2020

The 2nd Osaka University-OIST Symposium

X

OIST

Neuroscience, Immunology and cutting-edge technologies including imaging

Date

January 20th-21st (Mon-Tue) 2020

Venue

Symposium, Poster session

Sydney Brenner Lecture Theater

(Seminar Room B250), Center Bld.

Preface

Graduate School of Medicine/
Faculty of Medicine,
Osaka University

Eiichi Morii

Dean

We are happy to hold the second joint symposium between OIST and Osaka University, and to visit a beautiful and comfortable campus of OIST.

We look forward to meeting again OIST members and enjoying nice talks and discussions.

This opportunity will be a good memory for us and the students from Osaka University.

Let's enjoy such a nice symposium.

OIST

Mary Collins

Provost

Welcome to our friends from Osaka University for this second joint Osaka University OIST Symposium. We look forward to excellent talks and to the meeting between young researchers from OIST and Osaka University, in this way more collaborations will start. Please enjoy your stay at OIST in the beautiful island of Okinawa.

Schedule

Day 1

January 20th (Mon) 2020

Time: 13:30-18:10

Venue: B250

	Lunch at Grano	
13:30-13:40	OIST Provost. Mary Collins	Greetings
Chair: OIST Provost. Mary Collins		
13:40-14:10	OIST Prof. Hiroki Ishikawa	“Regulation of T cell functional states by AP-1 transcription factors”
14:10-14:40	OIST Prof. Erik De Schutter	“Modeling the cell biology of learning”
14:40-14:55	Break	
Chair: Graduate School of Medicine/Faculty of Medicine, Osaka University Vice Dean Masaru Ishii		
14:55-15:25	Osaka University Prof. Toshihide Yamashita	“Development of therapeutic strategies to repair neuronal network for the central nervous system diseases”
15:25-15:55	Osaka University Prof. Kazuyo Moro	“Role of group 2 innate lymphoid cells in idiopathic interstitial pneumonias”
15:55-16:25	Osaka University Prof. Yukinori Okada	“Statistical genetics, disease biology, and drug discovery”
16:25-16:40	Break	
Chair: OIST Prof. Erik De Schutter		
16:40-17:10	OIST Distinguished Prof. Tomoyuki Takahashi	“Multi-disciplinary approach to presynaptic black box”
17:10-17:40	OIST Prof. Bernd Kuhn	“Voltage imaging from Purkinje neurons in awake animals”
17:40-18:10	Osaka University Prof. Shigeru Kitazawa	“In search of allocentric coordinates–where they are and how they emerge”

Schedule

Day 2

January 21st (Tue) 2020

Time: 08:45-13:40

Venue: B250

Chair: Graduate School of Medicine/Faculty of Medicine, Osaka University Dean Eiichi Morii		
08:45-09:15	OIST Dr. Charles Plessy	"Rapid changes of gene order in closely related species of Oikopleura zooplankton"
09:15-09:45	Osaka University Prof. Makoto Sato	"Neocortical neurons develop neuronal circuits for cooperative tasks by multiple-targeting with collateral formation"
09:45-09:55	Break	
09:55-10:25	Osaka University Vice Dean Masaru Ishii	"Intravital multiphoton imaging dissecting immune celluar dynamics in vivo"
Chair: OIST Dean of Research Tadashi Yamamoto		
10:25-11:25	Flash Talk by Poster presenter [2min/person]	
11:25-11:50	Business Meeting on OSAKA-OIST Collaborations	
11:50-12:00	OIST Dean of Research Tadashi Yamamoto	Closing Remarks
12:00-13:40	Lunch & Poster presentation	
13:50-14:50	Campus Tour	
15:00	End	

H-1 **Yumiko Ueno**

Phosphorylation of hepatitis B virus core protein regulates its subcellular localization

Yumiko Ueno, Keiji Ueda, Tomoyuki Honda

Division of Virology, Department of Microbiology and Immunology, Osaka University Graduate School of Medicine

Hepatitis B virus (HBV) is one of major agents of hepatitis, cirrhosis and hepatocellular carcinoma. Most of the core protein is found in the nuclei of hepatocytes, and is rarely present in cytoplasm. However, patients with cytoplasmic core protein tend to have more severe hepatitis. It has been reported core protein carries nuclear localization signals. In this study, we identified other sites relating to core protein subcellular localization. We found that core protein is translocated into the nucleus by dephosphorylation of one phosphorylation site of core protein. On the other hand it is maintained in the cytoplasm by phosphorylation of this site. This result suggests that phosphorylation of core protein regulates its subcellular localization.

