Yun Zhang

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Dear Faculty Search Committee,

I am a postdoctoral fellow in Dr. Cori Bargmann's lab at the Rockefeller University. I am applying for the Assistant Professor position in the Department of Biology at Indiana University at Bloomington. My research interest focuses on the function of neural circuits that regulate behaviors.

As a graduate student in Dr. Martin Chaflie's lab at Columbia University, I studied the differentiation of the *C. elegans* mechanosensory neurons. To reach a complete understanding of cell fate regulation in these neurons, I developed and optimized molecular and cellular techniques to solve the challenging problem that only 0.5% of all cells in an animal are touch neurons. This work generated the first expression profile for a single type of neuron and was published in *Nature* with me as the first author.

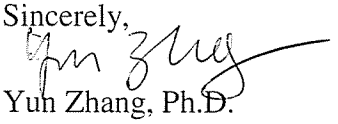
As a postdoc in Dr. Cori Bargmann's lab, I studied molecular and cellular mechanisms by which neural circuits regulate learning. Using the *C. elegans* olfactory system as a model, I established an associative learning paradigm and demonstrated that *C. elegans* is able to learn to avoid pathogenic bacteria, a physiologically and ecologically relevant natural stimulus. This work opened an independent research area. Using this model, I identified a serotonergic circuit that regulates the learning and showed that enhanced serotonin signaling in a single pair of neurons promotes learning. These results provide a direct link between a molecular change of a specific neuron and an associative learning behavior. This work was accepted by *Nature* as an Article with me as the first author.

One of the fundamental questions in neurobiology is to understand how experiences modify behavior. Using *C. elegans* as a genetic model with a well-defined neuronal network, I intend to lead a lab that combines molecular genetics with system neuroscience to address this fundamental question at the level of individual neurons in three directions:

1. Assemble the complete olfactory learning circuits and identify the cellular sites where serotonergic modulatory inputs converge with and modify olfaction.
2. Characterize the mechanisms by which pathogen infections upregulate serotonin signaling. Serotonin probably functions as a negative reinforcing signal in this associative learning paradigm.
3. Characterize the mechanisms by which increased serotonin signaling modifies olfaction.

I also look forward to teaching both graduate and undergraduate students. I am well prepared to teach neurobiology, molecular biology, cell biology and genetics, and I am also ready to face the challenge of teaching outside of my major training. Thank you for considering my application.

Sincerely,



Yun Zhang, Ph.D.

I have enclosed my *curriculum vitae*, research proposal and four reprints and manuscripts of publications from my postdoc work in Dr. Cori Bargmann's lab and my graduate work in Dr. Martin Chalfie's lab. You should also receive letters of recommendation from the following:

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Molecular and Cellular Understanding of Neural Circuits Regulating Olfactory Learning

Learning is a conserved function of the nervous system that enables animals to modify behaviors based on prior experiences. A central goal in neuroscience is to understand the mechanism of learning processes. One of the most common learning behaviors in all animals is olfactory learning, which plays an important role in social recognition, maternal care, foraging and predation. Research on olfactory learning using mammals and flies as model systems suggests that neuromodulatory inputs modify olfaction. Several questions are fundamental to a full understanding of the molecular and cellular mechanisms of olfactory learning:

1. What are the neural circuits that underline olfactory learning behaviors?
2. How does experience regulate neuromodulatory pathways?
3. How do neuromodulatory inputs modify olfaction?

During my postdoctoral research in Dr. Cori Bargmann's lab, I have used the *C. elegans* olfactory system as a model to study these fundamental questions. I have established an associative olfactory learning paradigm and demonstrated that *C. elegans* is able to learn to avoid pathogenic bacteria, a physiologically and ecologically relevant natural stimulus. I have also shown that animals that have experienced both pathogenic bacteria and innocuous food bacteria during growth not only learn to decrease their attraction to the pathogen but also learn to increase their attraction to the innocuous bacteria. To understand this learning process, I partially mapped a serotonergic circuit that regulates aversive pathogen learning (Figure 1). I demonstrated that experience with pathogens activates the key enzyme for serotonin synthesis, TPH-1, in a single pair of serotonergic sensory neurons ADF and that the enhanced serotonin signaling promotes learning. I found that a serotonin-gated chloride channel, MOD-1, functions in several interneurons to regulate learning. I hypothesize that enhanced serotonin signaling following pathogen exposure modulates the function of olfactory neurons, resulting in an acquired olfactory aversion from the infecting pathogen.

