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Effects of Hypoxia on the Survival of Large Yellow Croaker (*Larimichthys crocea*) under Different Temperature and Fish Weight Conditions

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Abstract

Hypoxia zones (low dissolved oxygen, DO < 2 mg/L) exceeding 20,000 square kilometers occur near the Yangtze River Estuary in the East China Sea, with a minimum DO below 1 mg/L. Large yellow croaker was once one of the four major fisheries in China, yet its wild resources have declined markedly. In summer, large yellow croaker populations in some habitats of the East China Sea may be exposed to hypoxia. Hypoxia may exert adverse effects on large yellow croaker, but the associated impacts remain poorly understood. We examined hypoxia-induced mortality in large yellow croaker to quantify how mortality changes with hypoxia severity and to explore how hypoxia vulnerability varies with fish body size and water temperature. Hypoxia tolerance of large yellow croaker was tested at two temperatures (25˚C and 30˚C) and three dissolved oxygen (DO) concentrations (1.2, 1.4, and 1.6 mg/L). Three size groups of fish were used, with mean body weights of 58.6, 112.5, and 167.3 g. In middle size fish (mean body weight 112.5 g) mortality test, Large yellow croaker exposed to 1.6 mg/L DO exhibited no mortality within 24 h at 25˚C and only 7% mortality at 30˚C. Fish exposed to 1.4 mg/L DO showed 87% mortality within 24 h at 25˚C and 93% at 30˚C. Large yellow croaker exposed to 1.2 mg/L DO reached 100% mortality at 30˚C within 3.7 h and at 25˚C within 5.5 h. Linear regression analysis showed that the 24-h LC_{50} (lethal oxygen concentration causing 50% mortality) was 1.37 mg/L at 25˚C and 1.41 mg/L at 30˚C. Cox regression survival analysis was conducted to evaluate relationships between survival rate and DO concentration, fish body size, and temperature. DO exerted a significant effect on mortality hazard: 1 unit DO increase reduced the hazard by approximately 38.3%; 1˚C rise in temperature increased the mortality hazard by 8.5%; and each 1 g increase in body weight

decreased the mortality hazard by 0.1%. Our experiments demonstrated that 24-h acclimation to sublethal hypoxia significantly reduced mortality during subsequent exposure to lethal hypoxia. Our findings indicate that direct hypoxia-induced mortality varies with body size, water temperature, and pre-acclimation.

Keywords

Larimichthys crocea, Hypoxia, Mortality, Survival Analysis

1. Introduction

Over the past few decades, near-shore seas hypoxia has been increasing worldwide in frequency, severity, and area due to increased nutrient loading [1]. Low oxygen can lead to habitat loss of fishery resources by changing direct mortality and migration, decreasing suitable habitats, altering food resources, increasing risk to predation, and disturbing life cycles [2].

The degradation of fishery habitats by hypoxia is well documented in systems such as the Neuse River estuary, where seasonal summer oxygen depletion renders broad areas unoccupied by most fish species. The consequent crowding of survivors into oxygen-sufficient pockets not only raises fish densities but also amplifies competition for space and food within these refuges [1].

Although estuarine hypoxia most often develops in the deeper bottom layer, it is not static. Wind forcing and lateral tidal action intermittently carry oxygen-poor bottom water upward and into shallow nearshore habitats, creating sudden low-oxygen events that can trap fish when conditions shift too quickly or when escape routes to normoxic water are absent [3] [4]. The speed of these fluctuations is striking: in Chesapeake Bay, DO measurements have been observed to vary by 0.5 mg/L or more within only 15 minutes [3].

Assessing the consequences of oxygen depletion in estuaries is especially critical, because these systems function as vital nursery habitats that sustain many species of major commercial and ecological importance [5]. There are more than 20,000 square kilometers of low-oxygen zones (DO < 2 mg/L) near the Yangtze River Estuary in the East China Sea, with a minimum value of <1 mg/L [6]-[8]. Large yellow croakers used to be an important economic fish in China, and its wild resources had declined significantly, more importantly, the large yellow croakers might suffer from hypoxia in some distribution areas of the East China Sea in summer. In recent years, large yellow croakers have been become an important economic species of aquaculture in Marine cages under high density condition near the East China Sea [9] [10]. The objective of this research is to consider the mortality effects due to hypoxia on large yellow croaker (*Larimichthys crocea* = *Pseudosciaena crocea*), quantifying how mortality scales with the severity and duration of oxygen depletion, and identifying how fish body size, water temperature, and prior acclimation collectively shape vulnerability to hypoxic conditions.

