HCO3-, Na+, and Cl- as well as osmoregulatory and acid-base genes, indicating sustained disturbance of ion balance and buffering physiology even at relatively low CO_2 concentrations (Guo et al., 2023). Chronic handling or crowding stress also amplifies these effects at the whole-animal level: repeated handling elevated cortisol and progressively depressed innate immune indicators, while high-density exposure combined with ammonia intensified Na+ imbalance, Na+/K+-ATPase activity, and pro-inflammatory and heat-shock gene expression, showing that poor water quality and husbandry stress often interact rather than act in isolation.

6 Pathogen Dynamics under Environmental Stress

6.1 Bacterial infections

Bacterial disease is a persistent constraint in turbot aquaculture, and *Vibrio* spp. have been especially prominent across larval and grow-out systems. Early reports identified *Vibrio*sis as a common problem in turbot production and documented the involvement of several taxa, including *Vibrio anguillarum* serogroups O1, O2a, and O2b, as well as members of the *V. splendidus*/*pelagius* group. Farm-level microbiological surveys similarly found that *Vibrio* spp. were among the most prevalent bacteria recovered from diseased turbot, although lesion type could not always be linked to a single bacterial species.

More specific evidence shows that some *Vibrio* isolates are highly virulent under culture conditions. A *Vibrio pelagius* strain recovered from larval mass mortality was highly pathogenic to larvae and post-larvae, with an LD50 below 5 bacteria mL^-1 in larvae, and the isolate was also able to grow in sterile seawater at room temperature or 15°C, indicating both strong host pathogenicity and environmental persistence. In parallel, *Aeromonas salmonicida* is an important cause of furunculosis-related losses in turbot farming, and infection triggers early up-regulation of pro-inflammatory and innate immune genes such as *tnf-α*, *il-1β*, *il-10*, and *c3*, showing that even early-stage bacterial challenge rapidly perturbs host immune homeostasis (Fajardo et al., 2023).

6.2 Viral and parasitic outbreaks

Viral diseases in turbot range from larval neurologic syndromes to emerging hemorrhagic conditions in grow-out fish. A picornavirus-like agent was associated with encephalomyelitis in turbot larvae, with large numbers of virus particles observed in the brain and medulla and with outbreaks ending in extremely heavy, ultimately complete mortality in affected batches. More recently, turbot acute hemorrhage disease in China was linked to a novel circovirus, and diseased stocks showed rapid spread, mortality exceeding 90% within one to two weeks, and increasing viral copy numbers after experimental infection (Figure 2) (Jiang et al., 2024).

Parasitic diseases are also major components of pathogen dynamics in stressed turbot populations. *Enteromyxum scophthalmi* causes a severe enteric disease characterized by cachexia, high morbidity, and mortality, while early infection is marked by interferon-related responses together with down-regulation of complement and acute-phase genes, suggesting active immune evasion during colonization (Spinos et al., 2024). Likewise, *Philasterides dicentrarchi* causes scuticociliatosis with systemic tissue invasion, including severe encephalitis, hepatic necrosis, branchial lesions, and muscular degeneration, confirming that parasitic outbreaks in turbot can progress beyond surface infestation to multisystemic disease.

6.3 Environmental drivers of disease transmission and virulence

Environmental stress does not simply weaken the host; it also changes transmission opportunity and pathogen virulence. A classic fisheries disease framework showed that infectious outbreaks arise when susceptible fish encounter virulent pathogens under stress caused by temperature, eutrophication, sewage, metabolic wastes, industrial pollution, or pesticides (Spinos et al., 2024). In turbot specifically, simultaneous environmental and biological stress can act synergistically: low water depth elevated cortisol and metabolic disturbance, and the additional presence of *A. salmonicida* or *P. dicentrarch*i under these conditions further amplified several stress responses.

Temperature is one of the clearest environmental drivers of bacterial virulence and outbreak timing. In fish-pathogenic bacteria, temperature regulates virulence gene expression, and in *Aeromonas hydrophila* lower host-