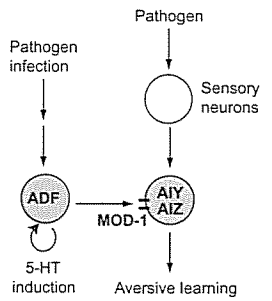

Figure 1. Aversive olfactory learning circuit.

This work will lead to research in three directions to understand the mechanisms that regulate both neuromodulatory signaling and olfactory pathways during olfactory learning.

First, I will map the olfactory circuit regulating learning. I will use cell-specific rescue of learning mutants and genetic ablations to identify chemosensory neurons required for learning. I will use cell ablation in combination with candidate gene approach to identify relevant olfactory interneurons.

Second, I will characterize the mechanisms by which pathogen exposure upregulates serotonin signaling. I will identify conserved TPH-1 regulatory domains that activate TPH-1 activity in response to pathogen infections. Then I will use genetic screens in combination with genomic approaches to identify molecules that upregulate ADF serotonin signaling in response to pathogen exposure. These studies directly address the question of how experience regulates neuromodulatory pathways.

Third, I would like to know how enhanced serotonin signaling in ADF modifies olfactory responses to pathogenic bacteria. I will use chemotaxis and navigation assays to characterize the functions of olfactory sensory and interneurons in the learning circuit during learning. I will also use imaging approaches to understand the changes in neural activity in these cells after pathogen exposure. Led by these studies, I will design genetic screens using chemotaxis and navigation behaviors as read-outs to identify molecules that regulate neural plasticity of the olfactory circuits. These studies should provide insights on how neuromodulatory inputs modify olfaction.

Molecular and Cellular Understanding of Neural Circuits Regulating Olfactory Learning

Background

Animals in a natural environment interact with different ecological cues and modify their behaviors based on their experiences. A central goal of neurobiology is to understand the regulation of dynamic learning processes.

Olfactory learning plays important roles in many behaviors. In *Drosophila*, coupling of olfactory cues with electrical shocks induces release of a neuropeptide neurotransmitter that activates a cAMP pathway to regulate learning in mushroom body neurons[1]. Studies of taste aversion in rats suggested that glutamate release triggered by visceral malaise converges and interacts with acetylcholine release triggered by novel tastes at insular cortex[2,3]. In female mice, increased release of noradrenaline in response to mating is associated with the exposure to the pheromone of a mating male. This association regulates pregnancy block[4].

During my postdoctoral research, I used *C. elegans* as a model system to develop a model for olfactory learning. The development and function of the *C. elegans* nervous system share basic features with those of other organisms. *C. elegans* senses a variety of odorants and water-soluble compounds through a well characterized olfactory system[5,6]. The simple nervous system contains 302 neurons and generates reliable and quantifiable behaviors[7]. The well-defined morphology and connections of the neuronal network allows behaviors to be dissected at the level of individual neurons and circuits. Therefore *C. elegans*, as a genetic model, offers unique strengths to integrate mutant analyses and molecular genetics with system neuroscience to understand information flow and processing in neuronal networks.

Significance

Many experiments suggest that neuromodulatory inputs modify olfaction during olfactory learning. However, the complexity of the mammalian and fly nervous systems limits a detailed understanding of olfactory learning at the level of individual neurons. As a free-living nematode that feeds on bacteria in soil, *C. elegans* can distinguish different bacteria[8]. Pathogenic bacteria are prominent environmental factors affecting *C. elegans'* survival[9]. As a postdoc working with Dr. Cori Bargmann at the Rockefeller University, I showed that *C. elegans* is capable of associative olfactory learning to avoid pathogenic bacteria. I identified a serotonergic circuit that converges with olfactory pathways and regulates this learning process. I also demonstrated that pathogen exposure enhances serotonergic signaling in one pair of serotonergic neurons and that increased serotonin signaling accelerates learning, probably functioning as a negative-reinforcing cue. I hypothesize that serotonergic modulatory inputs modify the functions of olfactory neurons to change an animal's olfactory behaviors based on its experience with the training pathogen. This work identified a direct link between the learning behavior and a molecular change in a single pair of neurons.

