Reviews in Aquaculture

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Global Warming Affects the Pathogenesis of Important Fish Diseases in European Aquaculture

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ABSTRACT

Global warming remains a neglected environmental challenge for the sustainability of primary production, particularly aquaculture, which is highly susceptible to the spread of established pathogens and the induction of emerging infectious diseases under warming conditions. Over the past decade, Europe has experienced dramatically high temperatures that may impact both farmed fish and their pathogens in a largely unpredictable manner. While, in general, warming may boost the rate of disease transmission and its virulence by increasing pathogens' fitness in weakened hosts, some diseases characteristic of cooler environments may become rare. Field data is still largely fragmented, but in vitro experiments reveal that almost 28 microbial diseases in European finfish farming could be facilitated by climate warming. Innovative mitigation tools, such as fish selective breeding, epigenetic programming, the development of new vaccines, and alternative treatments, may prove essential in coping with the effects of rising water temperatures on fish diseases in Europe.

1 | Introduction

1.1 | The Increase in Temperature in European Water Bodies

Climate change (CC) is undoubtedly the most significant environmental challenge facing the world today, resulting in long-lasting environmental changes from the tropics to the poles [1]. Global warming (GW) is perhaps the best-known and most severe effect of climate change (CC), representing one of the main long-term drivers of economic, social, and environmental changes that affect all types of primary production, including aquaculture [2]. Global warming raises air [3] and sea surface temperatures [4], with water temperature increases projected to reach between 1°C and 4°C by the year 2100 [5]. The European temperature is already ~2.3°C above the pre-industrial average and 0.8°C above its 1991–2020 average [6], affecting seas, lakes, and rivers [7] at a faster rate than in other continents. For example, temperatures in the Mediterranean Sea were significantly over average during the last decade, with anomalies reaching 5°C [8]. This region, a semi-enclosed basin with low water exchange, appears exceptionally susceptible [9], with a warming rate of 0.6°C per decade, compared to the average ocean warming of 0.1°C [10].

Moreover, European lakes are facing a significantly faster temperature shift of 0.5°C per decade [8], leading also to high evaporation.

1.2 | The Effect of Global Warming on Aquaculture

While the effects of global warming seem detrimental in all production sectors, they can be more complex in aquaculture than in livestock due to the greater variety of species produced, as well as the diminished ability to control farming conditions [11]. The general effects of GW on aquaculture have been reviewed in recent years at both regional and global levels and therefore will not be discussed here [11–17]. Direct and indirect GW drivers are now regarded as responsible for changes in aquaculture at both short- and long-term levels [18]. Short-term changes include the loss of production and infrastructure due to extreme events, increased disease outbreaks, and the emergence of alien pathogens, as well as increased incidences of toxic algal tides, as short-term consequences. In contrast, water chemistry changes, scarcity of wild marine seed, and limited access to materials from marine and terrestrial sources, decreased productivity and eutrophication are some of the long-term consequences [18].

Although GW has emerged as a serious threat to most developmental and production activities [15], it is essential to consider that the predicted impacts will not necessarily be negative in all cases. Due to an uneven distribution of GW across the globe, some regions will experience potentially detrimental changes, while others may suffer less impactful changes or even potential improvements. Similarly, aquaculture is not practiced uniformly throughout the world, and this heterogeneity must be considered to assess the possible impacts of GW objectively. The varied climatic regimes, environments, and broad range of farmed taxa, with different vulnerabilities to climate effects, present a serious challenge in devising mitigation policies. For example, most aquaculture production is concentrated in inland freshwaters, mainly in the tropical and subtropical regions of Asia [19], where the GW effects are considered relatively low [20], as the rise in water temperatures tends not to exceed the optimal range for cultured species [18]. In contrast, open water systems, such as cage fish farming, the predominant practice in European and global marine aquaculture, are expected to suffer more from the GW effects, and adaptive measures will necessitate the introduction of improved technologies to withstand extreme weather events [11, 21].

Considering that temperature plays a critical role in the performance of aquatic farmed species, including growth and reproduction, increasing water temperatures may significantly impact aquaculture practices in temperate zones by exceeding the optimal temperature range of the species currently cultured [18]. The cost of aquaculture is already being affected, as GW is impacting the supply of fishmeal and fish oil [22], increasing the feed price and thus the overall cost of the final products.

Diseases of cultured fish induced by various types of pathogens are inevitably affected by changing thermal regimes, but in a largely unpredictable manner. The impact of rising temperatures on fish diseases remains to be seen. Higher temperatures are known to increase the virulence of specific pathogens [23]. In addition, reduced resistance due to stress and immunosuppression in farmed animals, as well as consequent increased disease transmission, further aggravate the situation. Therefore, it has been suggested that the challenges related to diseases in cultured fish induced by various types of pathogens will be inevitably exacerbated by a changing thermal regime, and warmer conditions may facilitate the establishment of exotic diseases [24].

The higher-than-global-average warming of European air and surface water may disproportionately affect its primary production. European farmed fish production in open surface waters remains a growing industry (2,865,072 tons in 2022) [25], with marine cold-water species representing approximately 70% of total production, freshwater species making up 14%, and marine Mediterranean species (warm-water) accounting for 16%. Norway remains the dominant producer in Europe, particularly in the marine environment, with more than 50% of the total supply (Figure 1), mainly consisting of Atlantic salmon (*Salmo salar*) and large rainbow trout (*Oncorhynchus mykiss*). Other major European farmed fish species cultured in saltwater

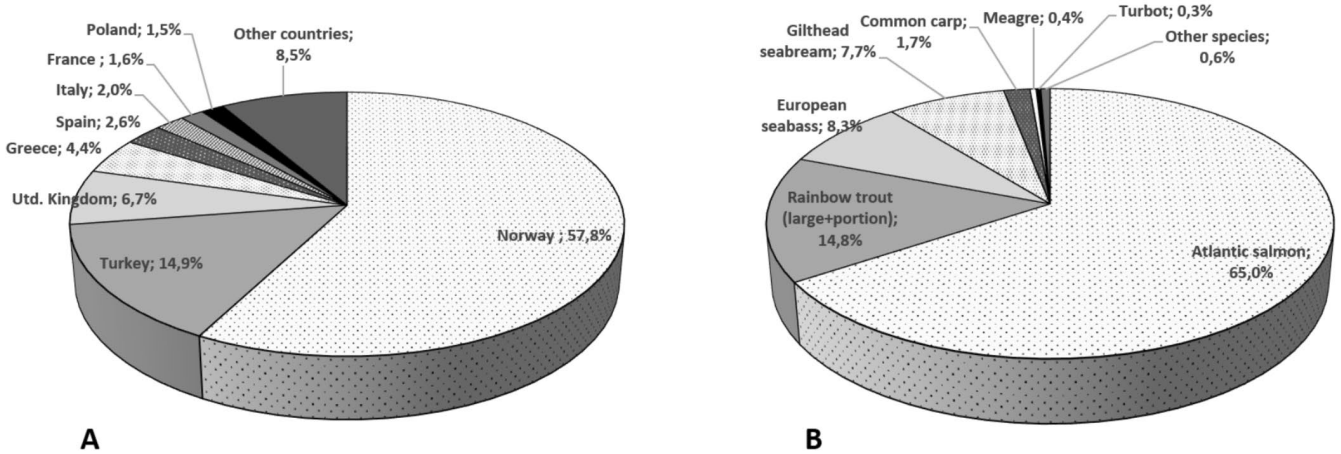

FIGURE 1 | A. Leading aquaculture production countries in Europe; B. European farmed fish production percentage per species (modified from FEAP 2023; https://feap.info/index.php/data/).

include gilthead seabream (*Sparus aurata*), European seabass (*Dicentrarchus labrax*), meagre (*Argyrosomus regius*), and turbot (*Scophthalmus maximus*) (Figure 1). Common carp (*Cyprinus carpio*), and rainbow trout farmed in land facilities represent the most commercialized farmed finfish European species in freshwater. In addition, the Senegalese sole (*Solea senegalensis*), Atlantic bluefin tuna (*Thunnus thynnus*), and greater amberjack (*Seriola dumerili*) represent emerging marine farmed finfish species that may potentially be affected by GW.

Global effects of CC and GW have been previously reviewed for many aquaculture aspects [11, 13, 14, 26–27], therefore, this study primarily focuses on two currently neglected aspects of disease in aquaculture: the impact of increased temperature on (a) pathogen incidence and (b) the pathogenesis of important diseases infecting both the most commercialized, as well as emerging farmed fish species in European aquaculture. This information will help farmers, veterinarians, and policymakers develop a joint health management strategy against GW-influenced diseases in European finfish farming.

2 | Literature Search and Data Collection

Given the broad scope of the topic on climate effects on diseases in aquaculture, where specific, exact filters may not be fully effective, and considering the goal of capturing as much relevant literature as possible, we employed the following strategy. As neither Scopus nor Web of Science databases utilize well-curated controlled vocabularies, we used the MeSH thesaurus, maintained by the National Library of Medicine, and searched PubMed. From the Basic Veterinary List of 123 journals, we found that PubMed currently indexes 78% of journals, with only 14 journals being relevant to the topic and not indexed (the journals on veterinary law, economics, or terrestrial animals were not considered). In our search, performed in April 2023 we combined MeSH terms and subheadings with specific-field criteria (to enhance sensitivity). The search strategy is described in detail in a Supporting Information entitled 'complete search strategies'. Overall, the search strategy was structured around three main lines of inquiry, with an additional AND term introduced to increase specificity, followed by the application of exclusion criteria.

1. Fish relevant to aquaculture: The initial list of species was compiled by experts in the field (GR, FPB, IM). The search criteria included both scientific and commercial names of identified species, and allowed for singular and plural forms.
2. Relevant climate effects: We conducted a comprehensive search of the MeSH thesaurus to identify terms related to climate effects. The terms mapped in the MeSH were then used to construct field-specific criteria, for example, "Temperature [MeSH] OR Temperature [Title/Abstract] OR Temperature [OT]"
3. Diseases: To identify studies addressing outbreaks, or emerging pathogens and diseases we used terms such as *outbreak*, or combined keywords *emerging* or *new* with etiological keywords (e.g., *pathogen*, *virus*, *parasite*, *bacteria*, and *disease*) using Boolean operators (AND, OR) across title, abstract, and authors' keyword fields.
4. After combining the first three queries as (#1 AND (#2 OR #3)), we further increased the specificity of the retrieved records by requiring that all hits include general pathogen-related terms such as *parasite*, *virus*, or *bacteria*, or general keyword such as *disease*; and their derivates.
5. Exclusion: Finally, to further increase specificity of findings we excluded papers exclusively referring to unrelated topics such as pollution or fish consumption.

We replicated the search on the OVID platform, which enabled searches across multiple databases, including AGRICOLA, a database maintained by the National Agricultural Library, and Evidence-Based Reviews that may also cover veterinary topics. The platform can map MeSH terms to corresponding subject headings or keywords within other databases, ensuring more comprehensive results. The last search was performed in October 2025, with no limit of language, year of publication, or study type limitations. Overall, the search strategy was structured around three main lines of inquiry, with an additional AND term introduced to increase specificity, followed by the application of exclusion criteria. The complete search strategy, including all search queries and the way they were combined, is presented in Table S1. We then conducted a hand search of reference lists of included studies, relevant journals and conference proceedings to identify additional studies that may not have been indexed in databases. In total 560 relevant records were retrieved, including mainly journal articles, but also books and book sections, technical reports, and conference papers.

The use of non-EU examples in Section 5 suggests that there are no equivalent studies within the EU.

3 | Global Warming Is Affecting the Fish and the Environment in Aquaculture

Most fish species are considered ectotherms, or more commonly described as "cold-blooded animals," as their internal metabolic activity is unable to produce enough heat to control their internal body temperature [28]. There are a few notable exceptions, such as partially homeothermic fish (like moonfish) or mesothermic fish (like tunas and swordfish), which have a partial ability to regulate their internal body temperature. The inability to produce enough heat through their metabolism has significant repercussions, as in most fish species, the body's internal temperature is the same as the surrounding water temperature. This specific characteristic has significant effects on their metabolism and may also impact various physiological functions, including growth, feeding, respiration, reproduction, and behavior. As fish inhabit aquatic habitats with a wide range of temperatures, they have developed adaptive allostatic responses to maintain their homeostatic ranges. However, the capacity to build and maintain these responses is limited and specific to each fish species. When environmental conditions, especially temperature, exceed their allostatic capacities, the physiological functions and resilience of the fish are compromised. This is typically observed under thermal stress, with adverse effects of specific temperature profiles on the immune system, leading to lower resilience and a higher risk of disease.

However, environmental temperature also has a substantial impact on the physico-chemical properties of water, as well as several water parameters that have a direct interaction with fish physiology. Among the most relevant ones, temperature strongly affects the solubility of dissolved gases, primarily oxygen, as well as carbon dioxide and nitrogen. In general, lower temperatures increase gas solubility; therefore, increases in water temperature associated with GW reduce oxygen availability for living organisms. Temperature increase can also increase the toxicity of several chemicals in fish, mainly freshwater [29]. Additionally, GW increases the rates of water evaporation, resulting in higher salinities and mineral concentrations in the marine environment. This consequently alters the hardness in freshwater, with relevant collateral effects on the solubility of gases and pH of the water. In some cases, GW may also cause unpredictable heavy rains in certain areas, resulting in the opposite effects.

Additionally, the impact of increased water temperature on both microscopic and macroscopic aquatic organisms should not be overlooked. Temperature is one of the main drivers for the development of most microscopic (bacteria, microalgae, aquatic microfauna) and macroscopic plant, algal and animal communities, including primary or opportunistic pathogenic species, harmful species (toxic microalgae, jellyfish), as well as predators and other species whose presence in the vicinity of the farm may cause stress to cultured species [30–32].

Lastly, an essential aspect to consider is the direct impact of temperature on the fish's immune system. Much scientific work has clearly demonstrated the effect of temperature modulation of the immune response of fish [33]. A transient increase in temperature can have a positive impact on the clearance of infections by inducing what is called environmental fever where fish voluntarily migrate to water with a higher temperature [34]. But in contrast, acute and chronic thermal stress has been recognized as relevant factors associated with diseases, poor health, and compromised welfare in fish [35]. GW may induce a shift in the magnitude of temperature and exposure length to high temperatures, which clearly increases the risk of diminished health and welfare conditions and, consequently, the risk of disease in aquaculture and wild fish populations.

4 | Effects of Global Warming on Pathogens and Disease Epidemiology in Aquaculture

The introduction of new aquaculture species, the intensification of production systems, and GW are the main concerns for the sustainable development of modern aquaculture, particularly in terms of the emergence and frequency of disease outbreaks. The transmission of established infectious diseases and the emergence of new pathogens are both affected by GW. However, although most host-pathogen systems are expected to experience more frequent disease impacts with warming, pathogens that favor lower water temperatures may decline with warming [36, 37]. Nonetheless, GW can directly affect the biology of the etiological agent, but also importantly, the host susceptibility [38] by directly inducing stress-driven immunosuppression, or indirectly by deteriorating the quality of the farming environment (e.g., higher accumulation of particulate matter, hypoxia, etc.). Notably, aquatic animals infected with microbial pathogens mostly show higher mortality at higher temperatures [37, 39–40], therefore, the increase in global average temperature over this century is an alarming risk factor affecting the frequency of disease outbreaks in farmed aquatic animals. All pathogen groups, including viruses, bacteria, fungi, and parasites, may be affected by GW, although in varying ways.

4.1 | Viral Pathogens

Viruses, being ultimately "parasitic" particles hijacking the host cells for replication, are highly dependent on temperature in poikilothermic organisms such as fish. The incidence and frequency of viral infections in farmed aquatic animals tend to increase with GW in some cases, resulting in economic consequences (e.g., fish mortalities and accumulation of uneaten fish feed) on a global scale [39]. Based on the ranges of the optimum water temperatures associated with outbreaks of the most important viral infections in European aquaculture (Table 1), it is assumed that increasing water temperatures may not directly affect the onset of viral diseases, as many viral pathogens prefer relatively low temperatures. However, GW can shift the season of viral outbreaks, as seen in the case of several viral diseases affecting farmed European salmonids. Additionally, the increased thermal stress leading to a deficiency in the effectiveness of immune responses of cold-water fish hosts, combined with higher host susceptibility to co-infections with other microbial groups (e.g., bacteria or parasites), facilitated by the viral primary infection, cannot be disregarded in the overall impact. Viruses are often underestimated effectors in complex diseases in fish [62]; changing water temperatures can shift or exacerbate the occurrence of these diseases by affecting the viral component, making diagnosis and treatment more challenging.

4.1.1 | Viruses Affecting Marine Farmed Fish Species

Infectious Salmon Anaemia Virus (ISAV) (Orthomyxoviridae) is a virus that affects farmed Atlantic salmon. It is transmitted horizontally, as well as through water and farm equipment [63]. Infection with ISAV is a World Organisation for Animal Health (WOAH) listed infection and is notifiable within the EU, and the European Economic Area (EEA), which includes Norway, the largest Atlantic salmon producer facing challenges from the ISAV. The virus is adapted to cold-water salmonids and shows optimum growth at $<15°C$. Excluding the fact that increasing coastal temperatures may physiologically alter the optimal environment and the physiology of farmed Atlantic salmon, it is unlikely that pathogen virulence will be enhanced by GW. Indeed, during a challenge trial with Atlantic salmon, viral load and fish mortality due to ISAV exposure were significantly higher at 10°C compared to 20°C [46].

Viral hemorrhagic septicemia virus (VHSV) (Rhabdoviridae) is one of the most severe finfish pathogens worldwide in terms of the width of the host range, pathogenicity, disease course, and mortality rates [64]. The disease was first documented in the 1930s in Europe in rainbow trout and has also been listed in WOAH, being notifiable within the EU zone. VHSV is the biggest threat to rainbow trout aquaculture, but it is also one of the most threatening pathogens affecting farmed turbot in Europe

TABLE 1 | Optimum temperatures for the emergence of viral fish pathogens affecting important European fish species.

