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REVIEW

REVIEWS IN Aquaculture

# Diseases of the giant river prawn *Macrobrachium rosenbergii*: A review for a growing industry

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## Abstract

The giant river prawn, *Macrobrachium rosenbergii*, is a major focus of aquaculture in tropical and sub-tropical regions around the globe. Over the last 30 years, culture of *M. rosenbergii* has increased exponentially as demand has risen both for domestic consumption and for international export trade. As with many aquaculture species increases in production have been accompanied by the emergence of diseases affecting yield, profit and trading potential. Disease-causing agents include pathogens infecting other crustaceans, such as *Decapod Iridescent Virus* (DIV1), as well as pathogens only known from *M. rosenbergii* such as White Tail Disease caused by Macrobrachium rosenbergii nodavirus (MrNV) and extra small virus (XSV). Here, we review the pathogenic agents associated with the culture of *M. rosenbergii* since commercial culture began in earnest during the 1970s. Particular emphasis is given to pathogens first identified in other aquaculture host species, but which have subsequently been shown to infect and cause disease in *M. rosenbergii*. As polyculture of *M. rosenbergii* with other aquaculture species is common practice, including culture with other decapods, crabs and fish, increased pathogen transfer among these farmed species may occur as *M. rosenbergii* aquaculture increases in the future.

**KEYWORDS**
aquaculture, emerging disease, giant river prawn, polyculture

## 1 | INTRODUCTION

Aquaculture is the fastest-growing farmed food sector, with the proportion of cultured to caught seafood increasing year-on-year.[1,2] Global aquaculture production for fish, crustaceans and molluscs surpassed 87 million tonnes in 2020, just under half of all world production.[2] Crustacean production by aquaculture far surpasses that by capture, with more than 11,000,000 tonnes in 2020 from aquaculture compared to ~6,000,000 tonnes by capture.[2] Despite these considerable production figures, it is estimated that up to 40% of tropical shrimp aquaculture production is lost annually,[3] equating to losses of over 3 billion USD, and this is primarily due to infection with viral agents.[4] Losses on this scale threaten global food security, with most of the consumption of cultured crustaceans being outside of producing countries, making management and mitigation of disease of key importance to the future success of crustacean aquaculture.[4] Stentiford et al. (2012) outlines a holistic strategy involving both producer and consumer nations to improve husbandry and farm management, better understand pathogens and their spread (either by movement of animals or alternative hosts) and learn lessons from previous disease outbreaks. This review focuses on these points to outline the pathogenic agents in *Macrobrachium rosenbergii* culture, identify other hosts

that these agents are able to infect, and discuss how culture practices can mitigate disease transfer.

The freshwater prawn genus *Macrobrachium* comprises over 200 species, distributed throughout the world's tropical and subtropical regions.[5] The giant river prawn, *M. rosenbergii*, is indigenous to the Indo-Pacific area,[6] but has been transferred from its native locations to almost every continent for farming purposes following the development of methods to mass-produce postlarvae.[5] *M. rosenbergii* is primarily found in inland freshwater areas, including but not limited to rivers, swamps and canals, particularly where turbidity makes the water cloudy or opaque. Most species of *Macrobrachium*, including *M. rosenbergii*, require brackish water in the larval stages of development before moving to freshwater as postlarvae, and thus are frequently found in habitats that are connected to the sea.[5]

*M. rosenbergii* is cultured worldwide in tropical and sub-tropical climate regions.[7] Its culture provides significant income, as well as both direct and indirect employment and a food source in areas of poverty.[7] The first countries to report production of *M. rosenbergii* to FAO of the UN (Food and Agriculture Organisation of the United Nations) were Thailand and Vietnam in 1975, followed over the next two decades by Myanmar, Taiwan, India, Bangladesh and China.[2] Prior to 1980, global production was below 3000 metric tonnes per year[2]; since then, the species has become a desirable target for commercial aquaculture due to its large size, tolerance to some diseases that cause mass mortality in shrimp aquaculture, and considerably higher market price than the same weight of marine penaeid shrimp.[7,8] In 2020, worldwide production of *M. rosenbergii* reached 294,081 tonnes (~$2.4bn USD), with over 50% of global production in China.[2]

The expansion of *M. rosenbergii* culture has resulted in numerous technological advancements and investments to facilitate the sector. These include the production of monosex cultures of *M. rosenbergii* by RNA interference (RNAi) or injection of suspended hypertrophied androgenic gland cells in order to reduce aggression (reducing mortality by injury, decreasing losses), increase yield and to make harvest size more uniform[9,10]; the construction of intensive hatcheries[11] and, the production of specific-pathogen-free (SPF) stocks.[12] However, despite these significant advances, increased incidence of disease has accompanied expansion of the industry, with the emergence of novel pathogens that current SPF stock production programmes do not consider.

Polyculture of *M. rosenbergii* is common in many countries including China, India, Bangladesh and Brazil, which all employ fish-prawn polyculture systems with species of carp and/or tilapia.[13–16] One of the reasons for deploying polyculture of *M. rosenbergii* with other aquaculture species is to mitigate losses from disease in one species by maintaining yield and profit from the others.[17] Another potential advantage derives from the hypothesis that the effect/impact of pathogens of one cultured species can be modified/quenched by the presence of (an)other(s).[18]

Fish-prawn polyculture systems are thought to reduce the frequency of algal blooms commonly observed in finfish monoculture.[19] In some scenarios, it is common for rice to be farmed alongside prawns and fish.[13–16] Prawn–shrimp polyculture systems are less common but have been reported in China and Bangladesh with the former culturing *M. rosenbergii* with the shrimp *Penaeus (Litopenaeus) vannamei*, and the latter farming *M. rosenbergii* with *P. monodon*.[13,20] In China, prawn–prawn polyculture with *M. rosenbergii* and *Macrobrachium nipponense*, the oriental freshwater prawn, also occurs.[13] China is the world's largest producer of the Chinese mitten crab, *Eriocheir sinensis*[2]; in 2012, 90% of all *M. nipponense* polyculture was with *E. sinensis*, with over 300,000 ha of land dedicated to this type of culture.[13] Reports suggest that *E. sinensis*–*M. rosenbergii* polyculture as well as *M. rosenbergii*–*Anodonta woodiana* (Chinese pond mussel) polyculture also occurs in China.[21,22]

Detection of known and emerging pathogens has become more accessible through advances in molecular biology techniques that have allowed the rapid characterisation of novel pathogens without relying on traditional pathogen culturing techniques.[23] Numerous disease-causing agents are known to infect *M. rosenbergii*, including several bacterial, viral, fungal and other eukaryotic pathogens, causing losses to the aquaculture sector either by mortality, or by the production of a smaller or substandard animal with decreased market value.[24]

When new aquaculture practices emerge concurrently with a concerted effort to improve the productivity of freshwater prawn farming, similar to marine shrimp farm operations, the risk of novel prawn diseases also increases (Figure 1). Since the last review of disease in *M. rosenbergii* in 2012,[24] several agents have been newly identified as pathogenic in giant river prawn culture, mainly aided by enhancement in molecular detection. In this review, we describe pathogenic agents of disease that have impacted *M. rosenbergii* since the origins of commercial culture of this species; further, we discuss the potential host range of these pathogens and their possible impact on the health of *M. rosenbergii* and those species with which it is often co-cultured.

## 2 | VIRUSES

As the culture of freshwater prawns (Genus: *Macrobrachium*) and penaeid shrimp (Family: Penaeidae) has increased, the incidence of viral infections has also increased. It has also become apparent that viral host specificity is not bound to fresh- or saline water, with several viruses of penaeid shrimps able to infect freshwater prawn species and vice versa. A summary of viruses known to infect *M. rosenbergii* is provided in Table 1.

Hepatopancreatic parvovirus (HPV) was the first reported virus of *M. rosenbergii*,[25] and has since been reclassified in the genus *Aquambidensoviridae*.[26] The 25–30 nm icosahedral virus, discovered in giant river prawns in Malaysia, was not associated with mortalities and primarily infected the hepatopancreatic tissue of postlarvae, with basophilic intranuclear inclusions present in tubule epithelial cells. It was first thought that this parvo-like virus was the same as another parvovirus infecting the Korean *Penaeus (Fenneropenaeus) chinensis*, a species of marine shrimp. However, differences in virus size and infected

