The Tri-lateral NAIST-TLL-CU Joint Symposium 2016

Animal-Plant-Microbe Ecological Networks

28 March, 2016

Organized by
Nara Institute of Science and Technology

Organizing committee

Toshiro Ito, NAIST/TLL

Taro Kawai, NAIST

Program

March 28 (Mon), 2016

The Tri-lateral NAIST-TLL-CU Joint Symposium 2016
"Animal-Plant-Microbe Ecological Networks"
@ Large Auditorium (L11), Lecture Hall, Grad. Sch. of Biological Sciences, NAIST

9:30- **Registration**

10:00-10:10 **Welcome Address**
Toshio Hakoshima
Dean of Graduate School of Biological Sciences, NAIST

Session 1 **Chair: Toshiro Ito (*NAIST/TLL*)**

10:10-10:35
Yusuke Saijo, NAIST
Danger peptide receptor signaling in plants ensures basal immunity upon pathogen-induced depletion of the co-receptor kinase BAK1

10:35-11:00
Zhongchao Yin, TLL
TAL effector-dependent disease resistance of rice to *Xanthomonas oryzae* pv. *oryzae*

11:00-11:25
Supaart Sirikantaramas, CU
Response of 'KDML 105' and salt-tolerant 'UBN 02123-50R-B-2' rice *Oryza sativa* L. under menadione-induced oxidative stress

11:25-11:45
Nobutoshi Yamaguchi, NAIST
A molecular framework for hormonal control of floral meristem termination in *Arabidopsis thaliana.*

11:45-13:00 **Lunch**

Session 2 **Chair: Hisaji Maki (*NAIST*)**

13:00-13:25
Noriaki Sasai, NAIST
Development and maintenance of the central nervous system

13:25-13:50
Gregory Jedd, TLL
Understanding the evolution of complex multicellularity using functional comparative genomics

13:50-14:15
Kanoktip Packdibamrung, CU
L-Phenylalanine production from glycerol by recombinant *Escherichia coli*

14:15-14:35
Hisashi Tatebe, NAIST
Role of Sin1 in TORC2 signaling

14:35-15:30 **Tea Break & Poster session**

Session 3 **Chair: Taro Kawai (*NAIST*)**

15:30-15:55
Naoyuki Inagaki, NAIST
Moleculr mechanics for axon outgrowth and navigation

15:55-16:20
Katsutomo Okamura, TLL
Regulation of miRNA biogenesis

16:20-16:45
Kunlaya Somboonwiwat, CU
Antiviral action of antilipopolysaccharide factor isoform 3 on white spot syndrome virus

16:45-17:05
Yasukazu Nakahata, NAIST
Intracellular NAD+ regulates circadian genes expression pattern

17:05-17:15 **Closing Remarks**
Hisaji Maki
Professor, Graduate School of Biological Sciences, NAIST

18:00-20:00 **Banquet**

Abstract

Danger peptide receptor signaling in plants ensures basal immunity upon pathogen-induced depletion of the co-receptor kinase BAK1

Yusuke Saijo[1,2,3]

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[3]*Max Planck Institute for Plant Breeding Research, Germany*

Pathogens infect a host by suppressing defense responses induced upon recognition of molecular structures typical of microbes, designated microbe-associated molecular patterns (MAMPs). Despite this suppression, MAMP receptors mediate basal resistance to limit host susceptibility, via a process that remains elusive. We have pursued functional interactions between cell-surface receptors for MAMPs and danger-associated molecular patterns (DAMPs) in immune activation against pathogens. In the reference plant *Arabidopsis thaliana*, different leucine-rich repeat (LRR) receptors for MAMPs/DAMPs physically associate and function with the LRR receptor kinase (RK) BAK1 in plants. We report that BAK1 depletion is linked to defense activation through the endogenous PROPEP-derived DAMP peptides (Pep epitopes) and their LRR-RKs PEPR1/PEPR2, despite critical defects in MAMP signaling. In *bak1*-knockout plants, PEPR elicitation results in extensive cell death and salicylate-based defenses over jasmonate-based defenses, which are associated with elevated proligand and receptor accumulation. Loss of *BAK1* stimulates the release of PROPEP3, produced in response to Pep application and pathogen challenge, and renders PEPRs necessary for fungal resistance. These findings are biologically relevant, since specific BAK1 depletion coincides with PEPR-dependent resistance to the fungal pathogen *Colletotrichum higginsianum*. Thus, the PEPR pathway ensures basal immunity in plants when MAMP-triggered defenses are compromised by BAK1 depletion.

