



Review

Carp Pathogens of Recreational and Ornamental Aquaculture: What Lies Beneath the Surface?

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Abstract

The farming and rearing of freshwater fishes is increasing in scale in response to growing global food demands, but notably also through the increasing popularity of sport angling and expansion of ornamental hobbyist practices globally. Alongside rises in numbers of fish and the intensity of their rearing, increases in aquatic and ecosystem stressors have expedited the emergence and motility of many pathogenic diseases. Cyprinids are the most popular species for culturing, and although largely considered hardy, are susceptible to a diverse array of pathogens. Common carp (*Cyprinus carpio*) and ornamental koi carp (*Cyprinus rubrofuscus*) are valued for their ease of cultivation, fast growth rates, and variety of genetic appearances. Vaccination programmes have become a mainstay of industrial aquaculture where financially viable, but their use in fish reared as companion animals or for sport angling is not cost-effective and despite the magnitude and growth of these markets, much less is known about current threats and trends in this area of aquaculture. Here, we review the bacterial, viral, and fungal infections commonly found within carp bred for angling and ornamental aquaculture in the United Kingdom and Europe, alongside current threats such as antimicrobial resistance and the potential impact of climate change on temperature-dependent infections. We highlight the evolution of diagnostic techniques, alternative therapies to reduce antibiotic dependence, and the environmental impact of available treatments as priority areas for cyprinid research. Additionally, there is an urgent need for novel therapeutics in this area, as innovation is currently lacking.

Keywords: carp pathogens; aeromonas; pseudomonas; columnaris; edwardsiellosis; mycobacteriosis; cyprinid herpes virus; edema virus; viraemia; saprolegniasis; branchiomycosis



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1. Introduction

The practices of angling and aquaculture are intertwined in history, with no exact date for their conception many thousand years ago. Although ancient in origin, both have exploded in popularity in the past three decades and now represent significant sources of food, recreation, and also employment globally. Cyprinid species, most notably the common carp (*Cyprinus carpio*) and their ornamental counterparts (*Cyprinus rubrofuscus*), play a crucial role in recreational angling (the practice of angling/coarse fishing for carp, and the rearing of carp for angling) and ornamental aquaculture (the keeping and rearing

of koi carp as pet/ornamental pond fish). Carp are by far the most popular of freshwater fishes for recreational applications and have grown exponentially in popularity worldwide. They are highly valued for their fast growth rates, long life span (up to 70 years) strength, intelligence, companionship behaviour, adaptability, ease of breeding, and varied visual presentation. Individual specimens can fetch significant prices from both anglers and Koi keepers, often in the tens of thousands in angling, and even millions in koi rearing. However, although hardy, carp are susceptible to a diverse array of pathogens that can result in significant economic losses and heavily compromised fish welfare that can affect both disciplines [1]. People and carp are spending more time in close contact as both sport and hobby numbers rise, increasing the potential for the anthropogenic spread and emergence of disease.

Pathogen selection for this review was guided by relevance and prevalence to UK and European recreational and ornamental aquaculture. Agents with documented disease in common carp and koi within the aforementioned regions were prioritized, supported by peer-reviewed outbreak reports, surveillance data, and regulatory listings. Input from the UK Institute of Fisheries Management (IFM) guided our identification of pathogens viewed as major threats within carp angling and koi-keeping specialisms, thus retaining end-user as well as academic relevance. The selected pathogens should not, however, be interpreted as a complete list of bacterial, viral, fungal, or oomycete agents affecting carp; those reviewed here were selected for their applied relevance to fish welfare, disease management, and biosecurity within recreational aquaculture only. Evidence citing other cyprinids or non-carp species is included only where it provides relevant context and is not implied to infer direct evidence for effects in common or koi carp.

Pathogenic bacterial species such as *Aeromonas*, *Pseudomonas*, and *Vibrio* species pose significant threats to cyprinid health, causing diseases such as haemorrhagic septicaemia and vibriosis with high mortality rates [2]. Viral infections, including koi herpesvirus disease (KHVD) and spring viraemia of carp (SVC) are of particular concern due to their potential for devastating outbreaks [3], and fungal infections, such as saprolegniasis, can also be detrimental to cyprinid populations [4]. Despite the prevalence and impact of these pathogens, treatment options remain limited, and conventional approaches raise concerns about antimicrobial resistance, environmental damage, and toxicity issues [5]. As outbreaks of these pathogens occur often at elevated temperatures, there are global concerns regarding the effect climate change may eventually have on the prevalence of these diseases [6]. But climate is a complex issue; different pathogens respond to rising temperatures in different ways, with some preferring higher temperatures, some preferring rapid changes in temperature, and others preferring cooler conditions. Host-immune competence also displays temperature dependencies; therefore, climate is not explored in detail in this review. In addition, the threat posed by zoonotic pathogens remains an underacknowledged risk within aquaculture [7]. Potential pathogen transfer between carp and humans necessitates further research and comprehensive transmission mapping to mitigate the risk of zoonotic transmission.

Recent advances in molecular diagnostics, sequencing, and alternative disease-management approaches, including bacteriophage-based interventions, may improve the detection, understanding, and control of some carp pathogens [8,9]. However, these technologies are out of reach for most hobbyists and enthusiasts; hence, alternative therapies and tools are required to ensure adequate biosecurity and safety levels in the angling and ornamental disciplines.

This review will focus on the major bacterial, viral, fungal, and oomycete pathogens affecting common carp and ornamental koi in recreational angling and ornamental koi pond settings, with emphasis on pathogen biology, clinical presentation, transmission, diagnosis, prevention, and evidence-based disease management. It highlights knowledge gaps and the need for accessible, proportionate, and environmentally responsible strategies for UK and European carp systems.

Note on the literature search and scope: Search databases included PubMed, Google, Google Scholar, and Mendeley, accessed March 2026 to July 2026. Search terms included Carp/Koi pathogens, carp/Koi disease, carp/Koi infections, carp/Koi pathology, zoonotic transmission in carp, carp care treatments, carp vaccination strategies, antimicrobial resistance in carp, carp aquaculture. Papers were selected for inclusion primarily based on documented evidence of relevance to carp; where none existed but the potential relevance was clear, papers were included that covered the same pathogens in other fish species. A balance of original and emerging research was targeted, highlighting gaps in the literature as we perceived them as well as established or documented findings/observations of relevance to carp in the context of recreational aquaculture.

2. Bacterial Diseases

Carp may be affected by a diverse range of bacterial agents, with disease expression shaped by pathogen virulence, host susceptibility, co-infection, water quality, stocking density, handling, and other environmental stressors [10]. The bacterial groups considered in this review were selected for their relevance to recreational and ornamental carp systems in the United Kingdom and Europe and include *Aeromonas*, *Pseudomonas*, *Vibrio*, *Flavobacterium*, *Edwardsiella*, and *Mycobacterium* spp. These taxa should not be interpreted as the only bacterial agents capable of affecting carp; rather, they represent selected pathogens of practical relevance to fish welfare, disease management, and biosecurity in the defined scope of this review. Many of these pathogens are Gram-negative, with their prevalence being linked to the ability to form biofilms and therefore persist in harsh environments, although some Gram-positive bacteria such as *Streptococcus iniae* can cause equally devastating systemic infections in carp [11,12]. Pathogenic bacteria are most frequently found in water with high organic load, particularly the static water commonly associated with aquaculture growth ponds [13]. Outbreaks of such bacterial infections are often temperature-dependent, with higher mortality rates reported during the spring and summer months, and more sudden temperature changes further increasing the susceptibility of fish [1]. But pathogen-temperature relationships are complex and varied, in addition, environmental influences are also nuanced and complex, with interconnectivity often compounding outcomes, as illustrated in Figure 1, below.

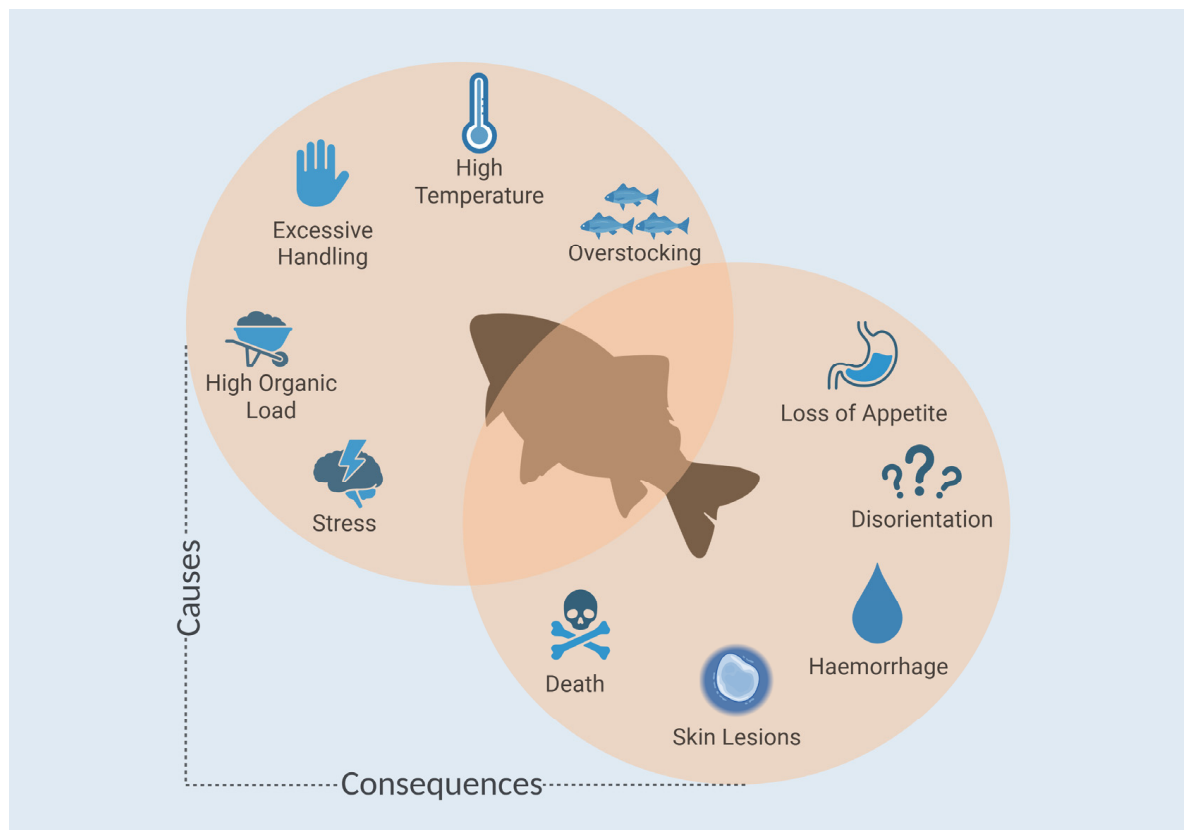


Figure 1. Illustration depicting the causes and consequences of disease within carp populations due to environmental and human factors.

