Cyanobacteria can produce toxins that can have negative effects on aquatic organisms, wildlife, domestic animals and humans. Lakes and ponds in urban parks, due to their location and importance raise important attention. They are frequented by people and pets, and, from an urban ecology perspective, they are important biodiversity spots and climate regulators in large cities. However, along with the increasing temperatures driven by global changes, the fact that lakes are usually shallow, tend to have stagnant water and may receive input of urban effluents makes them prone to cyanobacterial blooms to proliferate. In this work, the effects of a cyanobacterial bloom, collected from an urban park in Aveiro (Portugal), on zebrafish embryos (*Danio rerio*) were assessed, evaluating toxicity in terms of mortality, hatching, embryo development, behavior endpoints (total distance, slow and rapid movements, peripheral distance and different angle classes) and biochemical effects (oxidative stress - Glutathione-S-transferase (GST), Catalase (CAT), Glutathione Peroxidase (GPx) and Glutathione Reductase (GR); neurological damage - Acetilcholinesterase (AChE) and indicator of tissue damage - Lactate dehydrogenase (LDH)) after five days of exposure to cyanobacteria extract. The results showed that the exposure affected development (hatching and delayed development) and caused neurotoxicity, translated into hypoactive behavior, increased thigmotaxis and inhibition of ChE in zebrafish embryos. This study showed that cyanobacteria blooms in urban parks may pose risks to wildlife, pets and people that must be evaluated.

Keywords: Cyanotoxins, climate change, anthropogenic pressures, neurotoxicity, urban ecology, *Danio rerio* embryos

4.14.P-Th459 Influence of Temperature on Acute and Chronic Toxicity of Marine Algal Toxins — A Case Study with Copepod Nitokra spinipes

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Harmful algal blooms (HABs) – proliferated algae densities with often a toxin producing ability – have been found increasingly in both northern and southern oceans. Recent studies have established that increasing temperatures contribute to HABs occurrence. But the broader influence of climate change on these outbreaks is less well quantified. Of particular concern is the limited research on HABs toxin effects under varying temperatures, especially concerning zooplankton, a crucial component of aquatic ecosystems. They do not only consume algae but also serve as prey for organisms at higher trophic levels, hence, are pivotal in energy transfer and nutrient cycles in aquatic food webs. Therefore, we examined the impact of marine toxins on marine zooplankton in the context of climate change. We designed a series of laboratory experiments using filtered seawater to assess the toxicity of four commonly occurring algal toxins, purified and sourced from CIFGA Laboratory, on a model organism for ecotoxicological studies, *Nitokra spinipes*, exposed to three different temperatures. We evaluated acute toxicity of domoic acid and yessotoxin, respectively. Adult females were exposed to these toxins at 15, 20, and 25°C for 48 hours. EC50 values of domoic acid arranged from 11.08±3.81 to 88.51±164.89 μg/L, respectively. Also, juveniles, aged 48 to 72 hours, were exposed at 18, 20, and 22°C for the same duration. The EC50 of domoic acid in this case arranged from 65.36±10.66 to 102.76±9.52 μg/L. Mortality rates across temperatures showed no significant difference. In chronic toxicity test, larval development ratio (LDR), brood size and inter-brood time of domoic acid, yessotoxin, saxitoxin, and microcystin-LR were examined at 18, 20, and 22°C. We observed that with increasing temperatures, LDR for domoic acid increased, whereas brood size significantly decreased as toxin concentration rose. While these results are preliminary, they indicate a temperature dependent sensitivity of copepods towards toxins produced by HABs.

4.14.P-Th460 Gene Expression Response in Marine Mussels Exposed to Toxic Cyanobacteria

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The increase in cyanobacterial blooms poses a challenge to environmental and human health due to their potential ability to produce cyanotoxins. Exposure to these cyanotoxins can affect different marine trophic levels, including marine bivalves which have been shown to accumulate microcystin (MC) and cylindrospermopsin (CYN), two of the main cyanotoxins in freshwaters. The impact of cyanobacteria and cyanotoxins on this organism is of great relevance given its worldwide distribution and the fact that mussels are consumed by both humans and animals. Hence, the main objective was to analyze the expression of genes involved in xenobiotic metabolism in marine bivalves (*Mytilus galloprovincialis*) after short exposure to different cyanobacteria strains. Animals treatment consisted in the exposure to MC-producing *Microcystis aeruginosa* cells (LEGE-CC 91094) or CYN-producing *Chrisosporum ovalisporum* cells (LEGE-CC X-001). Two different control groups were used, consisting of non-feeding animals and animals feeding with the green algae mixture *Tetraselmis* and *Isochrysis*. After treatment for 3 and 5 days, samples were taken from the gills and digestive glands of the mussels and the expression of following genes were analyzed by quantitative polymerase chain reaction (qPCR): four nuclear receptor 1 family J identified in Mediterranean mussels (NRj1jα, NRj1jβ, NRj1jγ and NRj1jδ), three cytochrome P450s genes (CYP3L1, CYP3L2 and CYP3L3) and a membrane transporter gene (ATP binding cassette subfamily B member 1, ABCB1). The expression of genes was different depending on the organ analyzed, the time of exposure to algae or algae intake and the type of cyanobacteria. Thus, *C. ovalisporum* caused a significant increase in the expression of NRj1jα, NRj1jβ, NRj1jγ, CYP3L1 and CYP3L3 genes in gills after 5-days of exposure in comparison to both control groups, while *M. aeruginosa* mainly produced a significant increase in ABCB1 expression in the digestive gland after both exposure periods. The results indicated that cyanobacteria may produce changes in the expression of genes involved in the xenobiotic pathways.