제 24 회 한국분자·세포생물학회 예쁜꼬마선충분과 심포지움

◆ 일 시 : 2017 년 1 월 17 일 (화) ~ 2017 년 1 월 18 일 (수)

◆ 장 소 : 강원도 평창군 대관령면 올림픽로 715 용평리조트

◆ 주 최 : 한국분자·세포생물학회 예쁜꼬마선충분과

◆ 주 관 : 서울시립대학교 환경공학부 환경시스템독성학 연구실

◆ 후 원 : 자연과학, NGB, 국순당

◆ 인 원 : 91 명

◆ 내용 : 초청 연사 강연 및 실험실 연구 활동 발표

◆ 등록 :

등록비 : 교수 및 연구원 15만원

: 학생 7 만원

등 록 : 2017 년 1 월 17 일 (화) 13 시부터, 강연장 앞

퇴 실 : 2017 년 1 월 18 일 (수) 11 시까지, 물품 정리 후 카운터에 키 반납

◆ 강연장 : 용평리조트 그린피아콘도 레인보우홀

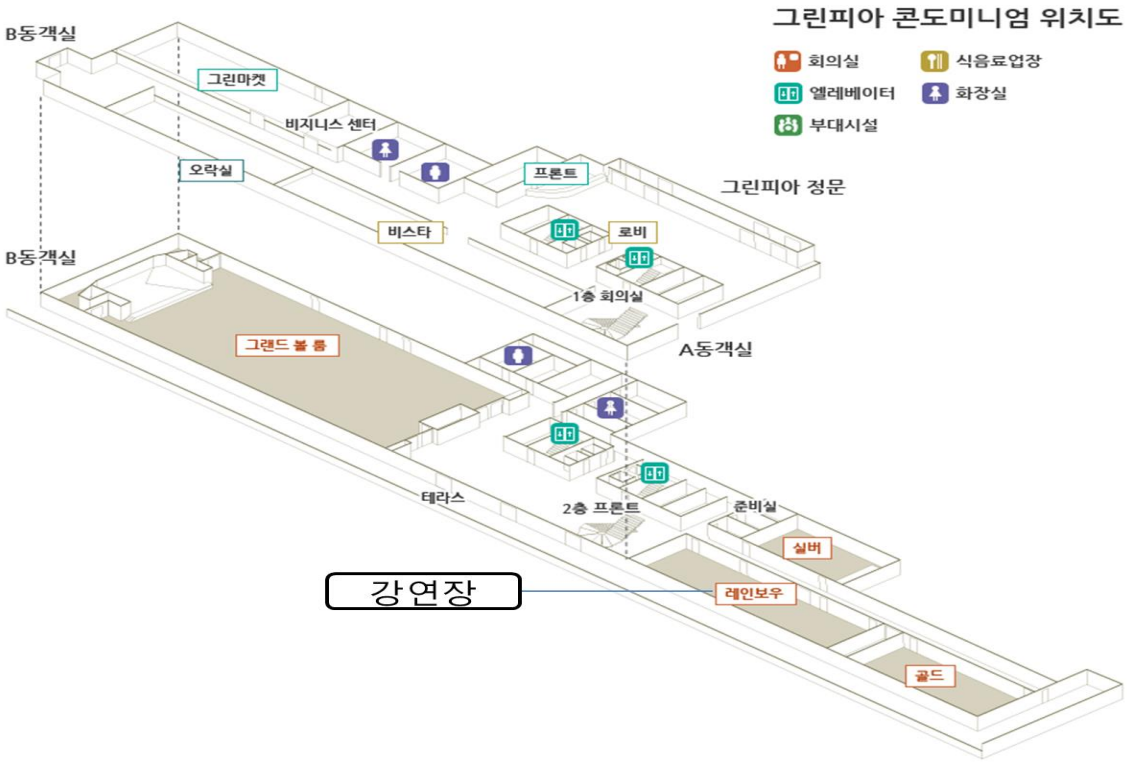
◆ 숙 소 :

교 수 : 그린피아콘도 25타입 (2인 1실)

학 생 : 그린피아콘도 38타입 (6인 1실)

- 등록처 및 강연장, 숙소 위치

  - 등록처 및 강연장, 숙소



제 24 회 한국분자·세포생물학회 예쁜꼬마선충분과 심포지움 스케쥴

2017 년 1 월 17 일 (화)		
13:00 ~ 14:00	등록 및 방배정, 포스터 전시	
14:00 ~ 14:05	Opening Remark (Prof. Jinhee Choi)	
Young Investigator Lecture (Chair : Young-Ki Paik)		
14:05 ~ 14:35	Seongkyun Kim (KAIST)	Network topology analysis of the *C. elegans* connectome: Vulnerability and Asymmetry
Session I : Development (Chair : Prof. Sun-Kyung LEE)		
14:35 ~ 15:15	Hana Jung (Hanyang University)	A calcineurin interacting protein functions in ovulation, male mating, and sperm activation of *C. elegans.*
	Tae-Woo Choi (Hanyang University)	Calcineurin regulates body length by controlling cholinergic transmission in *Caenorhabditis elegans*
	Esther Youn (Konkuk University)	cdc-25.2 is required for the spermatheca development and its expression is regulated by NHR-6
Session II : Neuroscience (Chair : Prof. Jin Il LEE)		
15:15 ~ 15:55	Leesun Ryu (DGIST)	The nutrient-responsive insulin signaling modulates chemosensory responses in *C. elegans*
	Woochan Choi (DGIST)	Chemosensory GPCR SRI-14 are required for concentration dependent odor preference in *C. elegans*
	Saraswathi Kalichamy (Yonsei University)	Hypergravity hinders axonal development of motor neurons in *Caenorhabditis elegans*
15:55 ~ 16:20	Coffee Break	
Session III : Stress response and Molecular biology (Chair : Prof. Seung-Jae V. Lee)		
16:20 ~ 17:15	Youngho Kim (University of seoul)	Immune and stress response crosstalk to xenobiotic exposure by pathogen infection in the nematode *Caenorhabditis elegans*
	Dae-Eun Jeong (POSTECH)	Mitochondrial chaperone HSP-60 up-regulates p38 MAP kinase signaling to enhance anti-bacterial immunity
	Soungyub Ahn (Seoul national university)	wts-1, the LATS kinase homolog of Hippo pathway regulates RNT-1 stability in *C. elegans*
	Bala Murali Krishna Vasamsetti (Chungbuk national university)	*Caenorhabditis elegans* VIG-1 protein is required for genomic stability

Session IV : Survival and Reproduction (Chair : Dr. Eun-Soo Kwon)		
17:15 ~ 17:55	Jun-Seok Han (KRIBB)	Genome-wide screening to find potential pro-longevity gut microbes using *Caenorhabditis elegans/Escherichai coli* system
	Sanghyun Sung (Seoul national university)	Using internal genomic regions as a conserved telomere maintenance mechanism
	Haelim Jeong (Yonsei University)	Identification of MGL-1 Function in Reproductive Plasticity of *C. elegans* in Response to Starvation Signals
17:55 ~ 18:05	단체사진촬영	
18:05 ~ 19:30	Dinner	
19:30 ~ 19:40	Jinhee Choi (University of seoul)	New Approach in *C. elegans* Research
Technical Lecture (Chair : Prof. Junho Lee)		
19:40 ~ 20:00	Won Bae (Unio biometrica)	COPAS Applications for High Throughput Screenings using *Caenorhabditis elegans*
Special Lecture (Chair : Yhong-Hee Shim)		
20:00 ~ 20:30	Jennifer H. Shin (KAIST)	How an engineer sees *C. elegans*
20:30 ~ 21:00	졸업생 talk 및 선물증정, 사진, 포스터 컨테스트 선물 증정	
21:00 ~ 22:00	자유토론 (포스터)	

2016 년 1월 18 일 (수)	
08:00 ~ 09:00	조식
09:00 ~ 11:00	자유토론
11:00 ~	퇴실

제 24 회 한국분자·세포생물학회 예쁜꼬마선충분과 심포지움 스케쥴

Young Investigator Lecture

Session I. Development

Session II. Neuroscience and Behavior

Session II. Stress response and Molecular biology

Session III. Survival and Reproduction

Technical Lecture

[Won Bae]

Special Lecture

[Jennifer H. Shin]

Poster Abstract

Young Investigator Lecture

Network topology analysis of the *C. elegans* connectome: Vulnerability and Asymmetry