H-2 **Ryo Oi**

Evaluation of Mitochondrial Mass in Colorectal Cancer

Ryo Oi, Kenji Ohshima, Satoshi Nojima, Takahiro Matsui, Shinichiro Tahara, Masako Kurashige, Eiichi Morii

Department of Pathology, Graduate school of Medicine/ Faculty of Medicine, Osaka University

Cancer cells acquire completely different metabolic pathways from normal tissues so as to be advantageous for their own survival. The Warburg effect is one of the most famous altered metabolic state in cancer cells, in which the glycolysis is enhanced and the function of mitochondria is decreased in cancer cells even in an aerobic environment. Especially in colorectal cancer, the Warburg effect has been considered to be strong. However, we hypothesized that mitochondria actually do function in colorectal adenocarcinoma, and examined the amount of mitochondria in colorectal adenocarcinoma tissue. Immunohistochemical staining of translocase of outer membrane 20 (TOMM20); a mitochondrial outer membrane protein, and mitochondrial transcription factor A (TFAM); a mitochondrial DNA transcription factor, were performed in 52 colorectal adenocarcinoma resected specimens respectively. Both TOMM20 and TFAM showed an increased expression level in the colorectal adenocarcinoma compared to the adjacent non-neoplastic mucosa. These results indicated that the amount of mitochondria in the colorectal adenocarcinoma was higher than that in the adjacent non-neoplastic mucosa. Further experiments are to be conducted whether increased mitochondria actually have the metabolic activity in colorectal adenocarcinoma.

H-3 **Kyotaro Nohata**

Distinct role of the 182nd amino acid residue between HIV-1 and HIV-2 capsids in CPSF6 binding

Kyotaro Nohata1), Akatsuki Saito1), Hirotaka Ode2), Hisaki Ohmori1), Emi E Nakayama1), Yasumasa Iwatani2), Tatsuo Shioda1)

Research Institute for Microbial Diseases, Osaka University
Clinical Research Center, National Hospital Organization Nagoya Medical Center

[Background]CPSF6 binding of viral capsid(CA) is strictly conserved among primate lentiviruses(PLVs). The CPSF6 binding interface locates to PLV CA N-terminal domain as well as its C-terminal domain, especially 182nd amino acid (lysine; K). Therefore, introduction of K182R mutation into HIV-1 CA diminishes CPSF6 binding. In contrast, we recently found that HIV-2 CRF01_AB isolates(NMC842) with K182R mutation in CA. Here, we examined the impact of the K182R mutation on CPSF6 binding among PLVs.

[Results]We found that CPSF6-358 sensitivity of NMC842 virus was comparable to that of HIV-1. We therefore introduced K182R mutation into HIV-1 and HIV-2 lineage viruses and found that CPSF6-358 sensitivity of HIV-1 lineage viruses was greatly decreased by the K182R mutation, that of HIV-2 lineage virus was minimally affected.

[Conclusion]K182R mutation has limited impact on CPSF6-358 sensitivity of HIV-2 lineage, suggesting a distinct role of the 182nd amino acid residue between HIV-1 and HIV-2 CAs in CPSF6 binding.

H-4 **Akira Mizuno**

Tissue-specific opposite directional effects of expression quantitative trait loci (eQTLs) may contribute to the genetics of complex traits

Akira Mizuno,1 Yukinori Okada2,3

[1]Faculty of Medicine, Osaka University, [2]Department of Statistical Genetics, Graduate School of Medicine, Osaka University, [3]Laboratory of Statistical Immunology, Immunology Frontier Research Center (WPI-IFReC), Osaka University

Analyzing tissue-specific effects of expression quantitative trait loci (eQTLs) is a promising approach to reveal the biological meanings of the susceptible loci reported by genome-wide association studies (GWASs). Here we describe “opposite eQTL effects”, i.e., gene expression effects of eQTLs that are in the opposite direction between different tissues, as the biologically meaningful annotations of genes and genetic variants for understanding the GWAS loci. The genes and single nucleotide polymorphisms (SNPs) associated with the opposite eQTL effects (opp-multi-eQTL-Genes and opp-multi-eQTL-SNPs) were extracted from the largest eQTL database provided by the Genotype-Tissue Expression (GTEx) project. A significant proportion of the genes having eQTLs were annotated as the opp-multi-eQTL-Genes (2,323 out of 31,212; 7.4%). The opp-multi-eQTL-SNPs were enriched at transcription start sites, and significantly associated with the SNPs reported in GWASs (2,275 out of 9,290; 24.5%). Based on the results, the opposite eQTL effects can be a common phenomenon in the tissue-specific gene regulation with a possible contribution to the development of complex traits.

