structure. The model network was subjected to synaptic damage representative of PD pathology, and the ensuing responses to simulated odor input were characterized and compared to the baseline response. Results show increased localization of model network activity in response to odors, as well as disruption to network oscillations. The severity of the effects depends on the size and structure of the network. Future work aims to implement recent models that incorporate other OB features likely affected by PD, such as dopaminergic input. By computationally investigating the OB under conditions caused by PD, this work attempts to better understand OB function and the mechanisms by which PD impairs the OB and other regions of the brain.

1405-Pos
Predicting the Morphology of Class IV Neurons from the Dynamics of Dendritic Growth in *Drosophila*
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The development of neurons into their stereotyped morphology is paramount to their function. The class IV neurons of the Drosophila melanogaster larva are sensory neurons that develop a complex, highly branched dendritic arbor sensitive to mechanical stimuli. The fully-developed dendritic tree results from a multitude of stochastic processes including dendritic tip growth, branching and self-avoidance. Previous studies have identified key molecular players involved in these processes, which include actin, microtubules, molecular motors, Golgi outposts, endosomes and the Down Syndrome cell adhesion molecule (DSCAM). However, it is yet unknown how these dendritic processes can produce the observed morphologies of the class IV neurons. Here, we formulated an agent-based model of dendritic growth that takes inputs from our previous measurements and analysis of the tip dynamics and branching process. The rules of the agent are based on the observed local behavior of dendritic tips: 1) Branches elongate stochastically by visiting three dynamical states, i.e, growing, paused and shrinking; 2) New branches are created at a rate that is proportional to the local density of dendrites; 3) Branches stop growing when they collide with another branch, which mimics contact-based retraction. Using these rules constrained by experimental observations, we show that the model recapitulates the morphology of class IV neurons. This allows us to bridge the scales of class IV dendritic growth by connecting the local tip dynamics ($\sim$1 μm) to the global tree growth ($\sim$1 mm). In summary, our results establish a mechanistic approach to understand how the small-scale growth dynamics of class IV neurons can shape the large-scale morphology of the dendritic tree.

1406-Pos
A Kinetic Model to Account for Selective Trapping of Weak Bases Inside Acidic Intra-Cellular Vesicles
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Recent work from our group has characterized selective trapping of nicotinic acetylcholine receptor (nAChR) ligands within neurons in the brain (Govind et al. eLife 2017). The ligands are weak bases that bind with high-affinity to *α4β2*-type nAChRs (*α4β2R*s), such as the anti-smoking drug varenicline (Chantix) and epibatidine. Selective trapping occurs within *α4β2*s-containing acidic vesicles of cells and neurons. Slow release of trapped varenicline reduces effects of long-term nicotine exposure. Selective trapping is further regulated by nicotine exposure, which increases the number of *α4β2*s-containing acidic vesicles. Nicotine, also a weak base, is not trapped due to its lower pKa and lower affinity for *α4β2R*s. Here, we present a kinetic model that accounts for the biphasic behavior of these weak bases leaving the cell after accumulating in the acidic vesicles and binding to the nAChRs on the surface of the vesicles, within intracellular membranes, and on the exterior of the cell. Intracellular accumulation and release of molecules by diffusion into and out of cytosol and vesicles is calculated using Fick's Law of diffusion (neutral molecule) and the Nernst-Planck equation for ions (Trapp and Horobin 2005). Ligand binding to *α4β2R*s is modeled using empirical receptor-ligand kinetics. Solving our coupled differential equations numerically has accurately approximated experimental data for epibatidine accumulation and release in vesicles. The model is mainly limited by inclusion of pKa and binding affinity as the only parameters directly causing the trapping. Our model also suggests that the vesicle membrane receptor concentration is an important factor, a notion that we hope to verify experimentally. Using this model, we have been able to model kinetic trapping of the weak-base *α4β2* ligands Nifene and 2-F-A8538, and to explain differences in their kinetics during positron emission tomography.

1407-Pos
Ketone Diets Can Reverse Some Brain Activities that are Lost in Aging
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We study how the neuronal activity in human brains differs with age, young vs. old, and with diet, glucose-based vs. ketogenic. fMRI studies have previously shown that the functional connectivity among brain regions decreases in old age. Here, using ultra-high field (7T) fMRI in humans, in conjunction with an Ising model of network activity, we show that this functional signature of age-related cognitive decline is partially reversed by ketogenic diets. These dependencies can be modeled as a low-dimensional component of network connectivity, interpreted as a degree of synchronization of activity across the brain network, and expressed in the language of thermodynamic critical points. Brains that are young or glucose fed are poised at a critical point. Older brains are in the subcritical region where there is a unimodal distribution of synchrony. Ketone-fed brains are supercritical, where synchrony is bimodal. The present work gives new quantitative insights into how brain network activity changes with aging and diet, and indicates a relationship between neurocognition and energy utilization.

Posters: Neuroscience: Experimental Approaches and Tools

1408-Pos
Optical Modulation of Receptor Tyrosine Kinase Signaling during Cell Differentiation and Embryonic Development
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The neurotrophic signaling pathway regulates a wide spectrum of cellular functions such as cell survival, proliferation, differentiation, and apoptosis. It also plays a key role in cell fate determination during embryonic development. Signaling output of the neurotrophic pathway varies with its temporal kinetics and spatial localization. However, a quantitative delineation of signaling kinetics is limited due to a lack of tools that allows precise control of the neurotrophic signaling in time and space. Non-neuronal optogenetics, an emerging technology that utilizes light to control intracellular signaling pathways, offer an alternative solution to address this challenge. In this presentation, I will introduce optogenetic systems recently developed in our laboratory that allow for reversible and bidirectional optical control of neurotrophic signaling pathway in intact cells and in developing *Xenopus laevis* embryos. I will also discuss the limitations of current non-neuronal optogenetics and update you with current progress in the field in addressing these challenges.

1409-Pos
An *in vitro* System for Studying Nematode Mechanosensory Neurons
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The primary function of mechanosensory neurons is to detect and convey information about mechanical cues, including touch and self-generated changes in muscle length. This function depends on mechanosensitive ion channels, but also the interaction between mechanosensory neurons and their surrounding tissues. With its transparent body, easily manipulated genome, and hundreds of known mutants, the roundworm C. elegans is an ideal model animal for studying the biophysics of mechanosensory neurons in vivo. Calcium indicators, expressed under cell-specific promoters, enable imaging of neural impulses in response to mechanical cues. The elements of mechanosensitivity that are intrinsic to these neurons or dependent upon interactions with surrounding tissues are not known. We seek to address this question by developing an in vitro system that provides greater control over the neuron's environment while preserving the advantages of studying mechanosensation in C. elegans. We present a cell culture system consisting of a purified population of identified primary neurons from dissociated C. elegans embryos, grown on geometrically defined patterns of adhesion proteins that control neurite outgrowth and cell shape. Using a CRISPR/Cas-9 genetic approach, we introduced puromycin resistance