3. Motile *Aeromonas* Septicaemia (MAS)

Also known as haemorrhagic septicaemia, motile *Aeromonas* septicaemia (MAS) is a syndrome associated with disease caused by motile members of the genus *Aeromonas*. *Aeromonas hydrophila* is frequently implicated, although other motile aeromonads, including *Aeromonas veronii*, *Aeromonas sobria*, and *Aeromonas caviae*, have also been associated with disease in freshwater and brackish-water fish [14]. *Aeromonas* species are found abundantly in polluted waters, particularly in areas where large amounts of organic waste are present. Aquaculture pond systems such as those used for the rearing of carp present optimal conditions for the growth and spread of *Aeromonas* due to readily available nutrients, therefore resulting in an ever-present threat to carp stock as a result of biofilm formation [15]. Devastating outbreaks of *Aeromonas* infections are common when carp are exposed to stress, often due to overstocking or handling. Temperature increases during the spring and summer also contribute to significant losses [16].

Motile aeromonads, such as *A. hydrophila*, are characterised by their use of single polar flagellum and therefore active motility. *A. hydrophila* is a Gram-negative, opportunistic bacillus that produces virulence factors including aerolysin, enterotoxins, cytotoxins, and various haemolysins, which bind to and disrupt cell membranes [17,18]. Mesophilic *Aeromonas* species possess two complex protein secretion mechanisms, the Type II secretion system (T2SS) and the Type III secretion system (T3SS), both crucial to the export of virulence factors and therefore pathogenicity. *A. hydrophila* infections are rarely primary, as infection mainly occurs in previously compromised hosts either as a result of stress or prior infection [18].

The clinical manifestation of MAS includes minor blood vessel rupture and haemorrhage, with ulcerative skin lesions due to the production of haemolysin and the resultant

lysis of red blood cells [19]. Whilst the aforementioned skin lesions are usually shallow, secondary fungal infections of the ulceration are common and often further complicate the diagnostic and treatment process [20]. Infected fish may show abnormal behaviours prior to any physical symptoms, including lethargy and loss of appetite. Abdominal swelling or edema may be observed due to internal haemorrhage and excess fluid, whilst external haemorrhage is often most clearly visible on the fins and periphery of the fish [1]. In some cases, sudden death will occur before the presentation of any characteristic clinical signs [21].

Control measures employed to mitigate large-scale *A. hydrophila* infection are largely dependent upon addressing the environmental factors that initially allowed the infection to spread. Historically, water temperature, stocking densities, and organic material levels were all considered important variables to be controlled where possible [1]. Where bacterial disease is suspected, antimicrobial treatment should follow veterinary assessment and, where practicable, bacterial culture and species-specific antimicrobial-susceptibility testing. Antimicrobial selection must account for the identity and susceptibility profile of the isolate, permitted medicines, local or national legislation, the intended use of the fish, and relevant withdrawal periods for fish entering the food chain [3,22]. A compounding difficulty here is the spread of antimicrobial resistance (AMR) genes in farmed and wild carp microbiomes. As with many other farming practices, Antimicrobial Stewardship remains a significant and globally important driver of best practice in treating carp in recreational aquaculture environments. Although antibiotic misuse is less prevalent in recreational aquaculture due to the relatively small number of veterinarians specialising in fish health in the UK and UE, carp microbiomes could theoretically still be exposed to the spread of resistance genes through other routes, such as feed sources and proximity to farming run-offs.

Aquaculture, whether for food fish or recreational purposes, can be treated as a potential source of environmental contamination with antimicrobial-resistant bacteria; where specific to carp, tetracycline resistance and treatment have previously been linked [23]. AMR surveillance is crucial to addressing ecological and environmental impacts of all forms of aquaculture, but simple, accessible, and portable solutions are only starting to emerge, and useful tools such as whole-genome sequencing, deep sequencing, and non-lethal sampling and monitoring methods are not always readily available to fishery/farm owners. However, without the development of novel treatment management strategies, *A. hydrophila* may become largely untreatable due to antimicrobial resistance, with studies already showing over 70% isolates with Multiple Antibiotic Resistance Index values higher than 0.2 [22]. Whilst three different vaccination approaches have been developed, recombinant, live attenuated, and outer membrane-based vaccines, the worldwide uptake has been significantly lower than anticipated [24], with many external economic and practical considerations exerting significant influence.

4. *Pseudomonas* Infections

Pseudomonas spp. are Gram-negative bacteria commonly found in freshwater environments, sediments, and on the skin and gills of fish. Several species have been associated with disease in cyprinids, including *Pseudomonas fluorescens* and *Pseudomonas aeruginosa*. Infection is often associated with stress, poor water quality, high stocking density, handling, and concurrent parasitic or bacterial disease. In common carp and ornamental koi, *Pseudomonas* spp. may act as primary or secondary pathogens and can contribute to outbreaks of septicaemia and ulcerative disease [25,26].

Clinical signs are non-specific and may include lethargy, reduced appetite, respiratory distress, darkening of the skin, haemorrhages, scale loss, fin erosion, skin ulceration, abdominal distension, and increased mortality. Internally, affected fish may show haemorrhages in

the liver, kidney, and spleen, with enlargement or congestion of these organs in more severe cases. Similar clinical signs can occur during infections with *Aeromonas* spp., *Flavobacterium* spp., and other opportunistic bacteria, and mixed infections may occur. For example, *P. fluorescens* has been reported in common carp affected by concurrent *Dactylogyrus extensus* infection, where affected fish showed anorexia, respiratory distress, dermal ulceration, and septicaemia [25]. Diagnosis is based on clinical history, gross pathology, bacteriological culture, and identification of isolates from internal organs or lesions. Isolation of *Pseudomonas* spp. from skin, gills, or water alone should be interpreted with care, as these organisms are widespread in the aquatic environment. Examination for parasitic disease, poor water quality, injury, and other bacterial pathogens is therefore important when investigating suspected outbreaks.

Control should focus on improving husbandry conditions, reducing handling stress, maintaining suitable water quality, removing dead and moribund fish, and quarantining new stock before introduction into established populations. Any antimicrobial treatment should be based on bacterial culture and antimicrobial-susceptibility testing, under veterinary direction and in accordance with national medicine-use requirements. Antimicrobial-resistance patterns can vary between isolates, including those obtained from carp farms, and empirical treatment should therefore be avoided where possible [26].

5. Columnaris Disease

Columnaris disease is caused by a group of closely related *Flavobacterium* species historically referred to as *Flavobacterium columnare*. The earlier literature may also use the names *Flexibacter columnaris*, *Bacillus columnaris*, or *Cytophaga columnaris*. Taxonomic revision has since resolved the former *F. columnare* complex into four species: *Flavobacterium columnare*, *Flavobacterium covaie*, *Flavobacterium davisii*, and *Flavobacterium oreochromis* [27]. Accordingly, the term *F. columnare* is only used here more generally when discussing older studies in which isolates cannot be assigned reliably to one of the currently recognised species. These species should not be assumed to be interchangeable or equally relevant to common carp and ornamental koi; host association, virulence, and geographical distribution may differ between taxa and isolates.

Columnaris-causing bacteria are Gram-negative, rod-shaped bacteria that show gliding motility rather than flagella-mediated motility. On wet mounts, bacterial cells may form characteristic column-like aggregates, although diagnosis should not rely on morphology alone [1,27]. The progression and severity of disease are influenced by bacterial strain, host species and age, stocking density, water quality, organic loading, dissolved oxygen, and temperature. In experimental work involving common carp and rainbow trout, highly virulent isolates caused rapid invasion and destruction of gill tissue, whereas lower-virulence isolates produced less severe disease; these findings should be interpreted as isolate- and host-specific rather than universally applicable to all columnaris-causing bacteria [28]. Other virulence-associated mechanisms, including secretion systems and adhesion-related traits, have been described, but much of this evidence is derived from non-carp hosts and laboratory isolates [29].

Columnaris disease is characterised by acute to chronic systemic infections first presenting on the skin and fins, and then the gills [1]. Age is a significant determining factor of both the progression and manifestation of columnaris disease, with acute development being most common in younger fish and older fish being subjected to a more chronic condition. Visible symptoms include ulceration of the mouth and skin lesions, both of which are highly susceptible to secondary infection by fungi or other bacteria. Gill necrosis is apparent in both acute and chronic cases, whereas fin deterioration and skin discoloration may only occur if the disease progresses in a chronic manner [28]. Clinical signs alone are

not specific to columnaris disease; diagnosis should therefore be supported by microscopic examination, culture and identification of the causative organism, and, where available, molecular methods capable of distinguishing members of the former *F. columnare* complex.

Management is key in the prevention of columnaris disease, with a lower rearing density and higher water quality consistently linked with a lower risk of *F. columnare* outbreak. Control of the amount of organic matter in the stocking ponds can help to minimise the adherence capacity of *F. columnare*, further reducing the likelihood of mortality [28]. Medicated baths and topical treatments such as methylene blue (methylthioninium chloride) baths or swabs have been used historically; however, there is no scientific evidence for this kind of treatment being beneficial to carp. On the contrary, we recently highlighted the potentially toxic effects of using methylene blue and other historically unevidenced chemicals for topical applications [30]. Vaccine candidates have been investigated for columnaris-causing bacteria, particularly in other aquaculture species, but their efficacy, regulatory status, and practical applicability to common carp and ornamental koi require further assessment.

6. Edwardsiellosis

Edwardsiella spp. are considered to be one of the greatest pathogenic threats to world-wide aquaculture [31]. Formerly known as *Edwardsiella tarda* and later reclassified into three distinct groups, *Edwardsiella piscicida*, *Edwardsiella anguillarum* and the redefined *Edwardsiella tarda*, these bacteria are highly virulent and abundant in freshwater and aquaculture environments alike [32]. The severity of *Edwardsiella* outbreaks is exacerbated by the intensive fish farming methods employed in commercial aquaculture, including the overuse of antimicrobial and antibiotic chemicals such as florfenicol and oxytetracycline [9]. As a result, *E. piscicida* has been found to carry antibiotic-resistant genes with the ability to transfer these through horizontal gene transfer [33]. Additionally epidemiological evidence has shown that *E. tarda* infections in fish have zoonotic potential, resulting in gastroenteritis in humans, particularly in immunocompromised patients [34].

The genus *Edwardsiella* comprises Gram-negative, rod-shaped bacteria that can grow in both aerobic and anaerobic environments [35]. All species are highly adaptable to changes in environmental factors including salinity, pH, and temperature [36]. Whilst its virulence factors are not yet fully understood, *E. tarda* is currently considered to exhibit the highest pathogenic potential due to its ability to produce toxins including haemolysins and dermatotoxins, and resistance to serum- and phagocyte-mediated killing [35]. Further research into the virulence genes associated with *Edwardsiella* spp. is essential for understanding the threat edwardsiellosis poses to aquaculture.

Clinical manifestation of edwardsiellosis varies between strains; however, abnormal swimming behaviours such as spiralling and remaining closer to the surface, alongside visible lethargy, are often the first symptoms observed [1]. During the initial stages of infection, skin necrosis and haemorrhaging both under and on the surface of the ventral side of the body and fins becomes apparent, followed by the progression of surface lesions and further internal deterioration. In more advanced cases, organ haemorrhages and abdominal edema can be observed upon dissection [36].