H-5

Siripongvutikorn Yanakawee

Acquired TTP mouse model induced by ADAMTS13/MHC class II complex

Yanakawee Siripongvutikorn1,2, Tadahiro Suenaga2,3, Ryosuke Hiwa4, Koichiro Ohmura4 and Hisashi Arase2,3

[1]Graduate school of Medicine, Faculty of Medicine, Osaka University
[2]Department of Immunochemistry, Research Institute for Microbial Diseases, Osaka University
[3]Laboratory of Immunochemistry, World Premier International (WPI) Immunology Frontier Research Center, Osaka University
[4]Graduate School of Medicine and Faculty of Medicine Kyoto University

Acquired TTP is a potentially fatal disease as a result of autoantibody inhibition of VWF protease, ADAMTS13. However, the mechanism how the autoantibodies are developed has remained unclear. Recently, we found a unique function of HLA class II molecules - the ability to transport misfolded proteins to the cell surface without processing to peptides. This may be targets for autoreactive lymphocytes and lead to autoimmune diseases. In the current study, we showed that full length ADAMTS13 forms complexes with MHC classII. Then, we found that mice immunized with ADAMTS13/MHC class II complex developed autoantibodies against ADAMTS13 when compared to mice immunized with only ADAMTS13. The model supports our hypothesis that a specific MHC class II molecule binding to an unprocessed protein and presenting it to the cell surface is the key mechanism for inducing autoantibodies, providing a compatible insight to understanding the pathogenesis of acquired TTP.

O-1

Saahil Acharya

Transient dimerization of a postsynaptic cell adhesion molecule neuroligin and its implications in the regulation of trans-synaptic adhesion

Neuroligins are synaptic adhesion molecules located in the post-synaptic membrane and form calcium-dependent trans-synaptic complexes with neurexins located in the presynaptic membrane. The trans-synaptic neuroligin-neurexin complexes have been proposed to regulate synaptic development and transmission [1, 2]. Using single-molecule imaging, we found that neuroligin1 (with inserts in splice sites A and B) conjugated with the ACP-tag (inserted between amino acid 641 and 642 in the exoplasmic domain; conjugated with SeTau647-ACP ligand) expressed in the dendritic plasma membrane (PM) in transfected hippocampal neurons in culture, as well as that expressed in the PM of transfected CHO cells, exist as both monomers and dimers (possibly oligomers in the dendritic PM due to the presence of endogenous neuroligins). In CHO cells, the dimer fraction at any moment was increased with an increase of the number density of neuroligin1. In the PM, these dimers fell apart quickly in the time scale on the order of 0.2 s, namely, these dimers were transient dimers. Presently, we are examining the effect of neurexin binding on neuroligin dimerization and dimer lifetimes, as well as whether neurexin by itself forms dimers. In the dendrites of hippocampal neurons, neuroligin1 entered the spine membrane from the dendritic shaft PM and also exited from the spines, with various dwell times in spines. A project examining the effect of synaptic excitation on the dynamics of neuroligin1 and neurexin entering and exiting from the synaptic region is in progress.

O-2

Sigita Augustinaite

Complementary activity of sensory activated and suppressed layer 6 corticothalamic neurons reflects behavioral state

Layer 6 corticothalamic neurons project to thalamus where they are thought to regulate sensory information transmission to cortex in a behavioral state dependent manner. However, little is known about this feedback activity in vivo. Here, we imaged calcium changes in visual cortex layer 6 primary corticothalamic neurons with 2-photon microscopy in head-fixed mice in response to passive viewing during full range of behavioral states, from locomotion to sleep. We found sensory activated and suppressed neurons. The state dependence was heterogeneous with respect to locomotion or level of alertness, although the average activity was largest during highest vigilance within both populations. Interestingly, complementary activity of these distinct populations kept the overall corticothalamic output relatively constant during any given behavioral state. Thereby, in addition to sensory and non-sensory information, a constant activity level characteristic for behavioral state is conveyed to thalamus where it can regulate signal transmission from periphery to cortex.

O-3 **Laurent Guillaud**

ATP-dependent liquid phase transitions in synapse organization and neurodegeneration

Emerging evidences are showing that biological functions are highly regulated by liquid phase separation of proteins and molecules within cells and in extracellular environment. However, how liquid phase transitions can regulate neuronal organization and function in vivo remain largely elusive. Here, using real time fluorescent microscopy and FRAP experiments I demonstrate that the solubility of the pre-synaptic cytoplasm is highly heterogeneous and that both peri-active zone and active zone phase transitions are correlated to local mitochondrial activity and ATP production. Blocking ATP synthesis from mitochondria drastically induced liquid phase separations of pre-synaptic cytoplasm, active zones and synaptic vesicles. By simultaneous labeling and tracking of synaptic vesicles and post-synaptic receptors, and by monitoring local mitochondrial activity and ATP concentration, I present evidences showing that a decrease in mitochondrial activity near release sites, promotes vesicles relocation and receptors translocation to neighboring area. In addition, in vitro liquid phase transitions assays of various presynaptic proteins (a-synuclein, Tau) implicated in neurodegenerative diseases such as PD and AD undergo ATP-dependent liquid phase separations. Thus, local regulation of pre- and post-synaptic liquid phase transitions by ATP play pivotal roles during synapse formation, plasticity and synaptic transmission in physiological or pathological conditions.

