



**1st Chicago Area Worm Meeting**

**Thursday May 24th, 2018**

**University of Illinois – Chicago**

## **Keynote and Talks**

Moss Auditorium, Room 1020. College of Medicine Research Building (COMRB)

**Keynote: Stressing Worms to Reveal the Secrets of Health and Longevity (10:00-11:00)**  
Dr. Rick Morimoto, Northwestern University

1.- Behavioral search strategy optimization to cues of distinct physical nature (11:00-11:20)

Chance Bainbridge, Jocelyn McDonald, Zach Benefeld, Lucas Barickman, Wolfgang Stein, and Andrés Vidal-Gadea. Dept. of Biological Sciences, Illinois State University

2.- pirScan: a webserver to predict piRNA targeting sites and to avoid transgene silencing in *C. elegans* (11:20-11:40)

Wei-Sheng Wu, Wei-Che Huang, Jordan S. Brown, Donglei Zhang, Xiaoyan Song, Hao Chen, Shikui Tu, Zhiping Weng, and Heng-Chi Lee. Dept. of Molecular Genetics and Cell Biology, University of Chicago

3.- The ER protein ERG-28 and ERAD control the level of SLO-1 channels in *C. elegans* (11:40-12:00)

Timothy Cheung, Kelly Oh, and Hongkyun Kim. School of Graduate and Postdoctoral Studies, Chicago Medical School, Dr. William M. Scholl College of Podiatric Medicine. Rosalind Franklin University

### **Lunch (12:00-1:00. COMRB Lobby)**

4.- Dynamic SUMO remodeling drives a series of critical events during *C. elegans* meiotic divisions (1:00-1:20)

Amanda Davis-Roca, Nikita Divekar, Rachel Ng, and Sadie Wignall. Dept. of Molecular Biosciences, Northwestern University

5.- Developmental regulation of chromatin compaction by *C. elegans* synMuv B proteins (1:20-1:40)

Meghan Fealey and Lisa Petrella. Dept. of Biological Sciences, Marquette University

6.- A transcription factor titration mechanism for the establishment and maintenance of neuron identity (1:40-2:00)

Weidong Feng, Pauline Dao, and Paschalis Kratsios. Dept. of Neurobiology, University of Chicago

### **Coffee Break (2:00-2:30PM. COMRB Lobby)**

7.- Genetic basis of benzimidazole resistance in *Caenorhabditis elegans* wild isolates (2:30-2:50)

Steffen Hahnel, Stefan Zdraljevic, Briana Rodriguez, and Erik Andersen. Dept. of Molecular Biosciences, Northwestern University

8.- An autism-causing mutation disrupts axon termination by misregulating lysosome function (2:50-3:10)

Tyler Buddell and Christopher Quinn. Dept. of Biology, University of Wisconsin - Milwaukee

9.- Adapting tools from free-living nematodes to study chemosensory pathways in filarial nematode parasites (3:10-3:30)

Nicholas Wheeler, Troy Meikle, Lyric Bartholomay, and Mostafa Zamanian. Dept. of Pathological Sciences, University of Wisconsin - Madison

10.- A serotonergic circuit that couples behavioral and anti-aging effects of a social signal (3:30-3:50)

Erin Aprison and Ilya Ruvinsky. Dept. of Molecular Biosciences, Northwestern University

### **Posters (4:00-6:00PM. College of Medicine West Tower (CMWT) Room 220)**

Page numbers shown below also denote poster-board locations in exhibition room

Available area on poster-board is 4'x4'. Therefore, posters should be no larger than 46"x46" (116cm. x 116cm.)

11.- Dendrite morphology under well-fed and starvation conditions: a comparison between IL2 and FLP arbors

12.- Hermaphrodite recognition of sex pheromones

13.- EXC-4/CLIC and G-protein signaling during tubulogenesis in *C. elegans*

14.- IL2 neuronal arborization in different alleles of *daf-9* and *daf-12* mutants

15.- *C. elegans* adult males lacking *hlh-3* function show altered *daf-7* expression in the ASJ neurons

16.- Effects of Martian physics on terrestrial organisms

17.- Epistatic loci or small-effect variants might underlie bleomycin response variation in *C. elegans*

18.- Mechanisms involved in repression of germline genes in somatic cells of *C. elegans*

19.- AIR-2/Aurora B kinase activity is essential for key events in *C. elegans* oocyte meiosis

20.- The effect of *hlh-3(lof)* on body wall muscle in *C. elegans*

21.- Genetic variation underlies differential responses to docetaxel and zinc treatments in *Caenorhabditis elegans*

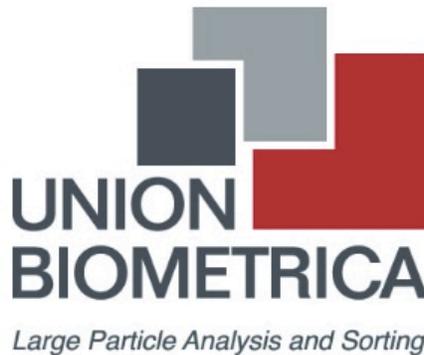
22.- *C. elegans* as a Neurological Model for Duchenne Muscular Dystrophy

23.- Oligomerization mediates self-stabilizing cortical asymmetry of the keystone polarity protein PAR-3

- 24.- The genetic basis of natural variation in chemical communication
- 25.- The role of histone modifications in shaping post-mitotic neuronal identity
- 26.- The miR-44 family of microRNAs are necessary for sperm function in *C. elegans*
- 27.- The PBAF chromatin-remodeling complex is required for cholinergic motor neuron subtype identity
- 28.- The search for magnetic particles in *C. elegans*
- 29.- Probing the molecular mechanism of receptor tyrosine kinase enzymatic activation through the analysis of heterodimers of the *C. elegans* fibroblast growth factor receptor (FGFR), EGL-15
- 30.- VCs require HLH-3 function to assume their terminal differentiation state
- 31.- Identifying new components that mediate fibroblast growth factor receptor signaling in *C. elegans*
- 32.- Development of a novel assay to identify genes involved in host infection by parasitic nematodes
- 33.- Decreased mating ability may play a role in *C. elegans* male low fertility at high temperature
- 34.- Cell to cell spreading of TDP-43 C-terminal fragments in *Caenorhabditis elegans*
- 35.- Dynamics and recruitment of the polarity protein PAR-3 in the P1 cell
- 36.- Natural variation in *C. elegans* arsenic-induced toxicity is explained by differences in branched chain amino acid catabolism
- 37.- Using *C. elegans* to teach the genetic basis of disease and behavior

Pages 38 – 40: List of Participants

**Many thanks to our sponsors:**

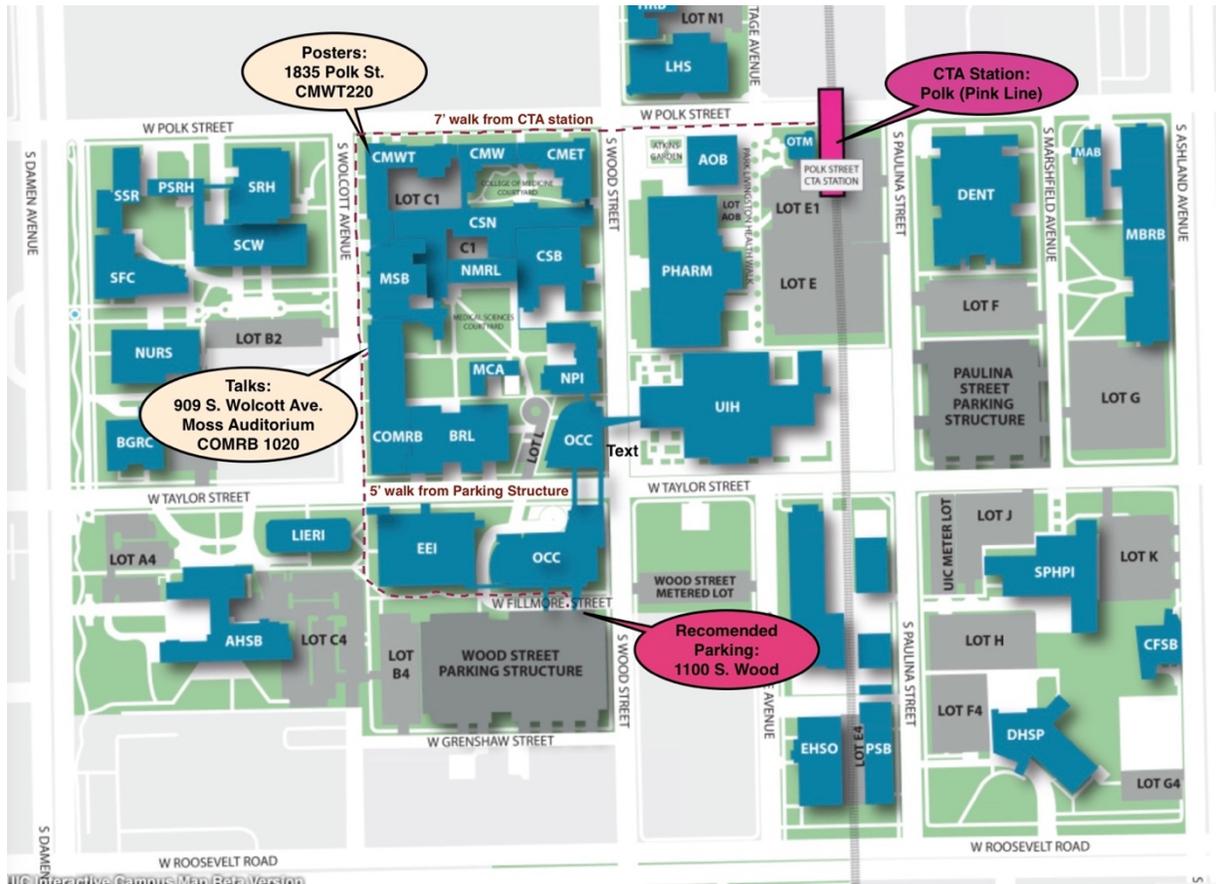


**The Rice Institute for Biomedical Research at Northwestern University**

Also, thanks to Cold Rain Studios, for designing our logo, and to Laura Scott and Monica Horga from the Dept. of Physiology and Biophysics at UIC College of Medicine for administrative assistance.