When bacterial infection is suspected, any antimicrobial treatment should be guided by veterinary assessment, bacterial culture, correct species identification, and antimicrobial-susceptibility testing. Recently, phage therapy has started to emerge as a potential alternative to antibiotics, with promising results being shown for both infection control and reduction in antibiotic resistance genes in zebrafish infected with *E. piscicida* [9]. Phyto-biotics and vaccination protocols both show theoretical promise for use in aquaculture; however, more research is needed to understand their efficacy and practical application [37].

7. Mycobacteriosis

Colloquially referred to as “fish tuberculosis”, piscine mycobacteriosis is a disseminated mycobacterial infection that was first isolated from carp in 1897 [38]. Mycobacterial infections in fish are caused by three primary etiological agents: *Mycobacterium marinum*, *Mycobacterium fortuitum*, and *Mycobacterium chelonae* [1]. *Mycobacterium* spp. are endemic worldwide, with a host range of over 160 species of freshwater, saltwater, and brackish fish species, posing a significant threat to both aquaculture and wild fishing alike [39]. The elevated temperatures and lowered pH frequently associated with intensive fish farming provide hospitable conditions for the overgrowth and outbreak of *Mycobacterium* spp. [1].

Mycobacterium spp. are aerobic, non-motile, Gram-positive bacteria [1]. The pathogenic potential varies between the etiological agents of piscine mycobacteriosis, with *M. marinum* being considered a true pathogen whilst *M. fortuitum* and *M. chelonae* are opportunistic pathogens [1]. Virulence determinants vary among piscine *Mycobacterium* species and remain incompletely understood. In *Mycobacterium marinum*, cell-wall-associated lipids, including phthiocerol dimycocerosates and phenolic glycolipids, together with the ESX-1 type VII secretion system, have been implicated in virulence and host infection; however, the relevance of these mechanisms may differ among piscine mycobacterial species and fish hosts [40]. *M. marinum* has been well documented as an opportunistic zoonotic fish-borne pathogen, causing “fisherman’s finger”, a generally superficial granulomatous inflammation of the extremities, but occasionally progressing into deeper tissues if untreated [8].

Piscine mycobacteriosis is characterised by the presence of necrotising granulomas, often most visible towards the later stages of infection [1]. Infection can occur with either an acute or chronic progression. In the rare acute cases, rapid morbidity and mortality occurs with few clinical indicators, whilst in the more common chronic manifestation, the disease may not become apparent for several months or years [39]. A lack of appetite and subsequent weight loss, abdominal edema, and granuloma formation in the liver, kidney, and spleen are considered to be end-stage symptoms in chronic cases [41].

The treatment of mycobacterial infections in fish are limited to control, in which all affected stocks must be destroyed and holding tanks must be thoroughly disinfected [1]. Whilst generally accepted practice in commercial aquaculture of “food fish”, the necessity of destruction is a particularly emotive issue in the culture of valuable ornamental species, further driving the need for research and the development of a therapeutic agent. Avenues of recombinant, DNA, and mRNA vaccines are currently being explored; however, whilst significant immune responses have been produced, the vaccines are far from commercial availability [42].

8. Viral Diseases

Viral diseases represent an important biosecurity concern in recreational and ornamental carp systems, as outbreaks can cause substantial morbidity and mortality, disrupt stocking and trade, and necessitate movement restrictions or enhanced disease-control measures. Prevention and biosecurity are the primary strategies for managing outbreaks [1]. Infection is often associated with seasonality, with outbreaks most commonly occurring in either the spring or autumn months [43]. Disease management relies primarily on prevention, early recognition, diagnostic testing, quarantine, biosecurity, and compliance with applicable reporting and movement-control requirements, rather than therapeutic intervention [1,4].

9. Cyprinid Herpes Virus

Two of the three closely related cyprinid herpesviruses are the etiological agents of highly destructive diseases in both *Cyprinus carpio* and *Cyprinus rubrofasciatus* [44]. *Cyprinid*

herpesvirus-1 (CyHV-1) causes CyHV-1 disease or “carp pox”, one of the oldest documented fish diseases, dating back to the Middle Ages [45]. *Cyprinid herpesvirus-3* (CyHV-3) is the causative agent of koi herpes virus disease (KHVD) and is highly virulent and contagious, having a mortality rate of between 80 and 100% in juvenile and adult koi [46]. *Cyprinid herpesvirus-2* (CyHV-2) is not considered in detail here as it is principally associated with diseases in goldfish and other *Carassius* spp. rather than clinical disease in common carp or ornamental koi; occasional detection in asymptomatic common carp is instead considered a potential biosecurity issue [47,48]. Infected fish are more susceptible to bacterial co-infection by *Aeromonas* or *Vibrio* strains, further complicating the management of potential outbreaks [49].

10. Cyprinid Herpesvirus-1 (CyHV-1)

Having previously thought to be a benign, seasonal skin disorder found primarily in older carp, carp pox has been shown to be lethal to both common carp fry and koi carp fry, although little research has emerged in this area over the last 35 years [50]. Mortality has been shown to be between 86 and 97% in two-week-old *Cyprinus carpio* and 20% in four-week-old *Cyprinus rubrofuscus*, with the surviving carp showing recurring cancerous growths from six months post-infection onwards [50]. Adult survivors of the disease can be characterised by epidermal hyperplasia and cutaneous growths, most apparent in the winter and spring when the water temperature is between 9 and 16 °C [43]. Transmission is primarily through water, although some evidence suggests that vertical transmission may also occur in a similar manner to CyHV-2, but further research is required in this area [44].

Diagnosis of CyHV-1 can be confirmed via PCR assay, with the viral genome being detectable in both infected cell cultures and paraffin-embedded samples of skin lesions [43]. CyHV-1 has a large double-stranded DNA genome of approximately 291 kbp [51]. Comparative genomic analysis has identified a CyHV-1 gene family with sequence similarity to cellular JUNB; however, this finding should not be interpreted as CyHV-1-encoding conventional host JUNB oncogenes or these genes directly causing tumour formation, as such mechanistic links are still to be elucidated. Carp pox is associated with epidermal hyperplasia and tumour-like lesions, but the molecular mechanisms linking viral infection, viral gene expression, host transcriptional responses, and tumour development remain incompletely resolved [45,51]. As carp pox is most virulent between 9 and 16 °C, supportive care in the form of temperature increases and water quality management is the primary treatment option [45]. Although fish will visibly recover and become asymptomatic if kept at 22 °C, their growth and development will be stunted, leading to economic loss [5]. Currently, no approved vaccine against carp pox is available; however, a multi-epitope subunit vaccine for both CyHV-1 and CyHV-3 has been proposed [44], offering some insight into potential future treatment strategies.

Cyprinid Herpesvirus-2 (CyHV-2) is not discussed in detail here as common carp and koi are only asymptomatic carriers for this particular pathogen; pathogenic infections of CyHV-2 are confined to other carp species with less relevance to recreational aquaculture, such as goldfish (*Carassius auratus*) and Prussian carp (*Carassius gibelio*). Crucian carp (*Carassius Carassius*) are susceptible, and occasionally found alongside common carp in angling ecosystems; however, instances are low and popularity for the species is low in comparison to common carp; therefore, the topic is not covered in detail here.

11. Cyprinid Herpesvirus-3 (CyHV-3)

Koi herpesvirus disease (KHVD) is a listed notifiable disease in accordance with the International Office of Epizootics, due to its mobility and persistence [3]. Primarily spread around the world by the global fish trade and international showings of prized ornamental

koi, serious financial losses have been observed since the late 1990s as a result [52]. Whilst primarily host-specific to common and ornamental carp, hybrid species show partial susceptibility to infection, further increasing the reach and spread of KHVD [53].

Infection by koi herpesvirus is characterised by gill discoloration, necrosis, and then eventually loss of osmoregulation in the final stages of the disease [46]. Abnormal behaviours can be observed, such as lethargy and loss of appetite, with infected fish often seen gathered on the bottom of the tank or pond [51]. Different clinical signs are visible in the skin at different stages of infection, starting with hyperaemia at the base of the fins and abdomen with pale, peeling patches seen as a result of mucus hyper-secretion [54]. Fin erosion, gasping motions, and bilateral enophthalmia are the last clinical signs to manifest, with some fish showing neurological distress before mortality [46].

At approximately 295 kbp, CyHV-3 possesses the longest recorded genome within the *Herpesvirales* order [55]. Two distinct genotypes exist with several variants within each: the Asian genotype with variant A1 and variant A2, and the European genotype with variants E1 through to E7 [56]. Nested PCR followed by DNA polymerase gene sequencing is an effective strategy to identify nucleotide disparities between specific genomes and variants [57]. The two genotypes have two distinct lineages, and in order for an effective vaccine to be developed, the two genotypes must be more fully understood. No universally available or broadly applicable licensed vaccine currently provides a complete solution for the prevention of CyHV-3 infection in common carp or in ornamental koi. Experimental approaches have included controlled exposures, followed by transfer to non-permissive temperatures, although this should not be regarded as an acceptable vaccination strategy. Fish surviving exposure may develop latent infection, remain carriers, and reactivate or transmit virus following subsequent temperature change or stress [58,59]. Vaccine candidates must therefore be evaluated not only for protection against clinical disease but also for their ability to prevent infection, latency, carrier status, and onward transmission. More modern approaches have included conventional and recombinant live attenuated vaccines, inactivated vaccines, DNA vaccines, bacterial-vector vaccines, and subunit vaccine candidates [44,58,60]. Vaccination programmes inherently favour high-value fish stocks, making them of particular interest to recreational aquaculture markets, where the economics of vaccine usage is more viable than the high-volume, low-individual-value nature of farmed food fish sectors. Thus, particularly in the UK and EU, carp angling, often overlooked in comparison to carp or other food fish farming, is emerging as a high-value and high-impact arena for academic study and therapeutic development, but one that is currently significantly underfunded in comparison.

12. Carp Edema Virus

Carp edema virus (CEV) is the causative agent of koi sleepy disease (KSD), and belongs to the pox virus group *Poxviridae* [61]. Whilst juvenile koi are primarily affected, common carp are also susceptible to koi sleepy disease within stocking environments [62]. Manifestation of the disease is most commonly associated with stressful farming conditions, including sudden temperature changes and movement from earthen to concrete pools [63]. Mortality of between 80 and 100% has been observed in juvenile koi, causing significant economic losses in the ornamental trade [61].

Lethargy is the characteristic clinical indicator of CEV infection, with affected fish often seen lying at the bottom of their pond or tank [62]. Tissue edema with associated skin haemorrhages and pale swollen gills signify the beginning of the “gill rot”, with hypoxia and mortality occurring in the later stages of the infection [64]. Microscopic examination can often reveal enlarged cells within the respiratory system, primarily the epithelium of gill lamellae [61].