O-4 **Nishtha Ranawat**

Microglial colonization of developing neural retina of zebrafish

Nishtha Ranawat, Ichiro Masai

Developmental Neurobiology Unit, Okinawa Institute of Science and Technology Graduate University

Microglia are brain-resident macrophages of mesodermal origin that function as the first line of defense in brain. Microglia patrol the brain throughout life, eliminating dead or dying neurons to promote neuronal protection. In addition, microglia prune synapses during brain development. Embryonic microglia precursors, or primitive microglia, originate in peripheral mesoderm and migrate to different brain regions during development. However, the mechanisms by which microglia colonize the brain during development are not fully understood. The retina is one of the first brain regions to accommodate microglia. In zebrafish, the first microglial precursors appear to enter the optic cup around 30 – 32 hpf. These embryonic microglial precursors use intraocular hyaloid blood vessels as a ropeway to migrate into the optic cup through choroid fissure. Once retinal progenitor cells exit from the cell cycle, the microglia precursors associated with blood vessels around the lens starts to infiltrate the retina preferentially through neurogenic regions. Furthermore, microglia precursors fail to invade the retinal tissue when retinal neurogenesis is arrested, suggesting that colonization of retinal tissue depends upon the neurogenic state. Thus, blood vessels and neuronal differentiation create an essential migratory path for microglia migration. We propose that at least two guidance mechanisms are required for microglia to migrate from yolk into the developing neural retina in zebrafish.

O-5 **Zacharie Taoufiq**

Deep synaptic proteome exploration in mental health and disease

In the brain, the deep molecular diversity of synapses is fundamental for anatomical and functional specializations of neural circuits. However, powerful proteomics method that can detect and quantify the diversified synaptic composition is still lacking, yet it may help to find the molecular origins of the most integrative brain functions.Here we have established a new proteomics method using purified synaptic vesicle (SV) model, which serves as benchmark for quantitative synaptic proteomics (Takamori et al, Cell 2006). We have identified about 1,500 proteins, three times more than reported previously. This refined proteome not only includes all known canonical SV proteins, but also many novel SV accessory protein groups, isoforms and low abundant proteins.
One aspect of the analysis of this 'hidden' SV proteome was particularly unexpected: 210 distinct brain diseases were found associated with SV-interacting proteins, of which the majority (76%) were not detected in previous studies. Our results bring to light the immense but inevitable challenge we are facing for drug targeting of human neurological diseases in the 21st century.

O-6 **Olga Elisseeva**

Hedgehog signaling in human lymphocytes, an unexpected player in anti-cancer immunity

Olga Elisseeva1,2, Tadashi Yamamoto1,2

1. OIST, Cell Signal Unit
2. RIKEN IMS, Laboratory of Immunogenetics

Hedgehog (Hh) is a morphogen family signaling pathway indispensable during embryogenesis in metazoans. It is required for tissue repair in adults and the differentiation of intra-thymic T cells. Its role in adaptive and innate immune responses has not been investigated. Our work shows that immune synapse (IS) serves as a signaling center for this pathway. Sustained TCR signaling and mature IS formation between dendritic cell and naïve T lymphocytes can only happen when the Hh pathway is active. Furthermore, we find that rather than canonical transcription-guided, a non-canonical Hh signaling, via heterotrimeric G protein coupling of signal transducer Smoothened (SMO) takes place at immune synapse. We show that SMO competes with CXCR4 for the pool of available Gai proteins, and pushes CXCR4 to switch to Gα11/q, rendering it to be irresponsive to the migratory ques. This enables optimal inside out signaling of integrins, reinforcing the productive immune synapse formation between DC and naïve T lymphocytes. We also show that manipulations of the Hh pathway affect anti-tumor immune responses to spheroid tumors in 3D culture.

O-7

Dongqi Han

Self-Organization of Action Hierarchy and Compositionality by Reinforcement Learning with Recurrent Neural Networks

We propose a novel, multiple-timescale, stochastic RNN for RL. Empirical results show that the network can autonomously learn to abstract sub-goals and can self-develop an action hierarchy using internal dynamics in a challenging continuous control task. Furthermore, we show that the self-developed compositionality of the network enhances faster re-learning when adapting to a new task that is a re-composition of previously learned sub-goals, than when starting from scratch. We also found that improved performance can be achieved when neural activities are subject to stochastic rather than deterministic dynamics.

O-8

Viktoras Lisicovas

Improving two-photon temporal focusing microscopy with high repetition rate pulsed laser source

Wide-field temporal focused two-photon microscopy enables high-speed volumetric imaging via the simultaneous acquisition of a large sample area with high lateral and axial resolution. Implementations to date have been held back due to the requirement for high fluence laser sources for excitation of a large area, which typically operated at a low repetition. Achieving high signal intensity in this configuration necessitates increasing energy per pulse; however, this exacerbates the photobleaching in a power-law fashion. We demonstrate that the increase in the repetition rate of the laser system, within thermal constraints compatible with live imaging, strongly enhances fluorescence signal intensity without accelerating photobleaching. We apply these findings to volumetric imaging of the nematode C. elegans sensory neurons.

