

## 5 Dissolved Oxygen and Water Quality Impacts

### 5.1 Hypoxia-induced physiological and behavioral changes

Turbot is notably sensitive to reduced dissolved oxygen, with measurable behavioral disturbance appearing before lethal thresholds are reached. During progressive hypoxia, abnormal swimming behavior was observed when dissolved oxygen fell below 5 mg/L, respiration frequency increased at the critical oxygen tension, and loss of equilibrium occurred near 1.30 mg/L; related work identified a critical oxygen tension around 3.34 mg/L and showed that turbot under severe hypoxia first respond through increased ventilation amplitude and frequency to maintain oxygen uptake (Maxime et al., 2000). This initial compensatory phase reflects a relatively strong short-term regulatory capacity, but it does not prevent endocrine and metabolic stress as oxygen availability continues to decline. At low oxygen tension, plasma cortisol, glucose, and lactate increase markedly, and severe hypoxia triggers rises in adrenaline and noradrenaline together with reduced arterial oxygen partial pressure, indicating activation of both the stress axis and anaerobic metabolism (Jia et al., 2021).

Longer hypoxic exposure shifts the response from acute compensation to impaired growth and tissue-level remodeling. Under chronic hypoxia at about 3.5 mg/L for 8 weeks, turbot showed reduced digestibility, feed efficiency, weight gain, and body indices, along with poorer muscle texture, lower nutrient content, and reduced myofiber number, indicating that oxygen shortage depresses both production performance and flesh quality (Guo et al., 2026). Similar long-term exposure elevated plasma cortisol and glucose, increased glycolytic and lipolytic enzyme activities, altered gill morphology, and upregulated *hif-1α*, *hif-2α*, and *hif-3α*, suggesting that chronic hypoxia induces coordinated endocrine, metabolic, and branchial adjustments to sustain gas exchange and energy balance (Jia et al., 2021).

### 5.2 Ammonia, nitrite, and toxic metabolite accumulation

Nitrogenous wastes are major toxicants in intensive turbot culture, and both ammonia and nitrite disrupt physiological homeostasis within short exposure periods. High ammonia exposure elevated CRH, ACTH, and cortisol while reducing GH, lysozyme, complement factors, and IgM, demonstrating simultaneous activation of the hypothalamic-pituitary-interrenal axis and suppression of humoral immunity. Nitrite caused a partly overlapping but distinct syndrome: at 0.4-0.8 mM, it increased GPT, GOT, ALP, *c3*, and C4, reduced IgM and lysozyme, and upregulated gill *hsp70*, *hsp90*, TLR-3, *tnf-α*, and *il-1β*, indicating blood physiological dysfunction together with inflammatory and immune disturbance (Jia et al., 2024).

The toxic effects of these metabolites also extend to oxidative damage, ion imbalance, and reduced oxygen transport. In ammonia-exposed fish, liver SOD and CAT activities, HSP expression, and MDA increased while GSH and IGF-1 decreased, supporting a mechanism involving oxidative stress and impaired growth regulation. Nitrite and nitrate further compromise oxygen-carrying capacity: nitrite raised methemoglobin, cortisol, glucose, and K<sup>+</sup> while lowering hemoglobin and Na<sup>+</sup>, whereas chronic nitrate exposure increased plasma NO<sub>3</sub><sup>-</sup>, NO<sub>2</sub><sup>-</sup>, methemoglobin, cortisol, glucose, lactate, and K<sup>+</sup> and decreased Hb, Na<sup>+</sup>, and Cl<sup>-</sup>, indicating hypoxic stress combined with osmoregulatory and metabolic disruption (Yu et al., 2021).

### 5.3 Water quality deterioration and chronic stress effects

Beyond acute toxicants, chronic deterioration of water quality in recirculating or high-density systems imposes persistent stress that reduces growth and damages multiple organs. Elevated nitrate over 60 days reduced survival and growth, caused dose-dependent gill and liver histopathology, lowered hemoglobin, increased methemoglobin, and dysregulated the GH/IGF-1, thyroid, and HPI axes, showing that nitrate acts as a chronic systemic stressor rather than a benign end-product of nitrification (Yu et al., 2021). Chronic nitrate exposure also injured the intestine by causing microvillus atrophy and lamina propria necrosis, downregulated tight-junction and mucin genes, and shifted the microbiota toward lower intrinsic flora and more potential pathogens, indicating weakened barrier function and long-term health deterioration (Yu et al., 2020).

Other chronic water-quality stressors in RAS show similarly broad impacts. Prolonged CO<sub>2</sub> exposure reduced specific growth rate, increased feed conversion ratio, damaged gill, liver, and intestinal tissues, and altered plasma