Seongkyun Kim, Hyoungkyu Kim, Pyeongsoo Kim, Jerald D. Kralik, and Jaeseung Jeong

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To uncover the functional design principles of nervous systems, we used graph theoretical approaches to the hermaphrodite and the male *C. elegans* connectome. The connectomes consist of neurons (nodes) and synaptic connections (links). However, every element does not have same network properties and vulnerability due to the inhomogeneous degree distribution of the connectomes. Here we investigated the *C. elegans* connectomes to identify critical neurons and synapses using a vulnerability method by attacking each of 279 individual neurons and 2990 synaptic connections in the hermaphrodite *C. elegans* connectome and each of 360 individual neurons and 5295 synaptic connections in the male *C. elegans* connectome. We identified 12 neurons and 29 synapses of the hermaphrodite connectome and 9 neurons and 33 synapses of the male connectome that were critical to clustering, information integration and propagation. Then, we found that these critical elements have functional roles in dealing with adverse conditions and mating behaviors (hooking and backing during vulva search). Therefore, we suggest that a fundamental principle for the neuronal architectures is a negotiation between wiring cost, efficiency, vulnerability, and relative importance of biological functions to build a topological structure. Since we found the asymmetric critical neurons in the vulnerability results, we also investigated the hermaphrodite *C. elegans* connectome to uncover asymmetric features using graph theoretical approaches. We found that there are asymmetric features in the *C. elegans* connectome and these asymmetric features should have a correlation to asymmetric biological functions. Our novel approaches to identify asymmetric neurons well discriminated ASEL/R and AWCL/R as the asymmetric neurons. Therefore, we suggest that the other asymmetric neurons, which are identified by our novel approaches using motif ratio and relative difference of motif fingerprint, should have asymmetric functions. We believe that this computational study provides insights to understand the *C. elegans* nervous systems and to design further experimental investigations to identify biological functions.

Session I

Development

A calcineurin interacting protein functions in ovulation, male mating, and sperm activation of *C. elegans*

Hana Jung, Joohong Ahnn and Sun-Kyung Lee*

Department of Life Sciences, College of Natural Sciences, Hanyang University, Seoul 133-791, Republic of Korea

Calcineurin is a Ca^{2+}/Calmodulin-dependent Serine/Threonine phosphatase, which is involved in a variety of biological processes including development, behavior and reproduction. Candidates for calcineurin substrates in *C. elegans* have been screened by yeast two-hybrid assays. We identified CNP-2, calcineurin interacting protein-2, which is a substrate of TAX-6, a worm calcineurin ortholog. CNP-2 is expressed in the head neurons, posterior intestine, spermathecal valve, spermatheca-uterus valve and cell junction/membrane in spermathecal in hermaphrodites. In males, the expression of *cnp-2* is prominent in the SPC and SPD neurons in the spicule and rays, and the tip of the fan that plays a crucial role in mating behavior. Our analyses indicate that CNP-2 is required in ovulation, egg retention, ray structure, sperm activation and mating behavior. The *cnp-2* is present in only *Caenorhabditis* genus and shows 33% similarity to the KSP-rich domain of the *unc-89*, a homolog to mammalian obscurin that functions in the sarcomere formation and assembly. We find that KSP motifs are also present in human NEFM (neurofliment medium subunit) that is associated with axon development. We are currently asking a question whether the *cnp-2* is a functional KSP-like domain protein, and investigating the role of *cnp-2* in the *tax-6* pathway, regarding IP3 signaling pathway regulating ovulation. We suggest that CNP-2 is a *Caenorhabditis* specific protein, which plays a critical role in reproduction process, possibly by providing scaffolding platform as proposed in the KSP-rich domain of the *unc-89* and NEFM.

Keyword: Calcineurin interacting protein, KSP motif, ovulation, male mating, sperm activation

Calcineurin regulates body length by controlling cholinergic transmission in *Caenorhabditis elegans*

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Calcineurin, Ca2+/calmodulin-dependent serine/threonine phosphatase, has been reported to function in body size regulation in *C. elegans*. However, how calcineurin regulates growth is largely unknown. In this study, we studied the neuronal function of calcineurin in the regulation of body size. Through RNAi screening to identify a kinase counteracting calcineurin in growth regulation, we found that *mbk-2*, an ortholog of the Dual-specificity Yak1-Related Kinase (DYRK) family proteins, rescued the body size of *tax-6(lf)*. In addition, *oma-1* and *oma-2*, which is a zinc-finger transcription factor and a target of *mbk-2*, significantly reversed small body length of *tax-6(lf)*. This result suggests that calcineurin work with MBK-2 through OMA-1. Interestingly, exogenous acetylcholine reversed small body size in *tax-6(lf)*, which is also resistant to aldicarb treatment. Cholinergic neuronal expression of *tax-6* also recovered small body length of *tax-6(lf)*. These results suggest that calcineurin modulate acetylcholine release to control worm growth. Currently, we are investigating what kinds of cholinergic neurons are responsible for calcineurin function in growth.

Keyword: Calcineurin, Counteracting kinases, body size regulation.

cdc-25.2 is required for the spermatheca development and its expression is regulated by NHR-6

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Cell division cycle 25 (Cdc25) phosphatase promotes cell division by removing inhibitory phosphates of a Cyclin/CDK complex. In *C. elegans,* there are four *cdc-25* family members, *cdc-25.1, cdc-25.2, cdc-25.3* and *cdc-25.4.* Among *cdc-25* family members, only *cdc-25.2* has a ~5 kb long first intron where a regulatory element for gene expression may be located. To investigate whether the first intron of *cdc-25.2* regulates transcription, a transgenic strain containing either a Pcdc-25.2::GFP or a Pcdc-25.2::intron1::GFP transgene was generated and expression patterns of transgenes were examined. Interestingly, expression of *cdc-25.2* in the spermatheca was dependent on the presence of intron1 of *cdc-25.2,* suggesting that intron1 is necessary for the *cdc-25.2* expression in the spermatheca, and *cdc-25.2* is required for spermatheca development. Indeed, defective spermatheca morphology with the decreased number of spermatheca nuclei was observed in *cdc-25.2* mutants. Transcriptional regulators binding to intron1 were searched using database, ModEncode and MEME. Among seventy-eight putative transcription factors, CEH-1, CEH-39, DAF-12 and NHR-6 which are expressed in the spermatheca, were selected for further analyses. A loss-of-function *nhr-6* mutant showed defects in the spermatheca development, and RNAi depletion of *nhr-6* significantly suppressed expression of *cdc-25.2* in the spermatheca. Taken together, NHR-6 appears to activate expression of *cdc-25.2* by binding to intron1, and *cdc-25.2* promotes cell divisions during spermatheca development. This study was supported by grants NRF-2013R1A1A2009090 and 2015R1D1A1A01057488.

Keyword: *cdc-25.2, nhr-6,* spermatheca, transcriptional regulation

Session II

Neuroscience

The nutrient-responsive insulin signaling modulates chemosensory responses in *C. elegans*

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Neural modulation of sensory responses is essential for animal's survivals in changing environment. However, the cellular and molecular mechanisms how neuromodulators regulate sensory responses are still unclear. *C. elegans* hermaphrodites exhibit avoidance behaviors to a pheromone (called ascr#3 or C9) via the ADL neurons. Here, we show that ascr#3 avoidance behavior is modulated by insulin signaling in different feeding conditions. daf-2 insulin-like receptor mutants showed decreased ascr#3 avoidance, and daf-16 FOXO transcription factor mutants suppressed the daf-2 phenotype, indicating that ascr#3 avoidance behavior is regulated by insulin signaling. Cell specific rescue experiments revealed that DAF-2 and DAF-16 work in ADL. The expression of synaptic transmission molecules, Ras GTPase (rab-3) and synaptobrevin (snb-1) was reduced in daf-2 mutants indicating that daf-2 regulates synaptic transmission. INS-18 insulin which was expressed in intestine antagonized daf-2 function in ascr#3 avoidance. We also found that 6hr starvation increased ascr#3 avoidance by causing DAF-16 translocation in cytosol. These results suggest that insulin/IGF-1 signaling modulates sensory transduction in different nutrient status.