O-9

Bianca Sieveritz

Ventral motor thalamic input to prelimbic cortex is involved in cost-benefit decision-making

Thalamocortical input is important in controlling the activity of cortical pyramidal neurons. For instance, input from the posterior medial thalamic complex can disinhibit these neurons and is crucial in long-term potentiation. Cortex also receives an extensive input from ventral motor thalamic nuclei (ventrolateral, ventral anterior and ventromedial thalamus) that target and drive pyramidal neurons in layers 2/3 and 5. We focus on the involvement of input from these ventral motor thalamic nuclei to prelimbic cortex in choice behaviour, specifically in cost-benefit decision-making. Prelimbic corticostriatal neurons project to striosomes and participate in cost-benefit decision-making, which provides animals with a choice between a high cost-high reward and low cost-low reward option. Optogenetic inhibition of prelimbic corticostriatal neurons increases the choice of rats for the high cost-high reward option, while optogenetic simulation increases the choice for the low cost-low reward option.

We trained five-week-old Sprague-Dawley rats on a benefit-benefit, cost-cost and cost-benefit decision-making task. These tasks offer animals a choice between: i- a high reward and a low reward ii- a high cost and a low cost, and iii- a high cost-high reward and low cost-low reward option. Once animals acquired all three tasks and reached 9 weeks of age, we stereotaxically injected an adeno-associated virus expressing archaerhodopsin (AAV5-CAG-ArchT-GFP) or a control virus (AAV5-CAG-GFP) into ventromedial thalamus (interaural AP +7.0, ML -1.2; from dura DV +6.56) and implanted a LED fibre optic into prelimbic cortex (bregma AP +3.24, ML 0.0; from dura DV 3.1). After two weeks the virus was expressed in ventral motor thalamic axon terminals in prelimbic cortex. The performance of animals was compared between two conditions; with and without administering optogenetic inhibition to ventral motor thalamic axon terminals in prelimbic cortex. On the cost-benefit decision-making task optogenetic inhibition significantly increased the preference of animals for the high cost-high reward as compared to the low cost-low reward option. In the other two tasks changes were less pronounced. This result indicates that ventral motor thalamic input to prelimbic cortex is involved in evaluating cost against benefit, but not in processing cost or benefit as such. Interestingly, optogenetic inhibition of prelimbic corticostriatal neurons in the cost-benefit decision-making task induces disinhibition of high-frequency neurons in striosomes that in turn inhibit striatal projection neurons. These striatal projection neurons may receive an excitatory input from ventromedial thalamus that may itself be involved in cost-benefit decision-making; an option that we currently explore further.

O-10 **Tsung-Han Hsieh**

JunB controls cell viability and clonal expansion in Th1

Tsunghan Hsieh, Daiki Sasaki, Hiroki Ishikawa

Immune Signal Unit, Okinawa Institute of Science and Technology Graduate University

【諸言】Type 1 helper T cells (Th1) play pivotal roles in maintaining host immune response against pathogens, especially bacteria and virus. During infection, naïve T helper cells react to microbial components, undergo rapid clonal expansion and migrate to infected tissues to activate other immune cells. The whole process can help host to eradicate invading pathogens. Here, we have identified one AP-1 gene, JunB, plays important role in this process.
【方法】Here, a JunB-deficient T-cell-specific (Junbfl/flCD4cre, JunB-TKO) mouse model was generated for this experiment. Naïve CD4 T (naïve Th) cells were collected from pooled spleens, and differentiated under Th1/2 polarizing conditions. After 72h, cells are harvested, stained with antibodies, and analyzed by FACS. For adoptively transfer experiment, naive Th cells were harvested from spleens of both WT-OT2 and JunB-TKO-OT2 mice, in which transgenic T cell receptors specific for ovalbumin are expressed. Harvested naïve Th cells were transferred through i.v. injection into recipient mice with immunization of adjuvant and ovalbumin. 7 days after immunization, mice were sacrificed, and spleen and lymph nodes were taken for analysis.
【結果・考察】We have identified JunB, when being deleted from T helper cells, hampered cell growth and viability during in vitro activation, albeit deletion of JunB did not affect Th cell function. When JunB-deficient T helper cells were adoptively transferred into recipient mice, they could not undergo clonal expansion and become Th1 cells. Moreover, RNAseq analysis revealed that JunB regulates cell growth and viability through different molecular mechanisms among Th1 and Th2 cells. Taking these evidences together, I hypothesized that JunB is essential and plays distinct functions in maintaining Th1 cell viability and growth during clonal expansion.