2. Material and Methods

2.1. Animals: Maintenance and Feeding

Experimental fish were acquired from the Haiwan Fish Breeding Farm in Xiangshan County, Zhejiang Province, and all subsequent trials were conducted on the farm premises. Prior to experiments, fish were maintained in 800-L outdoor aquaria filled with 28‰ seawater under normoxic conditions (DO ≈ 6.5 mg/L), with water temperature regulated at 26˚C - 28˚C. The fish were fed regularly with iced fish pieces. These aquaria formed flow-through system and a thermostat to maintain the experimental temperatures. The walls of the aquaria were covered by an opaque plastic sheet to keep the fish calm. The fish were acclimated over a minimum period of 48 hours prior to the experiments and were starved 24 h before measurements were made. The durations of exposure of hypoxia were 24 hours.

2.2. Experimental Design

Mortality trials

Hypoxia tolerance of large yellow croaker was studied through a probability of mortality method conducted at 28‰ salinity and at two temperatures, 25 and 30˚C, representing typical summer East China Sea conditions. Three size groups of fish (with mean body weights of 58.6, 112.5, and 167.3 g) underwent trials at both temperatures and at 3 different levels of dissolved oxygen [1.2, 1.4, 1.6 mg/L O_2]. Each temperature trial used 180 fish, a sample size determined by modeling confidence in survival-time estimates at different DO concentrations [1]. The design comprised 3 hypoxia levels × 3 weight levels, 10 fish per treatment; each treatment group was replicated twice, and each fish was used only once.

Another trial was conducted to test the effects of acclimation to hypoxia on mortality response. To do this, large yellow croakers were acclimated to 1.6 mg/L O_2 for 24 hours before exposed to 1.2 mg/L O_2 at 25˚C to test the tolerance of the fish to hypoxia. Fish were acclimated to the experimental aquaria, 12 (800-L) flow-through systems, at normoxia (6.5 mg/L DO) to minimize handling effects and to acclimate to the experimental temperature.

During experiments, water recirculation was switched off so that tanks remained completely static. Oxygenation systems were disconnected, and the water surface was covered with a floating plastic film to block air-water oxygen exchange. Target DO concentrations were achieved by direct N_2 injection into the tanks, requiring approximately 1 hour to reach the desired level. This rate of DO reduction was used because previous research showed that the rate of DO reduction does not affect the absolute DO concentration causing death to exposed fish [1] [11].

Ammonia nitrogen was controlled by passing tank water through zeolite-based filters that combined physical and chemical adsorption. Dissolved oxygen was measured with an oxymeter (HI9143, Hanna, Italy) equipped with an oxygen probe calibrated by the air-calibration procedure. Before each experiment, DO

was sampled at multiple locations within tanks to confirm vertical and horizontal uniformity; the water column was found to be well mixed with no significant spatial variation.

Throughout the exposure period, DO was tracked at a frequency of once every 15 min during the first 6 h, after which monitoring frequency was reduced to once every 30 min. The tanks were continuously monitored, and the time of death was recorded for each fish. Upon death, each fish was removed from the tank, weighed and measured, so that size could be used as a covariate in the analysis. The trials were terminated after 24 h, and any remaining live fish were weighed and measured. The experimental duration was set at 24 h because previous work has demonstrated that the lethal effects of low DO in static exposure systems usually manifest within the first 24 hours [1].

2.3. Data Analysis

LC_{50} analysis

LC_{50} is lethal oxygen concentration at which 50% of the individuals of a group die. Linear regression method was used to calculated LC_{50}.

Cox regression analysis

The Cox proportional-hazards model is the most general framework for survival regression, since it imposes no parametric assumptions on the underlying survival distribution. The central goal of survival analysis is to quantify how covariates shape survival trajectories, which is most commonly achieved by specifying a linear model on the log-hazard scale. The Cox model is given by

$$h(t,x) = h_0(t)e^{\beta' x}$$

where x is the covariate vector, β is the unknown parameter vector and the $h_0(t)$ is called the baseline hazard. $h(t, x)$ denotes the resultant hazard, given the values of the covariates for the respective case and the respective survival time (t). Estimation of β relies on the partial likelihood, a technique that bypasses the unknown baseline hazard $h_0(t)$ while properly incorporating censored survival data into the likelihood [1] [12].

