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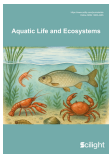

Review

Physiological Adaptations and Stress Responses of Juvenile Yellowfin Tuna (*Thunnus albacares*) in Aquaculture: An Integrative Review

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Abstract: Yellowfin tuna (*Thunnus albacares*) is a fast-growing, economically valuable pelagic species with increasing potential for aquaculture. However, its high metabolic demands and sensitivity to environmental fluctuations pose significant challenges for closed-cycle cultivation. This review synthesizes a series of recent studies conducted by our research team on the physiological responses and of juvenile yellowfin tuna under key aquaculture-relevant stressors, including thermal fluctuation, hyposalinity, ammonia nitrogen accumulation, and seawater acidification. We detail the mechanisms of oxidative stress, immunometabolic dysfunction, and osmoregulatory disruption across stress conditions, highlighting tissue-specific antioxidant responses, enzyme activity modulation, and microbiota remodeling. Collectively, these physiological responses underscore both the adaptability and vulnerability of yellowfin tuna juveniles in dynamic aquaculture environments. The insights gained from this integrative assessment provide a scientific foundation for optimizing environmental management, nutritional strategies, and system design in yellowfin tuna aquaculture. By aligning culture conditions with the physiological capacities of the species, these findings contribute to improving health, performance, and the long-term sustainability of yellowfin tuna farming operations.

Keywords: yellowfin tuna; environmental stressors; temperature; low salinity; ammonia toxicity; pH; size-dependent adaptation

1. Introduction

Yellowfin tuna (*Thunnus albacares*), a highly migratory pelagic species of significant economic value, has attracted increasing attention in aquaculture research due to its rapid growth rate, coupled with a metabolically active physiology and heightened responsiveness to environmental fluctuations, which render it both a promising candidate and a physiologically informative model for aquaculture research [1–4]. Compared with other commercially important tuna species, such as Atlantic bluefin tuna (*Thunnus thynnus*) and bigeye tuna (*Thunnus obesus*), yellowfin tuna was found to have a broader tropical and subtropical distribution, earlier maturation age,

and faster juvenile growth, making it particularly well-suited for aquaculture production [5–7]. These attributes, together with its ecological role as an apex predator and its high market demand, position it as both a promising aquaculture species and a physiologically informative model for studying pelagic fish biology [8–10]. Despite notable progress in broodstock management and larval rearing for various tuna species, the stable cultivation of yellowfin tuna—particularly through the juvenile stage—remains technically challenging [11–15]. Juvenile fish represent a critical developmental stage where rapid somatic growth is accompanied by high metabolic rates, active foraging behavior, and heightened susceptibility to environmental stressors [16–18]. Understanding their physiological responses is therefore essential for improving survival, performance, and welfare under farming conditions.

In recent years, our research group has conducted a series of systematic investigations to elucidate the physiological responses of juvenile yellowfin tuna to key environmental stressors—namely thermal fluctuation, salinity shifts, ammonia nitrogen accumulation, and seawater acidification—with a focus on oxidative stress, immune modulation, metabolic regulation, and behavioral adaptation. To better understand the species' physiological responses and support aquaculture development, we have synthesized recent research progress in this review.

This review consolidates our recent findings and provides an integrated perspective on the multifaceted physiological responses of yellowfin tuna juveniles under aquaculture-relevant stress conditions (Table 1). Specifically, we examine redox homeostasis and immunometabolic dysregulation under thermal extremes; antioxidant and osmoregulatory responses to acute hyposalinity; ammonia-induced oxidative damage, immune perturbation, and tissue degeneration; and the disruption of metabolic and immune function under seawater acidification, all of which have direct implications for husbandry practices in land-based and offshore aquaculture systems.

The overarching aim of this review is to provide a scientific foundation for the refinement of yellowfin tuna aquaculture. By elucidating the physiological mechanisms underpinning stress resilience and vulnerability in juvenile yellowfin tuna, we offer practical insights into environmental control, nutritional management, and system design. These findings serve as a critical reference for optimizing fish health, performance, and welfare, thereby supporting the sustainable and scalable development of yellowfin tuna aquaculture.

Table 1. Summary of research in juvenile yellowfin tuna (*Thunnus albacares*) under acute environmental stressors.

