Genetic suppressors of str-2 serotonin response defects in Caenorhabditis elegans

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C. elegans, a nematode, is a model organism to study animal behavior and development. The genome of the *C. elegans* shows that there is a similarity between the genes of the nematode and that of humans. *C. elegans* are widely used because of its rapid life cycle and its small size which makes it easy for laboratory cultivation. The str-2 gene in *C. elegans* is predicted to be responsible for receptor activity linked to olfactory responses. Through the binding of odorants on specific olfactory receptors, str-2 allows them to detect pheromones, environmental threats, and nutrition—essentially playing a vital role in their behavioral functioning. The egg-laying behavior of *C. elegans* is regulated by its surroundings and can be activated or inactivated through various environmental cues. However, without a properly functioning olfactory system, we hypothesize that *C. elegans* will be unable to initiate standard egg-laying activity through its inability to pick up on these environmental cues—even if they are in the presence of serotonin. In this study, we created a mutagenized str-2 *C. elegans* that was induced by ethyl methanesulfonate (EMS) which would also be resistant to the egg-laying ability response to serotonin. The *C. elegans* were treated and screened to ensure that they represented their ability to lay the least eggs in response to serotonin. Egg laying assays were repeated until the offspring was uniform. We found that in the life cycle of the the str-2 mutants were found have a decreased life cycle compared to the non-mutants. These mutants had crippled olfactory responses to environmental transmitters, and lacked sensory abilities that hindered life. In addition, we found that the number of offspring produced by the mutants were significantly less than those of the non-mutants.