In this method, parameters are estimated by maximizing the partial likelihood function. The partial likelihood is defined as

$$L(\beta) = \prod_{i=1}^{k} \frac{\exp\left(\beta' x_{(i)}\right)}{\sum_{l \in R\left(t_{(i)}\right)} \exp\left(\beta' x_{(l)}\right)}$$

In this expression, the denominator accumulates over every individual belonging to the risk set $R(t_i)$ at time t_i, the product spans the k distinct event times ordered in sequence, and $x_{(i)}$ denotes the covariate value associated with the individual whose ordered survival time equals $t_{(i)}$ [1] [13] [14].

All statistical computations were carried out with SPSS 13.0. The analytical dataset comprised exclusively fish that had experienced hypoxic conditions. Those still alive when the trial ended were classified as right-censored, with only their

minimum observed survival time incorporated into the model.

The survivor function is

$$s(t;x) = \left[s_0(t)\right]^{e^{(\beta' x)}}$$

where

$$s_0(t) = e^{\left(-\int_0^t h_0(u)du\right)}$$

is the baseline survivor function. The fitted model was used to construct cumulative hazard plots, enabling visualization of how the hazard function changed across the observation period.

3. Results

3.1. DO Measurement in Tests

The realized DO concentrations in all treatments agreed closely with the nominal targets, and within-treatment variability was well controlled (Table 1). In total, 90% of DO measurements deviated from the target concentration by no more than 0.03 mg/L.

Table 1. Descriptive statistics of recorded DO levels in each treatment, presented as mean ± standard deviation.

DO treatment (mg/ L)	Mean ± 1SD (n = 60)
1.2	1.23 ± 0.013
1.4	1.43 ± 0.012
1.6	1.62 ± 0.015

Control fish maintained at normoxia experienced no mortality at all, verifying that the deaths observed in hypoxic groups could be attributed directly to reduced dissolved oxygen.

3.2. LC_{50} Analysis

Given that preliminary experiments revealed that the vast majority of mortality took place within the opening hours of hypoxic exposure, LC_{50} was estimated solely on a 24-h basis, consistent with the observation period shared by all trials.

large yellow croaker exposed to 1.6 mg/L DO showing no mortality in 24 h at 25˚C, and only 7% mortality at 30˚C. The exposed to 1.4 mg/L DO showing 87% mortality in 24 h at 25˚C, and 93% at 30˚C. large yellow croaker exposed to 1.2 mg/L DO experiencing 100% mortality in in 5.5 h, at 25˚C, and 3.7 h at 30˚C.

Linear regression results showed that relation between mortality (Y) and DO (X) at 25˚C were $Y = -1.67X + 2.79$ and $Y = -1.55X + 2.68$ at 30˚C, and 24-h LC_{50} were 1.37 mg/L at 25˚C and 1.41mg/L at 30˚C.

3.3. Survival Analysis Base on Cox Regression Method

The hazard function for large yellow croaker was influenced by dissolved oxygen,

water temperature, and fish body weight, with DO and temperature reaching statistical significance and body weight showing a marginally significant effect (Table 2). The direction of each effect is encoded in its hazard ratio: a hazard ratio exceeding 1 signifies that a rise in the covariate elevates mortality hazard, whereas a ratio below 1 signifies that a rise in the covariate suppresses it. Hazard decreased with increasing DO, but increased with increasing temperature and weight.

Table 2. Cox regression parameter estimates for large yellow croaker.

