Title:	A genetic approach towards understanding endoplasmic reticulum stress modulators using caenorhabditis elagans
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Abstract:	Homeostasis is a key trait for the survival of an organism. Cellular homeostasis is maintained by different organelles. The endoplasmic reticulum (ER), a cellular organelle, is the major site of protein folding, maturation, and post-translational modification in the cell. Homeostasis in ER is disrupted by an increase in protein folding demand compared to the protein folding capacity of ER. This disruption causes ER stress which leads to the accumulation of unfolded proteins. ER stress has been associated with a plethora of diseases, including diabetes, cancer, and neurodegenerative diseases like Alzheimer's, Parkinson's, etc. Evolutionarily conserved pathways are activated to cope with ER stress, but the various factors that can modulate the ER stress and ER stress responses are unknown. In this research project, we are trying to understand different modulators of ER stress using the model organism Caenorhabditis elegans. There are two aspects of ER stress that we are trying to understand. First, we wanted to know how microbial metabolites affect ER stress, so we carried out a genome-wide bacterial mutant screen to identify metabolites affecting ER stress. Secondly, we wanted to understand the regulation of R08E5.3 gene expression, which encodes for an S-adenosylmethionine (SAM)-dependent methyltransferase and has increased expression when exposed to dithiothreitol (DTT), an ER stressor. We carried out forward genetic screens to find mutants having upregulation of R08E5.3 expression even in the absence of DTT and mutants in which R08E5.3 expression does not increase even upon exposure to DTT. This study would lay the foundation for unravelling mechanisms by which microbial metabolites can affect ER stress and can be used in the future for the development of potential probiotic therapeutics. Also, it would shed light on the regulation of an important gene, R08E5.3, by the ER stressor DTT.
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