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INVITED REVIEW

Mitigating human impacts including climate change on proliferative kidney disease in salmonids of running waters

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Abstract

Over the last two decades, an increasing number of reports have identified a decline in salmonid populations, possibly linked to infection with the parasite *Tetracapsuloides bryosalmonae* and the corresponding disease, that is, proliferative kidney disease (PKD). The life cycle of this myxozoan parasite includes sessile bryozoan species as invertebrate host, which facilitates the distribution of the parasite in running waters. As the disease outcome is temperature dependent, the impact of the disease on salmonid populations is increasing with global warming due to climate change. The goal of this review is to provide a detailed overview of measures to mitigate the effects of PKD on salmonid populations. It first summarizes the parasite life cycle, temperature-driven disease dynamics and new immunological and molecular research into disease resistance and, based on this, discusses management possibilities. Sophisticated management actions focusing on local adaptation of salmonid populations, restoration of the riverine ecosystem and keeping water temperatures cool are necessary to reduce the negative effects of PKD. Such actions include temporary stocking with PKD-resistant salmonids, as this may assist in conserving current populations that fail to reproduce.

KEYWORDS
conservation, global warming, malacosporea life cycle, management, salmonid disease, *Tetracapsuloides bryosalmonae*

1 | INTRODUCTION

There is growing consensus among aquatic wildlife managers and scientists that urgent action should be taken to safeguard freshwater species and their habitats from the effects of anthropocentric climate change (Dauwalter et al., 2020; Dudgeon, 2019; Reid et al., 2019; Tickner et al., 2020). Over the period of 1970 unti1 2020, water temperatures in central Europe have already increased with 0.3–0.4°C per decade (Daufresne & Boët, 2007; Kędra, 2020; Michel et al., 2020; Webb & Nobilis, 2007). Increased evaporation together with changes in patterns and seasonality of precipitation (Masson-Delmotte et al., 2019) will further intensify water use demands, which already conflict with managing the integrity of freshwater ecosystems for aquatic species (Dudgeon, 2019). This will strongly affect salmonid fish which are still the dominant fish species in many elevated and northern situated European and American river systems. Salmonid populations are critically dependent on pristine, cool and well-oxygenated riverine and lake environments (Jonsson & Jonsson, 2011), and therefore are strong indicators of good health or 'homeostasis' in these ecosystems (Costanza & Mageau, 1999). Additionally, salmonids provide important ecosystem services as, for example, they are highly appreciated by anglers and thus also

have recreational and economical value (Holmlund & Hammer, 1999; Worthington et al., 2020). Raising awareness about the threats to these valuable species (Burkhardt-Holm & Zehnder, 2018; IUCN, 2021) might provide the momentum to scientifically and politically tackle larger issues like climate change, which is necessary to protect these species and the fragile habitats they inhabit.

Salmonids are ectotherms that have specialized within a narrow range of temperatures (i.e. stenotherm) and therefore have limited capacity to adapt to variations in temperature (Angilletta et al., 2002; Crisp, 1996). It is first and foremost species with these characteristics that are sensitive to the current rapid changes that are taking place in their thermal environment due to anthropogenic climate change (Rahmstorf, 2008), and losses of more than 50% of their suitable environments might be expected if the world does not divert from a carbon-intensive pathway (Ros et al., 2021). Seasonal and daily changes in temperature, which are parameters that are strongly changing under global warming, affect salmonids via controlling metabolic processes that drive life history traits, like reproduction but that also drive important physiological traits like immunity (Fenkes et al., 2016; Slater & Schreck, 1998). As a consequence, the increase in global temperatures due to climate change have an augmenting effect on occurrence and severity of several infectious diseases (Jonsson & Jonsson, 2011). This effect was clearly shown in the example of proliferative kidney disease (PKD), which is caused by a life-threatening parasitic infection in salmonids (Okamura et al., 2011).

