

Laboratory of Immune Regulation, Institute for Frontier Life and Medical Sciences, Kyoto University, Kyoto, Japan

1-H-W6-19-O/P

**Accumulation of leukemic cells to the spleen is regulated by a transcription factor Tlx1 expressed in perfollicular mesenchymal cells**Yusuke Amemiya<sup>1,2)</sup>, Shogo Okazaki<sup>1)</sup>, Chiharu Nishiyama<sup>2)</sup>, Takuro Nakamura<sup>3)</sup>, Ryo Goitsuka<sup>1)</sup>Division of Development and Aging, Research Institute for Biomedical Sciences, Tokyo University of Science, Chiba, Japan<sup>1)</sup>, Laboratory of Molecular Biology and Immunology, Department of Biological Science and Technology, Tokyo University of Science, Tokyo, Japan<sup>2)</sup>, Division of Carcinogenesis, Cancer Institute, Japanese Foundation for Cancer Research, Tokyo, Japan<sup>3)</sup>

1-H-W6-25-O/P

**Intercellular communication between hematopoietic cells through Dectin-1**Shojiro Haji<sup>1)</sup>, Takashi Shimizu<sup>1)</sup>, Taiki Ito<sup>1)</sup>, Masato Tanaka<sup>2)</sup>, Sho Yamasaki<sup>1)</sup>Division of Host Defense, Department of Molecular Immunology, Research Institute for Microbial Diseases, Osaka University, Osaka, Japan<sup>1)</sup>, Laboratory of Immune Regulation, The School of Life Sciences, Tokyo University of Pharmacy and Life Sciences, Tokyo, Japan<sup>2)</sup>



## W7 Systemic lupus erythematosus and other systemic autoimmune diseases

15:25 ~ 16:45 Room B

Chairpersons: Sachiko Miyake, Fujio Keishi

Systemic autoimmunity including systemic lupus erythematosus (SLE) is an autoimmune disorder characterized by loss of tolerance to self-antigens. Although enormous progress has been made in the immunological and genetic understanding of systemic autoimmunity, controlling life-long disease activity without comorbidity is a clinically tough challenge. In order to develop a treatment with fewer side effects than the current nonspecific immunosuppressive therapy, it is essential to clarify the responsible pathological condition. Recent advance of immunological technology enables us to analyze immune response in detail for both animal models and human. Novel exciting roles of innate and adaptive immune system presented in this workshop will greatly contribute to the development of new therapy and patient stratification. We expect active discussion and communication by all participants.

2-B-W7-1-O/P

### Human TLR7 deficiency results in autoimmune conditions

Natsuko Yamakawa<sup>1,2</sup>, Anthony Hayward<sup>3</sup>, Charlotte Cunningham-Rundles<sup>4</sup>, Eric Meffre<sup>2</sup>

Tsukuba Primate Research Center, National Institutes of Biomedical Innovation, Health and Nutrition, Tsukuba, Japan<sup>1</sup>, Department of Immunobiology, Yale University, New Haven, USA<sup>2</sup>, The Alpert Medical School of Brown University, Rhode Island, USA<sup>3</sup>, Department of Medicine, Mount Sinai Medical Center, New York, USA<sup>4</sup>

2-B-W7-2-O/P

### IRF5 activation persists in SLE patients undergoing standard therapies and a prototype IRF5 inhibitor restrains autoantibody increment in mouse SLE models

Go R Sato<sup>1</sup>, Tatsuma Ban<sup>1</sup>, Masako Kikuchi<sup>1,2</sup>, Akio Manabe<sup>1</sup>, Ryusuke Yoshimi<sup>3</sup>, Shuichi Ito<sup>2</sup>, Tomohiko Tamura<sup>1</sup>

Department of Immunology, Yokohama City University Graduate School of Medicine, Yokohama, Japan<sup>1</sup>, Department of Pediatrics, Yokohama City University Graduate School of Medicine, Yokohama, Japan<sup>2</sup>, Department of Stem Cell and Immune Regulation, Yokohama City University Graduate School of Medicine, Yokohama, Japan<sup>3</sup>

2-B-W7-12-O/P

### Imbalance between memory Th1 and Th1-Treg cells depends on differential regulation of cell metabolism in patients with SLE

Shigeru Iwata<sup>1</sup>, Mingzeng Zhang<sup>1</sup>, Maiko Hajime<sup>1</sup>, Yusuke Miyazaki<sup>1</sup>, Naoaki Ohkubo<sup>1</sup>, Hiroko Miyata<sup>1</sup>, Yasuyuki Todoroki<sup>1</sup>, Shingo Nakayamada<sup>1</sup>, Kei Sakata<sup>2</sup>, Yoshiya Tanaka<sup>1</sup>

First Department of Internal Medicine, University of Occupational & Environmental Health, Japan, Kitakyushu, Japan<sup>1</sup>, Mitsubishi Tanabe Pharma, Yokohama, Japan<sup>2</sup>

2-B-W7-14-O/P

### Immune cell profiling of systemic lupus erythematosus reveals a key regulator of its pathogenesis and contributes to patient stratification

Masahiro Nakano<sup>1</sup>, Yukiko Iwasaki<sup>1</sup>, Yusuke Takeshima<sup>1,2</sup>, Mineto Ota<sup>1,2</sup>, Yasuo Nagafuchi<sup>1</sup>, Shuji Sumitomo<sup>1</sup>, Tomohisa Okamura<sup>1,2</sup>, Keishi Fujio<sup>1</sup>

Department of Allergy and Rheumatology, Graduate School of Medicine, The University of Tokyo, Tokyo, Japan<sup>1</sup>, Department of Functional Genomics and Immunological Diseases, Graduate School of Medicine, The University of Tokyo, Tokyo, Japan<sup>2</sup>

2-B-W7-15-O/P

### MAIT cells enhance autoreactive B cell responses in a murine model of lupus

Taiga Kuga<sup>1</sup>, Gou Murayama<sup>1</sup>, Asako Chiba<sup>2</sup>, Tomohiro Mizuno<sup>2</sup>, Atsushi Nomura<sup>2</sup>, Hirofumi Amano<sup>1</sup>, Sachiko Hirose<sup>3</sup>, Ken Yamaji<sup>1</sup>, Naoto Tamura<sup>1</sup>, Sachiko Miyake<sup>2</sup>

Department of Rheumatology and Internal Medicine, Juntendo University School of Medicine, Tokyo, Japan<sup>1</sup>, Department of Immunology, Juntendo University School of Medicine, Tokyo, Japan<sup>2</sup>, Toin Human Science and Technology Center, Department of Biomedical Engineering, Toin University of Yokohama, Yokohama, Japan<sup>3</sup>

2-B-W7-16-O/P

### Multiple tolerance checkpoints suppress generation and activation of B cells producing low-affinity germline precursors of SLE patient-derived high-affinity anti-dsDNA antibody in BCR knock-in mice

Marwa Ali El Hussien, Shuhei Sakakibara, Chao-Yuan Tsai, Hitoshi Kikutani

Immune Regulation, Immunology Frontier Research Center, Osaka University, Suita, Japan

2-B-W7-17-O/P

### **Stratification of SLE based on the BLyS biological activities**

Eri Itotagawa, Jeonghoon Park, Kohei Tsujimoto, Hachiro Konaka, Hyota Takamatsu, Atsushi Kumanogoh  
Department of Respiratory Medicine and Clinical Immunology, Graduate School of Medicine, Osaka University, Osaka, Japan

2-B-W7-18-O/P

### **The inhibitory B cell co-receptor CD72 recognizes various lupus self-antigens including C1q and inhibits B cell responses to these self-antigen**

Chizuru Akatsu<sup>1)</sup>, Quan-Zhen Li<sup>2)</sup>, Hideharu Sekine<sup>3)</sup>, Teizo Fujita<sup>4)</sup>, Takeshi Tsubata<sup>1)</sup>

Medical Research Institute, Department of Immunology, Tokyo Medical and Dental University, Tokyo, Japan<sup>1)</sup>, Department of Immunology and Internal Medicine, UT Southwestern Medical Center, Dallas, USA<sup>2)</sup>, Department of Immunology, Fukushima Medical University, Fukushima, Japan<sup>3)</sup>, Fukushima Prefectural General Hygiene Institute, Fukushima, Japan<sup>4)</sup>

T cells are generated through stepwise processes for development and selection in the thymus as well as activation and functional differentiation in the periphery, to exert their critical functions in pathogen clearance and tissue homeostasis. In this session, we would like to discuss several key events in the T cell development and activation, including early T cell development and positive/negative selection in the thymus, TCR signaling, chemokine/cytokine signaling, and differentiation into effector/regulatory T cells. We hope interactive discussion in this workshop will advance our understanding of T cells in relation to both biological and medical aspects.

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**2-C-W8-2-O/P****GATA3 and Runx1 repress PU.1 expression in early T cell development**Hiroyuki Hosokawa<sup>1,2)</sup>, Katsuto Hozumi<sup>1)</sup>, Ellen Rothenberg<sup>2)</sup>Department of Immunology, Tokai University School of Medicine, Kanagawa, Japan<sup>1)</sup>, Division of Biology & Biological Engineering, California Institute of Technology, Pasadena, USA<sup>2)</sup>**2-C-W8-4-O/P****The Establishment of 3D Genome Structure for Rag1/Rag2 Expression Mediated by E2A/E-protein Enhancer Activity**

Masaki Miyazaki, Hiroshi Kawamoto, Kazuko Miyazaki

Department of Immunology, Institute for Frontier Life and Medical Sciences, Kyoto University, Kyoto, Japan

**2-C-W8-6-O/P****A role of extracellular vesicles in T cell development**Tomoyoshi Yamano<sup>1)</sup>, Xiabing Lyu<sup>1)</sup>, Iriya Fijitsuka<sup>1)</sup>, Yoshinori Hasebe<sup>1)</sup>, Rikinari Hanayama<sup>1,2)</sup>Department of Immunology, Kanazawa University Graduate School of Medical Sciences, Kanazawa, Japan<sup>1)</sup>, WPI Nano Life Science Institute (NanoLSI), Kanazawa University, Kanazawa, Japan<sup>2)</sup>**2-C-W8-10-O/P****THEMIS functions as a cytoplasmic adaptor and nuclear transcription factor in developing thymocytes and mature T cells**

Kiyokazu Kakugawa, Hilde Cheroutre

RIKEN Center for Integrative Medical Science, Division of Human Immunology, Laboratory for Immune Crosstalk, Yokohama, Japan

**2-C-W8-11-O/P****Different requirement of the coreceptors CD4 and CD8 for initiation of T cell activation**Hiroaki Machiyama<sup>1)</sup>, Ei Wakamatsu<sup>1)</sup>, Kikumi Hata<sup>1)</sup>, Noriko Yanase<sup>1)</sup>, Tomohiro Takehara<sup>1,2)</sup>, Masae Furuhashi<sup>1)</sup>, Hiroko Toyota<sup>1)</sup>, Tadashi Yokosuka<sup>1)</sup>Department of Immunology, Tokyo Medical University, Tokyo, Japan<sup>1)</sup>, Division of Pulmonary Medicine, Department of Internal Medicine, Keio University School of Medicine, Tokyo, Japan<sup>2)</sup>**2-C-W8-13-O/P****Rap1 accelerates T cell polarity formation via the enhancement of Rho-Rock pathways**Yoshihiro Ueda<sup>1)</sup>, Koichiro Higasa<sup>2)</sup>, Yuji Kamioka<sup>1)</sup>, Naoyuki Kondo<sup>1)</sup>, Tatsuo Kinashi<sup>1)</sup>Molecular Genetics, Institute of Biomedical Science, Kansai Medical University, Hirakata, Osaka, Japan<sup>1)</sup>, Department of Genome Analysis, Institute of Biomedical Science, Kansai Medical University, Hirakata, Osaka, Japan<sup>2)</sup>**2-C-W8-17-O/P****STAP-2 positively modulates TCR-mediated T cell activation**Kodai Saitoh<sup>1)</sup>, Jun-ichi Kashiwakura<sup>1)</sup>, Akihiko Yoshimura<sup>2)</sup>, Kenji Oritani<sup>3)</sup>, Tadashi Matsuda<sup>1)</sup>Department of Immunology, Hokkaido University, Hokkaido, Japan<sup>1)</sup>, Department of Microbiology and Immunology, Keio University School of Medicine, Tokyo, Japan<sup>2)</sup>, Department of Hematology, International University of Health and Welfare, Tochigi, Japan<sup>3)</sup>**2-C-W8-27-O/P****High dimensional analysis of T-follicular regulatory cells**James B Wing<sup>1)</sup>, Shimon Sakaguchi<sup>1,2)</sup>IFReC, Osaka University, Osaka, Japan<sup>1)</sup>, Department of Experimental Pathology, Institute for Frontier Medical Sciences, Kyoto University, Kyoto, Japan<sup>2)</sup>

Innate inflammation, which is characterized by cytokine responses, may be driven by alterations in interactions between host and its environment, such as through microbial invasion, chemical exposures, stress, and an altered microbiome. The inflammasome is one of the major factors that mediate the release of key pro-inflammatory cytokines. Inflammasomes are multimeric cytosolic protein complexes that are assembled upon the sensing of a variety of pathogens or endogenous damages, leading to interleukin-1 (IL-1)- and IL-18-mediated inflammation and gasdermin D-dependent pyroptosis. It has also been reported that dysregulated inflammation is provoked by mutations that cause the constitutive activation of innate sensors such as NLRs or RLRs, which may play important causative roles in autoinflammatory, autoimmune, and metabolic diseases. In this session, we will discuss about newly identified inflammasome activators, regulation of inflammasome-related cytokine responses as well as genetic mutations of innate sensors linking to dysregulated hyperactivation of cytokine induction.

