

Multigenerational and Transgenerational Effects of Environmentally Relevant Concentrations of Endocrine Disruptors in an Estuarine Fish Model

Bethany M. DeCourten^{1,2,3}, Joshua P. Forbes¹, Hunter K. Roark¹, Nathan P. Burns¹, Kaley M. Major², J. Wilson White², Jie Li³, Alvine C. Mehinto⁴, Richard E. Connon³, and Susanne M. Brander²

¹*University of North Carolina Wilmington, Wilmington, NC*

²*Oregon State University, Corvallis, OR*

³*CONACYT, Departamento de Recursos del Mar, Cinvestav Unidad Mérida, Mexico*

⁴*Southern California Coastal Water Research Project Authority, Costa Mesa, CA*

ABSTRACT

Many pollutants cause endocrine disruption in aquatic organisms. While studies of the direct effects of toxicants on exposed organisms are commonplace, little is known about the potential for toxicant exposures in a parental (F0) generation to affect unexposed F1 or F2 generations (multigenerational and transgenerational effects, respectively), particularly in estuarine fishes. To investigate this possibility, we exposed inland silversides (*Menidia beryllina*) to environmentally relevant (low ng/L) concentrations of ethinylestradiol, bifenthrin, trenbolone, and levonorgestrel from 8 hpf to 21 dph. We then measured development, immune response, reproduction, gene expression, and DNA methylation for two subsequent generations following the exposure. Larval exposure (F0) to each compound resulted in negative effects in the F0 and F1 generations, and for ethinylestradiol and levonorgestrel, the F2 also. The specific endpoints that were responsive to exposure in each generation varied, but included increased incidence of larval deformities, reduced larval growth and survival, impaired immune function, skewed sex ratios, ovarian atresia, reduced egg production, and altered gene expression. Additionally, exposed fish exhibited differences in DNA methylation in selected genes, across all three generations, indicating epigenetic transfer of effects. These findings suggest that assessments across multiple generations are key to determining the full magnitude of adverse effects from contaminant exposure in early life.

Due to distribution restrictions, the full-text version of this article is available by request only.

Please contact pubrequest@sccwrp.org to request a copy.