Variable in the equation						
Variable	β	*df*	S.E.	Wald	Sig.	Exp(β)
Temperature (˚C)	0.082	1	0.033	6.11	0.013	1.085
DO concentration (0.1 mg/L)	−3.79	1	0.455	69.40	0.000	0. 617
Weight (g)	−0.001	1	0.000	3.19	0.074	0.999

The hazard ratio for each covariate can be roughly interpreted as a measure of relative risk (Table 2). All of the covariates in the models for the species are quantitative (*i.e.*, no categorical covariates), so the estimated percent change in the hazard for a 1-unit increase in a covariate can be obtained by subtracting 1 from the hazard ratio and multiplying by 100 [15]. Thus, for every 1˚C increase in temperature the mortality hazard increases by 8.5% (*i.e.*, [1.085 − 1] × 100). With 1 gram increases in weight the mortality hazard decreases 0.1%. Of all three variables, DO has the greatest effect on mortality hazard, an increase in DO concentration of 0.1 mg/L reduces hazard about 38.3%.

3.4. Effects of Temperature

To examine the temperature effect in detail, predicted survival probabilities for mid-size large yellow croaker were generated from the fitted model and graphed at 25˚C versus 30˚C for every experimental DO level (Figure 1). Regardless of oxygen concentration, fish at 25˚C consistently showed greater survival than those at 30˚C.

In the 1.2 mg/L treatment, survival reached zero at 5.5 h under 25˚C but at only 3.7 h under 30˚C. At 1.4 mg/L, fish at 25˚C had a 6% higher survival rate after 24 h than fish at 30˚C; at 1.6 mg/L, this gap widened to 7%. These results demonstrate that elevated temperature intensified the lethal effects of hypoxia on large yellow croaker.

3.5. Effects of Fish Size

Similarly, we examined weight effects by graphing predicted survival probabilities at 25˚C and 30˚C with DO concentration at the mid-range treatment level of 1.4 mg/L (Figure 2).

Smaller fish consistently exhibited weaker hypoxia tolerance than larger conspecifics, yet the difference remained modest within the limited size range of this

study, with predicted survival for small fish falling 3% - 7% below that of large fish at both 25˚C and 30˚C.

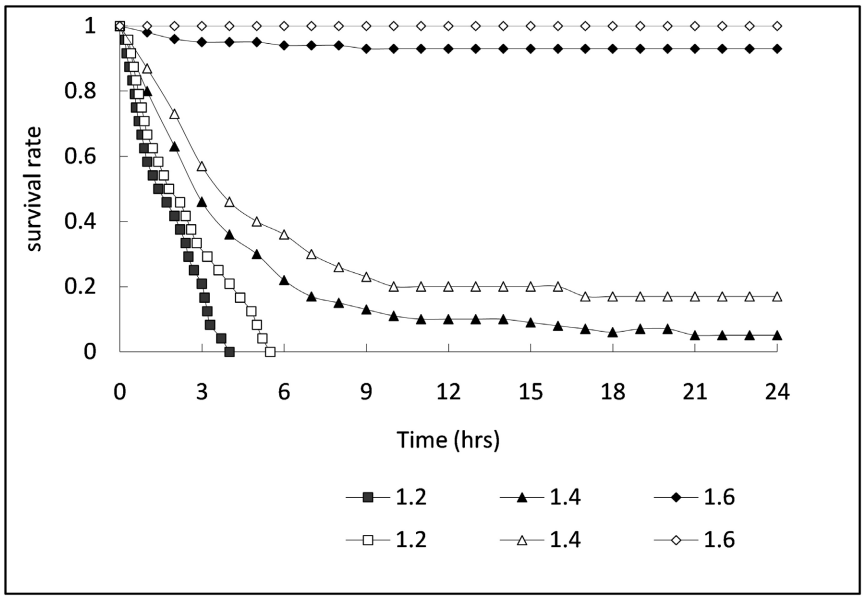


Figure 1. Differences in hypoxia tolerance at 25˚C and 30˚C for large yellow croakers. Filled markers correspond to 30˚C and open markers to 25˚C. Marker shape encodes the oxygen treatment: ▪ = 1.2 mg/L, ▲ = 1.4 mg/L, and ◆ = 1.6 mg/L. Middle size (mean body weight 112.5 g) large yellow croaker weights were used for test.