## 2-E-W9-1-O/P

**Molecular mechanisms of Imiquimod (IMQ)-induced cell death and its role in inflammation**

Haruna Okude<sup>1)</sup>, Daisuke Ori<sup>1)</sup>, Takumi Kawasaki<sup>1)</sup>, Masatoshi Momota<sup>1,2)</sup>, Ken J. Ishii<sup>2,3,4)</sup>, Masahiro Yamamoto<sup>5,6)</sup>, Taro Kawai<sup>1)</sup>

Division of Biological Science, Laboratory of Molecular Immunobiology, Nara Institute of Science and Technology, Nara, Japan<sup>1)</sup>, Laboratory of Mockup Vaccine, National Institutes of Biomedical Innovation, Health and Nutrition (NIBIOHN), Osaka, Japan<sup>2)</sup>, Division of Vaccine Science, The Institute of Medical Science, The University of Tokyo, Tokyo, Japan<sup>3)</sup>, International Research and Development Center for Mucosal Vaccines, The Institute of Medical Science, The University of Tokyo, Tokyo, Japan<sup>4)</sup>, Laboratory of Immunoparasitology, Immunology Frontier Research Center, Osaka University, Osaka, Japan<sup>5)</sup>, Department of Immunoparasitology, Research Institute for Microbial Diseases, Osaka University, Osaka, Japan<sup>6)</sup>

## 2-E-W9-2-O/P

**Roles of endoplasmic reticulum stress in cholera toxin B-induced interleukine-1 $\beta$  production from resident peritoneal macrophages**

Izumi Sasaki<sup>1)</sup>, Shuhei Morita<sup>3)</sup>, Daisuke Okuzaki<sup>4)</sup>, Takashi Orimo<sup>1)</sup>, Hiroaki Hemmi<sup>5)</sup>, Koichi Furukawa<sup>6)</sup>, Tsuneyasu Kaisho<sup>1,2)</sup>

Department of Immunology, Institute of Advanced Medicine, Wakayama Medical University, Wakayama, Japan<sup>1)</sup>, Laboratory for Immune Regulation, Immunology Frontier Research Center, Osaka University, Suita, Japan<sup>2)</sup>, First Department of Medicine, Wakayama Medical University, Wakayama, Japan<sup>3)</sup>, Genome Information Research Center, Research Institute for Microbial Diseases, Osaka University, Suita, Japan<sup>4)</sup>, Faculty of Veterinary Medicine, Okayama University of Science, Imabari, Japan<sup>5)</sup>, Department of Lifelong Sports and Health Sciences, Chubu University College of Life and Health Sciences, Kasugai, Japan<sup>6)</sup>

## 2-E-W9-5-O/P

**Gasdermin D mediates IL-1 $\alpha$  maturation during inflammasome formation**

Kohsuke Tsuchiya<sup>1,2)</sup>, Takashi Suda<sup>1)</sup>

Division of Immunology and Molecular Biology, Cancer Research Institute, Kanazawa University, Kanazawa, Japan<sup>1)</sup>, Institute for Frontier Science Initiative (InFiniti), Kanazawa University, Kanazawa, Japan<sup>2)</sup>

## 2-E-W9-6-O/P

**Lamtor1/p18 is required for activation of inflammasome**

Kohei Tsujimoto, Hyota Takamatsu, Atsushi Kumanogoh

Respiratory Medicine and Clinical Immunology, Graduate School of Medicine, Osaka University, Osaka, Japan

## 2-E-W9-8-O/P

**Exaggerated inflammasome activation by a novel NLRP1 mutant derived from a rare disease with severe liver fibrosis**

Taiki Ando<sup>1,2)</sup>, Akie Maehara<sup>1)</sup>, Tomoaki Ando<sup>1)</sup>, Kumi Izawa<sup>1)</sup>, Ayako Kaitani<sup>1)</sup>, Enzhi Yin<sup>1)</sup>, Takuma Ide<sup>1,3)</sup>, Keiko Maeda<sup>1)</sup>, Nobuhiro Nakano<sup>1)</sup>, Naoto Tamura<sup>2)</sup>, Ko Okumura<sup>1)</sup>, Jiro Kitaura<sup>1)</sup>

Atopy (Allergy) Research Center, Juntendo University Graduate School of Medicine, Tokyo, Japan<sup>1)</sup>, Department of Internal Medicine and Rheumatology, Juntendo University School of Medicine, Tokyo, Japan<sup>2)</sup>, Department of Otorhinolaryngology, Juntendo University School of Medicine, Tokyo, Japan<sup>3)</sup>

## 2-E-W9-12-O/P

**Constitutive RIG-I activation causes IL-23/IL-17 axis skin lesion resembling human psoriasis in mice**

Ahmed S.A Abu Tayeh<sup>1)</sup>, Hiroki Kato<sup>1,2)</sup>, Takashi Fujita<sup>1,2)</sup>

Graduate school of bio studies, Laboratory of Molecular and Cellular Immunology, Kyoto University, Kyoto<sup>1)</sup>, Institute for Frontier Life and Medical Sciences, Laboratory of Molecular Genetics, Kyoto University, Kyoto, Japan<sup>2)</sup>

2-E-W9-11-O/P

**TNFR-associated factor (TRAF) 5 expressed by intestinal epithelial cells promotes NF- $\kappa$ B-mediated inflammation via controlling TRAF2 stability in dextran sulfate sodium-induced colitis**

Hai T Phung<sup>1)</sup>, Hiroyuki Nagashima<sup>1)</sup>, Shuhei Kobayashi<sup>1)</sup>, Tomoaki Machiyama<sup>1)</sup>, Tsuyoshi Sakurai<sup>1)</sup>, Shun-ichi Tayama<sup>1)</sup>, Atsuko Asao<sup>1)</sup>, Yuko Okuyama<sup>1)</sup>, Naoto Ishii<sup>1)</sup>, Takanori So<sup>1,2)</sup>

Department of Microbiology and Immunology, Graduate School of Medicine, Tohoku University, sendai, Japan<sup>1)</sup>, Laboratory of Molecular Cell Biology, Graduate School of Medicine and Pharmaceutical Sciences, University of Toyama, Toyama, Japan<sup>2)</sup>

2-E-W9-15-O/P

**Regulation of intestinal bacteria-mediated diseases by induction of antigen-specific mucosal immunity**

Kosuke Fujimoto<sup>1,2)</sup>, Yunosuke Kawaguchi<sup>1,2,3)</sup>, Satoshi Uematsu<sup>1,2)</sup>

Department of Immunology and Genomics, Osaka City University Graduate School of Medicine, Osaka, Japan<sup>1)</sup>, Division of Innate Immune Regulation, International Research and Development Center for Mucosal Vaccines, The Institute of Medical Science, The University of Tokyo, Tokyo, Japan<sup>2)</sup>, Department of Pediatric Surgery, Graduate School of Medicine, Chiba University, Chiba, Japan<sup>3)</sup>

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**W10 Regulation of B cell development, activation, and function**

15:25 ~ 16:45 Room F

Chairpersons: Wataru Ise, Kei Haniuda

Ultimate goal of humoral immune response is to generate high affinity antibodies to neutralize invading pathogens. For this, development of B cells in bone marrow, activation by antigens in the peripheral lymphoid tissues, and differentiation into memory B cells or plasma cells, need to be tightly regulated, otherwise undesired B cells response might result in autoimmunity or allergy. On the other hand, modification of B cell activation or function would be beneficial to treat such diseases or enhance vaccine efficacy. We thus need to understand the critical checkpoints or mechanisms to ensure high quality humoral response. In this workshop, we will discuss recent progress or novel findings regarding B cell development, activation, or function. We welcome your participation and active discussion. Presentation will be 8 minutes followed by 2 min Q&A.

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**2-F-W10-1-O/P****A single microRNA rescues arrested B cell lineage commitment induced by early B cell factor 1 deficiency**Yuji Miyatake<sup>1)</sup>, Ryutaro Kotaki<sup>2)</sup>, Tomokatsu Ikawa<sup>3)</sup>, Ken-ichi Hirano<sup>4)</sup>, Hiroshi Kawamoto<sup>5)</sup>, Tomohiro Kurosaki<sup>6)</sup>, Katsuto Hozumi<sup>4)</sup>, Ai Kotani<sup>1)</sup>

Department of Hematological Malignancy, Tokai University School of Medicine, Kanagawa, Japan<sup>1)</sup>, Department of Immunology, Duke University School of Medicine, Durham, United States of America<sup>2)</sup>, Department of Immunobiology, Tokyo University of Science, Tokyo, Japan<sup>3)</sup>, Department of Immunology, Tokai University School of Medicine, Kanagawa, Japan<sup>4)</sup>, Institute for Frontier Life and Medical Sciences, Kyoto University, Kyoto, Japan<sup>5)</sup>, Immunology Frontier Research Center, Osaka University, Osaka, Japan<sup>6)</sup>

**2-F-W10-7-O/P****Essential role of late redoxosome in BCR ligation-induced B cell activation**Yang-Yang Feng<sup>1)</sup>, Jun Liu<sup>2)</sup>, Ji-Yang Wang<sup>2)</sup>, Takeshi Tsubata<sup>1)</sup>

Department of Immunology, Medical Research Institute, Tokyo Medical and Dental University, Tokyo, Japan<sup>1)</sup>, Department of Immunology, School of Basic Medical Sciences, Fudan University, Shanghai, China<sup>2)</sup>

**2-F-W10-18-O/P****The quantity of CD40 signaling determines the differentiation of B cells into functionally distinct memory cell subsets**Takuya Koike<sup>1,2)</sup>, Shu Horiuchi<sup>1)</sup>, Daisuke Kitamura<sup>1)</sup>

Division of Molecular Biology, Research Institute for Biomedical Sciences, Tokyo University of Science, Noda, Japan<sup>1)</sup>, Laboratory of Lymphocyte Differentiation, WPI Immunology Frontier Research Center, Osaka University, Suita, Japan<sup>2)</sup>

**2-F-W10-19-O/P****Requirement for memory B cell activation in protection from heterologous influenza virus reinfection**Sarah G Leach<sup>1)</sup>, Ryo Shinnakasu<sup>1)</sup>, Yu Adachi<sup>2)</sup>, Masatoshi Momota<sup>3)</sup>, Chieko Makino-Okamura<sup>4)</sup>, Takuya Yamamoto<sup>3)</sup>, Ken J. Ishii<sup>3)</sup>, Hidehiro Fukuyama<sup>4)</sup>, Yoshimasa Takahashi<sup>2)</sup>, Tomohiro Kurosaki<sup>1,4)</sup>

Osaka University Immunology Frontier Research Center, Osaka, Japan<sup>1)</sup>, National Institute of Infectious Diseases, Tokyo, Japan<sup>2)</sup>, National Institute of Biomedical Innovation, Osaka, Japan<sup>3)</sup>, RIKEN Center for Integrative Medical Sciences, Yokohama, Japan<sup>4)</sup>

**2-F-W10-20-O/P****The protein and glycolipid vaccine induce long-term protection against pneumococcal infection through differentiation of follicular helper NKT cells**Koji Hayashizaki<sup>1,2)</sup>, Shogo Takatsuka<sup>2)</sup>, Shun Kawakubo<sup>2)</sup>, Yasuhiro Kamii<sup>1)</sup>, Yoshimasa Takahashi<sup>3)</sup>, Kazuyoshi Kawakami<sup>4,5)</sup>, Masato Kubo<sup>6)</sup>, Yuki Kinjo<sup>1,2,5)</sup>

Department of Bacteriology, The Jikei University School of Medicine, Tokyo, Japan<sup>1)</sup>, Department of Chemotherapy and Mycoses, NIID, Tokyo, Japan<sup>2)</sup>, Department of Immunology, NIID, Tokyo, Japan<sup>3)</sup>, Department of Medical Microbiology, Mycology and Immunology, Tohoku University Graduate School of Medicine, Sendai, Japan<sup>4)</sup>, Intelligent Network for Infection Control, Tohoku University Graduate School of Medicine, Sendai, Japan<sup>5)</sup>, Laboratory for Cytokine Regulation, RIKEN Center for Integrative Medical Sciences, Yokohama, Japan<sup>6)</sup>

**2-F-W10-21-O/P****Zymosan as a novel adjuvant that directs IgA production in the airway tract**Mizuki Higashiyama<sup>1)</sup>, Kei Haniuda<sup>1)</sup>, Yoshihito Nihei<sup>2)</sup>, Daisuke Kitamura<sup>1)</sup>

Division of Molecular Biology, Research Institute for Biomedical Sciences, Tokyo University of Science, Chiba, Japan<sup>1)</sup>, Department of Nephrology, Juntendo University Faculty of Medicine, Tokyo, Japan<sup>2)</sup>

**2-F-W10-23-O/P****MZB1 promotes the secretion of J chain-containing IgA and is critical for the suppression of gut inflammation**Ermeng Xiong<sup>1)</sup>, Chaoqun Cao<sup>1)</sup>, Qing Min<sup>1)</sup>, Jun Liu<sup>1)</sup>, Nannan Lai<sup>1)</sup>, Ying Wang<sup>1)</sup>, Ryohtaroh Matsumoto<sup>2)</sup>, Daisuke Takahashi<sup>2)</sup>, Koji Hase<sup>2)</sup>, Reiko Shinkura<sup>3)</sup>, Takeshi Tsubata<sup>4)</sup>, Ji-Yang Wang<sup>1,4)</sup>

Department of Immunology, School of Basic Medical Sciences, Fudan University, Shanghai, China<sup>1)</sup>, Division of Biochemistry, Faculty of Pharmacy, Keio University, Tokyo, Japan<sup>2)</sup>, Laboratory of Immunology and Infection Control, Institute of Quantitative Biosciences, University of Tokyo, Tokyo, Japan<sup>3)</sup>, Department of Immunology, Medical Research Institute, Tokyo Medical and Dental University, Tokyo, Japan<sup>4)</sup>

## **TRPM5 Suppresses LPS-Induced Cytokine Production in B Cells**

Taiki Sakaguchi<sup>1,2)</sup>, Ryu Okumura<sup>1,2)</sup>, Kiyoshi Takeda<sup>1,2)</sup>

Laboratory of Immune Regulation, Department of Microbiology and Immunology, Graduate School of Medicine, Osaka University, Suita, Japan<sup>1)</sup>,  
Mucosal Immunology, WPI Immunology Frontier Research Center, Osaka University, Suita, Japan<sup>2)</sup>



Mucosal and skin surface constitutively interact with numerous numbers of microorganisms including bacteria, fungi, parasites, and viruses. Host epithelia-immune cell network create a surface barrier system for the maintenance of homeostasis of gut microbiota and gastrointestinal tracts. Recent studies highlight the role of resident gut microbes and their metabolites in the regulation of the local and systemic immune system. Dysbiosis of gut microbes predisposes to the development of host diseases such as inflammatory bowel diseases, allergy, diabetes, arthritis, skin diseases, and cancer. In this workshop, we will discuss recent findings on molecular and cellular mechanisms of the host-microbe interaction at the mucosal surface and the role of gut microbes and their metabolites in the pathogenesis of the diseases.