Viruses	Host	Replication temperature-lab (°C)	Production system	Optimum water temperature (°C)	References
Marine water					
Infectious haematopoietic necrosis virus	Rainbow trout			8–15	Enzmann et al. [41] and Dixon et al. [42]
Infectious pancreatic necrosis virus	Atlantic salmon	15–22		10–15	Dopazo [43]
	Rainbow trout (fresh)				Ørpetveit et al. [44]
Infectious salmon anemia virus	Atlantic salmon	15	Challenge facilities	10	Falk et al. [45] and Groves et al. [46]
Lymphocystis virus	Gilthead seabream	25	Floating cages, pre-fattening facilities	22	Kvitt et al. [47] and Labella et al. [48]
Nervous necrosis virus	European seabass, gilthead seabream	25	Floating cages, hatcheries, and pre-fattening facilities	20–25	Vendramin et al. [49], Pereiro et al. [50], and Toffan et al. [51]
Piscine myocarditis virus	Atlantic salmon		Floating cages	8–10	Rennemo et al. [52] and Rodger et al. [53]
Piscine orthoreovirus	Atlantic salmon Rainbow trout		Floating cages Challenge facilities	10–12 5	Sørensen et al. [54]
Salmon pancreas disease virus	Atlantic salmon	15	Floating cages	10–15	Jansen et al. [55]
	Rainbow trout				Jarungsriapisit et al. [56]
Viral hemorrhagic septicemia virus	Senegalese sole	12	Challenge facilities	22	Souto et al. [57]
Fresh water					
Carp edema virus	Common carp		Ponds	15–25	Pikula et al. [58]
Koi herpesvirus	Common carp	15–25		16–25	Michel et al. [59]
Sleeping disease virus	Rainbow trout	10		10	Villoing et al. [60]
Spring viremia of carp virus	Common carp	20	Ponds	10–17	Ahne et al. [61]

[65] and elsewhere, as neither prevention nor therapy is commercially available [66]. The susceptibility of turbot to VHSV has been demonstrated since the 80s [67]. Mortalities usually appear when water temperature is lower than 15°C [68], therefore, the ideal temperature for experimental challenges of turbot has been set at 10°C [65]. At higher water temperatures, it takes a short course with moderate cumulative mortality in rainbow trout, and the virus survives for more extended periods in the environment at 4°C compared to 20°C [69], therefore, it is unlikely that GW will enhance the incidence of VHSV.

Infectious pancreatic necrosis virus (IPNV) (Birnaviridae) has a significant impact on cultured salmonids (including Atlantic salmon and rainbow trout) worldwide, mainly affecting juvenile fish. It has been considered the most widespread virus, infecting more than 60 different species of fish, mollusks, and crustaceans [70]. It can survive in aquatic environments for a considerable time; therefore, it is among the most resilient viruses recognized [71]. Predisposing factors for IPNV outbreaks, apart from age and stress, also include water temperature. The maximum mortalities may occur at various temperatures, whose effects may be obscured by the strain of the virus and the host species [72]. However, IPNV is a ubiquitous virus that has been isolated from both cold- and warm-water environments, despite the virus's optimum temperature being reported as between 10°C and 15°C in salmonids [43]. This indicates a high adaptability of the virus to a wide range of water temperatures and salinities (0‰–40‰), suggesting that IPNV incidence may be affected by GW in specific hosts and environments.

Salmon pancreas disease virus (SPDV) (Togaviridae), often referred to as salmonid alphavirus (SAV), causes pancreas disease (PD) in European salmonids. It is highly contagious and affects both Atlantic salmon and rainbow trout reared in sea.

Clinical signs of PD include anorexia, lethargy, and increased fecal casts [73]. The optimal temperature for the virus coincides with the winter and spring periods (6°C–10°C), along with high salinity and minimal organic load [74]. The viability of SAV is inversely correlated with temperature, such that the lowest infectivity of the virus is observed at 16°C [56]. However, although warmer water temperatures seem less favorable for virus viability, stress-related immunosuppression in susceptible hosts may lead to increased virus replication and SPDV outbreaks [75]. Consequently, rising water temperatures due to GW may indirectly favor the virulence of SAV in farmed salmonids.

Piscine orthoreovirus (PRV) (Reoviridae) is an emerging virus that infects Atlantic salmon, causing heart and skeletal muscle inflammation (HSMI). Disease occurrence has been reported during all seasons, with spring and early summer being the most common seasons for PRV outbreaks. Challenge trials with PRV in Atlantic salmon are typically conducted at 12°C [76]. Another genotype of PRV, which primarily infects rainbow trout and was first discovered in Norway in 2013 [77] has been isolated lately in Denmark [78]. The decrease in water temperature (5°C) appears to enhance the replication of this genotype and the extent of heart pathology in experimentally infected rainbow trout [54], suggesting that its virulence is inversely related to water temperature rise. Therefore, GW could potentially affect the incidence and virulence of different PRV genotypes infecting farmed salmonids in different ways.

The piscine myocarditis virus (PMCV) (Totiviridae) is the causal agent of cardiomyopathy syndrome (CMS), a severe cardiac disease of farmed Atlantic salmon. The PMCV outbreaks typically occur in the second farming year [53] following stressful events such as handling, net cleaning, lice treatment, weather changes, or other diseases [52]. The virus is transmitted horizontally among marine cages and farms, while vertical transmission has also been suspected [79]. So far, there is no clear indication of temperature-related effects on virulence in the pertinent literature, although experimental trials have been conducted at 10°C [80].

Infectious hematopoietic necrosis virus (IHNV) (Rhabdoviridae) is a causative agent of a widespread disease of farmed salmonids in continental Europe and elsewhere. Horizontal transmission of IHNV is typically achieved through direct exposure; however, invertebrate vectors may play a role in some cases. Water temperature is a crucial environmental factor affecting IHNV outbreaks. While clinical disease occurs between 8°C and 15°C under natural conditions, experimental infection typically occurs at water temperatures below 12°C [81]. In addition, IHNV persists for a shorter time at warmer temperatures (15°C) and results in an overall lower fish mortality compared to lower temperatures (< 10°C) [82]. Consequently, it seems unlikely that GW will directly affect IHNV outbreaks in susceptible farmed fish in Europe.

Nervous necrosis virus (NNV) (Nodaviridae) is the causative agent of the most important viral nervous necrosis disease (VNN) affecting farmed marine fish in the Mediterranean region. VNN infects fish in all production phases, but it is especially severe in larval and juvenile stages, causing up to 100% mortality [83]. Farmed European seabass (mainly on-growing) is the most susceptible species since the first report of the disease decades ago [49]. More recently, the disease has become an emerging problem in farmed gilthead seabream, primarily affecting the early stages [84]. Typical signs in the infected fish include irregular swimming, anorexia, lethargy, with internal manifestations of brain congestion and swim bladder hyperinflation. The outbreaks in European seabass occur at high water temperatures (> 25°C), typically in late summer or early autumn [85]. Although losses in gilthead seabream are also observed at lower temperatures, these are not as prevalent [84]. NNV is also affecting farmed *Senegalese sole* [57, 86] with optimum water temperature during experimental infections reported to be 22°C. NNV replication is a composite process regulated by both the genetics of the viral strain and water temperatures, suggesting that clinical disease and fish losses are more apparent at higher temperatures (25°C–30°C) [51]. Given the versatility of NNV species and genotypes in infecting different hosts over a wide temperature range (18°C–28°C), it is plausible that the GW might increase the virulence of this pathogen and support its spread over new geographic areas and host species.

Lymphocystis disease virus (LCDV) (Iridoviridae) is a cosmopolitan virus that affects more than 140 species of marine and freshwater fish [87]. The disease caused by LCDV is a chronic and self-limiting pathology described in cultured gilthead seabream. Healing from the disease occurs in a temperature-dependent manner. The infection is contracted horizontally through contaminated water [88], although vertical transmission has also been demonstrated [89]. The progression of the disease depends on the host and the water temperature; outbreaks usually appear at water temperatures > 22°C [90]. This, coupled with the fact that the course of the disease is more rapid at warmer water temperatures, suggests that rising water temperatures due to GW might increase the virulence and incidence of outbreaks of LCDV in farmed gilthead seabream.

4.1.2 | Viruses Affecting Freshwater Farmed Fish Species

Sleeping disease virus (SDV), a variant of SAV (Togaviridae), causes a syndrome in farmed freshwater rainbow trout that has been observed in Europe over the last few decades [60]. SDV replicates at low temperatures (Metz et al. 2011), so that the natural outbreaks appear almost exclusively at water temperatures around 10°C, possibly also affected by the temperature-compromised efficiency of the immune system. In contrast to SPDV, the assumption that water temperatures higher than those characterized as optimal for fish may induce stress-related immunosuppression and consequently increase susceptibility to this SAV variant has not been investigated. Thus, it is unclear whether higher water temperatures may directly favor the virulence of SDV in rainbow trout.

Koi herpesvirus (KHV) (Alloherpesviridae) is a highly contagious viral pathogen of the common carp [91] and other cyprinids, with mortality rates sometimes reaching 80%–100% [92]. The disease caused by KHV, which was first reported in 1999, has spread throughout the world, becoming a WOAH-listed infection and notifiable within the EU region. The optimal water temperatures for KHV outbreaks range from 16°C to

25°C. However, higher water temperatures may be effective in reducing fish mortality [93] and the risk of viral dissemination, which is higher at relatively low temperatures (16°C) [94]. Based on the available data, it is unlikely that the GW and consequent increase in water temperatures will increase the virulence of the virus and trigger more severe outbreaks in common carp.

Spring viremia of carp virus (SVCV) (Rhabdoviridae) causes the homonymous disease affecting cyprinids, with farmed common carp being the most susceptible. The disease is notifiable by the WOAH. The virus can be spread by fomites and parasitic invertebrates, as well as fish-eating birds inhabiting carp ponds. High mortality occurs at water temperatures between 10°C and 17°C, but the mortality rate decreases at higher temperatures (www.cfsph.iastate.edu), during which infected carp can develop an efficient immunity response and neutralize the virus. Therefore, it is unlikely that SVCV outbreaks in European farmed carp will increase with rising water temperatures.

Carp edema virus (CEV) (Poxviridae), is one of the significant threats to common carp in European aquaculture [95] and elsewhere. CEV can cause massive mortalities between 15°C and 25°C during infections with CEV genogroup II [96], while outbreaks of CEV genogroup I may also occur at lower water temperatures [58]. The disease exhibits a seasonal biphasic pattern, being frequently detected in spring and early summer, with a second peak in the fall. Mortality events correlate with changes in water temperature [97]. However, the disease is not exacerbated during the high summer temperatures, suggesting that it is unlikely that increasing water temperatures caused by GW will increase CEV virulence and incidences of the disease. The existence of two genogroups of the virus with clearly different temperature optima may indicate that the virus has the ability to adapt to changing temperature conditions and may continue to threaten carp as inland water temperatures rise [97–99].

The above information suggests that the relationship between temperature and the onset of viral pathogens in European finfish farming remains complex, often case-specific, and further research is required to understand the mechanisms better. In most cases, especially for viruses affecting cold-water farmed fish species, viral incidence and virulence are likely to decrease as water temperature increases. However, the evidence from warm-water environments suggests that increases in temperature outside the normal range may directly promote warm-water viral outbreaks and/or compromise the immune system of farmed fish [33], making them more vulnerable to viral infection.

4.2 | Bacterial Pathogens

Like all living organisms, bacterial pathogens are constantly exposed to various environmental challenges, including GW. One of the relevant factors influencing the biology of bacteria is the water temperature [100]. All bacterial species have an optimal temperature range for growth and replication, outside of which their survival is dramatically reduced. Temperature adaptation may also be an essential evolutionary event in bacterial ecology, affecting membrane-associated functions and consequent changes in bacterial gene expression, which can lead to altered virulence [101]. In farmed fish, a key environmental stress factor in outbreaks of most bacterial fish diseases is water temperature. However, increasing water temperatures do not always favor bacterial disease incidences. In some cases (mainly cold-water bacterial diseases), outbreaks occur when the water temperature drops to a certain value, while in others, it is the opposite [100]. An increase in the temperature of bacterial culture can directly enhance bacterial virulence (e.g., damselysin cytotoxin) [102]; however, the optimal temperature used for the laboratory culture of a pathogen can vary considerably compared to the temperature at which the disease occurs in the field (Table 2). In fact, the environmental temperature that causes the disease in a susceptible host tends to be lower than the optimal growth temperature of the pathogen in the laboratory [103]. The optimum temperatures for the emergence of crucial bacterial fish pathogens in European finfish farming ([126–129]; https://www.eurl-fish-crustacean.eu/) are given in Table 2. The most relevant bacterial pathogens are further discussed.

4.2.1 | Bacteria Affecting Marine Water Farmed Fish Species

Aeromonas salmonicida (Aeromonadaceae) is a gram-negative bacterial fish pathogen and the etiological agent of furunculosis in several farmed fish species, including salmonids. Even though the disease was initially diagnosed in farmed Atlantic salmon in Norway as early as the 1980s, it remains a significant threat to the development of intensive Atlantic salmon farming [130]. Similarly, furunculosis still causes considerable problems in rainbow trout farmed in sea cages in Denmark, although it was initially described in freshwater rainbow trout in Denmark several decades ago [131]. Growth of *A. salmonicida* above 20°C may lead to plasmid rearrangements and other alterations, such as the loss of the A-layer protein or secreted proteolytic activity [132], however, the outbreaks in farmed rainbow trout appear during stress-associated periods with elevated temperatures during the summer period (20°C) [105]. Similar epizootics were observed during warm periods in farmed salmon in Finland [133]. Thus, it is likely that GW may increase the incidence of furunculosis outbreaks in farmed salmonids in Europe.

Renibacterium salmoninarum (Micrococcaceae) is a gram-positive facultative intracellular and cosmopolitan bacterium causing bacterial kidney disease at low temperatures in salmonids, including rainbow trout and Atlantic salmon [125]. Salmonids in temperate and cold-water areas are susceptible to the disease at temperatures ranging from 7°C to 15°C [134]. Although the disease develops slowly, its progress depends on environmental factors such as water temperature. Indeed, cooler water temperatures (12°C) contribute to the progression of infection and increased transmission of the disease in challenged salmon [135]. Therefore, rising water temperatures caused by GW may not directly affect the onset of bacterial kidney disease in farmed salmonids in Europe.

Lactococcus spp. (Streptococcaceae) are gram-positive bacterial pathogens causing piscine lactococcosis, which affects many fish species and causes important economic losses in both marine and freshwater aquaculture [136]. *L. garvieae* has traditionally been considered the primary species responsible

TABLE 2 | Optimum temperatures for growth and emergence of important bacterial fish pathogens in farmed European fish species.

Bacteria	Host	Growth temperature (°C)	Production system	Optimum water temperature (°C)	References
Marine Water					
Aeromonas salmonicida	Atlantic salmon	20–25		11–15	Uddin et al. [103]
	Rainbow trout		Floating cages		Boily et al. [104] and Pedersen et al. [105]
Aeromonas veronii	European seabass	35–37	Floating cages	> 21	Smyrli et al. [106]
Lactococcus garvieae	European seabass	> 23	Inland farm		Salogni et al. [107]
	Gilthead seabream	> 18	Floating cages, land-based tank farms		Esposito et al. [108]
Mycobacterium marinum	European seabass, gilthead seabream	20–30	Floating cages	23–25	[109]
Photobacterium damselae subsp. *piscicida*	Gilthead seabream, European seabass, meagre	22–28	Floating cages	> 18	Romalde [110] and Varvarigos [111]
Tenacibaculum maritimum	European seabass, gilthead seabream, turbot, Senegalese sole	15–34	Floating cages, Land-based facilities-tanks	15–20	Kolygas et al. [112], Piñeiro-Vidal et al. [113], Vilar et al. [114], and Mabrok et al. [115]
Vibrio anguillarum	Several marine and fresh water farmed fish spp.	25–30	Numerous production systems	> 15	Lages et al. [116] and Frans et al. [117]
Vibrio harveyi	European seabass, gilthead seabream	22–25	Challenge facilities	> 20	Firmino et al. [118]
	Greater amberjack		Challenge facilities		Minami et al. [119]
Fresh water					
Aeromonas hydrophila	Common carp	25–26	Ponds	22–32	Uddin et al. [103] and Semwal et al. [120]
Flavobacterium psychrophilum	Rainbow trout	15–21	Challenge facilities	< 10	Holt et al. [121] and Uddin et al. [103]
Lactococcus garvieae	Rainbow trout	37		> 15	[122] and [123]
Pseudomonas fluorescens	Common carp	30		< 12	Uddin et al. [103]
	Rainbow trout				Pekala-Safinska [124]
Renibacterium salmoninarum	Atlantic salmon, Rainbow trout	15–18		< 15	Delghandi et al. [125]

for causing disease; however, *L. petauri*, a newly identified species, has also been implicated in field outbreaks in rainbow trout [137]. Both species cannot be distinguished by routine diagnostic methods, resulting in their misidentification [138]. Recently, *L. garvieae* was identified as the etiological agent of mortalities in gilthead seabream at temperatures above 18°C [108]. Losses due to the same bacterium have also been recorded in European seabass at temperatures above 23°C [107]. Since increasing water temperature appears to be a key variable for *L. garvieae* outbreaks, GW may further trigger incidences of marine lactococcosis in finfish species farmed in the Mediterranean.