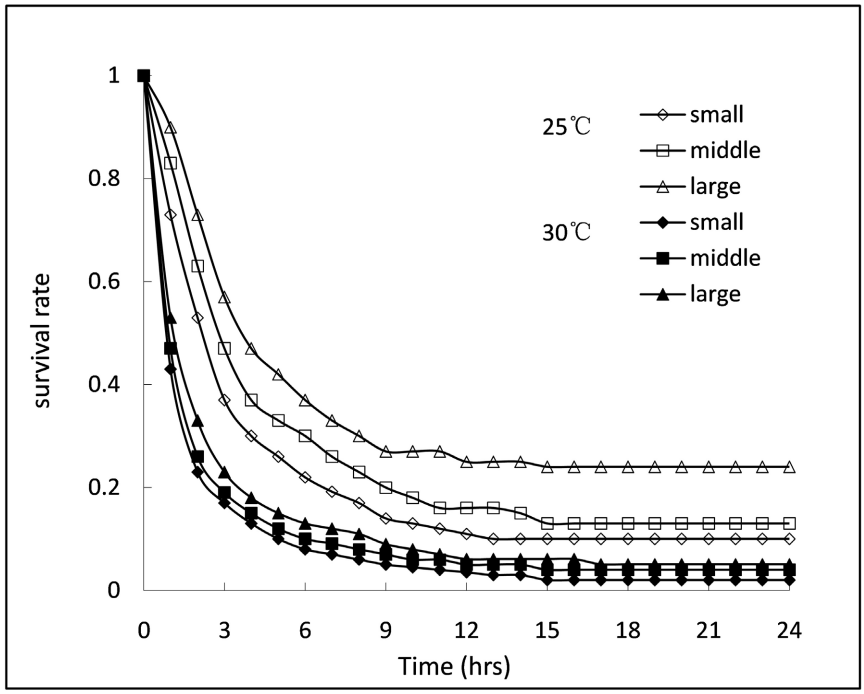


Figure 2. Differences in hypoxia tolerance of large yellow croaker at different weight levels at 1.4 mg/L DO. Filled markers correspond to 30˚C and open markers to 25˚C. Marker shape encodes the oxygen treatment: The symbol shape denotes the DO treatment: ◆ = small (mean weight 58.6g), ▪ = middle (112.5 g), ▲ = large (167.3 g).

3.6. Acclimation Effects on Hypoxic Survival

Prior acclimation to a sublethal DO concentration of 1.6 mg/L over 24 h produced a significant prolongation of survival in large yellow croaker held at 25˚C following subsequent exposure to 1.2 mg/L DO (Log-rank test: $P < 0.0001$).

After 6 h, survival rate for acclimated large yellow croaker was 26.7%, while survival rate for fish that went from normoxia directly to 1.2 mg/L O_2 was 0 after 5.5 h (Figure 3).

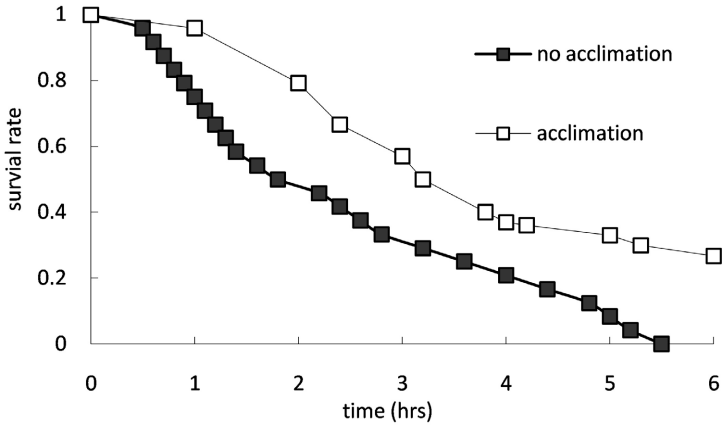


Figure 3. Effect of pre-acclimation on hypoxia tolerance in large yellow croaker. Tests were performed under 1.2 mg/L DO at 25˚C. Filled markers correspond to non-acclimated controls, open markers to acclimated fish; mid-size fish (mean body weight 112.5 g) were used.

4. Discussion

4.1. Effects of DO

Large yellow croaker, a near-bottom species, exposed to 1.2 mg/L O_2 experienced 100% mortality in 6 h (Figure 1), and exhibited less tolerance to hypoxia than benthic species previously studied. For example, naked gobies—benthic fish—exhibited no escape response until DO declined below 0.75 mg/L, and adult males achieved 100% survival when subjected to 0.7 mg/L DO for 7 h each day over 7 days [16]. Elizabeth observed that Spot (*Leiostomus xanthurus*), likewise a benthic species, showed zero mortality during 24 h at 25˚C under 1.2 mg/L DO. [1].