TAL effector-dependent disease resistance of rice to *Xanthomonas oryzae* pv. *oryzae*

Dongsheng Tian, Xuan Zeng, Keyu Gu, Chengxiang Qiu, Xiaobei Yang, Zhiyun Zhou, Meiling Goh, Yanchang Luo, **Zhongchao Yin**

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Bacterial blight of rice, caused by *Xanthomonas oryzae* pv. *oryzae* (*Xoo*), is one of the most destructive bacterial disease in rice. Transcription activator-like (TAL) type-III effectors from *Xoo* strains are important virulence factors for bacterial infection on rice plants. To against bacterial infection, rice has evolved dominant disease resistance (*R*) to recognize TAL effectors from incompatible *Xoo* strains. The rice *R* gene *Xa10* confers race-specific and whole-developmental resistance to *Xoo* strains harboring the corresponding *avrXa10* gene. Upon bacterial infection, AvrXa10 binds to the effector binding element (EBE) in the *Xa10* promoter and activates *Xa10* expression, which triggers hypersensitive response. The *Xa10* gene product, XA10, is localized to the ER membrane and triggers cell death by disrupting the ER and cellular Ca^{2+} homeostasis in plant cells. Transgenic rice plants carrying the modified *Xa10* gene containing 5 EBEs in its promoter ($Xa10^{E5}$) conferred broad-spectrum resistance to *Xanthomonas oryzae* pv *oryzae* strains.

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Response of 'KDML 105' and salt-tolerant 'UBN 02123-50R-B-2' rice *Oryza sativa* L. under menadione-induced oxidative stress

Apidet Rakpenthai, **Supaart Sirikantaramas**

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Various environmental stresses eventually lead to oxidative stress condition. To understand metabolic changes under this condition, we used menadione to induce oxidative stress in two cultivars, salt-sensitive KDML 105 and salt-tolerant UBN, of five-day-old rice (*Oryza sativa* L.) seedlings for 0.5, 2, and 6 hours. The assessment of oxidative stress by aconitase activity assay indicated that UBN is able to endure the stress better than KDML 105. Major changes in both cultivars under the stress were a reduction of inorganic content and primary metabolite levels. However, the higher basal levels of 4-amino-butanoic acid and asparagine were found in UBN. In addition, the increased flavonoid contents were found in UBN. Interestingly, we detected changes in sulfur metabolism under oxidative stress. Quantitative RT-PCR analysis of genes involved in sulfur assimilation revealed a higher response in mRNA levels of sulfur transporter genes and sulfur-responsive genes. These findings might suggest superior capabilities in stress tolerance of UBN.

A molecular framework for hormonal control of floral meristem termination in *Arabidopsis thaliana*.

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Flower formation requires termination of stem cell program (determinacy) to develop female reproductive structure or gynoecium, which is a key for plant reproductive success and crop yield. In *Arabidopsis*, the MADS box transcription factor AGAMOUS (AG) is necessary and sufficient for this vital termination by regulating a direct downstream target, *KNUCKLES* (*KNU*). Floral meristem determinacy defects in the *knu* flower is much weaker than in *ag* flower, suggesting that AG induces the expression of additional, as yet known gene(s) to terminate floral meristem at stage 6. We have found that a direct AG downstream gene*, CLABS CLAW* (*CRC*) functions in parallel with *KNU* to terminate floral meristem. CRC restricts auxin transport capacity to generate its proper gradients, which is critical for gynoecium development. This regulation is partially through direct transcriptional repression of *TORNADO2* (*TRN2*). Ectopic expression of *TRN2* under the control of *CRC* promoter enhanced floral meristem termination defects in the *knu* mutant background. In contrast, loss-of-function mutation of *TRN2* partially rescued the floral meristem termination defects in the *crc knu* mutant. TRN2 protein, localized at plasma membrane, promotes auxin export. Our study reveals a transcriptional regulatory network downstream of the master regulator AG and a molecular link between floral meristem termination and the subsequent auxin-dependent gynoecium development. This network contributes to the robustness of floral meristem.

Development and maintenance of the central nervous system

Noriaki Sasai

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The neural tube is comprised of more than ten different cell types along the dorsal-ventral (D-V) axis and they are precisely assigned and patterned. Many classical studies have shown that the pattern formation of the neural tube is mainly dependent on the special type of secreted factors that are collectively called morphogens. Among them, Sonic Hedgehog (Shh) is emanated from the floor plate (FP), which is located to the ventral-most part of the neural tube, and the underlying notochord, and plays essential roles for the patterning of the ventral subtypes. Several studies have indicated that Shh induces differential gene expression in a graded manner.