I propose to study a fundamental question in the learning field: how neuromodulatory inputs shape the activity of neural circuits to regulate an animal's responses to conditioned stimuli. I will pursue research in three directions to achieve a molecular understanding of this question at the level of individual neurons:

1. Identification of the olfactory learning circuit and the cellular sites at which olfactory pathways converge with serotonergic neuromodulatory inputs.
2. Characterization of mechanisms by which pathogen exposure enhances serotonin signaling in the negative-reinforcing pathway.
3. Characterization of mechanisms by which enhanced serotonin signaling modifies the function of the olfactory pathways.

Postdoctoral Research to Date

1. *C. elegans* learns to change olfactory preferences to avoid pathogenic bacteria. On low-nutrient media, the opportunistic pathogens *Pseudomonas aeruginosa* strain PA14 and *Serratia marcescens* kill *C. elegans* slowly over several days by intestinal infections[10,11]. Using a binary choice assay between a bacterial pathogen lawn and an *E. coli* (OP50) lawn (Fig. 1A), I showed that wild-type animals grown on OP50 alone do not have a preference between PA14 and OP50; but animals exposed to PA14 strongly prefer OP50 to PA14 (Fig. 1B, C). Similarly, animals grown on OP50 prefer *S. marcescens* to OP50, but animals trained on *S. marcescens* are significantly repelled by the pathogen (Fig. 1B, C). Thus *C. elegans* modifies its olfactory preferences to avoid pathogens based on experiences. Non-virulent derivative strains of PA14 do not induce learning, suggesting that pathogenesis is essential for olfactory learning.

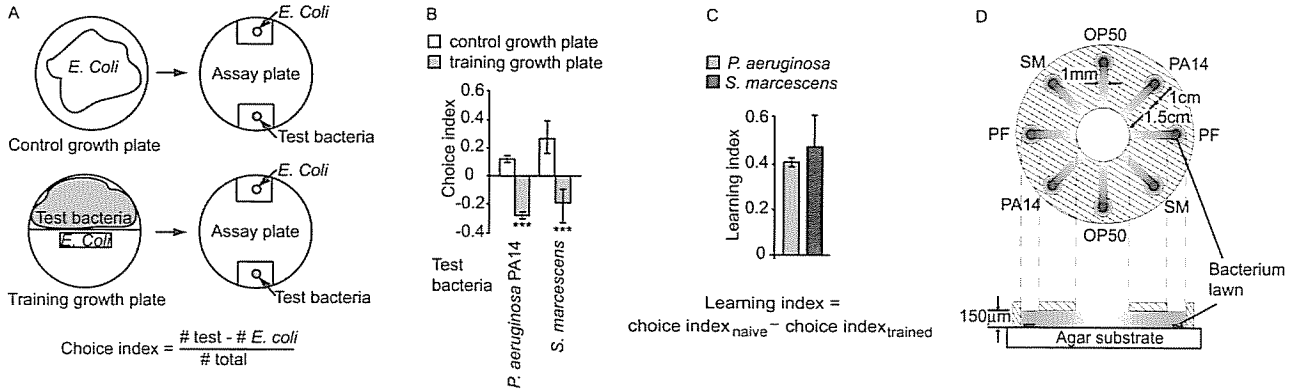

Figure 1. A, Animals are grown on *E. coli* alone or grown on test bacteria with *E.coli*, then tested in population assays for olfactory preferences. B, *C. elegans* changes olfactory preferences after exposure to pathogen *P. aeruginosa* PA14 or *S. marcescens*. C, Learning indices for avoidance of the pathogens. D, Diagrams of the maze assays for olfactory learning.

2. Olfactory learning on pathogen combines attractive and aversive learning processes. I next asked if the olfactory learning induced by pathogens is an induced aversion from pathogens or an increased attraction to *E.coli*. Together with another postdoc, Hang Lu, I developed a multiple-choice assay using an eight-arm worm maze made from polydimethyl siloxane elastomer (Fig. 1D). The distinctive smells of the bacteria in the eight chambers converge at the decision area at the center, where animals experience different smells of the bacteria and enter the chamber containing bacterium with relatively more attractive smell. I used four bacteria strains in the maze, two non-pathogenic ones, *Pseudomonas fluorescens* (PF) and OP50, and two pathogenic ones, *Pseudomonas* PA14 and *S. marcescens* (SM).

