182 | The metabotropic glutamate receptor homologs MGL-1 and MGL-2 are key for sensing nutritional status in C. elegans

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The mechanisms that allow the nervous system to sense nutritional state and adapt animal behavior are poorly understood in most species. The simplicity of its NS and its known connectome make C. elegans a useful system to study these mechanisms. Results from our laboratory showed that inhibition of the tyraminergic neuron RIM during fasting, enhances serotonin release from other neurons when the animal reencounters food, allowing it to slow down locomotion and start feeding. Mutations in the GPCRs, MGL-1 and MGL-2, located in two presynaptic interneurons to RIM have been reported to induce autophagy even in well-fed animals. Here, we performed behavioral assays on mgl-1; mgl-2 mutants. We found that these animals, even when well fed, show a significant decrease in locomotion when they find food similar to fasted wild-type animals. Moreover, when we exposed these mutants to GFP-expressing bacteria, the fluorescence in the intestine is higher than that of wild-type animals, suggesting a higher feeding rate. These initial results suggest that the metabotropic receptors MGL-1 and MGL-2 are key for C. elegans to censor satiation molecules. We propose, therefore, to determine what these satiety signals are and the neuronal circuits involved. Given that this behavioral plasticity modulated by the nutritional state is observed throughout the animal kingdom, and that several fundamental processes are highly conserved, these results may provide universally relevant information.