Vibrio anguillarum (Vibrionaceae) is perhaps the most cosmopolitan gram-negative bacterial pathogen, being pathogenic to a variety of farmed fish, crustaceans, and bivalves, and affecting more than 90 susceptible aquatic organisms [139]. *V. anguillarum* causes hemorrhagic septicemia in a wide temperature range, affecting cold- and warm-water farmed fish species. Atlantic salmon, rainbow trout, European seabass, gilthead seabream, and meagre are the relevant European farmed fish susceptible to this bacterium [140, 141]. Infections are contracted through the skin and the oral intake of the pathogen through contaminated water or food [117]. Chemical stress and the density of fish population are essential factors that induce the disease, but sudden temperature alterations are the most crucial parameter for its onset, occurring at temperatures above 15°C [142]. The degree of virulence of *V. anguillarum* peaks around 15°C [116], although the severity of disease caused by *V. anguillarum* is multifactorial in a temperature-dependent manner and in response to iron levels. Thus, an increase in water temperatures due to GW in the different environments where *V. anguillarum* causes vibriosis may affect differently susceptible finfish species.

V. harveyi (Vibrionaceae) is a gram-negative bacterial pathogen of several marine fish and invertebrates. It is a major concern for farmed fish, especially in the Mediterranean region, as it is currently becoming the principal cause of vibriosis [143], mainly in the more susceptible European seabass and gilthead seabream [118]. Other European farmed fish, such as rainbow trout and Atlantic salmon, are also susceptible to vibriosis caused by *V. harveyi* [144]. The bacterium is also causing epizootics at inland pre-ongrowing tanks and cages hosting the Mediterranean greater amberjack (unpublished observations) and its Japanese counterpart [119]. The most severe incidences in the western Mediterranean occur during the warmest months [143], suggesting that rising water temperatures due to GW may affect the onset of the disease at least in the Mediterranean region. Indeed, elevated temperatures (30°C) promote the expression of many virulence genes of *V. harveyi* (lytic enzymes, components of the T3SS secretion system, and iron-chelating compounds), potentially increasing its pathogenicity, although negatively influencing its survival, indicated by a loss of in vitro cultivability [145].

Aeromonas veroni (Aeromonadaceae) is a Gram-negative bacterium that has emerged as a serious concern, causing severe pathology and mortality in European seabass farmed in the eastern Mediterranean [106]. Outbreaks occur during the warm months of the year, when water temperature is over 21°C, and peak during the summer period when temperatures range between 24°C and 26°C [106]. Since *A. veroni* outbreaks seem to be favored by rising water temperatures, GW may directly affect *A. veroni*-related epidemics in European seabass.

Photobacterium damselae subsp. *piscicida* (Vibrionaceae) is a gram-negative bacterial pathogen responsible for pseudotuberculosis (pasteurellosis) in a variety of farmed fish species (Austin [142]). Gilthead seabream, the most susceptible species in European aquaculture, especially at juvenile stages, is primarily affected over summer temperatures (25°C–26°C) [146]. However, the disease later diagnosed in European seabass also occurs at lower water temperatures (18°C–19°C) [147]. In contrast to the gilthead seabream, European seabass is more susceptible after the nursery stage and during the on-growing phase. Meagre has also shown susceptibility to *P. damselae* subsp. *piscicida* under experimental in vivo challenge infection [148]. Pasteurellosis is clearly a temperature-dependent disease, with outbreaks in cages occurring at warmer water temperatures. Therefore, GW and increasing water temperatures may lead to increased incidence of pasteurellosis in Mediterranean farmed fish.

Tenacibaculum maritimum (Flavobacteriaceae), formerly known as *Flexibacter maritimus*, is a gram-negative opportunistic bacterium responsible for tenacibaculosis, an ulcerative disease causing high mortalities in various marine fish species worldwide [115]. Water temperatures exceeding 15°C have been linked to mortality due to *T. maritimum* in the eastern Mediterranean [149]. The same serotype has been isolated from gilthead seabream [112, 150]. The severity of the disease has also been evident during natural infections of farmed flatfish [113, 114], and *T. maritimum* virulence in turbot and Senegalese sole was demonstrated in experimental infections [151, 152]. However, the causative isolate was antigenically and genetically different from the common *T. maritimum* serotype. In addition to poor management conditions, water temperature plays a key role in *T. maritimum* outbreaks. A significant rise in the severity and incidence of tenacibaculosis has been reported at increasing water temperatures (> 15°C) and salinities (> 30‰) [153], although winter outbreaks of tenacibaculosis have also been documented [154]. Therefore, it is unclear what effect GW might have on the incidence of tenacibaculosis in European farmed fish.

Mycobacterium marinum (Mycobacteriaceae) is a Gram-positive, opportunistic pathogen that causes mycobacteriosis (piscine tuberculosis) in freshwater and marine species. Notably, *M. marinum* is one of the most common atypical mycobacteria that can cause human opportunistic infection [155]. Although there have been some sporadic incidences of the disease in meagre [156], European seabass is the most susceptible euryhaline species, for which the first outbreak was reported during the 1990s in the eastern Mediterranean at 24°C [157]. Later events of mycobacteriosis were reported in both European seabass and gilthead seabream, in the eastern Mediterranean at high water temperatures (25°C) [109]. *M. pseudoshottsii* has also been identified as the etiological agent for losses in European seabass, gilthead seabream, and red drum (*Sciaenops ocellatus*) during warm periods [158, 159]. Since high water temperatures appear to trigger the onset of mycobacteriosis in Mediterranean farmed fish,

increasing temperatures due to GW may enhance the incidence in Mediterranean aquaculture.

4.2.2 | Bacteria Affecting Freshwater Farmed Fish Species

R. salmoninarum (Micrococcaceae) is a gram-positive facultative intracellular and cosmopolitan bacterium causing bacterial kidney disease at low temperatures in salmonids, including rainbow trout and Atlantic salmon [125]. Salmonids in temperate and cold-water areas are susceptible to the disease at temperatures ranging from 7°C to 15°C [134]. Although the disease develops slowly, its progress depends on environmental factors such as water temperature. Indeed, cooler water temperatures (12°C) contribute to the progression of infection and increased transmission of the disease in challenged salmon [135]. Therefore, rising water temperatures caused by GW may not directly affect the onset of bacterial kidney disease in farmed salmonids in Europe.

Flavobacterium columnare (Flavobacteriaceae) is a Gram-negative bacterium that causes columnaris disease in fish. The disease exhibits an acute to chronic form, has a worldwide freshwater distribution, and infects many different fish species, including salmonids and common carp [160]. Outbreaks due to *F. columnare* are commonly observed in infected salmonids during warm temperatures [161], characterized by skin lesions, fin erosion, and gill necrosis. In addition to the virulence of the strain, which appears to be a key factor in the development of columnaris in susceptible cold-water and temperate fish, age also plays a significant role [162]. Moreover, the virulence of *F. columnare* and associated fish mortalities in experimentally infected salmonids increase progressively with increasing temperature, with losses peaking at 20°C [163]. Even higher temperatures (23°C) have been associated with losses in salmonids [160, 164]. Consequently, GW-induced rising water temperatures may cause increased incidences and losses due to columnaris in European farmed salmonids.

F. psychrophilum (Flavobacteriaceae) is a ubiquitous Gram-negative bacterium (mainly found in freshwater) and the etiological agent of cold water disease and rainbow trout fry syndrome [165]. *F. psychrophilum* can also cause disease in non-salmonid fish, such as eel (*Anguilla anguilla*) and three species of cyprinids, that is, common carp, crucian carp (*Carassius carassius*), and tench (*Tinca tinca*) in Europe [166]. Infection may occur both horizontally and vertically [167]. The disease typically occurs at water temperatures below 16°C, and is most severe at <10°C [168]. Therefore, rising water temperatures due to GW are unlikely to affect epizootics of the disease in European farmed rainbow trout; in contrast, it can act against the development of the disease.

Pseudomonas fluorescens (Pseudomonadaceae) is a Gram-negative bacterial pathogen common in aquatic and other environments [169], reported from a wide range of fish species [170], including common carp and rainbow trout. The bacterium belongs to the group of psychrophiles, which typically develop diseases at low water temperatures (<10°C). However, *P. fluorescens* was also isolated in a co-infection with *Yersinia ruckeri* during an outbreak in farmed rainbow trout during the summer period [171], and in a concurrent episode in farmed common carp in early spring [172]. Thus, although *P. fluorescens* is commonly considered a psychrophilic pathogen, it can cause outbreaks even during warm periods; hence, it is unclear to what degree GW will affect the future incidence of *P. fluorescens* infection in farmed rainbow trout and common carp.

A. hydrophila (Aeromonadaceae) is a freshwater Gram-negative, mostly opportunistic bacterium that causes disease in fish, amphibians, reptiles, birds, and mammals (Austin & [142]), indicating the ability to infect a wide variety of homeothermic and poikilothermic hosts. In farmed common carp, it causes hemorrhagic septicemia, resulting in significant losses [173]. Water temperature plays an essential role in the development of this disease: infections below 12°C remain asymptomatic, while clinical signs become evident above 22°C [174]. Thus, it is possible that the rising water temperature may favor epidemics of the disease.

Lactococcus garvieae is responsible for multiple outbreaks in European farmed rainbow trout [122]. Typically, the disease produces hyperacute and hemorrhagic septicemia, and early symptoms of infection include anorexia, melanosis, and erratic swimming [175]. Water temperature is a predominant factor in the development of the disease, which emerges at temperatures above 15°C during the warm months [136]. The most acute outbreaks in rainbow trout occur at water temperatures above 18°C [176]. Therefore, increasing water temperatures due to GW may directly influence the incidence and severity of piscine lactococcosis in farmed rainbow trout.

The collected data indicate that the relationship between increasing water temperatures due to GW and outbreaks of bacterial diseases in European finfish farming is, similar to the majority of disease scenarios, a multi-complex event, being both host- and environment-specific. Additional evidence, supported by the experimental challenges, is needed to gain a deeper understanding of this interaction. In several cases, bacterial outbreaks are favored by increasing water temperatures (examples of bacterial pathogens are listed in Table 3); however, the possibility of a lack of effect or even a negative impact cannot be dismissed. As in the majority of disease outbreaks, the effects of temperature increase beyond the normal range of the poikilothermic fish, along with the consequent immunosuppression, should be considered in the complex interaction of the changing environment-host-pathogen triangle.

4.3 | Parasitic Pathogens

Parasites, like other pathogenic microorganisms, are natural components of the aquatic ecosystems that may occasionally behave as etiological agents for farmed organisms. In contrast to bacterial and viral pathogens, parasites tend to be in balance with their hosts in the natural environment, rarely causing significant diseases or mortality. However, under aquaculture conditions, this balance may be shifted, resulting in substantial damage to the host by a parasite. Importantly, the host–parasite interactions leading to disease in fish are

TABLE 3 | Microbial pathogens of European farmed fish likely to be affected by global warming.

Pathogens	Hosts
Marine water	
Viruses	
Infectious pancreatic necrosis virus	Atlantic salmon, rainbow trout
Lymphocystis virus	Gilthead seabream
Nervous necrosis virus	European seabass
Piscine orthoreovirus	Atlantic salmon
Salmonid alphavirus	Atlantic salmon, rainbow trout (sea)
Bacteria	
Aeromonas salmonicida	Atlantic salmon, rainbow trout (sea)
Aeromonas veroni	European seabass
Lactococcus garviaeae	European seabass, gilthead seabream
Mycobacterium marinum	European seabass, gilthead seabream
Photobacterium damselae subsp. *piscicida*	European seabass, gilthead seabream
Vibrio anguillarum	European seabass, gilthead seabream
Vibrio harveyi	European seabass, gilthead seabream
Parasites	
Amyloodinium occelatum	European seabass, gilthead seabream, meagre
Ceratothoa ostreoides	European seabass, gilthead seabream, meagre
Cryprocaryon irritans	Gilthead seabream
Diplectanum aequans	European seabass
Gyrodactylus salaris	Atlantic salmon
Lepeophtheirus salmonis & Caligus elongates	Atlantic salmon
Lernanthropus kroyeri	European seabass
Neobenedenia girellae	Greater amberjack
Neoparamoeba perurans	Atlantic salmon
Philasterides dicentrarchi	Turbot
Fresh water	
Bacteria	
Aeromonas hydrophilla	Common carp
Flavobacterium columnare	Atlantic salmon, rainbow trout
Lactococcus garviaeae	Rainbow trout
Parasites	
Dactylogyrus extensus	Common carp
Ichthyophthirius multifiliis	Rainbow trout
Sphaerospora molnari	Common carp
Tetracapsuloides bryosalmonae	Rainbow trout

influenced by several environmental factors [177]. Water temperature is the most crucial among them, and changes due to GW may have a profound impact on parasite load in aquaculture. However, while the effect of temperature on specific and nonspecific immune defense in fish has been discussed [178], the extent to which water temperature affects fish simultaneously burdened by parasitic exposure is poorly understood. As already noted, increasing the water temperature can cause fish to experience additional environmental stress and consequent suppression of their immunological response against pathogens [33]. Parasites have an optimal temperature range in which they thrive, but the conditions outside of this range may be lethal [179]. In contrast, for those parasites for which increases in temperature do not surpass their lethal limits, a higher infection rate might be expected, suggesting that the temperature rise can directly affect parasite fitness, as well as exert an indirect influence on their hosts [23, 180]. Therefore, when considering the effect of GW on parasitic infections it is necessary to evaluate multiple host–parasite variables, such as the potential overlapping of parasite and host thermal preferences, intensity and mode of parasite transmission (including thermal range of the intermediate/paratenic hosts, if any), and geographic distribution of the parasite [181].

Parasites are more adept at adapting to environmental challenges than their hosts due to their evolutionary history and plasticity, which enable them to persist and adapt to unpredictable and extreme conditions [182]. However, the complexity of the parasite life cycle will play a significant role in the outcome of parasite adaptation to GW. Parasites exhibiting a direct life cycle might be more obviously affected as their parasitism success depends on a single fish host. Those with an indirect life cycle, which involves multiple hosts often connected through trophic interactions [183], may be impacted at several developmental stages, consistent with the impact on their respective host at each specific trophic level. In fact, Wood et al. [184], after extracting data on metazoan parasite abundance from marine fish specimens held in natural history collections, found a decline in the abundance of some parasites with complex life cycles and a correlation between this decline and increases in sea surface temperature. However, we hypothesize that the phylogenetic "strength" of the coevolution between the parasite and its host will play a crucial role in the parasite's ability to overcome GW for its benefit. Namely, in cases where host and parasite lineages are phylogenetically less congruent, the parasite may respond more easily to climate challenges by host switching, duplication, or sorting [185]. This plasticity enables microevolutionary dynamics at the individual level, allowing the switch

of the host, establishing a new association, and ultimately leading to a macroevolutionary process, such as speciation, which may result in new parasite species adapted to climate change-resistant hosts. Parasite species that fail to speciate may likely develop some other coping mechanisms. Moreover, it is possible that GW will alter the prerequisites for parasite transfer, for example, through changes in phenological relationships and the pressure of selection in the host [186].

In aquaculture, temperature has been identified as a crucial factor in regulating the seasonality and outbreaks of fish parasites, particularly monogeneans. However, it is essential to note that some taxonomic groups of parasites are artificial groupings of several independent clades that evolved from the last eukaryotic common ancestor (e.g., protists and helminths); therefore, their reaction to temperature may vary considerably [187]. The effects of environmental parameters (mainly water temperature) on biological characteristics of important parasites infecting European farmed fish species are presented in Table 4 and discussed below, while examples of affected parasites are listed in Table 3.

4.3.1 | Parasites Affecting Seawater Farmed Fish Species

4.3.1.1 | Ciliates. *Cryptocaryon irritans* is the marine counterpart of *I. multifiliis*, causing marine ich or marine white spot disease in a variety of cultured fish species at sea temperatures between 15°C and 30°C [226]. The detrimental effects of *C. irritans* infection on gilthead seabream broodstock in tanks and other less commercialized Mediterranean fish species were reported to occur at 21°C–24°C [188]. As in the case of *I. multifiliis*, the biological cycle of *C. irritans* and host infectivity are temperature-dependent, with the optimal water temperature being around 27°C [227]. Therefore, increasing water temperature due to GW may favor the onset of either fresh or marine white spot disease, particularly in land-based facilities such as ponds, lagoons, and raceways for freshwater fish or pre-growing facilities housing marine fish species.

Philasterides dicentrarchi (Philasteridae) is a histophagous ciliate and the causative agent of scuticociliatosis in several wild and farmed fish species [228], the latter including turbot [189, 229] and olive flounder (*Paralichthys olivaceus*) [230]. This histophagous and opportunistic parasite has also been considered a pathogen of the lagoon-reared European seabass [190]. Mortalities due to scuticociliatosis seem to occur in periods of high-water temperatures (20°C) in farmed turbot [189], and the temperature range of 18°C–23°C has been proposed as optimal for the proliferation of *P. dicentrarchi* [231]. Higher water temperatures due to GW may therefore enhance the propagation of scuticociliates, or in contrast, increase host susceptibility to this ciliate.

4.3.1.2 | Flagellates. *Amyloodinium ocellatum* (Thoracosphaeraceae) is a dinoflagellate with a very low species specificity that parasitizes marine fish [232]. The disease has a significant economic impact on temperate and warm-water aquaculture, particularly in Mediterranean countries [233]. Amyloodiniosis [233] occurs in earthen ponds and other semi-intensive land systems, with outbreaks also reported in improperly installed sea cages, as in the case of the European seabass farmed in shallow waters exposed to 24°C–26°C [191]. Although less susceptible compared to gilthead seabream, earth-raised meagre is also infected by the flagellate at high water temperatures (>27°C) [192]. Although this flagellate tolerates a wide range of salinities and temperatures, water temperature has a strong modulating effect on the pathogen and its lifecycle duration. In general, the lifecycle is completed in 5–7 days when the temperature rises between 23°C and 27°C. Consequently, *A. ocellatum* outbreaks may become more frequent in land-based aquaculture facilities in the Mediterranean due to increasing water temperatures associated with the GW.