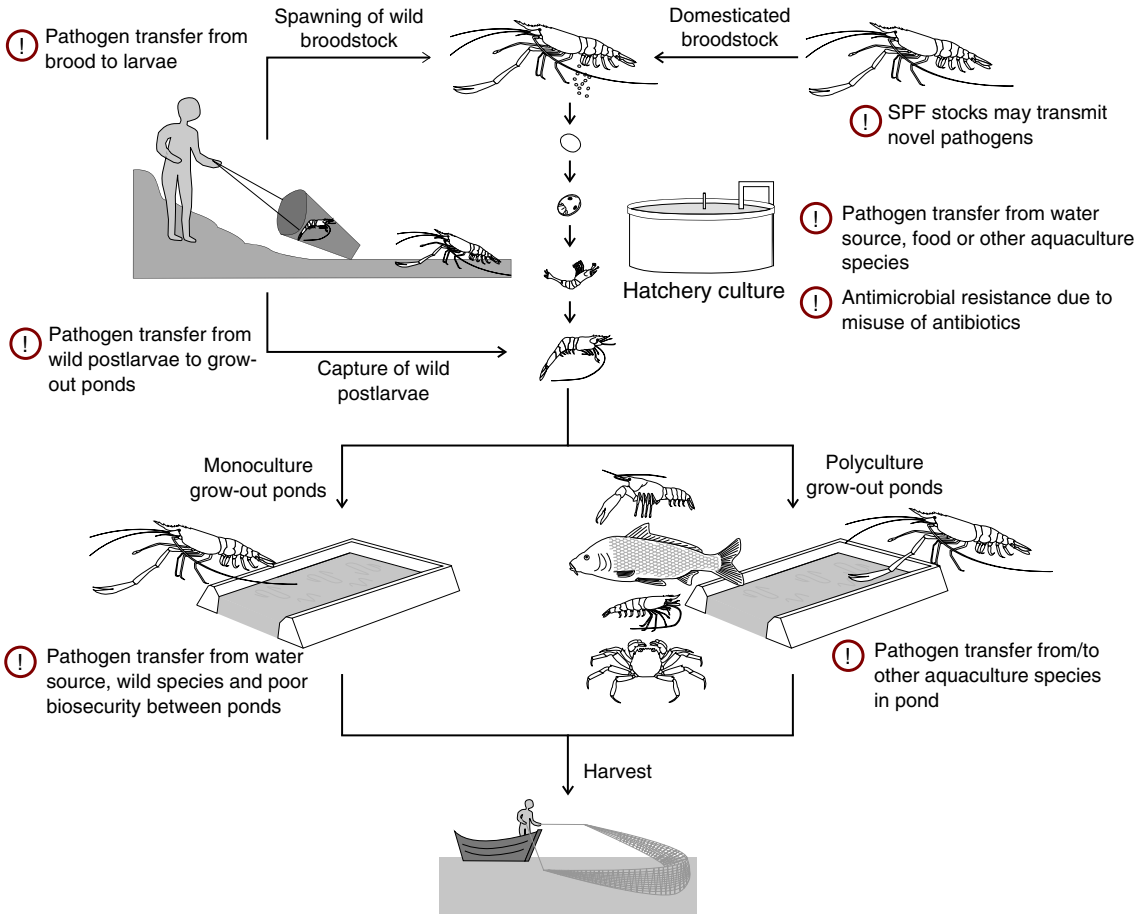



**FIGURE 1** Culture cycle of *M. rosenbergii* highlighting the potential for introduction of disease into the system

host cell histopathology justified its designation as separate species.[27] Since its discovery, there have been few reports of HPV in *M. rosenbergii*, most likely due to the lack of mortality associated with infection; however, HPV is associated with mortalities in penaeid shrimp.[28] The only reports of HPV infection of *M. rosenbergii* since the distinction between the two parvoviruses have been in postlarvae from Thailand and Malaysia in 2007 and 2009, respectively.[29,30] A more recent study attempted to detect HPV in *M. rosenbergii* collected from wild populations in India, but could not amplify the virus by PCR.[31]

Another parvovirus, *Penaeus stylirostris penstyldensovirus 1* (PstDV1),[26] commonly named infectious hypodermal haematopoetic necrosis virus (IHHNV), a 20–22 nm icosahedral-shaped virus, has been shown to cause disease in penaeid shrimp.[32] Gross clinical signs of infections vary with species, from runt deformity syndrome in *P. vannamei* and *P. monodon* to a whitish colour and opaque abdominal musculature resulting in mortality of *Penaeus stylirostris* postlarvae and adults.[32] In 2004, a pond in Taiwan reported mortality of *M. rosenbergii* postlarvae and juveniles.[33] The mortality was linked to the slow growth of animals, later determined to be infected with IHHNV, which manifested as muscular atrophy, a reddish colouration, and deformities to the cuticle and opaque musculature. Histopathological examination identified Cowdry type A and B eosinophilic intranuclear inclusions in hepatopancreatic epithelial cells, a different presentation to the basophilic inclusion seen in infection with HPV.

By far the biggest threat to the viability of *M. rosenbergii* aquaculture worldwide has been the emergence of a novel nodavirus, Macrobrachium rosenbergii nodavirus (MrNV) (Family: *Nodaviridae*) and an associated satellite virus, extra small virus (XSV), infecting *M. rosenbergii* and causing white tail disease (WTD).[34] The impact this nodavirus has had on the culture of *M. rosenbergii* has resulted in a large number of research publications, from pathogenicity of the virus to host response. The first report of MrNV was in hatchery-reared *M. rosenbergii* postlarvae in the French West Indies in 1994.[35] However, MrNV is likely synonymous with Macrobrachium muscle virus (MMV), a virus of the same size that caused similar clinical signs of disease and post-larval mortalities since 1992 in Taiwan.[36] The clinical signs of WTD are whitish colouration of the abdominal and tail muscle, with discolouration spreading from the tail towards the head as the infection progresses.[35] Histopathological signs of WTD include hyaline necrosis of muscle fibres, with moderate oedema, necrosis, haemocyte infiltration and fibrosis in affected muscles; with the presence of pale to dark basophilic intracytoplasmic inclusions in muscle cells and hepatopancreatic connective tissue cells.[35] MrNV is a 26–27 nm non-enveloped, icosahedral virus with a genome composed of two

**TABLE 1** Viruses infecting *M. rosenbergii*

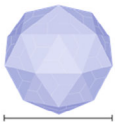
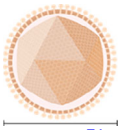
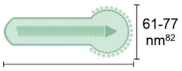
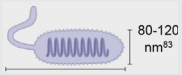
| Virus | Family | Structure | Clinical signs of infection | Histopathological signs of infection |
|---|---|---|---|---|
| Covert mortality nodavirus (CMNV)[63] | *Nodaviridae*[63] |  32 nm[63] | Slow growth, pale hepatopancreas, soft shell, muscle whitening and necrosis, mortality.[65] | Muscle fragmentation, muscular lysis and myonecrosis in whitish muscle lesions. Eosinophilic inclusions in the tubular epithelium of hepatopancreas and vacuolisation of hepatopancreocytes.[65] |
| Crustacea hepe-like virus 1 (CHEV1)[48] | [a]*Hepeviridae*[48] |  ? | Slow growth (Iron Prawn Syndrome).[48] | Unknown |
| *Decapod iridescent virus 1* (DIV1)[76] | *Iridoviridae*[76] |  ~160 nm[76] | White triangle under the carapace at the bottom of the rostrum, yellow gills, loss of swimming ability, migration to deep water, mortality.[79] | Eosinophilic inclusions and karyopyknosis in haemopoietic tissue.[79] |
| [b] *Gill-associated virus* (GAV)[52] | *Roniviridae*[52] |   | Unknown.[53] | Unknown. |
| Hepatopancreatic parvovirus (HPV)[28] | *Parvoviridae*[28] |  25–30 nm[28] | None.[28,29] | Basophilic intranuclear inclusion in hepatopancreas epithelial cells.[29] |
| [b]Infectious hypodermal haematopoietic necrosis virus (IHHNV)[32] AKA *Penaeus stylirostris penstyldensovirus 1* (PstDV1)[26] | *Parvoviridae*[32] |  20–22 nm[32] | Slow growth, muscular atrophy, reddish discolouration, deformities to cuticle, opaque musculature, mortality.[33] | Cowdry type A and B eosinophilic intranuclear inclusions in hepatopancreatic epithelial cells.[33] |
| Infectious precocity virus (IPV)[49] | [a]*Flaviviridae*[49] |  40–60 nm[49] | Slow growth (Iron Prawn Syndrome).[49] | Eosinophilic inclusions in neurosecretory cells of the organ of Bellonci, globuli cells of the hemielipsoid body, lamina ganglionaris, fasciculated zone, the onion body and the sinus gland. Eosinophilic and granular cytoplasm in the laminar ganglionaris and ganglia tissues.[49] |
| Macrobrachium rosenbergii Golda virus (MrGV)[50] | [a]*Roniviridae*[50] |   | Problems with swimming and feeding, slow growth, white discolouration, mortality.[50] | Unknown. |
| [b] Macrobrachium rosenbergii nodavirus (MrNV)[35] | *Nodaviridae*[37] |  24–26 nm[37] | White opaque discolouration of abdomen that gradually progresses towards the head, abnormal exuviae, mortality.[35] | Muscle tissues show hyaline necrosis of muscle fibres, with moderate oedema, necrosis, haemocyte infiltration and fibrosis. Presence of pale to dark basophilic intracytoplasmic inclusions in muscles, hepatopancreatic connective tissue cells.[35] |
| [b]Extra small virus (XSV) AKA *Macrobrachium satellite virus 1*[34] | *Sarthroviridae*[34] |  15 nm[39] | | |

**TABLE 1** (Continued)

| Virus | Family | Structure | Clinical signs of infection | Histopathological signs of infection |
|---|---|---|---|---|
| Macrobrachium rosenbergii Taihu virus (MrTV)[42] | [a]*Dicistroviridae*[42] |  25–29 nm[42] | Problems with feeding and moulting, reddish exuviae, decreased response to stimuli, mortality.[42] | Pale to dark basophilic cytoplasmic inclusions in the cuticle epithelium.[42] |
| Monodon-type baculovirus (MBV)[60] AKA *Penaeus monodon Nudivirus* (PmNV)[58] | *Baculoviridae*[60] |   234–316 nm[82] | Unknown[62] | Large nuclei in hepatopancreatic tubule epithelial cells with diffuse, central, eosinophilic inclusions and marginated nucleoli and chromatin.[62] |
| *White spot syndrome virus*[b] (WSSV)[67] | *Nimaviridae*[67] |   250–380 nm[83] | White spots on carapace, lethargy, increased cannibalism, mortality.[70–73] | Hypertrophied Feulgen-positive nuclei in target tissues, basophilic and Cowdry A type inclusion bodies, chromatin margination and karyorrhexis.[70] |

[a]Predicted family classification based on sequence similarity and/or virus ultrastructure.
[b]Pathogens listed by the World Organisation for Animal Health (WOAH).

fragments of linear single-stranded RNA (ssRNA) of 2.9 and 1.3 kb, respectively.[37] Qian et al. (2003) identified a 15 nm non-enveloped icosahedral satellite virus associated with MrNV infection in prawns with WTD.[34] Extra small virus (XSV), also known as *Macrobrachium satellite virus 1* (Family: *Sarthroviridae*, Genus: *Macronovirus*),[38] has a 796 nucleotide (nt) linear ssRNA genome with a short poly(A) tail, encoding two capsid proteins CP-17 and CP-16.[39] WTD is the first record of a satellite-nodavirus association.[40] A recent study discovered that experimentally MrNV alone could mortality associated with WTD lesions, with no significant differences in mortality compared to MrNV + XSV infection[41]; however, it is unknown whether if, in nature, MrNV can occur on its own. The same study also determined that XSV alone cannot induce WTD lesions or mortality, providing evidence that MrNV is the causative agent of WTD. The role of XSV in WTD is yet to be determined.