In my presentation, I will first demonstrate some of key experiments that proved the morphogen model. However, in addition to the gradient of the concentration, our recent study has shown that the timing of the Shh signal also influences the cell types generated. For example, the assignment of FP cells requires the early exposure to Shh during the neurogenesis.

In this symposium, I will be discussing how to generate specific types of neurons from the stem cells, and will also discuss the possibilities of the generated neurons into the clinical applications.

Understanding the evolution of complex multicellularity using functional comparative genomics

Tu Anh Nguyen[1+], Ousmane H. Cissé[2+], Jie Yun Wong[1], Peng Zheng[1], David Hewitt[3], Minou Nowrousian[4], Jason E. Stajich[2], **Gregory Jedd**[1]

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Understanding the genetic basis of the transition form simple to complex multicellularity (CM) is a major challenge for evolutionary biology. In the fungi, CM is based on hyphal filaments where septal pores allow intercellular transport, communication and cooperation. The genus Neolecta defines an enigma: phylogenetically placed with early diverging yeasts in the Ascomycota, Neolecta nevertheless possesses features characteristic of CM seen in the later diverging Pezizomycotina. We sequenced the Neolecta genome and employed comparative genomics to study CM evolution. Innovations known to be specific to other fungal CM taxa are absent in Neolecta. This indicates that a subset of its CM-associated traits are independently derived. By contrast, ancient CM-associated functions are present in Neolecta and other CM taxa, and these genes are lost or highly divergent in yeasts. Using this phylogenetic pattern as a search criterion, we identify and characterize new CM-associated functions in the model system *Neurospora crassa*. These act at various endomembrane compartments, which include peroxisomes, endosomes, mitochondria and the late secretory pathway. Some of these gene families appear to have arisen before the divergence of animals and fungi, while others define subsequent evolutionary transitions. Together, these data suggest that retention of an organismal body plan can profoundly constrain the evolution of organelles and related functions. They further identify endomembrane complexity resulting from a gradual accumulation of genetic innovations as a prerequisite for complex multicellularity.

L-Phenylalanine production from glycerol by recombinant *Escherichia coli*

Kanoktip Packdibamrung

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L-Phenylalanine (L-Phe), an essential amino acid, is mainly used as a reactant for production of important substances in medicine and food industry such as aspartame. An increase in demand for aspartame leads to the improvement of L-Phe production in well-known microorganisms, especially *Escherichia coli*. To improve L-Phe production from glycerol, a by-product from biodiesel production, phenylalanine dehydrogenase gene (*phedh*) from *Bacillus lentus* was cloned and expressed in *E. coli* BL21(DE3) to increase the conversion of phenylpyruvate to L-Phe in the last step of L-Phe biosynthesis pathway. In addition, various genes from *E. coli* encoding pivotal proteins involving in L-Phe biosynthesis pathway (*tktA aroB, aroL* and *pheA*), glycerol uptake (*glpF* and *glpK*) and L-Phe excretion (*yddG*) were co-expressed with *phedh* gene using both one-plasmid and two-plasmid systems. The clone containing *aroB, aroL*, *phedh* and *tktA* in pRSFDuet-1 vector and *glpF yddG* in pBAD33 vector showed the highest production of L-Phe at 746 mg/L when the recombinant clone was cultured in minimum medium containing 3.1% glycerol and 6.3% $(NH_4)_2SO_4$ as carbon and nitrogen sources, respectively, at 37 ^oC after induction by 0.02% arabinose for 240 hours.

Role of Sin1 in TORC2 signaling

Hisashi Tatebe[1], Shinichi Murayama[1], Toshiya Yonekura[1], Tomoyuki Hatano[1], David Richter[2], Tomomi Furuya[1], Saori Kataoka[3], Kyoko Furuita[3], Chojiro Kojima[3], Kazuhiro Shiozaki[1,2]

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TOR (Target Of Rapamycin) kinase is an evolutionarily conserved serine / threonine protein kinase that forms structurally and functionally distinct protein complexes TOR complex 1 (TORC1) and TOR complex 2 (TORC2). From yeast to human, TORC1 merges various input signals, including those emitted from nutritional sources, and regulates multiple downstream targets to modulate anabolic and catabolic cellular activities. On the other hand, TORC2 transmits growth factor signals in mammals and glucose signals in the fission yeast *Schizosaccharomyces pombe*, by phosphorylating and activating the specific subset of the AGC-family protein kinases.