In the maze, animal populations trained with PA14 and OP50 (Fig. 1A) have an increased fraction of animals that choose OP50 and a decreased fraction of animals that choose PA14 compared to animals raised on OP50. Therefore, the olfactory learning induced by the pathogens is a combination of both attractive learning towards *E. coli* and an induced aversive learning away from the training pathogen.

3. Serotonergic neurons are required for olfactory learning on pathogenic bacteria and exposure to pathogens enhances serotonergic signaling in ADF neurons. In *C. elegans*, *tph-1* encodes the only tryptophan hydroxylase required for serotonin biosynthesis, and *tph-1* mutants are deficient in serotonin, but viable[12]. When trained with OP50 and PA14, *tph-1* mutants are completely defective both in attractive and aversive olfactory learning (Fig. 2A). In *C. elegans*, serotonin is mainly produced in chemosensory neurons ADF, pharynx neurons NSM, and egg-laying motor neurons HSN[13]. Expression of *tph-1* in ADF neurons alone using *srh-142* promoter is sufficient to fully restore aversive learning, but has no effect on attractive learning. Expression of *tph-1* in NSM neurons cannot rescue. Exposure to PA14 results in increased serotonin immunostaining in ADF neurons (Fig. 2B, C). I propose that exposure to pathogenic bacteria enhances serotonergic signaling in ADF neurons, which triggers aversive olfactory learning.

I next characterized signaling downstream of ADF neurons. *mod-1* encodes a serotonin-gated chloride channel that regulates the enhanced slowing of starved animals on food[14,15]. When trained with OP50 and PA14, *mod-1* mutants are specifically defective in aversive learning (Fig. 2D), and expression of a *mod-1* cDNA in interneurons AIY or AIZ/AIB fully rescues *mod-1* mutants. Therefore, I propose that enhanced serotonergic signaling in ADF neurons acts through MOD-1 in several interneurons to control aversive olfactory learning (Fig. 2E).

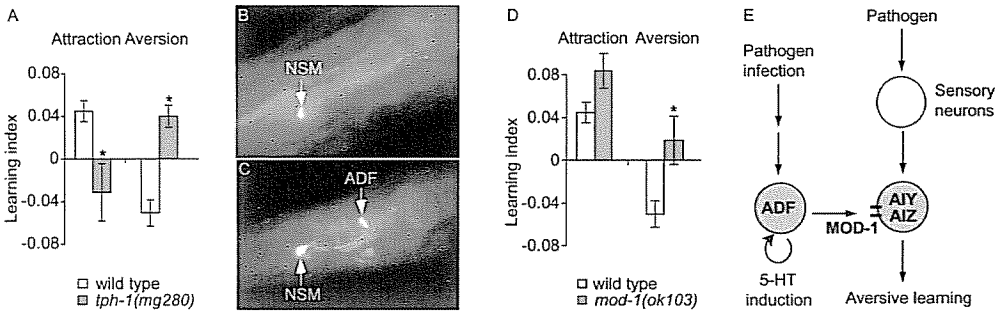

Figure 2. A, *tph-1* animals are defective in attractive and aversive learning. B, C, Anti-serotonin staining in ADF neurons is enhanced in PA14-trained wile type animals (C) compared to OP50-fed animals (B). D, *mod-1* animals are defective in aversive learning. E, Aversive olfactory learning circuit.

Proposed Research

1. From serotonergic neuromodulatory pathway to the olfactory learning circuit. *C. elegans* associates noxious infections resulted from ingestion of pathogenic bacteria with the odors of the pathogen. To understand how the olfaction is modified by the noxious stimuli, it is important to identify the cellular sites where neuromodulatory inputs converge with the olfactory pathway. The identification of the olfactory circuit should provide a cellular basis for mechanistic studies on olfactory learning.