Macrobrachium rosenbergii Taihu virus (MrTV), a proposed dicistrovirus, has been associated with mass mortalities (80%–90%) of the zoeal stage of *M. rosenbergii* in Chinese hatcheries in 2009.[42] Affected larvae had problems with feeding and moulting, had a reddish shed shell, a decreased response to stimuli, and sank to the bottom of tanks before death. Histology showed pale to dark basophilic cytoplasmic inclusions in the cuticle epithelium of infected larvae. MrTV forms 25–29 nm hexagonal non-enveloped virus particles and has a 10,303 nt positive sense ssRNA genome with a similar genome architecture to other *Dicistroviridae*. Phylogenetic analysis of the RNA-dependent RNA polymerase (RdRp) protein sequence of MrTV and other *Dicistroviridae* showed that MrTV is most closely related to the Taura syndrome virus (TSV), which causes mass mortalities of marine shrimp.[42]

In recent years, *M. rosenbergii* culture in China has been facing problems associated with growth retardation of prawns known as iron prawn syndrome (IPS)—prawns have body weights and lengths much smaller than normal.[43] Several studies have been conducted to determine a cause for this phenomenon, including environmental factors and known pathogens[44–46]; however, no one factor has been identified as the primary cause. The syndromic nature of this disease is discussed further in the Section 6. When IPS prawns are cultured with unaffected prawns, the latter begin to exhibit reduced growth, suggesting that the cause may be of pathogen aetiology.[47] One potential cause of IPS is a novel hepe-like virus identified in *M. rosenbergii* with slow growth from a farm in China.[48] Crustacea hepe-like virus 1 (CHEV1), identified via metatranscriptomics, has a 7750 nt positive sense ssRNA genome with three hypothetical open reading frames. Although CHEV1 could not be identified as the primary cause of iron-prawn by Dong et al. (2020), it highlights the importance of emerging pathogens and syndromic disease conditions in aquaculture.

Further to the discovery of CHEV1, another virus, infectious precocity virus (IPV) or *Crustaflavivirus infeprecoquis*, has been linked to IPS in *M. rosenbergii*.[49] The 12,630 nt single-stranded RNA virus is proposed to be of the family *Flaviviridae*, and was assembled from metatranscriptomic sequencing of IPS prawns. A disease challenge using a viral extract from IPS prawns was able to reproduce the IPS phenotype, with eosinophilic viral inclusions were seen in multiple tissue types (Table 1). The same study[49] also screened prawns from farms with and without IPS to determine whether the virus was present. All prawns collected from farms experiencing IPS were positive for IPS, whereas all farms with no gross clinical signs of IPS were negative by PCR.

The role of emerging pathogens in giant river prawn aquaculture was highlighted in Bangladesh in 2011. Hatcheries in southern Bangladesh experienced mass mortalities of larval *M. rosenbergii*, resulting in a decline in the number of hatcheries actively producing *M. rosenbergii* from >60 to 12, and a drop in the number of postlarvae produced from >200 million to 27.75 million in the last decade.[50] Affected larvae exhibited problems with swimming, feeding and growth, with moribund animals appearing whitish in colour compared to healthy animals. A recent study determined that a novel 29,110 nt positive-sense ssRNA virus, Macrobrachium rosenbergii Golda virus

(MrGV)—assembled from metatranscriptomic data, proposed to belong to *Nidovirales*, was present in hatcheries with mass mortality events.[50] Phylogenetically, the closest known relative of MrGV is *Gill-associated virus* (GAV), which comprises genotypes that cause mass mortalities in penaeid shrimp culture. Since the publication of MrGV, the virus has also been identified in metatranscriptomic data from postlarvae in China, but in the absence of disease.[51]

The yellow head viruses (YHV) are positive sense ssRNA viruses of the order *Roniviridae*.[52] To date, YHV comprises eight genotypes.[53] The most notable genotype of YHV is yellow head virus genotype 1 (YHV-1), the only genotype notifiable to the World Organisation for Animal Health (WOAH, previously OIE), which forms enveloped, rod-shaped virions and is the causative agent of yellow head disease (YHD) that has resulted in mass mortalities of cultured penaeid shrimp.[54] YHV genotype 2 (YHV-2), more commonly known as *Gill-associated virus* (GAV), is also associated with mortalities caused by gill-associated virus disease. Genotypes three to seven (YHV-3 to YHV-7) commonly occur in healthy *P. monodon* and are rarely or never associated with disease or mortality.[55] However, in recent years, yellow head virus genotype eight (YHV-8) has been detected in shrimp exhibiting acute hepatopancreatic necrosis disease (AHPND) in Chinese *F. chinensis*, but the pathology of YHV-8 in the absence of other pathogens is not known.[56] Longyant et al. (2005) screened for YHV-1 in *M. rosenbergii* collected from or nearby a YHV-affected *P. monodon* farm in Thailand as well as experimentally infected *M. rosenbergii*.[57] YHV-1 was not detected in either group of *M. rosenbergii*, suggesting that it may not be a susceptible species. However, Yang et al. (2016) were able to detect YHV in 21% of cultured *M. rosenbergii* ($n = 19$) using a novel LAMP assay designed to target a conserved region of the YHV complex in YHV-1, YHV-2 and YHV-8 genotypes.[53] The degrees to which *M. rosenbergii* is susceptible to different YHV genotypes, and can be a reservoir of YHV, are yet to be determined.

Monodon-type baculovirus (MBV) (recently re-named *Penaeus monodon nudivirus* [PmNV][58]); is an enveloped, rod-shaped, double-stranded DNA virus infecting the hepatopancreatic duct and tubule epithelium of penaeid shrimp.[59] First identified in Taiwanese *P. monodon* in 1977,[60,61] PmNV has since been shown to have a wide host range of penaeid shrimp and infections have been reported around the globe.[59] PmNV has been observed in all life stages of penaeid shrimp, but is most lethal to late larval, post-larval and juvenile shrimp.[59] Gangnonngiw et al. (2010) identified that *M. rosenbergii* postlarvae from Thailand had similar lesions in the hepatopancreas as that of *P. monodon* in the early stages of infection with PmNV.[62] Hepatopancreatic epithelial cells had enlarged nuclei with diffuse, central, eosinophilic inclusions. No mortalities were associated with infection with PmNV in *M. rosenbergii*; however, some individuals were noted to have co-infections of PmNV and HPV. As these infections have only been identified by histological observations, it is not clear whether the nudivirus infections seen in *M. rosenbergii* are caused by the same nudivirus (PmNV) that infects penaeid shrimp. As nudiviruses are quite diverse, genomic comparisons are needed to determine whether these viruses are the same or closely related.

Covert mortality nodavirus (CMNV), a 32 nm icosahedral nodavirus first identified in *P. vannamei*, causes viral covert mortality disease (VCMD) in penaeid shrimp.[63,64] CMNV, named after the behaviour of infected shrimp, causes moribund shrimp to move to deep water rather than swimming to the surface or shallow water, meaning that infections in ponds are difficult to detect until mortalities occur and shrimp float to the surface.[64] Infected shrimp have similar clinical and histopathological signs of infection to *M. rosenbergii* infected with MrNV, with hepatopancreatic atrophy and necrosis, empty stomach and guts, soft shells, slow growth and abdominal muscle whitening and necrosis.[63] Subsequent epidemiological studies have shown that CMNV is present in several aquaculture species including *F. chinensis*, *Penaeus (Marsupenaeus) japonicus*, *P. monodon* and *M. rosenbergii*,[65] suggesting that this virus is a threat to a number of crustacean species. *M. rosenbergii* infected with CMNV had a pale hepatopancreas, soft shell and muscle whitening and necrosis. These clinical signs of disease were shared with *P. vannamei* and *F. chinensis*; slow growth is also seen in *M. rosenbergii*, *P. vannamei*, *P. japonicus and P. monodon* infected with CMNV.[65] Liu et al. (2018) identified 11 species of co-habiting invertebrates, present in ponds with CMNV infections, to also be positive by RT–PCR and RT-LAMP assay for the nodavirus.[66] Of these 11 species, five were shown to be infected with CMNV by histopathology: *Columbarium sinense*, a species of seasnail; *Diogenes edwardii*, a hermit crab; *Ocypode cordimundus*, a ghost crab; the amphipod *Parathemisto gaudichaudi*, and fiddler crab *Tubuca arcuate*.