Three distinct genotypes of CEV have been isolated, although the majority of the DNA sequence that encodes the core protein P4a is not yet published [65]. A 6–10% genetic diversity between genogroups I, IIa, and IIb across Europe and Asia has been shown, which could suggest that further lineages of the virus are yet to be discovered [66]. Genogroup I is primarily detected in common carp that have been farmed, whereas koi are mostly infected by genogroup IIb [63]. Diagnosis is reliant upon the detection of viral DNA via PCR assay, using a nested PCR protocol designed by Tokyo University of Fisheries [67]. Treatment options are limited to mitigation, with a salt bath of 0.3–0.5% thought to minimise the effects of disease [68]. Any pond or tank with a suspected CEV outbreak should be quarantined, with strict biosecurity measures adhered to.

13. Spring Viraemia of Carp

Spring viraemia of carp virus (SVCV), currently classified within the genus *Springvivirus* (family *Rhabdoviridae*), is the causative agent of spring viraemia of carp (SVC), a listed disease of fish recognised by the World Organisation for Animal Health [3,69]. SVCV infections have been reported in several carp species and other susceptible cyprinid and ictalurid fish species; however, susceptibility, clinical expression, and outbreak risk vary among host species, life stages, isolates, and environmental conditions [3]. Although water-borne transmission is believed to be the primary route of infection, parasitic invertebrates such as leeches were previously also postulated as mechanical vectors of transmission [70].

SVCV exhibits the characteristic bullet-shaped morphology of a vertebrate rhabdovirus, measuring approximately 80–180 nm in length and 60–90 nm in diameter [71]. Containing five open reading frames encoding five structural proteins, the total genome length is approximately 11kb [72]. SVCV isolates are broadly divided into two clades, one Asian and one European [73]. These isolates are then further divided into four genogroups, each with their own geographical association: Ia, Ib, Ic, and Id [74]. Outbreaks are most common in spring as the name would suggest, with few adult fish becoming infected once the water temperature reaches over 17 °C [75]. However, juvenile fish can succumb to SVC even at water temperatures of 22–23 °C, exhibiting a mortality rate of up to 90% [72].

Clinical manifestation of SVC is often non-specific, proving difficult to diagnose before the disease progresses. As with many piscine viral diseases, exophthalmia, abdominal distension, and surface haemorrhages are often observed [76]. Additional symptoms may include degeneration of the gill lamellae, edema, and necrosis of the internal organs alongside a loss of structure within blood vessel walls [1].

Whilst there currently is not a commercially available vaccine against SVC, several experimental approaches are showing promising efficacy. DNA vaccines currently show the most potential for future commercialisation, with an intramuscular delivery vaccine based on the SVCV glycoprotein providing up to 100% protection in juvenile carp [77]. Alternative approaches include a chitosan-encapsulated DNA vaccine and a PEG-modified subunit vaccine, both of which are still undergoing experimental development [78,79].

14. Fungal and Oomycete Diseases

Fungal and oomycete diseases represent a significant but often overlooked threat to carp in recreational and ornamental systems, most notably in cultivation systems, where environmental stressors compromise fish immunity [1]. Often discussed together in aquaculture, true fungi (kingdom fungi) and oomycetes (phylum Oomycota) are evolutionarily distinct and differ in both cell biology and treatment susceptibility.

Unlike most viruses and bacteria, fungal pathogens are often opportunistic, with disease either following mechanical injury or previous infection with a primary pathogen [80]. Oomycetes, rather than true fungi, are responsible for the majority of common and orna-

mental carp “fungal” infections, with outbreaks primarily occurring in the late autumn and winter when cooler temperatures favour their growth and virulence [81]. Management of these diseases within commercial aquaculture is challenging due to the limited availability of effective, authorised anti-oomycete agents and the increase in regulation surrounding traditional chemical treatments such as malachite green [82]. As a result, strategic prevention is emphasised, with probiotic and other non-antimicrobial approaches gaining attention as alternative control measures [83].

15. Saprolegniasis

Saprolegnia species, particularly *Saprolegnia parasitica*, are important pathogens of carp and other freshwater fish, but their role is best described as context-dependent rather than true primary pathogens [84]. *Saprolegnia* is an aquatic oomycete, a primarily saprotrophic type of water mould that colonises dead organic matter and injured tissue, but can act as an opportunistic pathogen in an already compromised host [85]. Traditional morphology-based identification is often unreliable, as identifying characteristics such as sexual reproductive structures are not always produced in vitro; therefore, molecular identification is heavily relied upon for species-level identification [84]. Pathogenesis of saprolegniasis is complex, with the relative contribution of the oomycete as a primary invader versus a secondary coloniser remains debated, likely varying between isolates and production systems [1].

White, cotton-like lesions are a hallmark of saprolegniasis, with these patches of filamentous hyphae found on the fins or body of infected fish [86]. These hyphae can penetrate into underlying muscle and connective tissue, leading to mortality through tissue disruption or secondary infection in severe cases [84]. Early lesions are often circular, spreading out radially as the infection progresses until the lesions begin to merge [1]. In severe cases, between 50 and 80% of the surface of the fish may be covered by these hyphae [86]. Physiological symptoms include a loss of appetite and reduced mobility, often preceding any visible infection. Respiratory distress can lead to the observation of gasping and surface breathing as a result of the colonisation and haemorrhaging of the gill tissue [87].

A broad spectrum of chemical and management interventions are used to control saprolegniasis, but these are constrained by regulatory status, species-specific toxicity, and variable evidence of efficacy under field conditions. Malachite green treatment has historically shown high efficacy against *Saprolegnia*, but has since been banned for food-producing animals within the UK and EU, as it has been shown to be both carcinogenic and genotoxic in animal models [80,88]. Potassium permanganate (KMnO₄) is considered one of the most effective treatments currently available due to a higher fish survival rate in comparison with alternative treatments such as hydrogen peroxide (H₂O₂); however, outcomes are highly dependent on dose, water chemistry, and species tolerance [89]. Virkon-S shows promise in inhibiting growth of *Saprolegnia*; however, the minimum inhibitory concentration (MIC) of 1000 ppm is significantly higher than the 5 ppm MIC of malachite green, raising both practical and environmental considerations [90]. Further research is needed to ascertain the effect these treatments would have on the food chain, particularly in cases of human consumption.

16. Branchiomycosis

Commonly known as “gill rot”, branchiomycosis affects the gills of freshwater fish and is associated with the oomycetes *Branchiomyces sanguinis* and *Branchiomyces demigrans* [1]. Although historically described as a fungal disease, *Branchiomyces* spp. are oomycetes and should be distinguished from true fungi. Both species have been associated with branchiomycosis, but their reported distribution and therefore relative importance may

vary between host species, geographical settings, and management conditions [91]. Often referred to as a “poor management” disease, branchiomycosis outbreaks are primarily found in aquaculture systems with a high organic load and elevated temperatures. Infection is localised and acute, with fungal growth found within the vascular system of the gills. Branchiomycosis can devastate fish populations, with substantial mortality reported during outbreaks [1].

The two *Branchiomyces* species have traditionally been differentiated by their morphology and distribution within affected gill tissue. *B. sanguinis* has been reported principally within branchial blood vessels, whereas *B. demigrans* may occur more extensively within gill tissue and the interlamellar spaces [92]. A presumptive diagnosis can be made by macroscopic observation of the gill tissue, as a characteristic marbling will reveal the presence of infection, with the paler gill areas showing infected or necrotic tissue [93,94]. Confirmation of the visual diagnosis can then be made by microscopic examination of fresh gill tissue, with histopathology demonstrating branching, non-septate hyphae, and spores within the tissue. Molecular methods may provide additional support where suitable assays are available and validated for the relevant sample type and species [93]. Infected fish present with the symptoms of a respiratory disorder including lethargy, gasping motions at the water’s surface, and rapid movement of the operculum [91].

Treatment options are largely limited to prevention and control, with farmers advised to remove deceased fish from stocking ponds immediately to prevent further release of spores [94]. Pond drainage, drying, and disinfection have historically been used following outbreaks; however, any chemical intervention must comply with local regulatory requirements and should be selected with veterinary advice, taking account of fish species, intended use, water chemistry, and environmental safety [1]. Malachite green or formalin treatments have historically been used; however, such practices are outdated and malachite green has since been banned and formalin reclassified as a class one human carcinogen [80,95], supporting our own recent findings documenting lack of efficacy of unverified, old chemical treatments such as these [31]. Further research is needed to improve surveillance, clarify species-level epidemiology, and develop effective control measures suitable for recreational and ornamental carp systems.

17. Zoonotic Potential

Fish-borne zoonotic diseases present an evolving threat to public health, transmitting a range of pathogens from the aquatic environment to humans. Livestock, reptiles, and amphibians often act as carriers or reservoirs, further increasing transmission through natural migration or trade shipping. With recreational fishing and rearing koi carp as companion animals spiking in popularity, humans are in markedly closer contact with these pathogens, increasing the necessity of further research into the public health impact of fish-borne zoonoses. Little documented evidence exists for direct zoonotic transfer of carp pathogens, but several of the carp pathogens covered in this review are more widely accepted marine/estuarine pathogens, with documented zoonotic potential. Thus, feed-stocks originating from marine environments could be considered a potential transmission route into recreational aquaculture applications, maggot farming being the prime example. However, more research in this area is needed to further develop this emerging area of research in recreational aquaculture and the following is not based on direct evidence in carp or recreational aquaculture, instead taking inference from other environments where zoonotic potential has been documented for those species discussed here.

Four of the previously discussed pathogens are considered to have zoonotic potential, with the pathogenicity of two supported by epidemiological and molecular evidence, which could easily be extrapolated to recreational aquaculture as detailed in Table 1, below.

Table 1. Summary of potential bacterial agents of fish-borne zoonosis. Species are indicated as Gram-positive (G+), Gram-negative (G−), or acid-fast (AF). Evidence of zoonotic potential is indicated by '+', and '−' denotes lack of evidence or evidence to the contrary.

Organism	Type	Epidemiological Evidence	Molecular Evidence	Study
<i>Aeromonas</i> spp.	G−	+	−	[96]
<i>Vibrio vulnificus</i>	G−	+	+	[97]
<i>Edwardsiella tarda</i>	G−	+	−	[98]
<i>Mycobacterium</i> spp.	AF	+	+	[99]

Aeromonas species, in particular *A. hydrophila*, have been shown to be present in faecal samples of livestock known to drink from water contaminated with the pathogen. Samples were taken from 110 horses, 115 pigs, 111 sheep, and 123 cows, and 54 positive results for *A. hydrophila* were found, with infection most commonly occurring in the bovine sample group [100]. Transmission via migratory birds presents a concerning prospect, with a study showing the presence of antibiotic-resistant *Aeromonas* spp. present in birds within five provinces in China. Out of the 810 samples taken, a total of 176 strains of *Aeromonas* were isolated, with 30.1% of the samples identified as *A. hydrophila* [101]. The close proximity of wildfowl and carp in recreational angling is widespread, and faeces from infected birds can easily be envisaged as a route of transmission to carp fishery lake beds, on which stocked carp then feed. However, direct evidence of this link has not been shown to date.