## 2-G-W11-1-O/P

**The hydrolysis of commensal bacteria-derived ATP by ENTPD8 suppresses neutrophil-mediated colitis**Haruka Tani<sup>1,2)</sup>, Hisako Kayama<sup>1,2)</sup>, Kiyoshi Takeda<sup>1,2)</sup>Microbiology and Immunology, Graduate school of Medicine, Osaka University, Osaka, Japan<sup>1)</sup>, WPI Immunology Frontier Research Center, Osaka University, Osaka, Japan<sup>2)</sup>

## 2-G-W11-2-O/P

**Cigarette smoke has different effects on inflammatory bowel disease model through mucosal immunity and gut microbiota**Tatsuki Nishioka<sup>1)</sup>, Masahiro Kitabatake<sup>1)</sup>, Noriko Ouji-Sageshima<sup>1)</sup>, Tomoko Nishimura<sup>1,2)</sup>, Toshihiro Ito<sup>1)</sup>Department of Immunology, Nara Medical University, Kashihara, Japan<sup>1)</sup>, Center for Infectious Diseases, Nara Medical University, Kashihara, Japan<sup>2)</sup>

## 2-G-W11-7-O/P

**Identification of stomach ILC2-inducible commensal bacteria belonging S24-7 family of order Bacteroidales**Naoko Satoh-Takayama<sup>1)</sup>, Hiroshi Ohno<sup>2)</sup>Laboratory for Intestinal Ecosystem, Center for Integrative Medical Science, RIKEN, Yokohama, Japan<sup>1)</sup>, Intestinal Microbiota Project, Kanagawa Institute of Industrial Science and Technology, Kawasaki, Japan<sup>2)</sup>

## 2-G-W11-13-O/P

**Microbe-dependent metabolite of  $\alpha$ -linolenic acid alleviates contact hypersensitivity by inhibiting the development of inducible skin-associated lymphoid tissue**Takahiro Nagatake<sup>1)</sup>, Tetsuya Honda<sup>2)</sup>, Azusa Saika<sup>1,3)</sup>, Koji Hosomi<sup>1)</sup>, Ayu Matsunaga<sup>1)</sup>, Kenji Kabashima<sup>2)</sup>, Jun Kunisawa<sup>1,3,4,5,6)</sup>Laboratory of Vaccine Materials, Center for Vaccine and Adjuvant Research and Laboratory of Gut Environmental System, National Institutes of Biomedical Innovation, Health and Nutrition (NIBIOHN), Ibaraki-city, Osaka, Japan<sup>1)</sup>, Department of Dermatology, Kyoto University Graduate School of Medicine, Kyoto, Japan<sup>2)</sup>, Graduate School of Pharmaceutical Sciences, Osaka University, Osaka, Japan<sup>3)</sup>, Department of Microbiology and Immunology, Kobe University Graduate School of Medicine, Kobe, Japan<sup>4)</sup>, International Research and Development Center for Mucosal Vaccines, The Institute of Medical Science, The University of Tokyo, Tokyo, Japan<sup>5)</sup>, Graduate School of Medicine, Graduate School of Dentistry, Osaka University, Osaka, Japan<sup>6)</sup>

## 2-G-W11-14-O/P

**Anti-inflammatory microbial metabolites from linoleic acid suppress obesity-induced liver cancer development**Tomonori Kamiya<sup>1)</sup>, Fumitaku Kamachi<sup>1,2)</sup>, Tatsuya Arai<sup>2)</sup>, Naoko Ohtani<sup>1,2)</sup>Department of Pathophysiology, Osaka city University, Osaka, Japan<sup>1)</sup>, Department of Applied Biological Science, Tokyo University of science, Noda, Japan<sup>2)</sup>

## 2-G-W11-15-O/P

**Intestinal *Prevotella copri* isolated from rheumatoid arthritis patients exacerbates murine models of arthritis**Takuro Nii<sup>1,2)</sup>, Yuichi Maeda<sup>1,2)</sup>, Atsushi Kumanogoh<sup>2)</sup>, Kiyoshi Takeda<sup>1)</sup>Laboratory of Immune Regulation, Graduate school of medicine, Osaka University, Osaka, Japan<sup>1)</sup>, Department of Respiratory Medicine and Clinical Immunology, Graduate school of medicine, Osaka University, Osaka, Japan<sup>2)</sup>

## 2-G-W11-16-O/P

**Development of Nanogel-based Nasal Vaccine against Pneumonia**Rika Nakahashi<sup>1)</sup>, Yoshikazu Yuki<sup>1)</sup>, Kohtaro Fujihashi<sup>1)</sup>, Hiroshi Kiyono<sup>1,2,3,4)</sup>International Research and Development Center for Mucosal Vaccines, The Institute of Medical Science, The University of Tokyo, Tokyo, Japan<sup>1)</sup>, Mucosal Immunology and Allergy Therapeutics, Institute for Global Prominent Research, Graduate School of Medicine, Chiba University, Chiba, Japan<sup>2)</sup>, CU-UCSD Center for Mucosal Immunology, Allergy and Vaccines, Division of Gastroenterology, Department of Medicine, School of Medicine, University of California, San Diego, California, USA<sup>3)</sup>, Division of Mucosal Immunology, IMSUT Distinguished Professor Unit, The Institute of Medical Science, The University of Tokyo, Tokyo, Japan<sup>4)</sup>

T cells in cancer are often prone to become dysfunctional in terms of reduced proliferative capacity, exhausted effector function and overexpression of multiple inhibitory receptors. Several approaches have been attempted to improve the function of effector cells (T or NK cells) in cancer. In this workshop we will discuss not only the hallmarks and mechanisms of T cell dysfunction in cancer, but also therapeutic reactivation of cancer-specific T cells. Of course, we should also discuss new strategies for converting such a dysfunctional state into a functional one for cancer immunotherapy.

2-H-W12-1-O/P

**Sequestration of T cells in bone marrow in the setting of glioblastoma and other intracranial tumors**Shohei Koyama<sup>1</sup>, Atsushi Kumanogoh<sup>1</sup>, Peter Fecci<sup>2</sup>Department of Respiratory Medicine and Clinical Immunology, Osaka University Graduate School of Medicine, Suita, Japan<sup>1</sup>, Department of Neurosurgery, Duke University Medical Center, Durham, USA<sup>2</sup>

2-H-W12-4-O/P

**Resveratrol promotes fatty acid oxidation in CD8<sup>+</sup> T cells and enhances anti-tumor immunity exerted by PD-1 blockade**

Muna M. Al-Habsi, Kenji Chamoto, Rosemary J. Menzies, Tasuku Honjo

Immunology and genomic medicine, graduate school of medicine Kyoto University, Kyoto, Japan

2-H-W12-8-O/P

**Tumor-derived soluble CD155 inhibits DNAM-1-mediated antitumor activity of natural killer cells**Genki Okumura<sup>1,3</sup>, Akiko Iguchi-Manaka<sup>2</sup>, Rikito Murata<sup>1,4</sup>, Yumi Yamashita-Kanemaru<sup>1</sup>, Akira Shibuya<sup>1,5,6</sup>, Kazuko Shibuya<sup>1,6</sup>Faculty of Medicine, Department of Immunology, University of Tsukuba, Ibaraki, Japan<sup>1</sup>, Faculty of Medicine, Department of Breast and Endocrine Surgery, University of Tsukuba, Ibaraki, Japan<sup>2</sup>, Comprehensive Human Sciences, Doctoral Program of Biomedical Sciences, University of Tsukuba, Ibaraki, Japan<sup>3</sup>, School of Integrative and Global Majors, PhD Program in Human Biology, University of Tsukuba, Ibaraki, Japan<sup>4</sup>, Tsukuba Advanced Research Alliance, Life Science Center for Survival Dynamics, University of Tsukuba, Ibaraki, Japan<sup>5</sup>, R&D Center for Innovative Drug Discovery, University of Tsukuba, Ibaraki, Japan<sup>6</sup>

2-H-W12-11-O/P

**Extrinsic and intrinsic inhibition of T cell response by co-inhibitory receptors, TIGIT and CD96**Noriko Yanase<sup>1</sup>, Hiroaki Machiyama<sup>1</sup>, Hiroko Toyota<sup>1</sup>, Masae Furuhashi<sup>1</sup>, Kikumi Hata<sup>1</sup>, Tomohiro Takehara<sup>1,2</sup>, Ei Wakamatsu<sup>1</sup>, Tadashi Yokosuka<sup>1</sup>Department of Immunology, Tokyo Medical University, Tokyo, Japan<sup>1</sup>, Division of Pulmonary Medicine, Department of Internal Medicine, Keio University School of Medicine, Tokyo, Japan<sup>2</sup>

2-H-W12-16-O/P

**Single-cell RNA-sequencing reveals function of IL-17 produced by tumor infiltrating T cells**Koji Nagaoka<sup>1</sup>, Akihiro Hosoi<sup>1</sup>, Kiyomi Taniguchi<sup>3</sup>, Masataka Shirai<sup>3</sup>, Kazuhiro Kakimi<sup>1,2</sup>Department of Immunotherapeutics, The University of Tokyo Hospital, Tokyo, Japan<sup>1</sup>, Cancer Immunology Data Multi-Level Integration Unit, Medical Sciences Innovation Hub Program (MIH), RIKEN, Yokohama, Japan<sup>2</sup>, Research & Development Group, Hitachi, Ltd, Tokyo, Japan<sup>3</sup>

2-H-W12-20-O/P

**Optimization of CAR T cells recognizing malignant mesothelioma specific antigen**Taku Kouro<sup>1</sup>, Kohzoh Imai<sup>2</sup>, Tetsuro Sasada<sup>1</sup>Division of Cancer Immunotherapy, Kanagawa Cancer Center Research Institute, Yokohama, Japan<sup>1</sup>, Kanagawa Cancer Center Research Institute, Yokohama, Japan<sup>2</sup>

2-H-W12-21-O/P

**Screening of tumor-reactive TCRs from TILs of breast cancer patients using a patients' HLA-transduced breast cancer cell line**Hiroshi Hamana<sup>1</sup>, Kiyomi Shitaoka<sup>1</sup>, Kenta Sukegawa<sup>2</sup>, Shiori Saeki<sup>1,2</sup>, Takuya Nagata<sup>2</sup>, Eiji Kobayashi<sup>1</sup>, Tatsuhiko Ozawa<sup>1</sup>, Tsutomu Fujii<sup>2</sup>, Atsushi Muraguchi<sup>1</sup>, Hiroyuki Kishi<sup>1</sup>Department of Immunology, Graduate School of Medicine and Pharmaceutical Sciences, University of Toyama, Toyama, Japan<sup>1</sup>, Department of Surgery and Science, Graduate School of Medicine and Pharmaceutical Sciences, University of Toyama, Toyama, Japan<sup>2</sup>

2-H-W12-25-O/P

**Development of an efficient method to introduce TCR genes into the endogenous TCR locus by genome editing and cassette exchange**Koji Terada<sup>1</sup>, Ryohei Kondo<sup>1</sup>, Seiji Nagano<sup>2</sup>, Kyoko Masuda<sup>2</sup>, Hiroshi Kawamoto<sup>2</sup>, Yasutoshi Agata<sup>1</sup>Division of Molecular Physiology and Chemistry, Department of Biochemistry and Molecular Biology, Shiga University of Medical Science, Otsu, Japan<sup>1</sup>, Laboratory of Immunology, Institute for Frontier Life and Medical Sciences, Kyoto University, Kyoto, Japan<sup>2</sup>

**W13 Rheumatoid arthritis, Sjogren's syndrome and other autoimmune diseases**

13:30 ~ 14:50 Room B

Chairpersons: Isao Matsumoto, Shinsuke Yasuda

Cytokine-targeted therapeutics for rheumatoid arthritis (RA) have been remarkably progressed, although only half of the patients achieve clinical remission. In this session, novel exciting findings will be reported, concerning cellular and molecular mechanisms in RA pathophysiology. Other miscellaneous autoimmune diseases including Sjogren's syndrome, Behcet's disease, Castleman's disease, systemic sclerosis and Grave's disease will be discussed from translational aspects. Mechanisms of cell-specific activation will also be reported. Beyond cytokine suppression, cell-specific treatment would be an emerging strategy in difficult-to-treat RA and other autoimmune/inflammatory diseases. Intense and fruitful discussions are awaited.

3-B-W13-1-O/P

**FCγRIIIA-mediated activation of NK cells by IgG heavy chain complexed with MHC class II molecules**

Tadahiro Suenaga<sup>1,2</sup>, Yuta Shimizu<sup>1,2</sup>, Masako Kohyama<sup>1,2</sup>, Hideki Yorifuji<sup>1,2,3</sup>, Jin Hui<sup>2</sup>, Noriko Arase<sup>1,4</sup>, Hisashi Arase<sup>1,2</sup>  
Research Institute for Microbial Diseases, Osaka University, Osaka, Japan<sup>1</sup>, Immunochemistry, WPI Immunology Frontier Research Center, Osaka University, Osaka, Japan<sup>2</sup>, Department of Respiratory Medicine and Clinical Immunology, Graduate School of Medicine, Osaka University, Osaka, Japan<sup>3</sup>, Department of Dermatology, Graduate School of Medicine, Osaka University, Osaka, Japan<sup>4</sup>

3-B-W13-2-O/P

**Potential involvement of OX40 expressing Tfh cells on the regulation of autoantibody sialylation in experimental and rheumatoid arthritis**

Izumi Kurata, Isao Matsumoto, Ayako Ohyama, Atsumu Osada, Yuya Kondo, Hiroto Tsuboi, Takayuki Sumida  
Division of Rheumatology, Department of Internal Medicine, University of Tsukuba, Tsukuba, Japan

3-B-W13-11-O/P

**TNF-αevoked14-3-3etavia necroptosis-like cell death of macrophages**

Gulzhan Trimova<sup>1</sup>, Kaoru Yamagata<sup>1</sup>, Shigeru Iwata<sup>1</sup>, Shintaro Hirata<sup>2</sup>, Tong Zhang<sup>1</sup>, Fumi Uemura<sup>1</sup>, Minoru Satoh<sup>3</sup>, Norma Biln<sup>4</sup>, Michelle Zaharik<sup>4</sup>, Shingo Nakayamada<sup>1</sup>, Walter P. Maksymowych<sup>5</sup>, Yoshiya Tanaka<sup>1</sup>  
The first department of internal medicine, University of Occupational and Environmental Health Japan, Kitakyushu, Japan<sup>1</sup>, Department of Clinical Immunology and Rheumatology, Hiroshima University Hospital, Hiroshima, Japan<sup>2</sup>, Department of Clinical Nursing, School of Health Sciences, University of Occupational and Environmental Health, Japan, Kitakyushu, Japan<sup>3</sup>, Augurex Life Sciences Corp, 887 Great Northern Way, Suite 125-1, North Vancouver, BC, Canada<sup>4</sup>, Department of Medicine, University of Alberta, Edmonton, AB, Canada<sup>5</sup>

3-B-W13-16-O/P

**Lack of SATB1 leads toSjögren's syndromelike autoimmune manifestations in mice**

Yuriko Tanaka<sup>1</sup>, Akiko Inoue<sup>2</sup>, Taku Kuwabara<sup>1</sup>, Taku Naito<sup>1</sup>, Motonari Kondo<sup>1</sup>  
Department of Molecular and Immunology, Toho University School of Medicine, Tokyo, Japan<sup>1</sup>, Department of Otolaryngology, Toho University School of Medicine, Tokyo, Japan<sup>2</sup>

3-B-W13-19-O/P

**Reactivity of antibodies in the salivary glands of Sjögren's syndrome reveals affinity maturation in disease lesion and the significance of anti-centromere "complex" antibody in disease classification**

Masaru Takeshita, Katsuya Suzuki, Tsutomu Takeuchi  
Division of Rheumatology, Department Internal Medicine, Keio University School of Medicine, Tokyo, Japan

3-B-W13-20-O/P

**Investigation of immune cell subsets involved in Behcet's syndrome by immunophenotyping and gene expression analysis**

Mai Okubo<sup>1</sup>, Shuji Sumitomo<sup>1</sup>, Mineto Ota<sup>1,2</sup>, Yusuke Takeshima<sup>1,2</sup>, Satomi Kobayashi<sup>1</sup>, Yusuke Sugimori<sup>1</sup>, Yasuo Nagafuchi<sup>1</sup>, Yukiko Iwasaki<sup>1</sup>, Hirofumi Shoda<sup>1</sup>, Tomohisa Okamura<sup>1,2</sup>, Kazuhiko Yamamoto<sup>3</sup>, Keishi Fujio<sup>1</sup>  
Department of Allergy and Rheumatology, Graduate School of Medicine, The University of Tokyo, Tokyo, Japan<sup>1</sup>, Department of Functional Genomics and Immunological Diseases, Graduate School of Medicine, The University of Tokyo, Tokyo, Japan<sup>2</sup>, RIKEN Center for Integrative Medical Sciences, Yokohama, Japan<sup>3</sup>

