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Characteristics of Disease Occurrence in Oyster Farming

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Abstract Oyster aquaculture is an important component of global marine fisheries, yet disease outbreaks have become a major constraint on its sustainable development. This paper systematically analyzes the characteristics of disease occurrence in oyster farming by integrating biological susceptibility, pathogen diversity, environmental drivers, transmission pathways, host immune responses, and epidemiological patterns. The results indicate that oysters are highly vulnerable to pathogenic infections due to their filter-feeding behavior and limited adaptive immune capacity. Bacterial diseases, particularly those caused by Vibrio species, are the most frequently reported, followed by viral, parasitic, and fungal infections. Environmental stressors such as elevated water temperature, salinity fluctuation, dissolved oxygen depletion, and eutrophication significantly increase disease incidence by weakening host immunity and promoting pathogen proliferation. High stocking density further accelerates disease transmission through waterborne pathways and aquaculture facility-mediated spread. In addition, microbial community dysbiosis plays a critical role in disease development and progression. A case study of intensive farming systems demonstrates that poor environmental management and inadequate biosecurity measures can lead to rapid disease outbreaks and substantial economic losses. Based on these findings, integrated disease management strategies combining ecological regulation, pathogen monitoring, and improved farming practices are proposed to enhance disease prevention and control in oyster aquaculture.

Keywords Oyster aquaculture; Disease occurrence; Pathogen dynamics; Environmental stress; Disease management

1 Introduction

Oyster aquaculture has become one of the most important components of global seafood production, expanding rapidly as demand for sustainable animal protein grows. Oysters are now among the most widely farmed molluscs, with several commercial species cultivated worldwide, and global output has climbed into the multi-million-tonne range as producers respond to rising consumer interest in nutritious seafood (Okon et al., 2023). Within this expanding sector, the Pacific oyster Crassostrea gigas dominates production and underpins the economies of many coastal regions; yet the industry remains highly vulnerable to disease, environmental change and management constraints that limit producers' ability to fully exploit growing markets (Botta et al., 2020; Petton et al., 2021). Over the past decade, infectious diseases have emerged as a central constraint on oyster farming, with repeated mass mortality events reported from major producing regions. In Pacific oysters, outbreaks associated with Ostreid herpesvirus 1 and Vibrio bacteria have intensified since 2008, increasing in both severity and geographic extent and making disease a particularly sensitive issue for producers (Alfaro et al., 2019). Pacific oyster mortality syndrome (POMS), driven by OsHV-1 µVar and subsequent bacterial dysbiosis, has become panzootic and now affects juvenile oysters in multiple countries, illustrating how complex host-pathogen-environment interactions can produce recurrent, large-scale losses (Petton et al., 2021). The economic and social consequences of these disease events are profound, with massive mortalities sometimes causing near-total stock losses and threatening the viability of farms and coastal communities. For POMS alone, annual juvenile mortality in France has been estimated to exceed 35% of cultivated and natural oysters, illustrating the scale at which disease can erode production and income. At the broader aquaculture scale, diseases across species are estimated to cost billions of dollars annually, yet economic assessments remain sparse, underscoring the need for more systematic evaluations of losses and of the benefits of control strategies in oyster systems (Maezono et al., 2025). Against this backdrop, there is a pressing need to better characterize the patterns and drivers of disease occurrence in real farming contexts so that risk can be

managed more effectively. Current knowledge shows that oyster disease expression is influenced by a suite of interacting host, pathogen and environmental factors-such as oyster age and genetics, temperature, food availability and microbiota-but the mechanisms by which these variables jointly control outbreaks remain poorly resolved. The present study therefore focuses on describing the characteristics of disease occurrence in oyster farming-across space, time, production stages and environmental gradients-with the dual objectives of identifying key risk factors and providing an empirical basis for improved surveillance and management strategies (Petton et al., 2021).

2 Biological and Ecological Basis of Oyster Disease Susceptibility

2.1 Oyster physiological characteristics and immune defense systems

Oysters rely entirely on a sophisticated innate immune system to survive in pathogen-rich estuarine environments, compensating for the absence of adaptive immunity. Hemocytes circulating in the hemolymph execute core defenses including immune recognition, signal transduction, synthesis of antimicrobial peptides, phagocytosis and encapsulation, supported by apoptosis and autophagy as important immune mechanisms (Wang et al., 2018). Single-cell transcriptomic analyses have revealed at least seven functionally distinct hemocyte types and multiple hematopoietic lineages, with specialized roles such as phagocytosis, reactive oxygen species production, copper accumulation and antimicrobial peptide expression, underscoring the cellular complexity behind disease resistance (De La Forest Divonne et al., 2025). Oyster immunity also shows plasticity and memory-like features that shape disease outcomes in farming. Immune priming and maternal immune transfer have been reported, suggesting that prior exposure can modulate subsequent responses, and neuroendocrine systems (catecholaminergic, cholinergic, neuropeptide and GABAergic) further regulate these defenses (Wang et al., 2018). Experimental work shows that inactivated Ostreid herpesvirus-1 and related viral antigens can rapidly stimulate hemocyte functions (e.g. ROS production, immune-related gene expression) without cytotoxicity, highlighting the potential to exploit innate "immune memory" and hemocyte responsiveness to reduce mortality from Pacific oyster mortality syndrome (Delisle et al., 2023).