Environmental Factor/Topic	Species (Length/Weight)	Experimental Conditions/Stress Level	Exposure Duration	Sampled Tissues/Measured Parameters	Key Conclusion	Reference
Temperature	30.26 ± 2.79 cm; 384.88 ± 58.04 g	Control 30 °C vs. Low 24 °C vs. Ultra-low 18 °C	0, 12, 24, 36 h	Gut contents: 16S rDNA; α-diversity (Ace, Shannon); OTUs; composition (phylum/genus)	Cold stress reconfigures tuna gut microbiota: Verrucomicrobiota/Firmicutes/Bacteroidota ↑; Proteobacteria/Vibrio ↓ → metabolic adaptation.	Huang et al. (2025) [19]
Temperature	28.03 ± 1.78 cm; 503.23 ± 36.78 g	Control 28 °C vs. High temp 34 °C	0, 6, 24, 48 h	Serum: K^+, Na^+, Cl^-, cortisol, TG, CHO, ALP, C3/C4; Gill, liver, red/white muscle: SOD, MDA; Liver: ALP	Acute heat stress disrupts homeostasis and drives oxidative damage in juvenile yellowfin tuna, with gills most sensitive.	Liu et al. (2022) [20]
Temperature	29.97 ± 0.67 cm; 487.28 ± 29.26 g	Control 28 °C vs. High temp 34 °C	0, 6, 12, 24, 48 h	Stomach: pepsin; Pyloric caeca: trypsin; Intestinal tract: amylase, lipase; Liver: ACP, LDH, ALT, AST; Red and white muscle energy reserves	Juvenile yellowfin tuna restore hepatic energy balance within 24 h of acute heat stress, indicating short-term thermal resilience.	Liu et al. (2023) [21]
Salinity	38.39 ± 1.26 cm; 683.38 ± 39.05 g	Control 32 ‰ vs. Low salinity 26 ‰	0, 6, 12, 24, 48 h	Stomach: pepsin, α-amylase, lipase, chymotrypsin; Pyloric caeca: trypsin, α-amylase,lipase, chymotrypsin; Foregut: α-amylase, lipase, chymotrypsin, Histology;	Within 48 h at ~29‰, juveniles restore digestive enzyme activity—pyloric ceca are the most sensitive to low-salinity stress.	Zhang et al. (2023) [22]
Salinity	38.39 ± 1.26 cm; 683.38 ± 39.05 g	Control 32 ‰ vs. Low salinity 26 ‰	0, 6, 12, 24, 48 h	Gills, liver, red and white muscle: SOD, GPx, MDA	Juvenile yellowfin tuna rapidly adapt to ~29‰ low salinity, restoring liver, gill, and muscle antioxidant status to baseline within 48 h.	Zhou et al. (2023) [23]
Salinity	38.39 ± 1.26 cm; 683.38 ± 39.05 g	Control 32 ‰ vs. Low salinity 26 ‰	0, 6, 12, 24, 48 h	Serum: osmotic pressure, prolactin, lactic acid, glucose, K^+, Cl^-, Na^+, Ca^{2+}, H^+, HCO_3^-; Gill and trunk kidney: Ca^{2+} Mg^{2+}-ATPase, H^+/K^+-ATPase, Na^+/K^+-ATPase (activity & gene expression)	Under acute hyposalinity, juvenile yellowfin tuna restore ion–osmotic balance via prolactin-mediated ATPase responses in gill, trunk kidney, and anterior intestine.	Zhang et al. (2023) [24]
Ammonia nitrogen	38.39 ± 1.26 cm; 683.38 ± 39.05 g	Control 0 mg L^{-1} vs. High ammonia 5 and 10 mg L^{-1}	0, 6, 12, 24, 48 h	Trunk kidney: MDA, SOD, CAT, GPx, AKP, ACP; Head kidney: SOD2, CAT, GPX1b, IL-10, IL-6r expression level	Acute ammonia triggers oxidative–inflammatory stress—trunk kidney antioxidants ↑, phosphatases ↓; head-kidney antioxidant/immune genes ↓—supporting strict ammonia limits.	Sun et al. (2024a) [25]