3-B-W13-21-O/P

**Inflammasome drives release of mitochondrial DNA enclosed in extracellular membrane vesicles and propagation of inflammation in Behçet's disease**

Hachiro Konaka, Yasuhiro Kato, Hyota Takamatsu, Atsushi Kumanogoh  
Department of Respiratory Medicine and Clinical Immunology, Osaka University Graduate School of Medicine, Osaka, Japan

**Integrative analysis of proteomics and DNA methylation in orbital fibroblasts from Graves' ophthalmopathy**

Sita Virakul<sup>1,2,3</sup>, Poorichaya Somparn<sup>4</sup>, Trairak Pisitsuk<sup>4</sup>, Peter J. van der Spek<sup>5</sup>, Virgil A.S.H. Dalm<sup>2</sup>, Dion Paridaens<sup>6</sup>, P. Martin van Hagen<sup>2,3,6</sup>, Nattiya Hirankarn<sup>7</sup>, Tanapat Palaga<sup>1</sup>, Willem A. Dik<sup>2</sup>

Department of Microbiology, Faculty of Science, Chulalongkorn University, Bangkok, Thailand<sup>1</sup>, Department of Immunology, Laboratory Medical Immunology, Erasmus MC, Rotterdam, the Netherlands<sup>2</sup>, Department of Internal Medicine, Erasmus MC, Rotterdam, the Netherlands<sup>3</sup>, Center of Excellence in Systems Biology, Research affairs, Faculty of Medicine, Chulalongkorn University, Bangkok, Thailand<sup>4</sup>, Department of Bioinformatics, Erasmus MC, Rotterdam, the Netherlands<sup>5</sup>, Rotterdam Eye Hospital, Rotterdam, the Netherlands<sup>6</sup>, Center of Excellence in Immunology and Immune Mediated Diseases, Department of Microbiology, Faculty of Medicine, Chulalongkorn University, Bangkok, Thailand<sup>7</sup>

Organ-specific autoimmune diseases are caused, at least in part, by the breakdown of immune tolerance against tissue-restricted self-antigens. However, their pathogenesis has not been fully understood yet. In addition to autoreactive T cells and B cells, other immune cells such as dendritic cells, tissue-resident macrophages and monocytes are involved in the pathological condition. Furthermore, genetic factors and environmental factors such as food and smoking affect the disease onset and its progression. Therefore, comprehensive understanding of the mechanisms by which various factors are involved in the pathogenesis of the diseases is essential. In this session, we focus on several types of organ-specific autoimmune disease including multiple sclerosis, inflammatory bowel diseases, hepatitis, pemphigus vulgaris, psoriasis and diabetes. We are aiming to share recent insight into the pathogenic mechanisms of the diseases. We hope active participations and discussion of the audience to deepen our understanding of the pathogenesis of organ-specific autoimmune diseases.

3-B-W14-1-O/P

### Microglia-derived type I interferons promote chronic neuroinflammation by inducing ectopic prolactin production

Chenyang Zhang, Ben JE Raveney, Takashi Yamamura, Shinji Oki

National Institute of Neurosciences, Immunology, National Center of Neurology and Psychiatry, Tokyo, Japan

3-B-W14-2-O/P

### Survival of peripherally derived monocytes in the CNS is crucial for EAE relapse

Nobuhiko Takahashi, Daisuke Kamimura, Kotaro Higuchi, Yuki Tanaka, Masaaki Murakami

Molecular Psychoimmunology, Institute for Genetic Medicine, Graduate School of Medicine, Hokkaido University, Hokkaido, Japan

3-B-W14-9-O/P

### Cigarette smoking facilitates intestinal colonization of oral bacteria in patients with inflammatory bowel disease

Takashi Taida<sup>1)</sup>, Eiji Miyauchi<sup>1)</sup>, Hiroshi Ohno<sup>1,2,3)</sup>Laboratory for Intestinal Ecosystem, RIKEN Center for Integrative Medical Sciences, Yokohama, Japan<sup>1)</sup>, Graduate School of Medical Life Science, Yokohama City University, Yokohama, Japan<sup>2)</sup>, Intestinal Microbiota Project, Kanagawa Institute of Industrial Science and Technology, Kawasaki, Japan<sup>3)</sup>

3-B-W14-20-O/P

### IL-27 is crucial in anti-desmoglein 3 autoantibody production in pemphigus vulgaris mouse model

Aki Kamata<sup>1)</sup>, Hayato Takahashi<sup>1)</sup>, Hiroki Yoshida<sup>2)</sup>, Jun Yamagami<sup>1)</sup>, Masayuki Amagai<sup>1)</sup>Department of Dermatology, Keio University School of Medicine, Tokyo, Japan<sup>1)</sup>, Division of Molecular and Cellular Immunoscience, Department of Biomolecular Sciences, Faculty of Medicine, Saga University, Saga, Japan<sup>2)</sup>

3-B-W14-24-O/P

### Successful treatment of psoriatic inflammation improves hyperglycemia in humans and mice

Mizuyu Odanaka<sup>1)</sup>, Kyoko Ikumi<sup>1,2)</sup>, Hiroaki Shime<sup>1)</sup>, Masaki Imai<sup>1)</sup>, Emi Nishida<sup>2)</sup>, Hiroaki Hemmi<sup>3,4,5)</sup>, Tsuneyasu Kaisho<sup>3,4)</sup>, Akimichi Morita<sup>2)</sup>, Sayuri Yamazaki<sup>1)</sup>Department of Immunology, Nagoya City University Graduate School of Medical Sciences, Nagoya, Japan<sup>1)</sup>, Department of Geriatric and Environmental Dermatology, Nagoya City University Graduate School of Medical Sciences, Nagoya, Japan<sup>2)</sup>, Department of Immunology, Institute of Advanced Medicine, Wakayama Medical University, Wakayama, Japan<sup>3)</sup>, Laboratory for Immune Regulation, World Premier International Research Center Initiative, Immunology Frontier Research Center, Osaka University, Osaka, Japan<sup>4)</sup>, Faculty of Veterinary Medicine, Okayama University of Science, Okayama, Japan<sup>5)</sup>

3-B-W14-25-O/P

### Toll-like receptor 7 is a factor of type 1 diabetes in NOD mice

Ryutaro Fukui<sup>1)</sup>, Atsuo Kanno<sup>1)</sup>, Yuji Motoi<sup>1)</sup>, Yusuke Murakami<sup>1,2)</sup>, Kensuke Miyake<sup>1)</sup>Division of Innate Immunity, The Institute of Medical Science, The University of Tokyo, Tokyo, Japan<sup>1)</sup>, Department of Pharmacotherapy, Musashino University, Tokyo, Japan<sup>2)</sup>

3-B-W14-26-O/P

### Glutaminolysis and mitochondrial functions in plasmablast differentiation and its relevance to the pathogenesis of type 1 diabetes (1DM)

Maiko Hajime<sup>1)</sup>, Shigeru Iwata<sup>1)</sup>, Mingzeng Zhang<sup>1)</sup>, Hiroko Miyata<sup>1)</sup>, Yasuyuki Todoroki<sup>1)</sup>, Shingo Nakayamada<sup>1)</sup>, Kei Sakata<sup>2)</sup>, Kazuo Yamamoto<sup>3)</sup>, Yosuke Okada<sup>1)</sup>, Yoshiya Tanaka<sup>1)</sup>First Department of Internal Medicine, University of Occupational & Environmental Health, Japan, Kitakyushu, Japan<sup>1)</sup>, Mitsubishi Tanabe Pharma, Yokohama, Japan<sup>2)</sup>, Biomedical Research Support Center, Nagasaki University School of Medicine, Nagasaki, Japan<sup>3)</sup>

**Genome scale *in vivo*CRISPR screen identifies a novel gene as a modifier of beta cell vulnerability in type 1 diabetes**

Yuki Ishikawa<sup>1)</sup>, Erica P. Cai<sup>2)</sup>, Wei Zhang<sup>2)</sup>, Nayara C. Leite<sup>3)</sup>, Jian Li<sup>2)</sup>, Badr Kiaf<sup>1)</sup>, Jeniffer Hollister-Lock<sup>2)</sup>, Doug A. Melton<sup>3)</sup>, Peng Yi<sup>2)</sup>, Stephan Kissler<sup>1)</sup>

Section for Immunobiology, Joslin Diabetes Center, Harvard Medical School, Boston, USA<sup>1)</sup>, Section for Islet Cell and Regenerative Biology, Joslin Diabetes Center, Harvard Medical School, Boston, USA<sup>2)</sup>, Department of Stem Cell and Regenerative Biology, Harvard Stem Cell Institute, Harvard University, Cambridge, USA<sup>3)</sup>

This session will be focusing on presentations with novel target, technique, animal model or new modality of intervention as well as prevention of various infectious diseases.

3-C-W15-1-O/P

### **Extracellular vesicles secreted from HBV-infected cells inhibit the eradication of HBV-infected cells in the HBV-infection mouse model**

Masatoshi Kakizaki, Yuichiro Yamamoto, Ai Kotani

Tokai university school of medicine, Kanagawa, Japan

3-C-W15-2-O/P

### **The bacterially cleaved antibody recognition mechanism by immune activation receptor, LILRA2**

Rika Yamazaki<sup>1)</sup>, Atsushi Furukawa<sup>1)</sup>, Koyuki Hirayasu<sup>2)</sup>, Hisashi Arase<sup>3)</sup>, Katsumi Maenaka<sup>1)</sup>

Department of Biomolecular Science, Faculty of Pharmaceutical Science, Hokkaido University, Sapporo, Hokkaido, Japan<sup>1)</sup>, Advanced Preventive Medical Sciences Research Center, Kanazawa University, Kanazawa, Japan<sup>2)</sup>, Research Institute for Microbial Diseases, Osaka University, Suita, Osaka, Japan<sup>3)</sup>

3-C-W15-3-O/P

### **3D High-Resolution Imaging of Experimental Cerebral Malaria**

Julia Matsuo Dapaah<sup>1,2,3)</sup>, Michelle Sue Jann Lee<sup>3)</sup>, Cevayir Coban<sup>1,3)</sup>

Division of Malaria Immunology, Department of Microbiology and Immunology, the Institute of Medical Science, the University of Tokyo, Tokyo, Japan<sup>1)</sup>, Graduate School of Medicine, the University of Tokyo, Tokyo, Japan<sup>2)</sup>, Laboratory of Malaria Immunology, Immunology Frontier Research Institute, Osaka University, Osaka, Japan<sup>3)</sup>, Department of System Pathology for Neurological Disorders, Brain Research Institute, Niigata University, Niigata, Japan<sup>4)</sup>

3-C-W15-4-O/P

### ***Aedes aegypti* salivary protein AgBR1 antibodies protects mice from mosquito-borne zika virus infection by modulating local inflammatory responses**

Ryuta Uraki<sup>1,2)</sup>, Andrew K. Hastings<sup>2)</sup>, Alejandro Marin-Lopez<sup>2)</sup>, Tomokazu Sumida<sup>2)</sup>, Takehiro Takahashi<sup>2)</sup>, Jonathan R. Grover<sup>2)</sup>, Akiko Iwasaki<sup>2,3)</sup>, David A. Hafler<sup>2)</sup>, Ruth R. Montgomery<sup>2)</sup>, Erol Fikrig<sup>2,3)</sup>

Department of Immunology, Nagoya City University, Nagoya, Japan<sup>1)</sup>, Yale University School of Medicine, New Haven, USA<sup>2)</sup>, Howard Hughes Medical Institute, Chevy Chase, USA<sup>3)</sup>

3-C-W15-5-O/P

### **Circulating extracellular vesicle miR-451a, but not human papilloma virus vaccine, plays a crucial role in an autoimmune disorder**

Kana Ishikawa, Yoshimi Fukushima, Hiroyuki Oshiumi

Department of Immunology, Graduate School of Medical Sciences, Faculty of Life Sciences, Kumamoto University, Kumamoto, Japan

3-C-W15-6-O/P

### **Studies of incomplete control of Epstein-Barr virus infection in association with aging by using cynomolgus macaques model**

Emiko Urano, Yoshiko Murakata, Yusuke Tsujimura, Yasuhiro Yasutomi

Tsukuba Primate Research Center, National Institutes of Biomedical Innovation, Health and Nutrition, Tsukuba, Japan

3-C-W15-7-O/P

### **Antibody production and host protection from pneumococcal infection by a novel nanoparticle vaccine**

Ayako Nakahira<sup>1)</sup>, Hiroki Iwaoka<sup>1)</sup>, Ko Sato<sup>2)</sup>, Jun Kasamatsu<sup>2)</sup>, Keiko Ishii<sup>1)</sup>, Kazuyoshi Kawakami<sup>1,2)</sup>

Department of Medical Microbiology, Tohoku University Graduate School of Medicine, Miyagi, Japan<sup>1)</sup>, Department of Intelligent Network for Infection Control, Tohoku University Graduate School of Medicine, Miyagi, Japan<sup>2)</sup>

3-C-W15-8-O/P

### **Murine norovirus infection elicits cross-group neutralizing antibodies against human norovirus**

Kana Hashi, Taishi Onodera, Yoshimasa Takahashi

Department of Immunology, National Institute of Infectious Diseases, Tokyo, Japan



This session will hopefully provoke discussion about the critical cell types, receptors, signaling pathways and/or cytokines for immune-regulation by, and -response to, infectious organisms.