Large yellow croaker, exposed to 1.6 mg/L O_2 experienced no mortality in 24 h at 25˚C (Figure 1), and exhibited more tolerance to hypoxia than pelagic species previously studied, such as adult bay anchovies (*Anchoa mitchilli*), which show less tolerance to low DO, with an estimated 96-h LC_{50} of 1.85 mg/L O_2 [16].

4.2. Effects of Temperature

Our data reveal that elevated temperature intensified the lethal effects of hypoxia on large yellow croaker, with mortality responses at 30˚C exceeding those at 25˚C (Figure 2). The two temperatures tested, 25˚C and 30˚C, are within the range of temperatures large yellow croaker are likely to experience during summer in the

East China Sea. Reported temperature effects on fish hypoxia tolerance are inconsistent across studies: some species show no response, while others exhibit reduced tolerance as temperature climbs. Southern flounder (Paralichthys lethostigma) displayed no variation in DO sensitivity at 6.1, 14.4, and 25.3˚C under 0.68 - 1.09 mL/L O_2 [17], and Atlantic cod (*Gadus morhua*) similarly showed no temperature effect in lethal hypoxia trials, probably because the 2˚C - 6˚C gradient was too limited to manifest such a difference [18].

Conversely, a number of investigations have documented declining hypoxia tolerance with warming, aligning with the present results. For Atlantic cod, the lethal DO threshold for 50% mortality increased from 0.5 mg/L at 5˚C to 2.3 mg/L at 17˚C [19]. Juvenile Atlantic sturgeon (*Acipenser oxyrinchus*) maintained at 3 mg/L O_2 over 3 days showed only 22% mortality at 19˚C but 92% at 26˚C [20], and Spot (*Leiostomus xanthurus*) and menhaden (*Brevoortia tyrannus*) at 1.2 mg/L O_2 had no mortality at 25˚C but 30% - 40% at 30˚C [1]. This reduction in hypoxia tolerance under warming is mechanistically plausible: higher temperatures accelerate metabolic rate, which in turn increases oxygen demand [21].

4.3. Effects of Size

Hypoxia tolerance was modulated by body weight, with larger large yellow croaker exhibiting steadily increasing resistance to low oxygen as weight grew (**Figure 2**). Within the size range examined, this weight effect accounted for a survival difference of 4% - 7% between small and large fish at 25˚C, and 2% - 10% at 30˚C.

The literature on body-size effects on fish hypoxia tolerance yields conflicting results [1]. One proposed explanation is that larger fish are more hypoxia-tolerant than smaller ones, attributable to their superior anaerobic metabolic capacity. [22]. These findings are consistent with our results for large yellow croaker, in which larger individuals showed stronger hypoxia tolerance. Comparable evidence has been reported in the literature: Elizabeth found that smaller spot were consistently less resistant to low oxygen than larger ones [1], and Shephard observed that small brook trout (*Salvelinus fontinalis*) perished more quickly in hypoxic water than larger fish [23].

Nevertheless, a counterargument suggests that larger fish generally consume more oxygen per hour than smaller conspecifics [24], and may therefore be more vulnerable to low oxygen. Evidence supporting this alternative includes stress responses displayed by larger fish but not smaller ones under hypoxic conditions [25], significantly lower lethal DO concentrations in juvenile brown shrimp (*Penaeus aztecus*, 1.37 g) relative to sub-adults (6.12 g) [26], and Atlantic menhaden showing reduced hypoxia tolerance at larger body sizes [1]. In contrast, other investigations have reported no association between body size and hypoxia survival time in cutthroat trout (*Oncorhynchus clarki*) [27], rainbow trout (*Oncorhynchus mykiss*), or perch (*Perca fluviatilis*) [28]. The overall heterogeneity of findings indicates that body size can influence hypoxia tolerance, but the nature of this effect appears to be species-specific [1].