**For WiFi access during ChAWM:** connect to the “UIC-Guest” network, open a web browser, then authenticate with the login: chawm\_2 and the password: psyc4non

**Map and Directions:**



- **By Public Transportation:** the closest train station is “Polk” on the Pink Line. It’s a 5-10-minute walk from this station to the College of Medicine Research Building (COMRB) at 909 S. Wolcott Ave., where Moss Auditorium is located (COMRB1020). The "Illinois Medical District" stop on the Blue Line (Damen Ave. exit) is also near, but a longer (10-15 minute) walk. CTA Bus Routes 12 and 157 have several stops nearby.
- **Driving:** COMRB is accessible from Interstate 290 (Kennedy Expressway) and several major roads.
  - o From either direction on 290, take Exit 28A (Damen Ave./Illinois Medical District). Travel south on Damen Ave. then make a Left onto Taylor St. If dropping off at COMRB, make a Left onto Wolcott St. If driving to Parking Structures, make a Right on S. Wood St. or a Left on Paulina St.
  - o If travelling on local roads, UIC College of Medicine is accessible from several major streets: Ashland Ave. on the East, Roosevelt Rd. on the South, Damen Ave. on the West, Polk St. on the North, and it is traversed by Taylor St.
- **Parking:** There are several UIC lots open to the public.
  - o The rate for the full day (anything more than 4.5 hours) is \$13. For more rate details click [here](#).
  - o For information on all available visitor parking lots at UIC’s Westside campus, check [this page](#).
  - o The nearest visitor parking lots are B2 and C4 (see map), but these are small and may be full. Instead we recommend the Wood Street Parking Structure, at [1100 South Wood St.](#) (marked on map), or the Paulina Street Parking Structure at [915 South Paulina St.](#) (corner of Taylor St. and Paulina St.). For directions to either of these parking structures, click on their addresses above.

# ABSTRACTS

## **Behavioral search strategy optimization to cues of distinct physical nature**

Chance Bainbridge, Jocelyn McDonald, Zach Benefeld, Lucas Barickman, Wolfgang Stein and Andrés Vidal-Gadea

Dept. of Biological Sciences, Illinois State University

To successfully navigate their surroundings, animals must decide how much attention they dedicate to the variety of environmental stimuli they routinely encounter. This decision is often reflected on the nervous system allocation of resources to different stimuli (e.g. humans rely on vision more than touch). In nature, animals must reconcile the physical nature, and the saliency of stimuli to optimize search strategies using finite behavioral repertoires. Most work in the field has focused on orientation strategies specific to highly salient stimuli (e.g. temperature or chemical gradients). It remains unclear how animals optimize their behavioral strategies for stimuli of different physical nature.

Like many animals, the nematode *C. elegans* detects a large variety of stimuli and responds using a small well described repertoire of orientation strategies. Here we use *C. elegans* to investigate how it optimizes navigation to physically distinct stimuli of different salience. We recorded the worms orienting to temperature, chemical, or magnetic field stimuli and analyzed heading and orientation strategies. Worms appear to orient differentially in the presence of a temperature gradient by increasing velocity and clustered reversals (i.e. pirouettes) as a search strategy that was not observed in magnetic orientation or chemotaxis. Conversely, pirouettes appear suppressed in chemical gradients while gradual turning (weathervaning) is increased. These results suggest that *C. elegans* uses distinct orientation strategies depending on the physical nature of a stimulus.

**pirScan: a webserver to predict piRNA targeting sites and to avoid transgene silencing in *C. elegans***

Wei-Sheng Wu, Wei-Che Huang, [Jordan S. Brown](#), Donglei Zhang, Xiaoyan Song, Hao Chen, Shikui Tu, Zhiping Weng and Heng-Chi Lee

Dept. of Molecular Genetics and Cell Biology, University of Chicago

In diverse animals, PIWI Argonaute and its associated piRNAs play an essential role in genome defense against foreign nucleic acids, such as transposons. The vast majority of the 15,000 sequence-distinct piRNAs encoded by the *C. elegans* genome however are not complementary to transposons, and their targets have proven enigmatic. It has been known for decades that transgenes carrying foreign nucleic acids, such as GFP or mCherry, are frequently silenced in the germline of *C. elegans*. Recent studies have shown that the PIWI protein PRG-1 plays a critical role in triggering silencing of these transgenes. Our recent elucidation of the degenerate piRNA targeting rules suggests that piRNAs are able to target a diverse array of sequences and thereby guard against the expression of foreign nucleic acids. Specifically, piRNAs require near perfect complementarity to their targets within the 2nd to 7th nucleotide seed region. Outside of this seed region, piRNAs tolerate a few mismatches. In order to avoid the persistent silencing of transgenes in the *C. elegans* germline, we have developed pirScan. pirScan allows researchers to predict piRNA targeting sites within any input sequence and to find silent mutations that, when introduced, break targeting rules so that piRNA-dependent germline silencing will be much less likely to occur. Using pirScan's algorithm to introduce silent mutations, we have successfully expressed silencing-prone GFP, mCherry, and Cas9 transgenes in the worm germline. pirScan represents a new strategy that can be employed by the worm community to circumvent the decades-old barrier to germline transgene expression.

## The ER protein ERG-28 and ERAD control the level of SLO-1 channels in *C. elegans*

Timothy Cheung, Kelly Oh and Hongkyun Kim

School of Graduate and Postdoctoral Studies, Chicago Medical School, Dr. William M. Scholl College of Podiatric Medicine. Rosalind Franklin University.

The SLO-1 BK channel is the large conductance, voltage- and calcium-dependent potassium channel that controls synaptic transmission and muscle excitation. However, the mechanism underlying BK channel trafficking, thus controlling channel level, has yet to be clearly defined. In a previous *C. elegans* genetic study, the ER membrane protein ERG-28 was identified as a regulator of SLO-1 trafficking from the ER to the Golgi complex; SLO-1 level is drastically reduced in the absence of ERG-28. We hypothesize that without ERG-28, SLO-1 is recognized by the ERAD (ER associated degradation) system and inactivation of ERAD increases SLO-1 channel level. Using a candidate gene approach, we identified SEL-11, an ER-resident E3 ubiquitin ligase, as an important regulator of SLO-1 degradation in *erg-28* mutants. Introduction of *sel-11* mutation to *erg-28* mutant resulted in significant recovery of SLO-1 levels at the plasma membrane. Moreover, the recovered SLO-1 at the plasma membrane is functional as *sel-11* mutation suppressed *erg-28* mutant phenotype. We also examined the relationship between *sel-11* and *ddi-1*, a gene encoding an aspartic protease that participates in the degradation of SLO-1 in *erg-28* mutant. The SLO-1 channel levels of *ddi-1; sel-11 erg-28* triple mutant and *sel-11 erg-28* or *ddi-1; erg-28* double mutants were not significantly different, suggesting a shared pathway of SLO-1 degradation between SEL-11 and DDI-1. Together, our data show that the overall level of SLO-1 channel is regulated in the ER by the concerted action between ERG-28 and the ERAD machinery, in which SEL-11 and DDI-1 are main components.

## **Dynamic SUMO remodeling drives a series of critical events during *C. elegans* meiotic divisions**

Amanda Davis-Roca, Nikita Divekar, Rachel Ng and Sadie Wignall

Dept. of Molecular Biosciences, Northwestern University

*C. elegans* oocytes utilize a unique chromosome segregation mechanism to facilitate meiosis. Instead of end-on kinetochore attachments, chromosome segregation depends on a complex of proteins containing AIR-2/Aurora B kinase that forms a ring around the center of each homologous chromosome pair. These ring complexes (RCs) facilitate congression and then are released from chromosomes in anaphase and left at the spindle midzone, where they begin to disassemble as anaphase progresses. We have uncovered mechanisms underlying the dynamic regulation of these ring complexes (RCs), revealing a strategy by which protein complexes can be progressively remodeled during cellular processes. We find that the stability of the RC is regulated by a balance of SUMOylation and deSUMOylation activity. During prometaphase, the SUMO protease ULP-1 is targeted to the RCs but is counteracted by SUMO E2/E3 enzymes; then in early anaphase the E2/E3 enzymes are removed, enabling ULP-1 to trigger RC disassembly and completion of the meiotic divisions. Moreover, we found that SUMO regulation is essential to properly connect the RCs to the chromosomes and then also to fully release them in anaphase. Altogether, our work demonstrates that dynamic remodeling of SUMO modifications facilitates key meiotic events and highlights how competition between conjugation and deconjugation activity can modulate SUMO homeostasis, protein complex stability, and ultimately, progressive processes such as cell division.