*White spot syndrome virus* (WSSV) is a double-stranded DNA virus of the *Nimiviridae* family that causes mass mortalities in penaeid shrimp species,[67] but has also been shown to have an extensive host range, including crabs and lobsters.[68] Susceptibility of *M. rosenbergii* to WSSV has been a topic of debate, with different studies suggesting varying levels of susceptibility. WSSV was first detected in *M. rosenbergii* by Lo et al. (1996) in cultured prawns from Taiwan; prawns exhibited clinical signs of infection with WSSV and tested positive with nested PCR, but not single-round PCR.[69] A study in 1998 collected giant river prawns from culture farms and experimentally-infected adults, postlarval and larval *M. rosenbergii* with WSSV. Again, all WSSV-positive animals from the aquaculture farms were positive only by nested PCR and no mortalities were associated with the infections.[70] Experimental infection of *M. rosenbergii* led to clinical signs of disease, including white spots on the carapace; however, these spots appeared to have a smaller diameter, and a different shape and colour to those of *P. monodon* infected with the same strain of WSSV.[70] Peng et al. (1998) noted that experimentally infected larvae and postlarvae appear to be more susceptible to WSSV infection than adults; this was also reported in a study in 2002 along with increased cannibalism in WSSV-infected groups.[71] Interestingly, adult *M. rosenbergii* experimentally infected with WSSV can clear the virus within a few days of infection[72]; infected individuals exhibit lethargy and anorexia within the first few days of infection but recover within 24 h.[72,73] This ability to clear the virus is also seen from a molecular perspective: VP28 WSSV enveloping protein mRNA can only be detected up to 4 days post-infection, whereas WSSV DNA can be detected 40 days post-

**TABLE 2** Bacterial diseases of *Macrobrachium rosenbergii*

| Genus | Species | Reported life stage(s) affected | Clinical signs of infection | Histopathological signs of infection |
|---|---|---|---|---|
| *Aeromonas* | *A. hydrophila*[89,118] | Juveniles (GO), adults (GO, W) | "Black spot disease"—dark necrotic lesions on exoskeleton and appendages, mortality.[89] | Lesions heavily covered in bacterial cells, with many embedded in an amorphous mucoid material on the lesion surface.[89] |
| | *A. veronii*[91] | Juveniles (L), adults (L) | Shell lesions, opaque white musculature, hepatopancreatic erosion, mortality.[91] | Bacteria present in hepatopancreas and muscle.[91] |
| | *A. caviae*[91] | Adults (GO, L) | Mortality.[91] | Bacteria present in hepatopancreas and muscle.[91] |
| | sp.[84] | Larvae (H) | "Black-spot" bacterial necrosis and gill obstruction.[84] | Not described. |
| *Bacillus* | sp.[89] | Juveniles (GO, L), adults (GO) | "Black spot disease"—dark necrotic lesions on exoskeleton, mortality.[115] | Lesions heavily covered in bacterial cells, with many embedded in an amorphous mucoid material on the lesion surface.[115] |
| *Citrobacter* | *C. freundii*[108] | Juveniles (GO, L) | "Water bubble disease (WDB)—Formation of a "water bubble" with a diameter of *c.* 7 mm under the carapace, loss of appetite, inactivity, weight loss.[108] | Not described. |
| *Enterobacter* | *E. cloacae*[104,107] | Larvae (H, L), postlarvae (H), juveniles (GO, L) | Weakness, poor appetite, mortality in larvae.[104] Slow growth (iron prawn) in juveniles.[107] | Not described in larvae. In juveniles, vacuolation of columnar cells, partial dissolution of villi and loss of structure in the hepatopancreas; densely-packed muscle fibres.[107] |
| *Enterococcus* | *E. casseliflavus*[105] | Postlarvae (H, L) | Mortality.[105] | Not described. |
| *Exiguobacterium* | *E. profundum*[105] | Postlarvae (H, L) | Mortality.[105] | Not described. |
| *Klebsiella* | *K. pneumoniae*[105] | Postlarvae (H, L) | Mortality.[105] | Not described. |
| *Lactococcus* | *L. garvieae*[94]<br>*L. lactis*[95] | Juveniles (GO, L), adults (GO) | Anorexia, poor growth, inactivity, opaque, white musculature in cephalothorax and abdominal segments, mortality.[94,95] | Ovoid diplococci bacteria present in hepatopancreas and musculature. Necrosis of hepatopancreatic tubules with melanised, encapsulated granulomas in connective tissue of haemal sinuses. Necrotic musculature infiltrated with haemocytes.[94] |
| *Pseudomonas* | *P. aeruginosa*[96] | Juveniles (GO), adults (GO, L) | Opaque, white musculature in cephalothorax and abdominal segments, mortality.[96] | Musculature lesions containing bacteria, changes in myofibrillar arrangement, haemocyte aggregation in necrotic musculature, melanised haemocytic granulomas around haemal sinuses. Melanised hepatopancreatic tubules, haemocytic nodules and irregular luminal cavity of the hepatopancreas.[96] |
| *Spiroplasma* | *S. eriocheiris*[109] | Juveniles (GO), adults (GO, W) | Weakness, aggregation at the side of ponds, mortality.[109] | Basophilic to mixed basophilic/eosinophilic intracellular inclusions, most prominent in |

(Continues)

**TABLE 2** (Continued)

| Genus | Species | Reported life stage(s) affected | Clinical signs of infection | Histopathological signs of infection |
|---|---|---|---|---|
| | | | | the hepatopancreas, but also present in heart muscle, skeletal muscle and connective tissue.[110] Large numbers of *Spiroplasma* in inclusions in haemocytes and connective tissue.[109] |
| *Vibrio* | *V. alginolyticus*[102,106] | Larvae (H), postlarvae (L), adults (L) | Mortality in larvae and postlarvae.[102,106] Mortality, cloudy muscle appearance, loss of appendages and soft and dark-brown hepatopancreas in adults.[101] | Not described in larvae.<br>In adults, Haemocyte infiltration in hepatopancreas, muscle and gill tissue. Bacterial infiltration in the hepatopancreas, muscle and heart tissue. Loss of the epithelial layer, hyperplasia of epithelial cells and degeneration of the epithelium in the hepatopancreas. Hyperplasia in branchial arches of gills and deformed and necrotising lamellar. Nodular haemocytic reaction and mild melanisation in heart tissues.[101] |
| | *V. anguillarum*[97] | Juveniles (L) | Mortality.[97] | Not described. |
| | *V. campbelli*[103] | Larvae (H) | Mortality.[103] | Not described. |
| | *V. carchariae*[106] | Larvae (L), postlarvae (L) | Mortality.[106] | Not described. |
| | *V. cholerae*[100,106] | Larvae (L), postlarvae (L), juveniles (GO, L), adults (GO) | Mortality in larvae and postlarvae.[106] Red discolouration, anorexia, swimming alone in juveniles/adults.[100] | In adults, rupture of basal lamina of hepatopancreatic tubules, with severe necrosis and dilation of tubules, loss of structure, atrophy and vacuolisation. Disorganisation of intestinal villus and epithelial cells with severe necrosis and separation from the basal membrane of epithelial cells.[100] |
| | *V. harveyi*[119,120] | Larvae (H), juveniles (L) | "Luminescent larvae syndrome"—glowing appearance of dead and moribund larvae.[119]<br>In juveniles—swelling and deformation of the hepatopancreas with appearance of white spots. Mortality.[120] | Not described in larvae.<br>Chromatin condensation in hepatopancreatic cells of juveniles.[120] |
| | *V. mimicus* | Larvae (L), postlarvae (L) | Mortality.[106] | Not described. |
| | *V. neocaledonicus*[85] | Larvae (H) | Mortality.[85] | Not described. |
| | *V. parahaemolyticus* (non-AHPND strains)[99,102] | All (H, GO) | Red discolouration with black spots on carapace, loss of appendages and telson, brittle shells and mortality in juveniles/adults.[99] Mortality in larvae.[102] | In adults, infiltration of acinar cells into musculature. Deranged muscle bundles. Moderate necrosis in gill lamellae, with hyperplasia and severe haemolytic infiltration of branchial arches. Dilation of hepatopancreatic tubules, vacuolisation of hepatocytes and necrosis of acinar cells. |

**TABLE 2** (Continued)

| Genus | Species | Reported life stage(s) affected | Clinical signs of infection | Histopathological signs of infection |
|---|---|---|---|---|
| | | | | Infiltration of cells in intertubular spaces.[99]<br>Histopathology of larvae not described.[102] |
| | *V. parahaemolyticus*[a] (AHPND strains)[114,116] | Larvae (L), adults (L) | Mortality in larvae and adults.[114,116] | Not described in larvae. In adults, haematopoietic tissue showed karyorrhexis, pyknotic nuclei and accumulation of cells with eosinophilic structures.[116] |
| | *V. vulnificus*[98,121] | Larvae (H), postlarvae (L), juveniles (GO) | Dark brown focal lesions and necrosis on appendages of juveniles/adults.[98]<br>Infected larvae are weak, have poor appetite and slowed growth prior to mortality.[121] | In adults, necrosis of hepatopancreatic tubules and presence of bacterial haemocytic nodules with melanised and marked haemolytic enteritis. Accumulation of haemocytes in the haemocoelic space of gills and diffuse necrosis of gill lamellae. Erosion through the epicuticle of the exoskeleton extending to into the exocuticle.[98]<br>Histopathology of larvae not described. |

*Note*: Brackets in the 'reported life stage(s) affected' column indicate which settings infections were reported: H = Hatchery, N = Nursery, GO = Grow-out pond, W = Wild, L = Laboratory setting.