Table 1. *Cont.*

Environmental Factor/Topic	Species (Length/Weight)	Experimental Conditions/Stress Level	Exposure Duration	Sampled Tissues/Measured Parameters	Key Conclusion	Reference
Ammonia nitrogen	38.39 ± 1.26 cm; 683.38 ± 39.05 g	Control: 0 mg L^{-1} TAN; Stress: 10 mg L^{-1}	0, 6, 12, 24, 48 h	Liver: MDA, SOD, POD, CAT, GPx, AKP, ACP, Na^+/K^+-ATPase, $Ca2^+$ Mg^{2+}-ATPase, AST, ALT; Liver: SOD2, CAT, GPX1b, IL-10, IL-6r, atp1b1a expression level	Ammonia ≤ 5 mg/L (≤36 h) or ≤10 mg/L (≤24 h) preserves juvenile yellowfin tuna liver function.	Sun et al., (2024b) [26]
Ammonia nitrogen	38.39 ± 1.26 cm; 683.38 ± 39.05 g	Control 0 mg L^{-1} vs. High ammonia 5 and 10 mg L^{-1}	0, 6, 12, 24, 48 h	Serum: LDL-C, HDL-C, T-CHO, TG, C3, C4, AKP, ACP, BUN, CRE; Spleen: SOD2, CAT, GPX1b, IL-10, IL-6r, TNF-a, TNF-b, casp2, casp9 expression level	NH_3–N ≤ 5 mg/L (≤36 h) or 5–10 mg/L (≤24 h) avoids adverse serum/splenic responses in juvenile yellowfin tuna.	Sun et al. (2024c) [24]
Ammonia nitrogen	38.39 ± 1.26 cm; 683.38 ± 39.05 g	Control 0 mg L^{-1} vs. High ammonia 5 and 10 mg L^{-1}	0, 6, 12, 24, 48 h	Gills, red muscle, white muscle: SOD, CAT, GPx, MDA; Gill histology	NH_3–N induces gill injury and rising oxidative stress (red muscle more resilient than white); limit exposure to ≤5 mg/L for ≤36 h or 5–10 mg/L for ≤24 h.	Sun et al. (2024d) [27]
Ammonia nitrogen	38.39 ± 1.26 cm; 683.38 ± 39.05 g	Control 0 mg L^{-1} vs. High ammonia 5 and 10 mg L^{-1}	0, 6, 12, 24, 48 h	Foregut: AMS, LPS; SOD2, CAT, GPX1b, IL-6r, IL-10 expression level; Liver: AMS, LPS; Pyloric cecum: trypsin	NH_3–N ≤ 5 mg/L (≤36 h) enhances digestibility, whereas 5–10 mg/L should be limited to ≤ 24 h to avoid inhibition; the liver is less tolerant than the foregut.	Sun et al. (2025) [28]
Acidification	18.21 ± 1.09 cm; 307.49 ± 49.38 g	Control: pH 8.10; Stress: pH 6.6, 7.1, and 7.6	0, 6, 12, 24, 48 h	Liver: GR, T-AOC, LPO, LDH, HK, PK, Na^+/K^+-ATPase, Ca^{2+} Mg^{2+}-ATPase; Serum: GLU, LDL-C, HDL-C, TGs, TCH, GOT, GPT, AKP; Hepatic histology	Acute acidification (\~pH 6.6) depresses antioxidant/metabolic function, induces hepatic degeneration, and shifts metabolism toward glucose (lipids largely stable).	Wang et al. (2024) [29]
Acidification	18.21 ± 1.09 cm; 307.49 ± 49.38 g	Control: pH 8.10; Stress: pH 6.6, 7.1, and 7.6	0, 6, 12, 24, 48 h	Skin, red muscle, gill, liver: MDA, SOD, CAT, GSH-Px, POD, ACP, AKP, LZM.	48-h acidification: \~pH 7.1 threshold—regulated ≥ 7.1, dysregulated <7.1; liver/gill most responsive; antioxidants peak at 6.6.	Wang et al. (2024) [30]

2. Physiological Responses of Juvenile Yellowfin Tuna to Thermal Stress

Temperature plays a crucial role in shaping the physiological responses of pelagic fish, which are adapted to thrive in the vast and often fluctuating oceanic environments [31–33]. Increased frequency and magnitude of thermal anomalies in marine ecosystems—such as heatwaves and cold spells—have been widely reported, posing emerging challenges to the stability and productivity of aquaculture operations [34–37]. As ectothermic organisms, these fish are particularly sensitive to temperature changes, and therefore, their ability to maintain homeostasis in the face of thermal stress is critical for survival [38–41]. Temperature fluctuations significantly impact various physiological processes, including oxidative stress, immune function, metabolic activity, and gut microbiota composition [42–44]. Unlike typical teleosts, yellowfin tuna possess a degree of regional endothermy, enabling them to maintain elevated temperatures in metabolically important tissues—such as red muscle, brain, and eyes—via a counter-current heat exchange system (rete mirabile) [45–47]. This specialized thermoregulatory ability enhances their aerobic performance, visual acuity, and sustained swimming capacity, providing a physiological advantage in the thermally dynamic pelagic environment [48–51]. While pelagic fish have evolved adaptive mechanisms to cope with temperature variations, extreme temperature stress can still disrupt these processes, leading to physiological dysfunction and increased vulnerability to environmental challenges [52,53]. Understanding their physiological responses to temperature fluctuations is therefore crucial for optimizing rearing conditions, mitigating temperature-induced stress, and supporting the sustainable development of tuna aquaculture under changing oceanographic conditions.