## Developmental regulation of chromatin compaction by *C. elegans* synMuv B proteins

Meghan Fealy and Lisa Petrella

Dept. of Biological Sciences, Marquette University

A central question in development is how chromatin is organized and regulated to ensure proper gene expression and cell fate. During early embryo development, before gene expression is globally upregulated, chromatin is found in an open state. As development proceeds and cells differentiate, the genome compacts and becomes organized into open and closed domains. Genes required for a particular fate remain open and poised for transcription factor binding, while genes not needed for that fate are further compacted and sequestered to the nuclear periphery. Although this process is highly regulated, many of the proteins involved in this progression are unknown. Loss of *C. elegans* synMuv B proteins causes changes in the developmental regulation of chromatin and gene expression. Many synMuv B mutants, including *lin-15B*, show ectopic expression of germline genes in somatic cells and a high temperature larval arrest (HTA) phenotype. The HTA phenotype is rescued by knockdown of chromatin modifiers, suggesting that synMuv B proteins regulate gene expression programs epigenetically. To determine this, we investigated if synMuv B proteins function to regulate developmental chromatin compaction utilizing extrachromosomal arrays and fluorescent in-situ hybridization. SynMuv B mutants display a developmental delay in both general genome-wide chromatin compaction and compaction of tissue specific loci. The timing of compaction is sensitive to temperature in both wild type and mutant embryos but is delayed longer into mid-embryonic development in mutants. The delay in mutants results in open chromatin during the developmental window when ectopic expression of germline genes in somatic tissues begins. Using temperature shift assays, we found that the crucial developmental time period for the HTA phenotype is the same as the time period when synMuv B mutants display open chromatin at high temperature. Open chromatin during this period may allow germline genes to be poised for ectopic expression in somatic tissues of synMuv B mutants. Interestingly, we found that the most anterior cells of the intestine are the last cells to adopt compact chromatin, suggesting an anterior to posterior pattern of chromatin compaction that has not been previously described. Understanding this pattern and synMuv B regulation of chromatin compaction will help elucidate pathways used to achieve proper gene expression and correct development.

## **A transcription factor titration mechanism for the establishment and maintenance of neuron identity**

Weidong Feng, Pauline Dao and Paschalis Kratsios

Department of Neurobiology, University of Chicago

Neuron identity transformations have been widely described in nervous system development across species. Such transformations usually occur due to genetic removal of a specific regulatory factor with dual function. The regulatory factor can often activate genes that determine a specific neuronal identity and simultaneously repress genes that define an alternative identity. Here, we provide evidence for a novel, transcription factor (TF) titration mechanism of neuron identity transformation using the cholinergic motor neurons (MNs) of the *Caenorhabditis elegans* ventral nerve cord as a model. We find that removal of the conserved terminal selector-type transcription factor *unc-3/Ebf* results in partial neuron identity transformation characterized by loss of expression of cholinergic MN identity genes and concomitant gain of expression of GABAergic MN features. This dual UNC-3 function in cholinergic MNs is determined by the limited pool of available molecules of LIN-39 (Scr/Dfd/Hox4-Hox5), a mid-body Hox protein necessary for direct activation of both cholinergic and GABAergic MN identity genes. UNC-3 secures expression of cholinergic MN features through synergy with LIN-39 on the cis-regulatory region of cholinergic identity genes, thereby exhausting the pool of available LIN-39 molecules that could activate GABAergic MN features. Lowering UNC-3 or increasing LIN-39 protein levels in cholinergic MNs results in activation of GABAergic identity genes. This LIN-39-mediated titration mechanism operates continuously, from development throughout adulthood, to ensure maintenance of cholinergic and exclusion of GABAergic MN features. The mechanism of TF titration represents a simple, yet economical strategy for neuron identity transformation with implications for the evolution of neuronal cell types.

## **Genetic basis of benzimidazole resistance in *Caenorhabditis elegans* wild isolates**

Steffen Hahnel, Stefan Zdraljevic, Briana Rodriguez and Erik Andersen

Department of Molecular Biosciences, Northwestern University

Benzimidazoles (BZ) are essential components of the limited chemotherapeutic arsenal available to control the global burden of parasitic nematodes. In veterinary medicine, extensive treatment on livestock has generated geographically widespread BZ resistance and raised the concerns that selective pressure of mass drug administration may lead to reduced cure rates in human populations as well. Although single nucleotide mutations in beta-tubulin genes were identified to be major determinants of BZ resistance in parasitic nematodes, there is increasing evidence that BZ resistance is a polygenetic trait. To improve treatment strategies, a more detailed understanding of the mechanisms leading to BZ resistance is of high importance.

Since population genetics approaches are strongly limited in helminth parasites we performed genome wide association (GWA) studies on a wild isolates panel of the free-living model nematode *C. elegans*. We evaluated the variation in BZ responses of 240 genetically divergent strains using the COPAS BIOSORT platform, which allows high-throughput measurement of multiple fitness traits including brood size and growth rate. Subsequent association mapping was performed with a genome-wide set of 13,540 single nucleotide variants as genomic markers leading to the identification of several quantitative trait loci (QTL) that contribute to resistance. Beside QTL that were found to be common among different BZ, some QTL appear to be drug-specific.

Additionally, we did an in-depth analysis on *ben-1*, a beta-tubulin gene coding for the major drug target in *C. elegans*. This approach revealed unexpected genetic variation among strains at this locus including coding variants, insertions, and deletions that could be linked to resistance. These findings provide further evidence for the complexity of BZ resistance and have the potential to expand our knowledge of the genetic mechanisms that contribute to anthelmintic drug response in nematodes.

**An autism-causing mutation disrupts axon termination by misregulating lysosome function.**Tyler Buddell and [Christopher Quinn](#)

Dept. of Biology, University of Wisconsin - Milwaukee

The molecular mechanisms that underlie autism remain elusive, partly because of the large number of gene variants that contribute to the disorder. The Timothy syndrome mutation is a gain of function variant in a voltage gated calcium channel (VGCC) that can cause autism on its own, without contributions from other variants, providing a powerful avenue of investigation into the causes of autism. However, the Timothy syndrome mutation has not been linked to any specific defect in neurodevelopment and the mechanisms that underlie its role in autism are unknown. Here, we show that an *egl-19(gof)* mutation, which is equivalent to the Timothy syndrome mutation, causes defects in PLM axon termination in *C. elegans*. Our genetic analysis indicate that wildtype VGCCs negatively regulate PLM axon termination. Moreover, we find that VGCCs localize to a cellular compartment that is adjacent to lysosomes and that the axon termination defects caused by the *egl-19(gof)* Timothy syndrome mutation are suppressed by a mutation that disrupts lysosome function. Together, these results suggest that VGCCs can function with lysosomes to negatively regulate axon termination. Moreover, these results suggest that the Timothy syndrome mutation misregulates lysosomes to cause axon termination defects and reveal a potential role for lysosomes in autism.

## **Adapting tools from free-living nematodes to study chemosensory pathways in filarial nematode parasites**

Nicholas Wheeler, Troy Meikle, Lyric Bartholomay and Mostafa Zamanian

Dept. of Pathological Sciences, University of Wisconsin - Madison

Clade III filarial nematode parasites include the etiological agents of onchocerciasis (river blindness) and lymphatic filariasis (elephantiasis). These neglected diseases inflict high morbidity and mortality throughout much of the world and demand new methods of disease intervention. The mechanisms by which filarial nematode parasites invade and migrate through host tissues is not well understood. We explore the role that parasite chemosensory pathways play in mediating these critical behaviors. Our hypothesis, motivated by both experimental and genomic data, is that chemosensory signaling is required for the establishment of filarial infections and that parasite chemoreceptors are lucrative targets for disease intervention. Borrowing from methods established in the model nematode *Caenorhabditis elegans*, we show that the infective L3 stage of the human parasite *Brugia malayi* exhibits robust chemotactic preferences with respect to a panel of host-associated compounds. We have carried out phylum-wide discovery and classification of chemosensory proteins and have identified filarid-specific expansions of chemosensory receptors. Using RNA-Seq and droplet digital PCR (ddPCR), we show that many of these receptors are expressed only in specific stages and enriched towards the anterior region of the parasite. To build on these observations, we are working to 1) establish microfluidic assays to improve the throughput of chemotaxis assays, 2) use RNA interference (RNAi) to assess whether chemotactic preferences are modulated by conserved signaling molecules in the nematode chemosensory pathway, and 3) develop a heterologous expression platform in the model nematode *Caenorhabditis elegans* to associate chemosensory cues with chemoreceptor activation. We expect that chemosensory behaviors are relevant to the vector and definitive host stages of *B. malayi* parasitism, as well as to other closely-related filarial nematode parasites.

## **A serotonergic circuit that couples behavioral and anti-aging effects of a social signal**

Erin Aprison and Ilya Ruvinsky

Dept. of Molecular Biosciences, Northwestern University

Decrease in germline quality is a major cause of reproductive aging. Signals from the soma contribute to regulation of germline senescence, making it imperative to understand the underlying mechanisms. We discovered that, in *C. elegans*, a male sex pheromone ameliorates the effects of aging on the hermaphrodite germline via a serotonin circuit that also regulates behavioral states that govern how animals explore their environment. Thus, male pheromones facilitate reproductive success by altering behavior and physiology of potential mates. We argue that the shared reliance on the same neuronal circuit couples these two types of responses that unfold on different time scales. We also show that pharmaceuticals that potentiate serotonin signaling improve germline maintenance, suggesting that therapeutic interventions using available compounds could efficiently forestall reproductive aging.

## **Dendrite morphology under well-fed and starvation conditions: A comparison between IL2 and FLP arbors**

Rebecca Androwski, Janet Goelzer; Cassandra Smith and Nathan Schroeder

Dept. of Neuroscience. University of Illinois - Urbana-Champaign.