O-11 **Tsung-Yen Huang**

Analysis of Immunomodulatory Metabolisms involved in Th17 differentiation

Tsung-Yen Huang, Hiroki Ishikawa

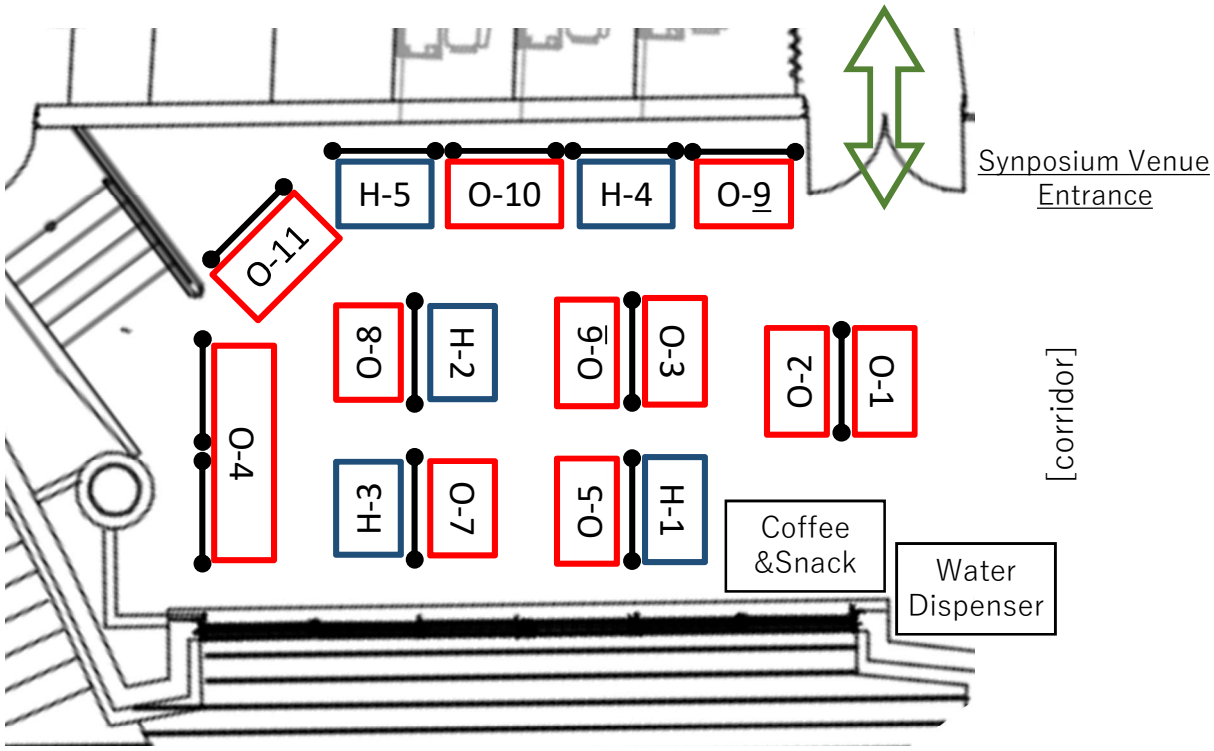
Immune Signal Unit, Okinawa Institute of Science and Technology Graduate University

T helper 17 (Th17) cells are capable to differentiate into various lineages which can either act as critical drivers of autoimmune diseases or participate in the intestinal homeostasis in a non-inflammatory manner. It remains unclear that how cellular metabolism regulates the fate commitment among different Th17 subtypes. To identify the metabolic pathways involved in the generation of pathogenic Th17 cells, we compared the metabolomic profiles between *in vitro* polarized non-pathogenic Th17 cells (referred to as Th17(B)) and pathogenic Th17 cells (Th17(23)). The metabolomic analysis showed that a broad spectrum of cellular metabolism, including glucose metabolism, fatty acid oxidation, and purine metabolism, were altered in the pathogenic Th17(23) cells. Furthermore, the inhibition of glycolysis through either 2-deoxy glucose (2-DG) treatment or glucose starvation could enhance the IL-17 expression in both Th17(B) and Th17(23) cells, and 2-DG altered the expression pattern of transcription factors which predominantly control the fate commitment in different Th17 lineages. These results indicate metabolic alteration is associated with Th17 differentiation, and modulation of glycolytic activity can control the immunofunction of Th17 cells.
Keywords: Th17, Immunometabolism, Glycolysis

Poster Board Placement

H- ＊ - Osaka University Poster number

O- ＊ - OIST Poster number



Poster Presentation Information

[Size of poster board]

＊Full size: W 975mm × H 1,910mm
＊Blue board part:
W 880mm × H 1,170mm
＊Pushpins will be available at the venue.