Keyword: Insulin, dietary restriction, sensory response, synaptic transmission, INS-18, DAF-2

Chemosensory GPCR SRI-14 are required for concentration dependent odor preference in *C. elegans*

Woochan Choi and Kyuhyung Kim*

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Animals must recognize and discriminate among thousands of chemicals in order to generate the correct behavioral responses. Understanding basic design of a sensory system in simple animals gives the opportunity to elucidate detailed molecular and neural mechanisms underlying sensory responses in higher animals. *C. elegans* detects a large number of odorants via three neurons pairs including the AWC, and elicit a multitude of olfactory behaviors (Bargmann, 1993, Cell). Previous genetic and behavioral experiments have identified set of signaling genes including olfactory receptors, but the knowledge is still limited. Specifically, the mechanisms of how the same odorants can elicit either attractive or aversive responses depending on the chemical concentrations are not known yet. First, we are trying to construct a comprehensive map of odorants and their receptors in *C. elegans*. We screened more than 50 volatile chemicals that are not tested previously, and found that animals respond to 13 volatiles. We further identified that the AWC neurons are required for chemotactic responses to these chemicals. We then performed candidate gene searches and found that the chemosensory GPCR mutants sra-13 or str-2, or un-linked mutant lsk46 exhibit specific defects in chemotactic responses to 2-Furyl methyl ketone, Ethyl pyruvate, or 1-propanol, respectively. Interestingly, we also found that chemosensory GPCR sri-14 are required for either attraction to low concentration DMTS and aversion to high concentration DMTS. Previous study showed that the sri-14 is expressed in the olfactory neurons AWC, and the nociceptive neurons ASH. We next found that the defect of DMTS chemotaxis in sri-14 mutants were restored when we expressed the wild-type sri-14 gene to the AWC neurons. We alsofound that Ca2+ response of AWC to low concentration DMTS was decreased in sri-14 mutants. These results indicate that sri-14 functions in the AWC neurons to mediate DMTS attraction. We are currently testing whether sri-14 also regulates ASH-mediated avoidance to DMTS.

Keyword: Neurobiology, Olfactory GPCR

Hypergravity hinders axonal development of motor neurons in *Caenorhabditis elegans*

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As space flight becomes more accessible in the future, humans will be exposed to gravity conditions other than our 1G environment on Earth. Our bodies and physiology, however, are adapted for life at 1G gravity. Altering gravity can have profound effects on the body, particularly the development of muscles, but the reasons and biology behind gravity's effect are not fully known. We asked whether increasing gravity had effects on the development of motor neurons that innervate and control muscle, a relatively unexplored area of gravity biology. Using the nematode model organism Caenorhabditis elegans, we examined changes in response to hypergravity in the development of the 19 GABAergic DD/VD motor neurons that innervate body muscle. We found that a high gravity force above 10G significantly increases the number of animals with defects in the development of axonal projections from the DD/VD neurons. We showed that a critical period of hypergravity exposure during the embryonic/early larval stage was sufficient to induce defects. While characterizing the nature of the axonal defects, we found that in normal 1G gravity conditions, DD/VD axonal defects occasionally occurred, with the majority of defects occurring on the dorsal side of the animal and in the mid-body region, and a significantly higher rate of error in the 13 VD axons than the 6 DD axons. Hypergravity exposure increased the rate of DD/VD axonal defects, but did not change the distribution or the characteristics of the defects. Our study demonstrates that altering gravity can impact motor neuron development. Currently, we are identifying the molecular mechanism of gravity's effect on axon development. Further studies will focus on synaptic development and age associated changes in DD/VD axons in hypergravity conditions. We are also planning to explore the microgravity effect on DD/VD axons using a 2D-clinostat. This study helps us to understand both microgravity's and hypergravity's roles on motor neurons.

Session III

Stress response and Molecular biology

Immune and stress response crosstalk to xenobiotic exposure by pathogen infection in the nematode *Caenorhabditis elegans*

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In the environment, most organisms are simultaneously faced with various chemical and biological stresses. Herein, we investigated how pathogen infection modifies an organism's response to chemical exposure. To explore this phenomenon, we conducted a toxicity study combined with pathogen infection, using the nematode, Caenorhabditis elegans; the pathogen, Pseudomonas aeruginosa; and various environmental chemicals. Increased toxicity was observed in *C. elegans*, pre-infected with the PA01, when subsequently challenged to nonylphenol (NP) and cadmium (Cd), whereas toxicity of silver nanoparticles (AgNPs) was rescued after pre-infection, which led to a mechanistic study focusing on AgNP exposure. A gene expression study revealed that most of the immune response genes activated by PA01 infection remained activated after subsequent exposure to AgNPs, suggesting that the acquired tolerance of *C. elegans* to AgNP exposure may be caused by boosted immunity resulting from PA01 pre-infection. Further functional genetic study revealed that the immune response pathway (i.e., PMK-1/P38 MAPK) were involved in the defense against AgNP exposure in PA01 pre-infected *C. elegans*, suggesting immune and stress response crosstalk to xenobiotic exposure. This study will aid in the elucidation of how pathogen infection impacts the way the defense system responds to subsequent xenobiotic exposure..

Keyword: *Caenorhabditis elegans*, *Pseudomonas aeruginosa*, Silver nanoparticle, Immunity, Stress response

Mitochondrial chaperone HSP-60 up-regulates p38 MAP kinase signaling to enhance anti-bacterial immunity

Dae-Eun Jeong[1#], Dongyeop Lee[1#], Sun-Young Hwang[1], Yujin Lee[1], Jee-Eun Lee[1], Mihwa Seo[2], Wooseon Hwang[1], Keunhee Seo[1], Ara B. Hwang[1], Murat Artan[3], Heehwa G. Son[1], Jay-Hyun Jo[1], Haeshim Baek[1], Young Min Oh[1], Youngjae Ryu[4], Hyung-Jun Kim[4], Chang Man Ha[4], Joo-Yeon Yoo[1*], and Seung-Jae V. Lee[1,2,3*]

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Mitochondria play crucial roles in innate immunity. How specific mitochondrial components modulate host immunity against pathogens is largely unknown. Here, we showed that HSP-60/HSPD1, a major mitochondrial chaperone, boosts anti-bacterial immunity through the up-regulation of PMK-1/p38 MAP kinase signaling in *C. elegans* and mammalian cells. We first identified 16 evolutionarily conserved mitochondrial components that affected the immunity of *C. elegans* against pathogenic P. aeruginosa (PA14). Among them, the mitochondrial chaperone HSP-60 was necessary and sufficient to increase resistance to PA14. We found that PMK-1 signaling, an evolutionarily conserved anti-bacterial immune pathway, was down-regulated by genetic inhibition of hsp-60, and up-regulated by overexpression of hsp-60. Overexpression of HSPD1, the mammalian ortholog of hsp-60, increased p38 MAP kinase activity in human cells, indicating an evolutionarily conserved mechanism. Further, HSP-60 that is localized in the cytosol physically bound and stabilized SEK-1/MAP kinase kinase 3, which in turn up-regulated PMK-1 and increased immunity. Our study suggests that molecular chaperones generated from bacteria-originated mitochondria protect host eukaryotes from pathogenic bacteria.

Keyword: anti-bacterial immunity, mitochondria, HSP-60, p38 MAP kinase signaling, *C. elegans, P. aeruginosa*

wts-1, the LATS kinase homolog of Hippo pathway regulates RNT-1 stability in *C. elegans*

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RUNX transcription factors have essential functions in mammalian development, cancer and stress response. The fine-tuned expression and regulation of RUNX is indispensable for proper development and response, which can prevent cancer development. We previously reported that RNT-1, the homolog of RUNX transcription family in Caenorhabditis elegans, is continuously degraded by Ubiquitin-Proteasome System in normal condition and stabilized by various stress conditions in the intestine. Especially, p38 MAPK pathway plays a crucial role for this RNT-1 stabilization. But the mechanisms for RNT-1 degradation, need to be discovered. To address this, we performed random mutagenesis experiments. We isolated a mutation in wts-1, the LATS kinase homolog of Hippo pathway, from this screening. Now we try to figure out this regulation by crosstalk between two signaling pathways and biological meaning. Our study can contribute to the understanding of regulatory mechanism of RUNX transcription factor, and this will help further understand development, cancer and stress response.