Dendrite morphology plays a key role in proper neural signaling. Environmental stress can significantly influence the shape of dendrites. The two FLP neurons located in the head begin to arborize rapidly during L4 and persist through adulthood. FLP branches cover the head of the animal laying between the muscle and hypodermis. During the stress-induced dauer, four quadrant IL2 neurons arborize similarly to the FLPs, extending arbors out to the body wall that cover the head of the worm. Unlike the FLPs, the IL2s resorb their arbors upon dauer recovery. Using a forward genetic screen, we identified the membrane bound receptor, DMA-1 as essential for dauer-specific IL2 arborization. DMA-1 was previously shown to regulate FLP branching. We found that DMA-1::GFP is expressed in the IL2 during dauer. Similar to results in the FLP neurons, IL2-specific expression of DMA-1 rescued the arborization defects during dauer; however, overexpression of DMA-1 in dauer IL2 neurons had no obvious effect on IL2 branching. DMA-1 interacts with several secreted and transmembrane proteins to regulate FLP arborization. We found that the IL2s use identical binding partners during IL2 dauer arborization. Our data suggest that all highly arborized neurons in *C. elegans* use a similar protein complex to regulate branching. Because several DMA-1 ligands localize to the surrounding hypodermis and are secreted from the muscle and these signals are present to bind with DMA-1 during FLP arborization in adult animals, we hypothesized that overexpression of DMA-1 in IL2 during adult could be sufficient to induce arbor formation outside of the dauer stage. However, overexpression of DMA-1 in adult IL2s did not cause obvious branching suggesting that additional dauer-specific components regulate IL2 arborization during dauer. The unfolded protein response (UPR) regulates DMA-1 production during FLP arborization, resulting in decreased branches when the UPR is blocked, but the UPR is not necessary for IL2 arborization. However, the FOXO transcription factor, DAF-16 is able to compensate for a blocked UPR by upregulating alternative protein degradation pathways. Consistent with this, we found that *daf-16(m27)*; *daf-7* partial dauers and autophagy mutant *unc-51* are defective for IL2 arborization.

## Hermaphrodite recognition of sex pheromones

Erin Aprison and Ilya Ruvinsky

Dept. of Molecular Biosciences, Northwestern University

Sex pheromones contribute to reproductive success by attracting potential mates and influencing their behavior and physiology. In *C. elegans*, the prominent difference between otherwise similar blends of pheromone molecules secreted by hermaphrodites and males is that the former contains more *ascr#3*, while the latter has more *ascr#10*. We have demonstrated that the ratio of concentrations of these two similar molecules is sufficient to convey the sexual identity of the pheromone blend. I will present our latest efforts to understand how this concentration ratio, and therefore the sexual identity of the pheromone, is decoded by the nervous system of hermaphrodites. The first set of results focuses on sensory neurons and argues that the processing of *ascr#10* and *ascr#3* signals relies on distinct neurons and molecular mechanisms. The second set of results deals with the fact that despite having nearly identical chemical structures, *ascr#10* and *ascr#3* have antagonistic functions. Our results suggest that this peculiar property may arise from the organization of the neuronal circuits that detect *ascr#10* and *ascr#3* and be a part of the ratio-detection mechanism. We expect our studies to provide a detailed picture of the neuronal circuits that process important social signals and serve as a useful paradigm for understanding how the nervous system integrates diverse, including contradictory inputs, *en route* to decisions.

## **EXC-4/CLIC and G-protein signaling during tubulogenesis in *C. elegans***

Anthony Arena and Dan Shaye

Dept. of Physiology and Biophysics. University of Illinois - Chicago.

Biological tube formation (tubulogenesis) is a key process during vascular development and angiogenesis. The chloride intracellular channel (CLIC) family was first implicated in tubulogenesis by the discovery that EXC-4, a worm CLIC, is required for *C. elegans* excretory canal (*CeEC*) tubulogenesis. Following this discovery, it was shown that two mammalian CLICs, CLIC1 and CLIC4, are expressed in vascular endothelial cells and are required for angiogenesis.

EXC-4 is constitutively localized to the apical plasma membrane in the *CeEC* and this localization is critical for function. Human CLIC1 can rescue *exc-4* null (*0*), but only when targeted to the apical membrane, demonstrating conservation of function and the importance of membrane targeting. In contrast to EXC-4, human CLICs accumulate in the cytoplasm, but are transiently recruited to the plasma membrane upon G-protein-coupled receptor (GPCR) activation. This transient localization appears to depend on the GPCR effector RhoA. To further understand the conserved role and regulation of CLICs in tubulogenesis we want to know whether EXC-4 activity and localization is regulated by GPCR and Rho-family signaling in *C. elegans*.

Previously-described *exc-4* alleles are nulls, exhibiting strong and fully-penetrant cystic phenotypes. As such, we cannot use them to analyze genetic interactions with mutants in GPCR and Rho-family signaling, because enhancement is not possible and suppression may not occur if *exc-4* is completely absent. By scanning the Million Mutation Project we found mutations that 1) affect conserved EXC-4 residues, 2) exhibit *CeEC* phenotypes, 3) are recessive, and 4) fail to complement *exc-4(0)*. We are currently analyzing genetic interactions between these new hypomorphic *exc-4* alleles and mutations that affect GPCR and Rho-family signaling. Finally, to further understand the regulation of EXC-4 localization we are examining the effect of mutants in GPCR-signaling, and in Rho-family members, as well as the effect of mutations in conserved residues, on EXC-4 accumulation.

## **IL2 neuronal arborization in different alleles of *daf-9* and *daf-12* mutants**

Nadeem Asad, Ali Hofer and Nathan Schroeder

Dept. of Crop Sciences. University of Illinois - Urbana-Champaign

Dauer formation is a survival strategy used by *Caenorhabditis elegans* under harsh environmental conditions. The choice between the two pathways is based on the environmental conditions at late L1 stage. These environmental cues include high levels of dauer pheromone and lack of food. Wild type dauer related phenotypes include development of a buccal plug, radial constriction of the body and terminal bulb, alae formation, and the arborization of the IL2 neurons. Invariably all of these phenotypes are sequentially orchestrated in wild type dauers. Several genes influence IL2 neuronal arborization. Depending on the genes and the environment, these changes can vary from fewer arbors to complete loss of arborization in the IL2 neurons and as well, as cell body displacement. The genes *daf-9* and *daf-12* that encode Cytochrome P450 and Human Vitamin D receptor homolog respectively are the final players in the dauer decision pathway. We hypothesized that alleles of *daf-9* and *daf-12* impact arborization of IL2 neurons. We found that alleles of *daf-9* and *daf-12* exhibit a diverse range of aberrant dauer phenotypes under varied environmental conditions. This study further brought to light roles of *ncr-1* and *ncr-2* in IL2 neuronal arborization. Consequently, *daf-9* and *daf-12* impact many dauer phenotypes including IL2 neuronal arborization.

### ***C. elegans* adult males lacking *hlh-3* function show altered *daf-7* expression in the ASJ neurons**

Kimberly Atteberry, Maryam Sabir and Aixa Alfonso

Dept. of Biological Sciences. University of Illinois - Chicago

The Alfonso laboratory has evidence that the function of *hlh-3* in *C. elegans* is necessary for the terminal differentiation of sex-specific neurons with sex-specific functions in hermaphrodites and males (Doonan et. al, 2008; Marquez, Perez, Raut, Alfonso, unpublished). This affects egg-laying behavior in hermaphrodites and mating behavior in males. The laboratory also knows that amphid wing “A” (AWA) neurons do not appropriately differentiate in *hlh-3* mutants (Marquez, Atteberry, Alfonso, unpublished). Though the AWA cells are not sex-specific, their function via the G-protein-coupled receptor ODR-10 is regulated differently in males. Thus, it appears that shared neurons with sex specific roles are functionally defective in *hlh-3* mutants. This project studies the shared amphid sensory “J” (ASJ) neurons to determine whether they are also affected by *hlh-3* deletion. Hermaphrodites and males express *daf-7* in the shared amphid sensory “I” (ASI) neurons, however there is dimorphic expression of *daf-7* in the ASJs. Only males express *daf-7* in the ASJs, where it has been shown by others to play a role in mate searching behavior (Hilbert & Kim, 2017). This project assays the differentiation state of the ASJs with the reporter *pdaf-7::gfp* in *hlh-3* mutants and wild type animals. We find that *daf-7* expression is altered in mutant males; most mutant males expressed *daf-7* in two or three neurons, as opposed to four. Because of the stereotypic location of neurons in the wild type, it appears the missing cells in the mutant males are the ASJs. Mutant hermaphrodites did not show altered expression. These results support the conclusion that the deletion of *hlh-3* function affects the differentiation of neurons with sex-specific roles, regardless of whether the neurons are shared between the sexes or unique.

## **Effects of Martian physics on terrestrial organisms**

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Over the next few years, several organizations have expressed their intention to place humans on the surface of Mars. Traveling to new planets will present new environments for organisms that evolved entirely within Earth's biosphere. For example, the importance of the earth's gravitational field for biological systems went unappreciated until the first space travelers returned from their mission displaying signs of muscle and bone loss. It is now apparent that the gravitational field of the earth is important for a number of biological functions. In addition to gravity, the earth generates a magnetic field that expands far into space, well beyond the limits of current populated missions. Life as we know it evolved in the presence of this force field. Because biological systems are comprised of polar molecules (e.g. water, proteins, nucleic acids), on earth, these molecules must interact with the geomagnetic field to some degree. The extent or biological relevance of this interaction is presently unknown. For example, the earth's magnetic field could potentially help coordinate the alignment of distant polar molecules within developing tissues, similar to how gravity coordinates growing direction in developing plants. Travelers to Mars will encounter a gravitational field one third of earth's, and a magnetic field at most one hundredths its intensity. To determine the potential effects of Martian physics on terrestrial life forms we constructed two planetary modules mimicking the gravity and magnetic fields of Mars (test), or the Earth (control). Compared to controls, we find that animals exposed to Martian gravitational and magnetic fields display several metrics pointing to developmental insults. Parameters affected included egg hatch rate, developmental time, survival to adulthood, body size, and locomotion velocity. Locomotion deficits appear to increase over successive generations. Understanding the potential effects of traveling to Mars, will allow us to generate insights into the role of Earth's magnetic field in biological systems.