3-C-W16-1-O/P

### The contribution of histone-lysine N-methyltransferase Setdb2 in high mortality of secondary bacterial pneumonia via regulating cytokines and chemokines in macrophages

Atsushi Hara<sup>1)</sup>, Masahiro Kitabatake<sup>1)</sup>, Noriko Ouji-Sageshima<sup>1)</sup>, Shota Sonobe<sup>1)</sup>, Natsuko Imakita<sup>1,2)</sup>, Ryutaro Furukawa<sup>1,2)</sup>, Akihisa Oda<sup>1,3)</sup>, Toshihiro Ito<sup>1)</sup>

Department of Immunology, Nara Medical University, Kashihara, Japan<sup>1)</sup>, Center for Infectious Diseases, Nara Medical University, Kashihara, Japan<sup>2)</sup>, Department of Pediatrics, Nara Medical University, Kashihara, Japan<sup>3)</sup>

3-C-W16-2-O/P

### $\gamma\delta$ T cells regulate humoral immunity against *Plasmodium berghei* infection

Shin-Ichi Inoue<sup>1)</sup>, Mamoru Niikura<sup>2)</sup>, Fumie Kobayashi<sup>3)</sup>, Katsuyuki Yui<sup>1)</sup>

Division of Immunology, Department of Molecular Microbiology and Immunology, Graduate School of Biomedical Sciences, Nagasaki University, Nagasaki, Japan<sup>1)</sup>, Department of Infectious Diseases, Kyorin University School of Medicine, Tokyo, Japan<sup>2)</sup>, Laboratory of Parasitology, School of Life and Environmental Science, Azabu University, Kanagawa, Japan<sup>3)</sup>

3-C-W16-3-O/P

### Critical role of Irgm2 in host defense mechanism against *Toxoplasma gondii*

Ariel Pradipta<sup>1,2)</sup>, Miwa Sasa<sup>2,3)</sup>, Jisu Ma<sup>2,3)</sup>, Youngae Lee<sup>2,3)</sup>, Masahiro Yamamoto<sup>2,3)</sup>

Department Immunoparasitology, Faculty of Medicine, Osaka University, Osaka, Japan<sup>1)</sup>, Department of Immunoparasitology, Research Institute for Microbial Diseases, Osaka University, Osaka, Japan<sup>2)</sup>, Laboratory of Immunoparasitology, WPI Immunology Frontier Research Center, Osaka University, Osaka, Japan<sup>3)</sup>

3-C-W16-4-O/P

### The suppression of host immune responses by mycobacteria through inhibitory C-type lectin receptors

Naoya Nishimura<sup>1,2)</sup>, Shota Torigoe<sup>1)</sup>, Chihiro Motozono<sup>1)</sup>, Satoru Mizuno<sup>3)</sup>, Kazuhiro Matsuo<sup>3)</sup>, Sho Yamasaki<sup>1,4)</sup>

Molecular Immunology, Research Institute of Microbial Diseases, Osaka University, Osaka, Japan<sup>1)</sup>, Department of Medicine and Biosystemic Science, Graduate School of Medical Sciences, Kyushu University, Fukuoka, Japan<sup>2)</sup>, Research and Development Department, Japan BCG Laboratory, Tokyo, Japan<sup>3)</sup>, Molecular Immunology, Immunology Frontier Research Center, Osaka University, Osaka, Japan<sup>4)</sup>

3-C-W16-5-O/P

### Sequential sensing by TLR2 and Mincle directs immature myeloid cells to afford protection against severe invasive group A *Streptococcus* infection

Takayuki Matsumura<sup>1)</sup>, Sho Yamasaki<sup>2)</sup>, Yoshimasa Takahashi<sup>1)</sup>, Manabu Ato<sup>3)</sup>

Department of Immunology, National Institute of Infectious Diseases, Tokyo, Japan<sup>1)</sup>, Division of Molecular Immunology, Research Institute for Microbial Diseases, Osaka University, Osaka, Japan<sup>2)</sup>, Department of Mycobacteriology, Leprosy Research Center, National Institute of Infectious Diseases, Tokyo, Japan<sup>3)</sup>

3-C-W16-6-O/P

### Interstitial-resident memory CD8 T cells sustain frontline epithelial memory in the lung

Shiki Takamura<sup>1)</sup>, Chihiro Motozono<sup>2)</sup>, Satoshi Ueha<sup>3)</sup>, Kazuhiko Matsuo<sup>4)</sup>, Kosuke Miyauchi<sup>5)</sup>, Takashi Nakayama<sup>4)</sup>, Michio Tomura<sup>6)</sup>, Kouji Matsushima<sup>3)</sup>, Masato Kubo<sup>5)</sup>, Masaaki Miyaawa<sup>1)</sup>

Department of Immunology, Kindai University Faculty of Medicine, Osaka, Japan<sup>1)</sup>, Department of Molecular Immunology, Research Institute for Microbial Diseases, Osaka University, Osaka, Japan<sup>2)</sup>, Division of Molecular Regulation of Inflammatory and Immune Diseases, Research Institute for Biomedical Sciences, Tokyo University of Science, Chiba, Japan<sup>3)</sup>, Division of Chemotherapy, Kindai University Faculty of Pharmacy, Osaka, Japan<sup>4)</sup>, Laboratory for Cytokine Regulation, Research Center for Integrative Medical Science, RIKEN Yokohama Institute, Kanagawa, Japan<sup>5)</sup>, Laboratory of Immunology, Faculty of Pharmacy, Osaka Otani University, Osaka, Japan<sup>6)</sup>

3-C-W16-7-O/P

### Chronic interferon-gamma signals impair memory CD8 T cell maintenance

Ruka Setoguchi<sup>1)</sup>, Tadashi Yamamoto<sup>1)</sup>, Shohei Hori<sup>2)</sup>

RIKEN IMS, Yokohama, Japan<sup>1)</sup>, Graduate School of Pharmaceutical Sciences, The University of Tokyo, Tokyo, Japan<sup>2)</sup>

3-C-W16-8-O/P

### *Salmonella* deletes humoral immune memory

Koji Tokoyoda

German Rheumatism Research Centre Berlin (DRFZ), Berlin, Germany

Type 2 immune responses are well known to be crucial for development of allergic disorders. In addition of type 2 cytokines such as IL-4, IL-5 and IL-13, many molecules and immune cells are involved in the development of IgE-dependent and -independent allergic disorders. In this session, we will focus on the molecular mechanisms for development of allergic dermatitis; Bach2 and TSLP in Th cells in acute and chronic contact dermatitis, ILC2 in nickel allergy, M2 macrophages in IgE-dependent chronic dermatitis, LAT1, CARD11 and Clec10a in atopic dermatitis, and neurokinin-N in IL-31-induced itch.

## 3-D-W17-2-O/P

**Critical role of TSLP receptor on CD4 T cells for exacerbation of Th2-type skin inflammation**

Masayuki Kitajima<sup>1)</sup>, Steven F. Ziegler<sup>2,3)</sup>, Harumi Suzuki<sup>1)</sup>

Department of Immunology and Pathology, Research Institute National Center for Global Health and Medicine, Chiba, Japan<sup>1)</sup>, Immunology Program, Benaroya Research Institute, Seattle, USA<sup>2)</sup>, Department of Immunology, University of Washington, Seattle, USA<sup>3)</sup>

## 3-D-W17-4-O/P

**A deficiency of Bach2 in Th cells aggravates allergic inflammation in the skin**

Miyuki Omori-Miyake<sup>1)</sup>, Makoto Kuwahara<sup>2)</sup>, Tomohiro Kurosaki<sup>3)</sup>, Masakatsu Yamashita<sup>1,2)</sup>

Department of Infections and Host Defences, Ehime University Graduate School of Medicine, Ehime, Japan<sup>1)</sup>, Department of Immunology, Ehime University Graduate School of Medicine, Ehime, Japan<sup>2)</sup>, Laboratory of Lymphocyte Differentiation, Immunology Frontier Research Center, Osaka University, Osaka, Japan<sup>3)</sup>

## 3-D-W17-7-O/P

**The role of ILC in metal allergy**

Shota Takigawa<sup>1,2)</sup>, Yasutaka Motomura<sup>1,3)</sup>, Kazuyo Moro<sup>1,3)</sup>

Laboratory for Innate Immune Systems, IMS, RIKEN, Yokohama, Japan<sup>1)</sup>, Department of Medical Life Science, Yokohama City University, Yokohama, Japan<sup>2)</sup>, Laboratory for Innate Immune Systems, Department of Microbiology and Immunology, Graduate School of Medicine, Osaka University, Suita, Japan<sup>3)</sup>

## 3-D-W17-10-O/P

**Suppressive effects of LAT1-specific inhibitor on T cell-mediated allergic skin inflammation**

Keitaro Hayashi<sup>1)</sup>, Kunie Matsuoka<sup>2)</sup>, Takachika Hiroi<sup>3)</sup>, Osamu Kaminuma<sup>3,4,5)</sup>

Department of Pharmacology and Toxicology, Dokkyo Medical University School of Medicine, Shimotsuga, Japan<sup>1)</sup>, Mammalian Genetics Project, Tokyo Metropolitan Institute of Medical Science, Tokyo, Japan<sup>2)</sup>, Allergy and Immunology Project, Tokyo Metropolitan Institute of Medical Science, Tokyo, Japan<sup>3)</sup>, Center for Life Science Research, University of Yamanashi, Yamanashi, Japan<sup>4)</sup>, Department of Disease Model, Hiroshima University, Hiroshima, Japan<sup>5)</sup>

## 3-D-W17-11-O/P

**Hypomorphic *CARD11* mutation developed inflammatory atopic disorders in mice**

Yusuke Nomoto<sup>1)</sup>, Shinsuke Yasukawa<sup>2)</sup>, Ei'ichi Iizasa<sup>3)</sup>, Shin-Ei Matsumoto<sup>3)</sup>, Masutaka Furue<sup>2)</sup>, Takuro Kanekura<sup>1)</sup>, Hiromitsu Hara<sup>3)</sup>

Department of Dermatology, Kagoshima University, Kagoshima, Japan<sup>1)</sup>, The Department of Dermatology, Kyusyu University, Fukuoka, Japan<sup>2)</sup>, The Department of Immunology, Kagoshima University, Kagoshima, Japan<sup>3)</sup>

## 3-D-W17-12-O/P

**Clec10a regulates mite-induced dermatitis**

Kazumasa Kanemaru<sup>1,7)</sup>, Satoko Tahara-Hanaoka<sup>1,2,7)</sup>, Kaori Denda-Nagai<sup>4)</sup>, Tatsuro Irimura<sup>4)</sup>, Satoru Takahashi<sup>2,3,5,6)</sup>, Kazuko Shibuya<sup>1,7)</sup>, Akira Shibuya<sup>1,2,7)</sup>

Department of Immunology, Faculty of Medicine, University of Tsukuba, Tsukuba, Ibaraki, Japan<sup>1)</sup>, Life Science Center for Survival Dynamics, Tsukuba Advanced Research Alliance (TARA), University of Tsukuba, Tsukuba, Ibaraki, Japan<sup>2)</sup>, Laboratory Animal Resource Center, University of Tsukuba, Tsukuba, Ibaraki, Japan<sup>3)</sup>, Division of Glycobiologics, Intractable Disease Research Center, Juntendo University School of Medicine, Bunkyo-ku, Tokyo, Japan<sup>4)</sup>, Department of Anatomy and Embryology, Faculty of Medicine, University of Tsukuba, Tsukuba, Ibaraki, Japan<sup>5)</sup>, International Institute for Integrative Sleep Medicine (WPI-IIS), University of Tsukuba, Tsukuba, Ibaraki, Japan<sup>6)</sup>, R&D Center for Innovative Drug Discovery, University of Tsukuba, Tsukuba, Ibaraki, Japan<sup>7)</sup>

## 3-D-W17-16-O/P

**Monocyte-derived M2-like macrophages induced by activated basophils dampen IL-1 $\alpha$ -mediated aggravation of allergic inflammation**

Kensuke Miyake<sup>1)</sup>, Soichiro Yoshikawa<sup>2)</sup>, Yoshinori Yamanishi<sup>1)</sup>, Hajime Karasuyama<sup>1)</sup>

Department of Immune Regulation, Tokyo Medical and Dental University (TMDU), Bunkyo-ku, Japan<sup>1)</sup>, Department of Cell Physiology, Okayama University Graduate School of Medicine, Dentistry and Pharmaceutical Sciences, Okayama, Japan<sup>2)</sup>

**Identification of the molecule required for transmission of IL-31–induced itch sensation in the spinal cord**

Daiji Sakata, Yshinori Fukui

Division of Immunogenetics, Medical Institute of Bioregulation, Kyushu University, Fukuoka, Japan

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## W18 Allergy-2: Anaphylaxis, airway inflammation/asthma and others (except dermatitis)

15:05 ~ 16:25 Room D

Chairpersons: Hiroshi Nakajima, Jiro Kitaura

Allergic diseases, including anaphylaxis and airway inflammation/asthma, are caused by allergen-induced unfavorable adaptive and/or innate immune responses involving a variety of immune cells (e.g., Th2 cells, Tfh cells, B cells, mast cells, basophils, dendritic cells, and ILC2). First, we focus on the regulatory mechanisms of antigen-specific high-affinity or natural IgE production and of mast cell or basophil activation in IgE-dependent anaphylaxis. Next, we discuss about the immunoregulatory roles of specific molecules such as Exophilin-5, TSLP receptor, IL-7, and Regnase-1 in airway inflammation.

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3-D-W18-15-O/P

### The importance of T<sub>H</sub>2 producing IL-13 in IgE response

Taiki Yajima<sup>1</sup>, Yasuyo Harada<sup>1</sup>, Masato Kubo<sup>1,2</sup>

Division of Molecular Pathology, Research Institute for Biological Science, Tokyo University of Science, Chiba, Japan<sup>1</sup>, Laboratory for Signal Network, Research Center for Allergy and Immunology, RIKEN Yokohama Institute, Yokohama, Japan<sup>2</sup>

3-D-W18-17-O/P

### Mast cell- and IgE-dependent anaphylaxis is regulated by the interaction between inhibitory LMIR3/activating LMIR7 and specific lipids

Kumi Izawa<sup>1</sup>, Ayako Kaitani<sup>1</sup>, Tomoaki Ando<sup>1</sup>, Akie Maehara<sup>1</sup>, Takuma Ide<sup>1,2</sup>, Moe Matsuzawa<sup>1,3</sup>, Saaya Fukase<sup>1,3</sup>, Anna Kamei<sup>1</sup>, Taiki Ando<sup>1,4</sup>, Nobuhiro Nakano<sup>1</sup>, Ko Okumura<sup>1</sup>, Jiro Kitaura<sup>1</sup>

Atopy (Allergy) Research Center, Juntendo University School of Medicine, Tokyo, Japan<sup>1</sup>, Department of Otorhinolaryngology, Juntendo University Graduate School of Medicine, Tokyo, Japan<sup>2</sup>, Department of Ophthalmology, Juntendo University Graduate School of Medicine, Tokyo, Japan<sup>3</sup>, Department of Internal Medicine and Rheumatology, Juntendo University Graduate School of Medicine, Tokyo, Japan<sup>4</sup>

3-D-W18-20-O/P

### Selective suppression of oral allergen-induced anaphylaxis by Allergin-1

Yu-hsien Lin<sup>1,2</sup>, Satoko Tahara-Hanaoka<sup>2,3,4</sup>, Soichiro Yoshikawa<sup>5</sup>, Shiro Shibayama<sup>6</sup>, Hajime Karasuyama<sup>5</sup>, Akira Shibuya<sup>2,3,4</sup>

Ph.D. Program in Human Biology, School of Integrative and Global Majors, University of Tsukuba, Tsukuba city, Japan<sup>1</sup>, Department of Immunology, Faculty of Medicine, Tsukuba city, Japan<sup>2</sup>, Life Science Center for Survival Dynamics, Tsukuba Advanced Research Alliance (TARA), Tsukuba city, Japan<sup>3</sup>, R&D center for Innovative Drug Discovery, University of Tsukuba, Tsukuba city, Japan<sup>4</sup>, Department of Immune Regulation, Tokyo Medical and Dental University, Tokyo, Japan<sup>5</sup>, Research Center of Immunology, Tsukuba Institute, Ono Pharmaceutical Co. Ltd, Tsukuba city, Japan<sup>6</sup>