Both high and low temperature extremes elicit significant oxidative stress in juvenile yellowfin tuna, altering redox homeostasis and triggering compensatory antioxidant responses. Acute heat exposure (34 °C) increased the activity of antioxidant enzymes such as superoxide dismutase (SOD) and catalase (CAT) in the gills and liver within the first 6 to 24 h, reflecting an immediate response to elevated reactive oxygen species (ROS) levels [20,21]. Similarly, acute cold stress (18 °C) induced oxidative responses, with malondialdehyde (MDA)—a biomarker for lipid peroxidation—being elevated in tissues such as the liver, suggesting membrane damage under hypothermic conditions [19]. However, these antioxidant defenses are time- and tissue-dependent, and their effectiveness diminishes under prolonged stress, resulting in cellular dysfunction. Thus, thermal extremes compromise oxidative equilibrium in juvenile tuna, with the liver showing dominant oxidative responses due to its key metabolic functions, while gill tissues may also contribute to stress sensing and early antioxidant activation.

Thermal stress significantly impairs immune and metabolic functions in yellowfin tuna juveniles. Acute high-temperature (34 °C) exposure leads to fluctuations in serum biochemical indicators such as alkaline phosphatase (AKP), alanine aminotransferase (ALT), and lactate dehydrogenase (LDH), indicative of hepatic stress and altered metabolic activity [20,21]. These enzymes showed early upregulation—suggesting a heightened metabolic state—and partial suppression over time, pointing to cellular fatigue. In acute cold-stressed (24 °C and 18 °C) individuals, changes in gut microbiota composition and diversity reflected systemic stress, with reductions in beneficial microbial populations likely compromising nutrient absorption and immune modulation [19]. Furthermore, shifts in enzyme activity (e.g., pepsin, trypsin, lipase) under both thermal extremes reflected disruptions in digestive capacity, further implicating temperature as a critical regulator of physiological homeostasis.

Thermal fluctuations drive substantial reorganization in energy metabolism and gut microbial communities. In response to acute heat (34 °C), muscle tissues showed decreased energy reserves (proteins, lipids, carbohydrates) and elevated energy expenditure, as evidenced by increased ETS (electron transport system) activity and reduced cellular energy allocation (CEA) [20,21]. The red and white muscle tissues respond differently, with red muscles increased energy utilization early on, while white muscles exhibited lipid depletion. Acute cold stress (24 °C and 18 °C), on the other hand, enhanced α-diversity in gut microbiota but reduced the abundance of dominant phyla such as Proteobacteria, while increased Firmicutes and Verrucomicrobiota, suggesting microbial adaptations to reduced host metabolism [19]. These metabolic and microbial shifts indicate that temperature extremes not only impair cellular and enzymatic efficiency but also alter symbiotic microbial dynamics, collectively reducing the resilience of juvenile yellowfin tuna in aquaculture settings.

Collectively, these findings demonstrate that thermal stress induces a multifaceted physiological response in juvenile yellowfin tuna, encompassing oxidative imbalance, immune and metabolic disruption, and shifts in energy allocation and gut microbial composition. These effects are highly tissue-specific, time-dependent, and influenced by the direction and severity of temperature change. The resulting impairments in homeostasis not only compromise the fish's ability to cope with environmental stress but also present significant challenges for maintaining health and performance in aquaculture systems. Understanding these integrated responses is essential

for designing temperature management strategies that align with the species' physiological limits and developmental needs.

3. Physiological Responses of Juvenile Yellowfin Tuna to Salinity Stress

Salinity stress has significant physiological impacts on marine pelagic fish, which are adapted to the vast and stable salinity conditions of open ocean environments [53–55]. In natural habitats, these species—often classified as stenohaline—rarely encounter abrupt salinity changes and thus have limited salinity tolerance ranges; however, in land-based or nearshore aquaculture systems, they may be exposed to suboptimal or fluctuating salinity levels due to rainfall, evaporation, water exchange practices, or system failures [56–58]. Such deviations from optimal salinity represent a form of salinity stress, which may involve either acute or chronic exposure to hypo- or hyper-saline conditions, or frequent salinity fluctuations. These forms of salinity stress are particularly challenging for stenohaline pelagic species like yellowfin tuna, which lack the osmoregulatory plasticity seen in more euryhaline species [59–61]. These variations can challenge the osmoregulatory systems of pelagic fish, which rely on coordinated physiological mechanisms to maintain ionic and osmotic balance, with the gills, kidneys, and intestines playing key regulatory roles [22,62,63]. Under hyper- or hypo-saline conditions, ion transporters like Na^+/K^+-ATPase in gill tissues adjust their activity to manage ion gradients. However, in stenohaline species, extreme changes in salinity can overwhelm these mechanisms, leading to ion imbalance, dehydration, or overhydration. Additionally, salinity-induced oxidative stress was well-documented in pelagic or narrow-salinity-tolerant fish, marked by elevated reactive oxygen species (ROS), which can damage cellular structures [64–66]. This oxidative damage further impairs metabolic processes, enzyme activity, and energy allocation, affecting growth, reproductive health, and immune function [59,67,68]. Moreover, studies in pelagic and stenohaline fish have shown that salinity shifts could disrupt gut microbiota composition, compromising digestion and nutrient absorption [69–71]. These physiological disturbances are particularly concerning in aquaculture systems rearing oceanic species, where even modest salinity deviations may trigger pronounced stress responses. Understanding how pelagic fish adapt to salinity stress is crucial for predicting the impacts of climate change and maintaining the health of marine ecosystems.