## **Epistatic loci or small-effect variants might underlie bleomycin response variation in *C. elegans***

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Bleomycin is a potent anti-neoplastic agent isolated from the soil fungus *Streptomyces verticillus*. Cancer patients treated with this cytotoxic compound vary in responses from individual to individual. Although bleomycin sensitivity is heritable, genetic variants that underlie the differences in bleomycin response have not been identified in humans. To elucidate the genetic determinants of bleomycin variation, we study responses in the model nematode *Caenorhabditis elegans*. Natural strains differ in response to this compound, so we used linkage mapping with recombinant inbred advanced intercrossed lines (RIAILs) to identify a quantitative trait locus (QTL) on chromosome V that explains about half of the genetic contribution to bleomycin response variation across the RIAILs. We generated reciprocal near-isogenic lines (NILs) by introgressing a large part of chromosome V into the opposite genetic background. Upon bleomycin exposure, these NILs recapitulate the large effect of this QTL but also suggest that a genetic interaction might cause extreme bleomycin sensitivity.

The 162-kb QTL confidence interval contains five genes with nonsynonymous variants in protein-coding regions or changes in gene expression levels between parental strains. We generated CRISPR/Cas9-mediated deletions and reciprocal allele replacements for each of these five candidate genes. None of the reciprocal allele replacements alter bleomycin responses from that of the background parental strain. However, strains with a deletion in one of the five candidate genes display slight changes in bleomycin response compared to their respective parental strains. Interestingly, similar deletions in this candidate gene cause both increases in sensitivity and increases in resistance, depending on the parental strain into which the deletion was introduced. These results motivated us to consider more complex genetic models involving epistatic loci and/or small-effect variants within one QTL to understand how natural genetic variation might impact responses to bleomycin.

## **Mechanisms involved in repression of germline genes in somatic cells of *C. elegans***

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Organisms need to maintain proper gene expression at all times. In *C. elegans*, the DRM complex helps proper gene expression maintenance in somatic cells by repressing germline gene expression. DRM complex mutants can maintain close to normal gene expression at 20°C. However, at an elevated temperature of 26°C, DRM mutants have increased misexpression of germline genes ectopically in the soma and also show a High Temperature larval Arrest (HTA) phenotype. The mechanism by which germline genes become active at 26°C is still not fully known. Although data from our lab shows changes in histone modifications between mutants and wild type, these marks do not change between 20°C and 26°C. Therefore, I am investigating if changes in germline gene localization within the nucleus correlate to changes in gene expression at 26°C. Repressed genes are often associated with the nuclear periphery whereas active genes generally move away from nuclear periphery. I am testing temperature dependent changes in DRM target promoter localization using arrays containing DRM promoters. Initial results show that DRM promoter arrays localize to the nuclear periphery at 20°C and 26°C in both WT and mutants. Since DRM targets do not move away from the periphery when expressed, I hypothesize that they could be moving near to nuclear pore complexes, which is an expressive environment at the periphery. I have found that knock-down of several nuclear pore genes in a DRM mutant background was able to suppress the HTA phenotype. This suggests that disruption of the nuclear pore complex structure dampens ectopic germline gene expression in DRM mutants. I speculate that DRM targets are expressed by localizing to the nuclear pore region when faced with moderate temperature stress. In order to understand how DRM target loci are activated, I am also working on identifying transcription factors (TFs) involved in misexpression of the DRM target genes at 26°C. I conducted an RNAi screen to determine TFs that when knocked down result in suppression of the HTA phenotype in DRM mutants. I found 9 of 82 TFs tested were able to suppress HTA. Future experiments will confirm if the TF candidates can bind to DRM target promoters. Identifying TFs facilitating germline gene misexpression will elucidate local changes in chromatin structure at DRM target loci. Overall, this study will identify mechanisms involved in proper cell fate maintenance in *C. elegans* under environmental stress.

## **AIR-2/Aurora B kinase activity is essential for key events in *C. elegans* oocyte meiosis**

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Oocyte spindles assemble in the absence of centrosomes through unique, poorly understood mechanisms. In *C. elegans* oocyte meiosis, microtubules form lateral associations with chromosomes and a multi-protein ring complex forms at the center of each bivalent that is essential for both congression and segregation. The ring complex consists of more than fifteen proteins including the highly conserved chromosomal passenger complex (CPC), which contains AIR-2 (Aurora B kinase homolog). Previous studies have shown that the CPC is required for targeting all other known ring components, but how AIR-2 and the CPC function to promote ring assembly is not understood. One idea is that AIR-2 serves as a scaffold upon which other proteins can assemble. This idea stems from a study that proposed that SUMOylation of various ring components enables the recruitment of other components with SUMO-interacting motifs (SIMs), driving ring assembly; this study also demonstrated that AIR-2 can be SUMOylated *in vitro* and proposed that this SUMOylation event was crucial for ring assembly. However, whether AIR-2 simply serves as a scaffold or whether its kinase activity is also important for ring assembly is not known.

To address this question, we generated a *C. elegans* strain where the endogenous copy of AIR-2 was tagged with an auxin-inducible degron (AID) tag, and where a kinase-dead version of AIR-2 was expressed from a transgene; this strain therefore enables us to add auxin to degrade wildtype AIR-2, and then assess the consequences of only having a kinase-dead version of the enzyme. These studies revealed that AIR-2 kinase activity is essential for the overall assembly of the ring complex, demonstrating that it does not just play a scaffolding role. Notably, instead of localizing to the ring complex, we observed SUMO aggregates around the spindle under these conditions, suggesting a role for AIR-2 dependent phosphorylation in the SUMOylation pathway. In addition, we found that kinase activity is required for the accurate patterning of the CPC on chromosomes, acentrosomal spindle bipolarity, and chromosome alignment and segregation. Thus, AIR-2 kinase activity is essential for driving multiple key events in *C. elegans* oocyte meiosis.

## **The effect of *hlh-3(lof)* on body wall muscle in *C. elegans***

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Basic Helix-Loop-Helix (bHLH) proteins have been identified in a variety of multicellular organisms. These proteins are transcription factors that regulate cell fate in many tissues including the nervous system. They usually work as heterodimers comprised of a ubiquitously expressed Class I bHLH protein and a specific Class II bHLH protein. In the nematode *C. elegans* there are 14 Class II bHLH proteins. The Alfonso laboratory has shown that the function of the Class II bHLH protein encoded by the gene *hlh-3*, HLH-3, is important for terminal differentiation of neurons in both *C. elegans* hermaphrodites and males. This gene is expressed in all neural precursors but surprisingly only subsets of neurons, those with roles in sexual behaviors, appear affected in their function when HLH-3 function is compromised. Others have shown that *C. elegans* males are generally faster than hermaphrodites and this behavior requires the coordination of sensory neurons and body wall muscles shared between the two sexes. Given these observations we wondered whether *hlh-3* mutants have defects in muscle cell function too. Locomotion may be affected depending on the sex of the muscle. We hypothesized that if muscles are abnormal in these mutants a locomotion assay will reveal the defects. Levamisole is an acetylcholine receptor agonist. It is known that exposure to levamisole results in quicker paralysis in wild type male worms than hermaphrodite worms (Mowrey et al., 2014). This project addressed whether *hlh-3* mutant males and hermaphrodites responded to levamisole in the same or different way than wild type males and hermaphrodites. Multiple synchronized L4 staged males ( $n > 10$ ) and hermaphrodites were placed within a 30 microliter drop of levamisole; worms were separated by sex and genotype, and the total number of worms paralyzed was recorded every 15 minutes for a period of 90 minutes. We determined that *hlh-3* mutant hermaphrodites indeed responded differently to levamisole than WT hermaphrodites.

## **Genetic variation underlies differential responses to docetaxel and zinc treatments in *Caenorhabditis elegans***

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Zinc is an essential element for growth and development, acting as a co-factor for more than 300 enzymes and transcription factors in the cell. For this reason, altering intracellular zinc levels can produce dramatic phenotypes ranging from cell proliferation to cell death. Furthermore, zinc concentrations create a unique cellular environment that may also have an effect on how an organism responds to other stimuli, including chemical compounds. Docetaxel is a semisynthetic compound derived from the European yew tree, *Taxus baccata*, with effective antitumor activity against many cancers. Variation in response to docetaxel is the major limiting factor of this drug, leading to the continued investigation of its metabolism and drug interactions. Previous studies show that the intracellular zinc concentration might influence, both positively and negatively, the effectiveness of docetaxel. Furthermore, it is suggested that this interaction may be genetically regulated, making it important to study the effect of docetaxel in combination with zinc. Leveraging the power of *Caenorhabditis elegans* as a tractable metazoan model for quantitative genetics, we can identify genetic factors that underlie responses to both agents individually and in combination. Using a panel of recombinant inbred lines constructed from two genetically and phenotypically divergent strains, we mapped several quantitative trait loci (QTL) in response to each agent independently. Efforts to narrow these QTL have begun with the construction of near-isogenic lines (NILs) and the subsequent generation of CRISPR/Cas9-mediated deletions of prioritized candidate genes. Additionally, to assess the effects of both conditions in combination, we generated a dual-dose response curve and analyzed a panel of wild isolates for interactions between zinc and docetaxel. Here, we show that zinc supplementation has a range of effects on docetaxel-induced toxicity in *C. elegans*. Future work will explore this variation in response and will identify QTL for this interaction to elucidate the genetic mechanisms specific to this unique combination of treatments.

