The Effects of Glucose on the Behavior and Nervous System of C. elegans

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Type 2 diabetes rates are on the rise due to changing lifestyles. Diabetes can cause hyperglycemic events, which can damage cells. However, how hyperglycemic damage occurs is not completely understood. Animal models such as the nematode Caenorhabditis elegans can be utilized to understand such damage. It is known that glucose affects both the transcriptome and behavior of C. elegans, but the pathways involved are not fully understood. I find that C. elegans settles in greater numbers on the border of a bacterial lawn when exposed to bacteria which have been exposed to and interacted with glucose. Through assays of mutants defective in TRPV (Transient Receptor Potential Vanilloid-type) ion channels, serotonergic signaling, and bordering behavior, this behavior is characterized as an increased bordering response, dependent on the genes osm-9, ocr-2, tph-1, gcy-35, gcy-36, and tax-4. This behavior is also found to be inducible upon exposure to ROS (Reactive Oxygen Species) and able to be modulated by oxygen levels. It is also revealed that C. elegans can recover from glucose exposure in both egg-laying and bordering assays. Additionally, by studying GFP-tagged genes, it is found that glucose does not change the spatial expression of certain upregulated genes. I therefore propose that C. elegans displays behavioral plasticity in its responses to glucose, utilizing reversible neuronal pathways. I also propose the glucose bordering behavior arises from recognition of heightened ROS levels on glucose-supplemented bacteria. Investigating such behavior is important in understanding the recognition of and responses to hyperglycemia in biological systems and understanding how hyperglycemic damage occurs.

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