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Toxicological Analysis of Oxybenzone Effects

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Abstract

Oxybenzone is a new aquatic pollutant whose widespread use has made it easier to find in exposed biota's body burdens and surface waters. In this review, the sub-deadly poisonousness impacts of oxybenzone were learned at the earth pertinent groupings of 0.6 and 5.2 µg/L (counting 0.01% DMSO as the dissolvable control). The metabolic physiology of larval zebra fish (Danio rerio) after 7 days of exposure (from 2 to 9 days post fertilization) was investigated using an integrated in vivo and in silico experimental strategy. Whole-organ respirometry revealed a statistically significant twofold increase in metabolic rate for the 0.6 g/L treatment group; however, there were no significant differences observed between the solvent control and the oxybenzone treatment level of 5.2 g/L. Functional categories related to metabolism (metabolic enzymes) and biological regulation (genome regulation, cell signaling) were found to be highly deregulated in the analysis of whole-transcriptase RNA-sequencing data.

Keywords: Oxybenzone effects; Glycerol lipid metabolism; Toxicological analysis

Oxybenzone effects

In silico stoichiometric metabolic models were parameterized for each treatment group using the transcriptomics changes for metabolic enzyme genes. Glycerol lipid metabolism was found to be significantly impacted by low dose oxybenzone exposure in FBA simulations for each metabolic model. Further investigation of core metabolic pathways revealed elevated flux through triglyceride-producing esterification reactions in glycerol lipid metabolism [1].

The increased catalytic activities of the esterification reactions can be used to explain the elevated in vivo metabolic rate observed for 0.6 g/L oxybenzone. In addition, the disruption of glycerol lipid metabolism predicted by our in silico analysis is consistent with the findings of other authors' non-targeted metabolomics studies, which show that oxybenzone exposure affects glycerol phospholipid metabolism and causes dyslipidemia[2]. As a result, the framework for integrated toxicological analysis based on biological pathways is provided by our study's methods.

Oxybenzone or benzophenone-3 (BP-3) on embryo-larval zebra fish (Dania rerio). Specifically, we investigated how the metabolic physiology of embryo-larval zebrafish was affected by exposure to nominal concentrations of 1 or 10 g/L oxybenzone (including a solvent control of 0.01% dimethyl sulfide, or DMSO) for 7 days from 2 to 9 days post fertilization (dpf). The entirety of the metabolic pathways that influence organismal physiology (such as respiration or metabolic rate) is referred to as metabolic physiology. Oxybenzone is a prevalent and emerging pollutant that is frequently utilized as an organic UV-absorbing chemical in personal care products [3]. Its widespread use as photo stabilizers in plastics and cosmetics , which has contributed to its detection in fish tissues and surface waters as well as its release into the aquatic environment[4]. According to Burns et al., the typical level of oxybenzone found in the surface waters of inshore (freshwater) and near-shore coastal/estuarine systems is 5 g/L. 2021; Fent and others, 2010; ICRI, 2018).

Given such inescapable sea-going contamination with oxybenzone, there is extensive work to describe its consequences for piscine physiology. According to Bluthgen for instance, larval zebrafish exposure results in altered expressions of pollutant metabolism enzymes and endocrine genes, neurotoxicity (including behavioral effects), and toxicity effects on survival [5]. Non-targeted metabolomics studies of juvenile gilthead sea bream (Sparus aurata) exposed to oxybenzone have revealed widespread disruptions in lipid and amino acid metabolism [6]. However, these studies' lowest effect concentrations of oxybenzone ranged from 50 to 1000 g/L, well above the predicted no effect concentration of 18 g/L for fish toxicity. As a result, this study measured the mass-specific metabolic rate (or O2 consumption) of larval zebrafish using respirometry to test the sub-lethal effects of oxybenzone at levels that were within relative environmental relevance at 10 g/L. RNA-sequencing (RNA-seq) was also used to quantify changes across the transcriptase of an entire organism[7]. The benchmark dose (BMD) analysis platform, BMD Express (v2), was used to determine transcriptomics dose-responses, and the protein annotation through evolutionary relationship (PANTHER) database was used to map gene ontologies to biological functional categories. By incorporating experimental data on O2 consumption rate and changes in metabolic enzyme gene expression (as determined by RNA-seq) to constrain the minimum and/or maximum (min/max) catalytic bounds for specific metabolic reactions in an in silico stoichiometric model of zebrafish metabolism, deeper insights into the perturbations of metabolic pathways were sought. The metabolic capabilities of the solvent control or oxybenzone treatment group stoichiometric models were then characterized using a variety of constraints-based reconstruction and analysis (COBRA) methods We hypothesized that the disruptions of metabolic pathways that are responsible for maintaining organismal bioenergetics (i.e., oxidative metabolism, ATP synthesis, etc.) would result in the sub-lethal effects of oxybenzone exposure on metabolic rate[8]. As a result, the goal of our integrative strategy was to combine toxicological analyses performed in vivo and in silico to offer a mechanistic evaluation of the effects those changes in the underlying metabolic pathways have on the physiology of an organism.

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Water samples taken each day from each treatment beaker were used to measure the concentrations of oxybenzone in the exposure aquariums [9]. A 1 mL test was gathered from every measuring utensil, spiked with 10 μL of 1000 ng/mL d10-carbamazepine in methanol (Sigma-Aldrich, Cat# 1.06018) as interior norm (Sigma-Aldrich, Cat# C-094) (for a last grouping of 10 ng/mL). Prior to analytical analysis by means of liquid chromatography and tandem mass spectrometry (LC-MS/MS), a 200-milliliter sample was transferred to a glass insert and centrifuged at 2000 rcf for five minutes to pellet any debris. The LC-MS/MS framework included an Agilent (St Nick Clara, CA) 1260 super elite execution fluid chromatography (UHPLC) framework and a triple-quadrupole 6420 mass spectrometer (MS/MS) with an electrospray ionization (ESI) source [10]. The analytes, oxybenzone and d10-carbamazepine, were chromatographically separated onto an Agilent Poroshell EC-C18 column (3.0 50 mm, 2.7 m particle size), and all water samples were quantified against a 9-point standard curve ranging from 40 ng/mL to 0.156 ng/mL. Milli-Q water (A) and methanol (B), both of which contained 5 mm ammonium formate (Sigma-Aldrich, Cat#17843), made up the mobile phase [11].

Discussion

After remaining at 95% (B) for five minutes, the mobile phase gradient linearly decreased to 5% (B) for one minute and remained there for two more minutes. The total run time was 10 minutes, with a 2-minute pre-run duration at 95% (B). At a flow rate of 0.4 milliliters per minute, chromatographic separation was carried out. Both analytics were identified in sure electrospray ionization mode (ESI+) with a slim voltage of 3.5 kV, nitrogen desolation gas temperature of 350°C, and gas stream pace of 2 Liter/minute. With a collision energy of 20 V, multiple reaction monitoring (MRM) was used to monitor precursor > product ions with mass-to-charge (m/z) ratios of 229 > 151 for oxybenzone and 247.2 > 204.1 for d10-carbamazepine [12]. The retention time for d10-carbamazepine was 4.5 minutes, and it was 5.6 minutes for oxybenzone. Oxybenzone lowest limit of detection, which was determined to be 0.156 ng/mL, was used with a precision of 25% and accuracy of 70%.

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