Keyword: RUNX, *rnt-1*, LATS, *wts-1*, Crosstalk, Oncogene

Caenorhabditis elegans VIG-1 protein is required for genomic stability

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The genome integrity is essential to the organisms for their survival and reproductive success. Genomic DNA is constantly damaged by a wide range of chemical and physical agents, both from the environment and intracellular metabolites. *Caenorhabditis elegans* VIG-1 is considered to be a putative component of RISC (RNA-inducing silencing complex) and to play a role in the micro-RNA pathway. However, we observed that the *vig-1* (*tm3383*) deletion mutant is sensitive to dsDNA molecules just like the wild-type animal. In an effort to search for the function of VIG-1, we analyzed the phenotypes of two different *vig-1* deletion mutants (*tm3383*, *ok2536*). We found that these mutants exhibit similar phenotypes such as decreased brood size, high incidence of males (*him*), and increased embryonic lethality. These phenotypes become more evident in the succeeding generations. This progressive increase in embryonic lethality and *him* phenotype may indicate that the germ line of *vig-1* mutants accumulates DNA damage slowly over generations. We thus postulate that *vig-1* mutants are defective in eliciting DNA damage response (DDR) against spontaneous DNA damage.

Keyword: *vig-1,* DNA repair, genomic stability, embryonic lethality, male ratio

Session IV

Survival and Reproduction

Genome-wide screening to find potential pro-longevity gut microbes using *Caenorhabditis elegans/Escherichai coli* system

Mooncheol Park[1,#], Jun-Seok Han[1,2,#], Jae-woong Lee[1], Min-Gi Shin[1], Jin-Hyuck Jeong[1], Ki-Sun Kwon[1,2]and Eun-Soo Kwon[1]

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[#]These authors contributed equally to this work

Gut microbes regulate a variety of host physiology such as metabolism, immunity and gastrointestinal homeostasis. However, studies on their effects on host aging are poorly understood due to their complexity. Here, we used a simple model system consisting of a gut microbe (Escherichai coli) – host (Caenorhabditis elegans) to identify genetic interactions between microbes and host aging. Using Keio Collection, 3985 E. coli single gene knock out library, we performed genome-wide lifespan screening, with scoring the survival rate of *C. elegans* feeding on each E. coli single gene mutant. Consequently, we isolated 18 E. coli mutants which extend the lifespan of *C. elegans* and 35 E. coli mutants to reduce the lifespan of *C. elegans* by the extent of 20% thresholds. In this study, we showed that worms lived ~30% longer on E. coli lic1 mutant which encodes one of central glucose metabolic enzymes. Interestingly, this lifespan extension is completely dependent on *C. elegans* AAK-2, catalytic α subunit of AMPK (AMP-activated protein kinase). In addition we found that phosphorylation of AAK-2 was increased on lic1 E. coli mutant. To find molecular mechanisms of inter-species genetic regulation of longevity, these experiments are in progress: metabolomics to find metabolites changed in E. coli mutants, phospho-proteomics to find proteins phosphorylated by AAK-2 in lic1 mutant-dependent manner, and lifespan assays using RNAi of possibly involved signaling pathway. These data will provide molecular mechanisms regarding how gut microbes regulate host aging.

Keyword: Host aging, gut microbes, *C. elegans, E. coli,* AAK-2

Using internal genomic regions as a conserved telomere maintenance mechanism

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Daechan Park[2] and Junho Lee[1]

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Telomeres are specialized nucleo-protein complexes that integrate the chromosomal end DNA sequence and specific binding proteins. Telomeres are required for maintaining genome integrity and guaranteeing successive divisions of the cell. Because of the limitation of DNA polymerase, cells cannot replicate the linear chromosomes completely. If there is only canonical DNA replication machinery, cells are doomed to lose the genome information from the end, telomeres. Most of infinitely dividing cells utilize specific telomere maintenance mechanisms. The most well-known mechanism is mediated by reverse transcriptase, telomerase. However, some cancer cells use telomerase-independent mechanism called ALT (alternative lengthening of telomeres). We identified telomerase-independent survivors from Caenorhabditis elegans telomerase deletion mutants1. These survivors utilized internal unique sequences (TALTs; Template for ALTs) to protect chromosome ends. Two different ALT survivors have their own TALTs depending on their original genetic backgrounds. We are currently trying to identify the protein factors that mediate ALT mechanism. Another question to address is which conditions or events induce the activation of ALT mechanism. Interestingly, similar telomere maintenance mechanism was identified in telomerase-deficient mouse embryonic stem cells (mESCs)2. These surviving cells showed amplification of telomeric and nontelomeric sequences, which have a similar structure to that of TALT of *C. elegans*. These conserved phenomena of telomere maintenance will reveal the basis for telomere evolution and the ability to adapt hazardous crisis.

Keyword: Telomere, telomere maintenance, ALT (Alternative Lengthening of Telomeres), TALT(Template for ALT)

Potential Roles of Nematode MGL-1 in Reproductive Plasticity in Response to Starvation

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Reproductive plasticity describes the ability of animals to alter their reproduction in unfavorable conditions. *Caenorhabditis elegans*, a free-living nematode, can temporarily cease oogenesis and embryogenesis to allocate energy use to survival over reproduction during prolonged starvation. It has been reported that the regulation of reproductive quiescence during starvation is independent of GLP-1/Notch signaling. *C. elegans* can convert olfactory cues to delay reproductive timing using neuroendocrine signaling, but olfactory and starvation cues are two different stimuli that must be distinguished from each other by the mechanisms and *loci* of neuronal *perception*. Thus, the molecular mechanisms that translate starvation into the cessation of reproduction remain largely uncharacterized. Here, we show that MGL-1, a glutamate receptor homolog in *C. elegans*, acts as a switch-like molecule to initiate reproductive plasticity in response to starvation. Genetic analysis of *mgl-1 (tm1811)* suggests that the starvation cue perceived by MGL-1 at the AIY neuron signals the worm to delay the onsets of oogenesis and embryogenesis. Furthermore, the *mgl-1 (tm1811)* mutant showed a significantly shorter lifespan with reduction in fat accumulation than wild-type worms in starvation. Our findings reveal molecular insights into the MGL-1–triggered early stage of translating starvation cues into the delay of reproduction. This study also suggests the presence of multiple types of reproductive plasticity that can be exercised *in a stimulus-specific* and *cell type–specific manner* for animal survival in harsh conditions.

Technical Lecture

COPAS Applications for High Throughput Screenings using *Caenorhabditis elegans*

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C.elegans was first introduced about a half-century ago as a model organism and thereafter has been a powerful model platform used for the study of a wide range of biological processes, including neurobiology, cell death, ageing, development, and RNAi. In the past decade, *C.elegans* has become a prominent tool for drug discovery, microbial pathogenesis, innate immunity, toxicological test and cancer study. In order of scale-up of screening to large library screen, instrumentation is a critical component. Typical bottle-neck in high throughput screening with *C.elegans* is to prepare worms sample in a microtiter plate and detection of end-points, such as imaging acquisition and data analysis. One of main benefits of instrumentation and/ or automation is to handle large quantitis of samples without user's continuous attention. COPAS, also known as a worm sorter, has played a pivotal role in allowing *C.elegans* as a whole animal high throughput screening system, including for the libraries of chemical, natural product and RNAi, and for toxicological test.This presentation will summarize the applications of COPAS in high throughput screening with *C.elegans*.

Keyword: *Caenorhabditis elegans*, High throughput screening, COPAS

Special Lecture

Poster abstract

1a.Sensory Food Cues Shorten *C. elegans* Lifespan via Inducing Neuroendocrine INS-6/Insulin-like Peptide

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Sensory neurons regulate the lifespan of several organisms, including C. elegans, Drosophila and mice. Perturbation of sensory neurons prolongs the lifespan of C. elegans by activating the DAF-16/FOXO transcription factor. Here, we show sensory neurons modulate lifespan through an insulin-like peptide, INS-6. We found that mutations that disrupt sensory neural function extended lifespan by enhancing the nuclear localization and transcriptional activation of DAF-16 in both neurons and non-neuronal tissues. These data suggest a tissue non-autonomous regulation of lifespan by sensory neurons. We then tested potential roles of neuroendocrine signaling via insulin-like peptides in this systemic lifespan regulation. We found that down-regulation of ins-6 in ASI and ASJ sensory neurons mediated the longevity of sensory mutants. Conversely, we found that the activation of ASI or ASJ neurons by providing food cues or using optogenetics increased the expression of ins-6 and decreased DAF-16 activity, which shortened lifespan in food-deprived conditions. Thus, food cues appear to regulate INS-6 in sensory neurons, which relay the longevity signals to non-neuronal tissues by regulating the activity of DAF-16. Our study provides mechanistic insights into how environmental sensory cues regulate organismal longevity by altering insulin/IGF-1 signaling via neuroendocrine peptides.