(A) Identification of chemosensory neurons required for olfactory learning. Most *C. elegans* chemosensory neurons use either the cGMP-gated channel TAX-2/TAX-4 or TRPV channels OSM-9/OCR-2 for sensory transduction. My preliminary results showed that both *tax-4* and *osm-9* mutants are defective in learning. By testing mutants specifically defective in differentiation of major chemosensory neurons, I identified a requirement of chemosensory neurons AWC and ASI for learning. AWC and ASI neurons express *tax-4*. I will test the requirement of these cells by specifically rescuing the functions of these neurons in a *tax-4* background and ask if that restores learning. Serotonergic neurons ADF express *osm-9*, and I will test the function of ADF by asking if learning is rescued in *osm-9* animals specifically expressing *osm-9* in ADF. As a complementary approach, I will also use genetic ablation of specific cells by ectopically expressing the cell death activator EGL-1[16] in specific sensory neurons in the circuit and tested for learning. I hypothesize that ADF, AWC and ASI neurons function as chemosensory neurons in the learning circuit: serotonergic neurons ADF in the negative reinforcing pathway sense pathogen infections, and sensory neurons AWC and ASI in the olfactory pathway sense specific odors from the infecting pathogens.

(B) Identification of downstream neurons in the olfactory pathway for pathogen-learning. ADF neurons directly synapses onto AIY and AIZ interneurons, and AIY, AIZ, AIA, and AIB are major interneurons downstream of chemosensory neurons[7]. Since the serotonergic receptor MOD-1 acts in AIB, AIY and/or AIZ, neuromodulatory inputs induced by pathogen infections may converge at and modify activity of these olfactory interneurons. To identify downstream neurons that function in the olfactory pathway in the learning, I will use two different approaches.

- Test candidate genes. I will test *mgl-1*[17] and *gpa-2*[18] mutants to study function of AIA interneurons in learning, *glr-1*[19,20] and *odr-2*[21] for AIB neurons, *ttx-3*[22] for AIA/AIY and *lin-11*[23] for AIZ.

- Cell ablation experiments. Since mutations that compromise the function of interneurons usually affect multiple cells, I will develop a single-worm learning assay and use it in combination with cell-ablation techniques to identify the contribution of individual interneurons. I will train single animals with four hours of pathogen exposure, which is sufficient to induce aversive learning[24]. I will assay each individual animal for learning ability in a binary choice assay. Using this single-animal learning assay, I should be able to test animals that have individual interneurons killed by a laser microbeam to identify the olfactory interneurons required for pathogen learning.

2. Mechanisms that upregulate serotonin signaling in ADF neurons upon pathogen exposure. Our molecular and cellular experiments suggested that pathogen exposure enhances serotonergic signaling in ADF neurons and enhanced serotonin signaling accelerates learning[24]. In mammals, flies, and *Aplysia* [25,26], the molecular understanding of learning mechanisms has focused on the neurons postsynaptic to the neuromodulatory pathway; little has been done to understand the molecular changes in the modulatory pathway itself. Our model has a unique ability to fill this gap: we identified a single pair of serotonergic neurons whose enhanced signaling promotes learning. I propose the following projects to elucidate the mechanisms by which pathogen infections enhance serotonin signaling.

(A) Identification of regulatory domains of TPH-1 that up-regulate serotonin synthesis and control olfactory learning. I found that pathogen exposure upregulates TPH-1, the rate-limiting enzyme for serotonin synthesis, both transcriptionally and post-transcriptionally. I also showed that post-transcriptional activation of TPH-1 is sufficient to up-regulate serotonin and rescue learning. To characterize upregulation of serotonin in ADF, I will first identify TPH-1 protein domains required for enzyme activation and olfactory learning. The mammalian TPH-1 homolog consists of separable regulatory and catalytic domains; the regulatory domain can be phosphorylated by protein kinase A to regulate enzymatic activity[27]. *C. elegans* TPH-1 is highly homologous to mammalian TPH-1[12]. I hypothesize that the N-terminal regulatory domain is required for activation of TPH-1 by pathogens. TPH-1 clones lacking different N- terminal regions will be expressed in ADF neurons in *tph-1* animals. Since deletion of the N-terminal domain of mammalian TPH-1 does not abolish basal activity of the enzyme[27], I expect that some N-terminal truncated *C. elegans* TPH-1 still synthesize serotonin. I will first score enzymatic activity of the truncated proteins by antibody staining of serotonin in transgenic strains. Then among all the truncated proteins that maintain basal activity I will examine the ability of the truncated TPH-1 mutants to upregulate serotonin and regulate learning on pathogenic bacteria.