3-D-W18-23-O/P

### Natural IgE production requires cognate iNKT-B interaction via CD1d

Akihiko Kito<sup>1</sup>, Rintaro Shibuya<sup>1</sup>, Kenji Kabashima<sup>1,2</sup>

Department of Dermatology, Kyoto University Graduate School of Medicine, Kyoto, Japan<sup>1</sup>, Singapore Immunology Network (SIgN) and Skin Research Institute of Singapore (SRIS), Agency for Science, Technology and Research (A\*STAR), Singapore, Singapore<sup>2</sup>

3-D-W18-27-O/P

### I $\kappa$ B kinase complex-dependent Regnase-1 degradation is crucial for interleukin-33- and interleukin-25-mediated group-2 innate lymphoid cell activation

Kazufumi Matsushita<sup>1</sup>, Hiroki Tanaka<sup>2</sup>, Koubun Yasuda<sup>1</sup>, Shizuo Akira<sup>2</sup>, Etsushi Kuroda<sup>1</sup>

Department of Immunology, Hyogo College of Medicine, Nishinomiya, Japan<sup>1</sup>, Department of Host Defense, IFRc Osaka University, Osaka, Japan<sup>2</sup>

3-D-W18-33-O/P

### A novel regulatory role of the Rab27 effector exophilin5 in allergic airway inflammation

Katsuhide Okunishi<sup>1</sup>, Hao Wang<sup>1</sup>, Maho Suzukawa<sup>2</sup>, Susumu Nakae<sup>3</sup>, Tetsuro Izumi<sup>1</sup>

Laboratory of Molecular Endocrinology and Metabolism, Department of Molecular Medicine, Institute for Molecular and Cellular Regulation, Gunma University, Maebashi, Japan<sup>1</sup>, National Hospital Organization Tokyo National Hospital, Tokyo, Japan<sup>2</sup>, Laboratory of Systems Biology, Center for Experimental Medicine and Systems Biology, The Institute of Medical Science, The University of Tokyo, Tokyo, Japan<sup>3</sup>

3-D-W18-37-O/P

### Role of local IL-7 in maintenance of lung ILC2s

Daichi Takami, Shinya Abe, Akihiro Shimba, Takuma Asahi, Yuanbo Zhu, Aki Ejima, Shizue Tani-ichi, Takahiro Hara, Guangwei Cui, Koichi Ikuta

Immune Regulation, Institute for Frontier Life and Medical Sciences, Kyoto University, Kyoto, Japan

**Targeted deletion of TSLP receptor reveals cellular mechanisms underlying acute versus chronic type 2 inflammation**

Hiroki Kabata<sup>1,2)</sup>, David Artis<sup>2)</sup>

Pulmonary Medicine, Department of Medicine, Keio University School of Medicine, Tokyo, Japan<sup>1)</sup>, JILL Roberts Institute for Research in Inflammatory Bowel Disease, Department of Microbiology and Immunology, Weill Cornell Medicine, New York, USA<sup>2)</sup>

Innate lymphoid cells include all ILC subsets (NK cell, ILC1, ILC2, ILC3) and innate T lymphocytes (NKT cell, MAIT cell, gdT cell). Recent studies have been shown that ILC subsets contribute to tissue homeostasis as well as defense against pathogens and inflammatory diseases. In addition to ILC research, studies on innate T lymphocytes have also contributed to our understanding of the role of innate lymphocytes in whole-body homeostasis and in the pathophysiology of various diseases.

The aim of this WS is to provide an interactive platform for the exchange of new concepts, ideas and data between immunologists interested in ILCs and innate lymphocytes at large.

3-E-W19-1-O/P

**Protective role of type 1 innate lymphoid cells in acute liver injury**Tsukasa Nabekura<sup>1,2)</sup>, Akira Shibuya<sup>1,2)</sup>Life Science Center for Survival Dynamics, Tsukuba Advanced Research Alliance, University of Tsukuba, Tsukuba, Japan<sup>1)</sup>, R&D Center for Innovative Drug Discovery, University of Tsukuba, Tsukuba, Japan<sup>2)</sup>

3-E-W19-5-O/P

**The role of group 2 innate lymphoid cells(ILC2) in pulmonary fibrosis**Natsuko Otaki<sup>1,2)</sup>, Yasutaka Motomura<sup>2,3)</sup>, Tommy Walter Terootea<sup>4)</sup>, Tom Kelly<sup>4)</sup>, Akiko Minoda<sup>4)</sup>, Hideya Kitamura<sup>5)</sup>, Takashi Ogura<sup>5)</sup>, Koichiro Asano<sup>6)</sup>, Kazuyo Moro<sup>2,3)</sup>Department of Microbiology and Immunology, Keio University School of Medicine, Tokyo, Japan<sup>1)</sup>, Laboratory for Innate Immune Systems, IMS, RIKEN, Yokohama, Japan<sup>2)</sup>, Laboratory for Innate Immune Systems, Department of Microbiology and Immunology, Graduate School of Medicine, Osaka University, Suita, Japan<sup>3)</sup>, Epigenome Technology Exploration Unit, IMS, RIKEN, Yokohama, Japan<sup>4)</sup>, Kanagawa Cardiovascular and Respiratory Center, Yokohama, Japan<sup>5)</sup>, Division of Pulmonary Medicine, Department of medicine, Tokai University School of Medicine, Isehara, Japan<sup>6)</sup>

3-E-W19-7-O/P

**IQGAP1 regulates ILC2 apoptosis in the lung**Shun-ichi Tayama<sup>1)</sup>, Yuko Okuyama<sup>1)</sup>, Phung The Hai<sup>1)</sup>, Takeshi Kawabe<sup>1)</sup>, Takanori So<sup>2)</sup>, Naoto Ishii<sup>1)</sup>Graduate School of Medicine, Microbiology and Immunology, Tohoku university, Sendai, Japan<sup>1)</sup>, Toyama University, Toyama, Japan<sup>2)</sup>

3-E-W19-14-O/P

**The interaction of NKT cell and macrophage has regulatory function in obesity**

Masashi Satoh, Kazuya Iwabuchi

Department of Immunology, Kitasato University School of Medicine, Sagami-hara, Japan

3-E-W19-15-O/P

**Thymic IL-15 niche controls anti-tumor immunity by regulating a novel subset of iNKT cells**

Guangwei Cui, Koichi Ikuta

Laboratory of Immune Regulation, Institute for Frontier Life and Medical Sciences, Kyoto University, Kyoto, Japan

3-E-W19-16-O/P

**Capturing and identification of MR1-binding molecules using unfolded MR1 protein**Chihiro Motozono<sup>1,2)</sup>, Hendra Saputra Ismanto<sup>1)</sup>, Kensuke Shibata<sup>2,3,4)</sup>, Sho Yamasaki<sup>1,2)</sup>Department of Molecular Immunology, Research Institute for Microbial Diseases, Osaka University, Osaka, Japan<sup>1)</sup>, Laboratory of Molecular Immunology, Immunology Frontier Research Center, Osaka University, Osaka, Japan<sup>2)</sup>, Department of Microbiology and Immunology, Graduate School of Medicine, Yamaguchi University, Ube, Japan<sup>3)</sup>, Department of Ocular Pathology and Imaging Science, Graduate School of Medical Sciences, Kyushu University, Fukuoka, Japan<sup>4)</sup>

3-E-W19-17-O/P

**Involvement of CD96 in imiquimod (IMQ)-induced psoriasis through upregulation of IL-17 production by  $\gamma\delta$ T cells**Kyoko Oh-oka<sup>1,2)</sup>, Akira Shibuya<sup>1,2,3)</sup>, Kazuko Shibuya<sup>1,2,3)</sup>Department of Immunology, Faculty of medicine, University of Tsukuba, Ibaraki, Japan<sup>1)</sup>, Life Science Center for Survival Dynamics, Tsukuba Advanced Research Alliance (TARA), University of Tsukuba, Ibaraki, Japan<sup>2)</sup>, R&D Center for Innovative Drug Discovery, University of Tsukuba, Ibaraki, Japan<sup>3)</sup>

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**W20      Dendritic cells, macrophages, granulocytes : Immune regulation      13:30 ~ 14:50      Room F**

Chairpersons: Tsuneyasu Kaisho, Kenichi Asano

Dendritic cells and macrophages are the key components of first line of defense system against pathogen infection. They express sensors/receptors that recognize invaded pathogen, and regulate inflammatory cytokine secretion as well as antigen presentation that are crucial for the subsequent induction of adaptive immune responses. This session discuss about the recent progress in the understanding of immune regulation mediated by dendritic cells and macrophages.

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3-F-W20-1-O/P

**Lysosomal protein Lamtor1 (p18) regulates dendritic cell trafficking by actomyosin contraction**

Takeshi Nakatani<sup>1)</sup>, Park Jeong Hoon<sup>2)</sup>, Kohei Tsujimoto<sup>1)</sup>, Hyota Takamatsu<sup>1)</sup>, Atsushi Kumanogoh<sup>1)</sup>  
respiratory medicine and clinical immunology, Osaka university school of medicine, Osaka, Japan<sup>1)</sup>, Osaka Police Hospital, Osaka, Japan<sup>2)</sup>

3-F-W20-2-O/P

**Influences of immune activation on the transport of skin self-antigens to regional lymph nodes**

Miya Yoshino, Akihiko Murata, Shin-Ichi Hayashi  
Division of Immunology, School of Life Science, Faculty of Medicine, Tottori University, Yonago, Japan

3-F-W20-3-O/P

**Novel DC targeting by allogenic T-cells for multifocal prophylactic antibody production**

Hisashi Ueta<sup>1)</sup>, Yusuke Kitazawa<sup>1)</sup>, Yasushi Sawanobori<sup>1)</sup>, Tomoya Kataikai<sup>2)</sup>, Satoshi Ueha<sup>3)</sup>, Kouji Matsushima<sup>3)</sup>, Nobuko Tokuda<sup>1)</sup>, Kenjiro Matsuno<sup>1)</sup>  
Department of Anatomy (Macro), Dokkyo Medical University, Mibu, Japan<sup>1)</sup>, Department of Immunology, Niigata University, Niigata, Japan<sup>2)</sup>, Division of Molecular Regulation of Inflammatory and Immune Diseases, Research Institute for Biomedical Sciences, Tokyo University of Science, Chiba, Japan<sup>3)</sup>

3-F-W20-4-O/P

**NR4A3 plays a critical role in the induction of adaptive immunity via the DC migration from the periphery to second lymphoid organs**

Takuya Yashiro<sup>1)</sup>, Shiori Nakano<sup>1)</sup>, Akihiko Yoshimura<sup>2)</sup>, Chiharu Nishiyama<sup>1)</sup>  
Department of Biological Science and Technology, Tokyo University of Science, Tokyo, Japan<sup>1)</sup>, Department of Microbiology and Immunology, Keio University School of Medicine, Tokyo, Japan<sup>2)</sup>

3-F-W20-5-O/P

**Identification of a unique macrophage subset induced by repeated antigen painting in sublingual mucosa to regulate T cell tolerance**

Yue Yang, Shigenori Nagai, Miyuki Azuma  
Molecular Immunology, Tokyo Medical and Dental University, Tokyo, Japan

3-F-W20-6-O/P

**H3K4me3 Profile and the Effect of Lipopolysaccharide/Immune Complex-Activated Macrophages on Endotoxemia**

Tanapat Palaga<sup>1,2)</sup>, Vichaya Ruenjaiman<sup>1,2)</sup>, Yu-Wei Leu<sup>3)</sup>, Asada Leelahavanichkul<sup>2)</sup>, Patipark Kueanjinda<sup>4)</sup>  
Interdisciplinary Graduate Program in Medical Microbiology, Microbiology, Faculty of Science, Graduate School, Chulalongkorn University, Bangkok, Thailand<sup>1)</sup>, Center of Excellence in Immunology and Immune-Mediated Diseases, Chulalongkorn University, Bangkok, Thailand<sup>2)</sup>, Department of Life Science, National Chung Cheng University, Chiayi, Taiwan<sup>3)</sup>, Institute for Biomedical Sciences, Interdisciplinary Cluster for Cutting Edge Research, Shinshu University, Kamiina, Japan<sup>4)</sup>

3-F-W20-7-O/P

**Negative regulation of IL-10 transcription by calcium signaling in dendritic cells**

Xiuyuan Lu<sup>1)</sup>, Masatsugu Oh-hora<sup>2)</sup>, Sho Yamasaki<sup>1,3,4)</sup>  
Laboratory of Molecular Immunology, Immunology Frontier Research Center, Osaka University, Suita, Japan<sup>1)</sup>, Department of Biochemistry, Graduate School of Medicine, Juntendo University, Tokyo, Japan<sup>2)</sup>, Department of Molecular Immunology, Research Institute for Microbial Diseases, Osaka University, Suita, Japan<sup>3)</sup>, Division of Molecular Immunology, Medical Institute of Bioregulation, Kyushu University, Fukuoka, Japan<sup>4)</sup>

3-F-W20-8-O/P

**Fcγ-coupled receptor members induce different cytokines by altering the kinetics of Fcγ signaling**

Miyuki Watanabe<sup>1)</sup>, Sho Yamasaki<sup>1,2)</sup>  
Department of Molecular Immunology, Research Institute for Microbial Diseases, Osaka University, Osaka, Japan<sup>1)</sup>, Department of Molecular Immunology, Immunology Frontier Research Center, Osaka University, Osaka, Japan<sup>2)</sup>



## W21 Dendritic cells, macrophages, granulocytes : Ontogeny and disease perspectives

15:05 ~ 16:25 Room F

Chairpersons: Nobuyuki Onai, Yasutaka Okabe

Dendritic cells and macrophages are highly heterogeneous populations in terms of their phenotypes and functions. This session will discuss about differentiation, polarization, and activation of dendritic cell and macrophage lineages, and how these cells acquire heterogeneous phenotypes that fulfill immune regulation and homeostasis. Additionally, we discuss about the pathogenesis caused by the exaggeration or dysregulation of the functions of these cell types.

