Genetic accessions, treatment information, and methodology from laboratory experiments studying transcriptomic responses to saxitoxin in zebrafish (Danio rerio)

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Data Type: experimental
Version: 1
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Project
» WHCOHH - Cellular and Molecular Mechanisms Underlying Long-Term Effects of Early Life Exposure to HAB Toxins (WHCOHH HAB toxins)

Program
» Woods Hole Center for Oceans and Human Health (WHCOHH)

Contributors	Affiliation	Role
Aluru, Neelakanteswar	Woods Hole Oceanographic Institution (WHOI)	Principal Investigator
Hahn, Mark	Woods Hole Oceanographic Institution (WHOI)	Co-Principal Investigator
York, Amber D.	Woods Hole Oceanographic Institution (WHOI BCO-DMO)	BCO-DMO Data Manager

Abstract

This dataset contains genetic accessions, treatment information, and methodology from laboratory experiments involving developmental exposure to low-level saxitoxin and how it affects neuronal gene expression in zebrafish (Danio rerio).

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Coverage

Temporal Extent: 2022

Dataset Description

The data discussed in this publication have been deposited in NCBI's Gene Expression Omnibus (GEO) and are accessible through GEO Series accession number GSE204989 (https://www.ncbi.nlm.nih.gov/geo/query/acc.cgi?acc=GSE204989).

Sequence Read Archive (SRA) data, BioSamples, and GEO holdings can be accessed from the NCBI BioProject PRJNA843039 (http://www.ncbi.nlm.nih.gov/bioproject/PRJNA843039).

Methods & Sampling

Adult zebrafish (Tupfel-Long fin wild type strain) were raised under ambient conditions (temperature 28.5 degrees, photoperiod 10:14 light/dark) in Redfield zebrafish facility. Embryos used in the experiment were obtained by tank breeding of the adults. Embryos were injected with saxitoxin or vehicle control at 6 hours post-fertilization.

Zebrafish embryos were exposed to saxitoxin (STX; 24 or 48 pg) or vehicle (0.3 mM HCl) at 6 hours post fertilization (hpf) via microinjection. We examined transcriptional profiles in embryos at 24, 36 and 48 hpf using RNA sequencing.

Sampling and analytical procedures:
Total RNA was isolated from embryos at 3 developmental time points and RNAseq was carried out using Illumina platform.

Organism scientific name: Danio rerio
LSID (Life Sciences Identifier) = urn:lsid:marinespecies.org:taxname:1026595
NCBI:txid903980

Data Processing Description

RNAseq data was pre-processed and mapped to the zebrafish genome (GRCz10) using STAR aligner. HTseq-count was used to obtain read counts. Statistical analysis was done using edgeR.

Data Files

File
Treatment and NCBI accession information

filename: treatments_accessions.csv

(Comma Separated Values (.csv), 1.05 KB)
MD5:0de81a7c5514a6e42a314011f6f6d093

Treatment information, and NCBI accession identifiers for BioSamples, and holdings in the Sequence Read Archive (SRA).

Parameters:

Run,NCBI Sequence Read Archive (SRA) Run identifier

BioSample,NCBI BioSample identifier

Experiment,NCBI Sequence Read Archive (SRA) Experiment identifier

Sample_Name,Sample name

Treatment,Treatment type (Control or Saxitoxin)

Related Publications

Aluru Lab (2022). Developmental exposure to low-level saxitoxin affects neuronal gene expression in zebrafish (Danio rerio). 2022/05. NCBI:BioProject: PRJNA843039. Bethesda, MD: National Library of Medicine (US), National Center for Biotechnology Information; Available from: http://www.ncbi.nlm.nih.gov/bioproject/PRJNA843039.
IsRelatedTo

Parameters

Parameters for this dataset have not yet been identified

Instruments

Dataset-specific Instrument Name	Illumina HiSeq 2000
Generic Instrument Name	Automated DNA Sequencer
Generic Instrument Description	General term for a laboratory instrument used for deciphering the order of bases in a strand of DNA. Sanger sequencers detect fluorescence from different dyes that are used to identify the A, C, G, and T extension reactions. Contemporary or Pyrosequencer methods are based on detecting the activity of DNA polymerase (a DNA synthesizing enzyme) with another chemoluminescent enzyme. Essentially, the method allows sequencing of a single strand of DNA by synthesizing the complementary strand along it, one base pair at a time, and detecting which base was actually added at each step.

