Presentation Type: **(**Tracking # ORD-008689)

Platform Preferred (Invited)

Track:

Aquatic Toxicology and Ecology

Session:

Adaptation to stressors: Evolutionary and molecular toxicology and their potential for informing ecological risk assessment

Abstract Title:

The genetic basis for evolved tolerance to dioxin-like compounds in wild Atlantic killifish: more than the aryl hydrocarbon receptor.

Authors:

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Abstract:

Populations of Atlantic killifish (*Fundulus heteroclitus*) resident to some US urban estuaries have independently evolved extreme and inherited tolerance to toxic dioxin-like compounds (DLCs). To further understand the genetic basis for this trait, we densely genotyped families of F2 recombinant embryos derived from tolerant killifish resident to the New Bedford (NB) PCB Superfund site (MA, USA). Quantitative Trait Locus (QTL) analysis identified main, interacting and epistatic genetic markers of NB tolerance that together provide an unusually comprehensive accounting of this complex trait: ≥ 68% phenotypic variance explained, LOD (Logarithm of Odds) 23.68; p = 8.33e-15. An integrated genetic linkage map for the species was constructed to characterize physical relationships among QTLs across the killifish genome. This map and recently identified killifish gene models (*Fundulus* genome consortium) provided invaluable tools to explore syntenic and orthologous

comparisons across fish and other species, and infer potential causal relationships among loci identified through QTL associations. Consistent with mechanistic knowledge of DLC toxicity in fish and other vertebrates, the aryl hydrocarbon receptor (AHR2) accounts for 16.77% of phenotypic variation; however, QTLs on an independent linkage group have even greater explanatory power (43.74%). The species-wide relevance of some of these NB QTLs was suggested through genetic screening of three other DLC-tolerant populations, resident to highly contaminated urban estuaries. These results advance our understanding of intra- and inter-specific variation in DLC toxicity, and permit insight into rapid evolutionary mechanisms.