By dissecting TORC2 signaling in fission yeast, we have revealed that the Sin1 subunit of TORC2 plays a pivotal role in proper recognition of the TORC2 substrate kinases. While fission yeast Sin1 is dispensable for TORC2 assembly, Conserved Region In the Middle of Sin1 (Sin1CRIM) forms a discrete domain that physically binds the TORC2 substrate kinases. Moreover, in the fission yeast *sin1* null mutant, Sin1CRIM grafted onto another TORC2 subunit is sufficient for TORC2 to phosphorylate its substrate kinase Gad8. Solution structure analysis indicates that Sin1CRIM is a ubiquitin-like domain, of which a characteristic acidic loop is essential for binding the TORC2 substrate kinases.

MOLECULR MECHANICS FOR AXON OUTGROWTH AND NAVIGATION

Naoyuki Inagaki

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Neurons extend a long process, axons, to the right destinations and form complicated networks in the brain; all our brain activities depend on the neuronal networks. We recently identified novel proteins shootin1 and singar1, and are analyzing the molecular mechanisms for axon outgrowth and guidance, using up-to-date methods including cell biology, developmental biology and mechanobiology. We expect that these analyses will give us a new window into therapeutic strategies for neuronal diseases, such as nerve injury.

Axon guidance molecules can navigate axonal outgrowth. Actin filaments (F-actins) polymerize at the leading edge of axon (growth cone) and depolymerize proximally; this induces retrograde flow of F-actins. It has been proposed that the forces underlying axon outgrowth may be regulated by the modulation of coupling efficiency between F-actin flow and the extracellular substrate via “clutch” molecules. However, how cell signaling controls the coupling efficiency remains unknown. Shootin1 functions as a clutch molecule that couples F-actin retrograde flow and the substrate at neuronal growth cones to promote axon outgrowth. Here, we show that a soluble axon guidance molecule netrin-1 positively regulates forces at axonal growth cones via Pak1-mediated shootin1 phosphorylation. This phosphorylation enhanced the interaction between shootin1 and F-actin retrograde flow, thereby promoting F-actin-substrate coupling, force generation, and axon outgrowth. These results suggest that shootin1 is located at a critical interface, transducing a signal of netrin-1 into the forces for axon outgrowth. We also report that shootin1 is involved in the axon outgrowth mediated by a substrate-bound axon guidance molecule laminin.

Regulation of miRNA biogenesis

Mandy Yu Theng Lim[1,2], Alvin Wei Tian Ng[3], Li Zhou[1,3], Yuting Chou[4], Teck Por Lim[3], Amanda Simcox[5], Greg Tucker-Kellogg[3,6] , **Katsutomo Okamura**[1,2]

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miRNAs are processed from characteristic hairpin structures through sequential cleavage by conserved ribonucleases, and play regulatory roles in various biological processes including host-microbe interactions. In our lab, we aim to answer two key questions: 1. How do the miRNA processing factors distinguish miRNA hairpins and other structured RNA molecules? 2. How is biogenesis of miRNA regulated during animal development. Using Drosophila as a model system, we analyze biochemical properties of individual miRNA processing factors, and perform short- and long-RNA expression profiling to gain insights. At this symposium, I will present our recent findings regarding the molecular functions of the core miRNA processing factor Loqs, and the prevalence of post-transcriptional regulation of miRNA biogenesis.

Antiviral action of antilipopolysaccharide factor isoform 3 on white spot syndrome virus

Kunlaya Somboonwiwat

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Antilipopolysaccharide factor isoform 3 (ALF*Pm*3), the antimicrobial peptide from *Penaeus monodon,* is active against bacteria, fungi and a shrimp pathogenic virus, white spot syndrome virus (WSSV). Previously, ALF*Pm*3-interacting proteins such as WSSV186, WSSV189, WSSV395, WSSV458 and WSSV471 have been identified by the yeast two-hybrid assay. The interaction between ALF*Pm3* and WSSV proteins was confirmed by *in vitro* pull-down assay. Subsequently, we proved that pre-incubation of each WSSV protein with rALF*Pm*3 could interfere the neutralizing activity of rALF*Pm*3 on WSSV indicating by an increase in the mortality rate of WSSV-infected shrimp when compared to those injected with rALF*Pm*3 and WSSV. Next, WSSV proteins were analyzed as envelope and nucleocapsid proteins on the WSSV virion by Western blot analysis and immunoelectron microscopy. Furthermore, the direct effect of ALF*Pm*3 on WSSV was revealed. After WSSV virion and rALF*Pm*3 incubation, the morphological change of WSSV virion was determined by transmission electron microscopy. It was clearly observed that the envelope and nucleocapsid of WSSV virion were disrupted when compared with the control. The results indicated that the ALF*Pm*3 performs its anti-WSSV action by binding to WSSV structural proteins causing the WSSV virion damage.