[Flash Talk]
Date: January 21st (Tue) 2020
Time: 10:25-11:25
＊2min/person

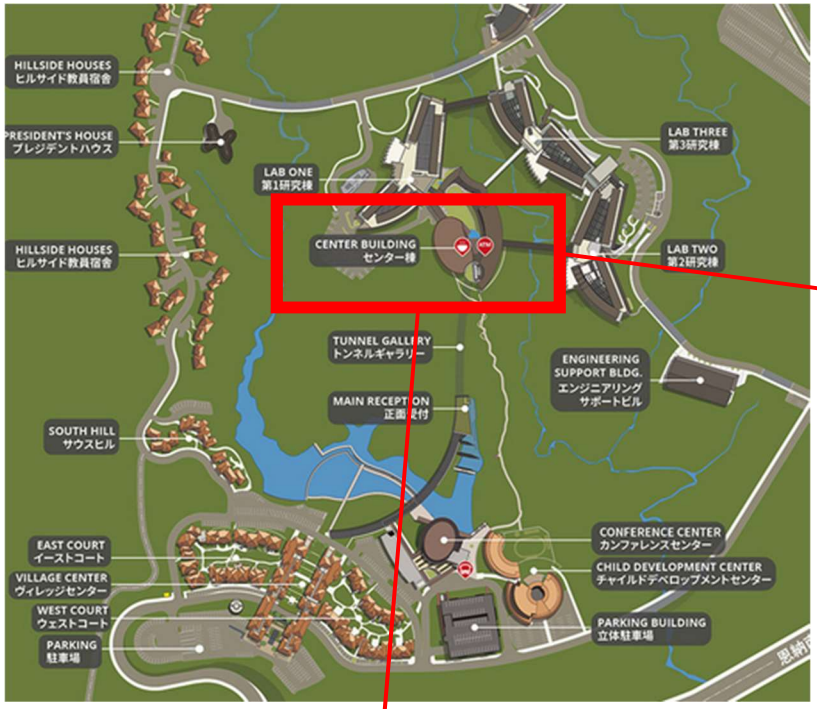
[Poster mounting]
Date&Time: Jan 21st (Tue) 11:30-11:50

[Poster removal]
Date&Time: Jan 21st (Tue) 13:40 ~ by 14:00

[Lunch & Poster sessions]
The lunch box will be provided at the venue for speakers and poster presenters.
You are free to have lunch or break or poster session from 12:00 to 13:40.
Date: January 21st (Tue) 2020
Time: 12:00-13:40
Venue: In front of B250

Venue

[Symposium, Poster Presentation]
Seminar Room B250 (Sydney Brenner Lecture Theater), Center Bld.
https://www.oist.jp/conference-venue#b250



Seminar Room B250
(Sydney Brenner Lecture Theater)

Lunch

- **January 20th (Mon)**: Please have lunch on your own.

- **January 21st (Tue)**: The lunch box will be provided at the venue for speakers and poster presenters.

There is a café and restaurant at OIST as well: Grano
https://www.oist.jp/ja/visitors-center-shops-restaurants

➢ Café Business Hours (Level C, Center Building)
- Weekdays: 09:00-18:30
- Weekends: 10:00-15:00 (Last Order 14:30)
- Holidays: 10:00-17:00 (Last Order 16:30)

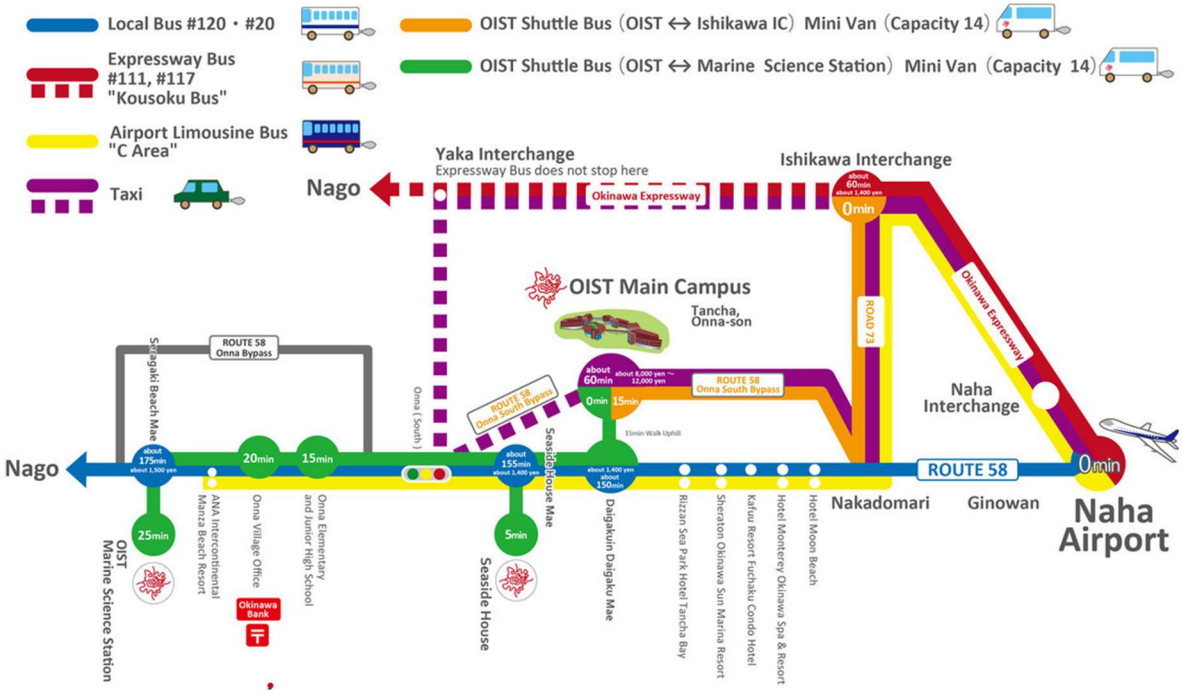
➢ Restaurant Business Hours (Level B, Center Building)
- Weekdays: 11:30-15:00
- Weekends and Holidays: 11:00-15:00 (Last Order 14:30)