[a]Pathogens listed by the World Organisation for Animal Health (WOAH).

infection, suggesting that the virus can persist in *M. rosenbergii*, but does not replicate at high levels.[72] WSSV has been shown to have reduced pathogenicity in other crustacean species, including the shore crab, *Carcinus maenas*.[74]

*Decapod iridescent virus 1* (DIV1) (Family: *Iridoviridae*) is a large dsDNA virus.[75] Members of *Iridoviridae* have been shown to infect amphibia, fish and invertebrates.[76] DIV1 is an icosahedral enveloped virus with a diameter of approximately 160 nm,[75] but dependent on infected tissue type, DIV1 can be enveloped or non-enveloped, determined by whether they were budded from the cell membrane or released by cell lysis.[75,76] To date, DIV1 comprises two strains: shrimp iridescent virus (SHIV) 20141215 and Cherax quadricarinatus iridovirus (CQIV) CN01,[77] with the former first identified in *P. vannamei*, and the latter in the red claw crayfish *C. quadricarinatus*.[75,78] However, both strains have been shown to be able to infect *P. vannamei*.[78] Natural infections of *M. rosenbergii* with DIV1 have been reported in China, with cumulative mortalities of ≥80%. Affected prawns migrate to deep water and exhibit a white triangle under the carapace at the bottom of the rostrum.[75,79] Mass mortalities due to infection with DIV1 have been reported from a pond in China where *M. rosenbergii* were co-cultured with *Procambarus clarkii*, a freshwater crayfish, on a farm that had seen high levels of mortalities from DIV1 in an adjacent *P. vannamei* pond a month prior to mortalities in *M. rosenbergii*.[79] qPCR analysis of viral load showed high copy numbers of DIV1 in *M. rosenbergii* and *P. clarkii*, as well as in dried dead *P. vannamei* from the side of the pond that had experienced mortalities. Wild crustaceans also in the *M. rosenbergii* pond, including *Macrobrachium superbum*, *M. nipponense* and a *Cladocera* sp., also had detectable levels of DIV1, with *M. nipponense* having comparable viral loads to that of *M. rosenbergii* and *P. vannamei*. DIV1 has also been detected in *F. chinensis* and *M. japonicus*, suggesting a wide host range.[80] DIV1 is a disease listed by WOAH.[81]

## 3 | BACTERIA

Since the start of *M. rosenbergii* culture, opportunistic bacterial infections have been associated with mortalities and clinical signs of disease in all prawn life stages. *Vibrio*, *Aeromonas* and *Pseudomonas* spp. have been associated with mortalities in the hatchery stage of culture through to adults in grow-out ponds.[5,84,85] A summary of bacteria known to infect *M. rosenbergii* is provided in Table 2.

In juvenile and adult prawns, bacterial infections commonly manifest as 'black spot' lesions on the animal's exoskeleton; melanised, necrotic lesions, primarily on the appendages.[86] As in other crustaceans, the causative agents *Vibrio*, *Aeromonas*, *Pseudomonas* and *Bacillus* have been associated with mortalities in all life stages of *M. rosenbergii*.[87,88] Even when animals can be harvested before mortalities occur, market value is still reduced due to the substandard appearance of the animals.[86] *Aeromonas hydrophila* has been associated with black spot disease in *M. rosenbergii*.[89] Experimental

infections with *A. hydrophila*, and other species, including *A. veronii* and *A. caviae*, have been shown to cause disease and mortality.[90–93]

Several other bacterial species have been associated with opaque and white musculature in the cephalothorax and abdominal segments of adult and juvenile *M. rosenbergii*. Two species of *Lactococcus*: *L. garvieae* and *L. lactis*, as well as *Pseudomonas aeruginosa* have been associated with these clinical signs during experimental infection.[94–96] *L. garvieae* and *L. lactis* were first reported a decade apart in association with mortalities in ponds in Taiwan, with 30%–40% and 25%–60% mortality occurring in farms infected with each species, respectively.[94,95]

Infection with *Vibrio* spp. was identified as a risk to *M. rosenbergii* farming when its culture was in its infancy. Huang et al. (1981) experimentally infected *M. rosenbergii* with a strain of *V. anguillarum* and observed mortalities of up to 100% with a high dose.[97] Since then, numerous *Vibrio* species infections have been reported including *V. vulnificus* causing dark brown local lesions and necrosis of appendages, *V. parahaemolyticus* and *V. cholerae*, which both cause red discolouration of infected prawns,[98–100] and *V. alginolyticus* causing mortalities in adult prawns.[101]

Larvae appear to be particularly susceptible to *Vibrio* spp. in hatchery settings. Ma et al. (2020) showed that four species of *Vibrio*, with multiple strains of each species, were prevalent in moribund hatchery-cultured zoea-stage larvae.[85] *V. parahaemolyticus*, *V. neocaledonicus*, *V. vulnificus* and *V. alginolyticus* could cause larval mortalities in a dose-dependent manner individually. However, mortalities in hatcheries were likely to be caused by co-infections of multiple *Vibrio* species and other opportunistic bacteria.[85] Previous studies have also identified other species of *Vibrio* that cause larval mortalities including *V. harveyi* and *V. campbellii*.[102,103] Other than *Vibrio* spp., the same study identified *Enterobacter* and *Bacillus* associated with moribund prawns. *E. cloacae* was previously identified as the causative agent of mortalities at the zoea stage of larval development in China, where mortalities exceeded 50% in hatcheries with affected larvae exhibiting reduced growth and rapid death when captured by netting.[104] Despite multiple studies investigating the role of bacteria in larval disease and mortality, very few studies have investigated the bacterial communities involved in postlarval mortality.[105,106]

Recently, *Enterobacter cloacae* has been associated with slow growth syndrome, also known as iron prawn syndrome (introduced in Section 2 - Viruses). Reports of *E. cloacae* in aquatic animals are rare; however, *E. cloacae* was detected in 100% of slow growth prawns collected from farms in China between 2017 and 2019. Challenging prawns experimentally with an isolate of *E. cloacae* reproduced the slower-growth phenotype, with slow-growth prawns significantly smaller in size than non-challenged prawns.[107]

A study carried out in 2000 challenged juvenile *M. rosenbergii* with *Citrobacter freundii* but found no clinical signs of disease or mortalities associated with infections.[91] Despite this, a 2022 study found *C. freundii* to be associated with juvenile mortalities of up to 30% in grow-out ponds in China, with laboratory challenges with *C. freundii* replicating clinical signs of disease seen in culture settings.[108] Typically, prawns infected with *C. freundii* develop a 7mm diameter bubble under the carapace, show loss of appetite, weight loss and inactivity. Clinical signs of disease are quickly followed by mortality and spread of the disease to other prawns. The discrepancies in the results of these two studies could be explained by different strains of the same species of bacteria showing differing pathogenicity to the host, or differences in host genetics of the challenged prawns resulting in resistance to infection.

Many bacterial infections in *M. rosenbergii* appear to be opportunistic and caused by bacteria that are ubiquitous in the aquatic environment and/or known as (opportunistic) pathogens of other aquatic animals. However, other bacteria appear to be more specifically host-associated. *Spiroplasma eriocheiris*, described from an infection of the Chinese mitten crab *Eriocheir sinensis*, has been identified as a pathogen of *M. rosenbergii* across Asia.[109,110] *S. eriocheiris*, a mollicute with a distinctive helical morphology, is the causative agent of tremor disease in *E. sinensis*, which manifests as paroxysmal tremors prior to mortality.[111] A *Spiroplasma* infection was first identified in *M. rosenbergii* in 2010 in China.[109] This strain of *Spiroplasma*, provisionally named MR-1008, caused disease and mortalities in juveniles and adults in infected ponds. Experimental infections with the MR-1008 strain of *Spiroplasma* resulted in >80% mortality.[112,113] A publication in 2013 investigating the proteomic response in *M. rosenbergii* to *Spiroplasma* infection confirmed that strain MR-1008 was *S. eriocheiris*.[113] There were no reports of *S. eriocheiris* infection outside of China until 2015, when naturally infected *M. rosenbergii* were reported from a pond in Thailand with unusually high mortality.[110] Infections of *S. eriocheiris* have also been identified in wild river populations of *M. rosenbergii* in Bangladesh in 2018 and 2019 by molecular and histopathological techniques (Our own data, not published).