Ichthyobodo spp. (Bodonidae) are flagellates that were first described affecting reared brown trout fry in France, but nowadays they are known to have a wide range of freshwater and marine fish hosts [234–236], including salmonids, flatfish, gilthead seabream, and European seabass, among others. Age is considered a risk factor, as it is more common in hatcheries. Additionally, malnutrition, stress (resulting from excessive handling or fish transfer), and temperature fluctuations are also risk factors. *I. necator* typically shows the highest prevalence in early summer with a lower water temperature [194], in agreement with other studies that observed a negative effect of temperature on *I. necator* infections in salmon [37]. However, a recent study in European seabass in Egypt correlates the speed of *I. necator* outbreaks with the respiratory distress of fish, due to higher temperatures and lower oxygen levels [237]. Therefore, it is possible to suggest that the effect of GW on this parasite appears to be host-specific, particularly in relation to the effect of GW on its host.

4.3.1.3 | Amoebozoae. *Neoparamoeba perurans* (Vexilliferidae) is an amphizoic amoeba causing amoebic gill disease (AGD) in farmed Atlantic salmon [238]. The disease was initially identified in Australia but is currently observed in various parts of the world where Atlantic salmon is farmed, including Norway, where the first documented case was reported in 2006 [239]. Later, *N. perurans* was identified as the causative agent of salmon losses in France, Ireland, and Scotland [240]. Temperature and salinity thresholds for the amoeba growth lie between 4°C and 8°C, and 20‰ and 25‰, respectively [241]. A relationship between increasing water temperature and the severity of AGD has been widely noted in outbreaks on Atlantic salmon farms worldwide [195]. Particularly, while the host response remained unaffected by increased water temperature (15°C), a stronger infection of *N. perurans* was evident, although previous studies have claimed that elevated sea temperatures may be an insignificant risk factor for AGD [240]. Since elevated water temperature appears to be a key risk factor in *N. perurans* outbreaks at least in the most recent literature, GW is likely to trigger more outbreaks and higher severity of this disease in farmed Atlantic salmon.

4.3.1.4 | Myzozoa. *Cryptosporidium* spp. are apicomplexan parasites that infect epithelial cells of the gastrointestinal tract of different vertebrates, including fish. *C. molnari* and *C. scophthalmi* have been reported from farmed fish in gilthead seabream, European seabass and turbot [242–244]. *C. scophthalmi* infections in turbot were also related to the age of the fish,

TABLE 4 | The effects of water parameters on significant biological factors of important fish parasites affecting farmed European fish species.

Parasite	Host	Production system	Optimum water temperature (°C)	Salinity (ppt)	Effects			References
					Fecundity/ hatching	Development/ cycle	Infection success	
Marine water								
Ciliates								
Cryptocaryon irritans	Several spp. broodstock	Tanks	20–24			Shorter duration of trophont residence and theront excystment	Species-specific massive losses	Rigos et al. [188]
Philasterides dicentrarchi	Turbot	Tanks	>20				Massive mortalities	Iglesias et al. [189]
	European seabass	Lagoon						Dragesco et al. [190]
Flagellates								
Amyloodinium ocellatum	Gilthead seabream	Cages	26				Massive mortalities	Rigos et al. [191]
	Meagre	Ponds	27				Emergence of mortalities	Soares et al. [192]
Ichthyobodo sp.	Silthead seabream	Open hatchery	18	21			Massive mortalities	Alvarez-Pellitero et al. [193]
	Atlantic salmon	Fingerlings	10–17				Heavy mortalities	Rintamäki-Kinnunen and Valtonen [194]
Amoebozoea								
Neoparamoeba perurans	Atlantic salmon	Challenge tanks	15				Higher attachment/ growth capacity	Benedicenti et al. [195]
Myzozoa								
Cryptosporidium scophthalmi	Turbot	Ongrowing tanks	11–16				Higher infection	Alvarez-Pellitero et al. [196]

(Continues)

TABLE 4 | (Continued)

Parasite	Host	Production system	Optimum water temperature (°C)	Salinity (ppt)	Effects			References
					Fecundity/ hatching	Development/ cycle	Infection success	
Microsporeans								
Clugea thunni	Atlantic bluefin tuna	Cages	November in east Mediterranean					López-Verdejo et al. [197]
Enterospora nucleophila	Gilthead seabream	Cages, ponds	Mainly low water temperatures				Peaked mortalities	Palenzuela et al. [198]
Myxosporeans								
Enteromyxum leei	Gilthead seabream	Tanks	25				1 week; 100% prevalence	Picard-Sánchez et al. [199]
Enteromyxum scophthalmi	Turbot	Tanks	Warm temperatures				Faster disease progression, peak of mortalities	Branson et al. [200] and Quiroga et al. [201]
Monogeneans								
Diplectanum aequans	European seabass	Cages	20–30		6 days; complete hatching			Cecchini [202]
Diplectanum sciaenae	Meagre broodstock	Tanks	Spring temperatures					Andree et al. [203]
Neobenedenia girellae	Greater amberjack	Cages	30		Faster life cycle and production of higher number of eggs			Hirazawa et al. [204]
Sciaenacotyle panceri	Meagre	Cages	Fall temperatures				5%–10% mortalities	Merella et al. [205]
Sparicotyle chrysophrii	Gilthead seabream	Tanks	18–22		> 93% hatching	Oncomiracidia	Higher rate	Villar-Torres et al. [206] and Villar-Torres et al. [207]
Zeuzapta seriolae	Yellowtail kingfish	Cages	21		Decreased hatching times and age at maturity			Tubbs et al. [208]

(Continues)

TABLE 4 | (Continued)

Parasite	Host	Production system	Optimum water temperature (°C)	Salinity (ppt)	Effects			References
					Fecundity/ hatching	Development/ cycle	Infection success	
Digeneans								
Cardicola spp.	Atlantic bluefin tuna	Cages	Spring temperatures					Palacios-Abella et al. [209]
	Gilthead seabream							Holzer et al. [210] and Palacios-Abella et al. [211]
Paradeontacylix grandispinus, Paradeontacylix kampachi	Greater amberjack	Cages						Repullés-Albelda et al. [212]
Copepods								
Caligus elongatus	Atlantic salmon	Tanks	9				Higher infection success	Michre [213]
Lepeophtheirus salmonis		Tanks	10				Higher infection success	[214]
Lerneanthropus kroyeri	European seabass	Cages	Spring–summer				Peak of outbreaks	Manera and Dezfuli [215]
Isopods								
Ceratothoa oestroides	Gilthead seabream	Tanks	22			Uncompleted reproduction cycle	Higher prevalence	Mladineo [216]
	Meagre	Cages	Low and high temperatures				Higher prevalence, peak of outbreaks	Čolak et al. [217]
	Gilthead seabream	Tanks, cages	21–23					Papapanagiotou and Trilles [218]

(Continues)

TABLE 4 | (Continued)

Parasite	Host	Production system	Optimum water temperature (°C)	Salinity (ppt)	Effects			References
					Fecundity/ hatching	Development/ cycle	Infection success	
Fresh water								
Ciliates								
Ichthyophthirius multifliis	Rainbow trout	Ponds	11–21	< 5		Higher released theront number (< 642)		Aihua and Buchmann [219]
	Common carp		24–25			Faster completed cycle (6–9 days)		
Amoebozoea								
Amoebae spp.	Rainbow trout	Flow-through tanks	< 10				> 60% mortalities in young fish	Quaglio et al. [220]
Myzozoa								
Goussia carpelli	Common carp	Experimental tanks	10–24				Shorter time to develop into oocyst	Steinhagen [221]
Myxosporeans								
Sphaerospora molnari	Common carp	Ponds						Dyková et al. [222]
Tetracapsuloides bryosalmonae	Rainbow trout	Tanks	> 15				< 2 weeks; 100% prevalence	Bettge et al. [223]
Monopisthocotylans								
Dactylogyrus extensus	Common carp	Ponds	25			Higher and faster hatching rate		Turgut [224]
Gyrodactylus salaris	Atlantic salmon		12			Increased parasitic infection rate		Bakke et al. [225]

poor condition factor, and higher temperatures during spring and summer [196], and *C. molnari* also exhibited maximum levels of infection in spring [244]. Thus, although more studies are needed to elucidate the effect of CC in coccidians and *Cryptosporidium* in fish, higher temperatures due to CC are likely to enhance the infection of these intracellular parasites.

4.3.1.5 | Microsporeans. *Glugea thunni* (Glugeidae) is a microsporidian recently described as a fungus that causes severe pathology of the visceral cavity of Atlantic bluefin tuna farmed in the Spanish Mediterranean Sea [197]. Whitish xenomas were mostly located at the cecal visceral mass. The episode was observed during the fall (November), but the water temperature was not specified.

Enterospora nucleophila (Enterocytozoonidae) is an emerging intranuclear microsporidian responsible for microsporidiosis in farmed gilthead seabream [245]. Since its first description in Spanish facilities of gilthead seabream [198], *E. nucleophila* has also been diagnosed in Italian and Greek farms, in both sea cages and land-based nurseries [245]. Infections with *E. nucleophila* have been noticed during all seasons [198], but clinical signs in the field are not conspicuous during warmer periods [246]. While low water temperatures inhibit microsporidian development in fish, during warmer periods, the parasite copes with the evasion of the immunological response of the host [247] and reaches the optimal temperature for reproduction [246]. Therefore, it is unclear to what degree the GW will affect the onset of microsporidiosis in farmed gilthead seabream.

4.3.1.6 | Myxosporeans. *Enteromyxum leei* (Enteromyxidae) (formerly *Mixidium leei*) is a severe myxosporean parasite reported since the early 90s as a causative agent of mortalities in Mediterranean farmed sparids [248], including gilthead seabream [249, 250], sharpsnout seabream (*Puntazzo puntazzo*), and red porgy (*Pagrus major*) [251]. Notably, due to uncontrollable acute summer mortalities (24°C–26°C) caused by *E. leei* in the sharpsnout seabream, its farming in the Mediterranean was eventually abandoned. Water temperature has been the most critical risk factor for the transmission and development of enteromyxosis in gilthead seabream, with optimal in vitro development of *E. leei* occurring at higher temperatures (20°C–25°C) [199]. For the development of a clinical form in the field, a minimum temperature of 18°C–22°C is necessary [252]. However, enteromyxosis development is suppressed below 15°C, possibly due to the low multiplication rate of the parasite [253]. Under such conditions, the parasite can remain latent in the host [254], with the ability to reinfect upon sudden temperature increases. Therefore, rising water temperatures due to GW are likely to affect the incidence of enteromyxosis in gilthead seabream.

E. scophthalmi is an enteric myxozoan responsible for severe losses in cultured turbot [200]. Apart from the influence of water temperature, other risk factors associated with *E. scophthalmi* infection included diminished water quality (unfiltered water) and the aggregation of infective stages in culture tanks [201]. The mortality pattern of infected turbot growers indicates that losses occur throughout the entire production year, peaking during warmer water temperatures; however, the seasonality may be altered depending on the time of introduction of the fish stock [201]. Since several factors are involved in the onset of turbot *E. scophthalmi* infection, it cannot be claimed that increasing water temperatures alone due to GW can enhance the pathogenesis of turbot enteromyxosis.

4.3.1.7 | Polyopisthocotylans. *Sparicotyle chrysophrii* (Microcotylidae) is a monogenean oviparous parasite that severely infects gilthead seabream, causing gill tissue damage, anemia, hypoxia, emaciation, and lethargy [255, 256]. While most reports agree on the correlation between the outbreaks and seasonally increased water temperatures, the monogenean has also been associated with epidemics in winter (> 13°C) [257]. Water temperature is especially crucial for the propagation of *S. chrysophrii* and development of its larval stages, as recently assessed [207]. The infection success is highest from 18°C to 22°C and declines at 14°C and 26°C, suggesting that 22°C is the ideal temperature for *S. chrysophrii* propagation and pathogenicity, while transmission is less successful outside this temperature range [207]. Moreover, increasing temperature from 22°C to 26°C favors embryonic development and egg hatching rates as observed by Villar-Torres et al. [206], where a decrease in the swimming activity and survival rate in *S. chrysophrii* oncomiracidia was noticed with increasing temperatures from 10°C to 26°C, resulting in a lesser ability to infect hosts. On the contrary, water temperature had a positive correlation with the intensity of adult *S. chrysophrii* infection, indicating that parasitic transmission was favored by increasing temperature [258]. Therefore, rising water temperatures due to GW in Mediterranean waters are likely to positively affect the incidence of sparicotylosis in farmed gilthead seabream.

Zeuxapta seriolae (Heteraxinidae) is a gill polyophystocotylean blood-feeding parasite which has been associated with anemia and severe mortalities in farmed greater amberjack in Europe [259, 260] and Japan [261]. High water temperature and fouled nets in the cages accelerate the propagation of *Z. seriolae* [262], although associated mortality in greater amberjack has also been described during the winter period in the Western Mediterranean [259]. Notably, hatching times and age at maturity of this polyophystocotylean were inversely related to water temperature (13°C–21°C) in yellowtail kingfish (*S. lalandi*), a close relative of greater amberjack [208]. Considering that Mediterranean water temperature during the warmest periods is higher (24°C–26°C) than the temperatures tested in the latter study, and *Z. seriolae*-related outbreaks may also occur at winter temperatures, it is unclear whether a further increase in water temperatures will favor outbreaks of *Z. seriolae* in greater amberjack.

4.3.1.8 | Monopisthocotylans. *Diplectanum aequans* (Diplectanidae) is a species-specific oviparous monophystocotylean causing gill pathology in European seabass [263, 264]. Diplectanosis was considered one of the most significant ectoparasitic diseases of the European seabass [265], but the lack of more recent reports in the Mediterranean farms suggests that the disease has become sporadic, with an endemic incidence in some farming sites. There are conflicting observations regarding the incidence of diplectanids and temperature: some authors have reported a higher intensity correlated with the seasonal increase in water temperatures [266],

while others have related the highest prevalence and mean intensity to winter [264]. Nonetheless, *D. aequans*' life cycle is temperature-driven [267], with high water temperatures (30°C) increasing the success and speed of the hatching time [202, 268]. This suggests that, although *D. aequans*' development has a wide temperature range (20°C–30°C) [268], increasing water temperatures caused by GW may trigger *D. aequans* outbreaks in the European seabass.

Sciaenacotyle panceri (Sciaenidae) is a monophystocotylean that parasitizes the gills of sciaenid fish [205]. It has been highlighted as an emerging pathogen of concern in sea-cage meager, a newly introduced fish species in the Mediterranean aquaculture [269]. *Diplectanum sciaenae* (Diplectanidae) is another monophystocotylean that causes considerable gill pathology and losses in meagre broodstock in spring [203]. In the fall, infected meagre displayed emaciation, pallid gills, hypersecretion of mucus, and anemic visceral organs [205]. Meanwhile, *S. pancerii* prevalence and intensity peak in November and December, coinciding with the lowest water temperature (14°C) [270]. The latter is likely related to reduced responsiveness of the fish immune response, which favors microcotylid outbreaks at low water temperatures. Therefore, without more recent observations from the field and in vitro studies, it is unclear whether the GW will have an impact on the pathogenesis of the aforementioned monophystocotylean in reared meagre.

Neobenedenia girellae (Capsalidae) is a skin monophystocotylean which exhibits low host specificity, infecting the fins and skin of several farmed fish species. Greater amberjack seems to be one of the most susceptible hosts [271] among numerous other farmed marine species [272]. Another *Neobenedenia* sp., described as *N. melleni* was found in greater amberjack raised in the Canary Islands [273], and *N. girellae* naturally infected fish have been used in experimental trials in the same area [274]. More recently, *N. girellae* was detected in cage-reared gilthead seabream from the North-eastern Atlantic area [275]. Since *N. girellae* undergoes a faster life cycle and produces a greater number of eggs, causing a more severe infection on greater amberjack with increasing temperatures (30°C vs. 20°C–25°C) [204], rising water temperatures due to GW will probably affect the pathogenesis of *Neobenedenia* spp. in caged greater amberjack.

4.3.1.9 | Digeneans. *Paradeontacylix* spp. (Sanguinicolidae) are blood flukes considered important pathogens in marine aquaculture [276]. These digeneans have been associated with mortalities in farmed greater amberjack in the Mediterranean region [277–280] and Japan [281–283]. Specifically, *P. ibericus* and *P. balearicus* [212] are affecting Mediterranean greater amberjack [277–279], causing severe mortality [277, 278, 283]. *P. ibericus* has been associated with heavy losses caused by mixed infections with *Epitheliocystis* sp. in 0+ fish [277]. The seasonality influences the abundance of fluke eggs in the gills of greater amberjack, increasing during winter and decreasing as summer approaches [281], which is essential to consider in parasite management. Thus, it is unlikely that rising water temperatures due to GW will favor outbreaks of *Paradeontacylix* spp. in greater amberjack.

Cardicola spp. (Aporocotylidae) are important blood flukes of mainly bluefin tuna ranched across Australia, Asia, and Europe [284]. To date, in Mediterranean aquaculture, four *Cardicola* spp. have been reported in Atlantic bluefin tuna [209], and two in gilthead seabream [210, 211]. Initially, the infection in gilthead seabream was described as sanguinicolid infection, causing trickling mortalities during the cold season in the western Mediterranean [285]. Since infections of gilthead seabream with *Cardicola* spp. occurs during medium water temperatures (spring), it is unlikely that increasing water temperatures due to GW may affect such parasitic infestations.

4.3.1.10 | Copepods. *Lepeophtheirus salmonis* and *Caligus elongatus* (Caligidae) are ectoparasitic copepods, commonly named salmon sea lice, that represent a key limitation to Atlantic salmon aquaculture and to the sustainability of populations of wild salmonids [286]. The duration of the parasitic planktonic stages depends on the water temperature, lasting up to 35 and 10 days at 5°C and 15°C, respectively [287]. Indeed, water temperature has been described as a crucial variable in the biological cycle of caligids. For example, salmon lice develop faster in warmer water but survive for a shorter time [288]. Importantly, the effects of sea lice on the growth rate and survival of Atlantic salmon worsen with increasing water temperatures (10°C–22°C) [289]. Moreover, the impact of *L. salmonis* gradually increases with rising temperature, with an estimated twofold effect if the temperature rises from 9°C to 11°C [290]. The modelled temperature increase in the order of 2°C is within a realistic scenario, with an estimated prediction that the annual mean sea surface temperature increase in the North Sea area by the end of the century will be in the range of 1°C–3°C [5]. A warmer climate due to GW will most likely increase the pressure of sea lice infection in farmed Atlantic salmon.

