The Efficacy of the Antioxidants, Ascorbic Acid, Glutathione, and α-Tocopherol, in Treating Fetal Alcohol Syndrome using Danio rerio as a Model Organism

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Fetal Alcohol Syndrome is a major cause of non-genetic developmental delays and neural and physical abnormalities. Free radicals, highly reactive by-products of ethanol (EtOH) metabolism, cause oxidative stress that affects cell development, survival, and migration leading to abnormal craniofacial development. Antioxidants detoxify free radicals and may play a role in reducing the effects of alcohol toxicity. This study examines the effects of the antioxidants, ?-tocopherol, glutathione, and ascorbic acid, on reducing ethanol's toxic effects in Fetal Alcohol Syndrome, using zebrafish as the model organism. Eggs were exposed to 100 μM tocopherol, 30 μM glutathione, 250 μM ascorbic acid, or no antioxidant at 0-24 hours post fertilization (hpf). At 6-24 hpf the eggs were exposed to 500 mM EtOH. At 5 days post fertilization (dpf) eye length, head length, body length, and heart rates were measured. Zebrafish eggs exposed to EtOH showed significant abnormalities in anterior body development which was evident in the head, body, and eye length measurements when compared to fish without EtOH exposure (P less than 0.01). To investigate the efficacy of prolonged antioxidant exposure, eggs were exposed to antioxidants for 0-5 dpf. This extended exposure with ascorbic acid and tocopherol further mitigated the toxic effects of EtOH on fetal development. Future research could focus on specific pathways affected by free radical by-products of EtOH. Antioxidants could be part of a multi-faceted treatment to reduce the teratogenic effects of EtOH.