

MHW impacts in productive boundary regions, such as the northeast Pacific, illustrate how biomass declines and rapid range shifts during heatwaves can be several times larger and faster than those driven by gradual warming alone. Such events impose “double strains” on fisheries management by overlaying acute shocks on chronic warming trends (Cheung and Frölicher, 2020). For aquaculture species, including large yellow croaker, extreme temperature episodes are becoming more frequent and intense, and temperature acts as the master abiotic factor controlling development and physiology across all life stages. Climate change-induced extremes drive changes in growth, metabolism, hemato-physiology and immune function, with impacts varying among species and depending on stress magnitude. Large yellow croaker farms in China’s main production areas are already close to or within risky temperature regimes during summer. Field data show maximum sea surface temperatures around 30.1 °C and rapid warming rates in summer, exposing cage-reared croaker to summer heatwaves (Wu et al., 2022). Although thermal safety margins relative to lethal limits appear currently positive, prolonged exposure to 30 °C induces energy redistribution, oxidative stress, and reduced body weight, indicating sublethal performance costs under chronic heat.

2.2 Physiological and cellular mechanisms of thermal stress

At the organismal level, temperature increases act as potent acute and chronic stressors for fish. Rapid warming elicits typical primary and secondary stress responses, including catecholamine and cortisol release, followed by rises in blood glucose and lactate, and changes in osmolality and hematological variables. These reactions are superimposed on elevated metabolic rate under chronic warming, potentially compromising the capacity to cope with additional stressors and leading to long-term impacts on fitness. Acute upper thermal limits are governed by direct thermal effects on reaction rates, protein structures and membrane fluidity, which then propagate through cellular and organ-level pathways. In fish, these mechanisms include mitochondrial dysfunction, oxygen limitation in some species, and impaired excitability of neural and muscular cells, leading to loss of equilibrium, failed homeostasis and ultimately heat death (Ern et al., 2023).

The limiting pathways vary among species, life stages and thermal histories, implying that a single universal failure mechanism is unlikely. At the cellular level, high temperature commonly induces oxidative stress, inflammation and apoptosis. In rohu carp, partial-lethal thermal exposure elevates antioxidant defenses (e.g. reduced glutathione, catalase, superoxide dismutase expression) and heat shock responses (hepatic hsp70), yet these responses are insufficient to prevent lipid peroxidation, DNA fragmentation, and pro-inflammatory activation, leading to hepatotoxicity and partial mortality. Similarly, in pufferfish, high temperature elevates serum enzymes, triggers oxidative damage, and upregulates pro-apoptotic genes (p53, caspase-9, caspase-3), indicating caspase-dependent and p53-mediated apoptosis under thermal stress. For large yellow croaker, prolonged exposure to upper-range temperatures (e.g. 30 °C) results in energy reallocation and oxidative adjustments. Experimental work shows that long-term high temperature reduces body weight and increases superoxide dismutase activity in gills, reflecting a trade-off between maintaining performance and mitigating oxidative stress (Wu et al., 2022). At the proteomic level, high-temperature stress in croaker liver alters thousands of proteins, with 442 differentially expressed proteins enriched in pathways related to translation, oxidative phosphorylation, ribosomes and lipid metabolism, indicating broad reprogramming of protein synthesis and energy use (Figure 1).

2.3 Overview of thermal stress response pathways in aquatic animals

Aquatic animals mount a conserved cellular stress response to temperature extremes that is centered on heat shock proteins (HSPs). In fish and shellfish, HSP families such as HSP70 and HSP90 act as molecular chaperones for protein folding, assembly, and protection against misfolding, and are upregulated not only by heat but also by hypoxia, toxins and infection. The HSP response contributes to thermotolerance and also modulates apoptosis, inflammation and immune function, linking environmental stress to disease resistance in finfish and shrimp (Jeyachandran et al., 2023). In teleost fish generally, HSP expression is induced in numerous tissues and cell types by both biotic (pathogens) and abiotic (heat, cold, contaminants) stressors, and is now recognized as a central component of the generalized stress response. In aquaculture, non-traumatic induction of HSPs (e.g. by mild heat or stimulants) is being explored as a strategy to enhance resilience, reduce handling trauma, and support vaccination and transport. For large yellow croaker specifically, multiple thermal stress pathways have been characterized.