

Evidence from related euryhaline fishes helps clarify which genes are likely central to this regulation. In hybrid tilapia gills, salinity stress differentially regulated canonical osmoregulatory genes including CA, AQP1, SLC4A4/NBC1, CLCN2, and SLC12A2/NKCC1, indicating that ion exchange, bicarbonate transport, and water permeability are coordinated at the transcriptional level. In black porgy, another *Acanthopagrus* species, acute salinity transfer increased gill NKA transcripts under both freshwater and seawater challenge, while prolactin receptor expression rose specifically during freshwater adaptation, showing that branchial ion transport is tightly linked to endocrine signaling. Dynamic salinity regimes also appear to rely on especially labile *cfr* and *aqp3* regulation, as shown in tidally reared Mozambique tilapia, suggesting that fluctuating estuarine conditions may favor flexible local transcript control rather than fixed expression states (Seale, 2022).

5.2 Energy metabolism and stress response mechanisms

Salinity adaptation in yellowfin seabream is not only an ion-regulatory process but also an energy-allocation problem, because osmoregulation requires sustained metabolic support. Comparative work in teleosts shows that salinity stress consistently reorganizes metabolites linked to energy production and osmolyte synthesis, indicating that metabolic reprogramming is a basic requirement for osmotic compensation (Mkulo et al., 2025). In yellowfin seabream, hypoosmotic stress caused the liver to experience the strongest oxidative disturbance among examined tissues, with changes in SOD, CAT, and MDA indicating that the metabolic cost of acclimation is closely tied to redox imbalance. This means that salinity-induced health effects are shaped not only by whether fish can regulate ions, but by whether they can supply ATP and limit oxidative damage during that regulation.

Transcriptomic studies in other fishes provide a mechanistic framework for these changes. In spotted sea bass liver, salinity-responsive genes clustered in categories including energy metabolism, ion transport, signal transduction, immune response, and structural reorganization, and the liver was identified as a major source of carbohydrate metabolites for osmoregulatory organs. In spotted seabass exposed to salinity gradients, Na^+/K^+ -ATPase and MDA both rose and then fell over time, while most differentially expressed genes were enriched in energy metabolism, osmoregulation, signal transduction, and immune response, indicating a coordinated time-dependent stress program rather than a single static response (Hu et al., 2024). More detailed multi-omics work in *Pseudobagrus ussuriensis* further showed enrichment of glycolysis, oxidative phosphorylation, HIF-1, TNF, and Jak-STAT pathways under salinity stress, supporting the idea that metabolic reprogramming and stress signaling jointly determine whether acclimation remains adaptive or progresses toward injury (Liu et al., 2025).

5.3 Immune regulation and changes in disease susceptibility

Salinity variation also reshapes immune regulation in yellowfin seabream, and current evidence suggests that this is a central part of its health response rather than a secondary by-product. Under hypoosmotic stress, transcriptomic analysis showed that immune-related differentially expressed genes were widespread across tissues and that T cell-mediated immunity pathways were important in the response of *A. latus*. Intestinal analysis under freshwater exposure likewise revealed differential expression of genes involved in pathogen recognition, antimicrobial function, pro-inflammatory cytokines, apoptosis, and antioxidant defense, indicating that reduced salinity can simultaneously alter barrier defense and inflammatory signaling (Su et al., 2020). These results suggest that salinity stress changes how yellowfin seabream allocates resources between osmoregulation and host defense, which can influence disease vulnerability.

Changes in disease susceptibility are also likely mediated through microbiota and immune trade-offs. In yellowfin seabream intestine, brackish water favored *Lactobacillus* and *Pseudomonas*, whereas freshwater increased potentially pathogenic *Vibrio* and *Aeromonas*, implying that low salinity can shift the microbial environment toward higher infection risk. Comparative studies support this interpretation: in black-chinned tilapia from hypersaline habitats, genes linked to the immune system and heat-shock response were strongly downregulated, suggesting an energetic trade-off between acclimation and immune protection (Blondeau-Bidet et al., 2024). Similar links between low-salinity adaptation, energy metabolism, and disease resistance were observed in *Litopenaeus vannamei*, where the low-salt-tolerant family showed stronger immunity and higher resistance to *Vibrio parahaemolyticus*, supported