Acute hepatopancreatic necrosis disease (AHPND) causes morbidity and mortality in post-larval penaeid shrimp, but it was not known until recently whether freshwater prawn species could be affected. AHPND, listed by WOAH in 2017, is caused by a strain of *Vibrio parahaemolyticus* made virulent by the acquisition of plasmids encoding the $PirA^{Vp}/PirB^{Vp}$ toxin ($VP_{AHPND}$).[114] Three different studies have addressed the effect of infection with AHPND on *M. rosenbergii*, the first infecting larvae, the second infecting postlarvae and the third infecting adults.[114–116] Larvae were shown to have dose-dependent mortality in a disease challenge setting, with high doses inducing 100% mortality after 36 h.[117] Postlarvae appear to be resistant to infection with $VP_{AHPND}$; however, they may have the potential to be a carrier to susceptible species that are commonly cultured together or in close proximity,[115] and as in the *C. freundii* example above, may be due to host genetics of the challenged prawns conferring resistance to infection. Despite postlarvae appearing to be tolerant to $VP_{AHPND}$, Pudgerd et al. (2021) demonstrated that adult *M. rosenbergii* could be infected with $VP_{AHPND}$ by intramuscular injection, and showed dose-dependent mortality.[116]

## 4 | FUNGI

Fungi were some of the first pathogenic agents identified in *M. rosenbergii* culture, discovered prior to the commercial expansion of the industry. Fungal agents have tended to cause lower mortality levels than infections with viruses and bacteria, but have resulted in the

**TABLE 3** Diseases of *Macrobrachium rosenbergii* caused by fungi and microsporidia

| Species | Reported life stage(s) affected | Clinical signs of infection | Histopathological signs of infection |
|---|---|---|---|
| *Batrachochytrium dendrobatidis*[123] | Juveniles, adults | Greyish-white body colouration, spongy appearance, lethargy, anorexia, abnormal swimming, fading of eye colour, dark spots and melanisation on cephalothorax and muscles, greyish-white filaments on claws and mortality.[123] | Cuticular erosion and atrophy with fungal cells penetrating the cuticular epithelium and intramuscular regions. Gill lamellae infiltrated by proliferating yeast cells causing distension and gross enlargement.[123] |
| *Candida sake*<br>*Candida mogii*[126,127] | Juveniles, adults | Yellowish-brown discolouration, white opaque eyes, swelling between cephalothorax and abdomen, cloudy white haemolymph, light yellow hepatopancreas, slow swimming, lethargic, anorexia, mortality.[126] | Vacuolisation of hepatopancreatic ducts and tubules with yeast aggregates in intertubular connective tissue. Large numbers of yeast cells present in haemolymph.[126] |
| *Debaryomyces hansenii*[125] | Juveniles, adults | Yellowish-brown discolouration, swollen hepatopancreas, cloudy haemolymph, whitish opaque musculature, mortality.[125] | Vacuolisation of hepatopancreatic epithelial cells, infiltration of yeast cells into the hepatopancreatic sinus. Necrosis of whiteish muscle tissue. Large numbers of yeast cells present in haemolymph.[125] |
| *Fusarium* sp.[122] | Juveniles, adults | Dark lesions on exoskeleton following injury, mortality[122] | *Fusarium* cells present in abdominal pleura, carapace, swimmerets, uropods and walking feet and appendages. Cuticular erosion, necrosis and melanisation of musculature underlying dark lesions on the exoskeleton[122] |
| *Metschnikowia bicuspidata*[128] | Juveniles, adults | Poor growth, anorexia, yellowish-brown discolouration of body, swelling between cephalothorax and abdomen, swollen hepatopancreas, milky haemolymph, whitish, opaque musculature with whitish-yellow spots, mortality.[128] | Accumulation of oedematous fluid between cuticle and muscle. Numerous yeast cells infiltrating the cuticle. Fragmentation of cardiac fibres and oedema in haemocyte nodules in the heart. Hepatopancreatic tubule epithelial cells vacuolised and sinuses contain thin membrane encapsulating yeast cells with large necrotic foci observed. Abdominal, pereiopod and pleopod muscle tissue has liquefactive necrosis and oedema with a large number of yeast cells. Necrosis of gill tissues with dilation and infarction of capillaries.[128] |
| Microsporidia sp.[136] | Juveniles, adults | Not described | Not described |
| *Enterocytozoon hepatopenaei* (EHP)[135] | Postlarvae | Not described—PCR positive only. | Not described |

discolouration of prawns, decreasing their market value.[122–125] Typically, yeast infections in *M. rosenbergii* have been shown to occur in the winter months when water temperatures are cooler.[122] A summary of fungi known to infect *M. rosenbergii* is provided in Table 3.

A *Fusarium* sp. was identified in 1977 as the causal agent of "black spot disease" in juvenile and adult *M. rosenbergii*.[122] Similar to infection with opportunistic bacteria, isolated black (melanised) lesions were apparent on the carapace and appendages of prawns, typically when the carapace had been damaged, leaving an open wound vulnerable to infection.[122] *Fusarium* spp. have also been associated with 'black gill' in penaeid shrimp and can cause mortality over a long period of time; however, there is no evidence that this fungus causes similar clinical signs in *M. rosenbergii*.[122]

In 1998, several yeast taxa were identified as causal agents of mortality in adult and juvenile *M. rosenbergii* in Taiwan.[126] Clinical signs of the disease included behavioural changes such as slow swimming, lethargy and anorexia accompanied by a yellow-brown discolouration, white opaque eyes and swelling between the cephalothorax and the abdomen. Upon dissection, infected prawns were seen to have cloudy-white haemolymph, a light yellow hepatopancreas and white opaque muscle tissue; under light microscopy, masses of Gram-positive yeast cells were observed.[126] *Candida sake* was the most abundant yeast species in moribund prawns and is a possible contribution to the mortalities seen in Taiwan, as a previous study showed that *C. sake* could cause 100% mortality in experimental infections of this host.[124,126] Another species of

*Candida*, *C. mogii*, has also been associated with infection of *M. rosenbergii* in China.[127]

In 2001, juvenile *M. rosenbergii* experienced mortality events associated with similar clinical signs to those of infection with *C. sake*. *Metschnikowia bicuspidata* was isolated from diseased prawns co-infected with *Enterococcus faecium*.[128] Experimental challenges of *M. rosenbergii* with *M. bicuspidata* alone and *M. bicuspidata* with *E. faecium* resulted in similar clinical signs, with the co-infection challenge only slightly increasing mortality.[125] A separate study in 2007 showed that a high prevalence of *M. biscupidata* was associated with juvenile prawn mortalities of up to 95% in some ponds.[129] Other yeast species have also been associated with mortalities, including *Saccharomyces cerevisiae* and *Candida albicans*, both of which were shown to be pathogenic by experimental infection.[129] *M. rosenbergii* infected with *Debaryomyces hansenii* also shows similar clinical signs to infection with *M. bicuspidata*.[125]

The chytrid *Batrachochytrium dendrobatidis* was identified as the causative agent of discolouration and prawn mortality in *M. rosenbergii* farms in southern India between 2007 and 2011.[123] Affected prawns were greyish white, had a 'woolly' appearance with greyish filaments on their claws, and had problems with feeding, moulting, movement and respiration—culminating in mortality, reaching up to 90%.

## 5 | MICROSPORIDIA

In penaeid shrimp culture, the microsporidian parasite, *Enterocytozoon hepatopenaei* (EHP) of the Enterocytozoon Group Microsporidia (EGM) causes slow-growth in *P. monodon* and *P. vannamei*.[130,131] EHP is associated with a wider range of syndromic conditions, for example, white faeces syndrome[132] and is now a major threat to the commercial shrimp farming industry, listed as an emerging disease by WOAH[133] First identified in *P. monodon* in ponds in Thailand, at the time, it was not considered to be causing significant problems in shrimp culture,[131] but since, although not linked to mortalities, it has spread globally, causing severe growth retardation and resulting in enormous losses for shrimp farmers.[134] A study in 2018 showed that *M. rosenbergii* cultured in the same pond as *P. vannamei* were positive by PCR for EHP; however, the histology was unable to demonstrate infection of *M. rosenbergii*.[135] Despite the inconclusiveness of this study, molecular detection of EHP from *M. rosenbergii* is a significant finding and should be explored in more depth to determine whether freshwater prawn species are susceptible to infection with EHP (or other EGM) to the same extent as penaeid shrimp.

Microsporidia infections have been observed in *M. rosenbergii*,[136] but have not been taxonomically described further than to the genus level.[137] A microsporidian parasite has been identified in *M. nipponense* as the novel species *Potaspora macrobrachium*, causing progressive whitening of musculature and reduced survivability during holding and transportation of the animals.[138] The description of this novel disease-causing microsporidian is an important discovery in freshwater prawn culture and should be considered as a potential threat to the culture of *M. rosenbergii*.

## 6 | OTHER EUKARYOTIC PATHOGENS

Eukaryotic pathogens other than fungi and microsporidia are not commonly reported in *M. rosenbergii* culture, likely due to the lack of, or low, mortalities associated with infection. The presence of epibionts, principally ciliates, are common in *M. rosenbergii* ponds predominantly associated with larger animals that moult less frequently, allowing the accumulation of symbionts on the exoskeleton.[8] The presence of these epibionts can cause problems with feeding and mobility when attached to appendages, and in cases with severe fouling of the gills, mortality can occur from insufficient gaseous exchange.[8]

Two publications have investigated the presence of eukaryotic parasites in wild populations and showed that *M. rosenbergii* could be infested by a range of epi- and endo-parasitic infections.[139,140] Both publications describe the presence of ciliates, with *Zoothamnium* spp. present at high prevalence on appendages and gills as well as *Acineta* spp., *Epistylis* spp. and *Vorticella* spp. The latter two were found both externally and within the gut. Jayasree et al. (2001) reported that apostome ciliate cysts were commonly seen attached to gill lamellae, with heavy infections frequently observed, causing a melanisation response at points of attachment.[140] Gut-dwelling gregarines were also present in both *M. rosenbergii* populations.[139,140] Other eukaryote parasites reported include bopyrid isopods and the larval digeneans *Opecoelid metacercariae* attached to pleopods, pereiopods and antennules and microphallid metacercariae in the musculature. Berried females may be a source of introduction of epibionts into hatchery culture systems, where they cause greater issues with feeding and swimming impairment due to the small size of *M. rosenbergii* larvae and postlarvae.[8]

Despite the infrequent reports of mortality caused by eukaryotic pathogens in *M. rosenbergii*, one ciliate, *Metanophrys sinensis*, has been associated with mass mortalities of larval stage *M. rosenbergii* in India; prawns exhibiting lethargy, erratic movement and discolouration prior to mortality. Under microscopic examination, large numbers of ciliates were seen in the coelom.[141]

Overwise, microeukaryote pathogens associated with *Macrobrachium* have received little attention. As awareness of the importance of host-associated eukaryotes (the 'eukaryome') increases, particularly with respect to host health, investigating this aspect of *Macrobrachium* health, particularly using the new approaches available to profile unknown pathogens,[142] will likely provide valuable information about symbiotic relationships contributing to health and disease in this host and those cultured with it.

