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Olfactory Transcriptional Analysis of Salmon Exposed to Mixtures of Chlorpyrifos and Malathion Reveal Novel Molecular Pathways of Neurobehavioral Injury.

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Abstract

Pacific **salmon** exposed to sublethal concentrations of organophosphate pesticides (OP) have impaired olfactory function that can lead to loss of behaviors that are essential for survival. These exposures often involve mixtures and can occur at levels below those which inhibit acetylcholinesterase (AChE). In this study, juvenile Coho **salmon** were exposed for 24 h to either 0.1, 0.5, or 2.5 ppb chlorpyrifos (CPF), 2, 10, or 50 ppb malathion (MAL), or binary mixtures of 0.1 CPF:2 ppb MAL, 0.5 CPF:10 ppb MAL, or 2.5 CPF:10 ppb MAL to mimic single and binary environmental exposures. Microarray analysis of olfactory rosettes from pesticide-exposed **salmon** revealed differentially expressed genes involved in nervous system function and signaling, aryl hydrocarbon receptor signaling, xenobiotic metabolism, and mitochondrial dysfunction. Coho exposed to OP mixtures exhibited a more pronounced loss in detection of a predatory olfactory cue relative to those exposed to single compounds, whereas respirometry experiments demonstrated that exposure to OPs, individually and in mixtures, reduced maximum respiratory capacity of olfactory rosette mitochondria. The observed molecular, biochemical, and behavioral effects occurred largely in the absence of effects on brain AChE. In summary, our results provide new insights associated with the sublethal neurotoxic effects of OP mixtures relevant to environmental exposures involving molecular and cellular pathways of injury to the **salmon** olfactory system that underlie neurobehavioral injury.

KEYWORDS: chlorpyrifos; malathion; microarray analysis; mitochondria; olfaction; **salmon**

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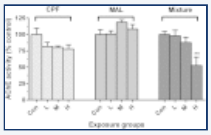

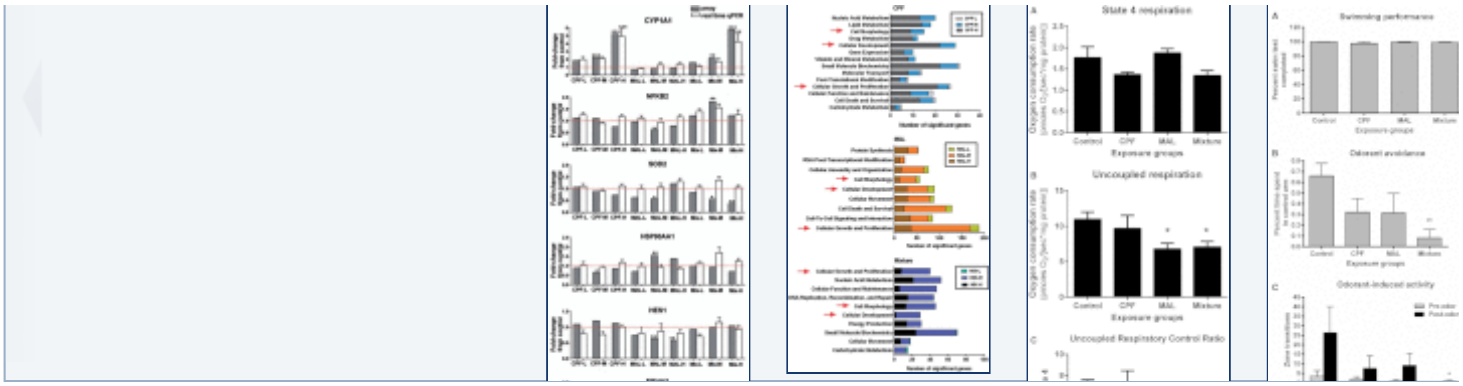

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