The influence of temperature on chemical toxicity in aquatic organisms

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Abstract. Water temperature is a key environmental factor modulating the toxicity of chemical contaminants in aquatic ecosystems. Aquatic organisms are simultaneously exposed to a wide range of pollutants and to increasing thermal stress driven by climate change, leading to complex interactions that alter toxicokinetic and toxicodynamic processes. As ectothermic organisms, fish and invertebrates exhibit temperature-dependent metabolic rates, which influence contaminant bioavailability, uptake, biotransformation, and elimination. Elevated temperatures can increase the solubility and mobility of many chemicals, enhance metabolic activation of xenobiotics, and exacerbate oxidative stress while impairing antioxidant and detoxification systems. These mechanisms often result in synergistic effects between thermal stress and chemical exposure, increasing toxicity beyond levels predicted under standard laboratory conditions. Consequences extend from molecular and physiological disturbances, such as altered enzyme activity and stress biomarker expression, to impaired development, reproduction, survival, and population stability. This paper highlights the critical role of temperature in shaping chemical toxicity in aquatic organisms and emphasizes the need to integrate realistic thermal scenarios into ecotoxicological testing and environmental risk assessment frameworks under ongoing climate change.
Key Words: aquatic organisms, chemical toxicity, climate change, ecotoxicology, temperature.

Introduction. Aquatic organisms are exposed to a wide range of chemical contaminants (heavy metals, pesticides, polycyclic aromatic hydrocarbons – PAHs, etc.), and the toxicity of these substances is not constant but can be strongly influenced by environmental factors, including water temperature. Temperature increases associated with climate change affect not only the physiology of organisms, but also the way they interact with environmental toxins (Noyes et al 2009).

In the current context of global climate change, aquatic ecosystems are exposed to multiple pressures, among which the increase in water temperature represents a major stress factor. Temperature directly influences the physiological processes of aquatic organisms, as well as the behavior and bioavailability of chemical contaminants present in the environment. This complex interaction between physical and chemical factors leads to changes in the toxicity of substances, increasing the vulnerability of aquatic fauna.

Aquatic organisms, being mostly ectothermic, cannot maintain a constant body temperature, which makes them dependent on water temperature for carrying out metabolic functions. In this context, thermal changes influence the absorption, biotransformation, as well as the physiological and ecotoxicological effects of toxic substances (Heugens et al 2001; Noyes et al 2009).

Climate change represents a significant threat to the integrity of aquatic ecosystems, especially through temperature changes occurring in freshwater and marine habitats. At the same time, human activity has led to the accumulation of a large number of chemical contaminants in aquatic environments. Critically, temperature acts as an interacting factor that can amplify or modulate the effects of these pollutants on organisms. Aquatic organisms, especially poikilothermic ones (with body temperature dependent on the environment), are particularly vulnerable to the synergistic effects of thermal stress and chemical toxicity (Noyes et al 2009). Understanding these interactions is essential for ecotoxicological predictions and for adapting conservation strategies.

Effects of temperature on the bioavailability of chemicals. Temperature influences the solubility, volatilization, and adsorption of toxic substances, thereby altering their bioavailability to aquatic organisms. For example, in the case of heavy metals, higher temperatures can increase their solubility and mobility in the water column, thus increasing the risk of uptake through fish gills or through the body surface of invertebrates (Heugens et al 2001). For instance, mercury becomes more bioavailable at higher temperatures, which intensifies its neurotoxic effects on fish (Boening 2000).

Temperature significantly influences the chemical behavior of contaminants in water, affecting essential properties such as:

Solubility and diffusivity. Increased temperature leads to higher solubility of many substances, especially heavy metals (e.g. copper, cadmium, mercury), thus increasing their bioaccessible concentration. At the same time, the diffusibility of contaminants in the water column increases, accelerating their transfer to biological compartments (Heugens et al 2001).

Volatilization and particulates. For semi-volatile organic contaminants (e.g. benzene, toluene, PCBs), higher temperature accelerates volatilization, reducing their concentration in water but increasing atmospheric emissions and inhalation risks for semi-aquatic organisms. On the other hand, for particle-bound substances (e.g. PAHs), temperature influences the desorption rate, increasing the potential for bioaccumulation.

Impact of temperature on xenobiotic metabolism. Aquatic organisms increase their metabolic rate as temperature rises, which can lead to:

- faster absorption of contaminants;
- more intense, but not necessarily more efficient, hepatic metabolism of xenobiotics;
- accumulation of toxic intermediate metabolites.

Detoxification enzymes, such as cytochrome P450, can be inhibited or overexpressed depending on the species and temperature level, which alters toxicokinetics (Madeira et al 2013).