Vibrio vulnificus is the etiological agent of vibriosis in fish species and humans, posing significant risk to both fish and their handlers. Specific strains within lineage II of the *V. vulnificus* species that belong to the zoonotic clonal-complex are capable of infecting homeothermic and poikilothermic animals, with eels being the primary host. These strains are found globally throughout aquaculture industries, and are contracted primarily through contact with contaminated seawater or diseased fish [102]. Although classically an estuarine/marine organism widely associated with oysters and other sea foods, *V. vulnificus* could be predicted to affect recreational angling sectors through its potential contamination of cheap/old fish meals and meats used in the production of angling baits. However, this is an emerging area of research and no documented evidence of this currently exists.

V. vulnificus samples have been isolated from aquatic and ground-foraging migratory birds consistently since monitoring began in 1965, indicating that birds may act as reservoirs and carriers of vibriosis [103].

Edwardsiella tarda infections in humans are primarily characterised by bacterial gastroenteritis, although systemic infections such as meningitis and septicaemia have been documented [35]. One case study illustrates an incidence of chronic osteomyelitis in a former fisherman and fishmonger in which the pathogen lingered for 15 years, slowly degrading and necrotising the tissue of his right femur. Possessing the ability to evade phagocytic killing and survive intracellularly for extended periods of time is a concerning prospect given the zoonotic potential of *E. tarda* [98]. Migratory birds can act as asymptomatic carriers; however, in some cases are also susceptible to the pathogen [35], offering the potential for the same close-contact zoonotic transfers to carp and carp anglers.

Whilst zoonotic transmission of marine *Mycobacterium* species to humans often results in the superficial granulomatous inflammation known as “fisherman’s finger”, this condition can also develop into severely necrotic lesions or deep tissue infections encompassing tendons or bones [7]. Immunocompromised patients may contract systemic infections putting particular strain on the respiratory system, occasionally resulting in death [40].

Classically, Leptospirosis, normally associated with *Leptospira Interrogans* infection and colloquially known amongst anglers as Weil's disease, is widely regarded as a significant and serious zoonotic threat in recreational aquaculture environments. Transfer occurs via rodent urine into still waters, where anglers inadvertently come into contact through open wounds, that can result in severe neurological complications, and can also be fatal. However, to our knowledge, no documented examples exist of carp carrying or being infected by *Leptospira*, but the exposure route is clear and further research is needed to elucidate other zoonotic vectors of *L. Interrogans* in the recreational angling.

18. Conclusions and Perspective

Whilst considerable progress has been made in the identification of pathogens impacting carp in both angling and aquaculture, substantial knowledge gaps still exist within host–pathogen dynamics and around sustainable treatment strategies. For recreational aquaculture specifically, prevention is far better than cure, and good fishery management practices, responsibly sourced feed strategies, and proper transmission control measures are an absolute priority for fishery owners and fish farmers. For anglers and koi keepers themselves, it remains difficult for individuals to gain access to proper advice and guidance, with only around twenty practicing veterinarians in the whole of the UK offering specialised fish-related services; antibiotic prescriptions are often impossible to source, leaving a gap in the market for ineffective, ill-conceived, and even toxic alternatives [30]. In addition, antibiotic and antimicrobial resistance in aquaculture has become more prevalent, increasing difficulties around controlling outbreaks within large aquaculture systems, thus making the availability of antibiotics to hobbyists increasingly scarce also [5]. With the rise in popularity of these pastimes, and the diversification of fish farms also leading to the increased availability and distribution of easily accessible carp angling venues, there is an urgent unmet need to research, identify, and monitor the microbiomes of carp and of the organisms on which they feed and with which they interact regularly in the UK and EU. Additionally, new research is needed to identify novel classes of next-generation non-toxic, biodegradable, and environmentally friendly antibacterials/antifungals that can be accessed as over-the-counter medicines, increasing their availability, and replacing many currently employed old, toxic chemical 'wound sealants' and 'topical carp care' products that have been developed off-label and with no supporting evidence base.

Zoonotic potential presents an interesting prospect in the recreational aquaculture industry, which is now perhaps the best place to study such mechanisms due to the significant numbers of people engaging in these popular pastimes. The increasing proximity of carp to livestock, humans, and wildlife brings into question every aspect of angling and fish care, including the food sources used to rear carp in the UK and EU, which currently are largely unregulated. Much research is needed to identify current variations in feeding practices, potential links to other aquaculture environments, particularly marine, and in particular, the microbiome composition of both the carp themselves and the animals they come into contact and may also feed upon. This is a truly emerging area, but one that could have significant implications for large-scale pathogen transmission routes given the network of fisheries and ponds holding carp throughout the UK and Europe.

Antibiotic resistance monitoring is an emerging priority area in recreational aquaculture, where lakes and carp ponds have the potential to act as sumps for the accumulation of and evolution of resistance genes through animal products used in angler bait, or through the livestock/wildlife that frequents such areas alongside the carp that reside there. Antibiotic-resistant strains of *A. hydrophila*, for example, have access to avian reservoirs and distribution routes, meaning the implementation of surveillance and monitoring programmes should be an essential focus alongside novel treatments [100]. Inter-species

transmission of treatment-resistant pathogens could devastate aquaculture, angling, and terrestrial livestock industries, with ever-closer contact to human populations increasing the threat of zoonotic transmission.

Future research endeavours should prioritise the development of advanced and rapidly deployable diagnostic tools/methodologies, more detailed studies into pathogen/host biology, and the formulation of novel ‘greener’ therapeutic treatment approaches, where both environmental and biological sustainability should be considered, ensuring low resistance levels to novel approaches. An interdisciplinary and collaborative approach is essential amongst user groups, trade/industry, and academia to monitor, research, and implement adaptations/strategies to address these threats, in order to safeguard the future of all carp-based aquaculture systems worldwide. Government agencies also need to recognise the economic contribution of recreational aquaculture, which far exceeds that of the salmon farming industry in the UK and Europe, with 8 million anglers in the UK, contributing £1.7 billion to the UK economy [104] and 25 million anglers in Europe, making the market there three times the size of the UK’s. Research funding currently significantly under-represents the contribution and importance of recreational aquaculture and the financial as well as cultural wealth it brings to these economies.

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References

1. Roberts, R.J. *Fish Pathology*; John Wiley & Sons, Incorporated: Newark, NJ, USA, 2012. Available online: <http://ebookcentral.proquest.com/lib/dmu/detail.action?docID=875436> (accessed on 17 February 2025).
2. Al-Jubouri, M.O.; Al-haider, S.; Mahdi Imran, S. Study of some Bacterial Pathogens that Infect Common Carp (*Cyprinus carpio*) in the Diwaniya River and their Relationship to Water Characteristics. *Adv. Anim. Vet. Sci.* **2024**, *12*, 1961–1968. [CrossRef]
3. Ernst, I. Infection with spring viraemia of carp virus. In *Manual of Diagnostic Tests for Aquatic Animals*; World Organisation for Animal Health (WOAH): Paris, France, 2024. Available online: https://www.woah.org/fileadmin/Home/eng/Health_standards/aahm/current/2.3.09_SVC.pdf (accessed on 2 September 2026).