2.2 Filter-feeding ecological habits and environmental exposure characteristics

As sessile filter feeders, oysters continuously pump large volumes of water, concentrating microorganisms and particles from their environment. An individual can retain up to 75% of microorganisms present in surrounding water, so naturally occurring bacterial communities, including potentially pathogenic taxa, accumulate in tissues and can pose health risks, particularly when animals originate from poor-quality environments (Mendes et al., 2023). Long-term monitoring at a commercial Pacific oyster area showed that, while oysters exhibited good depurating capacity for fecal bacteria (*E. coli*, enterococci) compared with surrounding waters, norovirus persisted seasonally in digestive glands, illustrating that filter feeding exposes oysters to diverse microbes and that clearance efficiency is pathogen-dependent (Rodrigues et al., 2023). Filter feeding also structures host-microbiota-pathogen interactions that influence disease susceptibility and food safety. Comparative analyses of water and oyster microbiota found that environmental conditions strongly shaped water communities and pathogen levels, whereas oyster digestive gland communities were more stable and showed "hot oyster" patterns, where individual oysters accumulated much higher Vibrio or fecal indicator loads than neighbors (Diner et al., 2023). In estuarine culture sites, surveys of Crassostrea gasar and surrounding waters have repeatedly detected human-pathogenic bacteria such as Escherichia coli, Salmonella, Vibrio spp. and others in both oysters and environment, confirming that filter-feeding oysters mirror-and can amplify-microbial risks associated with degraded water quality (Mendes et al., 2023).

2.3 Mechanisms linking environmental stress to disease susceptibility

Environmental stressors modulate both pathogens and host defenses, creating windows of high disease susceptibility in oyster farming. Large-scale field and modeling studies demonstrate that seawater temperature is a dominant driver of disease-induced mortality in Pacific oysters, with mortality risk sharply increasing between about 16 °C-24 °C and specific mean temperature thresholds (around 194 °C-24 °C over 12-21 days) corresponding to high probabilities of OsHV-1 outbreaks and substantial stock losses (Shi et al., 2024). A four-year, multi-site survival analysis similarly identified seawater temperature as the strongest predictor of mortality, with additional modulation by wind speed and humidity, and showed that spat were far more vulnerable than juveniles or adults, indicating that

environmental forcing interacts with life stage to shape disease patterns (Fleury et al., 2025). Other stressors, including salinity fluctuations, low flow and hypoxia, affect host-pathogen interactions and immune competence. Experimental manipulation of flow, temperature, salinity and dissolved oxygen showed that oysters in low-flow, high-sedimentation microhabitats had the greatest *Perkinsus marinus* infection prevalence, intensity and mortality, with disease responses significantly negatively correlated with flow, likely due to poorer physiological condition under chronic stress. At the cellular level, hypoxia experiments revealed that granulocytes under moderate low oxygen could transiently enhance phagocytosis and deploy antioxidant and mitophagy responses, but severe hypoxia led to ROS accumulation, mitochondrial dysfunction and impaired lysosomal degradation, suggesting that prolonged or intense hypoxia can overwhelm hemocyte resilience and thereby heighten disease susceptibility (Chen and Wang, 2025).

3 Major Pathogens and Disease Spectrum in Oyster Aquaculture

3.1 Bacterial diseases and the pathogenicity of Vibrio species

Bacterial diseases in oyster aquaculture are dominated by Vibrio spp., which occur as part of the normal microbiota but include lineages that cause severe mortality. Many Vibrio species from the Splendidus and Harveyi clades are frequently associated with bivalves, and several have been linked to mortality outbreaks as primary or opportunistic pathogens in cultured oysters (Destoumieux-Garzón et al., 2020. Even in non-intensive farming areas without recorded mass mortalities, Pacific oysters can naturally harbor potentially pathogenic Vibrio strains, indicating that virulence is present in the microbial community before overt disease is observed (Oyanedel et al., 2022). Specific pathogenic Vibrio species and lineages have been identified in large-scale mortality events. In China, mass summer mortality of Crassostrea gigas with over 60% losses was associated with Vibrio alginolyticus, whose virulence was confirmed experimentally and enhanced at higher temperatures, highlighting temperature-dependent pathogenicity (Wang et al., 2021; Yang et al., 2021). In Europe, Vibrio aestuarianus subsp. francensis emerged as a specialist oyster pathogen causing recurrent adult mortalities with ~25% mortality rates and shows genomic adaptations such as a copper-resistance island that may favor persistence in oyster hosts (Mesnil et al., 2023).

3.2 Viral diseases and their epidemiological characteristics

Viral infections, especially those caused by Ostreid herpesvirus 1 (OsHV-1) and its microvariants, are a major cause of disease in oyster aquaculture. OsHV-1 has been detected in at least 15 countries and is associated with massive mortalities in Crassostrea gigas, with microvariants such as OsHV-1 µVar driving Pacific oyster mortality syndrome (POMS) that has become panzootic (Alfaro et al., 2019). Descriptive epidemiology from the Hawkesbury River estuary in Australia showed that OsHV-1-associated mass mortalities affected all age classes but were most severe in spat and juveniles, with incubation periods of less than 4 days and evidence of subclinical infection months before overt mortality. Epidemiological studies highlight strong influences of temperature, host age and viral genotype on disease expression. Long-term sentinel monitoring in Australian estuaries found that OsHV-1 mortality typically began when mean water temperature exceeded ~20 °C, with consistent seasonal windows of high risk and spatial clustering at the scale of farms and even baskets. Experimental infections comparing OsHV-1 microvariants isolated between 2011 and 2015 in Australia showed significant reductions in virulence over time, with earlier isolates causing higher hazard of death and cumulative mortality, suggesting that phenotypic variation among genotypes may alter outbreak severity and offering prospects for management using low-virulence strains (Cain et al., 2021).