### ***C. elegans* as a Neurological Model for Duchenne Muscular Dystrophy**

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Duchenne muscular dystrophy (DMD) is a lethal degenerative disease that affects 1 in 3,500 males. DMD is caused by mutations in the dystrophin gene, which is expressed in muscle and nervous tissue. About one-third of DMD patients show developmental delays, among other neurological and muscular phenotypes. *C. elegans* is unique among animals used in DMD research in its ability to model not only the genetic insult, but also the behavioral, and cellular phenotypes observed in patients. To determine if *Caenorhabditis elegans* can also be used to model the neurological deficits of DMD, we ran dystrophic (*dys-1*) worms through a battery of neurological tests. Wild type animals are attracted to low concentrations of an attractant (1% diacetyl) but are repelled by high concentrations (100% diacetyl). Dystrophic worms detected and oriented normally towards low appetitive concentrations of a chemical cue. However, knockout mutants or worms with dystrophin specifically suppressed in nervous tissue also exhibited positive chemotaxis rather than being repelled by high concentrations. These findings suggest that lack of neuronal *dys-1* is responsible for the ability of the animals to be repelled by noxious concentrations of diacetyl. The current experiments suggest that, in addition to modeling the muscular aspects of this disease, *C. elegans* may be useful to model the neurological impairments associated with DMD as well.

## Oligomerization mediates self-stabilizing cortical asymmetry of the keystone polarity protein PAR-3

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The PAR proteins constitute a highly conserved biochemical module that forms and stabilizes cellular asymmetries in a wide variety of contexts and in response to a wide variety of cues. During polarization of the *C. elegans* zygote, a transient cue triggers the dynamic segregation of PAR proteins into complementary anterior and posterior domains. Mutual antagonism between anterior and posterior PAR proteins plays a central role in stabilizing this pattern. However, the anterior PAR protein PAR-3 is unique in that it is required for all other PAR asymmetries, but remains stably asymmetric when all other PAR proteins are either absent or uniformly distributed. A key property of PAR-3 is its ability to oligomerize, which is required for its cortical localization. Here we show that oligomerization mediates dynamically self-stabilizing PAR-3 asymmetries in the absence of mutual antagonism. Combining single-molecule imaging and particle tracking, we show that PAR-3 monomers bind weakly to the membrane where they assemble rapidly and reversibly into oligomers. We show that oligomer dissociation rates decrease sharply with oligomer size. We further show that recruitment rates of PAR-3 monomers are five-fold higher on the anterior cortex where PAR-3 oligomers are enriched and that this bias depends strongly on the ability of PAR-3 to oligomerize implying positive feedback in which cortical PAR-3 oligomers directly bind/recruit cytoplasmic monomers. Using a quantitative model with parameter values constrained by experimental measurements, we find that weak membrane binding of PAR-3 monomers, reversible assembly into slowly-dissociating cortical oligomers, and positive feedback on monomer recruitment is sufficient to explain the stable persistence of a PAR-3 enriched cortical domain. Our model predicts that the PAR-3 domain will self-focus over time through dynamic competition among PAR-3 oligomers for a finite pool of PAR-3 monomers. However, the domain size is effectively stable over the timescale of polarization given a sharp decrease in oligomer mobility with oligomer size and sufficiently slow exchange of oligomer subunits, which we confirm experimentally. Our results reveal a novel mechanism that underlies the dynamic stabilization of cortical polarity in the *C. elegans* zygote. We hypothesize that variants of this mechanism, involving dynamic exchange and oligomerization of membrane-associated proteins, may operate in other cells to stabilize polarized states.

## The genetic basis of natural variation in chemical communication

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From bacterial quorum sensing to human language, communication is essential for social interactions. Nematodes communicate through pheromones called ascarosides, which consist of a sugar ascarylose linked to diverse fatty acid-like side chains as well as derivatives of amino acids, folate, and other metabolites. These modular structures produce a unique and diverse chemical language. Nematodes release distinct combinations of ascarosides at specific concentrations, which can signal other nematodes to modulate a variety of biological processes, including developmental arrest, social and sexual behavior, olfactory learning, stress response, and longevity.

Intraspecific natural variation of ascaroside production has been reported previously, implying that a diversity in nematode languages exist. However, the genetic basis and molecular mechanisms underlying the observed diversity have been insufficiently explored. To study natural variation in the nematode chemical language, we profiled 16 excreted ascarosides from 110 divergent *Caenorhabditis elegans* wild strains using mass-spectrometric analysis. We discovered significant natural variation in the production of ascarosides and investigated the genetic architectures of these traits through genome-wide association analyses. We identified seven unique quantitative trait loci (QTL) that explain variation in production of ten ascarosides, including three loci underlying multiple ascaroside production traits. Additionally, because the ratios of various ascarosides serve as chemical cues, we used the ratios of pairwise combinations of ascarosides and identified 66 additional QTL. Fine mapping of these QTL suggests genetic variants in lipid and amino acid metabolism, as well as other regulatory pathways, all of which could contribute to the diversity of a nematode language. In summary, our study will facilitate (1) exploring the landscape of pheromone diversity in *C. elegans*, (2) discovering novel regulatory pathways involved in the modular production of ascarosides, and (3) elucidating the genetic underpinnings and molecular mechanisms for how evolution has shaped regulatory pathways to produce diversity in this pheromone language.

## The role of histone modifications in shaping post-mitotic neuronal identity

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Neuronal cell diversity fundamentally relies on precise spatiotemporal regulation of gene expression in the nervous system. Despite in-depth studies focusing on sequence-specific DNA-binding proteins (transcription factors [TFs]), the contribution of specific chromatin modifications in neuronal diversity remains poorly understood. Using the cholinergic motor neuron (MN) subtypes of the *C. elegans* ventral nerve cord (VNC) as a model, we previously showed that the phylogenetically conserved Collier/Olf/Ebf (COE)-type TF, UNC-3, is required for MN diversity by directly activating subtype-specific terminal differentiation genes. However, diversification of MNs into molecularly distinct subtypes requires additional TFs that antagonize UNC-3 by repressing its targets in a subtype-specific fashion. This interplay between a transcriptional activator and MN subtype-specific repressors provides a unique platform to reveal the contribution of chromatin modifiers in post-mitotic neuronal diversity. Here, we show that two histone 3 lysine 9 (H3K9) methyltransferases, *met-2* (SETDB1) and *set-25*, despite their ubiquitous expression, are required cell-autonomously and selectively repress an UNC-3 target (*glr-4*) in specific VNC MN subtypes. Similar cell-autonomous requirements are also observed for three other heterochromatin-related genes, *hpl-2* (HP1), *lin-61*, and *lin-13*, known to have binding affinity for H3K9 methylation marks. We hypothesize that MN subtype-specific TFs recruit H3K9 methyltransferases to UNC-3 target genes. Subsequently, H3K9me is recognized by HPL-2, LIN-61, LIN-13 proteins to facilitate transition of the chromatin structure to a more compact state, thus less accessible to UNC-3. Ongoing experiments test the temporal requirement of these and other (NuRD complex) chromatin factors to gain novel insights into the mechanisms of neuronal diversity.

## **The miR-44 family of microRNAs are necessary for sperm function in *C. elegans***

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microRNAs regulate diverse developmental and physiological processes in *C. elegans* by binding in the 3' UTR of mRNAs to repress translation. However, specific functions for most individual miRNAs have not yet been identified. We have identified that the miR-44 family of microRNAs regulate sperm function. While sperm in *C. elegans* is regulated both transcriptionally and post-transcriptionally, little is known about the post-transcriptional regulation. miR-44 family mutant phenotypes include reduced brood size, high levels of unfertilized oocytes, and fertilization defects, likely due to defects in sperm function. The miR-44 family comprises four miRNAs: *mir-44*, *mir-45*, *mir-61*, and *mir-247* that share a common seed sequence and thus are predicted to regulate shared target mRNAs. Interestingly, miR-44 and miR-45 share an identical mature sequence and are located only ~9kb apart on chromosome II previously precluding the generation of double mutants. Additionally, analysis of expression of dissected hermaphrodite and male germlines, using qPCR, showed that normalized expression of *mir-44* and *mir-45* are twenty times higher in males than hermaphrodites. Using CRISPR/Cas9, we generated a *mir-44/45* double mutant that we are currently analyzing. Our data indicate that mutants in members of the miR-44 family, particularly the *mir-44/45* mutants, have significantly increased numbers of unfertilized oocytes due to defects in sperm. Furthermore, mating of *mir-44/45* males to hermaphrodites produce significantly less cross progeny. *mir-44/45* male sperm also frequently fail to activate. Further analysis of *mir-44/45* males has revealed that *mir-44/45* sperm fail to transfer and migrate to the spermatheca after mating, signifying that miR-44 and miR-45 are required for proper sperm localization. miR-44 and miR-45 play essential roles in sperm function in *C. elegans*. Continued analysis will focus on identifying target genes and pathways that are regulated by the miR-44 family in sperm function.