Lerneanthropus kroyeri (Lernanthropidae) is a parasitic copepod that infects the gills of European seabass, causing mechanical damage [215]. A high prevalence is commonly observed during the warm periods [291], suggesting a preference for higher water temperatures [215], although there are cases of infections at lower water temperatures (18°C) [292].

4.3.1.11 | Isopods. *Ceratothoa oestroides* (Cymothoidae) is a ubiquitous isopod affecting European seabass, gilthead seabream, and meagre [217, 293–295]. The highest mortality caused by *C. oestroides* is noted in juvenile fish [295], and outbreaks commonly occur during the warmer months (21°C–23°C) [218]. Additional risk for the spread of isopods is the seasonal aggregation of wild fish that transfer the isopod to farmed fish, especially during warm periods when higher feed loads are provided within the cages [296]. Since the outbreaks of the isopod coincide with high water temperatures, increasing water temperatures due to GW are likely to influence the intensity of the outbreaks.

4.3.2 | Parasites Affecting Freshwater Farmed Fish Species

4.3.2.1 | Ciliates. *Ichthyophthirius multifiliis* (Ichthyophthiriidae) is a ciliate causing ich or white spot disease in a variety of freshwater fish maintained in land-based facilities, including rainbow trout and common carp [219]. Host-independent stages (tomonts) that are encysted on various substrates can survive

water temperature ranges from 2°C to 27°C [297], although development is possible even at higher temperatures (30°C) in farmed rainbow trout [219]. The life cycle of *I. multifiliis* is faster with increasing temperatures, and the ciliate can complete a full cycle in 6–9 days at 24°C–25°C in common carp. While it has a clear temperature-dependent life cycle, the associated outbreaks in farmed fish are also dependent on the resistance of the host.

4.3.2.2 | Amoebozoae. Nodular gill disease (NGD) is the freshwater counterpart of AGD, but the aetiology of this disease remains unclear. Although NGD was initially described in North America [298], it was also described in Europe, particularly in rainbow trout farms [220, 299, 300]. Some authors suggest that the most severe clinical signs appear during the cold months when the water temperature drops below 10°C [220], but others have recorded disease outbreaks when the water temperature rises in spring [301]. Therefore, the implications that GW may have on this disease cannot be ruled out.

4.3.2.3 | Myzozoa. Myxozoa are protists characterized by a myzocytotic feeder, and comprise dinoflagellates and apicomplexans among other minoritarian groups [302]. Eimeriid coccidians, especially of the genus *Eimeria* and *Goussia*, are described mainly as affecting fish [303, 304]. Little is known about the effect of CC in coccidian infection in fish, but Steinhagen [221] observed that the development of the oocyst of *Goussia carpelli* affecting carps was temperature dependent, needing 5–6 weeks to develop at 12°C but only 2–3 weeks at 20°C.

4.3.2.4 | Myxosporeans. *Tetracapsuloides bryosalmonae* (Saccosporidae) is a myxozoan parasite causing proliferative kidney disease (PKD), which is one of the most serious parasitic diseases of salmonids, including rainbow trout in Europe and elsewhere. Rainbow trout survivors of PKD can restore renal structure and reduce parasite intensity, while water temperature (25°C) influences the rate but not the outcome of the recovery process [305]. PKD seasonality is often linked to increased water temperatures [306], and clinical signs and mortalities increase at water temperatures above 15°C [307]. Elevated water temperatures likely promote bryozoan growth, which in turn leads to a greater number of infective stages of the parasite being released into the farming environment of salmonids [308]. Similarly, a higher water temperature also enhances parasite proliferation in the host [223]. Thus, it is likely that GW may trigger the incidence and severity of PKD in farmed rainbow trout in freshwater environments.

Myxobolus cerebralis (Myxobolidae) is a myxosporean that causes whirling disease in farmed and wild salmonids, among which the rainbow trout is the most susceptible member [309]. The development of the triactinomyxon stage of *M. cerebralis* and the release of mature spores from the tubificid oligochaete are temperature-dependent [310]. Temperatures between 15°C and 20°C enhance the development of the parasite, increasing the number of released spores; however, temperatures above 20°C suppress, or are lethal to the triactinomyxon stages. Similarly, the development in the oligochaete is also temperature-driven [311]. Since increasing water temperatures abrogate the development of the parasite in the intermediate host and consequently, the infection of the final host, it is unlikely that GW will affect the onset of whirling disease in farmed rainbow trout.

Sphaerospora molnari (Sphaerosporidae) is a myxozoan parasite causing gill, skin, and blood sphaerosporosis in European farmed common carp [312]. The disease has mainly been reported from Central Europe and less frequently from southeastern European countries. A link to increasing pond temperatures due to climate change was emphasized as a key factor in the increased occurrence of *S. molnari* [313]; *S. molnari* infects carp fry during summer temperatures, while parasite prevalence decreases in the winter months. Therefore, rising water temperatures due to GW may affect outbreaks of sphaerosporosis in farmed common carp.

4.3.2.5 | Monopisthocotylans. *Dactylogyrus extensus* (Dactylogyridae) is a host-specific oviparous gill monophystocotylean that causes significant economic losses in farmed common carp [314]. It is the dominant species of the parasitic communities in Iranian [315] and Balkanian carp farming [316]. Water temperature is the primary factor influencing egg production and oncomiracidia hatching [224]. A significantly higher number of eggs is produced at 17°C compared to 10°C, and the hatching rate is higher and the hatching is more rapid at 25°C. Therefore, increasing water temperatures due to GW is likely to affect *D. extensus* outbreaks in farmed common carp.

Gyrodactylus salaris (Gyrodactylidae) is an obligate viviparous monophystocotylean parasite of salmonids. It is a severe pathogen that causes significant losses in fry and parr of Atlantic salmon farmed in freshwater [317]. In contrast, it has a negligible impact on farmed rainbow trout [318]. Due to the severity of gyrodactylosis, *G. salaris* is listed by the WOAH as a notifiable disease. *G. salaris* has a wide temperature tolerance, surviving between 0°C and 25°C. The effect of water temperature (> 12°C) on gyrodactylid transmission rates is positively correlated [225], where lower temperatures reduce the rate of transmission. The survival of detached parasites or those attached to a dead host is also temperature-dependent [319]. *G. salaris* mainly thrives in freshwater, but it may reproduce at salinities up to 5–6 ppt, while survival at higher salinities is temperature-dependent [320, 321]. The increasing temperatures in freshwater due to GW may increase the incidence and severity of gyrodactylosis in the younger stages of Atlantic salmon farmed in freshwater.

5 | The Effect of Global Warming on the Pathogenesis of Viral, Bacterial, and Parasitic Pathogens in Fish Aquaculture

5.1 | General Mechanisms of Pathogen Evolution

Pathogenesis is the development of a disease through a chain of sequential biological mechanisms (exposure or contact, colonization, invasion, and infection) by which a pathogen causes a diseased state. These biological mechanisms are facilitated by the pathogen's virulence, that is, its ability to cause disease, exerted through the virulence factors of the pathogen. Disease development is not a one-sided process; host responses are essential for resolving the pathogenesis or progressing it to the ultimate demise of the host [322, 323]. It is hypothesized that the GW scenario will accelerate the evolution of pathogens and introduce a higher mutation rate in the pathogens' genome, especially in

the part encoding for virulence factors, such as peptidases and peptidase inhibitors, consequently selecting for isolates or strains with a mutated, higher virulence [324]. Namely, a particular abiotic factor (e.g., temperature) that extends its limit beyond the pathogen's "comfort zone" concomitantly exerts a selective pressure upon the pathogen. This may result in a part of the pathogen population dying out, while the other part adapts and passes the mutation on to subsequent generations, evolving reduced sensitivity to climate fluctuations [325]. When the abiotic factor remains in the upper limit of the pathogen's "comfort zone," it supports the increase of the pathogen population by intensifying the pathogen metabolism and cell division [326]. However, the former may result in the higher production of reactive oxygen species that negatively affect the stability of the genome and induce a higher rate of mutation that need to be "corrected" by the activation of DNA damage and repair (DDR) mechanisms [327]. The pathogen's fate is directed either towards cell death (apoptosis) if the major genome mutation is unrepairable or too costly; cell survival with corrected mutation, therefore with no changes in the genome; or cell survival where the mutation is minor and has been neglected or has slipped from the DDR mechanism, consequently being passed further. From the published data, it appears that the correlation between virulence and temperature is much clearer for bacterial than for viral diseases, possibly due to host immune responses and genetics, that is, species susceptibility, which plays an important role in the latter. Species susceptibility, considered a genotypic feature of immunocompetence in relation to GW, is not addressed in this review, except to highlight a more complex interaction between immunity and temperature than initially assumed. For example, while higher fish immunocompetence was accepted as an asset to mitigate the increased virulence during the GW scenario, Kayansamruaj et al. [328] suggested that the resulting explosive immune reaction at higher temperatures accounted for increased mortalities of rainbow trout during *Streptococcus agalactiae* outbreaks. In contrast to cold-water rainbow trout, transcriptomes of tilapia infected with *S. agalactiae* at 22°C are enriched mainly with the immune response pathways. At 32°C, immune, as well as oxygen- and energy-related metabolic pathways, are prominent in infected tilapia, suggesting a high burden exerted on the fish to mitigate temperature stress [329]. In this scenario, high temperature evokes immunosuppression (observed by downregulation of toll-like receptor, chemokine, NF-kappa B, TNF, and cytokine-cytokine receptor interaction signaling pathways), damage, and dysfunction of the immune system under the condition of oxygen depletion [33]. This can lead to lower selective pressure control of the pathogens, resulting in longer persistent infections that allow pathogens to "experiment" with different mutations within virulence factors [330, 331]. Therefore, thermally compromised or dysregulated host immune responses cannot be excluded from the equation, which facilitates increased mutation rates and, consequently, the virulence of pathogens due to lower control over pathogens during disease development at higher temperatures. Below, we provide selected examples illustrating the impact of GW on pathogen virulence.

5.2 | Viral Pathogens

A recent simulation of future cross-species viral transmission between mammals under climate change and land use scenarios for the year 2070 estimated that this ecological transition will not be reduced even if the increase in global temperature is limited to 2°C within the twenty-first century [332]. While studies of mammalian viruses evidence a clear correlation with GW [333, 334], in aquatic environments, there are still many knowledge gaps to fill. For the latter, general conclusions have been drawn from an understanding of the increased mutation rate in negative-strand RNA viruses. These viruses exhibit a higher mutation rate of 10^{-3} to 10^{-6} mutations incorporated per nucleotide copied, which is orders of magnitude higher than estimated for replication of cellular DNA under normal metabolic conditions [335]. Consequently, it is hypothesized that an increased mutation rate due to rising global temperatures may ultimately lead to an improvement in the fitness or virulence of fish RNA viruses at higher temperatures, as observed in arthropod viruses [336]. Thermal adaptation under the selective pressure of increased environmental temperature can result in altered intra-host competition and viral strain selection [337], although this can be overlooked if studied solely in vitro [338].

In case of ISAV (Orthomyxoviridae), a representative of negative-strand RNA viruses, the average mutation rate of two virulence factors (fusion protein and haemagglutinin-esterase protein) is estimated to be 0.90×10^{-3} nucleotides per site per year, suggesting that the risk of new more virulent isolates is higher compared to other viruses [339]. Given that generally the negative-strand RNA viruses are more likely to emerge as new diseases or in new hosts [340], it can be speculated that these would be more prone to GW-triggered mutations, showing an advantage in interspecies spill over compared to other viruses.

In contrast, the potential for spreading a warm-water tilapia lake virus (TiLV), an orthomyxo-like virus from the newly established family Amnoonviridae, to cold EU waters proved to be ambiguous. No clear evidence was observed between the drop in temperature and TiLV virulence, but the research was unable to parse the influence of host responses from the virulence [341]. The experiment highlighted the fact that in countries where tilapia is originally farmed (29°C–31°C), other fish species, such as walking catfish (*Clarias macrocephalus*), striped snake-head fish (*Channa striata*), climbing perch (*Anabas testudineus*), silver barb (*Barbodes gonionotus*), or Asian sea bass (*Lates calcarifer*) appear to be non-susceptible. However, the presence of TiLV in these species cannot be fully ruled out, even in the absence of clinical signs [342]. Whether temperature increases the virulence of TiLV remains elusive; evidence gathered so far confirms that it does in the case of tilapia and crucian carp infections, in contrast to rainbow trout and common carp, suggesting that permissiveness to infection also depends on host genetics [343].

The red sea bream iridovirus disease (RSIVD) caused by the DNA iridovirus Sachun (IVS-1) infection in the rock bream (*Oplegnathus fasciatus*) reaches 100% at higher temperatures (18°C, 21°C, and 25°C), while at lower temperature (13°C) it slows the replication, but is never fully cleared from the host [344]. The virus progresses faster at increased temperatures until the onset of mortality. However, at 13°C, mortality can reach 100% only when the virus is directly injected; as the temperature progresses towards 25°C, it indicates that factors other than temperature contribute to the severity of the disease.

Models support the pathogenesis–temperature correlation: for cyprinid herpesvirus 3 (CyHV-3), which causes koi herpesvirus disease (KHVD), and Betanodavirus, responsible for VNN, a meta-analysis of cumulative host mortalities under fixed temperatures was undertaken to test whether increased water temperature increases the severity of viral infections, evaluated as the mortality rate [39]. The developed linear regression models based on data from 53 experimental studies corroborated that higher mortality rates were associated with increasing water temperatures. An increase in water temperature of 1°C would result in an increase in mortality of 2.55%–6.98% in CyHV-3-infected carp, and 2.18%–5.37% in NNV-infected fish. Under such a scenario, CyHV-3 and VNN mortality is 1.09–1.89 times higher than the mortality increase due to bacterial infection in warm waters, and 1.05–1.26 times lower than the mortality due to bacterial infection in temperate environments. However, different members of Betanodavirus exhibit variations in virulence in response to temperature challenges. Betanodavirus in Senegalese sole and grouper (*Epinephelus akaara*) exacerbates 100% mortality at 22°C and only 8% at 16°C [57, 345]. In contrast, RGNNV in fingerlings of the humpback grouper (*Cromileptes altivelis*) subjected to increased temperature (29°C–31°C, and 35°C) shows lower mortality (50%, 20%, and 10%, respectively). In this case, histopathological changes (vacuolization of the retina) were observed only at 29°C, suggesting that high temperature inhibits viral replication [346]. While the results could have been affected by a limited sample size of the study ($n = 10$), the potential confounding effect of the host immunity and virus genotype should be closely evaluated. Namely, some betanodavirus genotypes (BFNNV, TPNNV) cause disease in cold-water fish, while others (red-spotted grouper NNV, RGNNV; striped jack NNV, SJNNV) infect warm-water fish [51]. Moreover, the reassortants of the latter two, that is, RGNNV/SJNNV and SJNNV/RGNNV, also show different virulence under an increasing temperature regime in juvenile European seabass, confirming that their replication has been affected by the polymerase gene and the temperature. At 20°C, the highest mortality is induced by SJNNV genotype, and by RGNNV at 25°C and 30°C. In contrast, the reassortant RGNNV/SJNNV and SJNNV/RGNNV achieve lower mortality with the increase in temperature (i.e., 3.3% and 4.0% mortality, respectively at 20°C, both 1.5% mortality at 25°C, and 2.3% and 1.5% at 30°C). However, it appears that the pathogenicity of the strains is not influenced only by the temperature: the SJNNV strain is weakly pathogenic, and it replicates in the brain only at 20°C. Reassortant strains cause low mortality; however, the viral load in the brain is temperature- and polymerase type-dependent [51]. Therefore, temperature-dependent virulence and pathogenesis of fish viral diseases are topics that warrant further research.

Association between different temperatures and fish viruses important for aquaculture in terms of mortality, clinical signs, and/or histopathology is presented in Table 5.

5.3 | Bacterial Pathogens

The virulence genes in bacteria that are directly or indirectly affected by temperature are mostly associated with pathways involved in the synthesis of flagellar elements, thereby influencing motility, quorum sensing signaling, biofilm formation, and adhesion [347–353]. In culturable bacteria that can be studied in vitro, extensive literature assessing the temperature effect is available; however, far less data have been generated from experimental laboratory and field in vivo studies. While the latter can be confounded by other inevitable conditions, such as the general host immune status, the limited number of specimens, and their genetic background, these can still offer a more realistic insight into virulence display within host–pathogen interactions.

The high plasticity of *V. harveyi* genomes has been attributed to its ability to adapt to rapidly changing environmental factors [354]. This is facilitated by mobile extrachromosomal elements (e.g., genomic islands, bacteriophages, and plasmids), as well as the conjugation mechanism that enables efficient gene exchange and transfer, not only within, but also between different Gram-negative bacterial species [355]. For example, temperature has an impact on quantum sensing of vibrios, a universal system for bacterial cell-to-cell communication induced by production and secretion of autoinducers, signal molecules that are correlated to cell density, enhancing vibrios' virulence in black-band disease of corals [356]. Despite the observation that in vitro temperature has a correlation with the emergence of pathogenic *Vibrio* spp., there is limited data provided over long timescales or directly related to disease onset in aquatic vertebrates. Studies performed on archival formalin-fixed samples of microbial communities using molecular tools were invaluable to show increased vibrios' presence within the plankton-associated bacterial community [357, 358] and in seawater microcosms [145], but are not directly related to outbreaks in aquatic vertebrates, demonstrating only an indirect and consequential relationship.

