Manipulating Odor-Driven Behavior in Drosophila melanogaster: A Model to Investigate ?-Aminobutyric Acid (GABA) Deficiency Disorders

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Imbalances between the inhibitory and excitatory neurotransmitters, gamma aminobutyric acid (GABA) and glutamate, result in hyperexcitability disorders, such as anxiety. Many of these disorders are currently treated by the use of benzodiazepines, which restore the homeostasis between GABA and glutamate. Although benzodiazepines have anxiolytic effects, they have the capability to act as a full inverse agonist, and result in addiction and tolerance in a patient experiencing anxiety. The purpose of this study was to use odor recognition in Drosophila melanogaster as a model to investigate efficacious methods of treating anxiety. Olfactory receptor neurons (ORNs) are located in the antennal lobe of the fruit fly and are responsible for all olfactory processing and recognition. These ORNs are controlled by GABA. In this study, the ORNs were manipulated by either excitation or suppression of neuronal activity. Through the use of a controlled odor presenting mechanism, fly behavior and response to non-aversive 10% apple cider vinegar will be determined. Two fly lines were used during experimentation. The flies exposed to UAS-TNTe had inhibited production of GABA and therefore demonstrated a significantly weaker response to the odor than the parental control. Those exposed to UAS-Chrimson had neurons with increased GABA production. The TNTe line of flies demonstrated a significantly weaker response to the administered odor due to the lack of GABA in the synapse. Contrastingly, the flies exposed to UAS-Chrimson had an increased production rate of GABA in the synapse. Using genetic methods such as UAS-Chrimson, to increase GABA production in the synapse may replace the potentially hazardous effects of benzodiazepines.

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