The University of Chicago
Department of Statistics

Seminar Series

DAVID BIRON

Department of Physics
The University of Chicago

So, do worms sleep?

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Refreshments following the seminar in Eckhart 110.

ABSTRACT

Despite much progress in our understanding of *C. elegans* locomotion and navigation, little is known about the regulation of the absence of movement. Yet behavioral quiescent states are *universal* to the animal world, with the most famous and mysterious of these being sleep. The roundworm *C. elegans* is in many respects the simplest — and admittedly the most simplistic — model system that exhibits a behavior that resembles sleep. It has only 302 neurons, the connections of which have been anatomically mapped, a short life cycle and an optically transparent body. The worm develops through four larval stages before it reaches adulthood. At the end of each of these stages it exhibits a quiescent behavior called *lethargus*. David Raizen et al. recently demonstrated that lethargus bears behavioral similarities to sleep, such as *reversibility* (the worms wake up), *sensory gating* (an elevated threshold for responding to sensory stimuli) and *homeostatic control* (following deprivation, lethargus is resumed faster and "deeper").

Our basic characterization of the microarchitecture of the behavioral patterns during lethargus builds on the observation of individual bouts of quiescence and motion during its 2–3 hour duration. The duration of individual bouts ranges from a few to about 100 seconds. A two state Markov chain can be used to describe the statistics of these bouts, where the transition rates undergo slow modulation. The resulting statistics provide a robust set of phenotypic measures of wild-type and behavior and the results of genetic perturbations. We have also begun to characterize the body posture of the worm during lethargus. We found that the curvature along the lethargic body is lower than that of awake worms. Moreover, during lethargus the number of body bends exhibits stereotypical temporal dynamics. Van Buskirk and Sternberg showed that the activation of the EGFR LET-23 in the ALA neuron can suppress motion, and that EGF signaling promotes quiescence during lethargus. We ablated the ALA neuron and observed an increase in the mean duration of quiescence bouts. Expressing tetanus toxin in the AVE neurons, postsynaptic partners of ALA, resulted in the opposite effect. Our data is thus consistent with an inhibitory connection between ALA and AVE.

In summary, we observed that lethargus is a complex and highly regulated behavior: it can be dissected to quantifiable elements, which exhibit complex temporal dynamics and are modulated by molecular and neuronal activity. This suggests that it is unlikely that lethargus is merely the result of mechanical restriction during molting.
