

214. Mutations that Cause Food-Deprived Behavior in Well-Fed Animals

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Acute food deprivation causes N2 animals to exhibit a serotonin-dependent slowing response when entering a bacterial lawn. This *enhanced slowing response* is absent in well-fed animals which exhibit a dopamine-dependent *basal slowing response* upon entering a bacterial lawn. Acute food deprivation has no significant effect on N2 locomotion in the absence of food. Thus, the condition of being acutely food deprived causes animals to react very differently to a food stimulus. Our laboratory is interested in determining what genes are involved in this food-dependent modulation of locomotion.

To find genes involved in establishing and responding to a food-deprived state in *C. elegans*, we have performed a screen, using EMS mutagenesis, for mutants that inappropriately exhibit a *constitutive enhanced slowing response* in the absence of acute food deprivation. Our starting strain includes a null mutation in the serotonin reuptake transporter *mod-5(n3314)*. This mutation causes a *hyper-enhanced slowing response*, so that *mod-5(n3314)* animals become temporarily paralyzed upon entering a bacterial lawn specifically after acute food-deprivation. We screened for animals that exhibit a food-dependent paralysis in the absence of acute food deprivation. We then tested for serotonin dependence of this paralysis. Six of the 33 mutants isolated in the screen regain mobility when pre-treated with the serotonin receptor antagonist methiothepin, which antagonizes the serotonin-gated chloride channel MOD-1, a key protein in the enhanced slowing response. These six mutants grow at a normal rate.

Currently we are screening for constitutive hyper-enhanced slowing mutants using Mos 1 transposon mutagenesis and have isolated several mutants. Through the identification of genes that underlie the enhanced slowing response, we hope to understand more about the generation, storage, and retrieval of potential hunger signals in *C. elegans*.