Intracellular NAD^+ regulates circadian genes expression pattern

Yasukazu Nakahata, Yasumasa Bessho

Gene Regulation Research, Grad. Sch. Bio. Sci., NAIST

We and other groups have reported that intracellular NAD^+ amount demonstrates circadian oscillation, because gene expression of the rate limiting enzyme of NAD^+ salvage pathway “NAMPT” is regulated by circadian clock. Circadian NAD^+ oscillation regulates not only output physiological events but also circadian clock itself. NAD^+ consuming enzymes SIRT1 deacetylase and poly (ADP-ribose) polymerase 1 (PARP1) control acetylation status of histones/BMAL1/PER2 and poly ADP-ribosylation status of CLOCK to fine-tune the circadian genes oscillation. These findings suggest that controlling intracellular NAD^+ amount is crucial for maintaining circadian clock machinery precisely. To assess that, we tried to increase or decrease intracellular NAD^+ amount in NIH3T3 cells by genetically or pharmacologically. So far, we found that increasing NAD^+ up to 2-fold or decreasing by 50% rarely affect circadian machinery. However, the period of circadian clock was prolonged when NAD^+ amount was below 50% compared to control condition. We are now trying to reveal molecular mechanisms how NAD^+ amount modulates circadian period. In this talk, we would like to discuss how NAD^+ affects circadian clock machinery and when it happens under physiological or pathological conditions.

Poster session

P01
THAGUN Chonprakun, *Plant cell function (Hashimoto lab)*
Jasmonate-responsive ERF transcription factors regulate steroidal glycoalkaloid biosynthesis in tomato

P02
Wu Jinfeng and Satoshi Matsubara, *Plant Stem Cell Regulation and Floral Patterning (T. Ito lab)*
Heat and drought resistance by histone demethylases in Arabidopsis

P03
Shinsuke Yasuda, Yuko Wada, and Seiji Takayama, *Intercellular Communications (Takayama lab)*
Trans-acting small RNA controlling the dominance hierarchy among self-incompatibility alleles in Brassica rapa

P04
Teruki Sugiyama, Hirotomo Takatsuka, and Masaaki Umeda, *Plant Growth Regulation (Umeda lab)*
Control of the cell cycle in two distinct cell files of the root epidermis

P05
Takunori Minegishi, *Systems Neurobiology and Medicine (Inagaki lab)*
Functional Analysis of Shootin1b in Neuronal Migration

P06
Thanh Le Thi, Asako Furukohri, and Hisaji Maki, *Microbial Molecular Genetics (Maki lab)*
Molecular mechanisms of cruciform extrusion at long inverted repeats during DNA replication

P07
Suzianti Iskandar Vijaya and Hisaji Maki, *Microbial Molecular Genetics (Maki lab)*
Sublethal antibiotics induce reactive oxygen species (ROS) and 8-oxoG-related mutagenesis in Escherichia coli

P08
CHIA Kim Hou, *Cell Signaling (Shiozaki lab)*
The Rag GTPase ortholog binding (Rob) protein complex regulates TORC1 activity in fission yeast

P09
Shigehiro Iwaki, *Membrane Molecular Biology (Tsukazaki lab)*
Rapid estimation of monodispersity and stability of a drug-transporter for its structural biology

P10
Shuhei Horibe, *Molecular Signal Transduction (H. Itoh lab)*
The G-protein regulator Ric-8A ensures centriole duplication in mammalian cells

P11
Izumi Dateyama, *Molecular Signal Transduction (H. Itoh lab)*
Analysis of Serotonin Signaling Mediated through Primary Cilia

P12
Manabu Kitamata, *Molecular Medicine and Cell Biology (Suetsugu lab)*
Cellular function analysis of ANKHD1 and ANKRD17 with lipid binding ability

P13
Miki Nishio, Tomomi Kotoku, Yasumasa Ishida, Eishou Matsuda and Masashi Kawaichi, *Functional Genomics and Medicine (Ishida lab)*
Functional analysis of BTB-containing zinc finger protein, CIBZ, during cardiac inhibits differentiation of mouse ES cells

P14
Atsuki Yatsuzuka, *Developmental Biomedical Science (Sasai lab)*
An analysis of the molecular network in the ventral neural tube

P15
Tomoya Deguchi, *Molecular Immunobiology (Kawai lab)*
In vivo function of the kinase PIKfyve in T cell development

P16
Mizuka Nagayama, *Molecular Immunobiology (Kawai lab)*
Molecular mechanism of IL-33 production during allergic inflammation

P17
Takuya Sueyoshi, *Molecular Immunobiology (Kawai lab)*
Identification of HuR as a molecule that mediates antiviral innate immune responses

MEMO