Exposure to acute low salinity conditions (from 32–34‰ to 29‰) alters the antioxidant defense mechanisms in juvenile yellowfin tuna. Oxidative biomarkers indicated a time-dependent modulation of enzymatic responses, particularly in the gills and muscles. Activities of superoxide dismutase (SOD) and glutathione peroxidase (GSH-Px) increased significantly in gill tissues by 48 h post-stress, indicating a compensatory mechanism to elevated reactive oxygen species (ROS). In contrast, antioxidant enzyme activities in the liver and muscle tissues initially rise and then fall, reflecting redox imbalance and possible enzymatic exhaustion. Malondialdehyde (MDA) levels—an index of lipid peroxidation—peaked alongside GSH-Px before declining, suggesting transient oxidative damage followed by partial recovery. These findings highlight the localized and transient oxidative stress responses in tuna under salinity perturbation, with implications for their health in fluctuating estuarine and coastal aquaculture environments [23].

Salinity fluctuations induce complex changes in digestive enzyme activities across the gastrointestinal tract of juvenile yellowfin tuna. Acute hyposalinity (29‰ for 48 h) significantly reduced the activity of key proteolytic enzymes such as pepsin and trypsin in the stomach and pyloric ceca, potentially impairing protein hydrolysis and nutrient assimilation. Meanwhile, the activity of α-amylase exhibited a biphasic trend—initially declining, then recovering—particularly in the pyloric ceca, indicating region-specific adaptive responses. Lipase and chymotrypsin showed inconsistent changes but generally follow a decrease-increase-stabilize pattern. These enzymatic fluctuations suggest a transient disruption of digestive efficiency under salinity stress. Although yellowfin tuna demonstrates a degree of resilience at 29‰, sustained deviations from optimal salinity (31.2–33.3‰) could impair feeding performance, energy balance, and growth potential in intensive rearing systems [22].

Juvenile yellowfin tuna exhibit pronounced osmoregulatory disturbances under acute low-salinity stress. Serum osmotic pressure, Na^+, Cl^-, and K^+ concentrations declined significantly within the first 6 h of exposure to 29‰ salinity, indicating impaired ion homeostasis. Prolactin (PRL), a hormone involved in ion retention and freshwater adaptation, showed a biphasic response—initial suppression followed by a marked increase by 48 h—suggesting endocrine compensation for osmotic imbalance. Concurrently, lactate and glucose levels fluctuate, reflecting stress-induced metabolic shifts. The activities of ATPase enzymes, including Na^+/K^+-ATPase and Ca^{2+}/Mg^{2+}-ATPase, were modulated across gill, kidney, and intestinal tissues, implying altered energy allocation for active ion transport. These osmoregulatory and hormonal adjustments illustrate the physiological cost of hyposalinity adaptation, underscoring the importance of salinity stability for maintaining ionic and metabolic homeostasis in yellowfin tuna [24].

This knowledge is also vital for the aquaculture industry, where salinity fluctuations can affect fish growth, reproduction, and overall health. In aquaculture systems, maintaining stable salinity conditions is essential for optimizing fish production and minimizing stress-induced diseases. As demonstrated in juvenile yellowfin tuna, even modest reductions in salinity can lead to a series of coordinated physiological responses, including time-dependent activation and subsequent suppression of antioxidant enzymes, disruptions in digestive enzyme activity across the gastrointestinal tract, and impaired osmoregulatory function marked by ionic imbalance and hormonal compensation. These responses reflect the physiological burden imposed by hyposalinity on stenohaline pelagic species, which are poorly adapted to osmotic variability. Sustained deviations from the optimal salinity range (31.2–33.3‰) may further compromise nutrient assimilation, metabolic efficiency, and energy allocation. Therefore, a mechanistic understanding of these stress pathways provides a critical foundation for developing targeted aquaculture strategies—including salinity stabilization, early stress detection, and dietary modulation—to support the health, performance, and resilience of yellowfin tuna under intensive rearing conditions.

