BMC Pharmacology

Oral presentation

Open Access

A cGMP-dependent protein kinase plays a pivotal role in the control of behavioral quiescence in *C. elegans*

David M Raizen*[1,2], Allan I Pack[2] and Meera Sundaram[3]

Address: [1]Department of Neurology, U. of Pennsyvania Medical School, Philadelphia USA, [2]Center for Sleep, U. of Pennsylvania Medical School, Philadelphia, USA and [3]Department of Genetics, U. of Pennsylavnia Medical School, Philadelphia, USA

Email: David M Raizen* - raizen@mail.med.upenn.edu

* Corresponding author

from 2nd International Conference of cGMP Generators, Effectors and Therapeutic Implications
Potsdam, Germany, 10–12 June, 2005

Published: 16 June 2005

BMC Pharmacology 2005, **5**(Suppl 1):S8 doi:10.1186/1471-2210-5-S1-S8

Virtually all animals have periods of inactivity or behavioral quiescence, when locomotion stops. In Drosophila, behavioral quiescence corresponds to sleep and is controlled by some of the same genes and neurochemicals that affect mammalian sleep, suggesting that the genetic control of quiescence is phylogenetically ancient. We provide, for the first time, a quantitative description of behavioral quiescence in *C. elegans*, a nematode. As noted by others (Singh and Sulston '78), periods of quiescence occur during the transitions between the four larval stages, but not during the adult stage. We provide descriptive statistics for the normal onset, duration, and consolidation of these quiescent periods, as a baseline for studying mutants. We find that the quiescence is fully reversible upon mechanical stimulation, indicating that this behavioral state is under nervous system regulation. To begin to unravel the genetic machinery that controls quiescence, we have been studying *eat-7 (ad450sd)*, a *C. elegans* dominant mutant that displays increased behavioral quiescence during lethargus as well as an increase in quiescence during the adult period. When stimulated mechanically or when starved, *eat-7* mutants move normally, suggesting that the defect in *eat-7* is not in the muscles or motor neurons. *eat-7* has the same genetic map position but opposite phenotypes to those of mutants with loss of function of the EGL-4 cGMP-dependent protein kinase (PKG). We found that *eat-7* mutants contain a glycine to arginine mutation in PKG. This glycine, found in the cGMP-binding domain closer to the kinase domain, it conserved in all cyclic nucleotide-binding proteins, suggesting that this mutation would have major consequences for enzyme function.