Abstract/Session Information for Program Number 450C

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Abstract Content

Program Nr: **450C**

Mutations that Cause Food-Deprived Behavior in Well-Fed Animals. **Daniel Omura**, Bob Horvitz. HHMI, Dept. Biology, MIT, Cambridge, MA.

Acute food deprivation causes N2 animals to modulate their locomotion differently than well-fed animals upon entering a bacterial lawn[1]. Acutely food-deprived animals exhibit a serotonin-dependent 'enhanced slowing response' upon entering a bacterial lawn while well-fed animals exhibit a dopamine-dependent 'basal slowing response' upon entering a bacterial lawn. Acute food deprivation has almost no effect on N2 locomotion in the absence of food. We are interested in identifying genes that establish and respond to a food-deprived state.

Beth Sawin isolated mutants that do not exhibit food-deprived behavior after an acute food-deprivation event[1]. Conversely, we sought mutants mutants that constitutively exhibit food-deprived behavior in the absence of acute food-deprivation. To do so we screened for mutants that may inappropriately exhibit a constitutive enhanced slowing response in the absence of acute food deprivation. Our starting strain included a null mutation, *n3314*, in the *mod-5* gene which encodes a serotonin reuptake transporter. This mutation causes a hyper-enhanced slowing response in which *mod-5* animals become temporarily paralyzed upon entering a bacterial lawn specifically after acute food-deprivation[1,2]. We isolated a number of mutants that exhibit a food-dependent paralysis in the absence of acute food deprivation. We then tested for serotonin dependence of this behavior by assaying recovery of locomotion after preincubation with the serotonin receptor antagonist methiothepin. Methiothepin antagonizes the enhanced slowing response, at least in part, by inhibiting the the serotonin-gated chloride channel MOD-1[3]. We screened 5250 EMS mutagenized and 20375 Mos1 transposon mutagenized haploid genomes and isolated 12 mutants, six from each screen. These mutants exhibit a food-dependent paralysis that is antagonized by methiothepin. Most of the mutants grow at a normal rate. Some are slightly Egl.

By analyzing genes that underlie the enhanced slowing response, we hope to understand more about how acute food-deprivation modulates *C. elegans* behavior.

[1]Sawin, E.R., Ranganathan, R. & Horvitz, H.R., *Neuron* (2000); [2]Ranganathan, R., Sawin, E.R., Trent, C. & Horvitz, H.R., *J Neurosci* 2001; [3]Ranganathan, R., Cannon, S. C. & Horvitz, H. R., *Nature* (2000).

Session Information

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Abstract Information

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