4. Physiological Impacts of Ammonia Nitrogen Stress on Juvenile Yellowfin Tuna

Ammonia nitrogen, a primary form of nitrogenous waste in aquatic environments, poses significant physiological challenges to marine fish, particularly those adapted to oceanic conditions [72–74]. In marine ecosystems, ammonia exists predominantly as unionized ammonia (NH_3), which is highly toxic and can rapidly diffuse across gill membranes, disrupting cellular processes [75–77]. Elevated ammonia levels impair osmoregulation, leading to altered ion balance, dehydration, and reduced efficiency in nitrogen excretion. Furthermore, prolonged exposure to elevated ammonia concentrations results in oxidative stress, inflammation, and disruptions in mitochondrial function, which can compromise the immune system and metabolic processes. Studies on various marine species, such as the yellowtail (*Seriola rivoliana*), Atlantic cod (*Gadus morhua*) and large yellow croaker (*Larimichthys crocea*), demonstrate that ammonia exposure induces alterations in enzyme activities, including those related to antioxidative defense and cellular repair mechanisms [78–80]. In severe cases, sustained ammonia toxicity can lead to gill damage, reduced growth rates, and increased mortality. Thus, understanding the physiological impacts of ammonia nitrogen is crucial for managing aquaculture systems and ensuring the health and sustainability of marine fish populations in both natural and controlled environments.

Ammonia nitrogen (NH_3-N), a prevalent toxicant in high-density aquaculture systems, exerts profound oxidative stress on juvenile yellowfin tuna [25,26]. Under acute NH_3-N challenge, increasing concentrations elevate intracellular reactive oxygen species (ROS), triggering lipid peroxidation—evidenced by elevated malondialdehyde (MDA) levels in hepatic and gill tissues. The organism's antioxidant defense machinery, including superoxide dismutase (SOD), catalase (CAT), and glutathione peroxidase (GSH-PX), demonstrates a dose- and time-dependent biphasic response. While acute sublethal concentrations (e.g., 5 mg/L) may transiently activate antioxidant enzymes as a compensatory mechanism, sustained or higher concentrations (≥ 10 mg/L) suppress enzymatic activity, indicating oxidative exhaustion and impaired cellular homeostasis. This imbalance culminates in structural and functional damage to mitochondria, endoplasmic reticulum, and plasma membranes, particularly in metabolically active organs such as the liver and gills [25–27].

Beyond redox imbalance, acute NH_3-N stress alters immune function through both enzymatic and molecular pathways [24]. Changes in serum immune enzyme activities—particularly alkaline phosphatase (AKP) and acid phosphatase (ACP)—reveal a transient hyperactivation followed by downregulation, suggesting immunological dysregulation. On a transcriptomic level, acute ammonia exposure (≥5 mg/L) elicits marked changes in cytokine gene expression. Upregulation of pro-inflammatory markers such as *tumor necrosis factor alpha* (TNF-α), *interleukin-6 receptor* (IL-6r), and downregulation of anti-inflammatory cytokines like *interleukin-10* (IL-10) suggest an initial inflammatory cascade that fails to resolve effectively, potentially leading to chronic immune suppression [28]. Such immunotoxic effects compromise pathogen resistance and raise concerns regarding disease outbreaks in closed or semi-closed farming systems.

The gills, as primary osmoregulatory and respiratory organs, are particularly vulnerable to NH_3-N toxicity. Histopathological examinations in our previous study revealed epithelial lifting, lamellar fusion, and necrosis once NH_3-N reached 5–10 mg/L, all of which impair respiratory efficiency and ionic regulation [27]. Simultaneously, NH_3-N exposure disturbs skeletal muscle homeostasis. Red muscle, crucial for sustained swimming, displays greater resilience at acute low ammonia concentrations (5 mg/L) through transient increases in SOD and CAT activity. However, at elevated concentrations, both red and white muscle tissues show sharp declines in antioxidant capacity, resulting in oxidative damage, muscle fatigue, and compromised locomotor performance. These impairments are especially detrimental to yellowfin tuna, a highly active pelagic species reliant on continuous swimming for respiration, feeding, and waste excretion [24,27].

Ammonia nitrogen also disrupts the digestive physiology and metabolic equilibrium of juvenile yellowfin tuna [28]. Enzymatic assays indicate dose-dependent inhibition of key digestive enzymes—amylase (AMS), lipase (LPS), and pepsin—across the foregut, pyloric caeca, and hepatic tissues at 5–10 mg/L NH_3–N. These disruptions hinder macronutrient assimilation and compromise feed conversion efficiency. Concurrently, hepatic biomarkers such as aspartate aminotransferase (AST) and alanine aminotransferase (ALT) are elevated, indicating hepatocellular stress and potential hepatic dysfunction. The cumulative metabolic burden imposed by NH_3-N exposure not only retards somatic growth but also exacerbates susceptibility to environmental fluctuations and disease, posing a significant bottleneck to the sustainability of closed-cycle yellowfin tuna aquaculture operations.