3.3 Types and impacts of parasitic and fungal diseases

Protozoan parasites of the genera Perkinsus and Haplosporidium constitute some of the most significant non-viral, non-bacterial threats in oyster aquaculture. In eastern oysters along the U.S. Atlantic and Gulf coasts, *Perkinsus marinus* (dermo) and Haplosporidium nelsoni (MSX) are prevalent and have been identified as causative agents of fatal diseases that have triggered mass mortalities and constrained population recovery. (Batchelor et al., 2023). Recent qPCR-based surveys in Georgia and Maine show very high prevalence of P. marinus and H. nelsoni (often >80 °C-90%), with complex relationships to oyster condition that may include emerging resistance or tolerance in some populations despite historical die-offs (Marquis et al., 2020; Batchelor et al., 2023). Other parasitic diseases, notably bonamiasis, have caused severe localized impacts in flat oyster industries and may co-occur with

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 µVar 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

oyster mortality in Galveston Bay rose from 11% to 48%, reaching 100% at some reefs; mortality was significantly correlated with the duration of bottom salinity below 5 psu, while storm-induced sediment deposition showed no such relationship, implicating extended low-salinity exposure as the main driver (Du et al., 2021). A similar pattern was observed when a series of atmospheric rivers produced extreme freshwater discharge into San Francisco Bay, causing sustained salinities below ~6.3 and a near-100% mass mortality of wild oysters, closely matching critical salinity tolerances and highlighting how rare events can wipe out already depleted populations.

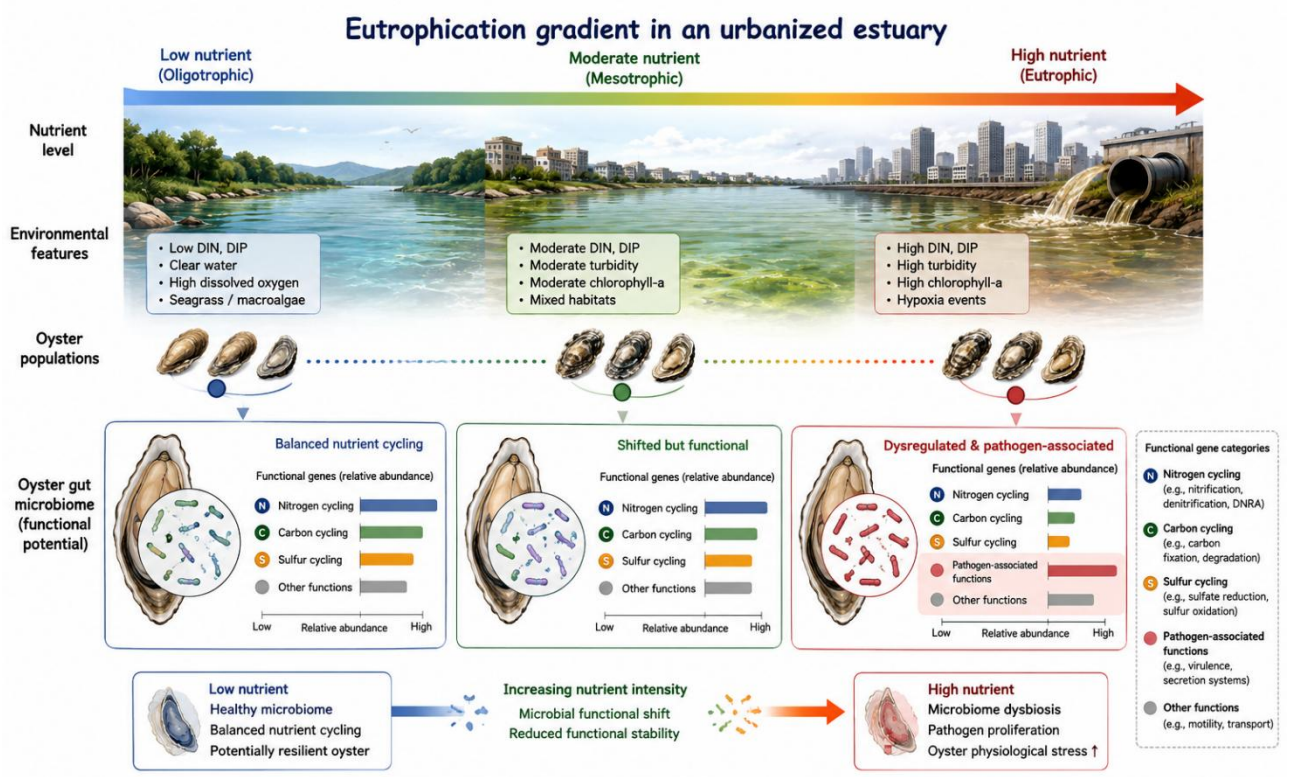

Figure 1 Changes in oyster gut microbiome functional profiles along an estuarine eutrophication gradient. Nutrient enrichment alters microbial functional genes associated with nutrient cycling, suggesting a link between eutrophication intensity, microbial dysbiosis, and potential host physiological stress

5 Disease Transmission and Epidemiological Characteristics

5.1 Waterborne transmission and density-dependent spread mechanisms

Experimental and modeling work for Vibrio aestuarianus shows clear waterborne transmission, with a basic reproduction number (R_0) around 2.9 and a generation time of ~5.5 days in small, closed oyster populations. (Lupo et al., 2020). Transmission is dose-dependent, driven by bacterial shedding and the concentration in seawater, implying threshold exposure levels needed to initiate an epidemic. At bay scale, hydrodynamic connectivity and temperature control the spatial spread of waterborne bacteria, allowing long-distance dispersal between distant farms over months. Theoretical models of marine infectious diseases further emphasize that highly infected oysters and the balance between pathogen release and removal in the water column determine outbreak thresholds and the existence (or not) of low-abundance refuges.

5.2 Transmission via aquaculture facilities and biological vectors

Recirculating and hatchery-like systems modify but do not eliminate waterborne viral circulation: OsHV-1 persists in seawater despite biofiltration and UV, and detection odds are higher where treatment is interrupted, showing facility design directly affects pathogen loads. Cohabitation experiments show higher donor biomass and feeding (algal addition) markedly increase OsHV-1 transmission and mortality, indicating facility management of stocking and feeding influences effective dose exposure. Field observations in Woolooware Bay show clustered OsHV-1 mortalities within farms and vertical stratification in the water column, consistent with infection from common

environmental sources shaped by local hydrodynamics and farm layout. Laboratory and epidemiological evidence support a role for particles and biofouling communities (e.g., plankton, fouled oysters on equipment) in transporting OsHV-1 and other pathogens, though successful transmission via fouling organisms appears rare and complex (Fuhrmann et al., 2021).