## The PBAF chromatin-remodeling complex is required for cholinergic motor neuron subtype identity

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The various motor neuron (MN) classes of the *Caenorhabditis elegans* ventral nerve cord (VNC) provide an ideal platform to probe the gene regulatory mechanisms that establish distinct neuronal cell identities. We have previously shown that the evolutionarily conserved COE (Collier, Olf, Ebf)-type transcription factor UNC-3 acts as a terminal selector and determines cholinergic MN identity in the majority of VNC cholinergic MN classes (SAB, DA, DB, VA, VB, AS). UNC-3 directly controls the expression of both shared (e.g., enzymes and transporters in the acetylcholine biosynthetic pathway) and class-specific terminal identity genes (e.g., ion channels, neurotransmitter receptors, neuropeptides). However, *unc-3* is expressed in all these MN classes, leading us to hypothesize the existence of repressor proteins that restrict the ability of UNC-3 to activate these class-specific genes more broadly, i.e., in all MN classes. To test this hypothesis, we performed a forward genetic screen using the UNC-3 target gene *glr-4*, which encodes a glutamate receptor subunit selectively expressed in the SAB class. We found that *pbrm-1*, the sole *C. elegans* ortholog of the evolutionarily conserved chromatin regulator BAF180, selectively prevents *glr-4* expression in DA, VA, and AS classes of MNs. However, the expression of multiple DA, VA, and AS class-specific features is unaffected, indicating that loss of *pbrm-1* leads to mixed MN identity. Since PBRM-1/BAF180 is a subunit of the PBAF, a chromatin remodeling complex of the SWI/SNF family, we reasoned that animals lacking gene activity for other PBAF subunits might display similar MN phenotypes. We indeed found that loss of *swn-9* (*C. elegans* ortholog of human BRD7 and BRD9), *swn-7* (*C. elegans* ortholog of human ARID2), and *phf-10* (ortholog of human PHF10) results in gain of *glr-4* expression in the DA, VA, and AS classes of MNs. Motor neuron-specific rescue and RNAi experiments further demonstrated that all four of these PBAF components, despite their ubiquitous expression, act cell-autonomously. Altogether, we provide novel insights on the epigenetic mechanisms that generate neuronal diversity by uncovering a previously unrecognized, neuron-specific role for the PBAF chromatin-remodeling complex in selective repression of terminal selector target genes.

**The search for magnetic particles in *C. elegans***

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Many organisms, from bacteria to whales can detect and orient to the magnetic field of the earth. While the mechanism for magnetic field detection in animals remains a mystery, it is known that many magnetosensitive organisms harbor magnetic particles in their tissues. Among these, magnetotactic bacteria are the most understood. The nematode *C. elegans* detects magnetic fields and has magnetic particles in its tissues. We are investigating the location, identity, and role in orientation of these magnetic particles.

**Probing the molecular mechanism of receptor tyrosine kinase enzymatic activation through the analysis of heterodimers of the *C. elegans* fibroblast growth factor receptor (FGFR), EGL-15**

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Receptor tyrosine kinases (RTKs) are a class of cell-surface receptors that play critical roles in cell-cell communication processes. Multicellular organisms rely on RTKs for the proper organization of cells into functional units during development and homeostasis in adult tissues. One sub-class of RTKs is the family of heparin-binding fibroblast growth factor receptors (FGFRs). Upon ligand binding, FGFRs dimerize, activating their intrinsic tyrosine kinase activity, which causes both autophosphorylation and phosphorylation of additional substrates. Misregulation of FGFR signaling in humans has been linked to developmental abnormalities and cancer. This study attempts to probe the molecular mechanism of kinase domain activation in RTKs. Genetic analysis of EGL-15, the *C. elegans* FGFR, has provided many insights into the molecular mechanisms of RTK signaling. The cumulative EGL-15 activity of an *egl-15* heterozygote can be used to understand how the structures of various EGL-15 heterodimers affect the mechanism of their activation. *egl-15(n1457)* is a nonsense mutation that truncates the carboxy-terminal domain ( $\Delta$ CTD) of EGL-15; the CTD contains several tyrosine phosphorylation sites important for EGL-15 signaling events. Based on the phenotype of the *egl-15(n1457  $\Delta$ CTD)* homozygote, the truncated EGL-15 $\Delta$ CTD forms active homodimers. Interestingly, heterozygotes between *egl-15(n1457 $\Delta$ CTD)* and various *egl-15* substitution mutants have significantly less EGL-15 activity. Our results suggest that certain EGL-15 mutant monomers can inhibit the activity of EGL-15( $\Delta$ CTD) monomers when the two dimerize. By studying the degree to which the specific structural changes inhibit EGL-15( $\Delta$ CTD), we hope to gain insight into the molecular mechanism by which kinase dimerization leads to kinase enzymatic activity.

## VCs require HLH-3 function to assume their terminal differentiation state

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Of interest to the field of neurodevelopment is to elucidate the mechanisms underlying acquisition of cellular identity and sub-type specific identity within a class of neurons. Our laboratory has found that the *C. elegans* bHLH, proneural-like protein HLH-3, plays a role in the terminal differentiation of neurons that are either sex-specific or have a sexually dimorphic function. We have evidence that *hlh-3* has a role in the differentiation of the sex-shared AWA neurons and sex-specific neurons including the CEMs (male specific chemosensory neurons) (Marquez, Atteberry and Alfonso, unpublished), and HSNs (hermaphrodite specific neurons) (Raut, Atteberry and Alfonso, unpublished, Doonan et al, 2008). We also have evidence that *hlh-3* has a role in the differentiation of the six hermaphrodite-specific ventral cord type C (VC) neurons (Perez, Vazquez and Alfonso, unpublished). The VCs are implicated in egg-laying circuitry and locomotion (Shafer, 2006; Yan et al., 2017). Additionally, these neurons have a subtype-specific gene expression pattern making VCs distinct dependent on their proximity to the vulva (Zheng et al., 2013). Because we aim to elucidate the mechanism underlying VC neuronal identity we have characterized the expression of several VC differentiation markers in the absence of *hlh-3* function. We will report on our findings that distal VCs are most affected in *hlh-3(lof)* hermaphrodites. While reporter expression is detected in all six VC neurons in WT animals, the distal VCs (1, 2, 3, and 6) fail to report expression of these markers. We set out to address whether the abnormal expression pattern was a result of inappropriate cell death in these lineages, however, our data suggests that this is not the case; *hlh-3 (lof); ced-3(lof)* animals do not express VC reporters in the distal VCs. These results suggest that HLH-3 function is required to induce terminal differentiation of these sex-specific neurons.

## Identifying new components that mediate fibroblast growth factor receptor signaling in *C. elegans*

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Fibroblast growth factor receptors (FGFRs) are a class of cell-surface receptors that play key roles in cellular functions such as cell proliferation, migration, and differentiation. FGFRs belong to a larger family of receptor tyrosine-kinase (RTK) cell-surface receptors that act by phosphorylating specific tyrosine residues to trigger downstream responses. The FGFR EGL-15 in the nematode *Caenorhabditis elegans* has long been used as a paradigm to understand principles of RTK signaling. Defects in the processes mediated by EGL-15 result in striking phenotypes that provide powerful genetic tools that have been used to discover many components that mediate RTK signaling. One such process is fluid homeostasis regulation. EGL-15 hyperactivation causes excessive accumulation of clear fluid inside the worm's body, known as the Clear (*Clr*) phenotype. Hyperactivation of EGL-15 signaling is typically accomplished by a mutation in the gene *clr-1*, which encodes a receptor tyrosine phosphatase that negatively regulates the EGL-15 signaling pathway. The isolation of suppressors of the *Clr* phenotype, termed Suppressor of *Clr* (*soc*) mutants, has led to the identification of many of the core components of EGL-15 signaling. For example, the original set of *soc* mutations identified the Grb2/SEM-5 adaptor protein that links RTK activation to the activation of the RAS/MAPK pathway. Although SEM-5 is required for the regulation of fluid homeostasis via EGL-15, a key signaling component that links activated EGL-15 to SEM-5 has yet to be identified. While activated EGL-15 can recruit SEM-5 via phosphorylated tyrosines in its carboxy-terminal domain (CTD), this mechanism cannot explain the SEM-5 requirement for fluid homeostasis, since an *egl-15* mutation (*n1457*) that truncates the CTD and eliminates these binding sites does not confer a *Soc* phenotype. To identify these missing components, a modified, "enhancer" *soc* screen in an *egl-15(n1457)* background was conducted, and 34 enhancer mutations were isolated. The characterization of an initial subset of these *soc* enhancer mutations suggests that they define at least two new *soc* genes. Genetic analysis and whole-genome sequencing will be used to identify the molecular identities of these new FGFR signaling genes.

## Development of a novel assay to identify genes involved in host infection by parasitic nematodes

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About one-quarter of the world population is estimated to be infected with parasitic nematodes. There is widespread resistance to currently used drugs in the veterinary field, and concern is growing that resistance may arise in humans parasitic nematodes. *C. elegans* is a free-living nematode that has been especially suggested as a good model for anthelmintic drug, and target discovery. This project takes advantage of *C. elegans*' burrowing behavior to model the need of parasitic worms to cross biological barriers during infections. We are searching for new molecular targets to prevent nematodes from infecting livestock and other hosts. Worms will be placed in multi-well plates and fed bacteria using RNA interference to target a specific gene in each well. We are targeting genes with no homology in humans, but with homology in the parasitic nematode *Haemonchus contortus*. This will allow us to identify genes likely to be involved in parasitic nematode biology, but with no similar targets in humans (or livestock). Burrowing will be assessed in RNAi silenced animals. This assay will allow us to identify potential drug targets that can be used to impede nematocidal infections.

