

## Integration of Fish Density Dependence and Concentration Response Models Provides a More Ecologically Relevant Assessment of Populations Exposed to Various Toxicants

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The assessment of toxic exposure on wildlife populations involves the integration of organism level effects measured in toxicity tests (e.g., chronic life cycle) and population models. These modeling exercises typically ignore density dependence, primarily because information on density dependence functional forms is lacking for most species. The interactions between population density and toxicant exposure can be synergistic or antagonistic, resulting in either under or overestimation of toxicant effects based on low density toxicity tests and density independent models. To explore the relationship between population density dependence and toxicant concentration, we simulated the exposure of a density dependent sheepshead minnow (*Cyprinodon variegatus*) population to five chemicals of various modes of action (estradiol, trenbolone, trifluralin, chlordane, pentachlorophenol). The functional form of density dependence was derived from laboratory density manipulation experiments that modeled the influence of adult density on juvenile survival and growth and adult survival and reproduction. Concentration-response models were developed from the chronic toxicity tests for all endpoints within each chemical where significant effects were measured. For each chemical, exposure was simulated across a wide range of densities and concentrations, applying density dependent mechanisms and concentration response curves at each density-concentration combination. The relationship between density and toxicant concentration for each chemical was modeled and described using population growth rate (PGR) contours. These contours identify compensatory and synergistic responses over a range of densities and concentrations for each chemical and demonstrate alterations to “carrying capacity” ( $PGR = 0$ ) relative to stress and density, which provides the boundary of a population’s tolerance to the model stressor. This work demonstrates how the integration of intraspecific interactions and organism toxicant response can provide a more ecologically relevant assessment of population response to toxicant exposure.