➢ Contact and Information
- Phone: 098-966-8413

Access

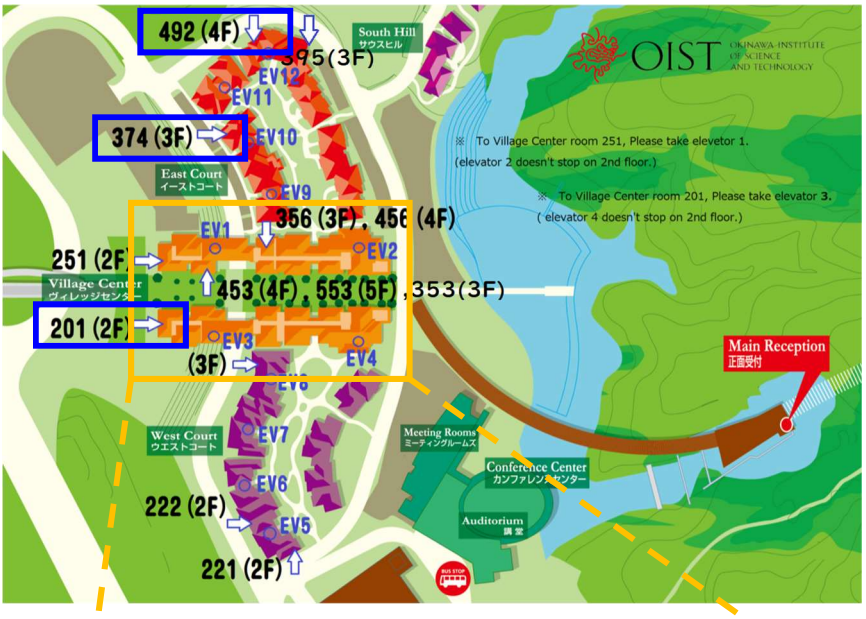
[Area Map] https://www.oist.jp/ja/page/30654

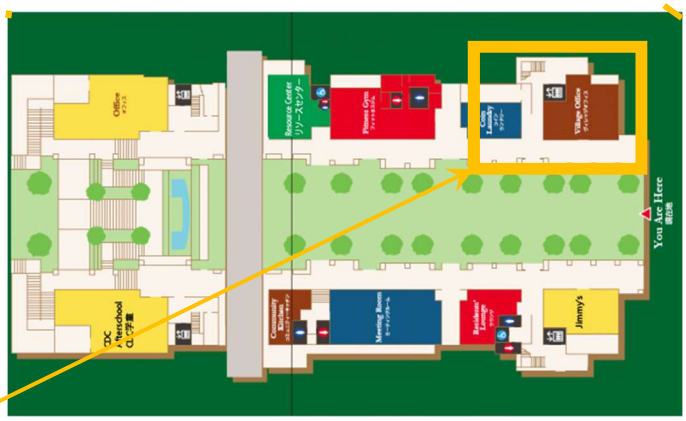
[How to Get to OIST from Naha Airport and Southern Okinawa]
https://www.oist.jp/directions-southern-okinawa



Accommodation

For Students





[Village Office]
https://www.oist.jp/community-facilities#village-office

➢ Office Hours
- Monday through Friday: 9:00-18:00,
- Saturday and Sunday: 9:00-17:00, closed on national holidays.
- Contact: 080-6342-6500 or 080-6342-6501

[Check-In]
- Check－In: 15:00
- Please receive your room key at Village Office: Please tell your name and reservation number to the reception.
- You can leave your luggage at Village Office at your responsibility. Please pick it up by 30 minutes before the Office closes.

[Check-Out]
- Check-Out: 10:00
- Please return your room key to the Village Office, or hand it to OIST staff(Suzuki).
- You can leave your luggage at Village Office at your responsibility. Please pick it up by 30 minutes before the Office closes.