New Evidence for a Potential Citalopram Hydrobromide Mediated Mechanism Influencing the Suppression of Serotonergic Neurons during Brain Development

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Citalopram hydrobromide, an antidepressant agent found in the drug Celexa, has led to numerous suicide attempts (Schneeweiss, 2010). Suicidal thoughts and actions have been directly associated with sleeplessness and recurrent depression in numerous studies (Melnick, 2010). Citalopram hydrobromide is a selective serotonin reuptake inhibitor (SSRI) that blocks the reuptake of serotonin (5-HT). In a previous study, Canton-S D. melanogaster exposed to citalopram hydrobromide during brain development exhibited impaired cognitive function and increased activity. To determine whether citalopram hydrobromide had similar effects on locomotion and cognition in w118+ flies, locomotor and fast phototaxis assays were conducted. The results from these assays were similar to those seen in Canton-S flies. To explain the decreased cognitive function and increased activity observed, serotonergic neurons of citalopram hydrobromide treated, w118+ flies were immunostained, imaged coronally, and analyzed. When compared to placebo, 45.2% fewer serotonergic neurons were present in the the treated group. Due to the artificial 5-HT levels in the synapse that seem to reach the projected level typically observed after the termination of brain development, 5-HT during neurogenesis in the citalopram hydrobromide treated group inhibit serotonergic growth factors. SERT downregulation and apoptosis induced by postsynaptic receptor over-activation are also important. Citalopram hydrobromide may relieve depression in the short term but leaves devastating long term side effects during brain development. Therefore, if these results are commensurate in humans, then citalopram hydrobromide should not be prescribed to patients under 30.