4. Yoshimizu, M.; Kasai, H.; Sakoda, Y.; Sano, N.; Sano, M. Oncogenic viruses: *Oncorhynchus masou* virus and *Cyprinid herpesvirus*. In *Fish Viruses and Bacteria: Pathobiology and Protection*; CABI Books: Wallingford, UK, 2017; pp. 51–67. [CrossRef]
5. Manzoor, K.; Rasool, F.; Khan, N.; Anjum, K.M.; Parveen, S. Resistance Patterns of Frequently Applied Antimicrobials and Occurrence of Antibiotic Resistance Genes in *Edwardsiella tarda* Detected in Edwardsiellosis-Infected Tilapia Species of Fish Farms of Punjab in Pakistan. *J. Microbiol. Biotechnol.* **2023**, *33*, 668. [CrossRef] [PubMed]
6. Jiang, M.; Chen, Z.-G.; Zheng, J.; Peng, B. Metabolites-Enabled Survival of Crucian Carps Infected by *Edwardsiella tarda* in High Water Temperature. *Front. Immunol.* **2019**, *10*, 1991. [CrossRef] [PubMed]
7. Khan, U.M.; Rittenberg, A. Fisherman's Dilemma: Disseminated *Mycobacterium marinum* in an Immunosuppressed Patient. *Am. J. Med.* **2020**, *133*, e549–e551. [CrossRef] [PubMed]
8. Abdelsalam, M.; Ewiss, M.; Khalefa, H.S.; Mahmoud, M.A.; Elgendy, M.Y.; Abdel-Moneam, D.A. Coinfections of *Aeromonas* spp., *Enterococcus faecalis*, and *Vibrio alginolyticus* isolated from farmed Nile tilapia and African catfish in Egypt, with an emphasis on poor water quality. *Microb. Pathog.* **2021**, *160*, 105213. [CrossRef] [PubMed]
9. Han, G.; Huang, T.; Liu, X.; Liu, R. Bacteriophage EPP-1, a potential antibiotic alternative for controlling edwardsiellosis caused by *Edwardsiella piscicida* while mitigating drug-resistant gene dissemination. *Sci. Rep.* **2024**, *14*, 9399. [CrossRef] [PubMed]
10. Food and Agriculture Organization of the United Nations (FAO). *Water Quality and Fish Health*; FAO: Rome, Italy, 1993. Available online: <https://www.fao.org/4/t1623e/t1623e00.htm> (accessed on 3 September 2026).
11. Locke, J.B.; Colvin, K.M.; Varki, N.; Vicknair, M.R.; Nizet, V.; Buchanan, J.T.; Vicknair, M.R. *Streptococcus iniae* β -hemolysin streptolysin S is a virulence factor in fish infection. *Dis. Aquat. Org.* **2007**, *76*, 17–26. [CrossRef] [PubMed]
12. Seike, S.; Kobayashi, H.; Ueda, M.; Takahashi, E.; Okamoto, K.; Yamanaka, H. Outer Membrane Vesicles Released from *Aeromonas* Strains Are Involved in the Biofilm Formation. *Front. Microbiol.* **2020**, *11*, 613650. [CrossRef] [PubMed]
13. Clois-Fuentes, J.; Nguinkal, J.A.; Unger, P.; Kreikemeyer, B.; Palm, H.W. Bacterial Communities from Two Freshwater Aquaculture Systems in Northern Germany. *Environ. Microbiol. Rep.* **2024**, *16*, e70062. [CrossRef] [PubMed]
14. Kozińska, A.; Pekała, A. Characteristics of Disease Spectrum in relation to Species, Serogroups, and Adhesion Ability of Motile *Aeromonads* in Fish. *Sci. World J.* **2012**, *2012*, 949358. [CrossRef] [PubMed]
15. Nascimento, M.; Rodrigues, J.; Matias, R.; Jordao, L. *Aeromonas* spp. in Freshwater Bodies: Antimicrobial Resistance and Biofilm Assembly. *Antibiotics* **2024**, *13*, 166. [CrossRef] [PubMed]
16. Moya-Salazar, J.; Díaz, C.R.; Cañari, B.; Badillo, R.X.; Verano-Zelada, M.; Chicoma-Flores, K.; Contreras-Pulache, H. Detection of pathogenic *Aeromonas hydrophila* from two rainbow trout (*Oncorhynchus mykiss*) farms in Peru. *Braz. J. Vet. Med.* **2022**, *44*, e000922. [CrossRef] [PubMed]
17. Epple, H.J.; Mankertz, J.; Ignatius, R.; Liesenfeld, O.; Fromm, M.; Zeitz, M.; Chakraborty, T.; Schulzke, J.D. *Aeromonas hydrophila* Beta-Hemolysin Induces Active Chloride Secretion in Colon Epithelial Cells (HT-29/B6). *Infect. Immun.* **2004**, *72*, 4848. [CrossRef] [PubMed]
18. Abdella, B.; Abozahra, N.A.; Shokrak, N.M.; Mohamed, R.A.; El-Helow, E.R. Whole spectrum of *Aeromonas hydrophila* virulence determinants and the identification of novel SNPs using comparative pathogenomics. *Sci. Rep.* **2023**, *13*, 7712. [CrossRef] [PubMed]
19. Chen, F.; Sun, J.; Han, Z.; Yang, X.; Xian, J.-A.; Lv, A.; Hu, X.; Shi, H. Isolation, Identification and Characteristics of *Aeromonas veronii* From Diseased Crucian Carp (*Carassius auratus gibelio*). *Front. Microbiol.* **2019**, *10*, 2742. [CrossRef] [PubMed]
20. Chang, P.H.; Wu, T.P.; Chung, H.Y.; Chien, C.Y. *Aeromonas hydrophila* and *Saprolegnia australis* isolated from Ayu, *Plecoglossus altivelis*, with ulcerative skin disease in Taiwan. *Bull. Eur. Assoc. Fish. Pathol.* **2002**, *22*, 393–399.
21. Bai, L.; Li, S.; Fu, X.; Wang, P.; Guo, Y.; Yu, D. Isolation and Identification of *Aeromonas hydrophila* from *Pangasius bocourti* with Bacterial Septicemia and Drug Screening for Treatment. *Bull. Eur. Assoc. Fish. Pathol.* **2023**, *43*, 114–128. [CrossRef]
22. Ninh, D.T.; Le, D.V.; Van Van, K.; Giang, N.T.H.; Dang, L.T.; Hoai, T.D. Prevalence, Virulence Gene Distribution and Alarming the Multidrug Resistance of *Aeromonas hydrophila* Associated with Disease Outbreaks in Freshwater Aquaculture. *Antibiotics* **2021**, *10*, 532. [CrossRef] [PubMed]
23. Ruzauskas, M.; Armalytė, J.; Lastauskienė, E.; Šiugždinienė, R.; Klimienė, I.; Mockeliūnas, R.; Bartkienė, E. Microbial and Antimicrobial Resistance Profiles of Microbiota in Common Carps (*Cyprinus carpio*) from Aquacultured and Wild Fish Populations. *Animals* **2021**, *11*, 929. [CrossRef] [PubMed]
24. Zhang, M.; Zhang, T.; He, Y.; Cui, H.; Li, H.; Xu, Z.; Wang, X.; Liu, Y.; Li, H.; Zhao, X.; et al. Immunogenicity and protective efficacy of OmpA subunit vaccine against *Aeromonas hydrophila* infection in *Megalobrama amblycephala*: An effective alternative to the inactivated vaccine. *Front. Immunol.* **2023**, *14*, 1133742. [CrossRef] [PubMed]
25. Attia, M.M.; Abdelsalam, M.; Elgendy, M.Y.; Sherif, A.H. *Dactylogyrus extensus* and *Pseudomonas fluorescens* dual infection in farmed common carp (*Cyprinus carpio*). *Microb. Pathog.* **2022**, *173*, 105867. [CrossRef] [PubMed]
26. Aly, S.M.; Ismail, M.M. Characteristics of infectious dropsy from an epizootic of cultured common carp (*Cyprinus carpio* L.) with special investigation to swim-bladder lesions. *Suez Canal Vet. Med. J.* **2016**, *21*, 185–195. [CrossRef]

27. LaFrentz, B.R.; Králová, S.; Burbick, C.R.; Alexander, T.L.; Phillips, C.W.; Griffin, M.J.; Waldbieser, G.C.; García, J.C.; Sebastião, F.d.A.; Soto, E.; et al. The fish pathogen *Flavobacterium columnare* represents four distinct species: *Flavobacterium columnare*, *Flavobacterium covae* sp. nov., *Flavobacterium davisii* sp. nov. and *Flavobacterium oreochromis* sp. nov., and emended description of *Flavobacterium columnare*. *Syst. Appl. Microbiol.* **2022**, *45*, 126293. [CrossRef] [PubMed]
28. Declercq, A.M.; Chiers, K.; Broeck, W.V.D.; Dewulf, J.; Eeckhaut, V.; Cornelissen, M.; Bossier, P.; Haesebrouck, F.; Decostere, A. Interactions of highly and low virulent *Flavobacterium columnare* isolates with gill tissue in carp and rainbow trout. *Vet. Res.* **2015**, *46*, 25. [CrossRef] [PubMed]
29. Thunes, N.C.; Conrad, R.A.; Mohammed, H.H.; Zhu, Y.; Barbier, P.; Evenhuis, J.P.; Perez-Pascual, D.; Ghigo, J.-M.; Lipscomb, R.S.; Schneider, J.R.; et al. Type IX Secretion System Effectors and Virulence of the Model *Flavobacterium columnare* Strain MS-FC-4. *Appl. Environ. Microbiol.* **2022**, *88*, e01705. [CrossRef] [PubMed]
30. Makin, E.; Shilton, G.; Brotherhood, O.; Rassool-Amin, A.; Gordon, K.; Satkunarasa, H.; Reynolds, P.; Wellby, I.; Locker, J.; Qutachi, O.; et al. Scale of concern: Efficacy of Commercially Available Topical Carp Care Formulations for Recreational Application in Carp. *Aquaculture* **2026**, *6*, 19. [CrossRef]
31. Oh, W.T.; Jun, J.W.; Kim, H.J.; Giri, S.S.; Yun, S.; Kim, S.G.; Kim, S.W.; Kang, J.W.; Han, S.J.; Kwon, J.; et al. Characterization and Pathological Analysis of a Virulent *Edwardsiella anguillarum* Strain Isolated from Nile Tilapia (*Oreochromis niloticus*) in Korea. *Front. Vet. Sci.* **2020**, *7*, 14. [CrossRef] [PubMed]
32. Reichley, S.R.; Ware, C.; Steadman, J.; Gaunt, P.S.; García, J.C.; LaFrentz, B.R.; Thachil, A.; Waldbieser, G.C.; Stine, C.B.; Buján, N.; et al. Comparative Phenotypic and Genotypic Analysis of *Edwardsiella* Isolates from Different Hosts and Geographic Origins, with Emphasis on Isolates Formerly Classified as *E. tarda*, and Evaluation of Diagnostic Methods. *J. Clin. Microbiol.* **2017**, *55*, 3466. [CrossRef] [PubMed]
33. Nakamura, Y.; Takano, T.; Yasuike, M.; Sakai, T.; Matsuyama, T.; Sano, M. Comparative genomics reveals that a fish pathogenic bacterium *Edwardsiella tarda* has acquired the locus of enterocyte effacement (LEE) through horizontal gene transfer. *BMC Genom.* **2013**, *14*, 642. [CrossRef] [PubMed]
34. Davies, Y.M.; de Oliveira, M.G.X.; Cunha, M.P.V.; Franco, L.S.; Santos, S.L.P.; Moreno, L.Z.; Gomes, V.T.d.M.; Sato, M.I.Z.; Nardi, M.S.; Moreno, A.M.; et al. *Edwardsiella tarda* outbreak affecting fishes and aquatic birds in Brazil. *Vet. Q.* **2019**, *38*, 99. [CrossRef] [PubMed]
35. Rahmawaty, A.; Chen, M.; Byadgi, O.V.; Wang, P.; Chen, S. Phenotypic and genotypic analysis of *Edwardsiella* isolates from Taiwan indicates wide variation with a particular reference to *Edwardsiella tarda* and *Edwardsiella anguillarum*. *J. Fish Dis.* **2022**, *45*, 1659–1672. [CrossRef] [PubMed]
36. Pandey, V.; Bhat, R.A.H.; Chandra, S.; Tandel, R.S.; Dubey, M.K.; Sharma, P.; Gehlot, B.; Dash, P.; Joshi, R. Clinical signs, lethal dose and histopathological lesions in grass carp, *Ctenopharyngodon idella* experimentally infected with *Edwardsiella tarda*. *Microb. Pathog.* **2021**, *161*, 105292. [CrossRef] [PubMed]
37. Ahmadifar, E.; Mohammadzadeh, S.; Kalhor, N.; Yousefi, M.; Moghadam, M.S.; Naraballoh, W.; Ahmadifar, M.; Hoseinifar, S.H.; Van Doan, H. Cornelian cherry (*Cornus mas* L.) fruit extract improves growth performance, disease resistance, and serum immune-and antioxidant-related gene expression of common carp (*Cyprinus carpio*). *Aquaculture* **2022**, *558*, 738372. [CrossRef]
38. Bataillon, E.; Dubard, L.; Terre, L. Un nouveau type de tuberculose. In *Comptes Rendus des Seances de la Societe Biologie (Paris, France)*; Biodiversity Heritage Library: Washington, DC, USA, 1897; Volume 49, pp. 446–449. Available online: <https://www.biodiversitylibrary.org/bibliography/169512> (accessed on 3 September 2026).
39. Pasnik, D.J.; Smith, S.A. Immunogenic and protective effects of a DNA vaccine for *Mycobacterium marinum* in fish. *Vet. Immunol. Immunopathol.* **2005**, *103*, 195–206. [CrossRef] [PubMed]
40. Hashish, E.; Merwad, A.; Elgaml, S.; Amer, A.; Kamal, H.; Esat, A.; El-Diasty, M. *Mycobacterium marinum* infection in fish and man: Epidemiology, pathophysiology and management; a review. *Vet. Q.* **2018**, *38*, 35–46. [CrossRef] [PubMed]
41. Swaim, L.E.; Connolly, L.E.; Volkman, H.E.; Humbert, O.; Born, D.E.; Ramakrishnan, L. *Mycobacterium marinum* infection of adult zebrafish causes caseating granulomatous tuberculosis and is moderated by adaptive immunity. *Infect. Immun.* **2006**, *74*, 6108–6117. [CrossRef] [PubMed]
42. Chen, D.; Huang, W.; Shen, L.; Zhang, J.; Pan, Z.; Zhang, C.; Tang, Y.; Zhou, Z.; Tao, J.; Luo, G.; et al. An mRNA vaccine induces antimycobacterial immunity by activating DNA damage repair and autophagy. *Mol. Ther. Nucleic Acids* **2025**, *36*, 102402. [CrossRef] [PubMed]
43. Rahmati-Holasoo, H.; Ahmadvand, S.; Shokrpour, S.; El-Matbouli, M. Detection of Carp pox virus (CyHV-1) from koi (*Cyprinus carpio* L.) in Iran; clinico-pathological and molecular characterization. *Mol. Cell. Probes* **2020**, *54*, 101668. [CrossRef] [PubMed]
44. Rani, N.A.; Robin, T.B.; Prome, A.A.; Ahmed, N.; Moin, A.T.; Patil, R.B.; Sikder, M.N.A.; Bappy, N.I.; Afrin, D.; Hossain, F.M.A.; et al. Development of multi epitope subunit vaccines against emerging carp viruses Cyprinid herpesvirus 1 and 3 using immunoinformatics approach. *Sci. Rep.* **2024**, *14*, 11783. [CrossRef] [PubMed]
45. Nagata, J.; Takita, K.; Kasai, H. Long-term detection of cyprinid herpesvirus 1 genome and tumorous transcriptome in common carp (*Cyprinus carpio*) infected with the virus. *Aquaculture* **2024**, *592*, 741158. [CrossRef]

