

further suggests that when aerobic metabolism becomes constrained, glycogen and lipids are mobilized, fatty acid and amino acid catabolism increase, and anaerobic metabolism becomes more prominent, which is consistent with the metabolic compensation expected under severe heat load (Jiang et al., 2024).

3.2 Oxidative stress and immune function responses

High temperature induces oxidative stress in swimming crab, and the initial physiological response is the activation of antioxidant and non-specific immune defenses. In embryos, increasing temperature caused superoxide dismutase, glutathione peroxidase, total antioxidant capacity, and malondialdehyde to rise first and then fall, with the strongest antioxidant response occurring at 31°C, indicating that protective systems are activated before being impaired at more severe temperatures (He et al., 2022). In adults exposed acutely to 34°C, hemocyanin, POD, CAT, and ACP increased transiently, whereas SOD and AKP declined early, showing that rapid warming can quickly disturb free-radical balance and alter immune enzyme activity.

When heat stress is prolonged or intensified, protective capacity appears to become insufficient, increasing the risk of cellular injury and immune dysfunction. Although direct long-term heat studies in swimming crab remain limited, work on this species under other environmental stressors shows a common trajectory in which antioxidant defense, heat shock response, and other cellular stress pathways are activated initially but can later be overwhelmed, leading to oxidative damage and apoptosis (Jiang et al., 2024). Related crab studies under heat stress support this interpretation: in Chinese mitten crab, ACP and AKP increased during early exposure but fell below control levels later, while total antioxidant capacity declined after 48 h, suggesting that sustained high temperature can convert an adaptive response into physiological exhaustion (Li et al., 2022).

3.3 Gut health and microbial communities

High temperature likely impairs gut health in swimming crab by weakening digestive performance, disturbing barrier function, and altering microbial community structure. In swimming crab embryos and newly hatched larvae, temperatures above 31°C reduced hatching success and were associated with depressed digestive enzyme activities, especially trypsin and cellulase at 33°C, suggesting that excessive warming compromises later digestive capacity and early-life intestinal function (He et al., 2022). Complementary evidence from ocean acidification experiments in *P. trituberculatus* shows that changes in gut bacteria were closely linked with digestion, stress response, immunity, metabolism, survival, and growth, indicating that gut microbial balance is a central component of physiological resilience in this species (Lin et al., 2020).

Evidence from crab heat-stress models further suggests that thermal stress drives intestinal dysbiosis toward a less favorable microbial profile. In Chinese mitten crab, acute heat stress altered intestinal microbial composition, decreasing beneficial taxa such as *Candidatus*, *Hepatoplasma*, and *Marinifilum* while increasing potentially pathogenic bacteria including *Rhodococcus* and *Morganella* (Li et al., 2022). In swimming crab, dietary lauric acid improved peritrophic membrane thickness, upregulated barrier-related factors, increased beneficial taxa such as *Actinobacteria* and *Rhodobacteraceae*, and reduced *Vibrio*, which indirectly supports the view that maintaining intestinal structure and microbiota stability is an important route for mitigating heat-associated physiological damage (Zhan et al., 2024).

4 Major Health Problems and Disease Risks Under High Temperature

4.1 High-temperature-related stress syndromes

High temperature is a primary environmental stressor for *Portunus trituberculatus* because, as a poikilothermic crustacean, its survival and growth are tightly constrained by water temperature (Qian et al., 2024). Acute heat exposure rapidly disturbs physiological homeostasis, causing metabolic maladjustment of free radicals and marked changes in non-specific immune indices such as hemocyanin, SOD, POD, CAT, ACP, and AKP activities.

At the mechanistic level, thermal stress in swimming crab is not only a passive injury process but also a regulated sensory and cellular response. Temperature-responsive TRP channels are broadly activated under temperature challenge, indicating that crabs possess a molecular system for detecting abrupt thermal fluctuations (Qian et al.,