5.3 Risks associated with seedstock circulation and inter-regional transmission

Network analyses of French oyster farming show highly dynamic, heterogeneous transfer patterns among farm categories and growing sites, with seasonal peaks in movements and specific sites acting as high-risk hubs for becoming infected or transmitting disease (Coralie et al., 2016). Spatial epidemiology of OsHV-1 shows mortality starting in dense farming areas and spreading kilometres outward, consistent with animal movements and waterborne export from seedstock hotspots, while connectivity to farms is a strong mortality risk factor for both juveniles and adults (Gangnery et al., 2019). Historically, large-scale imports of Pacific oyster stock into Europe in response to disease crises created a positive feedback loop between introductions of non-native species (including pathogens) and further stocking, with continuing vector activity inferred over decades. More broadly, anthropogenic vector analysis identifies movements of live growing stock and culture substrates as among the highest-risk vectors in shellfish aquaculture networks, logistically difficult to manage but central to disease spread pathways across regions (Lovett et al., 2024).

6 Host Immune Response and Pathophysiological Mechanisms

6.1 Innate immune system and changes in disease-resistance responses

The oyster innate immune system relies on circulating haemocytes and a highly expanded repertoire of recognition and effector genes, enabling pathogen detection, phagocytosis, oxidative killing, antimicrobial peptide production and apoptosis (Wang et al., 2018). During bacterial or viral outbreaks, these defences can be subverted; for example, Vibrio aestuarianus deregulates hemocyte oxidative metabolism, impairing cellular functions and survival (De Lorgeril et al., 2018). Disease resistance varies strongly among families and life stages. Resistant oysters show higher basal expression of stress and antiviral pathway genes (TLR-NFκB, JAK-STAT, STING-RLR), supporting more robust responses to Pacific oyster mortality syndrome (POMS). In contrast, susceptible juveniles in field outbreaks sense pathogens and activate receptors and signaling, but fail to induce key antimicrobial, apoptotic and redox-homeostasis genes, leading to microbial overgrowth and high mortality.

6.2 Characteristics of tissue damage and metabolic disorders

Complex pathogenesis is well illustrated in POMS, where OsHV-1 µVar first infects haemocytes, inducing an immunocompromised state that permits secondary bacterial invasion and fatal bacteraemia (Destoumieux-Garzón et al., 2024). Histopathological observations in experimental infections show invasion of connective tissues by opportunistic bacteria during dysbiosis, consistent with systemic sepsis as a terminal lesion pattern. (Delisle et al., 2022; Destoumieux-Garzón et al., 2024) Metabolic reprogramming is another key feature of pathophysiology. During intense OsHV-1 replication, oysters show increased glycolysis and altered mitochondrial porin (VDAC) levels, resembling a “Warburg effect” that redirects energy metabolism. Other pathogens induce broad metabolic and cellular stress responses; Vibrio coralliilyticus infection in larvae reduces feeding, mobilizes energy reserves, remodels fatty acids, and activates antioxidant and heat-shock defences alongside immune pathways.

6.3 Mechanisms of gut and microbial community imbalance

Oysters host tissue-specific, dynamic microbiota that differ from surrounding seawater and are shaped by both environment and host genetics. In healthy states, this microbiota contributes to immune education and homeostasis; antimicrobial peptides help fine-tune microbial communities (Destoumieux-Garzón et al., 2024). Under stress or disturbance, rare phylotypes are lost, overall diversity declines, and the coupling between host genetics and microbiome composition breaks down, suggesting stress-driven community destabilization (Lokmer et al., 2016). Dysbiosis is now recognized as central to major syndromes such as POMS and environmentally driven mass mortalities. In POMS, OsHV-1-induced immune suppression leads to microbiota destabilization, replacement of commensals by virulent communities, and spill-over of opportunistic bacteria into tissues, amplifying disease

severity (King et al., 2018; Destoumieux-Garzón et al., 2024). Abiotic stressors such as low salinity or high temperature further disrupt digestive microbiota, increase pathogenic taxa (e.g. Vibrio), and are linked with inflammatory dysregulation and higher mortality during Vibrio infections (Li et al., 2022; Li et al., 2023). Oyster disease in farming systems emerges from the interplay of innate immune capacity, tissue-level damage, metabolic reprogramming and microbiome stability. Viral and bacterial pathogens exploit immune weaknesses and energy constraints, while environmental stressors destabilize microbial communities and inflammatory control, tipping oysters from resistance toward dysbiosis, systemic infection and mortality.

7 Case Study: Disease Outbreak Characteristics in Intensive Oyster Farming Systems

7.1 Analysis of typical disease outbreak processes in high-density farming areas

In intensive Pacific oyster farming areas, mass mortality episodes typically begin within farming zones and then spread rapidly across leases and into surrounding waters, highlighting the amplifying effect of high host density and farm clustering on infection pressure. Outbreaks often show strong spatial and temporal dependence, with disease fronts progressing from intensively used farming sectors toward less stocked or peripheral sites, consistent with local transmission driven by waterborne dispersal and farm connectivity (Figure 2) (Richard et al., 2021). Outbreak timing is tightly linked to seasonal windows when temperature favours pathogen replication, and juvenile cohorts in dense nursery structures are usually affected first, often experiencing very high losses within short periods. Husbandry operations such as handling and high stocking densities can further intensify outbreaks, with evidence that recent manipulation and dense basket systems increase mortality relative to lower-density rope culture or standard stocking levels (De Kantzow et al., 2017).