## 7 | EMERGING AND SYNDROMIC DISEASES

The last review of diseases in *M. rosenbergii*[24] identified several diseases of uncertain aetiology. Many of these apparently syndromic conditions remain unresolved currently.

Many idiopathic diseases affect the early life stages of *M. rosenbergii*. Mid-cycle disease (MCD) affects larval stages of *M. rosenbergii*, typically between days 15 and 22 of production; larvae display clinical

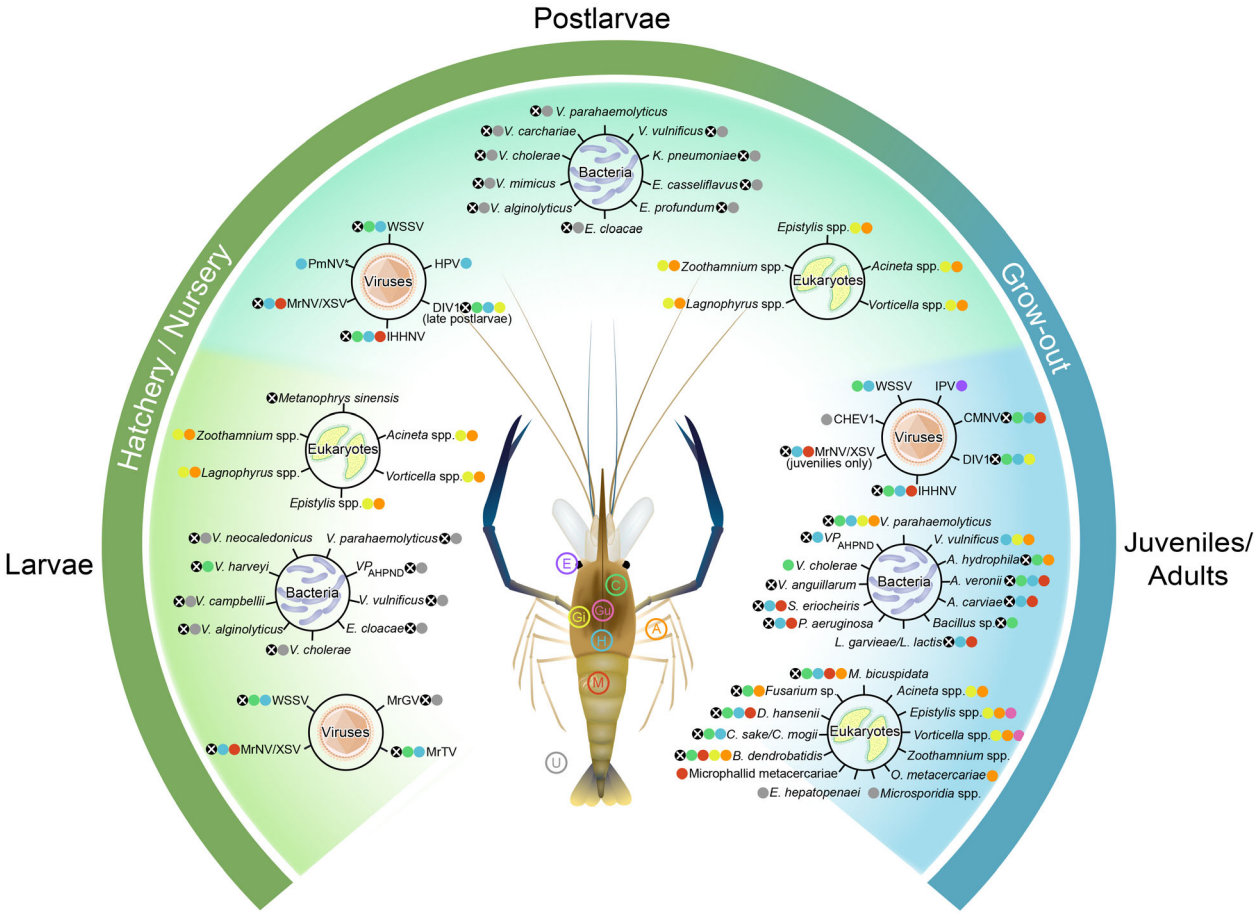



**FIGURE 2** Diseases of *Macrobrachium rosenbergii* and the life stage(s) affected. Black circle with cross denotes that this pathogen has been shown to cause mortality. Coloured circles denote the organ(s) affected: Green = cuticle, pink = gut, yellow = gill, orange = appendages, blue = hepatopancreas, red = muscle, purple = eye/eyestalk and grey = unknown

signs of lethargy, erratic swimming activity, reduced feeding and growth and a bluish discolouration of the body.[143] Moult death syndrome (MDS), also known as exuviae entrapment disease (EED), is a phenomenon that most commonly occurs during the metamorphic moult of larvae to postlarvae, where larval mortality occurs during unsuccessful moults.[86] Dietary supplements have been shown to reduce the occurrence of MDS.[144]

Branchiostegal blister disease (BBD), named by farmers as balloon disease (BD), since the disease is characterised by swelling of the gill flap or branchiostegal region and deformities of the appendages.[145] The disease occurs in grow-out ponds, typically within 30 days of stocking postlarvae from nurseries, with mortalities ranging from 70% to 80%. As the disease spreads rapidly between adjacent ponds, a viral aetiology was proposed; however, traditional methods of virus isolation did not detect the presence of any viruses.[145]

Appendage deformity syndrome (ADS) was reported to cause late mortalities in grow-out ponds in the Nellore district of India in 2004.[146] The disease typically affected female prawns more than males, and prevalence in affected ponds was ~50%. Affected prawns had appendage deformities, broken or bent rostrums, cut antennae and wrinkled carapaces. Cohabitation challenges were unable to cause ADS in unaffected prawns, suggesting that disease cannot be transferred from prawn to prawn. Interestingly, supplementation of feed with carotenoids was able to reverse the diseased phenotype, indicating that ADS is not of pathogenic aetiology.[146]

Growth retardation of prawns with the idiopathic condition known as iron prawn syndrome (IPS) affects the body weight and length of prawns in culture. Attempts to determine a cause of this abnormal phenotype have been significant compared to other idiopathic diseases, with studies investigating culture practices, pathogen-related disease candidates and host genetics. Studies assessing diet,[45] antibiotic use[147] and water quality[44] have not identified the leading cause of the condition.

Some studies investigated growth based on single-nucleotide polymorphisms (SNPs) and host transcriptomics.[148,149] However, these mainly focused on size differences present in populations due to social dominance hierarchy, not prawns affected by IPS. Jiang et al. (2020) investigated the genetic and epigenetic differences between prawns with a normal growth phenotype and IPS prawns and noted differences mainly in the germplasm of the two phenotypes.[43] Interestingly, the same study reported enrichment for host transcripts related to immune response in infected animals, suggesting that a host response may be being initiated due to pathogen infection. Since then, a study comparing the differences in proteomics between normal and IPS prawns also reported significant variations in the

expression of proteins involved in immune system functions.[150] Further evidence to suggest pathogen involvement was the identification of *E. cloacae* infections in growth-retarded prawns[107] (Section 3 - Bacteria), the presence of a novel hepe-like virus,[48] and the presence of a novel flavivirus[49] (see Section 2 - Viruses). Screening for agents associated with IPS is missing from the three recent investigations, for example, the study that identifies IPV did not screen for Enterobacter *cloacae* or CHEV1, therefore it is unclear whether these pathogens can cause IPS phenotype alone, or infections of multiple pathogens are needed.

## 8 | DISCUSSION

Since a review of diseases of *M. rosenbergii* in 2012,[24] the number of its characterised pathogens (Figure 2) has increased. Many of these pathogens are viruses that have either emerged directly within *M. rosenbergii* culture, or are known pathogens from other aquaculture sectors that have also been shown to infect *M. rosenbergii*. Identifying pathogens throughout aquaculture has become easier with the decreasing cost of next-generation sequencing techniques that have allowed culture-free identification of pathogens—particularly useful where the agent is novel and favourable culture environments are not known or possible to reproduce.

