

Tolerance limits nevertheless vary with life stage and endpoint. Sudden-change experiments in juveniles estimated incipient lethal limits at 7.06°C-26.54°C and 16.05-37.76 salinity, with highest short-term survival around 15°C-18°C and salinity 28-32, showing that survival tolerance is broader than the optimum for growth. Reproductive stages are more constrained: in Baltic turbot, fertilization success and viable hatch decline sharply below 7 psu, while egg survival is highest at 12°C-18°C and lower at 9°C and 21°C, indicating that successful reproduction near the species' brackish distribution limit depends on a narrower hydrographic window than juvenile persistence (Yang et al., 2020).

3 Key Environmental Temperature Effects on Health

3.1 Thermal stress and metabolic imbalance

Thermal stress rapidly disrupts energy metabolism in turbot and forces a shift toward compensatory catabolic pathways. In kidney tissue, exposure to 25°C-28°C increased cortisol, creatinine, *hsp70*, and *hsp90*, while also upregulating enzymes linked to aerobic metabolism and gluconeogenesis, including SDH, FBPase, MDH, cPEPCK, and G6Pase, indicating that maintenance of energy supply under heat load depends on metabolic reprogramming rather than simple metabolic depression (Yang et al., 2020). Consistent with this, integrated metabolome-transcriptome analysis showed that heat stress significantly affected pathways involved in steroid hormone biosynthesis, glycerophospholipid metabolism, sphingolipid metabolism, glycerolipid metabolism, and unsaturated fatty acid biosynthesis, suggesting that lipid homeostasis is also extensively remodeled during thermal challenge (Zhao et al., 2021).

At the whole-animal level, acute hyperthermal exposure at 27°C elevated serum cortisol, glucose, and respiratory frequency during the early stress phase, while hepatic glycogen, CAT, and GPx progressively declined and malondialdehyde increased, indicating rising oxidative cost and depletion of metabolic reserves as stress duration lengthened (Jia et al., 2020). Broader transcriptomic evidence supports this interpretation by showing that heat stress in turbot kidney enriches pathways related to fat metabolism, insulin signaling, apoptosis, FOXO, Jak-STAT, and P53 signaling, with hub genes such as AKT3, PIK3r2, and *mdm2* implicated in coordinating the balance between metabolic adjustment and cellular injury under elevated temperature.

3.2 Heat shock protein (HSP) expression and immune modulation

Heat shock proteins are among the most responsive molecular indicators of temperature stress in turbot, but their regulation is tissue-specific and linked to broader stress physiology. Under rapid cooling from 18°C to 1°C, red and white blood cell counts and hemoglobin fell significantly, while cortisol, cholesterol, and triglycerides increased; in parallel, *hsp70* and *hsp90* expression changed across tissues, and the authors identified 8°C-5°C as a critical low-temperature stress zone for aquaculture management. Under heat exposure, *hsp70* and *hsp90* transcripts in liver increased significantly within 6-12 h, accompanying early endocrine disturbance and the onset of hepatocyte apoptosis, which supports the view that HSP induction forms part of an acute protective response that is activated before prolonged thermal damage becomes established (Figure 1) (Jia et al., 2020).

Mechanistically, heat-induced HSP regulation in turbot is not merely descriptive but has defined signaling features. In turbot kidney cells, heat stress activated ERK1/2 and HSF1 and induced *hsp90* expression, while ERK inhibition attenuated this response, identifying an ERK-HSF1-dependent pathway in the cellular heat shock response (Yang et al., 2020). At the genome-wide level, 16 *hsp70* genes have been identified in turbot, and several members—especially *hspa1a*, *hspa1b*, and *hspa5*—show strong responses across heat, salinity, parasitic, bacterial, and viral stress datasets, indicating that the HSP network participates not only in thermal protection but also in immune modulation across diverse environmental and infectious challenges (Zheng et al., 2023).

3.3 Temperature-related disease susceptibility patterns

Temperature strongly shapes disease susceptibility in turbot by altering both host defense capacity and pathogen performance. In megalocytivirus challenge experiments, fish held at 14°C-18°C showed undetectable or moderate viral replication and no mortality, whereas fish held at 20°C-24°C showed robust viral replication and 100% mortality; furthermore, shifting temperature downward during infection improved survival, while upward shifts