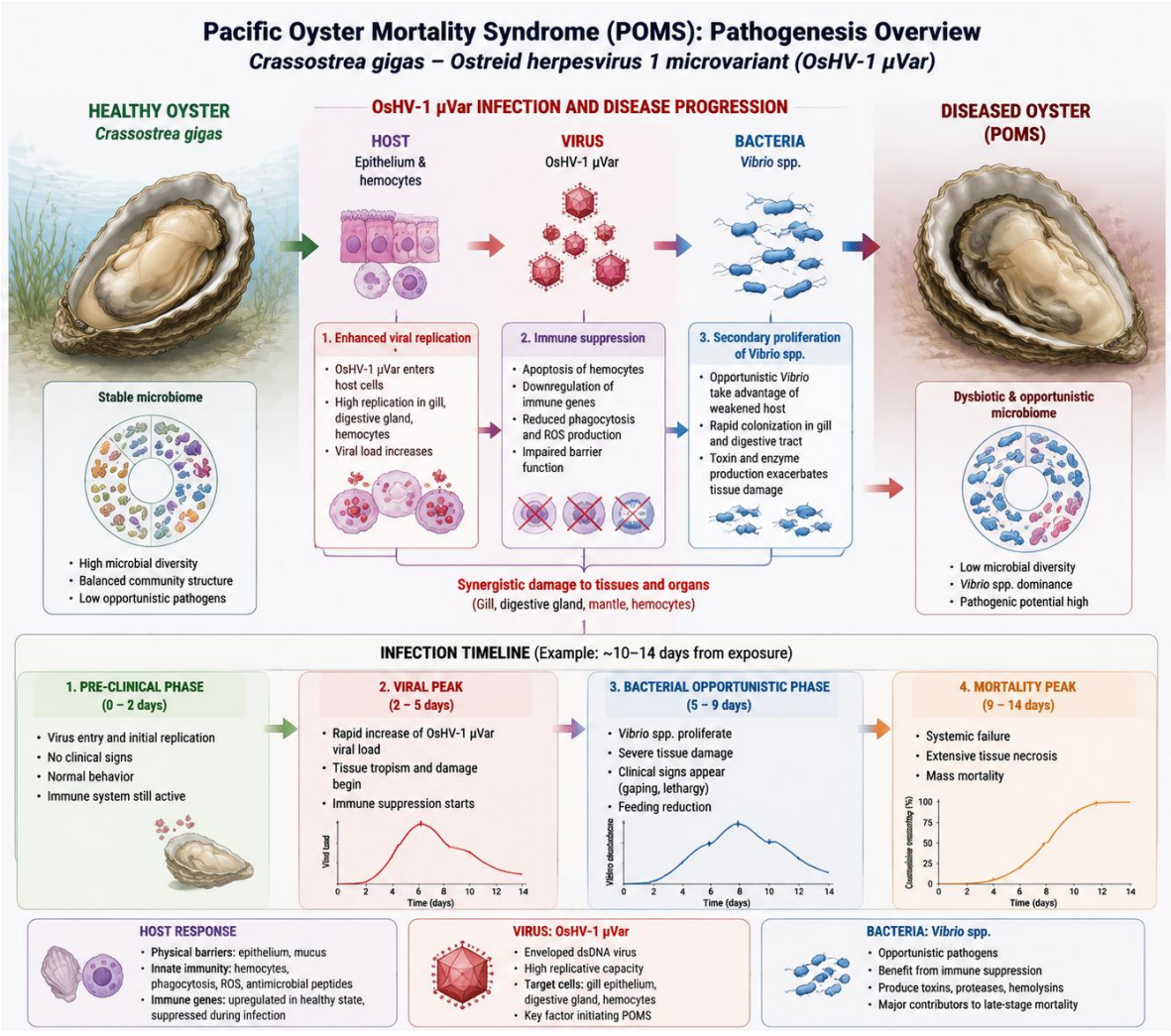

Figure 2 Pathogenesis cascade of Pacific Oyster Mortality Syndrome (POMS) in Crassostrea gigas

7.2 Identification of major pathogens and epidemiological dynamics

Across intensive systems, Pacific Oyster Mortality Syndrome (POMS) associated with Ostreid herpesvirus 1 microvariants (OsHV-1 µVar) is the dominant cause of recurrent mass mortalities of Crassostrea gigas since 2008, often followed by opportunistic bacterial infections (Pernet et al., 2016). Epidemiological surveys also frequently implicate Vibrio spp., particularly Vibrio aestuarianus and other pathogenic vibrios, as co-factors or primary agents in some mortality events, with pathogen diversity contributing to variable clinical outcomes (Alfaro et al., 2019). Outbreak dynamics often follow a sequence in which OsHV-1 titres rise in oyster tissues before visible signs, peak around the mortality maximum, and are accompanied by shifts in bacterial communities towards known pathogenic genera (Richard et al., 2021). Environmental factors such as temperature thresholds and confinement influence both virus activation and bacterial proliferation, while host age, size, and genetic background modulate susceptibility, creating complex interaction patterns in intensive farms.

7.3 Evaluation of control measure effectiveness and economic losses

Because curative treatments and vaccination are not feasible, control in intensive oyster systems relies on risk-based husbandry, spatial planning and biosecurity, including movement restrictions and zoning, which can reduce infection pressure but have not prevented the global spread of OsHV-1 µVar (Pernet et al., 2016). Adjustments in farming practices, such as optimizing stocking density, modifying culture structures or immersion regimes, and timing seeding to avoid high-risk temperature periods, have demonstrated reductions in mortality in some intensive settings. conomically, OsHV-1 microvariants have challenged the viability of Pacific oyster industries in both hemispheres, driving sector consolidation, loss of smaller farms, and shifts towards compensatory production strategies rather than purely technical disease solutions (Pernet et al., 2016; Fuhrmann et al., 2019). At a broader scale, marine diseases of farmed oysters and other aquaculture species impose economic burdens measured in billions of dollars annually, through direct losses, control costs, and regulatory compliance, underscoring the need for integrated, economically informed disease management in intensive oyster systems (Freitag et al., 2022).

