

Perkinsus infections. In Australia, bonamiasis in *Ostrea angasi* led to mass mortalities in a pilot native oyster industry in the 1990s, and a subsequent epizootic in 2015 was attributed to *Bonamia exitiosa*, with *Perkinsus olseni* also reported for the first time in this host, underscoring the potential for multiple protozoan agents to affect emerging aquaculture species (Bradley et al., 2025). Historical syntheses further identify *Roseovarius* oyster disease and disseminated neoplasia as additional important disease conditions in some regions, emphasizing that the pathogen spectrum in oyster aquaculture extends beyond vibrios and OsHV-1 to a diverse assemblage of protozoan and other agents whose distributions may shift with climate and industry expansion.

4 Environmental Drivers of Disease Occurrence in Oyster Farming

4.1 Effects of fluctuations in water temperature, salinity, and dissolved oxygen

Water temperature is a key driver of major oyster diseases, directly affecting both host susceptibility and pathogen replication. Controlled challenges with OsHV-1 μ Var showed mortality of Pacific oysters reaching 77 °C-84% at 22 °C-26 °C, but dropping to 23% at 18 °C and 0% at 14 °C, with a clear threshold between 14 °C-18 °C below which productive infection did not occur. Broader analyses indicate that climate-driven warming alters pathogen growth rates, spatial dispersion and host physiology, thereby increasing the severity and frequency of oyster disease outbreaks at global scale (Okon et al., 2023). Salinity and dissolved oxygen interact with temperature to modify disease risk. Experimental work on OsHV-1 showed that oysters acclimated to low salinity (10‰) had very high survival (>95%) after exposure, whereas those at 15-35‰ survived at only 43 °C-73%; however, non-acclimated oysters suffered 23% survival at 10‰, suggesting mortality from salinity shock rather than viral disease. In estuaries with diel-cycling hypoxia, oysters exposed to repeated low dissolved oxygen experienced increased acquisition and progression of *Perkinsus marinus* infections and reduced growth, implicating hypoxia-induced immune impairment as a mechanism enhancing disease susceptibility.

4.2 Role of eutrophication and water pollution in promoting disease

Nutrient enrichment and pollution reshape microbial communities in coastal habitats and can elevate pathogen loads relevant to oyster health. Along a eutrophication gradient in an urbanized estuary, oyster gut microbiomes showed functional shifts in nutrient-cycling genes that tracked local nutrient regimes, indicating that eutrophication alters oyster-associated microbial functions potentially linked to host physiology and disease risk (Figure 1) (Stevick et al., 2021). In seagrass sediments near nutrient sources, putative pathogen groups such as *Vibrio* spp. and *Pseudoalteromonas* spp. doubled in relative abundance compared with less enriched sites, suggesting that nutrient pollution can increase environmental reservoirs of bacterial pathogens affecting invertebrates and humans. More generally, climate-driven change and pollution jointly promote pathogen development and disease exposure in oysters. A global review highlights that climate change fosters proliferation of aquatic pathogens and harmful algal blooms, while pollution and other environmental burdens compound these pressures, disrupting oyster biochemical pathways and physiological functions and leading to more frequent outbreaks (Okon et al., 2023). Conceptual work on oyster disease further emphasizes that shifting environmental parameters-including nutrients and pollutants-can alter both oyster immunity and pathogen growth and virulence, often via changes in the microbiome that buffer or amplify disease expression.

4.3 Mechanisms by which extreme weather and sudden environmental changes trigger disease

Extreme events such as marine heatwaves and intense rainfall can rapidly push environmental conditions beyond oyster tolerance thresholds and trigger disease-linked mass mortalities. A 24-week experiment during a summer mortality event recorded three mortality phases in Pacific oysters; the transition to a sharp mortality increase coincided with heavy rainfall, a 13-day marine heatwave up to 27.7 °C, reduced salinity (34.6 to 31.4 psu) and increased *Vibrio* abundance, with mortality positively correlated to the heatwave and *Vibrio*, and negatively to salinity (Siboni et al., 2024). Experimental simulation of a marine heatwave (20 °C -25 °C) led to 77.4% mortality in *C. gigas*, whereas mortality dropped to 4.3% when antibiotics were added; heat stress caused large increases in *Vibrio harveyi* and *V. fortis* within the microbiome, indicating that sudden warming can trigger dysbiosis and opportunistic bacterial disease. Extreme precipitation and freshwater inflow can likewise induce large-scale mortality through prolonged low salinity and associated stress-disease interactions. After Hurricane Harvey, mean