4.4. Effects of Acclimation

Fish given a 24-h pre-exposure to sublethal hypoxia prior to transfer to 1.2 mg/L O_2 displayed a pronounced survival benefit relative to those experiencing an abrupt 1-h drop from normoxia to the same lethal concentration. By 6 h, the acclimated group still retained 26.7% survival, whereas the non-acclimated group had already reached 0% (**Figure 3**). Since the majority of our trials omitted the pre-acclimation step, they reflected a pattern in which more than 90% of mortality occurred during the initial 6 h. A similar early-mortality concentration has been documented by Magaud [29] in 24-h trials on juvenile rainbow trout under varying oxygen regimes and by Elizabeth [1] in 24-h hypoxia trials on spot. Fish typically either perished within the first few hours or survived through the end of the trial—a bimodal outcome that Magaud attributed to partial acclimation of trout to hypoxia [29]-[31].

The mechanisms underlying this rapid hypoxia acclimation remain unresolved [1]. Yet fish possess a suite of acute compensatory responses to oxygen deprivation. Besides increasing ventilation frequency [32], numerous species—menhaden included—promptly raise hematocrit and hemoglobin concentrations under hypoxic exposure [33] [34], which enhances the oxygen-carrying capacity of blood. In certain teleosts, the spleen operates as an effective blood reservoir: it holds blood cells under resting conditions and mobilizes them into systemic circulation through contraction during hypoxia [33] [35]-[37]. In addition, hypoxia-induced movement of water from plasma into surrounding tissues can itself produce a marked increase in hematocrit [33] [38].

Fish undergoing acclimation may additionally shift their behavior to reduce energy demands under oxygen stress [39]. In our experiments, hypoxic large yellow croaker displayed lower locomotor activity and elevated rates of aquatic surface respiration (ASR). ASR constitutes a widespread behavioral response to low DO among teleosts [40]. Short-term acute exposure to severe hypoxia may alter anaerobic capacity, whereas extended exposure to mild hypoxia may facilitate acclimation of aerobic metabolism [41].

The empirical basis for hypoxia acclimation in fishes is highly variable. Davis cited multiple indications that fish exhibit some degree of acclimation to declining DO [39], and Shephard reported that acclimation extended survival five-fold under lethal oxygen conditions [23]. In three freshwater species, fish pre-conditioned at low DO for 11 - 15 days showed greater hypoxia resistance than those subjected to an abrupt 1 - 2 h oxygen decline [42]. Nevertheless, juvenile striped bass (*Morone saxatilis*) displayed little difference in survival between gradual 24-h oxygen reduction and sudden low-oxygen exposure [43], and a prolonged repeated-exposure trial in rainbow trout likewise found no evidence of hypoxic acclimation [31]. Overall, these studies indicate that although acclimation may occur in certain species, its magnitude and characteristics appear to be species-specific.

In the present 25˚C trials, the DO gap between 0% and 100% mortality

amounted to only 0.4 mg/L O_2, spanning from 1.6 down to 1.2 mg/L. Such narrow lethal thresholds are broadly consistent with the fish literature, in which oxygen levels governing survival typically occupy a very limited interval. Downing and Merkens, in experiments on eight fish species, found the gap between 100% survival and 100% mortality to be approximately 0.5 mg/L O_2 [44]; juvenile Spot (*Leiostomus xanthurus*) and Atlantic menhaden (*Brevoortia tyrannus*) showed a corresponding gap of 0.6 mg/L O_2 [1]; rainbow trout moved from complete survival at 2.3 mg/L O_2 to complete mortality at 1.7 mg/L O_2 [30]; and Seager reaffirmed that this transition range in rainbow trout was at most 1 mg/L O_2 [31].

Marked mortality from hypoxia in fishes is generally not observed until DO declines to extremely low levels [1]. In our trials, the majority of large yellow croaker deaths occurred at the severely hypoxic concentration of 1.2 mg/L O_2. Tolerance thresholds vary widely across species: less tolerant forms such as cutthroat trout exhibit significant mortality near 2 mg/L O_2 [27]; more tolerant species such as Spot and Atlantic menhaden show major mortality only when DO falls below 1 mg/L O_2; and highly tolerant species such as roach (*Rutilus rutilus*) may not experience appreciable mortality until oxygen drops below 0.5 mg/L O_2 [31].

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Conflicts of Interest

The author declares no conflicts of interest regarding the publication of this paper.

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