Keyword: Aging, sensory neurons, *C. elegans*, insulin-like peptides, DAF-16, endocrine signaling

1b.DEAD-box RNA helicases regulate lifespan via regulating distinct RNA-mediated processes in *Caenorhabditis elegans*

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RNA helicases, which unwind RNA duplexes and regulate RNA-protein interactions, play essential roles in RNA biology. However, whether RNA helicases affect organismal aging or lifespan remains largely unknown. By performing a large scale genetic screen employing *Caenorhabditis elegans* lifespan assays, we identified several RNA helicases that were required for the lifespan extension conferred by reduced insulin/IGF-1 signaling (IIS) (Seo et al., 2015, PNAS). We found that HEL-1, a DEAD-box RNA helicase, promotes longevity by increasing the activity of DAF-16/FOXO transcription factor in IIS pathway. In addition, we demonstrated that SACY-1, another DEAD-box RNA helicase whose homolog regulates RNA splicing, was required for the longevity of IIS mutants. Interestingly, we found that SACY-1 was also required for lifespan extension by various longevity interventions such as sensory impairment, mitochondrial respiration defects, germline removal, and dietary restriction. Thus, SACY-1 appears to play general roles in lifespan regulation, whereas HEL-1 specifically functions in the IIS pathway. Our study suggests that RNA helicases contribute to organismal lifespan extension via distinct RNA-mediated biological functions.

Keyword: Calcineurin interacting protein, KSP motif, ovulation, male mating, sperm activation

1c.RNA surveillance through nonsense-mediated mRNA decay is crucial for *C. elegans* longevity

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Appropriate protein and DNA quality control is crucial for organismal longevity. However, the role of RNA quality control in aging remains unexplored. Here, we show that nonsense-mediated mRNA decay (NMD), which degrades abnormal transcripts, promotes longevity by enhancing RNA surveillance in C. elegans. We found that the activity of NMD decreased during aging. In addition, smg-2, which encodes an RNA helicase crucial for NMD, was required for the long lifespan of various longevity mutants, including insulin/IGF-1 receptor daf-2(e1370) mutants. Knock-down of other NMD components, smg-1 through smg-5, also suppressed the longevity of daf-2 mutants. By performing mRNA sequencing analysis, we found that the long-lived daf-2(e1370) mutants displayed enhanced NMD activity, in a SMG-2-dependent manner. Our results suggest that reduced insulin/IGF-1 signaling increases lifespan through up-regulation of NMD activity, which may enhance RNA quality control. As both insulin/IGF-1 signaling and NMD pathways are evolutionarily well conserved, mammals including humans may be equipped with similar RNA surveillance systems to achieve longevity.

Keyword: Calcineurin interacting protein, KSP motif, ovulation, male mating, sperm activation

1d.KIN-4/protein kinase influences insulin/IGF-1 signaling-mediated longevity via interacting with DAF-18/PTEN in *C. elegans*

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Insulin/IGF-1 signaling (IIS) regulates aging and lifespan in various species including *C. elegans*. Despite the identification of many components in IIS, the roles of protein-protein interactions mediated by scaffolding proteins in lifespan regulation are poorly understood. In this study, we identified and characterized proteins containing PDZ domains (PDZ proteins), which organize protein-protein interactions for signal transduction, in IIS-mediated lifespan regulation. We first performed a targeted RNAi lifespan screen by knocking down each of 49 genes that encode PDZ proteins. We found that the genetic inhibition of *kin-4*, which encodes a MAST family protein kinase containing a PDZ domain, specifically shortened the long lifespan of *daf-2*/insulin/IGF-1 receptor mutants. We then identified lifespan-regulatory proteins that bound the PDZ domain of KIN-4 by performing a yeast two-hybrid screen and a subsequent secondary RNAi lifespan screen. We found that three genes that encode proteins that interacted with the PDZ domain of KIN-4 were required for the longevity of *daf-2* mutants. Among them we have been focusing on DAF-18/PTEN, a lipid phosphatase that acts downstream of DAF-2/insulin/IGF-1 receptor. We showed that the KIN-4 bound to the C-terminal PDZ-binding motif of DAF-18/PTEN. Specific disruption of the interaction between KIN-4 and DAF-18/PTEN suppressed the long lifespan of *daf-2* mutants. Thus, KIN-4 appears to promote longevity conferred by reduced IIS via interacting with DAF-18/PTEN. We are currently testing whether KIN-4 binding affects the activity of DAF-18/PTEN and subsequently IIS. Our study may provide insights into aging-regulatory mechanisms by PTEN and IIS through PDZ-mediated protein-protein interactions.

Keyword: Insulin/IGF-1 signaling, Aging, *C. elegans*, PDZ, KIN-4

1e.SBP-1 and MDT-15 protect animals from glucose-induced short lifespan by decreasing saturated fatty acid levels

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Glucose-rich diets shorten lifespan in *C. elegans*. To identify genes that mediate the effects of glucose on aging, we performed a genome-wide RNAi screen using a glucose-responsive *far-3p::GFP* reporter *C. elegans*. We first functionally characterized the RNAi clones that decreased the *far-3p::GFP* intensity. We found that SBP-1/sterol regulatory element-binding protein (SREBP) and MDT-15/mediator 15 (MED15) were necessary and sufficient for ameliorating the toxic effects of glucose on aging. Dietary glucose up-regulated SBP-1 and MDT-15, which in turn increased the expression of fatty acid desaturases to convert saturated fatty acids to unsaturated fatty acids. We also obtained data suggesting that this metabolic flow by SBP-1/MDT-15 prevents the accumulation of toxic and lifespan-decreasing intermediate metabolites in glycolysis, such as dihydoxyacetone. Thus, SBP-1/MDT-15 appears to moderate the glucose toxicity by enhancing glucose-to-fat metabolic flow by. For a follow-up study, we are currently characterizing the lifespan-regulatory roles of genes whose RNAi knockdown increased the *far-3p::GFP* levels. Our study will help designing strategies against diseases caused by abnormal carbohydrate metabolism, including diabetes.

Keyword: *C. elegans*, aging, metabolism, glucose, MDT-15, SREBP, fatty acid desaturases

1f.A point mutation in *daf-18/PTEN* uncouples longevity and developmental defects in daf-2/insulin/IGF-1 receptor mutants

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Insulin/IGF-1 signaling (IIS) regulates various physiological processes such as longevity, metabolism, development, reproduction, and behavior in many species, including C. elegans. Several daf-2/insulin/IGF-1 receptor mutant worms display dramatically increased lifespan and pathogen resistance, while exhibiting defects in development and reproduction. However, it remains unclear whether this pleiotropy can be uncoupled at specific steps of IIS pathway. Through a mutagenesis screen, we found that a point mutation in daf-18/PTEN, which encodes a lipid phosphatase in IIS, significantly suppressed developmental and reproductive defects with little effect on pathogen resistance in daf-2 mutants. We then showed that this point mutation in daf-18 retained extended lifespan and enhanced oxidative stress resistance in daf-2 mutants. This is intriguing as other known daf-18 mutations indiscriminately influence longevity and fitness phenotypes. We are currently investigating molecular mechanisms by which this daf-18 mutation influences downstream IIS factors for exerting its effects on worm physiology. Our study will provide insights into how a single component in an evolutionarily conserved signaling pathway coordinates distinct physiological processes such as longevity and development.

Keyword: *C. elegans*, insulin/IGF-1 pathway, daf-2, daf-18/PTEN, dauer, stress resistance

2a.*che-1, a salt-sensing neuron-specific transcription factor, regulates egg-laying by controlling transcription in vulval muscle.*

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che-1 encodes a C2H2-type zinc-finger transcription factor which is required for specification and function of salt-sensing ASE neurons. Human endogenous retrovirus (HERV) proviruses comprise 8% of human genome, and play important biological roles in genome evolution. HERV long terminal repeat (LTR) elements contain several regulatory sequences including promoters, enhancers, polyadenylation signals, and transcription factor binding sites. HERV-K119 LTR drives transcription exclusively in vulval muscle in C.elegans, and its transcriptional activity is dependent on che-1. We found out that che-1 lays eggs, hypersensitive to levamisole, but not to serotonin, indicating that che-1 is required for normal vulval muscle function. Currently, we are investigating how che-1 and salt-sensing regulate vulval muscle development and function.