3-F-W21-1-O/P

### Chromatin architecture dynamics during dendritic cell development *in vivo*

Daisuke Kurotaki<sup>1)</sup>, Pedro P. Rocha<sup>2)</sup>, Kairong Cui<sup>3)</sup>, Jun Nakabayashi<sup>4)</sup>, Keita Saeki<sup>5)</sup>, Keji Zhao<sup>3)</sup>, Keiko Ozato<sup>5)</sup>, Tomohiko Tamura<sup>1,4)</sup>

Department of Immunology, Yokohama City University Graduate School of Medicine, Yokohama, Japan<sup>1)</sup>, Unit on Genome Structure and Regulation, NICHD, NIH, Bethesda, USA<sup>2)</sup>, Laboratory of Epigenome Biology, NHLBI, NIH, Bethesda, USA<sup>3)</sup>, Advanced Medical Research Center, Yokohama City University, Yokohama, Japan<sup>4)</sup>, Program in Genomics of Differentiation, NICHD, NIH, Bethesda, USA<sup>5)</sup>

3-F-W21-2-O/P

### Smad3 determines conventional versus plasmacytoid dendritic cell fates

Jeong-Hwan Yoon<sup>1,2)</sup>, Eunjin Bae<sup>1,2)</sup>, Jin Soo Han<sup>4)</sup>, Seok Hee Park<sup>5)</sup>, Susumu Nakae<sup>6)</sup>, In-Kyu Lee<sup>1)</sup>, Ji Hyeon Ju<sup>7)</sup>, Isao Matsumoto<sup>8)</sup>, Takayuki Sumida<sup>8)</sup>, Mizuko Mamura<sup>1,2,3)</sup>

Bio-medical Research Institute, Kyungpook National University Hospital, Daegu, Korea<sup>1)</sup>, Department of Molecular Pathology, Tokyo Medical University, Tokyo, Japan<sup>2)</sup>, Diversity Promotion Center, Tokyo Medical University, Tokyo, Japan<sup>3)</sup>, Department of Laboratory Animal Medicine, College of Veterinary Medicine, Konkuk University, Seoul, Korea<sup>4)</sup>, Department of Biological Sciences, Sungkyunkwan University, Suwon, Korea<sup>5)</sup>, Laboratory of Systems Biology, Center for Experimental Medicine and Systems Biology, The Institute of Medical Science, The University of Tokyo, Tokyo, Japan<sup>6)</sup>, Department of Rheumatology, Catholic University of Korea, Seoul St. Mary Hospital, Seoul, Korea<sup>7)</sup>, Department of Internal Medicine, University of Tsukuba, Tsukuba, Japan<sup>8)</sup>

3-F-W21-3-O/P

### High dimensional single cell analysis reveals unexpected immune cell types, and loss of motility of alveolar macrophages regulated by PPAR $\gamma$ in chronic obstructive respiratory disease

Wataru Fujii<sup>1,2)</sup>, Kevin Bassler<sup>2)</sup>, Theodoros Kapellos<sup>2)</sup>, Anna C. Aschenbrenner<sup>2,3)</sup>, Kristian Händler<sup>4)</sup>, Carmen Pizarro<sup>5)</sup>, Dirk Skowasch<sup>5)</sup>, Joachim L. Schultze<sup>2,4)</sup>

Inflammation and Immunology, Kyoto Prefectural University of Medicine, Kyoto, Japan<sup>1)</sup>, Life & Medical Sciences (LIMES) Institute, Genomics and Immunoregulation, University of Bonn, Bonn, Germany<sup>2)</sup>, Department of Internal Medicine and Radboud Center for Infectious Diseases (RCI), Radboud University Medical Center, Nijmegen, The Netherlands<sup>3)</sup>, German Center for Neurodegenerative Diseases, Platform for Single Cell Genomics and Epigenomics, University of Bonn, Bonn, Germany<sup>4)</sup>, Cardiology, Pneumology and Angiology, Department of Internal Medicine II, University Hospital Bonn, Bonn, Germany<sup>5)</sup>

3-F-W21-4-O/P

### Defective dendritic cell and monocyte development in proteasome subunit mutant miceDefective dendritic cell and monocyte development in proteasome subunit mutant mice

Hiroaki Hemmi<sup>1,2)</sup>, Takashi Orimo<sup>1)</sup>, Izumi Sasaki<sup>1)</sup>, Takashi Kato<sup>1)</sup>, Yuri Fukuda-Ohta<sup>1)</sup>, Noriko Kinjo<sup>3)</sup>, Satoru Hamada<sup>3)</sup>, Akira Kinoshita<sup>4)</sup>, Koh-Ichiro Yoshiura<sup>4)</sup>, Hidenori Ohnishi<sup>5)</sup>, Nobuo Kanazawa<sup>6)</sup>, Tsuneyasu Kaisho<sup>1)</sup>

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3-F-W21-5-O/P

### SLC15A3 inhibits autophagy in macrophage and dendritic cells

Dat Nguyen-Tien<sup>1)</sup>, Toshihiko Kobayashi<sup>1)</sup>, Naoshi Dohmae<sup>2)</sup>, Tomohiko Taguchi<sup>3)</sup>, Noriko Sorimachi<sup>1)</sup>

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3-F-W21-6-O/P

### A chemokine signal amplifier FROUNT regulates tumor cell-mediated macrophage activation and migration to tumor sites

Etsuko Toda<sup>1,2)</sup>, Yuya Terashima<sup>2)</sup>, Kouji Matsushima<sup>2)</sup>

Department of Analytic Human Pathology, Nippon Medical School, Tokyo, Japan<sup>1)</sup>, Division of Molecular Regulation of Inflammatory and Immune Diseases, Research Institute for Biomedical Sciences (RIBS), Tokyo University of Science, Chiba, Japan<sup>2)</sup>

3-F-W21-7-O/P

### **Lnk/SH2B3 contributes to the initiation and severity of STZ-induced diabetes**

Mari Tenno, Satoshi Takaki

Immune Regulation, Research Institute National Center for Global Health and Medicine, Chiba, Japan

3-F-W21-8-O/P

### **Neutrophils convert into atypical Ly6G<sup>+</sup>SiglecF<sup>+</sup> cells with neurosupportive properties in olfactory neuroepithelium**

Kei Ogawa<sup>1,2)</sup>, Kenichi Asano<sup>1)</sup>, Satoshi Yotsumoto<sup>1)</sup>, Tsuyoshi Yamane<sup>3,4)</sup>, Makoto Arita<sup>3,5,6)</sup>, Yoshihiro Hayashi<sup>7)</sup>, Hironori Harada<sup>7)</sup>, Hidehiro Fukuyama<sup>8)</sup>, Masato Tanaka<sup>1)</sup>

Laboratory of Immune Regulation, Tokyo University of Pharmacy and Life Sciences School of Life Sciences, Tokyo, Japan<sup>1)</sup>, Department of Otolaryngology - Head and Neck Surgery, University of Tokyo Graduate School of Medicine, Tokyo, Japan<sup>2)</sup>, Laboratory for Metabolomics, RIKEN Center for Integrative Medical Sciences, Kanagawa, Japan<sup>3)</sup>, Division of Gastroenterology & Hepatology, Department of Internal Medicine, Keio University School of Medicine, Tokyo, Japan<sup>4)</sup>, Cellular and Molecular Epigenetics Laboratory, Yokohama City University Graduate School of Medical Life Science, Kanagawa, Japan<sup>5)</sup>, Division of Physiological Chemistry and Metabolism, Keio University Graduate School of Pharmaceutical Sciences, Tokyo, Japan<sup>6)</sup>, Laboratory of Oncology, Tokyo University of Pharmacy and Life Sciences School of Life Sciences, Tokyo, Japan<sup>7)</sup>, Laboratory for Lymphocyte Differentiation, RIKEN Center for Integrative Medical Sciences, Kanagawa, Japan<sup>8)</sup>

Development of front-line defenses in mucosal and skin tissues has a substantial impact on immune system to regulate the physiological functions of various organs. In these peripheral tissues, both hematopoietic cells including innate immune cells, antigen presenting cells, T and B cells and non-hematopoietic cells such as epithelial cells, stromal cells and fibroblasts cooperatively interact and function as a barrier system for controlling robust immune responses and maintaining homeostasis of tissue interface. Recent studies also highlight the important role of the mucosal/skin resident immunity that influence the function of other tissues/organs mediated by the recruitment of unique populations of immune cells. This workshop aims to discuss recent findings on molecular and cellular machineries of the barrier immunity against invading microbes, environmental factors or external stimuli at the mucosal/skin surface.

3-G-W22-1-O/P

**The impact of fasting on immune cell dynamics and mucosal immune response**

Motoyoshi Nagai<sup>1,2)</sup>, Ryotaro Noguchi<sup>1,2)</sup>, Kouhei Koshida<sup>1)</sup>, Daisuke Takahashi<sup>1)</sup>, Yuki I Kawamura<sup>2)</sup>, Taeko Dohi<sup>1,2)</sup>, Koji Hase<sup>1)</sup>

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3-G-W22-3-O/P

**ROR $\alpha$  is crucial for the survival of IgA<sup>+</sup> plasma cells but not B220<sup>+</sup> IgA<sup>+</sup> cells**

Masaaki Hashiguchi, Yuji Kashiwakura, Yumiko Kanno, Hidefumi Kojima

Department of Immunology, Dokkyo Medical University School of Medicine, Mibu, Japan

3-G-W22-4-O/P

**Age-dependent decrease in induction of regulatory T cells is associated with decreased expression of RALDH2 in mesenteric lymph node dendritic cells**

Tomohiro Takano<sup>1)</sup>, Ryutaro Kotaki<sup>1)</sup>, Tadashi Yoshida<sup>2)</sup>, Kyoko Takahashi<sup>3)</sup>, Haruyo Nakajima-Adachi<sup>1)</sup>, Satoshi Hachimura<sup>1)</sup>

Research Center for Food Safety, Univ. Tokyo, Tokyo, Japan<sup>1)</sup>, Department of Applied Biological Chemistry, Tokyo University of Agriculture and Technology, Tokyo, Japan<sup>2)</sup>, Nihon University College of Bioresource Sciences, Kanagawa, Japan<sup>3)</sup>

3-G-W22-17-O/P

**One of PD-1 ligand, PD-L2 induced on Langerhans cells augments cutaneous T cell-mediated immune responses**

Emi Furusawa<sup>1,2)</sup>, Tatsukuni Ohno<sup>1)</sup>, Shigenori Nagai<sup>1)</sup>, Takuya Komiyama<sup>1)</sup>, Katsunori Kobayashi<sup>4)</sup>, Hidetoshi Hamamoto<sup>4)</sup>, Michiyo Miyashin<sup>2)</sup>, Hiroo Yokozeki<sup>3)</sup>, Miyuki Azuma<sup>1)</sup>

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3-G-W22-18-O/P

**Evaluation of anti-inflammatory activity of enantiomeric 17,18-epoxyeicosatetraenoic acid in contact hypersensitivity**

Azusa Saika<sup>1,2)</sup>, Takahiro Nagatake<sup>1)</sup>, Tetsuya Honda<sup>3)</sup>, Koji Hosomi<sup>1)</sup>, Kenji Kabashima<sup>3)</sup>, Jun Kunisawa<sup>1,2,4,5,6)</sup>

Laboratory of Vaccine Materials, Center for Vaccine and Adjuvant Research, and Laboratory of Gut Environmental System, National Institutes of Biomedical Innovation, Health and Nutrition (NIBIOHN), Osaka, Japan<sup>1)</sup>, Graduate School of Pharmaceutical Sciences, Osaka University, Osaka, Japan<sup>2)</sup>, Department of Dermatology, Graduate School of Medicine, Kyoto University, Kyoto, Japan<sup>3)</sup>, International Research and Development Center for Mucosal Vaccines, The Institute of Medical Science, The University of Tokyo, Tokyo, Japan<sup>4)</sup>, Graduate School of Medicine, Graduate School of Dentistry, Osaka University, Osaka, Japan<sup>5)</sup>, Graduate School of Medicine, Kobe University, Hyogo, Japan<sup>6)</sup>

3-G-W22-19-O/P

**Immunohistochemical analysis of class-switched subtype of primary cutaneous marginal zone lymphoma in terms of inducible skin-associated lymphoid tissue**

Toshiaki Kogame<sup>1,2)</sup>, Tomoya Takegami<sup>2)</sup>, Tatsuhiro Sakai<sup>2)</sup>, Tatsuki R Kataoka<sup>3)</sup>, Masahiro Hirata<sup>3)</sup>, Chiyuki Ueshima<sup>3)</sup>, Miho Matsui<sup>4)</sup>, Takashi Nomura<sup>2)</sup>, Kenji Kabashima<sup>2)</sup>

Department of Dermatology, Shiga General Hospital, Moriyama, Japan<sup>1)</sup>, Department of Dermatology, Kyoto University Graduate School of Medicine, Kyoto, Japan<sup>2)</sup>, Department of Diagnostic Pathology, Kyoto University Hospital, Kyoto, Japan<sup>3)</sup>, Department of Dermatology, Ijinkai Takeda Hospital, Kyoto, Japan<sup>4)</sup>

Chairpersons: Tomonori Nochi, Hayato Takahashi

In the both gastrointestinal tract and skin, innate/acquired immune cells and stromal/epithelial cells cooperatively interact and function as a barrier system for maintaining homeostasis of mucosal and cutaneous interface. Recent studies have also highlighted the important role of resident microbes and their metabolites in the regulation of epithelial barrier as well as mucosal immune system. Dysbiosis of gut microbes is closely associated with dysregulation of the barrier system, leading to systemic translocation of gut microbes and development of inflammatory bowel diseases. Peripheral nerve is an emerging player to regulate inflammation in the peripheral tissue. This workshop aims to discuss recent findings on molecular and cellular machineries of the barrier establishment and inflammatory disease induction in the gastrointestinal tract and skin.

3-G-W23-1-O/P

### Notch signal controls final differentiation of TCR $\gamma\delta^+$ CD8 $\alpha\alpha^+$ intraepithelial lymphocytes in the small intestine

Chieko Ishifune, Koji Yasutomo

Department of Immunology and Parasitology, Tokushima University Graduate School of Biomedical Sciences, Tokushima, Japan

3-G-W23-2-O/P

### Imaging of ulcerative colitis diseased area-development by using model mice with luciferase reporter gene and inoculation of causative gut bacteria

Atsushi Irie<sup>1)</sup>, Takahisa Imamura<sup>2)</sup>, Tatsuko Kubo<sup>2)</sup>, Yayoi Michibata<sup>1)</sup>, Yasuharu Nishimura<sup>1)</sup>Department of Immunogenetics, Graduate School of Medical Sciences, Kumamoto University, Kumamoto, Japan<sup>1)</sup>, Department of Molecular Pathology, Graduate School of Medical Sciences, Kumamoto University, Kumamoto, Japan<sup>2)</sup>

3-G-W23-4-O/P

### IgA-deficiency causes spontaneous enteritis

Takahiro Adachi<sup>1)</sup>, Taro Watabe<sup>2)</sup>, Takashi Nagaishi<sup>2)</sup>, Mamoru Watanabe<sup>2)</sup>, Hajime Karasuyama<sup>3)</sup>, Soichiro Yoshikawa<sup>3)</sup>Department of Immunology, Medical Research Institute, Tokyo Medical and Dental University, Tokyo, Japan<sup>1)</sup>, Department of Gastroenterology and Hepatology, Tokyo Medical and Dental University, Tokyo, Japan<sup>2)</sup>, Department of Immune Regulation, Tokyo Medical and Dental University, Tokyo, Japan<sup>3)</sup>

3-G-W23-10-O/P

### Cholecystokinin down-regulates psoriatic inflammation by its possible self-regulatory effect on epidermal keratinocytes

Atsuko Funakoshi, Taisuke Ito, Yoshiki Tokura

Department of Dermatology, Hamamatsu University School of Medicine, Hamamatsu, Japan

3-G-W23-11-O/P

### Essential role of CCL2-CCR2 pathway in the pathogenesis of chronic irritant contact dermatitis

Rintaro Shibuya<sup>1)</sup>, Akihiko Kitoh<sup>1)</sup>, Kenji Kabashima<sup>1,2)</sup>Department of Dermatology, Kyoto University, Kyoto, Japan<sup>1)</sup>, Singapore Immunology Network and Skin Research Institute of Singapore, Agency for Science, Technology and Research (A\*STAR), Singapore, Singapore<sup>2)</sup>

3-G-W23-13-O/P

### Homeostatic pruning and activity of epidermal nerves are dysregulated in barrier-impaired skin during chronic itch development

Sonoko Takahashi<sup>1)</sup>, Azusa Ishida<sup>1)</sup>, Akiharu Kubo<sup>1,2)</sup>, Hiroshi Kawasaki<sup>1,2)</sup>, Sotaro Ochiai<sup>1)</sup>, Takashi Watanabe<sup>1)</sup>, Manabu Nakayama<sup>3)</sup>, Haruhiko Koseki<sup>1)</sup>, Masayuki Amagai<sup>1,2)</sup>, Takaharu Okada<sup>1)</sup>RIKEN, Yokohama, Japan<sup>1)</sup>, Keio University, Tokyo, Japan<sup>2)</sup>, Kazusa DNA Research Institute, Kisarazu, Japan<sup>3)</sup>

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## W24 Tumor immunity 2 -Immunosuppression and Microenvironment in Cancer-

13:30 ~ 14:50 Room H

Chairpersons: Yasuharu Nishimura, Shin-ichiro Fujii

Cancer immunotherapy has attracted enormous attention, stimulated by the success of immune check point blockade drugs (ICB), such as CTLA-4 and PD-1/PD-L1 mAbs. For a successful response to ICB agents, clinical and preclinical data so far provide evidence that the presence of tumor antigen-, including neoantigen-specific CD8 T cells within the tumor microenvironment (TME) is a good prognostic indicator. Another major compartment affecting these therapies is the myeloid cell; there are various types of myeloid cells, some of which have pro-, others antitumor activity. The success of ICB depends on their composition in the TME. In this workshop, a more detailed and refined analysis of the contribution of tumor-resident myeloid cells and CTL to the local antitumor immune response will be discussed.