Project Information

WHCOHH - Cellular and Molecular Mechanisms Underlying Long-Term Effects of Early Life Exposure to HAB Toxins (WHCOHH HAB toxins)

The **overall objective** of the proposed research is to elucidate the cellular and molecular mechanisms by which early-life exposure to domoic acid (DA) and saxitoxin (STX)— HAB toxins with demonstrated human exposures—can cause neurobehavioral abnormalities later in life. Most HAB toxins are known to be acutely toxic, causing severe neurotoxicity soon after exposure to high doses. However, there is also extensive and possibly growing human exposure to HAB toxins at low levels that do not cause obvious acute signs of toxicity. Accumulating epidemiological and experimental evidence indicates that exposures during early life can have a profound effect on health later in life (the *developmental origins of adult health and disease* hypothesis). In rodents early life exposure to low levels of neuro-active chemicals, including some HAB toxins and chemical pollutants, can cause physiological abnormalities and behavioral defects such as altered cognitive function later in life. However, the mechanisms by which developmental exposure elicits effects in developing animals and later in life are not understood. DA and STX act on ionotropic glutamate receptors and voltage-gated sodium channels, respectively. Both of these protein families are expressed widely in the developing nervous system and critical to establishing proper neuronal function in the central nervous system. *The **central hypothesis** of the proposed research is that early life exposure to HAB toxins alters neurotransmitter receptors and ion channels changing prenatal programming of neurodevelopment, leading to altered gene expression and functional changes in neuronal and glial cells in the developing nervous system that ultimately contribute to altered neurobehavioral function in adults.* We will address this hypothesis using zebrafish, a powerful model organism for research on developmental mechanisms including those involved in neurodevelopment and developmental neurotoxicity. Our preliminary research has identified a novel mechanism by which early life exposure to DA disrupts neurodevelopment through effects on myelination. Additional studies demonstrated altered expression of genes involved in axonal extension in embryos exposed to low levels of STX. Linking these studies with past and future HAB occurrence and toxin exposure scenarios (Project 2) will provide an understanding of the long-term health consequences of developmental exposure to HAB toxins, critical for assessing public health risks associated with widespread exposure to these chemicals.

Program Information

Woods Hole Center for Oceans and Human Health (WHCOHH)

Website: https://www2.whoi.edu/site/whcohh/

Coverage: Western N. Atlantic, Arctic

NSF Award Abstract

The mission of the Woods Hole Center for Oceans and Human Health is to protect the public health through enhanced understanding of how oceanic and environmental processes including climatic variation affect the population dynamics of toxin producing organisms, and the risks from exposure to their potent neurotoxins. Factors affecting the distribution, survival, proliferation, and toxicity of harmful algal bloom (HAB) species still are poorly known, despite their enormous consequences for human health. Three research projects and two cores comprise the Center. The Center structure will facilitate the integration among projects, and the integration of research with education and community engagement activities. The Center will engage stakeholders, facilitate education on HAB science at many academic levels, and strengthen public knowledge about HAB blooms and their impacts. The Center is jointly supported by NSF and by the National Institute for Environmental Health Sciences (NIEHS).

The research activities of the Center will focus on two key HAB taxa: Alexandrium fundyense that produces the saxitoxins responsible for paralytic shellfish poisoning (PSP), and Pseudo-nitzschia spp. that produce domoic acid responsible for the amnesic shellfish poisoning (ASP) syndrome. Novel, targeted, efficient, and data-rich sampling approaches developed by the applicants and applied in situ have revealed that critical aspects of A. fundyense dynamics in natural settings differ dramatically from those inferred from laboratory studies, indicating plasticity in response to climate. The research proposed will build on these new and fundamental insights into what regulates blooms, and on the Center's established strengths in ocean observation technologies and modeling, to predict how environmental variables may influence population dynamics of known and emerging HAB threats. Hindcast simulations compared with climate data records in the Gulf of Maine will assess model performance and uncertainty. Forecasts run for a range of potential climate scenarios can help quantify future public health risks. Similarly, specific cells have been identified in the developing brain that are targets of HAB toxins, findings giving insights into developmental toxicological mechanisms. These will guide studies to address the scope of toxin effect in the developing central nervous system, potentially linking developmental exposures to adult consequences. Studies of new mechanisms of toxin action will include determination of the effects of combined or repeated exposure to sub-lethal levels of saxitoxin and domoic acid, and possible silent neurotoxicity, at different life stages in the zebrafish model.

This award reflects NSF's statutory mission and has been deemed worthy of support through evaluation using the Foundation's intellectual merit and broader impacts review criteria.

The data management plan for the program can be found here.

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Funding

Funding Source	Award
NSF Division of Ocean Sciences (NSF OCE)	OCE-1314642
National Institutes of Health (NIH)	NIH-P01ES021923

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