46. Hedrick, R.P.; Gilad, O.; Yun, S.; Spangenberg, J.V.; Marty, G.D.; Nordhausen, R.W.; Kebus, M.J.; Bercovier, H.; Eldar, A. A Herpesvirus Associated with Mass Mortality of Juvenile and Adult Koi, a Strain of Common Carp. *J. Aquat. Anim. Health* **2000**, *12*, 44–57. [\[CrossRef\]](#)
47. Thangaraj, R.S.; Nithianantham, S.R.; Dharmaratnam, A.; Kumar, R.; Pradhan, P.K.; Thangalazhy Gopakumar, S.; Sood, N. Cyprinid herpesvirus-2 (CyHV-2): A comprehensive review. *Rev. Aquac.* **2021**, *13*, 796–821. [\[CrossRef\]](#)
48. Panicz, R.; Sadowski, J.; Eljasik, P. Detection of Cyprinid herpesvirus 2 (CyHV-2) in symptomatic ornamental types of goldfish (*Carassius auratus*) and asymptomatic common carp (*Cyprinus carpio*) reared in warm-water cage culture. *Aquaculture* **2019**, *504*, 131–138. [\[CrossRef\]](#)
49. Okon, E.M.; Okocha, R.C.; Taiwo, A.B.; Michael, F.B.; Bolanle, A.M. Dynamics of co-infection in fish: A review of pathogen-host interaction and clinical outcome. *Fish Shellfish Immunol. Rep.* **2023**, *4*, 100096. [\[CrossRef\]](#) [\[PubMed\]](#)
50. Sano, T.; Morita, N.; Shima, N.; Akimoto, M. Herpesvirus cyprini: Lethality and oncogenicity. *J. Fish Dis.* **1991**, *14*, 533–543. [\[CrossRef\]](#)
51. Davison, A.J.; Kurobe, T.; Gatherer, D.; Cunningham, C.; Korf, I.; Fukuda, H.; Hedrick, R.P.; Waltzek, T.B. Comparative Genomics of Carp Herpesviruses. *J. Virol.* **2013**, *87*, 2908. [\[CrossRef\]](#) [\[PubMed\]](#)
52. Whittington, R.J.; Chong, R. Global trade in ornamental fish from an Australian perspective: The case for revised import risk analysis and management strategies. *Prev. Vet. Med.* **2007**, *81*, 92–116. [\[CrossRef\]](#) [\[PubMed\]](#)
53. Hedrick, R.P.; Waltzek, T.B.; McDowell, T.S. Susceptibility of Koi Carp, Common Carp, Goldfish, and Goldfish × Common Carp Hybrids to Cyprinid Herpesvirus-2 and Herpesvirus-3. *J. Aquat. Anim. Health* **2006**, *18*, 26–34. [\[CrossRef\]](#)
54. Ziarati, M.; Hassantabar, F. Chapter 29—Koi Herpesvirus Disease. In *Emerging and Reemerging Viral Pathogens*; Ennaji, M.M., Ed.; Academic Press: Cambridge, MA, USA, 2020; pp. 657–671. [\[CrossRef\]](#)
55. Bigarré, L.; Baud, M.; Cabon, J.; Antychowicz, J.; Bergmann, S.; Engelsma, M.; Pozet, F.; Reichert, M.; Castric, J. Differentiation between Cyprinid herpesvirus type-3 lineages using duplex PCR. *J. Virol. Methods* **2009**, *158*, 51–57. [\[CrossRef\]](#) [\[PubMed\]](#)
56. Kurita, J.; Yuasa, K.; Ito, T.; Sano, M.; Hedrick, R.P.; Engelsma, M.Y.; Haenen, O.L.M.; Sunarto, A.; Kholidin, E.B.; Chou, H.-Y.; et al. Molecular Epidemiology of Koi Herpesvirus. *Fish Pathol.* **2009**, *44*, 59–66. [\[CrossRef\]](#)
57. Engelsma, M.Y.; Way, K.; Dodge, M.J.; Voorbergen-Laarman, M.H.A.; Stone, D.M. Detection of novel strains of cyprinid herpesvirus closely related to koi herpesvirus. *Dis. Aquat. Org.* **2013**, *107*, 113–120. [\[CrossRef\]](#) [\[PubMed\]](#)
58. St-Hilaire, S.; Beevers, N.; Way, K.; Le Deuff, R.; Martin, P.; Joiner, C. Reactivation of koi herpesvirus infections in common carp *Cyprinus carpio*. *Dis. Aquat. Org.* **2005**, *67*, 15–23. [\[CrossRef\]](#) [\[PubMed\]](#)
59. Eide, K.E.; Miller-Morgan, T.; Heidel, J.R.; Kent, M.L.; Bildfell, R.J.; LaPatra, S.; Watson, G.; Jin, L. Investigation of koi herpesvirus latency in koi. *J. Virol.* **2011**, *85*, 4954–4962. [\[CrossRef\]](#) [\[PubMed\]](#)
60. Perelberg, A.; Ilouze, M.; Kotler, M.; Steinitz, M. Antibody response and resistance of *Cyprinus carpio* immunized with cyprinid herpesvirus 3 (CyHV-3). *Vaccine* **2008**, *26*, 3750–3756. [\[CrossRef\]](#) [\[PubMed\]](#)
61. Miyazaki, T.; Isshiki, T.; Katsuyuki, H. Histopathological and electron microscopy studies on sleepy disease of koi *Cyprinus carpio* koi in Japan. *Dis. Aquat. Org.* **2005**, *65*, 197–207. [\[CrossRef\]](#) [\[PubMed\]](#)
62. Adamek, M.; Baska, F.; Vincze, B.; Steinhagen, D. Carp edema virus from three genogroups is present in common carp in Hungary. *J. Fish Dis.* **2018**, *41*, 463–468. [\[CrossRef\]](#) [\[PubMed\]](#)
63. Zawisza, M.; Rebl, A.; Teitge, F.; Krzystyniak, B.; Piackova, V.; Gela, D.; Kocour, M.; Chadzinska, M.; Adamek, M.; Rakus, K. Stressing out—Carp edema virus induces stress and modulates immune response in common carp. *Front. Immunol.* **2024**, *15*, 1350197. [\[CrossRef\]](#) [\[PubMed\]](#)
64. Kafi, Z.Z.; Najafi, H.; Alishahi, M.; Rahmati-Holasoo, H.; Moulouki, A.; Ghalyanchilangeroudi, A. Detection and phylogenetic analysis of carp edema virus in common carp (*Cyprinus carpio*) in Iran; 2020–2021. *Aquaculture* **2022**, *558*, 738381. [\[CrossRef\]](#)
65. Matras, M.; Borzym, E.; Stone, D.; Way, K.; Stachnik, M.; Maj-Paluch, J.; Palusińska, M.; Reichert, M. Carp edema virus in Polish aquaculture—Evidence of significant sequence divergence and a new lineage in common carp *Cyprinus carpio* (L.). *J. Fish Dis.* **2017**, *40*, 319–325. [\[CrossRef\]](#) [\[PubMed\]](#)
66. Adamek, M.; Oschilewski, A.; Wohlsein, P.; Jung-Schroers, V.; Teitge, F.; Dawson, A.; Gela, D.; Piackova, V.; Kocour, M.; Adamek, J.; et al. Experimental infections of different carp strains with the carp edema virus (CEV) give insights into the infection biology of the virus and indicate possible solutions to problems caused by koi sleepy disease (KSD) in carp aquaculture. *Vet. Res.* **2017**, *48*, 12. [\[CrossRef\]](#) [\[PubMed\]](#)
67. Oyamatsu, T.; Matoyama, H.; Yamamoto, K.-Y.; Fukuda, H. A trial for the Detection of Carp Edema Virus by Using Polymerase Chain Reaction. *Aquac. Sci.* **1997**, *45*, 247–251.
68. Stevens, B.N.; Michel, A.; Liepnieks, M.L.; Kenelty, K.; Gardhouse, S.M.; Groff, J.M.; Waltzek, T.B.; Soto, E. Outbreak and treatment of carp edema virus in koi (*Cyprinus Carpio*) from Northern California. *J. Zoo Wildl. Med.* **2018**, *49*, 755–764. [\[CrossRef\]](#) [\[PubMed\]](#)
69. International Committee on Taxonomy of Viruses (ICTV). Genus: *Sprivivirus*. ICTV Report: *Rhabdoviridae*. 2025. Available online: <https://ictv.global/report/chapter/rhabdoviridae/rhabdoviridae/sprivivirus> (accessed on 3 September 2026).