V. alginolyticus loses its swarming capacity when subjected to high temperatures (28°C–39°C) and low NaCl (0.6%–1.5%), which causes the loss of its peritrichous flagella, even if the polar sheathed flagellum is intact [359]. Purified peritrichous flagella are stable at temperatures ranging from 10°C to 45°C in 0.5%–5% NaCl, but a lower pH value (6 and 7.2) appears as a main driver of their development. Under such pH, swarming is activated even at high temperatures (37°C) and low NaCl concentrations (1%), highlighting the importance of other factors in assessing pathogen virulence under climate change.

The importance of physical factors associated with higher temperatures has been demonstrated in the case of *V. anguillarum*. The bacterium also has a sheathed polar flagellum that enables the fastest chemotactic response to serine at 25°C (compared to 5°C and 15°C), which is its optimal growth temperature [360]. However, the temperature dependency of chemotaxis has been hypothesized to be linked to various physical and biochemical factors. First, the increased rate of phosphorylation and/or (de-)methylation of signal transduction proteins and receptors at higher temperatures can be attributed to an increased kinetics of enzymatic reactions. Secondly, the fluidity of membranes depends on temperature, which consequently affects the sheath of the *V. anguillarum* flagellum, a part of a distinct outer-membrane domain (McCarter, 2001). Lastly, swimming speed also depends on the correlation between the interaction of water physical parameters, such as viscosity and temperature; the viscosity being 1.7 times lower at higher temperatures (25°C vs. 5°C), which facilitates higher speed [361].

TABLE 5 | Association between different temperatures and fish viruses important for aquaculture in terms of mortality and clinical signs and/or exerted histopathology.

Viruses	Family	Isolate type	Host	Temperature (°C)	Testing system	Challenge type	Mortality (%)	Clinical signs and/or histopathology	References
Betanodavirus	Nodaviridae	BFNNV, TPNNV, RGNNV, SJNNV	Rock bream	1°C increase	Linear regression model	i/m	2.18–5.37 increase		Combe et al. [39]
		SpSs-IAusc160.03 (reassortant RGNNV/SJNNV)	Senegalese sole	22	Experimental in vivo	Immersion, cohabitation	100	Standard clinical signs	Souto et al. [57]
				18			75–80	Standard clinical signs	
				16			8	No clinical signs, but viral replication present	
		SJNNV	Grouper	28	Experimental in vivo	i/m, immersion	20, 0, respectively	Abnormal swimming influenced by higher temperature	Tanaka et al. [345]
				24		i/m	28.6		
				20			14.3		
				16			14.3		
				24–28, fluctuating			57.1		
		RGNNV	Humpback grouper	35	Experimental in vivo	Immersion	50	No vacuolization in retina	Yuasa et al. [346]
				31			20	No vacuolization in retina	
				29			10	Vacuolization of retina	
		SJNNV	European seabass	20	Experimental in vivo	Immersion	5.2	Fewer signs compared to RGNNV, replication in brain	Toffan et al. [51]
		RGNNV	European seabass	20	Experimental in vivo	Immersion	3.7	Standard signs	Toffan et al. [51]
				30			30.8	The most serious signs	
				25			30	The most serious signs	
		RGNNV/SJNNV, SJNNV/RGNNV reassortants		30			2.3 and 1.5, respectively	Fewer signs compared to RGNNV under all temperatures, brain viral load correlated with temp.	
				25			1.5, both		
				20			3.3 and 4.0, respectively		
Cyprinid herpesvirus 3	Alloherpesviridae	CyHV-3	Rock bream	1°C increase	Linear regression model	i/m	2.55–6.98 increase		Combe et al. [39]

(Continues)

TABLE 5 | (Continued)

Viruses	Family	Isolate type	Host	Temperature (°C)	Testing system	Challenge type	Mortality (%)	Clinical signs and/or histopathology	References
Red seabream iridovirus	Iridoviridae	IVS-1	Rock bream	25	Experimental in vivo	i/m	100	17 days to mortality	Jun et al. [344]
				21			100	20 days to mortality	
				18			100	30 days to mortality	
				13			0	No histopathology, slow viral replication	
Tilapia lake virus	Orthomyxoviridae	2017B, weekly virulent	Nile tilapia	22	Experimental in vivo	i/p	25	Mild liver necrosis, infiltration and syncytia	Bergmann et al. [341]
				22		Immersion	25	Liver necrosis	
				17		i/p	50	Liver necrosis	
				12		i/p	50	Liver necrosis	
			Crucian carp	22		Cohabitation with tilapia	25–50	Liver necrosis, Infiltration and syncytia stronger than in 2017A	
			Common carp	22		Cohabitation with tilapia	30	Weaker changes compare to above	
			Rainbow trout	17		Cohabitation with tilapia	0	No clinical signs, but virus detected in normal liver	
				12		Cohabitation with tilapia	0	No clinical signs, but virus detected in normal liver	
		2017A, virulent	Nile tilapia	22		i/p	75	Liver necrosis	
				22		Immersion	25	Liver necrosis	
				17		i/p	0	No clinical signs	
				12		i/p	100		
			Rainbow trout	17		Cohabitation with tilapia	0	No clinical signs, liver necrosis, virus reisolated from the liver	
				12		Cohabitation with tilapia	10	No clinical signs, liver necrosis, virus reisolated from the liver	
			Common carp	22		Cohabitation with tilapia	0	No clinical signs, liver necrosis, virus reisolated from the liver	
			Crucian carp	22		Cohabitation with tilapia	25–50		

(Continues)

TABLE 5 | (Continued)

Viruses	Family	Isolate type	Host	Temperature (°C)	Testing system	Challenge type	Mortality (%)	Clinical signs and/or histopathology	References
Tilapia lake virus	Orthomyxoviridae	Hyper-virulent Thailand strain VETKU-TV01	Rainbow trout	20	Experimental in vivo	i/p	0		Adamek et al. [343]
				20		Cohabitation	0		
				25		i/p	0	Syncytia, multifocal necrosis in liver	
				25		Cohabitation	0	Syncytia, multifocal necrosis in liver	
			Brown trout (*Salmo salar*)	20		i/p	0		
				20		Cohabitation	0		
				25		i/p	0	Syncytia, multifocal necrosis in liver	
				25		Cohabitation	0	Syncytia, multifocal necrosis in liver	

Abbreviations: BFNNV: Barfin flounder nervous necrosis virus; i/m: intramuscular; i/p: intraperitoneal; RGNNV: Redspotted grouper nervous necrosis virus; SJNNV: Striped jack nervous necrosis virus; TPNNV: Tiger puffer nervous necrosis virus.

Chemotaxis and subsequent adhesion of *V. anguillarum* and *V. alginolyticus* strains towards gilthead seabream mucus were positively correlated with increased temperature (15°C, 22°C, and 27°C), but were dependent on the origin of the mucus. Skin mucus was a strong attractant at higher temperatures, followed by gill and intestinal mucus [362]. In contrast, strains of other vibrios (*V. harveyi*, *V. fischeri*, and *V. tubiashii*) either exhibited chemotaxis and adhesion or failed to achieve adhesion at all. Some strains, for example, showed strong chemotaxis but lacked adhesion to the mucus, suggesting a lack of straightforward interaction between the chemotactic response to mucus and adhesive ability. The authors developed quadratic polynomial models for chemotaxis in relation to temperature and salinity, predicting that the chemotaxis of *V. alginolyticus* strain can be directed toward the intestinal mucus under temperature and salinity conditions found at the farming site, suggesting that the intestine represents the leading entry portal. However, no conclusive pattern was found that could be applied to all tested isolates across different combinations of temperature and salinity.

Temperature (25°C vs. 15°C) also conditions *V. anguillarum* virulence when cultured under high- and low-iron availability [116]. The bacterium exhibits profound metabolic adaptations to grow under low iron conditions, including down-regulation of its energetic metabolism and induction of virulence-related factors, such as the biosynthesis of LPS, production of hemolysins and lysozyme, membrane transport, heme uptake, and the production of siderophores. However, chemotaxis, motility, as well as the T6SS1 genes, are expressed at higher levels at higher temperature (25°C vs. 15°C). By contrast, hemolysin RTX pore-forming toxin, T6SS2, and the genes associated with exopolysaccharides synthesis are preferentially expressed at 15°C. Biofilm formation significantly increases at 15°C. The siderophore piscibactin system is strongly upregulated at 15°C, but downregulated at 25°C, while the iron supply is maintained by the vanchrobactin siderophore system. Although the lower temperature reduces the growth of *V. anguillarum*, the virulence achieved for fish kept in cold water (15°C) is significantly higher than that observed in warmer water (25°C). On the other hand, other important factors, such as hemolysin Vah1, T6SS1, ferrous iron transport, and the vanchrobactin siderophore system, show higher expression at the optimal growth temperature (25°C). This potentially suggests that *V. anguillarum* produces a different cocktail of virulence factors depending on temperature, and that its virulence at a particular temperature is more related to the modulation of virulence factor expression than to its higher or lower capacity to grow. It appears that *V. anguillarum* virulence-related factors are up-regulated under low iron compared to high iron, either at 25°C or 15°C, with only two exceptions (i.e., chemotaxis-related (*cheWYAZ*) and motility-related (*fli* and *flg*) genes decreased under low iron). This suggests that the relative importance of each virulence factor for a particular fish species may vary depending on the water temperature.

The clinical hemolysins-positive *V. parahaemolyticus* strain RIMD22 upregulates the expression of genes implicated in adhesion (multivalent adhesion molecule-7, GlcNAc-binding protein A) and biofilm formation on abiotic surfaces (type IV pili, mannose-sensitive hemagglutinin, chitin-regulated pilins) when exposed to increased temperature [363]. The strain is viable but non-proliferative at 21°C, while it exhibits transient

expression of the targets at 27°C and upregulates them at 31°C. Similarly, the biofilm formation, quantified by SEM, is also significantly higher at 31°C. Importantly, the thermostable direct hemolysin involved in the pathogenesis of human vibriosis is also upregulated at 31°C (compared to 27°C and 21°C) in the 03:K6 strain isolated from a patient. Although this suggests increased upregulation of virulence factors at higher temperatures, the experimental design involved a short exposure to the temperature range, up to 6 h, which limits the predictability of the temperature dependency over a long-term period. However, the effect of the global temperature trend monitored since 1950 on the thermolabile hemolysin gene in 241 *V. parahaemolyticus* strains isolated from the seafood and subjected to whole-genome sequencing, supported the virulence evolution of the bacterium over decades. The study led to the identification of seven so-called "high-frequency mutation hot-spots" and two clinically specific sites that may be used in the future as biomarkers for tracking the GW-induced changes [173].

Temperature also affects the adherence of *F. columnare* to common carp gill tissues as demonstrated by an in vitro gill perfusion model [364]. The strain AJS 1, which exhibits high virulence and adhesion capability, forms abundant thread-like filaments on the gills and localizes at the tips of primary lamellae at higher temperatures (28°C vs. 17°C).

Streptococcus agalactiae, belonging to group B streptococci, with an optimum between 35°C and 40°C [365] shows a higher hemolytic activity at higher temperature (35°C vs. 28°C) [328]. Under the same regime, its virulence factors, *cylE* (b-hemolysin/cytolysin), which helps the bacterium to penetrate the host epithelial and endothelial barrier and to resist phagocytosis during invasion; *cfb* (CAMP factor), important in invasion; and *PI-2b* (pili-backbone), important in adhesion, are significantly upregulated. The bacterium also alters its phenotype so that a higher number of capsule-covered bacteria are present at higher temperatures, as the capsular polysaccharide prevents the cells from being recognized by leukocytes. In the Nile tilapia (*Oreochromis niloticus*), infection at 35°C results in 85% cumulative mortality, compared to 45% at 28°C, inducing an explosive inflammatory reaction in the host that results in a 30–40-fold change of *cyclooxygenase-2*, *IL-1β*, and *tnf-α* expression. In this case, the increased pathogenicity of *S. agalactiae*, mediated via the upregulation of specific virulence factors, in combination with an overwhelming host immune response, both contribute to the associated acute mortality observed at the higher temperature. Similarly, mortality in fish infected with *S. agalactiae* at 22°C starts 1 week post-challenge and reaches 100% in the second week, while mortality at 32°C begins 1 day post-challenge, reaching 100% within 3 days [329]. Although both studies corroborated the higher pathogenicity of *S. agalactiae* at higher temperatures, the latter presented no tangible numbers of fish involved. At the same time, the former measured the expression of only proinflammatory genes, leaving the potential expression of anti-inflammatory targets to balance the massive inflammation unresolved and therefore inconclusive.

P. damselae subsp. *piscicida* induces only 4% mortality in gilthead seabream (*Sparus aurata*) (60-day-old and 0.3 g) maintained at a constant temperature (15°C for 5 weeks), but sudden temperature shifts dramatically increase the mortality. Fingerlings kept at 15°C for 2 weeks, then at 18°C for an additional 2 weeks, and finally exposed again to 15°C for 1 week, suffer a 46% mortality rate. This is aggravated to 93% mortality if fish kept at 15°C for 2 weeks are exposed to 20°C for 3 weeks. Although this is probably associated with other confounding risks, such as the stress response and cannibalism, the practical output of the experiment highlighted the importance of maintaining the temperature in tanks below 18°C until the fish are vaccinated. Even though this will slow the growth rate, it will mitigate the temperature stress [366].

P. damselae subsp. *damselae* shows better growth and higher upregulation of virulence factors at higher temperature (25°C vs. 15°C) [102]. At 25°C the bacterium significantly upregulates genes involved in growth and virulence processes, such as DNA synthesis, nutrient uptake, chemotaxis, flagellar motility, secretion systems, antimicrobial resistance, and plasmid PHDD1-encoded virulence factors (i.e., putative adhesin/invasin OmpU, a transferrin receptor engaged in iron acquisition; a serum resistance protein (Vep07-like); and a defense against competitor proteins). In contrast, this temperature induces the downregulation of transcription factor RpoS, as well as genes participating in the cold shock response, modulation of the cell envelope, and amino acid metabolism. These genes are generally involved in functions related to the cell envelope, metabolism, and stress response. Unexpectedly, growth at 25°C does not upregulate expression of the major cytotoxins of *P. damselae* subsp. *damselae*, Dly cytotoxin (damselysin), which is one of the 10 most expressed genes at 15°C. This may indicate that damselysin expression at 15°C alone is enough to trigger mortality, even when the expression of the other four major cytotoxins is lacking. However, the onset of *P. damselae* subsp. *damselae* outbreaks in fish farms under increased seawater temperatures is likely influenced by the upregulation of other cellular processes, that is, higher division rate, motility, chemotaxis, and other plasmid-encoded putative virulence factors.

The fish and human isolates of *L. garvieae* differentially express genes when cultured at their optimal temperatures (i.e., 18°C and 37°C, respectively), and the transition to 37°C minimally perturbs the core gene expression in both isolates [367]. Although the fish isolate in vitro upregulates its virulence factors at 18°C rather than at 37°C, the outbreaks in the field suggest that their expression occurs at 25°C, highlighting the thermal adaptability of the bacterium [107]. However, the recent attribution of lactococcosis to three very similar bacteria, *L. garvieae*, *L. petauri*, and *L. formosensis*, which has led to misidentification in some clinical cases [368], probably caused incorrect designation of host and geographical ranges in the past. Nonetheless, mortalities in cold-water fish (chinook salmon *O. tshawytscha* and rainbow trout) by *L. petauri* are exacerbated at > 18°C. In contrast, warm-water fish (tilapia, *Oreochromis niloticus*, white sturgeon, *Acipenser transmontanus*, ornamental koi, *Cyprinus rubrofuscus*) show no mortality at the tested temperatures [369], which is their optimum physiological temperature. Further studies are necessary to identify and differentiate temperature-related virulence factors among *Lactococcus* spp.

Association between different temperatures and fish bacteria important for aquaculture in terms of mortality, clinical signs, and/or histopathology is presented in Table 6.

TABLE 6 | Association between different temperatures and virulence factors involved in the pathogenesis of fish bacteria important for aquaculture.

