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ABSTRACT BOOK

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polarity and subsequent protrusion of growth cone lamellipodia and filopodia in response to this polarity (the Polarity/Protrusion model). In response to UNC-6/Netrin, UNC-40 stimulates protrusion dorsally, and UNC-5 inhibits protrusion ventrally, resulting in dorsally-directed growth cone migration away from UNC-6/Netrin. *unc-5* encodes full-length long isoforms, as well as a short isoform truncated at the C-terminus and lacking most cytoplasmic domains (*unc-5B)*. *unc-5* null mutant VD growth cones (lacking both long and short isoforms) display unpolarized and excessive protrusion, whereas activated *myr::unc-5* results in small growth cones with reduced protrusion. Long-isoform-specific mutants also display unpolarized and over-protrusive growth cones, but are less uncoordinated and have less severe axon guidance defects than nulls. This suggests that long-isoform-specific mutants are not null and that the *unc-5* short isoform contributes to axon guidance. Precise genomic deletion of *unc-5B* resulted in unpolarized growth cones with reduced protrusion. It also affects the dorsal asymmetrical accumulation of F-actin but not MT+ end accumulation. This suggest that in addition to differentially regulating growth cone morphology, UNC-5B plays a role in regulating cytoskeletal dynamics to maintain dorsal polarity of filopodial protrusions. Transgenic expression of *unc-5A* long isoform rescued uncoordinated locomotion, axon guidance defects, and growth cone protrusion of *unc-5*null mutants. Transgenic expression of *unc-5B* short rescued uncoordinated locomotion and axon guidance defects, but did not rescue excess protrusion of *unc-5* null mutants. In sum, these results show that the long and short isoforms of UNC-5 play differential roles in growth cone migration. The long isoform UNC-5A inhibits growth cone protrusions and the UNC-5B short isoform is pro-protrusive in nature and the balance between the two isoforms is crucial for the development of VD/DD neurons.

1007V **Neural modulation of behavioral state transitions in foraging strategies in *C. elegans*** Maria Gabriela Blanco[1], Jeremy Florman[2], Mark Alkema[3], María José De Rosa[1], Diego Rayes[11]Biología, Bioquímica y Farmacia, Universidad Nacional del Sur, Instituto de Investigaciones Bioquímicas de Bahía Blanca, [2]Departmen of Neurobiology, University of Massachusetts Chan Medical School, [3]Department of Neurobiology, University of Massachusetts Chan Medical School

Adequate feeding behavior is essential for animal survival and it is regulated not only by the digestive system but also by the nervous system (NS). The NS allows the animal to respond flexibly to changes in the environment depending on the availability of food and the nutritional internal state. Despite feeding behaviors have been studied for decades, understanding the mechanisms involved in different animals´ responses to food depending on its internal state (satiated or fasted/stressed) is still a major challenge. Referred to as the “happiness hormone”, serotonin (5-HT) has been shown to increase with food stimulus and modulate feeding in different animals, suggesting that the role of 5-HT is conserved in nature. On the other hand, noradrenaline (NA), implicated in triggering a stress response, is involved in appetite control by reducing food ingestion. Interestingly, there are reports showing that a lesion of the serotonergic system enhances the effect of noradrenergic drugs. These findings indicate an interaction between serotonergic and noradrenergic signaling. However, the mechanism and relevance of this interplay are not entirely clear. Therefore, our goal is to investigate the molecular processes underlying this interaction.

The complexity of the mammalian brain complicates the study of neuronal processes. The nematode *Caenorhabditis elegans* is suitable for understanding neuronal signaling because of its simple and well-described nervous system. We found that during prolonged fasting, animals decrease their locomotion, which can be resumed by adding tyramine (TA), the analog of NA in invertebrates. 5-HT produces the opposite effect by reducing locomotion, suggesting that 5-HT acts antagonistically to TA. Moreover, it has been shown that when the environment improves and fasted animals encounter food, they release 5-HT to slow their locomotion and promote feeding. Interestingly, we found that this slowing response and the activity of the serotonergic neurons upon food encounter are enhanced in TA-deficient mutants compared to wild-type animals. Given that we also show that TA levels decrease during fasting, we hypothesize that this disinhibits the serotonergic neurons and favors their activity upon refeeding, allowing the animal to exploit the new source of food. Considering the conservation of neuronal components, we believe that our results may contribute to the understanding of the nervous control of state dependent foraging strategies.

1008V **Temperature regulates glia morphogenesis through thermosensory circuits** Junyu Zheng[1], Shaochen Wang[1], Mengqing Wang[1], Zhiyong Shao[21]Fudan University, [2]Institutes of Brain Science, Fudan University

Astrocytes are the most abundant macroglia in the brain and play critical roles in regulating neural development and functions. The diversity of astrocyte functions is largely determined by the heterogeneity of its morphology. However, how the astrocyte morphology is established remains largely unknown. Temperature perturbation affects neuronal development and function in both vertebrates and invertebrates, and high temperature has been shown to suppress astrocyte division and viability in tissue culture. However, if and how the temperature affects astrocyte morphogenesis *in vivo* remain unknown. In this study, we found that high cultivate temperature (26℃) promoted *C. elegans* astrocyte CEPsh endfoot extension, which requires thermosensory AWC neurons and the postsynaptic AIY interneurons. We further demonstrated that two glutamate gated chloride channels, GLC-3 and GLC-4, were required for the glia extension. Finally, we identified that the guanyl-nucleotide exchange factor EPHX-1 and GTPase CDC-42 act cell-automatically in the glia to regulate endfoot extension under high temperature. Collectively, these data suggest a model that high temperature acts through a thermosensory circuit to modulate the glia morphogenesis in *C. elegans*, which provides a novel mechanism underlying glia morphogenesis and may provide insights into high-temperature related