Alleviating DNA Demethylation of 5hmC in Neurons and Zebrafish Embryos

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More than seventy conditions are attributed to oxidative stress, including neurodegenerative diseases, cancer, and chronic pathological processes. The exposure of DNA demethylation by the introduction of cells to environmental stressors that induce oxidative stress can lead to the depletion of a central compound of the epigenetic network and gene regulation known as 5hmC. The purpose of the experimentation is to use environmental stress reducers, S-adenosylmethionine (SAM) and Tocopherol, to combat the effects of environmental stress inducers (Bisphenol A, Hydroquinone, Pentachlorophenol) on neurons and hydrogen peroxide (H2O2) on zebrafish embryos. Serial dilutions of SAM and Tocopherol added to the oxidative stress inducers were applied to neurons to observe their effectiveness. Dilutions of SAM and Tocopherol were applied to zebrafish embryos exposed to H2O2. The average optical densities (calculated by the SpectraMax program) of neurons exposed to all concentrations of SAM and Tocopherol were higher than that of the control trials, indicating both SAM and Tocopherol were successful in combating oxidative stress inducers, which was deemed statistically significant by an ANOVA test. In the zebrafish embryo trials, the embryos exposed to both SAM and Tocopherol progressed further into development than the embryos exposed to solely oxidative stress. Higher concentrations of SAM and Tocopherol and exposure to different oxidative stress inducers could be tested in the future to further gauge the effectiveness of the two treatments. SAM and Tocopherol's success in combating oxidative stress inducers can have real-world implications to potential serve as alternatives to the traditional, exploited methods of oxidative stress reduction.

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