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3-H-W24-1-O/P

### Inhibition of myeloid-derived suppressor cell differentiation by palmitic acid

Masashi Tachibana<sup>1,2)</sup>, Koji Kobiyama<sup>3)</sup>, Ken J. Ishii<sup>3)</sup>, Shizuo Akira<sup>4,5)</sup>, Fuminori Sakurai<sup>1)</sup>

Graduate School of Pharmaceutical Sciences, Osaka University, Osaka, Japan<sup>1)</sup>, Global Center for Medical Engineering and Informatics, Osaka University, Osaka, Japan<sup>2)</sup>, The Institute of Medical Science, The University of Tokyo, Tokyo, Japan<sup>3)</sup>, World Premier International Immunology Frontier Research Center, Osaka University, Osaka, Japan<sup>4)</sup>, The Research Institute for Microbial Diseases, Osaka University, Osaka, Japan<sup>5)</sup>

3-H-W24-2-O/P

### FROUNT inhibitor disulfiram as a new class of macrophage-targeting anti-cancer drug synergizing with immune-checkpoint blockade

Yuya Terashima, Etsuko Toda, Kouji Matsushima

Division of Molecular Regulation of Inflammatory and Immune Diseases, Research Institute for Biomedical Sciences (RIBS), Tokyo University of Science, Chiba, Japan

3-H-W24-7-O/P

### An inhibitory immunoreceptor CD300a suppresses tumor infiltrating Treg cells and tumor development through binding to phosphatidylserine on tumor-derived exosomes

Yuta Nakazawa<sup>1)</sup>, Chigusa Nakahashi-Oda<sup>1,3)</sup>, Akira Shibuya<sup>1,2,3)</sup>

Department of immunology, University of Tsukuba, Ibaraki, Japan<sup>1)</sup>, Life Science Center for Survival Dynamics, Tsukuba Advanced Research Alliance (TARA), University of Tsukuba, Ibaraki, Japan<sup>2)</sup>, R&D Center for Innovative Drug Discovery, University of Tsukuba, Ibaraki, Japan<sup>3)</sup>

3-H-W24-14-O/P

### Pharmacological activation of p53 enhances NK cell activity and inhibits the development of AML

Yasutaka Hayashi<sup>1,2)</sup>, Susumu Goyama<sup>1)</sup>, XiaoXiao Liu<sup>1)</sup>, Shuhei Asada<sup>1)</sup>, Yosuke Tanaka<sup>1)</sup>, Tomofusa Fukuyama<sup>1)</sup>, Mark Wunderlich<sup>3)</sup>, Eric O'Brien<sup>3)</sup>, Benjamin Mizukawa<sup>3)</sup>, Satoshi Yamazaki<sup>4)</sup>, Akiko Matsumoto<sup>5)</sup>, Satoshi Yamasaki<sup>5)</sup>

Division of Cellular Therapy, The Institute of Medical Science, The University of Tokyo, Tokyo, Japan<sup>1)</sup>, Department of Hematology-Oncology, Institute of Biomedical Research and Innovation, Foundation for Biomedical Research and Innovation at Kobe, Kobe, Japan<sup>2)</sup>, Cancer & Blood Diseases Institute, Cincinnati Children's Hospital Medical Center, University of Cincinnati College of Medicine, Cincinnati, USA<sup>3)</sup>, Division of Stem Cell Therapy, The Institute of Medical Science, The University of Tokyo, Tokyo, Japan<sup>4)</sup>, Laboratory of Molecular Medicine, The Institute of Medical Science, The University of Tokyo, Tokyo, Japan<sup>5)</sup>

3-H-W24-19-O/P

### Effective combination immunotherapy for chemoresistant mouse bladder cancer using peptide vaccines and PD-1 blockade

Shohei Ueda<sup>1,2)</sup>, Atsushi Irie<sup>1)</sup>, Satoru Senju<sup>1)</sup>, Masatoshi Eto<sup>2)</sup>, Yasuharu Nishimura<sup>1)</sup>

Department of Immunogenetics, Graduate School of Medical Sciences, Kumamoto University, Kumamoto, Japan<sup>1)</sup>, Department of Urology, Graduate School of Medical Sciences, Kyushu University, Fukuoka, Japan<sup>2)</sup>

3-H-W24-20-O/P

### Newly identified neoantigens by utilizing PD-L1-deficient tumor for an effective DCs vaccine

Masahiro Okada, Kanako Shimizu, Tomonori Iyoda, Shin-ichiro Fujii

Laboratory for Immunotherapy, RIKEN Center for Integrative Medical Sciences, Yokohama, Japan

3-H-W24-23-O/P

### Arid5a orchestrates immunosuppressive environments via the Ido1-kynurenine-Treg axis in malignant pancreatic cancer

Gyanu Parajuli, Shigeru Hashimoto, Tadamitsu Kishimoto

Immune regulation, Immunology Frontier Research Center (iFReC), Osaka University, Osaka, Japan

### **Immunosuppression caused by IDO protein stability in tumor microenvironment**

Nobuo Tsukamoto<sup>1)</sup>, Sou Kurosaki<sup>1)</sup>, Jun-ichiro Inoue<sup>2)</sup>, Yutaka Kawakami<sup>1)</sup>

Institute of Advanced Medical Research, School of Medicine, Keio University, Tokyo, Japan<sup>1)</sup>, The Institute of Medical Science, The University of Tokyo, Tokyo, Japan<sup>2)</sup>

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## W25 Tumor immunity 3 -Cancerimmunotherapy (Preclinical and clinical study)-

15:05 ~ 16:25 Room H

Chairpersons: Akira Yamada, Tetsuro Sasada

Cancer immunotherapy, particularly with immune checkpoint inhibitors and adoptive cell therapy (ACT), has recently been added as a fourth treatment option, in addition to/in combination with surgery, chemotherapy, and radiotherapy. To develop this field, in this workshop, we will focus on the assessment of the T cell response, particularly in cancer immunotherapy-treated patients as an aspect of immune monitoring. Next, we will focus on improvements in T cell therapy by using genetic modification of ACT technology and iPS-derived cells and novel adjuvants. In addition, recent advances in clinical research will also be discussed. Since the topics in this session are cutting edge, we encourage active discussion by all participants.

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3-H-W25-1-O/P

### Immunological mechanism of synergistic anti-cancer activities by activation of TLR9 and STING

Burcu Temizoz<sup>1,3)</sup>, Kou Hioki<sup>1)</sup>, Takayuki Shibahara<sup>2,3)</sup>, Shingo Kobari<sup>2)</sup>, Nao Jounai<sup>2)</sup>, Takato Kusakabe<sup>2)</sup>, Michelle Sue Jann Lee<sup>4)</sup>, Cevayir Coban<sup>4)</sup>, Etsushi Kuroda<sup>2)</sup>, Ken J. Ishii<sup>1,2,3)</sup>

The Institute of Medical Science, The University of Tokyo (IMSUT), Department of Microbiology and Immunology, Division of Vaccine Science, Tokyo, Japan<sup>1)</sup>, Laboratory of Adjuvant Innovation, Center for Vaccine and Adjuvant Research (CVAR), NIBIOHN, Osaka, Japan<sup>2)</sup>, WPI Immunology Frontier Research Center (IFReC), Osaka University, Laboratory of Vaccine Science, Osaka, Japan<sup>3)</sup>, WPI Immunology Frontier Research Center (IFReC), Osaka University, Laboratory of Malaria Immunology, Osaka, Japan<sup>4)</sup>

3-H-W25-3-O/P

### Generation of CTLs from iPSCs transduced with TCR genes: development of “TCR cassette” method

Seiji Nagano<sup>1)</sup>, Koji Terada<sup>2)</sup>, Ryohei Kondo<sup>2)</sup>, Yasutoshi Agata<sup>2)</sup>, Kyoko Masuda<sup>1)</sup>, Hiroshi Kawamoto<sup>1)</sup>

Institute for Frontier Life and Medical Sciences, Kyoto University, Kyoto, Japan<sup>1)</sup>, Shiga University of Medical Science, Shiga, Japan<sup>2)</sup>

3-H-W25-5-O/P

### Analysis of intra-tumoral immune response in gastric cancer by RNA sequencing and immunostaining

Noriyuki Saito<sup>1,2)</sup>, Yoshihiro Kushihara<sup>2,5)</sup>, Akihiro Hosoi<sup>2)</sup>, Koji Nagaoka<sup>2)</sup>, Yukari Kobayashi<sup>2)</sup>, Yasuyoshi Sato<sup>2,4)</sup>, Takahiro Karasaki<sup>2,6)</sup>, Koichi Yagi<sup>1)</sup>, Hiroharu Yamashita<sup>1)</sup>, Kazuhiro Kakimi<sup>2,3)</sup>

Department of Gastrointestinal Surgery, Graduate School of Medicine, The University of Tokyo, Tokyo, Japan<sup>1)</sup>, Department of Immunotherapeutics, The University of Tokyo Hospital, Tokyo, Japan<sup>2)</sup>, Cancer Immunology Data Multi-Level Integration Unit, Medical Sciences Innovation Hub Program (MIH), RIKEN, Kanagawa, Japan<sup>3)</sup>, The Cancer Institute Hospital, Tokyo, Japan<sup>4)</sup>, Department of Neurosurgery, Graduate School of Medicine, The University of Tokyo, Tokyo, Japan<sup>5)</sup>, Department of Thoracic Surgery, The University of Tokyo Hospital, Tokyo, Japan<sup>6)</sup>

3-H-W25-8-O/P

### Pan-cancer analysis of tumor-infiltrating T cells reveals differential CD39 and CD103 expression among CD4<sup>+</sup>CD8<sup>+</sup> double-positive T cells in renal cell carcinoma

Yoshinobu Koguchi<sup>1)</sup>, William Miller<sup>1)</sup>, Brian D. Piening<sup>1)</sup>, Venkatesh Rajamanickam<sup>1)</sup>, Brady Bernard<sup>1)</sup>, Zhaoyu Sun<sup>1)</sup>, Yaping Wu<sup>1)</sup>, Johanna K. Kaufmann<sup>2)</sup>, William L. Redmond<sup>1)</sup>

Earle A. Chiles Research Institute, Providence Cancer Institute, Portland, OR, United States<sup>1)</sup>, TESARO, a GSK company, Waltham, MA, United States<sup>2)</sup>

3-H-W25-11-O/P

### The investigation of personalized immunotherapy targeting neoantigen for liver, pancreas, and biliary tract cancer

Toshiro Suzuki<sup>1,2)</sup>, Tetsuya Nakatsura<sup>2)</sup>

General Medical Education and Research Center, School of Medicine, Faculty of Medical Technology, Teikyo University, Tokyo, Japan<sup>1)</sup>, Division of Cancer Immunotherapy, Exploratory Oncology Research & Clinical Trial Center, National Cancer Center, Kashiwa, Japan<sup>2)</sup>

3-H-W25-14-O/P

### PD-1 expressing *ex-vivo* cultured tumor infiltrating lymphocytes confers tumor reactivity in cervical cancer: possible implication for adoptive cell therapy

Mohammad A Sayem, Nuchsupha Sunthamala, Tomonori Yaguchi, Takashi Iwata, Tomonobu Fujita, Yutaka Kawakami

Division of Cellular Signaling, Institute for Advanced Medical Research, Keio University School of Medicine, Tokyo, Japan

3-H-W25-16-O/P

### Novel HTLV-1 Env epitope-specific CD4<sup>+</sup> T cells boost Graft-Versus-ATL effects in post-HSCT patients

Undrakh Ganbaatar<sup>1)</sup>, Yotaro Tamai<sup>1)</sup>, Ryuji Tanosaki<sup>2)</sup>, Yasuhiro Maeda<sup>3)</sup>, Ilseung Choi<sup>4)</sup>, Youko Suehiro<sup>5)</sup>, Mari Kannagi<sup>1)</sup>, Atsuhiko Hasegawa<sup>1)</sup>

Department of Immunotherapeutics, Tokyo Medical and Dental University, Tokyo, Japan<sup>1)</sup>, Center for Transfusion Medicine and Cell Therapy, Keio University, Tokyo, Japan<sup>2)</sup>, Department of Hematology, Osaka Minami Medical Center, Osaka, Japan<sup>3)</sup>, Department of Hematology, National Kyushu Cancer Center, Fukuoka, Japan<sup>4)</sup>, Department of Cell Therapy and Hematology, National Kyushu Cancer Center, Fukuoka, Japan<sup>5)</sup>



**Antibody-mediated transient depletion of CD4<sup>+</sup> cells enhances the remodeling of the T-cell receptor repertoire and promotes anti-tumor immune responses in cancer patients**

Hiroyasu Aoki<sup>1,2)</sup>, Satoshi Ueha<sup>1)</sup>, Shigeyuki Shichino<sup>1)</sup>, Toshihiro Suzuki<sup>3)</sup>, Tetsuya Nakatsura<sup>3)</sup>, Shigehisa Kitano<sup>4)</sup>, Kouji Matsushima<sup>1)</sup>

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