In summary, our studies reveal that ammonia nitrogen exposure triggers a cascade of physiological disturbances in juvenile yellowfin tuna, encompassing oxidative stress, immunosuppression, tissue degeneration, and metabolic disruption. The biphasic antioxidant responses, pro-inflammatory cytokine activation, gill and muscle histopathology, and digestive enzyme inhibition observed under varying ammonia concentrations collectively highlight the species' vulnerability to nitrogenous waste accumulation. These findings underscore the systemic nature of ammonia-induced stress and its organ-specific manifestations, particularly in metabolically active tissues such as the liver, gills, and muscle. For a pelagic species like yellowfin tuna—characterized by high oxygen demand, continuous swimming behavior, and limited tolerance to water quality fluctuations—these impairments pose significant threats to health, performance, and survival in high-density recirculating aquaculture systems. Therefore, effective ammonia management is imperative for sustaining physiological stability and optimizing production outcomes in intensive tuna farming.

5. Physiological Impacts of Seawater Acidification on Juvenile Yellowfin Tuna

Ocean acidification, driven by increased atmospheric CO_2 absorption, poses a significant threat to marine ecosystems, particularly pelagic fish species [81–83]. As seawater pH declines, critical physiological processes—such as respiration, ion regulation, and neurological function—are disrupted. Acid-base imbalances can impair metabolic efficiency, while altered ion gradients affect otolith development and sensory perception, potentially leading to behavioral changes and reduced survival [84–86]. Moreover, evidence suggests species-specific vulnerability to seawater acidification, with early life stages being particularly susceptible. Understanding these physiological impacts is crucial not only for predicting population dynamics and ecosystem responses in an acidifying ocean, but also for assessing how declining pH may compromise fish health, performance, and survival in marine aquaculture systems.

Juvenile yellowfin tuna exposed to acidified seawater conditions experience significant alterations in their antioxidant defense systems. A decline in seawater pH to 7.1 or lower induced oxidative stress, evident from increased levels of malondialdehyde (MDA) in the liver, a marker of lipid peroxidation and cellular membrane damage [29,30]. Antioxidant enzymes such as superoxide dismutase (SOD), catalase (CAT), and glutathione peroxidase (GSH-Px) displayed tissue-specific and pH-dependent activity patterns. Notably, SOD and CAT activities peaked in gill tissues at pH 7.1, suggesting an adaptive response to elevated reactive oxygen species (ROS), while GSH-Px activity was maximized in the skin. However, at pH 6.6, overall antioxidant activity declined or became inconsistent, indicating enzymatic exhaustion and impaired redox homeostasis under more severe acidification.

Acidification also compromised innate immunity in juvenile yellowfin tuna by modulating key immune-related enzymes. Activities of acid phosphatase (ACP), alkaline phosphatase (AKP), and lysozyme (LZM) vary across tissues and pH levels. ACP activity, which plays a critical role in phagocytic function, was highest in the liver under control conditions (pH 8.1) but decreases significantly with lower pH, indicating impaired lysosomal activity and immunocompetence. AKP activity in the red muscle reached its nadir at pH 7.1, while LZM activity—particularly prominent in gill tissues, peaked under extreme acidification (pH 6.6), potentially reflecting a stress-induced inflammatory response [30]. These findings suggest that while yellowfin tuna exhibits some resilience to moderate acidification, its immune regulation deteriorates in more acidic environments.

Seawater acidification exerts deleterious effects on energy metabolism and tissue integrity in juvenile yellowfin tuna. Enzyme activities associated with glycolysis and ion regulation—such as lactate dehydrogenase (LDH), hexokinase (HK), pyruvate kinase (PK), and Na^+/K^+-ATPase—peaked at pH 7.1, reflecting heightened metabolic compensation in response to acid stress. However, at pH 6.6, this upregulation is unsustainable, leading to systemic energy imbalance. Histological analysis reveals progressive liver damage under decreasing pH, including hepatocyte vacuolation, disorganized cell architecture, and increased sinusoidal gaps, indicative of lipid accumulation and impaired liver function [29]. These structural and biochemical disturbances underscore the

vulnerability of yellowfin tuna to acidification stress, which, if prolonged, may hinder growth, immunity, and overall fitness in aquaculture environments.

In summary, ocean acidification exerts multifaceted physiological impacts on juvenile yellowfin tuna, including oxidative stress, compromised immune function, metabolic dysregulation, and structural liver damage. The observed tissue-specific and pH-dependent responses—such as the transient upregulation of antioxidant and metabolic enzymes at moderate acidification (pH 7.1) and subsequent decline at more severe levels (pH 6.6)—highlight the species' limited capacity for physiological compensation. These findings underscore the potential vulnerability of yellowfin tuna to acidification stress within aquaculture settings, particularly under prolonged exposure. Continued research integrating molecular, cellular, and organismal approaches is essential to assess long-term resilience and to inform mitigation strategies that support sustainable tuna farming in an acidifying ocean.