Bacterium	Disease	Aquaculture host	Temperature (°C)	Testing system	Effect on pathogen	References
Gram-negative bacteria						
Aeromonas salmonicida	Furunculosis	Salmonids (Atlantic salmon, rainbow trout)	25 (vs. 20)	In vitro	Plasmid rearrangements, loss of A-layer protein and proteolytic activity, loss of Type III secretion system (TTSS)	Daher et al. [132]
Flavobacterium columnare	Columnaris	Steelhead trout (*Salmo gairdneri*), coho salmon (*O. kisutch*), chinook salmon (*O. tshawytscha*)	20 (vs. 12 or 17)	In vitro (gill perfusion), in vivo	Stronger adhesion to gills, formation of abundant thread-like filaments on the gills, and localizes at the tips of primary lamellae	Decostere et al. [364]
		Rainbow trout	23 (vs. 18)	In filed	Higher virulence that increased the mortality %	Suomalainen et al. [164]
Photobacterium damselae subsp. *damselae*	Pasteurellosis	Gilthead seabream	25 (vs. 15)	In vivo	Upregulation of nutrient transport genes, Fadl, lipase, NupC, RNR, porins, trypsin, hsp, peroxidases, flagellin proteins, transcriptional regulators (XRE, DeoR), T2SS, T6SS, OmpU-like, Vep07-like, TolC AcrAB system, Vep20-like. Downregulation of Opp, glycine betaine transporter, operon VDA001578, 1579, VDA001580, cyanophycin, cold shock protein VDA003169, DedA superfamily protein, PlpV, GABA, RpoS. PlsC, Nqrm, MsrPQ, Krebs cycle genes.	Matanza and Osorio [102]
Photobacterium damselae subsp. *piscicida*			20 (vs. 15 or 18)		Increase to 93% of mortality (vs. 4% or 46%, respectively)	Magariños et al. [366]
Vibrio alginolyticus	Vibriosis		37, pH 6, 1% NaCl	In vitro	Presence of peritrichous flagella and swarming	Ulitzur [359]
			37, pH 7.2, 3% NaCl			
			28–39, 0.6% NaCl		Presence of peritrichous flagella, no swarming	

(Continues)

TABLE 6 | (Continued)

Bacterium	Disease	Aquaculture host	Temperature (°C)	Testing system	Effect on pathogen	References
Vibrio anguillarum	Vibriosis		25 (vs. 5 or 15)	In vitro	Higher chemotactic response to serin (sheathed polar flagellum), increased swimming speed	Larsen et al. [360]
			25 (vs. 15), low or high Fe	In vitro	Intensive chemotaxis, motility, upregulation of T6SS1 genes, hemolysin Vah1, ferrous iron transport, and vanchrobactin siderophore. Downregulation of hemolysin RTX pore-forming toxin T6SS2, siderophore piscibactin and exopolysaccharides-associated genes, decreased biofilm formation	Lages et al. [116]
Vibrio harveyi	Black band disease	Corals	30 (vs. 24)	In vitro	Increase production of quorum-sensing signal molecules: AHLs	Bhedi et al. [356]
Vibrio anguillarum, *V. alginolyticus*	Vibriosis	Gilthead seabream	27 (vs. 15 or 22)	In vitro (skin mucus)	Higher adhesion	Bordas et al. [362]
Vibrio spp., Vibrio harveyi, V. fischeri, V. tubiashii					No differences in chemotaxis, and/or adhesion	
Vibrio parahaemolyticus			31 (vs. 21 or 27)	In vitro	Stronger biofilm formation, upregulation of adhesion genes: multivalent adhesion molecule-7, GlcNAc-binding protein A, and biofilm genes: type IV pili, mannose-sensitive hemagglutinin, and chitin-regulated pilins.	Billaud et al. [363]
		241 seafood isolates (fish, crabs, and shellfish)		Long-term data analysis (2005–2010)	Mutation in *tdh*, *tdh*-related hemolysin, efflux pump class (ABC antibiotic efflux pump, RND antibiotic efflux pump), β-lactamase resistance, and fluoroquinolone antibiotic resistance, CARB.	Zhang et al. [324]

(Continues)

TABLE 6 | (Continued)

Bacterium	Disease	Aquaculture host	Temperature (°C)	Testing system	Effect on pathogen	References
Gram-positive bacteria						
Lactococcus garvieae	Lactococcosis	Rainbow trout	18 (vs. 37)	In vitro	Upregulation of virulence factors RpoE, encoding the delta subunit of RNA polymerase, and three genes, encoding autolytic enzymes	Aguado-Urda et al. [367]
Streptococcus agalactiae	Streptococcosis	Nile tilapia	35 (vs. 28)	In vivo	Higher hemolytic activity via upregulation of virulence factor cylE, cfb and PI-2b, higher abundance of capsule-covered cells	Kayansamruaj et al. [328]

Abbreviations: AHLs: short- to medium-chain acyl homoserine lactone; CARB: carbenicillin-hydrolyzing β-lactamase; Cfb: CAMP factor; cylE: b-hemolysin/cytolysin; DeoR: deoxyribose operon repressor; FadL: long-chain fatty acid transport protein; GABA: gamma-aminobutyric acid; hsp: heat shock proteins; MsrPQ: methionine sulfoxide reductases system; Nqrm: Na+-NQR maturation; NupC: nucleoside permease; OmpU-like: outer membrane protein U; Opp: oligopeptide permeases; PI-2b: pili-backbone; PlpV: phospholipase; RNR: ribonucleotide reductase; RpoE: RNA polymerase delta factor; RpoS: RNA polymerase sigma factor; T2SS: type II secretion system; T6SS1: *Vibrio parahaemolyticus* type VI secretion system 1; tdh: thermostable direct hemolysin; *tlh*: tdh-related hemolysin (trh); TolC AcrAB: multidrug efflux pump with outer-membrane channel TolC and transporters AcrA and AcrB; Vep07-like: serum resistance protein; Vep20-like: transferrin receptor; XRE: xenobiotic response element.

5.4 | Parasitic Pathogens

Chaianunporn and Hovestadt [370] suggested that interspecific interactions, such as parasitic interactions, may play a crucial role in accurately predicting responses to GW, because the evolutionary adjustment of temperature preference (optimal habitat) is slower in parasitism than in the commensalism scenario. Consequently, parasitism selects for higher temperature tolerance (or niche width) and increased dispersal. This aligns with a model where limited parasite dispersal favors lower parasite growth rates and, hence, reduced virulence because it (1) decreases the direct benefit of producing offspring (dispersers are worth more than non-dispersers, because they can go to patches with no or fewer parasites), and (2) increases the competition for hosts experienced by both the focal individual ("self-shading") and their relatives ("kin-shading") [371]. Therefore, this demonstrates that reduced virulence can be understood as an individual-level adaptation by the parasite to maximize its inclusive fitness. However, for aquatic parasitic organisms, there is no conclusive evidence that temperature affects changes in virulence. Still, the higher infection intensity (here considered as the average number of parasites in an infected population) consequential to the higher temperature induces stronger pathogenicity, a stronger immune reaction, and consequently stronger damage to the tissues. Concomitantly, this may be complicated by secondary bacterial infections, which can lead to a confounding increase in mortality. Interestingly, a higher temperature (37°C) negatively affected the survival of infective larval stages of the nematode *Anisakis pegreffii* when introduced into a non-target final host, even though the same high temperature in the target host triggers larval molting and development into the adult stage [372]. Authors assumed that, in addition to temperature and other physicochemical conditions (pH), successful infection is also mediated through interaction with the host-associated microbiome. More studies capitalizing on molecular tools to prospect for temperature-mediated virulence factors in parasites would help fill the gaps in knowledge that are lagging behind those of other pathogens.

5.4.1 | Protist

The prevalence of *Ichthyophthirius multifiliis*, the causative agent of the whitespot diseases in mosquitofish (*Gambusia holbrooki*) is not affected by temperature or UVB; however, an interaction between high UVB and high temperature (25°C vs. 18°C) caused a threefold increase in whitespot intensity (i.e., approximately 18 parasites per infected fish were counted at 18°C, compared to 57°C at 25°C) [373]. Since the study focused on the susceptibility of the host to the ciliate, the absolute values for the parasite infection parameters were not provided. Additionally, the interchangeable use of terms'intensity' and 'abundance' introduced bias in data interpretation.

Neoparamoeba pemaquidensis, the causative agent of AGD elicits a stronger pathology at the higher temperature (15°C), probably due to a higher associated number of amoebae [195]. The experiment challenged a group of Atlantic salmon smolts (150 g) with *P. perurans* (500 cell/L) at 10°C, which reached and remained at a median gill score of 2 (based on McCarthy et al. [374]) and exhibited 70%–90% gill coverage without

impairment. The second group was held at 15°C after being acclimated at a rate of 1°C/day increase over 5 days, 10 days prior to the amoeba challenge. Three weeks post challenge in the latter group, the gill score reached 3, consisting of multifocal stratification, multifocal hyperplasia, multifocal lined up of mucous cells, multifocal fusion of lamellae, focal fusion of filaments, focal spongiosis, focal to multifocal vesicles or lacunae, multifocal epithelial and general hyperplasia, focal to multifocal epithelial lifting and desquamation, focal to multifocal necrosis, focal to multifocal circulatory disturbance indicating a mild inflammatory response, showing 50%–70% gill without impairment.

5.4.2 | Myxosporidae

In parasitic cnidarians, arrested development at low temperatures has been widely recorded. Infection with *E. leei* in cultured tiger puffer (*Takifugu rubripes*) progressed with an increase in temperature: prevalence reached 80% at 20°C and increased to 100% at 25°C on the 19th day post-exposure, compared to 0% at 15°C. However, the study examined only five fish in the experimental group (out of a total of 20 fish) [254]. The effect of varying versus constant temperature increase was also evaluated. When the temperature was gradually increased from 15°C to 20°C, prevalence reached 33% on the 63rd day post-exposure. The prevalence was 0% at 15°C, while all fish kept at 20°C were dead by day 63, indicating a delay in the disease rate when the temperature is gradually raised. When the temperature was increased from 10°C to 20°C, the prevalence reached 29%. At a constant temperature of 10°C, it reached 11%, and at a constant temperature of 20°C, it reached 77%. Consequently, increased prevalence was followed by more intensive tissue damage. Exfoliation of the intestinal epithelium was evident on the 19th day post-exposure at 20°C and 25°C, but was stronger at 25°C on the 26th day post-exposure, sometimes completely detaching and abundant with various developmental stages. In contrast, *E. fugu* did not cause any pathology. *E. leei* challenge in gilthead seabream at an average of 25.6°C, simulating the natural summer temperature, resulted in a 100% prevalence 1 week post-infection, in contrast to 58.3% at a constant 18°C 1 week post-infection [245]. Interestingly, due to higher production of specific IgM at higher temperatures, the progression of the cnidarian along the intestine was limited, leading to lower intensity of infection [the latter was evaluated semi-quantitatively on Giemsa-stained histological sections using a scale from 1 (lowest) to 6 (highest), as previously described] [253], suggesting that opposing forces and additional circumstances, for example, longer time of exposure and fish density, need to be considered when evaluating the interaction between temperature and pathogenicity.

The mortality of the rainbow trout in vivo infected with *T. bryosalmonae* at 12°C, 14°C, 16°C, 18°C, or 19°C reached 100% at 5–14 days post-exposure. Although mortalities differed among temperatures, the parasite load was significantly different only between 12°C and all other temperatures [223]. Cumulative mortality at 14°C on the 49th day post-exposure was 35.7% (0% in the control), at 16°C, it was 45.5% (0% in the control), and at 19°C, it was 85% (5.9% in the control group). Pathology was more prominent at 19°C; the kidney-swelling index increased significantly over a longer time (42nd day) compared to 14°C and 16°C (35th day). To test the effect of a sudden temperature shift, the same authors performed an additional experiment in which they exposed the fish to river water for 5 days, without adapting them to laboratory conditions, and then exposed them to a more pronounced temperature difference. Those exposed to 18°C suffered 77.1% mortalities, and at 12°C, only 5.6% [223]. Similarly, kidney lesions were more pronounced at 18 than at 12°C, potentially linked to a more intense immune reaction. Namely, the latter leads to a proliferation of interstitial tissues and a regression or displacement of glomeruli and tubules, which likely causes kidney dysfunction [223]. Such dysfunction at high temperature that itself increases the fish need for water excretion and elevates the metabolism, and is additionally burdened by an increased oxygen demand, would probably compromise the physiological osmoregulation, and/or hematopoiesis. The result is damaged kidney tissues, as evidenced by the marked anemia in the clinical phase of proliferative kidney disease.

5.4.3 | Platyhelminthes

Diplostomum spathaceum belongs to the Trematoda and exhibits an indirect life cycle in freshwater [375]. The development of eggs, infection of the snail, and the release of cercaria are all continuous processes, with more generations occurring when the summer temperature exceeds 10°C. The peak of cercarial release and transmission to fish in Finland is through June–August, while with the drop in temperature in autumn, the development of metacercaria in fish is inhibited [375]. Authors suggested that under GW conditions, the prolongation of the season for snail development, cercarial release, fish transmission, and/or increased snail populations in August–September could facilitate the adaptation and colonization of new paratenic hosts, in addition to increasing the incidence in fish. Pathological changes in aquaculture fish, such as cataract, blindness, and secondary bacterial infections, are intensity-dependent. Therefore, a higher intensity of cercaria over a longer period (late autumn) could induce heavy infections and higher fish losses, necessitating limnocides treatment twice a year.

In the greater amberjack, the monophystocotylean *N. girellae* exhibits a lower intensity at 30°C, compared to 20°C and 25°C at 16 days post-exposure, attributed to a shorter life span of the adult under high temperatures [204]. Larvae and adults seem to adapt differently to the increase in temperature. The extrapolated mean intensity of *N. girellae* larvae 13 days post-exposure was zero at 20°C, 2.4 at 25°C, and 3602 at 30°C; however, the extrapolated mean intensity of adults 13 days post-exposure was 124 at 20°C, 124 at 25°C, and 59 at 30°C. Contrastingly, the pathological effect on the fish was exacerbated at higher temperatures. Assessed by measuring epidermis thickness in histological sections, a marked thinning was observed at 25°C and 30°C, and no thinning at 20°C. Authors suspected that such inconclusiveness could also be due to the measurement of thickness in histology-processed samples. However, the thickness varied within a particular temperature over 16 days post-exposure, for example, when sampled 8 days post-exposure, it decreased in thickness by increase in temperature, while on day

12 and 16 post-exposure, it decreased from 20°C to 25°C, only to become thicker on the 16th day at 30°C compared to 25°C. This was probably due to a reduced infection level at the end of the experiment. The association between different temperatures and fish parasites important for aquaculture in terms of mortality, clinical signs, and/or histopathology is presented in Table 7.

5.4.4 | Copepoda

The crustacean *Argulus coregoni* exhibits a direct life cycle in freshwater fish, with a peak of transmission in the wild occurring in summer. However, in Finland, the copepod exhibits a "cohort-type" transmission, producing one generation per year, and displays facultative diapause, where the first batch of eggs hatches normally, and the second batch takes more time [375]. Namely, eggs overwinter and hatch after the temperature increases above 10°C, so that the highest abundance of *A. coregoni* is found in fish in May–July. Subsequently, the parasite detaches and lays eggs from early July up to September, and the parasite abundance in fish declines. The extended period of eggs hatching from May to September is considered a bet-hedging strategy, that is, the parasite's decreased fitness in its typical conditions in exchange for increased fitness in stressful conditions, rather than the production of multiple generations. In contrast, in Japan, where the water temperature is higher, there are two generations per year, suggesting that under the GW scenario, the population cycle will become more rapid. Hakalahti et al. [375] also highlighted that more parasites inducing skin damage and stress reactions cause more frequent secondary bacterial infections (*F. columnare*) and indirectly increase mortality. Based on observed transmission and hatching patterns, authors highlighted the importance of egg destruction in the farm and treatment by emamectin benzoate to be performed twice per year to mitigate the cohort-type transmission, taking into consideration the environmental concerns.

6 | Future Perspectives and Mitigation Strategies

Climate change adaptation is a broad term that encompasses efforts to mitigate the adverse effects of CC and entails modifying policies and behaviors in response to observed or predicted climate changes [21]. Risk management techniques or practices at different spatial and temporal levels are crucial for mitigating the negative effects of CC on cage farming, and adopting a combination of technical, economic, and social risk management measures. Climate-smart technologies in agriculture are seen as strategies with extensive mitigation and adaptation potential that have been established in many countries worldwide; however, their implementation depends on the suitability of the technology to the region, people's perception, economic viability, and technical complexity [376]. Similar to agriculture, climate-smart aquaculture (CSA) seeks to improve food security while addressing the need to adapt and develop methods that mitigate the effects of climate change. CSA encompasses various strategies designed to foster sustainable food production, mitigate the sector's vulnerability to CC impacts, and enhance its resilience to cope with the effects of climate variability, ultimately contributing to greenhouse gas emission reductions throughout the entire production system and aquaculture value chain. The increased sea surface temperature in European water bodies is likely to be tolerated by most pathogens described in this review; moreover, these pathogens are expected to increase their incidence and pathogenicity (Table 3). Indeed, several of the discussed microbial pathogens affecting important European farmed finfish species are likely to be more pathogenic due to climate warming in the coming years. The temperature of the water will not only increase, but also undergo important and rapid oscillations, which most fish, due to their poikilothermal condition, will find difficult to resist.

The aquaculture industry should be prepared to develop new strategies, tools, and management practices to mitigate the impact of climate change and, more specifically, the increase in seawater temperature.

6.1 | Infrastructure Alterations

In marine cage farming, if water temperatures reach certain levels, the only realistic option is to relocate the cages to geographical areas with lower temperatures, depending on the site's characteristics and presence of thermoclines. This possibility is only feasible for certain offshore mobile farms, but not for classical coastal fish farms, which use cages moored on the bottom and depend on a specific administrative coastal concession. Alternatively, submerged cages [377] or cages that force the fish to move to the bottom of the cage, using a complementary system of nets in the upper part of the cage to avoid the typically warmer surface water layers [378], could be used. However, in several cases, this strategy resulted in suboptimal conditions for production and welfare [379]. Fish with closed swim bladders, such as European seabass or Atlantic cod, are more suitable for submerged culture than fish with open swim bladders, such as salmonids. However, further research is needed to learn how to combine submerged culture with favorable environmental conditions to improve fish growth and welfare throughout a commercial production cycle [377].

In more sophisticated cage designs with closed containment, water can be pumped from higher depths, although these designs can only operate in areas protected from bad weather conditions. For semi-intensive or intensive flow-through ponds or raceways, the alternative could be to change the water source. Usually, underground phreatic water is colder than surface temperatures, but it highly depends on the geological characteristics of the site and the depth of the wells. In addition, these phreatic waters may have different physical and chemical characteristics, but their cooling capacity can be used through a heat exchanger system.

Additional options include recirculation aquaculture systems (RAS), integrated multi-trophic aquaculture (IMTA), and aquaponics systems that operate with recirculation. Although GW can also affect these systems, the lower dependency on water renewal and the ability to incorporate water temperature control can result in improved temperature stability. This temperature control can be implemented through the most efficient water cooling/heating systems and can also be complemented by the thermal insulation of the facilities. One of the main handicaps for cooling or heating water is its high specific heat capacity

TABLE 7 | Association between different temperatures and pathogenesis of fish parasites important for aquaculture in terms of mortality and clinical signs and/or exerted histopathology.