8 Integrated Disease Management Strategies for Sustainable Oyster Farming

Ecologically informed farm placement and water-quality management can reduce disease risk. Spatial epidemiology of OsHV-1 shows that mortality is highest in inshore, high-biomass intertidal farming zones and decreases offshore, where better food quality and lower turbidity align with improved oyster health, indicating that maintaining good ecological status of coastal waters is a practical disease-mitigation strategy. Broader ecosystem-based perspectives argue that sustainable aquaculture must explicitly integrate organism health, environmental integrity, and human health, embedding disease control in wider ecosystem and food-system planning. Well-designed oyster culture can also act as a regulating service, filtering pathogens and reducing disease pressure on wild stocks when harvests occur before infected farmed oysters shed large quantities of parasites. At the farm scale, healthy-farming models emphasize carrying-capacity assessment, climate-change adaptation, and use of habitat features (e.g. mangroves, ecological floats) to buffer environmental stress and disease risk.

Selective breeding provides a powerful tool to reduce disease impacts where environmental control is limited. Reviews of breeding programmes show significant gains in resistance to major oyster pathogens (OsHV-1, Haplosporidium nelsoni, Roseovarius crassostreae, Marteilia sydneyi) after only a few generations, demonstrating that resistance is heritable and can be exploited to stabilize production. Family-based experiments in Pacific oysters confirm a strong genetic basis for resistance to OsHV-1 across life stages and show that improving OsHV-1 resistance does not compromise resistance to Vibrio aestuarianus, supporting multi-trait resistance breeding. Field mass-selection programmes have achieved dramatic survival improvements under OsHV-1 pressure, with selected lines of Crassostrea gigas reaching about 69% survival compared with 7% in controls after four generations, while also improving growth and yield. More recently, genomic selection has been shown to further accelerate gains in resistance to vibriosis, with genomic breeding values enabling discrimination between resistant and susceptible oysters and producing double-digit increases in survival and survival time in progeny, indicating a route to faster development of robust seedstock.

Effective integrated management requires surveillance systems designed for early detection rather than only proof of disease freedom. Expert-guided design for OsHV-1 highlights that observational (farmer-based) surveillance, when carefully structured and combined with targeted active sampling and strong industry-government partnerships, can greatly enhance system sensitivity and confidence in disease freedom, whereas active testing alone is unlikely to detect outbreaks in time. Farmer reporting is central to such systems; behavioural studies in France show that financial compensation, awareness of reporting objectives, and participatory education strongly influence whether and how quickly farmers notify unexplained mortalities, directly affecting early-warning performance. New molecular and environmental tools expand early-warning capacity. Suites of droplet digital PCR assays enable high-resolution, simultaneous quantification of multiple oyster-relevant pathogens in farm-proximal waters, revealing spatiotemporal hotspots where mortality risk increases and allowing farmers to adjust practices as pathogen loads rise. In parallel, temperature-based outlooks and spatial risk maps that integrate sea-temperature thresholds, turbidity and terrestrial inputs can guide targeted surveillance and proactive management of marine diseases, illustrating how environmental monitoring can be coupled with aquaculture health systems as oceans warm.

References

Alfaro A., Nguyen T.V., and Merien F., 2019, The complex interactions of Ostreid herpesvirus 1, Vibrio bacteria, environment and host factors in mass mortality outbreaks of Crassostrea gigas, Reviews in Aquaculture, 11(4): 1148-1168.
https://doi.org/10.1111/raq.12284

Batchelor S., Harrison J.S., Greiman S., Treible L.M., and Carroll J.M., 2023, Assessment of infection prevalence and intensity of disease-causing parasitic protozoans *Perkinsus marinus* and Haplosporidium nelsoni in Georgia oysters, Microorganisms, 11(7): 1808.
https://doi.org/10.3390/microorganisms11071808

Botta R., Asche F., Borsum J., and Camp E., 2020, A review of global oyster aquaculture production and consumption, Marine Policy, 117: 103952.
https://doi.org/10.1016/j.marpol.2020.103952

Bradley T., Mohr P.G., Humphrey J.D., Moody N., Cummins D., Slater J., and Crane M., 2025, Bonamia exitiosa: the cause of bonamiasis in native oysters Ostrea angasi in Australia in 2015, Diseases of Aquatic Organisms, 162: 99-113.
https://doi.org/10.3354/dao03853

Cain G., Liu O., Whittington R., and Hick P., 2021, Reduction in virulence over time in Ostreid herpesvirus 1 (OsHV-1) microvariants between 2011 and 2015 in Australia, Viruses, 13(5): 946.
https://doi.org/10.3390/v13050946

Chen Y., and Wang W.X., 2025, Immune resilience and subcellular adaptation of oyster hemocytes under hypoxic stress, Environmental Science and Technology, 59(31): 16332-16343.
https://doi.org/10.1021/acs.est.5c05254

De Kantzow M.C., Hick P., Dhand N., and Whittington R., 2017, Risk factors for mortality during the first occurrence of Pacific Oyster Mortality Syndrome due to Ostreid herpesvirus-1 in Tasmania, 2016, Aquaculture, 468: 328-336.
https://doi.org/10.1016/j.aquaculture.2016.10.025

De La Forest Divonne S., Pouzadoux J., Romatif O., Montagnani C., Mitta G., Destoumieux-Garzón D., Gourbal B., Charrière G., and Vignal E., 2025, Diversity and functional specialization of oyster immune cells uncovered by integrative single-cell level investigations, eLife, 13: RP102622.
https://doi.org/10.7554/elife.102622