Keyword: HERV-K, transcriptional regulation, *che-1*, vulva muscle

2b.*Mitochondrial Dynamics and habituation in Caenorhabditis elegans*

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Habituation, the simple non-associative learning, is a progressive decrease of behavioral response with repeated harmless stimulus. Habituation usually occurs when synaptic transmission is gradually decreased. Neurotransmitter release is affected by mitochondria which can produce ATP and regulate Ca2+ homeostasis. Mitochondria are dynamic organelles controlling their size and morphology via fission and fusion. Mitochondrial fission is regulated by Drp1 and Fis1, whereas mitochondrial fusion is regulated by Fzo1, OPA1, and Mgm1 in mammalian cells. In C. elegans, DRP-1 and FIS-1 are required for the fission whereas EAT-3 and FZO-1 are required for the fusion. In this research, we asked a question how mitochodrial dynamics affect habituation using C. elegans as a model system. To do this, we observed tap-mediated habituation in drp-1, eat-3, fis-1 and fzo-1 mutants. This research will provide the insight about the relationship between mitochodrial dynamics and animal behavior.

3a.Stretch sensitive neurons generate rhythmic motor patterns during forward movement in *C. elegans*

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Coordinated voluntary and involuntary movements produce rhythmic motor activities to drive locomotory behaviors of animals. In C. elegans, forward locomotion, which is initiated by contraction and relaxation of head muscles, exhibits the rhythmic motor pattern. However, the neuronal and molecular mechanisms to generate rhythmic motor pattern are not fully understood. Previously, it was shown that ablation of the SMB or SMD neurons caused increased reversal and in addition, SMB ablation increased wave width of sinusoidal movement (Gray et al., 2005). To confirm whether the SMB or SMD neurons function in forward movement, we genetically ablated the SMB or SMD neurons. Either SMB or SMD ablated worms exhibited increased reversal rate compared to wild-type animals. We next found that SMB or SMD exhibited rhythmic calcium transients that were induced by the head bending. To gain further insight into which molecules generate rhythmic calcium transient in SMB, we examined SMB neuronal calcium transients in synaptic transmission mutants including unc-13 (neurotransmitter release regulator), unc-31 (dense-core vesicle fusion activator), or unc-9 (innexin). All three mutants showed normal calcium dynamic in SMB, indicating that the rhythmic calcium influx of SMB is independent of synaptic transmission, and SMB may act as a stretch sensitive/proprioceptive receptor neuron to sense head muscle contraction. To identify the stretch receptor(s) in SMB, we examined expression pattern of 29 TRP and DEG/ENaC channels and found that few genes including unc-8 (DEG/ENaC channel) were expressed in SMB. UNC-8 protein is localized to cell bodies and processes around nerve ring. We also observed that SMB specific unc-8 RNAi causes increased wave width. We are currently analyzing unc-8 mutant phenotypes.

Keyword: SMB, SMD, UNC-8, stretch receptor neuron

3b.*crh-1 CREB influences developmental decision in Caenorhabditis elegans*

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Animals adapt to ever-changing environmental conditions via modulating developmental programs and metabolic status. In early C. elegans larvae, environmental cues including dauer pheromones, food and temperature regulate neuroendocrine signaling and govern entry into the dauer developmental stage. Previous studies showed that feeding state regulates dauer formation and this feeding state is not properly integrated in cmk-1 mutants to inappropriately enter into dauer stage under well-fed conditions (Neal et al., 2015). To investigate molecular mechanisms by which CMK-1 regulates dauer decision, we first examined whether crh-1 CREB influences dauer formation; CREB is a target of CaMKI (Kimura et al., 2002). We found that in well-fed and high concentration of pheromone conditions, crh-1 mutants inappropriately and transiently enter into pre-dauer (L2d) stage and exit into L2 or L3 normal developmental stages (Golden and Riddle, 1984; Avery, 2014). Compared to crh-1 mutants, cmk-1 mutants did not form transient L2ds or dauers in these conditions. The L2d formation of crh-1 mutants was induced by ascr#5 (C3) but not by ascr#2 or ascr#3 pheromones and was rescued by introducing wild-type crh-1 cDNA in the ASI neurons, suggesting that crh-1 has roles in the C3 pheromone signal transduction. Furthermore, srg-36/37 G-protein coupled receptors that mediate C3-induced dauer formation were required for the L2d formation of crh-1 mutants. To investigate the role of crh-1 in L2ds formation, we next tested several genes which are shown to be genetically interacted with crh-1 gene and found that calcineurin subunit a and b (cna-1 and cnb-1) mutants showed similar or even stronger transient L2d formation phenotype, and unc-43/Ca(2+)-calmodulin-dependent protein kinase II (CaMKII) mutants also induced transient L2d entry. Therefore, our results indicate crucial roles of crh-1 in L2d formation.

Keyword: crh-1, L2d, C3 pheromone

3c.*trp-1 and trp-2 TRPC channels modulate locomotive behavior of C. elegans*

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Locomotion is mediated by coordinated processes between sensory and motor system in animals. C. elegans generates sinusoidal locomotion via periodic bending of its head and body. The putative proprioceptive SMD motor neurons, that innervate to head muscles and project posterior processes to the tail, have been proposed to sense the body stretch and regulate head locomotion (White et al., 1986; Hendricks et al., 2012; Shen et al., 2016). However, the molecular mechanisms by which SMD regulates head movement are still unclear. To identify factors that mediate SMD-mediated head bending, we performed candidate gene search and found that TRPC channels, trp-1 and trp-2, are co-expressed in SMD (Feng et al., 2006). Since we did not observe altered locomotion defects in single mutants of either trp-1 or trp-2, we next generated trp-1 trp-2 double mutants and found that these animals exhibit ventral-directed circles during forward movement; we name this phenotype as ventral circling. Expression of either TRP-1 or TRP-2 by using a SMDD specific promoter rescued ventral circling phenotype of trp-1 trp-2 mutants, and SMDD axonal morphological defect mutants also showed the ventral circling. These results indicate that the ventral circling phenotype of trp-1 trp-2 mutants is due to the functional defects of SMDD. Ca2+ activity of SMD is correlated with head bending direction in wild-type animals, whereas Ca2+ activity SMDD but not SMDV is not correlated with head bending in trp-1 trp-2 mutants. These impaired correlation Ca2+ dynamic of SMDD with head bending in trp-1 trp-2 double mutants was restored by expressing trp-1 cDNA using the SMDD specific promoter. Furthermore, ectopic expression of the known stretch receptors, C. elegans trp-4 or Drosophila TRPγ in SMDD were sufficient to rescue ventral circling locomotion of trp-1 trp-2 double mutants. Currently, we are investigating stretch-activation of TRP-1 or TRP-2 by performing electrophysiology in heterologous systems. Taken together, we propose that trp-1 and trp-2 act as stretch receptors in the SMD motor neurons to sense the dorsal head movement and to correlate SMDD motor neuronal activity with head bending.

Keyword: *trp-1*, *trp-2*, TRPC channels, Stretch receptor, SMD, proprioceptive neurons

3d.*Gene regulatory networks underlying cell fate specification of a C. elegans sensory/inter/motor neuron-type*

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Neuronal specification and differentiation are orchestrated through external and internal molecules including transcription factors. However, the mechanisms of how specific transcription factors determine or specify neuronal cell fate during development are not fully understood. In C. elegans, the IL1 sensory/inter/motor neurons consist of six neurons that regulate the rate and pattern of spontaneous foraging movement (Kaplan et al., 1997). To identify molecular and cellular mechanisms by which the IL1 neurons are terminally differentiated, we searched for cis- and trans-acting factors that are necessary and sufficient for specification of the IL1 neurons. First, we performed promoter analysis of the set of genes that are specifically expressed in IL1s, including flp-3 neuropeptide gene (Kim et al., 2004). We found several cis-regulatory regions of which deletion caused decreased flp-3 expression in the IL1 neurons. More specifically, we identified a motif (referred to as the IL1L/R motif) that is necessary for flp-3 expression specifically in the IL1L/R neurons. We are currently testing whether this motif exists in promoters of other IL1-expressed genes and this motif is also sufficient for the expression in IL1L/R. In addition, we performed candidate gene searches and mutagenesis screens to identify trans-acting factor of IL1s, and found that several genes including ceh-43 or unc-86 regulate flp-3 expression in the IL1 neurons. We also isolated several mutant animals which show decreased flp-3 expression in the IL1 neurons. We are currently identifying molecular lesions in these mutants.