## Decreased mating ability may play a role in *C. elegans* male low fertility at high temperature

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The germline, especially the spermatogenic germline, is temperature-sensitive, such that as the temperature increases, germline function and fertility drop rapidly. Our previous work has shown wild type strains of *C. elegans* males raised at 27°C are almost all sterile, while males raised at 20°C but upshifted to 27°C for mating are almost all fertile (Petrella, 2014). High temperature loss of male fertility could be due to loss of sperm function, reduced sperm number, changes in mating ability, or all three. To test sperm function, we have analyzed the rates of in vitro sperm activation in males that were raised at 27°C. We found that activation rates are reduced by approximately 10-15%. This level of decreased activation is not sufficient to explain the almost complete loss of fertility seen at this temperature. We also performed experiments to quantify the number of sperm produced by males at 27°C. We found that sperm count is reduced in some strains at this temperature. Male mating interest and execution were tested with males raised at 27°C. All wild type strains show declines in multiple steps of male interest and mating execution during timed assays. Although there were differences between strains, all strains had a high percentage of mating failure when raised at 27°C. Finally, to examine mating ability further and distinguish between successful insemination and migration of male sperm to hermaphrodite spermatheca, we performed additional mating experiments over a longer timeframe (1.5 hours). We found differences in wild isolate male mating ability and sperm migration at 20°C; but in all strains sperm migrated over 60% of the time. These experiments also confirmed that males raised at 20°C but upshifted to 27°C for mating could mate as well as worms raised at 20°C. However, we found that sperm from these males did not migrate to the spermatheca as well, with successful migration occurring less than 50% of time in all strains. Our data indicate that reduced sperm activation, and sperm count, are not the main cause of low fertility at 27°C but that decreased mating and sperm migratory ability may play an important role. As loss of fertility at high temperature is conserved across phyla, our research may uncover conserved temperature-sensitive germline pathways that account for the germline temperature sensitivity.

## Cell to cell spreading of TDP-43 C-terminal fragments in *Caenorhabditis elegans*

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Many neurodegenerative disorders such as Alzheimer's disease (AD), Parkinson's disease (PD), Amyotrophic Lateral Sclerosis (ALS) and prion diseases are characterized by abnormal accumulation of disease proteins in nerve cells leading to selective neurotoxicity. Moreover, a prion-like spreading mechanism might play a role in disease progression, where misfolded disease proteins spread from affected to unaffected neurons. Interestingly, ALS exhibits a focal clinical onset followed by a regional spreading of protein misfolding and cell death. Evidence points towards TAR DNA-binding protein 43 (TDP-43) as the major pathological protein in sporadic and certain familial forms of ALS where aggregates in affected neurons contain full length and fragmented forms of phosphorylated and ubiquitinated TDP-43. Despite recent advances in biomedical research on ALS disease associated proteins like TDP-43, a mechanistic explanation of cell to cell transmission remains unclear.

To explore whether TDP-43 spreads from cell to cell, we established a *C. elegans* model that expresses a human TDP-43 C-terminal fragment (TDP-25) fused to red fluorescent protein in the body wall muscle cells. We employed high-resolution time-lapse imaging and observed the intercellular movement of TDP-25::RFP from body wall muscle cells to the hypodermis, intestinal cells and gonad in living animals. These results confirm that at least certain fragments of TDP-43 are released from donor cells into neighboring receiving cells. To determine the effect of expression of TDP-43 C-terminal fragments in body wall muscles, we measured the thrashing activity of the animal and observed no difference compared to wild type animals. To assess the effect of accumulation of TDP-43 C-terminal fragments in the receiving tissue, we monitored the function of the gonad. Presence of TDP-25::RFP had no significant effect on fecundity or embryogenesis compared to wild type animals. Currently, we are testing whether TDP-43 C-terminal fragments are phosphorylated, a post-translational modification associated with toxicity. Furthermore, we are mapping the movement of TDP-43 C-terminal fragments from donor cells to receiving cells using strains expressing tagged lysosomal and endosomal components. Evidence of phosphorylation and co-localization would increase our understanding of the pathway and mechanism of cell to cell transmission of TDP-43 fragments.

## Dynamics and recruitment of the polarity protein PAR-3 in the P1 cell

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The ability to polarize and divide asymmetrically is fundamental to embryonic development. A set of highly conserved proteins, known as the PAR proteins, governs polarity in a wide range of organisms and cellular contexts. Two sets of PAR proteins (anterior aPARs and posterior pPARs) localize to complementary domains in polarized cells. These asymmetries are induced by polarizing cues, and maintained by mutual inhibition between aPARs and pPARs. In embryos of the nematode *Caenorhabditis elegans*, PAR polarities control a sequence of asymmetric divisions of the posterior blastomeres, P1, P2, and P3. In the well-studied case of the zygote P0, aPARs initially are universally enriched on the membrane, while pPARs are cytoplasmic. A transient sperm-derived cue induces polarity by inducing actomyosin-based cortical flows, that segregate the oligomeric scaffold PAR-3 and other aPARs to the anterior pole, and by promoting local accumulation of the pPAR PAR-1, which phosphorylates and displaces PAR-3 from the posterior membrane. The posterior daughter of P0, called P1, also polarizes due to the action of PARs, but from opposite starting conditions, in which pPARs rather than aPARs are enriched on the cortex. How aPARs can bind the cortex and polarize to an anterior domain in the presence of inhibitory pPARs remains unclear.

To address this question, we performed live near-TIRF imaging on 2-cell embryos containing GFP-tagged forms of PAR-3 and PAR-1. We find that soon after the first cell division, PAR-3 oligomers begin to appear everywhere on the P1 membrane; they increase steadily in both number and size, and then become enriched at the anterior pole. These events occur in the continuous and universal presence of PAR-1, indicating a previously unknown mechanism that allows PAR-3 to overcome inhibition by PAR-1. Removing PAR-1, or blocking cortical flows by removing the RhoA activator NOP-1, fail to prevent polarization of PAR-3, suggesting that additional mechanisms beyond cortical transport and PAR-1 dependent exclusion govern the formation of PAR-3 asymmetries in P1. Based on our unpublished studies of PAR-3 oligomerization dynamics, we propose that enhanced growth of PAR-3 oligomers and spatial competition among PAR-3 oligomers controls the emergence of PAR-3 asymmetries. The dynamics of PAR-3 recruitment may thus be sufficient to allow P1 polarization even under conditions significantly different from the established mechanisms of zygotic polarity regulation.

## **Natural variation in *C. elegans* arsenic-induced toxicity is explained by differences in branched chain amino acid catabolism**

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Organisms have long been forced to adapt to the environmentally ubiquitous toxic metalloid arsenic. Currently, an estimated 100 million people are at risk of chronic exposure to arsenic. Recent evidence from genome-wide association studies suggest that polymorphisms in the AS3MT gene, which are present in human subpopulations exposed to elevated arsenic, result in an increased ability to metabolize arsenic. Given the difficulty in functional characterization of human variation, we have no experimental evidence that links polymorphisms in the AS3MT gene to arsenic metabolism or toxicity. However, these results do suggest that standing genetic variation can be used to identify mechanisms by which organisms tolerate arsenic exposure. In the present study, we take advantage of the genetic diversity present in the tractable model organism *Caenorhabditis elegans* to identify a novel mechanism of arsenic toxicity that might have arisen as a result of differential exposure to arsenic among subpopulations of this free-living nematode species.

Using two genetic mapping approaches, we show that a major source of variation in *C. elegans* responses to arsenic trioxide is caused by natural variation in the *dbt-1* gene. This gene encodes for the E2 subunit of the branched-chain  $\alpha$ -keto acid dehydrogenase (BCKDH) complex, which is a core component of branched-chain amino acid (BCAA) catabolism. We used CRISPR/Cas9-mediated allele editing to show that a single non-synonymous variant (C78S) in the highly conserved lipoyl domain of DBT-1 is the causal polymorphism underlying variation in response to arsenic trioxide. We used unbiased metabolomics and chemical supplementation experiments to show that differences in *C. elegans* responses to arsenic trioxide result from differential depletion of mono-methyl branched chain fatty acids, metabolites with a central role in developmental progression. We hypothesize that the presence of the thiol group in the sensitive DBT-1(C78S) allele mediates the coordination of arsenic binding to the reduced lipoic acid cofactor, thereby inhibiting the catalytic cycle of the BCKDH. Our study marks the first time that the BCKDH complex and BCAA metabolism have been implicated in the response to arsenic. These results demonstrate the power of using natural genetic diversity of *C. elegans* in combination with comparative metabolomics to identify mechanisms by which environmental toxins affect organismal physiology.

### Using *C. elegans* to teach the genetic basis of disease and behavior

Hughes K, Ahlers S, Arteman C, Burdette H, Hanrahan D, Kurczewski J, Malinski J, Melvin R, Mota J, Niswonger D, Olszowy P, Omotunwashe S, Packard C, Penza M, Perry R, Robledo S, Shawd K, Smith T, Webb T, Westbrook M, Winter H and Vidal-Gadea, Andrés

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The experimental amenability of *C. elegans* makes this organism an excellent tool for teaching basic biological principles and techniques. We designed a course capitalizing on the advantages offered by *C. elegans* to teach junior level students molecular techniques, as well as basic genetic and cellular principles. We assume only high school level biological knowledge, and no previous experimental experience. In the first part of the course students learn to conduct different behavioral assays covering basic sensory biology (e.g. thermotaxis; chemotaxis; magnetotaxis; phototaxis) or disease processes (e.g. muscular dystrophy;, Parkinson's and Alzheimer's; alcoholism; cancer). In the second part of the course, students learn to perform forward and reverse genetic screens, backcrosses, complementation, and mapping. During the third part of the course they are introduced to molecular techniques (e.g. DNA purification; PCR; electrophoresis; Topo, BluntEnd, and restriction cloning; PCR fusion; RNAi; transformations, minipreps, colony PCR, etc.). For the final portion of the course, students learn optogenetics, and other molecular-based cellular manipulations before designing a project to answer a meaningful biological question using the knowledge acquired. They conduct experiments, analyze data, and present their findings in class presentations. Here we present the outcome of student generated projects. The advantages of *C. elegans* as a research and teaching tool have consistently continued to surprise the most seasoned researchers in the field. We strongly believe and advocate for its use in the training of students at every educational level

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