202. Some Dauer Formation, Social Feeding, and Chemotaxis Mutants are Abnormal in the Enhanced Slowing Response

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Well-fed animals encountering a bacterial lawn exhibit a dopamine-dependent basal slowing response mediated by the mechanosensory ADE, PDE, and CEP neurons. Acutely food-deprived animals exhibit a serotonin-dependent enhanced slowing response mediated by an undefined set of sensory neurons (Sawin, E. R., et al., *Neuron* 26, 619-31, 2000). The serotonin-gated chloride channel MOD-1 (modulation of locomotion defective) and the serotonin reuptake transporter MOD-5 are involved in propagating and attenuating the enhanced slowing response, respectively (Ranganathan, R., et al., *J Neurosci* 21, 5871-84, 2001; Ranganathan, R., et al., *Nature*408, 470-5, 2000). The mutation *mod-6(n3076)* causes animals to have a reduced enhanced slowing response and is likely an allele of *che-3* (chemotaxis defective). Some but not all *che-3* alleles cause defects in the enhanced slowing response. Our investigation of mutants bearing defects in sensory neurons in Mod behavior has led to the hypothesis that mutants broadly defective in sensory neuron function exhibit increased slowing, while mutants defective in a subset of chemosensory neurons are Mod and exhibit reduced enhanced slowing. Defects in a subset of chemosensory neurons that inhibit locomotion may specifically reduce the enhanced slowing response, while defects in a larger group of neurons that affect locomotion may result in greater slowing on bacteria. We are currently attempting to identify specific neurons and genetic pathways involved in the detection of bacteria when animals are food deprived.

Acute food deprivation results in an internal state change that can be detected when animals are re-exposed to their food source, as outlined above. Mutants were previously identified that have a reduced enhanced slowing response after acute food deprivation. We have more recently screened for mutants that exhibit constitutive enhanced slowing. Such mutants would exhibit a serotonin-dependent slowing response in both the well-fed and food-deprived states, which could reflect a defect in the generation, storage, or signaling of a well-fed state. In this screen we sought mutations that conferred paralysis upon entering a bacterial lawn in the absence of acute food deprivation in a *mod-5(n3314)* background. Mutants that moved well in the absence of bacteria, grew at a roughly normal rate, and displayed a paralysis that was antagonized by the serotonin receptor antagonist methiothepin were saved for further analysis. Ten such mutants were isolated including a Mos1 transposon insertion allele of *mrp-1* (multidrug resistance protein familiy). Studies by others have found that *mrp-1* is synthetically dauer constitutive with *unc-31* (Yabe, T, et al., 2002 Japanese Worm Meeting abstract 5151). This finding led us to investigate the role of dauer genes in the enhanced slowing response. Thus far we have found dauer mutants that exhibit constitutive enhanced slowing as well as mutants that have reduced enhanced slowing. We are currently investigating the role of *mrp-1* and dauer pathway genes in the enhanced slowing response.