(B) Identification of mechanisms by which pathogen exposure upregulates serotonin signaling in ADF neurons.

- Effects of innate immunity pathways on upregulation of serotonin signaling. A number of signaling pathways are known to regulate the *C. elegans* innate immunity response to pathogens. Since TPH-1 activity is regulated during learning on pathogens, it could interact with these signaling pathways. I will examine the upregulation of serotonin by pathogen exposure in several mutant backgrounds: (1) Mutants in the TGF-β like pathway[28] (*dbl-1, sma-2, sma-3, sma-4,* and *sma-6*), and mutants in the p38 MAP kinase pathway[29] (*nsy-1* and *sek-1*). All these mutants are hypersensitive to *P. aeruginosa* PA14 infections; (2), mutants of an insulin-like receptor (*daf-2*), which increase animals' resistance to PA14[30]; (3) mutants of CaM Kinase II *unc-43,* whose mammalian homologs affects TPH-1 activity. If some of the proteins affect the regulation of serotonin by pathogens, I will use genetic and biochemical approach to study the detailed mechanisms of regulations.
- Worm genetics. If none of the known innate immunity pathways affect upregulation of serotonin, I will seek novel signaling pathways using genetic screens. Since serotonin immunoreactivity is induced in ADF neurons in *P. aeruginosa* PA14-trained animals compared to animals fed on *E. coli* OP50, I will use antibody staining to identify mutants that fail to upregulate serotonin levels after

pathogen exposure. I expect to identify genes that function to recognize pathogens, or to sense infections, or to transduce signaling from infected tissues to neurons. Isolation and characterization of the mutants will likely lead to the mechanisms that enhance serotonin signaling.

- Genomic approach. I showed that expression of *tph-1* in ADF neurons is regulated both transcriptionally and post-transcriptionally in trained animals. To identify molecular responses of ADF to pathogen exposure, I will use whole genome DNA microarrays to analyze transcripts of ADF neurons, isolated by a micropipet from trained and naïve animals (Goodman, M. B., personal communications). Transcription profiles of mature mouse olfactory sensory neurons and olfactory progenitor cells have been generated from single isolated neurons[31]. I will identify genes whose expression in ADF is regulated by pathogen training, and I will use *tph-1* gene as a positive control because expression of *tph-1* in ADF neurons is upregulated by pathogen infections. I will study function of identified genes using RNAi approach and deletion mutants.

3. Mechanisms by which enhanced neuromodulatory signaling modifies olfaction. I propose a simple model for *C. elegans* olfactory learning on pathogens: pathogen exposure increases serotonergic signaling in ADF neurons, which strengthens or weakens responses to chemosensory inputs of AWC and ASI neurons relative to other olfactory inputs. These modifications result in avoidance of the infecting pathogens. How neuromodulatory inputs modify the functions of learning circuits is a central question in the learning field. Using the simple learning circuit that directs *C. elegans* to avoid pathogens, I will test my hypothesis and study the mechanisms of this process.

(A) Characterize functional modifications on cellular components of the learning circuit.

- Quantitative behavioral assays for individual chemosensory neurons. My preliminary results suggested a requirement of chemosensory neurons AWC and ASI for pathogen learning. I hypothesize that pathogen exposure would result in changes in an animal's innate responses to environmental stimuli, generating behavioral changes to the training pathogens. To test functional modifications of AWC neurons, I will compare dose-response curves of trained animals with those of naïve animals, using chemotaxis assays on AWC-sensed attractive odors. AWC neurons sense a variety of attractive odors through cGMP-gated TAX-2/TAX-4 channels. Although the molecular nature of odors that animals sense from bacteria is not clear, chemotaxis assays on AWC-sensed odors serves as a behavioral read-out for responses to AWC inputs. If dose-response curves of trained animals shift to left, it would suggest a decreased sensitivity to AWC inputs; and if it shifts to the right, it would suggest increased sensitivity. I will use chemotaxis assays on AWA-sensed odorants as a control, because AWA chemosensory neurons are not required for pathogen learning and I do not expect to see a significant change in olfactory responses to AWA inputs. I will use similar approaches to test functional modifications of ASI neurons.
- Quantitative behavioral assays for interneurons. I found that serotonin receptor MOD-1 functions in AIY, AIB and possibly AIZ interneurons to regulate olfactory learning. It is possible that serotonin signaling through MOD-1 modifies neural activity of AIB, AIY and AIZ interneurons. Since these cells also control specific aspects of *C. elegans* navigation behaviors, such as the frequency of reversals and turns[32], I will use navigation assays to analyze functional modifications of these neurons. When animals navigate off-food, AIB neurons increase turning frequency and AIY neurons decrease turns. AIZ cells increase an animal's turning frequency on-food. I will examine navigation behavior in trained and naive animals to ask if pathogen training results in differences in turning frequencies. To confirm the results, I will use a laser microbeam to kill the interesting neurons in individual animals and perform the regular training. I will test if the modifications in navigation after training would disappear in animals that have specific neurons killed.
- Functional analyses of olfactory pathways by imaging approaches. To directly characterize functional modifications of sensory neurons and interneurons in the olfactory pathway, I will

