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NYSSEF Abstract

Project Title: Ecotoxicity of Bisphenol S Through Proteomic and Genomic Changes in C. elegans: Preliminary Findings

Bisphenol S (BPS), an industrial ingredient used in plastic synthesis, has been an increasingly common pollutant in the world. As it has now replaced the function of its close cousin Bisphenol A (BPA), its usage is only rising and has been detected at higher levels, especially within aquatic systems, as plastics and their industrial byproducts enter systems.

BPA has now been well-characterized as an endocrine disruptor and as such has casted doubt on BPS' safety. We determined how BPS affects *C. elegans*, specifically at mitochondrial and endocrine disruption in our model organisms. *C. elegans* has genes homologous to the human estrogen receptors ERα, suggesting that it is not unreasonable to predict BPS' effects on higher organisms by investigating its effects on the nematode. By performing a co-IP, targeting BPS and thereby any proteins the chemical is interacting with, we can figure out if BPS is somehow inhibiting the ERα receptors. We specifically looked to investigate mitochondrial disruption caused by BPS exposure through determining the ratio of NAD+/NADH, an important factor of the electron transport chain, ATP, the ETC's ultimate byproduct, and mutations within mitochondrial DNA. We screened a portion of the fifth subunit of the type I NADH dehydrogenase complex, which catalyzes the transfer of electrons from NADH to coenzyme Q10, and maintains the mitochondrial proton gradient, via amplifying worm DNA by PCR before sequencing. We concluded that acute BPS exposure induced mutations in C. *elegans*, noticing BPS-ERa receptor interactions and mtDNA damage, thereby disturbing the worm's metabolism as indicated ATP levels and a disruption of the normal ratio of NAD/NADH.