Keyword: *flp-3,* IL1, transcription factor

4a.Gluten uptake induces stress responses and germline apoptosis

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Gluten ingested from wheat products causes gluten toxicity because of incomplete digestion. Food proteins are supposed to be digested into small peptides and amino acids by peptidases. However, when gliadin was produced from gluten ingestion, gliadin peptides are incompletely digested in human, which affects intestinal cell structure and functions, and other cellular responses. Although several previous studies suggested possible underlying mechanisms of gluten toxicity at a cellular level, how gluten uptake affects reproduction in individuals at an organismal level remains elusive. In this study, we investigated effects of gluten uptake on stress responses and reproduction using *C. elegans* as an animal model. Induction of stress responding genes, *gst-4*, *cyp-35*, *hsp-4 and hsp-6* were examined after gluten feeding using transgenic *C. elegans.* Expressions of these genes were increased in intestine after gluten uptake, suggesting that ingested gluten is also a stressor in *C. elegans* and causes gluten toxicity. In addition, gluten uptake reduced fertility of adult hermaphrodites (16.3% ± 1.2) and increased germline apoptosis (2.37-fold ± 0.9) compared to control. These findings suggest that gluten toxicity induces stress responses not only in the digestive organ but also in the germline in *C. elegans*. Furthermore, *C. elegans* can be an excellent animal model system to investigate effects of food toxicity on reproduction.

Keyword: *flp-3,* IL1, transcription factor

5a.Toxicological Evaluation of Topoisomerase Inhibitors in a Model Animal, *Caenorhabditis elegans*

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Topoisomerase inhibitors inhibit the DNA replication of cancer cells, and therefore, they are used for the treatment of various human cancers such as colorectal, ovarian, small-cell lung, and testicular cancers. But topoisomerase inhibitors also inhibit rapidly dividing normal cells, are known to have side effects including the hematological toxicity and the development of secondary leukemia. In the present study, we evaluated the toxicity of etoposide, a clinical anticancer drug, in a model nematode *Caenorhabditis elegans* and 3T3-L1 normal murine cell line. Etoposide significantly retarded the growth, egg laying rate, and egg hatching rate in *C. elegans*. Etoposide injured reproductive tissue of *C. elegans*, so decreased the number of gonad cells and induced the enlargement of gonad cell nuclei. We also evaluated the toxicity of etoposide in 3T3-L1 murine preadipocyte cells. Etoposide potently inhibited the proliferation of 3T3-L1 cells, whose IC_{50} value was 9.81±1.83 μM. Etoposide also induced the cell and nuclear enlargement in 3T3-L1 cells. Reproductive toxicity and abnormal nuclear morphological changes seemed to correlate with the side effects of etoposide. In addition, in the present study, we also tested other topoisomerase inhibitors, irinotecan and daurinol in *C. elegans*. We suggest these experimental system, toxicological evaluation using both nematodes and 3T3-L1 cells, may be useful to study mechanism underlying the side effects of chemicals including topoisomerase inhibitory cancer drugs.

Keyword: *Abnormal nuclei, C. elegans, etoposide, irinotecan, germ cell toxicity*

6a.Neural circuit in the nictation behavior

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We previously reported that IL2 ciliated sensory neuron is important for nictation, a dauer-specific behavior(1). However, it still remains elusive which inter- or motor-neurons are involved in IL2-mediated nictation behavior. In this study, in order to determine the neural circuit involving IL2 neurons, we developed a PDMS microfluidic device to immobilize dauers and stimulate IL2 physically and quantitatively. For a more sophisticated calcium imaging, we activate IL2 optogenetically by expressing Chrimson, a red-shifted Channelrhodopsin(2). We selected candidate neurons for expressing GCaMP6 based on IL2 post-synaptic neurons in adult stage and close spatial proximity with IL2, and current results will be presented. Our study will contribute to elucidating the IL2 neural circuit in dauer for nictation behavior.

Keyword: Nictation, IL2, circuit, dauer, GCaMP, calcium imaging

6b.Reverse genetic screens identify new players of alternative lengthening of telomeres in *C. elegans*

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In the linear chromosome replication, DNA polymerase cannot replicate completely chromosome ends, thus cells are faced with 'end replication problem'. Shortened chromosome ends can be considered as DNA damage, and chromosome fusion may occur through DNA repair pathways. Therefore, eukaryotic cells, which have linear chromosomes, have specific repetitive sequence at the end of chromosomes to protect them: 'telomeres'. Telomeres can be maintained by two different ways. One is by telomerase that is a reverse transcriptase, and the other is by telomerase-independent mechanism. In human cancers, about 10 to 15 percent of cancer maintain telomere length by alternative lengthening of telomeres (ALT), which is a kind of telomerase-independent mechanism. ALT mechanism is not fully understood, but it is known that DNA recombination pathway is involved. We used Caenorhabditis elegans as a model organism to study ALT mechanisms. We performed reverse genetic screens via RNA interference against various factors which are related (are predicted to be related) with telomerase in telomerase deficient worms. Silencing two genes among candidate genes resulted in extending trans-generational lifespan in telomerase deficient worms. Although their telomere length was not heterogeneous, they maintained their telomere length over 30 generations. As expression pattern and function of these two genes are rarely known, we are performing experiments to identify the expression pattern and possible genetic interactions.

Keyword: Telomere, Alternative lengthening of telomeres (ALT), Reverse genetics

6c.Theoretical and experimental network analysis for novel functions of a neural subnetwork in *Caenorhabditis elegans*

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With its neurons and synapses identified, the neural system of *Caenorhabditis elegans* has become a nice platform to apply network analysis to uncover structure and function of a neural network. Inclusion of neurobiological information such as neuronal bilateral symmetry, inhibitory synapses and informational significance of gap junctions in the analysis of *C. elegans* connectome through community detection algorithm revealed 11 cluster modules, most of which were experimentally identified. However, one cluster module that has PVP neuron as a hub had no known function. Simulations through *C. elegans* neural interactome showed a potential role of PVP neuron in locomotion and oxygen sensing. Currently we seek to experimentally identify the role of the PVP-hubbed neural cluster module by genetic and laser ablation of PVP neurons. Successful observation of related *in vivo* phenotypes of PVP neuron-ablated animals will demonstrate significance of neural network studies in neurobiology.

Keyword: Connectome, neuron ablation, community detection algorithm, interactome

6d.Developmental decision of *C. elegans* L2/L2d affected by sensing environmental uncertainty

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How will living organisms make decisions when they encounter ‘uncertainty’ of the environment? *Caenorhabditis elegans* appears to make its first developmental decision in the L1 stage. It is obvious that the L1 larva would decide to enter the L2 pathway if steadily good environment is guaranteed. In harsh condition, the L1 larvae would decide to enter the L2d pathway to become dauer. But how would L1 worm decides when it is in an uncertain environment? An uncertain state in which the environment is unpredictable, it would be advantageous to enter L2d pathway. Although L2d stage larva has to pay slow development as a trade-off cost, only L2d stage larva can have an option for the choice between L3 stage and dauer. How L1 larvae can sense uncertainty and make developmental decision, and its underlying molecular mechanisms is not well known. We plan to check the hypothesis by comparing of development in steady and uncertainty state. Then we will investigate how uncertainty is sensed and regulated by sensory neurons using neuron ablation lines and reverse genetics of chemoreceptors.