express G-CaMP[33] in the olfactory neurons and visualize neuronal responses to odors before and after learning. Reliable G-CaMP fluorescence responses to odors have been recorded in olfactory sensory neurons (AWC) and interneurons (AIY and AIZ) (Chalasani, S., Chronis, N., Zimmer, M. and Bargmann, C. I., unpublished results). Imaging approaches should allow me to visualize functional modifications of relevant olfactory neurons.

- Serotonin receptors. In the mammalian nervous system, AMPA receptor trafficking to and away from synaptic plasma membrane is essential for LTP and LTD, two physiological correlates of learning[34]. I will ask if a similar process regulates the serotonin receptor MOD-1 during olfactory learning in *C. elegans*. I will examine the subcellular localization and expression intensity of MOD-1 before and after learning. As an inhibitory receptor in a serotonergic learning circuit, MOD-1 is very different from the AMPA receptor; therefore, these studies could provide new insights into neural plasticity.

(B) Characterize mechanisms that regulate neural plasticity of the learning circuit using genetic screens. After I characterize the functional modifications of olfactory circuits by pathogen exposure using different behavioral assays (see 3A), I will use behavioral assays as a read-out in genetic screens to identify mutants that fail to modify their neuronal functions after learning. If trained wild type animals acquire increased sensitivity to AWC inputs, I will look for mutants that fail to increase AWC responses after training. Similarly, I will also be able to identify mutants that fail to modify functions of ASI. If pathogen exposure changes navigation behaviors by modifying the function of olfactory interneurons, I will design genetic screens to identify mutants that fail to modify navigation after learning. From these screens, I expect to identify genes that function in upregulating serotonin signaling, or that function in regulating neurotransmission, or that function in modifying neural activity of olfactory neurons. Among mutants that are able to upregulate ADF serotonin but fail to modify olfaction, I should be able to identify mutants defective in pairing the neuromodulatory inputs with olfactory pathways. Isolation and detailed characterization of mutants should reveal the mechanisms that regulate plasticity of the learning circuit.

Professional Goals

My graduate and postdoctoral research has prepared me to study olfactory learning using a variety of different approaches. I intend to work in an academic institute and lead a lab that uses *C. elegans* as a genetic and genomic model in combination with behavior and imaging approaches to understand the molecular and cellular mechanisms of olfactory learning. The research proposed above will greatly enrich our insights on the function of neural circuits.

I am also looking forward to my role as a mentor to both graduate and undergraduate students. While in the Chalfie and Bargmann's labs, I have supervised graduate students during their rotations. As a mentor, I would like to share my scientific interests and insights with students and provide research guidance and support at the early stages of their careers. I am also looking forward to interacting with my peer scientists. During both my graduate and postdoctoral research, I had successful and pleasant collaborations with my colleagues and scientists outside of the laboratory or my field. I would like to continue these relationships and develop new ones in my future scientific career.

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Teaching Interests

I am looking forward to teaching students of all levels. I am trained in neuroscience. My graduate study focused on developmental neurobiology and my postdoc work focuses on the function of neural circuits. I am ready to teach neurobiology, cell biology, genetics and molecular biology. I am also ready to accept the challenge of teaching outside of my major training. I would like to teach students not only the knowledge, but also the scientific ways to critically analyze the results from scientific research. I think that it is important for young students to learn both the creative and the stringent ways to think about and conduct research.