De Lorgeril J., Escoubas J., Loubière V., Pernet F., Le Gall P., Vergnes A., Aujoulat F., Jeannot J., Jumas-Bilak E., Got P., Guéguen Y., Destoumieux-Garzón D., and Bachère E., 2018, Inefficient immune response is associated with microbial permissiveness in juvenile oysters affected by mass mortalities in the field, Fish and Shellfish Immunology, 77: 156-163.
https://doi.org/10.1016/j.fsi.2018.03.027

De Lorgeril J., Petton B., Lucasson A., Perez V., Stenger P., Dégremont L., Montagnani C., Escoubas J., Haffner P., Allienne J., Leroy M., Lagarde F., Vidal-Dupiol J., Guéguen Y., and Mitta G., 2020, Differential basal expression of immune genes confers Crassostrea gigas resistance to Pacific oyster mortality syndrome, BMC Genomics, 21(1): 63.
https://doi.org/10.1186/s12864-020-6471-x

Delisle L., Laroche O., Hilton Z., Burguin J., Rolton A., Berry J., Pochon X., Boudry P., and Vignier J., 2022, Understanding the dynamics of POMS infection and the role of microbiota composition in the survival of Pacific oysters Crassostrea gigas, Microbiology Spectrum, 10(6): e01959-22.
https://doi.org/10.1128/spectrum.01959-22

Delisle L., Rolton A., and Vignier J., 2023, Inactivated Ostreid herpesvirus-1 induces an innate immune response in the Pacific oyster Crassostrea gigas hemocytes, Frontiers in Immunology, 14: 1161145.
https://doi.org/10.3389/fimmu.2023.1161145

Destoumieux-Garzón D., Canesi L., Oyanedel D., Travers M., Charrière G., Pruzzo C., and Vezzulli L., 2020, Vibrio-bivalve interactions in health and disease, Environmental Microbiology, 22(10): 4323-4341.
https://doi.org/10.1111/1462-2920.15055

Destoumieux-Garzón D., Montagnani C., Dantan L., Nicolas J., Travers M.-A., Duperret L., Charrière G., Toulza E., Mitta G., Cosseau C., and Escoubas J., 2024, Cross-talk and mutual shaping between the immune system and the microbiota during an oyster's life, Philosophical Transactions of the Royal Society B: Biological Sciences, 379(1901): 20230065.
https://doi.org/10.1098/rstb.2023.0065

Diner R.E., Zimmer-Faust A., Cooksey E., Allard S., Kodera S., Kunselman E., Garodia Y., Verhougstraete M., Allen A., Griffith J., and Gilbert J., 2023, Host and water microbiota are differentially linked to potential human pathogen accumulation in oysters, Applied and Environmental Microbiology, 89(7): e00318-23.
https://doi.org/10.1128/aem.00318-23

Du J., Park K., Jensen C., Dellapenna T., Zhang W., and Shi Y., 2021, Massive oyster kill in Galveston Bay caused by prolonged low-salinity exposure after Hurricane Harvey, Science of the Total Environment, 774: 145132.
https://doi.org/10.1016/j.scitotenv.2021.145132

Fleury E., Mat A.M., Petton S., and Pernet F., 2025, Unraveling multifactorial risks in Pacific oyster mortality: a four-year study of environmental and age-related impacts, Aquaculture Environment Interactions, 18: 1-13.
https://doi.org/10.3354/aei00512

Freitag A., Ellett A.N., Burkart H., and Jacobs J., 2022, Estimating the economic burden of Vibrio parahaemolyticus in Washington State oyster aquaculture: implications for the future, Journal of Shellfish Research, 40(3): 555-564.
https://doi.org/10.2983/035.040.0312

Fuhrmann M., Castinel A., Cheslett D., Nozal F.D., and Whittington R., 2019, L'impact des microvariants du virus herpétique ostreid herpesvirus 1 sur la culture d'huîtres creuses dans les hémisphères Nord et Sud depuis 2008, Revue Scientifique et Technique (OIE), 38(2): 491-509.
https://doi.org/10.20506/rst.38.2.3000

Fuhrmann M., Georgiades E., Cattell G., Brosnahan C., Lane H., and Hick P., 2021, Aquatic pathogens and biofouling: pilot study of Ostreid herpesvirus 1 translocation by bivalves, Biofouling, 37(9-10): 949-963.
https://doi.org/10.1080/08927014.2021.1985474

Green T., Vergnes A., Montagnani C., and De Lorgeril J., 2016, Distinct immune responses of juvenile and adult oysters (*Crassostrea gigas*) to viral and bacterial infections, Veterinary Research, 47(1): 72.
https://doi.org/10.1186/s13567-016-0356-7

King W., Jenkins C., Seymour J., and Labbate M., 2018, Oyster disease in a changing environment: decrypting the link between pathogen, microbiome and environment, Marine Environmental Research, 143: 124-140.
https://doi.org/10.1016/j.marenvres.2018.11.007

Li X., Shi C., Yang B., Li Q., and Liu S., 2023, High temperature aggravates mortalities of the Pacific oyster (*Crassostrea gigas*) infected with Vibrio: a perspective from homeostasis of digestive microbiota and immune response, Aquaculture, 568: 739309.
https://doi.org/10.1016/j.aquaculture.2023.739309

Li X., Yang B., Shi C., Wang H., Yu R.-H., Li Q., and Liu S., 2022, Synergistic interaction of low salinity stress with Vibrio infection causes mass mortalities in the oyster by inducing host microflora imbalance and immune dysregulation, Frontiers in Immunology, 13: 859975.
https://doi.org/10.3389/fimmu.2022.859975

Lokmer A., Kuenzel S., Baines J., and Wegner K.M., 2016, The role of tissue-specific microbiota in initial establishment success of Pacific oysters, Environmental Microbiology, 18(3): 970-987.
https://doi.org/10.1111/1462-2920.13163

Lovett B., Cahill P., Fletcher L., Cunningham S., and Davidson I., 2024, Anthropogenic vector ecology and management to combat disease spread in aquaculture, Environmental Management, 73(4): 895-912.
https://doi.org/10.1007/s00267-023-01932-8