Keyword: uncertainty, changing environment, L2/L2d, development

7a.Genome-wide screening to find potential pro-longevity gut microbes using *Caenorhabditis elegans/Escherichai coli* system

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In humans, the number of gut microbes reaches to 10 times of human cells. Gut microbes play a variety of roles in metabolism of non-digestible sugars, defense against pathogenic bacteria, supplying vitamin, and the development of the host immune system. Imbalance of gut microbes can cause many diseases such as not only gastrointestinal disorders but also obesity, cancer, metabolic syndrome, autoimmune diseases. Although most of these diseases are associated with aging, gut microbes and host correlation in aging is unknown. Here, we suggested that *Escherichia coli* and *Caenorhabditis elegans* as simple model system to study genetic interaction between gut microbes and host. Using Keio Collection, *E. coli* single gene knock out library, we performed genome-wide lifespan screening, measuring the survival rates of *C.elegans* fed *E.coli* single gene mutant. Consequently, we isolated 18 *E. coli* mutants which extend the lifespan of *C. elegans* and 35 *E. coli* mutants to reduce the lifespan of *C. elegans* by the extent of 20% thresholds. In this study, we show that *E. coli lic2* mutant extend the lifespan by activating *C. elegans daf-16* (mammalian FOXO ortholog). Currently, we are trying to reveal molecular mechanisms of this lifespan extension phenomenon. To find upstream of *daf-16* which is activated by *E. coli lic2* mutant, lifespan assay is in progress. And RNA seq is performed to find *daf-16* downstream target genes. These data will provide molecular mechanisms regarding how gut microbes regulate host aging.

Keyword: Host aging, gut microbes, *C. elegans, E. coli,* DAF-16

8a.Examining novel *C. elegans* behaviors in 3D cultivation conditions

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The nematode *C. elegans* is one of the premier experimental model organisms today. In the laboratory, they display characteristic development, fertility, and behaviors in a 2D habitat. In nature, however, C. elegans is found in three dimensional environments such as rotting fruit. To investigate the biology of C. elegans in a 3D controlled environment we designed a nematode cultivation habitat which we term the nematode growth tube or NGT-3D. NGT-3D allows for the growth of both nematodes and the bacteria they consume. Worms show comparable rates of growth and reproduction and lifespan when bacterial colonies in the 3D matrix are abundant. Using NGT-3D, we are observing feeding and egg laying behaviors. We noticed differences in feeding behavior patterns in 3D compared with 2D. We are currently quantifying these behaviors. In addition, NGT-3D allows us to cultivate worms for much longer periods than in 2D NGM. Normally adult worms will only develop until the 2^{nd} generation. However in NGT-3D, adult worms develop until the 3^{rd} or even 4^{th} generation. This allows us to observe behaviors and social patterns that we are not able to observe in 2D. Overall, NGT-3D allows us to broaden our understanding of nematode biology and ecology.

9a.Interaction of Nanomaterials with Telomere in *Caenorhabditis elegans*

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Alterations in telomere, DNA-protein structures that cap the ends of chromosomes, dynamics and length are associated with genomics instability, cancer and various age related diseases. Evidences are rapidly increasing that telomere homeostasis might be affected by environmental and occupational exposures of chemicals, such as, air pollution (i.e, particulate matter (PM), benzene/ toluene, polycyclic aromatic hydrocarbons (PAHs), N-nitrosamines, pesticides, lead, chromium, arsenic etc). Now-a-days, it is well established fact that the enormous nano-biological interactions could possess a potential threat to environmental and human health due to the increasing use of naomaterials. The aim of the present study was to investigative the effect of engineered nanomaterials on telomere homeostasis in the *in vivo Caenorhabditis elegans*, the nematode, models. We screened the life span of the *trt-1* (the telomerase mutant) and *mrt-2* (possess shortening of telomere with each generations) mutants with several nanomaterials (metallic nanoparticles – AgNP, AuNP; carbon nanomaterials – MWCNTs, graphene nanos, oxide nanomaterials – TiO2, SiO2) with. Among all the tested nanomaterials SiO2 and single layer graphene oxide (SLGO) were found as the most sensitive one to cause decrease in life span of the telomere related mutants. Moreover, both SiO2 and SLGO displayed lipofuschin accumulation, an aging biomarker, in a dose dependent manner in both *trt-1* and *mrt-2* strains. Conversely, the elongated telomere containing mutant worms [*pot-1(tm1620)*] and [*pot-2(tm1400)*] mutant did not displayed significant decrease in lifespan due to SiO2 and SLGO exposure. In addition, the SLGO showed more telomere specific effects by not affecting wild type (N2) lifespan in reference of their respective control. Long term exposure (10days), transgenerational (only mother exposed to SLGO) effects on telomere length, ageing related endpoints and related genes experiments are currently ongoing with wild type worms. Taken together, our results showed that nanomaterials could accelerate telomere shortening and in turn aging or age related diseases.

Keyword: Telomere, *Caenorhabditis elegans,* SiO2 and single layer graphene oxide (SLGO)

9b.Comparative effect between soil microbial extracts and OP50 on Cd toxicity in *C. elegans*

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In this study, we aimed to investigate how the modulation of gut microbiome can affect the response of the host to the environmental chemicals. To this end we selected *Caenorhabditis elegans* as model organism and cadmium(cd) as model chemicals *C.elegans* is very useful model to study the microbes-host response relationship not only because it is bacteriovore but also possess simple body plan with the intestine as the major body cavity Most importantly, it is easy to establish diverse gut microbiota containing worms starting with germ-free bleached eggs followed by exposing germ-free hatchlings to different microbial environments and it's self-fertilizing potentiality provides genetically homogenous populations onwards(M Berg et al. 2016). In lab condition, the experimental group of worms were fed with diverse microbes extracted from soil containing rotten fruits and vegetables while the control group was fed with only OP50 Next, survival and reproduction potentialities of the both groups were examined after exposing to cadmium at same dose The experiments are currently ongoing and we expect the marked differential response of experimental group, such as, longer life span and lesser sensitivity towards cadmium exposure than OP50 treated groups. Moreover, our future perspective is to identify the microbial communities and elucidate the mechanism by using OMICS techniques (metagenomics).

9c.Epigenotoxicity screening of various chemicals with transgenic worms *pkIs1582* (*let-858::GFP*) strain

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Epigenetic modifications, may or may not be transgenerational, cause modulation of gene expressions without changing the DNA sequence. Several epigenetic mechanisms including DNA methylation, histone modifications, non-coding RNAs (ncRNAs), and chromatin structure induced by various environmental stimuli such as light, temperature, food, chemicals etc. *Caenorhabditis elegans* is a good model system for studying epigenetic mechanisms because detailed epigenetic mechanisms involving ncRNAs and histone modifications are being elucidated, although it is considered that DNA methylation is absent. In this study, to evaluate the histone modification (methylation) potentiality of various environmental chemicals, we performed GFP florescence expression in a high-copy transgene array containing strain (*NL2507*) *pkIs1582*[*let-858::GFP + rol-6(su1006)*]. The strain *pkIs1582* (*let-858::GFP*) is a model of transcriptionally repressed locus bearing the heterochromatic histone modifications H3K9me3 and H3K27me3. We use *hpl-2* (*RNAi*), de-silencing of inactive transgene arrays in germ cells and cause higher GFP fluorescence in gonad arms of *pkIs1582* (*let-858::GFP*), as a positive control, and chemicals, such as, metals, endocrine disruptors, POPs and nanomaterials are exposed for 24 hours on young adult stage worms. As a result, a number of chemicals showed epigenetic changes on the germ cells′ histone modification status. For further study, we are planning to do transgenerational study to identify whether these epigenetic changes can pass through the next generations.

10a. Approach to anti-filarial drug discovery using molecular modeling of *Caenorhabditis elegans* calumenin

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Calumenin is a Ca^{2+} binding protein which localizes at the ER and functions in normal muscle and cuticle development. *Caenorhabditis elegans* calumenin protein shares 45% identity with its human counterpart and 72% identity with ones of two other nematodes (*Brugia malayi* and *Onchocerca volvulus*). These filarial worms cause vector-born infectious diseases such as lympathic filariasis and onchocerciasis, which show clinical symptoms like severe thickening of the skin and blindness. Diethylcarbamazine (DEC), albendazole, and ivermectin have been used for years to treat the diseases in a single or combinational use. However, none of them is effective in killing the long-lived adult worms (macrofilariae) and the evidence of drug resistance is growing. Therefore, it is urgent to develop novel macrofilaricidal drugs that affect new molecular targets. *C. elegans* calumenin mutant exhibited severe cuticle defects and hypersensitivity to the above microfilaricidal drugs at the adult stage. Molecular modeling predicted considerably distinct protein structures between human and *C. elegans* calumenin. Based on the predicted structure of *C. elegans* calumenin, chemical libraries were screened to identify possible inhibitors as novel anti-filarial drug candidates.

Meeting Participants

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