Parasite	Disease	Aquaculture host	Experimental host	Temperature (°C)	Testing system	Effect on host	Mortality (%)	Clinical signs and/or histopathology	References
Ciliophora									
Ichthyophthirius multifiliis	White spot		Mosquitofish	25 (vs. 18)	Experimental in vivo	No effect	N/A	N/A	Cramp et al. [373]
				UVB + 25 (vs. UVB +18)		3-fold intensity increase			
Amoebozoa									
Neoparamoeba pemaquidensis	Amebic gill disease	Atlantic salmon	Atlantic salmon	15 (vs. 10)	Experimental in vivo	30%–50% gill impairment	N/A	Gill score 3: multifocal stratification, lamellar fusion, focal filament fusion, focal spongiosis, focal to multifocal vesicles or lacunae, multifocal epithelial hyperplasia	Benedicenti et al. [195]
Myxosporidae									
Enteromyxum fugu	Enteromyxosis	Tiger puffer	Tiger puffer	20 (increase from 10)	Experimental in vivo	100% P	N/A	No pathology	Yanagida et al. [254]
				10 (constant)		100% P			
				15 (only above *s)		60%			
				20 (constant)		100% P			
				20 (increase from 15)		100% P			
				25 (increase from 20)		100% P			
Enteromyxum leei	Enteromyxosis	Tiger puffer Gilthead seabream	Tiger puffer Gilthead seabream	20 (increase from 10)	Experimental in vivo	29% P			Yanagida et al. [254]
				10 (constant)		11% P			
				15		0% P			
				20 (constant; only above *s)		77% P (19th dpe)		Exfoliation of intestinal epithelium	
				20 (increase from 15)		33% P	100% on 63rd dpe	Complete epithelial detachment, abundant with parasite	
				25 (increase from 20)		100% P (26th dpe)			

(Continues)

TABLE 7 | (Continued)

Parasite	Disease	Aquaculture host	Experimental host	Temperature (°C)	Testing system	Effect on host	Mortality (%)	Clinical signs and/or histopathology	References
		Gilthead seabream	Gilthead seabream	25.6 (average)		100% P (7 dpi)		Lower infection intensity and limited expansion along the intestine (due to higher IgM)	Picard-Sánchez et al. [199]
				18		58.3% (7 dpi)			
Tetracapsuloides bryosalmonae	Proliferative kidney disease (PKD)	Rainbow trout	Rainbow trout	14 (vs. 12)	Experimental in vivo		35.7% (49th dpe)		Bettge et al. [223]
				16 (vs. 12)			45.5% (49th dpe)		
				19 (vs. 12)			85% (49th dpe)	Increased kidney-swelling index	
				18 (directly from river)			77.1%	Proliferation of the interstitial tissues, regression or displacement of glomeruli and tubules, causing kidney dysfunction	
				12 (directly from river)			5.6%		
Monogenea									
Neobenedenia girellae	Neobenedeniosis	Greater amberjack	Greater amberjack	20	Experimental in vivo	0 m.i. (13th dpe) larvae 124 m.i. adults	No epidermal thinning Epidermal thinning Marked epidermal thinning		Hirazawa et al. [204]
				25		2.4 m.i. larvae 124 m.i. adults			
				30		3603 m.i. larvae 59 m.i. adults			
Trematoda									
Diplostomum spathaceum	Diplostomatosis	Common carp	Rainbow trout	> 10 (June–August, Finland)	In field study	More generations (peak of cercarial release and transmission)		Intensive cataract formation	Hakalahti et al. [375]
				< 10 (autumn, Finland)		Inhibition of metacercarial development			

(Continues)

TABLE 7 | (Continued)

Parasite	Disease	Aquaculture host	Experimental host	Temperature (°C)	Testing system	Effect on host	Mortality (%)	Clinical signs and/or histopathology	References
Copepoda									
Argulus coregoni	Argulosis	Rainbow trout, brook trout (*Salvelinus fontinalis*), common roach (*Rutilus rutilus*)	Rainbow trout (*S. trutta*), Atlantic salmon	10 (in Finland)	In field study (farm)	Single generation			Hakalahti et al. [375]
			Masu salmon (*O. m. masou*), rainbow trout	20 (in Japan)		Two generations			

Abbreviations: Dpe: days post-exposure; dpi: days post-infection; m.i.: mean intensity; P: prevalence; *s: sporulation.

(4.186 J/g °C), which results in a very high energy demand in systems such as the flow-through. In comparison, RAS systems have a lower need for water renewal due to the continuous reuse and reconditioning of water, resulting in significantly lower energy costs. New technologies that allow more efficient use and exchange of energy make temperature control in RAS-operated facilities much more feasible. If farms can also combine these strategies with local energy generation (solar, wind), the cost of the energy required to keep temperatures under control, even in a warmer scenario, could be reasonable.

6.2 | Species Diversification

Another potential strategy is to change farmed species according to the profile of the temperature changes. It is known that each fish species has specific thermal preferences and tolerances, so in the face of an expected increase in temperatures, it is advisable to adjust the target species accordingly. However, this is only possible in farming sectors with a relevant level of diversity, and not in sectors based on monoculture, such as salmon farming. In this regard, it is essential to note that Mediterranean fish farming primarily relies on three main species: European seabass, gilthead seabream, and meagre, which exhibit distinct thermal preferences and tolerances. For example, European seabass has a lower temperature tolerance regime than gilthead seabream [380]. In farming conditions, European seabass tends to experience more problems during temperature peaks, which are also related to lower oxygen concentrations. In contrast, gilthead seabream has a lower capacity to cope and perform at temperatures below 15°C, as demonstrated by the so-called winter syndrome, a condition that is no longer observed in the Mediterranean, likely due to GW. Meagre, being more resistant to higher temperatures, is claimed to have a larger thermal window or a better response to thermal stress [381, 382].

However, these premises must be taken with caution, since the window of thermal tolerance that a species can tolerate can be affected by prolonged exposure, as has been seen in the meagre, which, despite being tolerant of higher temperatures, can decrease its growth performance [383]. The substitution of farmed local species by other more adapted species is another possibility. Farming tropical farmed fish, such as cobia or tilapia, can be an option in some instances, but should not be recommended if these species can escape, establish, and reproduce in local aquatic ecosystems, becoming problematic exotic species. Other species may be able to prey on locals, compete for resources, or introduce new pathogens in naïve geographic areas. In these cases, farming in Europe may be advisable only under strict isolation (e.g., RAS) and with strains that are unable to reproduce. Several examples of thermotolerant invader fish species are recognized in marine and freshwater environments in Europe, particularly the lessepsian migrating fish that have been known to migrate in the Mediterranean [384, 385]. However, the risks associated with pathogen dispersion and transmission have not been thoroughly addressed.

6.3 | Selective Breeding and Epigenetics

There are also potential strategies to mitigate the risks associated with high water temperatures, such as reducing feeding

rates or lowering metabolic demand during these risk periods. However, as the primary target of fish farming is fish growth, this is not a sustainable solution. In contrast, selective breeding can facilitate the development of heat-tolerant varieties of aquatic species. Breeders' selection is the process of choosing breeding candidates to increase the occurrence of desired phenotypic, physiological, morphological and/or behavioral traits in a population through the accumulation of advantageous alleles [386]. It is a common practice in European aquaculture, particularly to enhance growth, carcass yield, delay maturation, or improve disease resistance. However, selecting for many traits at once is complex or difficult due to phenotypic variance that cannot be explained alone by genetics, but also by the environment and their interaction, that is, Genotype by Environment interaction (GxE), particularly relevant for polygenic traits. This concept has been explored to evaluate the environmental sensitivity of aquaculture species [387].

Genome-wide association studies (GWAS) are commonly used in aquaculture to identify genomic regions associated with commercially important traits, such as growth and disease resistance [388]. This method has already proven helpful in identifying quantitative trait loci (QTLs) associated with diseases in farmed species [389, 390], which can then be incorporated into selective breeding programs using marker-assisted selection (see Yáñez et al. [391] for a recent review). Yet, its use in relation to thermal tolerance is comparatively limited, with only a handful of studies having been carried out in aquaculture species [392–394]. Moreover, the already mentioned uncertainty about the direct consequences of GW, which can vary in different regions and environments, makes it difficult to decide on a clear target for selection, that is, tolerance to higher or lower temperatures. This uncertainty, coupled with the difficulties of selecting for several traits simultaneously, has led to the suggestion that selective breeding for robustness could be a better strategy for making farmed fish more resilient to environmental change, including GW, and disease [387]. This would imply selecting families that can maintain functionality in the face of extreme changes [395], although the practicalities of applying this strategy are unclear. Moreover, even in species with extensive genomic information (such as Atlantic salmon or Nile tilapia), the identification of specific genes associated with polygenic traits (such as disease resistance or temperature tolerance) remains elusive, even in genome-wide studies [396]. This can be attributed to a lack of consideration for the differential gene expression under local conditions, which can be controlled, at least partially, by epigenetic mechanisms [397]—an area already exploited in plant agriculture but still incipient in aquaculture [398].

Epigenetic mechanisms mediate changes in gene expression and function that do not involve DNA mutations [399, 400], and can influence offspring development, creating phenotypic variation that can be heritable across generations [401–404]. DNA methylation, histone modification, and non-coding RNAs are the best-studied epigenetic mechanisms, resulting in phenotypic variation that is free from the limitations typically associated with genetic inheritance. For example, epimutations induced by the environment can simultaneously arise in different individuals of the population [405], providing a plastic response to the environment that can prepare the offspring for the conditions experienced by their parents. In this way, epigenetic mechanisms can extend the range of tolerance to temperature stress, affecting not just one but several generations [406]. Experiments simulating temperature scenarios of climate change in coral reef fish have already revealed epigenetic signatures associated with within- and trans-generational plasticity in thermal tolerance and restoration of the aerobic scope, although the affected genes do not appear to be conserved across the geographical range of the species [407]. There are also numerous studies examining epigenetic regulation in response to high-temperature exposure [408], as well as the potential role of epigenetics in thermotolerance in animal farming [409], investigating the intergenerational effects of temperature, and the development of epigenetic markers to monitor the effects of temperature [410]. Previous work on salmonids has shown that early rearing conditions related to the farm environment, including temperature stress, result in epigenetic modifications that can have long-lasting effects [411]. Thus, environmentally modified phenotypes could be obtained by altering the rearing conditions, either at the juvenile stage or by conditioning the broodstock [398]. The incorporation of epigenetic markers into breeding plans, in combination with genetic markers such as SNPs [412] has the potential to accelerate the adaptation of stocks to different thermal conditions, expanding the scope of selective programs focused on genomic improvements for commercial traits [413, 414]. For this, epigenetic biomarkers could be used for both the diagnosis and prognosis of individual fish performance under specific thermal conditions, as well as to predict the potential long-term consequences of exposure to extreme temperatures during early development [410].

The relationship between epigenetics and immune response in fish is also well established. For example, non-coding RNAs have been shown to be involved in the modulation of gene expression in response to *A. salmonicida* infection in Atlantic salmon [415], and vaccination of turbot against the same pathogen also results in methylation changes [416], opening an avenue to identify diagnostic and prognostic biomarkers that could be combined with those for thermal resistance.

From the point of view of host-pathogen interactions, epigenetic mechanisms are not only involved in the phenotypic plasticity of the host but also control the plasticity of parasite life-history traits. Pathogen-induced effects can have transgenerational consequences for the host [417], and, critically, the interaction between host genetics, environment, and parasite loads can influence epigenetic diversity [418]. The potential of epigenetics goes beyond providing a mechanistic explanation for tolerance to extreme conditions or for host and pathogen plasticity. Thus, in the context of aquaculture, epigenetics could (a) explain phenotypic plasticity, (b) explain some of the missing heritability, (c) constitute a non-pharmacological approach to disease resistance, (d) be the basis for programming (i.e., epigenetic programming) and (e) be added on top of classic genetic selection [398]. While epigenetics is not yet being specifically used to achieve thermotolerant or disease-resistant species, it represents a promising avenue that warrants further exploration, particularly in Mediterranean species, some of which are understudied in this area. Further studies should focus on species-specific developmental plasticity to clarify the thermal limits within which juveniles can be adapted in the hatchery before being exposed to offshore environmental temperatures. Good models for

studying adaptability to extreme changes are species of euryhaline fish, such as the killifish (*Fundulus heteroclitus*) (Shaw et al. 2014), or naturally inbred species, like the mangrove killifish, for which the relative roles of genetic background and environmental factors can be disentangled from epigenetic effects [419].

6.4 | Welfare Assessment

Rather than being a relevant tool in mitigating CC effects, welfare assessment becomes crucial for controlling CC's detrimental effects. Among the Operational Welfare Indicators (OWIs) described and used in fish farming, the so-called environmental OWIs are represented by the classical physico-chemical water quality parameters. Temperature and pH are the two primary physical parameters, and most other parameters (dissolved gases, salinity, ionic load, etc.) are closely correlated with temperature. Therefore, in many cases, temperature oscillations should be considered holistically in conjunction with the other parameters. For this reason, other OWIs, especially those related to behavior, are of paramount importance for the early detection of undesirable effects of temperature increases on fish welfare [378, 420].

6.5 | Impacts in Therapeutics, Antimicrobial Resistance and Immunoprophylaxis

Disease treatments, primarily targeting bacteria and parasites, can also be impacted by GW. As mentioned before, temperature can affect most aquatic pathogens, as well as the metabolism and immune system of farmed aquatic organisms. Therefore, the temperature during treatments is also very relevant to the results, particularly in terms of efficacy and safety. In fish therapeutics, treatments are typically administered orally with antimicrobial substances or in baths, primarily targeting ectoparasites [421, 422]. Antimicrobial treatments are delivered through medicated feeds, and these treatments can also be affected by temperature. The water temperature affects appetite; at certain higher temperatures, appetite may decrease, and consequently, feeding rates should be reduced [423]. In addition, the pharmacokinetics and pharmacodynamics of the medicines used in treatment can also be significantly affected, as all the metabolic processes involved in digestion, absorption, distribution, and detoxification are highly dependent on temperature [424]. Therefore, the recommended dosing values at lower temperatures may not be applicable at higher temperatures. In bath treatments, the chemical reactivity, stability, and permanence in the environment of certain products used as therapeutics, such as oxygen peroxide [425], formalin [425], or paracetic acid [426] can change at certain temperatures. Under the GW scenario, these substances tend to be more reactive and unstable, so the outputs in terms of drug efficacy can change, and moreover, can introduce a high risk for safety levels in fish. The AMR, derived from aquaculture practices and also due to terrestrial animal farming and human uses, is one of the main environmental concerns nowadays. As stated before, the high occurrence of diseases due to CC can further exacerbate the improper use of antimicrobials [427]. Several authors have already addressed the environmental impacts of treatments in Mediterranean aquaculture [428, 429]. These are expected to increase with CC if more efficient treatments, adjusted to the sensitivity of each pathogen, the characteristics and metabolism of each fish species, and its environment, are not developed.

The negative effects of temperature increase can also be observed in fish prevention, particularly in immunoprophylaxis. This is due to the significant role of temperature in the immune system of fish [33], especially when planning vaccination programs [430–432]. As previously commented, if fish are vaccinated at a temperature higher than the homeostatic temperature for the species, a lower performance of the immune system could be expected; therefore, a reduced efficacy of the vaccination could occur.

6.6 | Disease Modeling

A large body of literature over the last decades has demonstrated the relationship between CC and the disease dynamics. Therefore, climate modelling could be used to help prepare for future outbreaks of both terrestrial and aquatic diseases. Both CC and infectious disease epidemiology are complex systems, and effective modelling requires knowledge from different disciplines. Software tools and packages have been developed in various programming languages (R, Python, and Julia) from a range of perspectives and specializations, reflecting the numerous disciplines that contribute to this field [433]. The latter includes health geography, disease ecology, applied climate science, data science, epidemiology, and public health. A recent review identified as many as 37 fully developed software tools for modeling climate-sensitive infectious diseases; the majority of these were created for vector-borne diseases [433]. Few of the tools described above had a visually intuitive interface. Specifically, most tools were developed for geographical regions where the infectious disease of interest is currently endemic. While a tool for modeling climate–disease interactions in aquaculture has yet to be developed, it would certainly facilitate more efficient aquaculture management in mitigating the impacts of CC on fish pathogens.

Assessing the potential economic impacts of climate-driven fish diseases at the European or global level is a timely and relevant topic. However, the extensive literature review conducted for this study revealed that no publicly available data exist to enable a quantitative assessment of the burden of the selected diseases (Table 3), whether in the published or grey literature, at regional, national, or European scales. For notifiable diseases, information and data collected by national competent authorities through official surveillance programs—once authorized—can be compiled, curated, and analysed for these purposes. In contrast, for non-notifiable diseases, the situation is and will remain more complex, as data held by farms and companies are considered highly sensitive and confidential, and are rarely shared unless specific national or regional surveillance plans are in place. The complexity of such undertakings, particularly in relation to the concept of disease burden in European fish farm management, has recently been reviewed {Padrós, 2025 #1501}. This challenge has also been highlighted by the World Organisation for Animal Health (WOAH) through its GBADS project, as well as in the EUPAH&W initiative (https://www.eupahw.eu/projects/integrated-approach-including-socio-economic-aspects-of-animal-health-and-welfare/assess-the-econo

mic-and-societal-burden-of-selected-priority-diseases-and-production-diseases) and other related efforts. These initiatives may, in the future, help address the current scarcity of comprehensive assessments.

Author Contributions

George Rigos: conceptualization, writing, data curation, editing. **Francesc Padrós:** writing, data curation, editing. **Maria Constenla:** writing, data collection, editing. **Ana Jeroničić:** writing, data collection, data curation, analysis. **Dimitra Kogiannou:** data curation, editing. **Sofia Consuegra:** writing, editing. **Mikolaj Adamek:** writing, editing. **Ivona Mladineo:** writing, data curation, editing.

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Conflicts of Interest

The authors declare no conflicts of interest.

Data Availability Statement

The data that support the findings of this study are available on request from the corresponding author. The data are not publicly available due to privacy or ethical restrictions.

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Supporting Information

Additional supporting information can be found online in the Supporting Information section. **Table S1:** Complete search strategies.