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376. FOOD-DEPRIVATION AND MODULATION OF BEHAVIOR: *mod-6* AND A SCREEN FOR NEW GENES

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Upon entering a bacterial lawn, well-fed hermaphrodites exhibit a basal slowing response, while acutely food-deprived animals exhibit enhanced slowing. The basal and enhanced slowing responses are genetically separable and involve different neurotransmitters. A number of cloned genes define a molecular genetic pathway in which serotonin signaling is critical for the enhanced slowing response. Food-deprived animals are no more sensitive to exogenous serotonin than well-fed animals, suggesting that food-deprivation induces a physiological change that modulates serotonin release rather than altering sensitivity to endogenous serotonin.

mod-6(n3076) (MODulation defective) mutants are defective in the enhanced slowing response, serotonin positive, slightly hypersensitive to exogenous serotonin, and fluoxetine (Prozac) resistant. These characteristics suggest that *mod-6* may be involved in modulating serotonin release in response to food deprivation. We mapped *mod-6* to a small interval on chromosome I and rescued the serotonin hypersensitivity phenotype of these mutants with cosmid F18C12. F18C12.1 corresponds to the cloned gene *che-3* (CHEmotaxis defective). We are presently determining the sequence of F18C12.1 in *n3076* mutants. *che-3* encodes a cytosolic dynein heavy chain required for the growth and maintenance of the ciliated processes of a subset of sensory neurons in *C. elegans*. Thus, *mod-6* mutants may be defective in the enhanced slowing response because they cannot detect a bacterial lawn by chemosensation.

We are also performing a *mod-5(n3314)* enhancer screen for mutants that display a constitutive hyperenhanced slowing response when entering a bacterial lawn. *mod-5* encodes a SErotonin Reuptake Transporter (SERT). SERTs have been shown to clear serotonin from the synapse after pre-synaptic release of the neurotransmitter, thus attenuating the serotonin signal. *mod-5(n3314)* mutants maintain a normal rate of locomotion in the absence of bacteria and exhibit a normal basal slowing response. However, upon food-deprivation these mutants become nearly immobilized upon entering a bacterial lawn. We have termed this more pronounced enhanced slowing response the hyperenhanced slowing response. We have isolated a number of *mod-5(n3314)* enhancers that cause the mutants to become immobilized when entering a bacterial lawn in the well-fed state while maintaining a nearly wild-type locomotory rate in the absence of bacteria. In this screen, we hope to isolate mutants that are defective in establishing and signaling the well-fed state but remain normal for the ingestion and absorption of food.