

ALP also rose transiently after transfer and recovered within 24 h, which supports the view that endocrine and biochemical responses are driven more by the timing of challenge than by sustained blood failure.

Although detailed hematology in yellowfin seabream remains limited, studies in related and comparative fishes show a consistent pattern of salinity-induced changes in circulating cells, proteins, and ions. In black sea bream, serum ion concentration and osmotic pressure were actively adjusted within 6-12 h under acute low salinity, showing that blood homeostasis is maintained through rapid regulation rather than passively preserved. In common carp exposed to hypersalinity, total protein, albumin, and globulins decreased while blood glucose, cortisol, and sodium increased, indicating combined osmotic, metabolic, and endocrine strain (Ahmed et al., 2023). A freshwater catfish model showed an even clearer hematological cost, with higher white blood cells but lower red blood cells, hemoglobin, and packed cell volume under salinity exposure, which implies that blood indices are sensitive markers of osmotic challenge severity (Okomoda et al., 2024).

4.2 Effects of salinity variation on antioxidant systems and immune function

Salinity variation strongly affects antioxidant defenses and immune function in yellowfin seabream, but the response depends on tissue and salinity level. Under hypoosmotic stress, the liver showed the most severe oxidative disturbance in *A. latus*, as reflected by fluctuations in SOD and CAT activities together with changes in MDA content. Intestinal physiology was also clearly affected: freshwater exposure significantly inhibited Na^+/K^+ -ATPase activity and challenged the antioxidant defense system, indicating that reduced salinity can impair both ion regulation and redox stability in the gut. At the transcriptional level, hypoosmotic stress altered intestinal expression of genes linked to pathogen recognition, antimicrobial activity, pro-inflammatory cytokines, apoptosis, and antioxidant defense, showing that oxidative and immune responses are tightly coupled in this species.

The immune consequences of salinity stress extend beyond enzyme activity to broader host-microbe and tissue-level regulation. In yellowfin seabream intestine, brackish conditions favored *Lactobacillus* and *Pseudomonas*, whereas freshwater increased potentially pathogenic *Vibrio* and *Aeromonas*, suggesting that low salinity can shift the microbiota toward a less favorable immune context. A newer study likewise found that 30 ppt water enriched *Vibrio* and activated immune and redox-homeostasis genes, while 10 ppt favored *Cetobacterium* and metabolism-related genes, indicating that different salinity regimes bias the fish toward either defense-oriented or metabolic states (Peng et al., 2025). Evidence from black seabream supports the same time-dependent pattern: acute low salinity increased reactive oxygen species, MDA, and total antioxidant capacity early in exposure, then declined as adaptation progressed, showing that oxidative stress is often strongest in the initial adjustment phase (Figure 2).

4.3 Effects of salinity variation on tissue structure and cellular functions

Salinity variation affects tissue structure and cellular function in yellowfin seabream most clearly in organs directly involved in osmoregulation. In *A. latus*, the gill was the most sensitive tissue under hypoosmotic stress and responded by increasing Na^+/K^+ -ATPase activity to reduce Na^+ and K^+ loss, indicating that branchial ion transport is a primary compensatory mechanism. The intestine also showed functional disruption under freshwater exposure, with depressed Na^+/K^+ -ATPase activity and coordinated shifts in microbes, gene expression, and physiological traits, which indicates that salinity stress modifies epithelial function at both cellular and community levels. These findings fit the general teleost model in which gill, intestine, kidney, and other epithelia dynamically switch between salt absorption and salt secretion strategies as salinity changes.

Comparative structural studies show that salinity stress can progress from functional adjustment to overt tissue remodeling when exposure is stronger or more prolonged. In black sea bream, low salinity reduced the number and volume of gill chloride-secreting cells and was accompanied by more developed glomeruli and wider renal tubules, consistent with reduced salt secretion and enhanced ion reabsorption. Across fishes more broadly, salinity challenge can induce gill and liver damage, alter proteins involved in ion transport, metabolism, immunity, and apoptosis, and make gill and intestine the earliest and most responsive organs to structural stress (Su et al., 2025). Acute and chronic salinity experiments in other species similarly show dose- and time-dependent lamellar fusion, intestinal mucosal change, renal stress, and hepatocellular swelling, reinforcing that cellular adaptation has clear structural limits when