Role of NSY-1 and SKN-1 in Microplastic-Induced Reproductive Toxicity and Luteolin Protection in C. elegans

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Microplastic pollution is an emerging environmental health threat, with polystyrene microplastics detected in water, food, and even our blood. Their toxicity arises from leachable endocrine-disrupting additives such as bisphenol-like chemicals and phthalates. These additives can amplify oxidative stress, mitochondrial dysfunction, and inflammatory signaling. Despite growing links between environmental exposures and immune dysregulation, the genetic basis of individual susceptibility remains unclear.

Using Caenorhabditis elegans as a model organism, I can study whether variation in conserved stress-response pathways alters susceptibility to microplastic-induced reproductive toxicity and whether the dietary flavonoid luteolin can rescue this damage in a pathway-dependent manner. Three strains are compared: N2 wild-type, VC390 (nsy-1 loss-of-function mutant with impaired p38 MAPK stress sensing), and a skn-1 loss-of-function mutant with impaired antioxidant transcriptional execution. Each strain is exposed to polystyrene microplastics at 0, 50, and 100 mg/L, with and without 50 µM luteolin, for three days. Fecundity, quantified as total offspring per adult, serves as the primary endpoint. I hypothesize that luteolin will partially rescue microplastic-induced loss of fecundity. Specifically, I expect significant rescue in N2 and VC390 due to preserved SKN-1-mediated detoxification capacity, but minimal rescue in skn-1 mutants. This would identify SKN-1 as the critical pathway bottleneck and support a model in which genetic background determines both environmental vulnerability and dietary intervention efficacy.

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