Shrimp aquaculture (*P. monodon* and *P. vannamei*) has seen more large-scale disease outbreaks than other groups of cultured aquatic animal species.[151] These disease outbreaks, causing total losses exceeding 40% of global capacity,[152] have a devastating economic and socio-economic impact on shrimp farms.[153] In *M. rosenbergii* culture, disease incidence, in particular diseases caused by viral infections, has increased with the expansion of the industry, but with no clear cause. A review of the vulnerabilities in aquatic animal production identified a number of factors contributing to increase in disease incidence, one being climate change.[154] Short-term climate events such as increased incidences of storms, floods and droughts can cause instabilities in pond systems, resulting in poor water quality and a chance for opportunistic pathogens to cause disease in stressed animals. These events are likely to become more frequent and extreme as the climate crisis continues.[155] Both climate change and intensification of culture has been shown to drive increased pathogen virulence, host susceptibility and incidence of disease.[156,157] Inherent production and management risks also contribute to the rate of disease incidence, with the increase in production resulting in wild stocks being transported into aquaculture settings with few biosecurity measures to manage potential pathogens they may bring into the system.[154]

Opportunistic bacterial infections have the potential to cause significant losses in all stages of *M. rosenbergii* culture. To limit the losses due to this type of infection, use of antibiotics is common practice in culture settings, with recommendations on the type of antibiotic suitable for treating certain disease phenotypes outlined in the FAO culture handbook.[5] Despite cautions surrounding the misuse of antibiotics in the handbook, these compounds have been reported to be used as a preventative measure against bacterial infections in both shrimp and other aquaculture species.[158,159] Commonly used antibiotics in *M. rosenbergii* culture include oxolinic acid, chloramphenicol, erythromycin and oxytetracycline.[8] Several studies have identified many bacteria, isolated from both prawns[160,161] and the culture environment they are grown in,[161] to be resistant to a number of antimicrobial agents, including those commonly used to treat opportunistic bacterial infections in culture settings. Antimicrobial resistance in global shrimp aquaculture is reviewed in Thornber et al. (2020), and suggests risk mitigations surrounding antibiotic use, including improved disease diagnosis to ensure that antibiotics are only used to treat bacterial infections, and alternatives to antimicrobials such as phage therapy.[162]

Polyculture of *M. rosenbergii* with other crustaceans and fish is popular throughout Asia; however, the wide host range of many pathogens raises important questions, for example, whether (1) culturing multiple aquaculture species together in ponds accelerates the ability of pathogens to infect new hosts and (2) co-occurring biota (e.g. bioaccumulating filter-feeders) act as reservoirs or vectors of pathogens of cultured species. A study in 2011 reported that feeding of live molluscs to *P. monodon* increased the risk of WSSV infection, suggesting that molluscs were able to concentrate the virus by filter-feeding, delivering an infectious dose to shrimp when the molluscs were ingested.[163]

In China, *M. rosenbergii* polyculture with *E. sinensis* poses a significant risk of infection with *S. eriocheiris*, shown to infect both *E. sinensis* (causing tremor disease) and *M. rosenbergii*, with infections in both species leading to mass mortalities.[109,111] Other species of crustaceans have been shown to be susceptible to infection with *Spiroplasma* spp., including *P. vannamei*[164] and *P. clarkii*,[165] with infections associated with mortalities. Given that *M. rosenbergii* is grown in polyculture systems with *E. sinensis*, *P. vannamei* and *P. clarkii*,[13,21,79] infection with *S. eriocheiris* or another *Spiroplasma* species is a significant risk.

A review in 2017 listed known viruses of crustaceans, with a large number infecting crab, including *E. sinensis*.[166] Little is known about whether *M. rosenbergii* is also a susceptible species to these pathogens but given the broad host range of some crustacean pathogens, these risks should be considered when choosing this culture method. Interestingly, AHPND, caused by a halophilic bacterial pathogen, does not appear to pose a significant concern to *M. rosenbergii* under polyculture with *P. vannamei* at low salinities, with the prawn postlarvae and juveniles shown to exhibit resistance to $VP_{AHPND}$, unlike *P. vannamei*, which is more susceptible to AHPND at higher salinities (e.g. 10, 15 and 20 ppt) than at 5 ppt.[115]

In many countries, *M. rosenbergii* is cultured in the same or adjacent ponds as penaeid shrimp, such as *P. monodon* and *P. vannamei*.[13,20,79] Some hatcheries also produce freshwater prawns and penaeid shrimp within close proximity, increasing the likelihood of cross-contamination of species and the pathogens they are potentially carrying.[167] An obvious concern relates to viruses with a wide host range such as WSSV, CMNV, IHHNV and DIV1 that are known to infect both marine and freshwater species. However, a greater

concern is infection of supposedly non-susceptible species by these and other pathogens—white tail disease caused by MrNV in Indian hatchery-cultured penaeid shrimp, *P. monodon* and *Penaeus (Fenneropenaeus) indicus*, is a good case study that highlights this issue.[167] Seed of the *P. monodon* and *P. indicus* was produced in very close proximity to the seed of *M. rosenbergii*; the postlarvae of the two penaeid shrimp species exhibited whitish abdominal muscle and lethargy, with mortality reaching 100% after the appearance of clinical signs.[167] Since this disease incident, WTD has been reported in *P. vannamei* in China and Vietnam,[168,169] suggesting that this is not an isolated case and WTD is a threat to both freshwater and marine hosts. Experimental infection of juvenile penaeid shrimps with MrNV and XSV was unable to cause WTD, but juveniles and adults could act as virus reservoirs for larval and post-larval life stages.[170] The finding that older life-stages could act as a reservoir is important in the context of berried females being used as broodstock to produce larvae in hatchery settings—many countries prefer to use berried females from rivers, canals and lakes,[5] potentially unknowingly transporting pathogens into the culture system. For almost all pathogens of *M. rosenbergii*, it is unknown whether berried females and their eggs can act as a reservoir, therefore caution should be taken when transporting animals with an unknown disease status into hatcheries and other culture settings.

Given the major problems currently being experienced in penaeid shrimp culture caused by the microsporidian parasite EHP, it would be naïve to assume that *M. rosenbergii* are not at risk from microeukaryotic pathogens—at the time of its identification, EHP was considered an incidental hazard but has since emerged as a significant disease risk to the industry.[171] Despite microsporidia being ubiquitous within the environment and being able to infect vertebrates and invertebrates,[172] no microsporidian parasites have been formally described to infect *M. rosenbergii*, despite reports of microsporidiosis in the literature. Other eukaryotic pathogens, such as members of the Ascetosporea class of parasites, also cause problems in crustacean culture, such as the haplosporidian parasite that has been associated with high mortality and slow growth in *P. vannamei*.[173]

In addition to a gap in our understanding of eukaryotic pathogens of *M. rosenbergii*, there is also information missing from the literature surrounding the life-stages that pathogens affect, especially whether viruses that are lethal in postlarvae/juveniles can also infect larvae and adults. This is especially important at the stages in culture when animals are moved to new locations e.g. from hatchery to grow-out pond, where pathogens could be transferred from one life stage to another.

As the cost of sequencing continues to decrease, sequencing of all host and non-host genetic material in a sample by either metagenomics or metatranscriptomics is being applied ever more widely. As these technologies become more accessible, a greater understanding of pathogen lifecycles can be obtained by sequencing of hosts, intermediate and potential hosts of pathogens, as well as the surrounding environment. A holistic approach is beginning to be applied to pathogens causing the biggest problems in prawn aquaculture, such as MrNV and XSV. For example, natural insect carriers of viable MrNV and XSV particles have been identified in nursery ponds containing *M. rosenbergii* with clinical signs of WTD,[174] adding to the information currently available on transmission of the virus. A better understanding of pathogen transmission and lifecycle will enable decisions to be made, considering the risks of pathogen transfer between species and the environment, and the implementing of measures to mitigate disease.

Other molecular tools to investigate host genetics to improve *M. rosenbergii* culture are in their infancy—SNPs have been identified by mining transcriptomic data and have been utilised to search for markers of growth performance.[149] However, there is much greater potential for this type of tool to be used to investigate genetic markers of resistance to diseases. Breeding resistance to diseases based on genetic markers could potentially replace SPF stocks that are still susceptible to infection. The most notable use of this type of technology in aquaculture has been in the identification of a single quantitative trait locus (QTL) in Atlantic salmon (*Salmo salar*) that confers resistance to infection with infectious pancreatic necrosis virus (IPNV), allowing the production of resistant stocks using marker-assisted selection programs.[175] A recent study has also shown that susceptibility of *P. monodon* to infection with GAV is heritable,[176] suggesting that the identification of the genetic markers involved could potentially lead to selective breeding programs to produce animals that are more tolerant, or even resistant, to diseases that cause major problems in aquaculture.

Other than the utilisation of molecular tools and breeding for disease resistance, further techniques to mitigate the risk of disease in systems have been tested and applied to giant prawn culture. Such techniques include the addition of probiotics[177] and dietary supplements[178] to the aquaculture environment, as well as changing culture systems to Recirculating Aquaculture Systems (RAS) or biofloc systems.[179] These changes have seen some success, including observations that the addition of probiotics and dietary supplements can protect against disease.[177,178] As a result of these positive outcomes, a more holistic approach, combining multiple methods determined to be beneficial to giant prawn culture, is starting to be adopted.[180] Further adoption of a combination of methods to improve the health of *M. rosenbergii* is likely to reduce disease risk to prawns and animals they are cultured with.

## AUTHOR CONTRIBUTIONS

**Chantelle Hooper:** Conceptualization; formal analysis; writing – original draft; writing – review and editing. **Partho P. Debnath:** Writing – review and editing. **Grant D. Stentiford:** Writing – review and editing. **Kelly S. Bateman:** Writing – review and editing. **Krishna R. Salin:** Writing – review and editing. **David Bass:** Funding acquisition; supervision; writing – original draft; writing – review and editing.

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## DATA AVAILABILITY STATEMENT

Data sharing is not applicable to this article as no new data were created or analysed in this study.

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