Lupo C., Dutta B.L., Petton S., Ezanno P., Tourbiez D., Travers M., Pernet F., and Bacher C., 2020, Spatial epidemiological modelling of infection by Vibrio aestuarianus shows that connectivity and temperature control oyster mortality, Aquaculture Environment Interactions, 12: 511-527.
https://doi.org/10.3354/aei00379

Maezono M., Nielsen R., Buchmann K., and Nielsen M., 2025, The current state of knowledge of the economic impact of diseases in global aquaculture, Reviews in Aquaculture, 17(3): e70039.
https://doi.org/10.1111/raq.70039

Marquis N., Bishop T.J., Record N., Countway P., and Robledo J.F.A., 2020, A qPCR-based survey of Haplosporidium nelsoni and Perkinsus spp. in the eastern oyster Crassostrea virginica in Maine, USA, Pathogens, 9(4): 256.
https://doi.org/10.3390/pathogens9040256

Mendes D.C.S., Rodrigues D.T., Gomes H.M., Lenz T., Silva C.M., and Antonio Í., 2023, Pathogens and microorganisms in the mangrove oyster Crassostrea gasar cultivated in an estuarine environment in Northeast Brazil, Brazilian Journal of Biology, 83: e272789.
https://doi.org/10.1590/1519-6984.272789

Mesnil A., Jacquot M., Garcia C., Tourbiez D., Canier L., Bidois A., Dégremont L., Cheslett D., Geary M., Vetri A., Roque A.I.V., Furones D., Garden A., Orozova P., Arzul I., Sicard M., Charrière G., Destoumieux-Garzón D., and Travers M., 2023, Emergence and clonal expansion of Vibrio aestuarianus lineages pathogenic for oysters in Europe, Molecular Ecology, 32(11): 2869-2883.
https://doi.org/10.1111/mec.16910

Okon E., Birikorang H.N., Munir M., Kari Z., Téllez-Isaías G., Khalifa N., Abdelnour S., Eissa M.E.H., Al-Farga A., Dighiesh H., and Eissa E.H., 2023, A global analysis of climate change and the impacts on oyster diseases, Sustainability, 15(17): 12775.
https://doi.org/10.3390/su151712775

Oyanedel D., Rojas R., Brokordt K., and Schmitt P., 2022, Crassostrea gigas oysters from a non-intensive farming area naturally harbor potentially pathogenic *Vibrio strains*, Journal of Invertebrate Pathology, 191: 107856.
https://doi.org/10.1016/j.jip.2022.107856

Pernet F., Lupo C., Bacher C., and Whittington R., 2016, Infectious diseases in oyster aquaculture require a new integrated approach, Philosophical Transactions of the Royal Society B: Biological Sciences, 371(1689): 20150213.
https://doi.org/10.1098/rstb.2015.0213

Petton B., Destoumieux-Garzón D., Pernet F., Toulza E., De Lorgeril J., Dégremont L., and Mitta G., 2021, The Pacific Oyster Mortality Syndrome, a polymicrobial and multifactorial disease: state of knowledge and future directions, Frontiers in Immunology, 12: 630343.
https://doi.org/10.3389/fimmu.2021.630343

Richard M., Rolland J., Guéguen Y., De Lorgeril J., Pouzadoux J., Mostajir B., Bec B., Mas S., Parin D., Le Gall P., Mortreux S., Fiandrino A., Lagarde F., Messiaen G., Fortune M., and D'Orbcastel E.R., 2021, In situ characterisation of pathogen dynamics during a Pacific oyster mortality syndrome episode, Marine Environmental Research, 165: 105251.
https://doi.org/10.1016/j.marenvres.2020.105251

Rodrigues I.C., Santos-Ferreira N., Silva D.L.D., Da Silva C.C., Inácio Â.S., Nascimento M., and Da Costa P.M., 2023, A one-year systematic study to assess the microbiological profile in oysters from a commercial harvesting area in Portugal, Microorganisms, 11(2): 338.
https://doi.org/10.3390/microorganisms11020338

Shi J., Kajtar J.B., Hayashida H., and Ugalde S., 2024, Relationships between high temperatures and Pacific oyster disease and mortality in southeast Tasmania, Australia, Continental Shelf Research, 273: 105173.
https://doi.org/10.1016/j.csr.2024.105173

Siboni N., King W.L., Williams N.L., Scanes E., Giardina M., Green T.J., Ostrowski M., O'Connor W.A., Dove M.C., Labbate M., and Seymour J.R., 2024, Increased abundance of potentially pathogenic Vibrio and a marine heatwave co-occur with a Pacific oyster summer mortality event, Aquaculture, 583: 740618.
https://doi.org/10.1016/j.aquaculture.2024.740618

Stevick R.J., Post A., and Gómez-Chiarri M., 2021, Functional plasticity in oyster gut microbiomes along a eutrophication gradient in an urbanized estuary, Animal Microbiome, 3(1): 5.
https://doi.org/10.1186/s42523-020-00066-0

Wang H., Yang B., Li X., Li Q., and Liu S., 2021, Screening of bacterial pathogens associated with mass summer mortality of the Pacific oyster *Crassostrea gigas* in China, Aquaculture Reports, 20: 100672.
https://doi.org/10.1016/j.aqrep.2021.100672

Wang L., Song X., and Song L., 2018, The oyster immunity, Developmental and Comparative Immunology, 80: 99-118.
https://doi.org/10.1016/j.dci.2017.05.025

Yang B., Zhai S., Li X., Tian J., Li Q., Shan H., and Liu S., 2021, Identification of Vibrio alginolyticus as a causative pathogen associated with mass summer mortality of the Pacific oyster Crassostrea gigas in China, Aquaculture, 535: 736363.
https://doi.org/10.1016/j.aquaculture.2021.736363