6. Future Directions

Building upon the physiological insights consolidated in this review, several key areas emerge as promising and necessary directions for future research and development in yellowfin tuna aquaculture. While substantial progress has been made in characterizing individual stress responses, the complexity of real-world farming systems, where multiple stressors and developmental transitions co-occur, calls for a more integrative and applied research paradigm. Key priorities include:

Toward Multi-Stressor Integration: While individual environmental stressors such as temperature extremes, salinity shifts, and ammonia accumulation have been well characterized in isolation, future studies should aim to unravel the compound effects of co-occurring stressors. In practical aquaculture systems, fish are often exposed to multiple environmental fluctuations simultaneously, and the physiological trade-offs and cross-tolerance mechanisms under multi-stressor conditions remain poorly understood. Addressing this gap will enable the development of more effective aquaculture management strategies to predict and mitigate stress-induced vulnerabilities in cultured yellowfin tuna. From an application standpoint, multi-stressor experiments should be parameterized using co-variations commonly observed on farms and executed in representative system types (e.g., land-based RAS and offshore cages). Deliverables should include actionable interaction maps that indicate the sequence of operational responses, along with practical checklists and short training modules for technicians so that early compound stress signals can be recognized and mitigated during routine production.

Advancing Mechanistic Understanding Through Multi-Omics: The physiological responses highlighted in this review are deeply interconnected at the molecular level, yet most existing studies remain focused on single-indicator endpoints. Integrating transcriptomics, metabolomics, and microbiomics across tissues and time scales will be essential to map regulatory networks that govern redox signaling, immune modulation, and metabolic control. Such holistic approaches can also help identify early-warning biomarkers and therapeutic targets to enhance stress resilience in juvenile tuna. To enhance translational value, multi-omics efforts ought to converge on a minimal set of robust, low-burden biomarkers that can be measured with farm-compatible assays and interpreted in real time.

Incorporating Ontogenetic Context in Aquaculture Design: Developmental stage strongly modulates physiological plasticity, with enzyme activities, ion balance, and behavioral rhythms changing substantially with growth. Future research should emphasize the dynamic nature of physiological responses across size classes and formulate growth-phase-specific management strategies. Tailoring environmental control, dietary regimes, and tank design to ontogenetic demands could significantly enhance both welfare and production efficiency.

Precision Farming Strategies: Translating physiological insights into aquaculture practice represents a critical step forward. Real-time monitoring systems that adjust temperature, pH, and salinity in response to physiological thresholds; functional feed additives targeting oxidative balance and gut integrity; and behavioral conditioning protocols aligned with diel activity rhythms all hold promise for advancing precision aquaculture. These innovations should be designed not only to mitigate stress but also to actively align environmental inputs with the evolving physiological capacities of yellowfin tuna, ultimately enabling scalable, health-oriented farming under diverse and changing conditions. For adoption in industry, precision approaches should be packaged as light-touch monitoring protocols and decision-support tools that translate sensor and behavioral data into clear husbandry actions. Demonstrations with producers—including side-by-side comparisons to current practice and brief staff training—will help quantify benefits, reduce perceived risk, and accelerate scale-up.

7. Conclusions

This review synthesizes a series of recent studies conducted by our research team on the physiological responses of juvenile yellowfin tuna under key environmental stressors relevant to aquaculture. By examining the effects of temperature extremes, salinity fluctuations, ammonia nitrogen accumulation, and seawater acidification, we have identified a spectrum of redox imbalances, immune perturbations, metabolic disruptions, and behavioral alterations that collectively shape the resilience and vulnerability of this species in controlled rearing environments.

Our findings demonstrate that thermal and salinity stressors significantly compromise oxidative and osmoregulatory homeostasis, leading to tissue-specific antioxidant exhaustion and impaired ion balance. Ammonia nitrogen exposure further disrupts mitochondrial function, immune regulation, and skeletal integrity, while acidified conditions induce metabolic instability and immune suppression. These physiological impairments are compounded by changes in gut microbiota and digestive enzyme activity, revealing complex interactions between environmental stress and host nutritional physiology.

Together, these results underscore the necessity of environment-specific aquaculture management strategies. Optimizing thermal regimes, salinity control, water quality, and feeding protocols according to the physiological capacities of juvenile yellowfin tuna is critical for promoting growth, minimizing disease susceptibility, and enhancing overall performance. As yellowfin tuna continues to emerge as a candidate species for high-value aquaculture, our integrative physiological insights provide a foundational reference to guide sustainable and scalable cultivation practices.

Author Contributions

Z.F.: Conceptualization, Investigation, Writing—original draft, Visualization. J.B.: Visualization, Writing—review & editing. Z.M.: Supervision, Project administration, Writing—original draft, Writing—review & editing, Funding acquisition. All authors have read and agreed to the published version of the manuscript.

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Conflicts of Interest

The authors declare no competing interests.

Use of AI and AI-assisted Technologies

No AI tools were utilized for this paper.

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