One of the most important ways in which temperature influences chemical toxicity is by altering xenobiotic metabolism. Aquatic organisms have a complex set of enzymes involved in contaminant detoxification (e.g. the cytochrome P450 family – CYP), whose activity is regulated by temperature. Higher temperatures increase the overall metabolic rate, thereby enhancing the absorption of toxic substances. However, increased temperature can have a negative effect on phase I and II enzymes, disrupting normal biotransformation processes and promoting the accumulation of reactive metabolites.

Studies carried out on estuarine fish (*Dicentrarchus labrax*) have shown that high temperatures (>25°C) increase the expression of cytochrome P4501A, but reduce overall enzymatic activity due to oxidative stress (Madeira et al 2013).

Poikilothermic aquatic organisms regulate their metabolism according to environmental temperature. This physiological flexibility can become a vulnerability when facing chemical contaminants, which may interfere with metabolic processes.

Xenobiotic metabolism. Hepatic enzymes (e.g. cytochrome P450, glutathione S-transferase) are essential for the biotransformation of contaminants. At high temperatures, the activity of these enzymes can be:

- increased (promoting faster detoxification);
- inhibited (favoring the accumulation of reactive and toxic metabolites).

For example, studies on estuarine fish have shown that CYP1A activity (a marker of exposure to hydrocarbons) is sensitive to temperature variations, which can completely change the toxicity profile (Madeira et al 2013).

Oxygen consumption and oxidative stress. Higher temperatures lead to increased oxygen consumption, but at the same time oxygen solubility in water decreases. This creates an imbalance that may cause functional hypoxia and induce oxidative stress. Under the combined influence of contaminants and temperature, large amounts of reactive oxygen species (ROS) are produced, damaging DNA, membrane lipids, and structural proteins (Lushchak 2011).

Synergy between thermal stress and chemical stress. High temperature can enhance the toxicity of certain substances by:

- compromising the antioxidant system → increasing oxidative stress;
- reducing the efficiency of cellular repair mechanisms;
- depleting the energy reserves of organisms exposed simultaneously to heat and toxins.

This synergistic interaction between thermal stress and chemical exposure is well documented for organophosphate pesticides, whose lethal effects increase significantly at higher temperatures (Patra et al 2015).

Temperature acts as an amplifier of chemical stress, leading to synergistic rather than additive effects. The interaction mechanisms include:

- disruption of redox homeostasis, resulting in ROS accumulation;
- inhibition of antioxidant enzymes (SOD, CAT, GPx), reducing the ability to neutralize contaminant-induced oxidative stress;
- depletion of energy resources, limiting adaptive and survival responses.

For example, Patra et al (2015) showed that the toxicity of the insecticide diazinon was 2-3 times higher at 28°C than at 20°C in freshwater crustaceans.

Effects on development, **reproduction**, **and survival**. Temperature influences embryonic and larval development in aquatic organisms, and in the presence of contaminants, the risk of malformations, early mortality, and infertility increases.
Studies have shown, for instance, that fish larvae exposed to polychlorinated biphenyls (PCBs) have a higher rate of malformations at temperatures of 25–28°C compared to 18°C (Barron et al 2004).

Organisms exposed simultaneously to thermal stress and contaminants develop a wide range of physiological responses, including:

- suppression of the immune system, with reduced phagocytosis, antibody synthesis, and lysozyme activity;
- reproductive alterations, such as delayed sexual maturation, reduced gamete production, and impaired embryonic development;
- chronic oxidative stress, with cumulative effects on vital organs (liver, gills, kidneys).

These effects were observed in *Danio rerio* larvae exposed to mixtures of metals and pesticides at temperatures above 26°C, where significant increases in mortality and malformation rates were recorded (Barron et al 2004).

Molecular and physiological responses: **stress biomarkers**. Aquatic organisms respond to thermal and chemical stress through the activation of complex molecular cascades aimed at preventing or repairing biological damage. These responses can be measured using cellular stress biomarkers, which reflect physiological, biochemical, and genetic disturbances.

Heat shock proteins (HSPs). Heat shock proteins (HSPs) are molecular chaperones that help maintain protein structure or refold denatured proteins. Under combined thermal and chemical stress, the expression of HSP70 and HSP90 is increased, indicating severe cellular distress. For example, in fish of the genus *Oncorhynchus*, simultaneous exposure to high temperatures and pesticides (e.g. diazinon) led to overproduction of HSP70, suggesting enhanced defense against protein denaturation (Iwama et al 2004).