70. Ahne, W. *Argulus foliaceus* L. and *Piscicola geometra* L. as mechanical vectors of spring viraemia of carp virus (SVCV). *J. Fish Dis.* **1985**, *8*, 241–242. [\[CrossRef\]](#)
71. Fijan, N.; Petrinc, Z.; Sulimanović, D.; Zwillenberg, L.O. Isolation of the viral causative agent from the acute form of infectious dropsy of carp. *Vet. Arh.* **1971**, *41*, 125–138.
72. Zhang, N.Z.; Zhang, L.F.; Jiang, Y.N.; Zhang, T.; Xia, C. Molecular analysis of spring viraemia of carp virus in China: A fatal aquatic viral disease that might spread in East Asian. *PLoS ONE* **2009**, *4*, e6337. [\[CrossRef\]](#) [\[PubMed\]](#)
73. Miller, O.; Fuller, F.; Gebreyes, W.; Lewbart, G.; Shchelkunov, I.; Shivappa, R.; Joiner, C.; Woolford, G.; Stone, D.; Dixon, P.; et al. Phylogenetic analysis of spring viremia of carp virus reveals distinct subgroups with common origins for recent isolates in North America and the UK. *Dis. Aquat. Org.* **2007**, *76*, 193–204. [\[CrossRef\]](#) [\[PubMed\]](#)
74. Stone, D.M.; Kerr, R.C.; Hughes, M.; Radford, A.D.; Darby, A.C. Characterisation of the genomes of four putative vesiculoviruses: Tench rhabdovirus, grass carp rhabdovirus, perch rhabdovirus and eel rhabdovirus European X. *Arch. Virol.* **2013**, *158*, 2371–2377. [\[CrossRef\]](#) [\[PubMed\]](#)
75. Ahne, W. The influence of environmental temperature and infection route on the immune response of carp (*Cyprinus carpio*) to spring viremia of carp virus (SVCV). *Vet. Immunol. Immunopathol.* **1986**, *12*, 383–386. [\[CrossRef\]](#) [\[PubMed\]](#)
76. Ouyang, P.; Li, Q.; Liu, S.; Li, Y.; Li, S.; Zhou, Y.; Jia, P.; Chen, D.; Huang, X.; Geng, Y. Histopathology and transcriptome profiling reveal features of immune responses in gills and intestine induced by Spring viremia of carp virus. *Fish Shellfish Immunol.* **2024**, *152*, 109726. [\[CrossRef\]](#) [\[PubMed\]](#)
77. Embregts, C.W.E.; Rigau, D.; Veselý, T.; Pokorová, D.; Lorenzen, N.; Petit, J.; Houel, A.; Dauber, M.; Schütze, H.; Boudinot, P.; et al. Intramuscular DNA Vaccination of Juvenile Carp against Spring Viremia of Carp Virus Induces Full Protection and Establishes a Virus-Specific B and T Cell Response. *Front. Immunol.* **2017**, *8*, 1340. [\[CrossRef\]](#) [\[PubMed\]](#)
78. Zhao, Z.; Jiang, F.-Y.; Zhou, G.-Q.; Duan, H.-X.; Xia, J.-Y.; Zhu, B. Protective immunity against spring viremia of carp virus by mannose modified chitosan loaded DNA vaccine. *Virus Res.* **2022**, *320*, 198896. [\[CrossRef\]](#) [\[PubMed\]](#)
79. Duan, H.; Zhao, Z.; Jin, Y.; Wang, Z.; Deng, J.; He, J.; Zhu, B. PEG-modified subunit vaccine encoding dominant epitope to enhance immune response against spring viraemia of carp virus. *J. Fish Dis.* **2021**, *44*, 1587–1594. [\[CrossRef\]](#) [\[PubMed\]](#)
80. Shin, S.; Kulatunga, D.C.M.; Dananjaya, S.H.S.; Nikapitiya, C.; Lee, J.; De Zoysa, M. *Saprolegnia parasitica* Isolated from Rainbow Trout in Korea: Characterization, Anti-Saprolegnia Activity and Host Pathogen Interaction in Zebrafish Disease Model. *Mycobiology* **2017**, *45*, 297. [\[CrossRef\]](#) [\[PubMed\]](#)
81. Lang-Yona, N.; Pickersgill, D.A.; Maurus, I.; Teschner, D.; Wehking, J.; Thines, E.; Pöschl, U.; Després, V.R.; Fröhlich-Nowoisky, J. Species Richness, rRNA Gene Abundance, and Seasonal Dynamics of Airborne Plant-Pathogenic Oomycetes. *Front. Microbiol.* **2018**, *9*, 2673. [\[CrossRef\]](#) [\[PubMed\]](#)
82. Song, J.; Han, G.; Wang, Y.; Jiang, X.; Zhao, D.; Li, M.; Yang, Z.; Ma, Q.; Parales, R.E.; Ruan, Z.; et al. Pathway and kinetics of malachite green biodegradation by *Pseudomonas veronii*. *Sci. Rep.* **2020**, *10*, 4502. [\[CrossRef\]](#) [\[PubMed\]](#)
83. Mazlumi, A.; Panahi, B.; Hejazi, M.A.; Nami, Y. Probiotic potential characterization and clustering using unsupervised algorithms of lactic acid bacteria from saltwater fish samples. *Sci. Rep.* **2022**, *12*, 11952. [\[CrossRef\]](#) [\[PubMed\]](#)
84. Erdei, N.; Hardy, T.; Verebelyi, V.; Weiperth, A.; Baska, F.; Eszterbauer, E. New Insights into the Morphological Diversity of *Saprolegnia parasitica* (Oomycota) Strains under In Vitro Culture Conditions. *J. Fungi* **2023**, *9*, 982. [\[CrossRef\]](#) [\[PubMed\]](#)
85. Ke, X.L.; Wang, J.; Gu, Z.M.; Li, M.; Gong, X.N. Morphological and molecular phylogenetic analysis of two *Saprolegnia* sp. (Oomycetes) isolated from silver crucian carp and zebra fish. *Mycol. Res.* **2009**, *113*, 637–644. [\[CrossRef\]](#) [\[PubMed\]](#)
86. Wang, Y.; Gao, X.; Wang, T.; Zhang, Y.; Hu, K. Effects of *Saprolegnia parasitica* on pathological damage and metabolism of *Epithelioma papulosum cyprini* cell. *Dev. Comp. Immunol.* **2025**, *162*, 105311. [\[CrossRef\]](#) [\[PubMed\]](#)
87. Ali, F.F.; AL-Tae, S.K.; AL-Jumaa, Z.M. Isolation, molecular identification, and pathological lesions of *Saprolegnia* spp. isolated from common carp, *Cyprinus carpio* in floating cages in Mosul, Iraq. *Vet. World* **2020**, *13*, 2759. [\[CrossRef\]](#) [\[PubMed\]](#)
88. EFSA (EFSA Panel on Contaminants in the Food Chain). Scientific opinion on malachite green in food. *EFSA J.* **2016**, *14*, e04530. [\[CrossRef\]](#)
89. Sherif, A. Treatment trails of saprolegniosis in *Oreochromis niloticus*. *Alex. J. Vet. Sci.* **2016**, *49*, 99–104. [\[CrossRef\]](#)
90. Tedesco, P.; Fioravanti, M.L.; Galuppi, R. In vitro activity of chemicals and commercial products against *Saprolegnia parasitica* and *Saprolegnia delica* strains. *J. Fish Dis.* **2019**, *42*, 237–248. [\[CrossRef\]](#) [\[PubMed\]](#)
91. Khalil, R.H.; Saad, T.T.; Selema, T.A.M.; Abdel-Latif, H.M.R. *Branchiomyces demigrans* infection in farm-reared common carp (*Cyprinus carpio* L.) and Nile tilapia (*Oreochromis niloticus*) at different localities in Egypt, with special emphasis to the role of environmental stress factors. *Int. J. Innov. Stud. Aquat. Biol. Fish.* **2015**, *1*, 15–23.
92. Goodwin, A.E. Branchiomycosis. In *FHS Blue Book: Suggested Procedures for the Detection and Identification of Certain Finfish and Shellfish Pathogens*, 2012 Ed.; American Fisheries Society, Fish Health Section: Bethesda, MD, USA, 2012; pp. 1–8.
93. Al-Jumaa, Z.M.; Al-Tae, S.K.; Jaber, M.T.; Rahhawi, A.M. Histopathological Alteration and Molecular Detection of Gills Rot Fungus in Carp Fish. *J. Appl. Vet. Sci.* **2024**, *9*, 54–59. [\[CrossRef\]](#)

94. Sheikh, G.; Mankodi, P. A case report of Branchiomyces sp. infection in carp (*Catla catla*) from Vadodara, Gujarat. In *Proceedings of the National Conference on Present Day Biology: Recent Advances in Biological Research*; The M. S. University of Baroda: Vadodara, India, 2022. Available online: <https://www.researchgate.net/publication/361255617> (accessed on 17 February 2025).
95. Cogliano, V.J.; Baan, R.; Straif, K.; Grosse, Y.; Lauby-Secretan, B.; El Ghissassi, F.; Bouvard, V.; Benbrahim-Tallaa, L.; Guha, N.; Freeman, C.; et al. Preventable Exposures Associated with Human Cancers. *J. Natl. Cancer Inst.* **2011**, *103*, 1827–1839. [[CrossRef](#)] [[PubMed](#)]
96. Cremonesini, D.; Thomson, A. Lung colonization with *Aeromonas hydrophila* in cystic fibrosis believed to have come from a tropical fish tank. *J. R. Soc. Med.* **2008**, *101*, S44–S45. [[CrossRef](#)] [[PubMed](#)]
97. Long, D.; Li, M.; Ma, L.; Huang, J.; Lv, C.; Chen, Y.; Cheng, Z.; Liu, C.; Huang, H.; Guo, X.; et al. Epidemiological and genetic characteristics of *Vibrio vulnificus* from diverse sources in China during 2012–2023. *Commun. Biol.* **2025**, *8*, 9. [[CrossRef](#)] [[PubMed](#)]
98. Ng, Q.X.; Seng, C.; Chan, F.Z.Y.; Yeo, W.S. Zoonosis: An unusual case of chronic osteomyelitis. *Singap. Med. J.* **2019**, *60*, 379–381. [[CrossRef](#)]
99. Mah, J.; Walding, K.; Liang, B.; Rinsky, L.; Mathew, R.; Budvytiene, I.; Banaei, N. Mycobacterium marinum infection after iguana bite in Costa Rica. *Emerg. Infect. Dis.* **2023**, *29*, 1278–1280. [[CrossRef](#)] [[PubMed](#)]
100. Gray, S.J. *Aeromonas hydrophila* in livestock: Incidence, biochemical characteristics and antibiotic susceptibility. *Epidemiol. Infect.* **1984**, *92*, 365–375. [[CrossRef](#)] [[PubMed](#)]
101. Liang, B.; Ji, X.; Jiang, B.; Yuan, T.; Gerile, C.L.M.; Zhu, L.; Wang, T.; Li, Y.; Liu, J.; Guo, X.; et al. Virulence, Antibiotic Resistance, and Phylogenetic Relationships of *Aeromonas* spp. Carried by Migratory Birds in China. *Microorganisms* **2023**, *11*, 7. [[CrossRef](#)] [[PubMed](#)]
102. Hernández-Cabanyero, C.; Sanjuán, E.; Mercado, L.; Amaro, C. Evidence that fish death after *Vibrio vulnificus* infection is due to an acute inflammatory response triggered by a toxin of the MARTX family. *Fish Shellfish Immunol.* **2023**, *142*, 109131. [[CrossRef](#)]
103. Ayala, A.J.; Ogbunugafor, C.B. When *Vibrios* Take Flight: A Meta-Analysis of Pathogenic *Vibrio* Species in Wild and Domestic Birds', in *Vibrio* spp. Infections. In *Advances in Experimental Medicine and Biology*; Springer International Publishing: Cham, Switzerland, 2023; Volume 1404, pp. 295–336. [[CrossRef](#)]
104. Marsh, D. *Natural Capital of Freshwater Fisheries in England*; Fresh Water Natural Capital Report, The Rivers Trust: Callington, UK, 2021. Available online: <https://storymaps.arcgis.com/stories/db08425aeca4409eab0f38fa74a4fc97> (accessed on 3 September 2026).

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