5 Effects of Temperature on Survival and Physiological Stress

5.1 Patterns of survival rate and mortality risk

Elevated temperatures sharply increase mortality risk in abalone, especially when they approach or exceed upper tolerance limits. Continuous exposure of *Haliotis discus hannai* to temperatures above 26 °C reduced survival, increased falling rates, and produced abnormal foot structures, indicating impaired attachment and higher risk of mortality under chronic heat stress (Gao et al., 2024). Field-relevant thermal pollution from nuclear power plant discharge demonstrates that even a 2 °C rise above already warm ambient conditions can cause partial mortality and reduced adhesion in hybrid abalone, underscoring the narrow margin between stressful and lethal temperatures in summer (Barkan et al., 2025). Cold stress also elevates mortality risk and narrows the safe operating window for culture. In *Pacific abalone* exposed for 7 days, survival remained near 100% at 8 °C-10 °C but dropped to 25%-55% at 4 °C across salinities, accompanied by strong oxidative and cellular stress signals in hemolymph. Acclimation history modifies acute survival outcomes; juveniles pre-acclimated at 30 °C for 62 days showed higher survival than 10 °C-acclimated counterparts during a 31 °C heat challenge, highlighting the role of prior temperature exposure in shaping mortality risk under extreme events (Xu et al., 2020).

5.2 Mechanisms of heat stress and oxidative stress responses

At the cellular level, heat stress in abalone disrupts mitochondrial function and elevates oxidative load. Metabolomic analysis of *Haliotis discus hannai* juveniles showed that acute exposure to 31 °C after acclimation led to mitochondrial failure, incomplete oxidative metabolism of amino acids and fatty acids, and accumulation of unstable intermediates, particularly in cold-acclimated animals. In disk abalone, both elevated (25 °C) and depressed (15 °C) temperatures, especially when combined with low pH, increased H_2O_2, malondialdehyde, and antioxidant enzyme activities, demonstrating that departures from optimal temperature trigger oxidative stress and lipid peroxidation (Kim et al., 2023). Protective responses to heat stress are mediated by antioxidant systems and heat shock proteins (HSPs). Reviews and experimental work describe how failure to match increased metabolic demand at higher temperatures leads to excess reactive oxygen species, which are countered by antioxidases and antioxidants as a first defense, followed by induction of HSPs to refold or remove damaged proteins (Xu et al., 2020). Differential expression studies in *Pacific abalone* identify large HSP gene families, with many members up-regulated under heat and cold stress; HSPs in hemocytes in particular are highlighted as reliable markers of thermal condition (Kyeong et al., 2019).

5.3 Changes in immune function and stress resilience

Thermal stress reshapes abalone immune function, often in ways that increase susceptibility to pathogens. In farmed hybrid Greenlip × Blacklip abalone acutely heated from 16 °C to 26 °C and held for a week, antibacterial activity, phenoloxidase activity and neutral red retention times declined significantly and did not recover, while total haemocyte counts rose initially, indicating immunosuppression and persistent cellular stress. Chronic exposure of *Haliotis discus hannai* to sub-optimal temperatures (8 °C, 14 °C, 26 °C) for 30 days altered core immune metrics: low temperature increased haemocyte counts and reactive oxygen species, while both low and high temperatures reduced phagocytic capacity and modulated expression of protease inhibitor genes, especially when combined with bacterial challenge. Temperature also mediates resilience by shaping host-pathogen interactions at the molecular level and biasing immune resource allocation. In *Pacific abalone* hemocytes co-cultured with *Vibrio harveyi*, exposure at 25 °C (vs. 20 °C) caused stronger pro-inflammatory and apoptotic transcriptional responses, with up-regulation of caspase-3 and caspase-7, alongside higher expression of multiple virulence genes in the bacteria, demonstrating that warming simultaneously stresses host cells and enhances pathogen aggressiveness (Lee et al., 2023). Field and laboratory work on *Haliotis* rubra similarly shows that while some immune parameters (e.g., antiviral activity) increase with elevated temperature, prolonged warming depresses antibacterial activity and reveals a negative correlation between antiviral and antibacterial responses, suggesting trade-offs that may leave abalone more vulnerable to bacterial disease under future warming scenarios.