Oxidative stress and antioxidant enzymes*.* Genetic and epigenetic changes caused by oxidative stress are key effects of the interaction between contaminants and temperature. These changes are characterized by excessive ROS accumulation, which damages cellular macromolecules:

- superoxide dismutase (SOD): converts superoxide into hydrogen peroxide;
- catalase (CAT) and glutathione peroxidase (GPx): break down hydrogen peroxide into water;
- glutathione S-transferase (GST): involved in xenobiotic detoxification;
- imbalances between ROS production and antioxidant capacity lead to membrane lipid peroxidation, measured by malondialdehyde (MDA) levels. For example, in bivalve mollusks, high temperatures increased sensitivity to heavy metals (e.g. Cu, Cd), causing significant increases in MDA levels and SOD activity (Livingstone 2001).

Chronic exposure to combined stress may result in altered gene expression, disrupted cellular signaling pathways, or even mutagenesis. Involved elements include: stress response genes (e.g. hsp70, sod1, mt1); pro-inflammatory genes (e.g. il-1β, tnf-α); microRNAs and epigenetic modifications (DNA methylation), with long-term effects on organism adaptability.

These biomarkers are widely used to assess ecosystem health and are correlated with ecotoxicological risks (Van der Oost et al 2003).

Ecological implications and risk assessment relevance. In the context of climate change, ecotoxicological risk assessment must include variable temperature scenarios, as data obtained under standard conditions (20°C) may seriously underestimate real risks.
The impact of combined thermal and chemical stress goes beyond individual organisms, affecting higher levels of biological organization: populations, communities, and ecosystems. Therefore, modern ecotoxicology must integrate these interactions into ecological risk assessment models.

From molecular effects to population-level effects*.* Biological responses at the molecular level (e.g. oxidative stress, mitochondrial dysfunction) can lead to: reduced survival and fertility; delayed larval development or sexual maturation; decreased adaptive capacity to environmental conditions. These effects result in: reduced population density; disruptions in trophic structure (e.g. decline of top predators); selection of resistant individuals, often with energetic and genetic costs.

Limitations of traditional toxicity models*.* Standard toxicity tests (OECD, EPA) assess chemical toxicity under static and optimal conditions (usually 20-22°C), which underestimates real environmental risk. In nature, organisms are exposed simultaneously to: multiple pollutants (chemical mixtures); physical stressors (temperature, salinity, pH); conditions (daily and seasonal cycles, heat waves). Therefore, there is a need for: multifactorial testing that simulates realistic climatic conditions; application of the Adverse Outcome Pathway framework, linking molecular changes to organism- and ecosystem-level effects; integration of biomarkers into biomonitoring programs for early detection of ecological stress (Van der Oost et al 2003).

Adaptive strategies in ecotoxicological risk assessment*.* To better reflect the interaction between temperature and toxicity, the following approaches are recommended:

- updating toxicological reference values (NOEC, LC50) according to climate scenarios;
- toxicokinetic–toxicodynamic (TKTD) modeling, integrating time, dose, and biological response;
- climate mesocosms: outdoor experimental ecosystems simulating real conditions;
- omics techniques (transcriptomics, proteomics, metabolomics), providing a comprehensive view of stress-induced changes.

Conclusions. Temperature is a critical factor influencing how chemical substances affect aquatic organisms. Rising environmental temperatures can increase contaminant toxicity through physiological, biochemical, and ecological mechanisms. It is essential that environmental assessments and ecosystem protection policies incorporate climatic variables into risk models.

The influence of temperature on chemical toxicity in aquatic organisms represents a complex ecological interaction with major implications in the context of global climate change. Temperature increase does not act in isolation but strongly modulates contaminant behavior in the environment, their bioavailability, and the physiological and molecular responses of aquatic organisms.

On one hand, higher temperatures can increase the solubility and uptake rate of toxic substances; on the other hand, they impair detoxification enzyme function, induce oxidative stress, and exhaust energy and antioxidant capacities. These combined effects not only raise individual mortality risk but may also cause severe population- and ecosystem-level consequences, affecting reproduction, larval development, trophic structure, and biodiversity.

Molecular responses, such as increased expression of heat shock proteins (HSPs) or imbalances in antioxidant systems, provide sensitive indicators for ecological stress assessment. However, traditional toxicological testing models, conducted under static and standardized conditions, fail to accurately reflect real environmental scenarios, where organisms face multiple and variable stressors.

A redefinition of ecotoxicological paradigms is therefore essential, through the integration of multifactorial testing, application of Adverse Outcome Pathway (AOP) models, use of molecular biomarkers, and adaptation of toxicological values to realistic climate scenarios. Only in this way can robust ecological risk assessments and effective conservation strategies for aquatic ecosystems be achieved in an era of accelerated climate change and persistent chemical pollution.

Conflict of interest. The author declares that there is no conflict of interest.

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