Proliferative kidney disease was first described in 1924 (Plehn, 1924). It took until the end of the 20th century to resolve the causes of the disease, when PKD was observed to inflict high mortalities in rainbow trout (*Oncorhynchus mykiss*) aquaculture in Europe. Research efforts showed that managing water temperatures was key to reduce the damage of the disease to the aquaculture industry (Clifton-Hadley et al., 1986; Feist, 2004; Ghittino et al., 2003). Changes in temperature affect the host immune responses towards the parasite and thereby modulate disease outcomes (Bailey et al., 2018), like susceptibility to develop PKD (Debes et al., 2017; Waldner et al., 2021), resistance to the parasite, meaning fish do not get infected and develop PKD (Foott & Hedrick, 1987), or tolerance to the disease, meaning fish do not develop PKD when they get infected with the parasite (Strepparava et al., 2018). Temperature also has a direct effect on the parasites themselves by influencing its proliferation and development (Okamura et al., 2011). Altered host response and parasite metabolism would then result in the highly increased virulence of the parasites at higher temperatures that might lead to increased mortality.

The unprecedented fast pace of environmental changes due to climate change may have put salmonid populations at the limits of their resilience and adaptability (Seebacher et al., 2015; Sunday et al., 2011), resulting in PKD (Figure 1a), becoming an important factor in current salmonid population declines (Arndt et al., 2019; Hutchins et al., 2019; Ros et al., 2021; Wahli et al., 2002; Waldner et al., 2020). This trend is expected to continue as in a recent study into the future effects of climate change on central European salmonids (Ros et al., 2021); it was predicted that especially those populations that are currently in areas where PKD is evident are vulnerable to further declines and that these declines are expected to be more severe under a high emission scenario (Figure 1b).

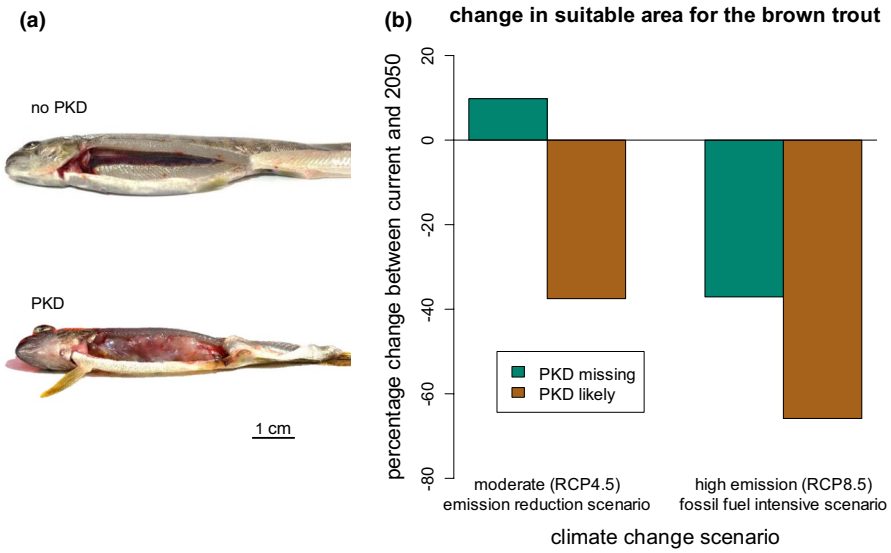

FIGURE 1 (a) Dissected brown trout: above without proliferative kidney disease (PKD) with healthy kidney and below with PKD resulting in severe kidney hyperplasia. (b) Brown trout populations that are currently living in areas that are likely for development of PKD show the highest risk to decline in 2050 in comparison to populations that inhabit areas where currently PKD is not likely to develop. In those latter areas that are restricted in size and mostly elevated, a small increase in suitable areas for salmonids might be expected under the moderate climate change scenario. The decline in suitable area is expected to occur under both moderate and high emission climate change scenarios, although the effect is more negative under the high emission climate change scenario. Percentage change in suitable areas was based on geographical projections taken from a study on salmonid populations in central Europe (Ros et al., 2021)

This review focusses on the impact of temperature on the salmonid disease PKD, on its emerging status due to climate change (Okamura et al., 2011) and on the possible management options to mitigate this disease. In order to understand these dynamics, an update on the biology and lifecycle of *Tetracapsuloides bryosalmonae* is given (see also: Sudhagar et al., 2019). The central hypothesis of this review is that due to the warming effect of climate change on water temperatures (Caissie, 2006; Sherwood et al., 2020; van Vliet et al., 2013), salmonid populations are increasingly under pressure of devastating effects of PKD (Waldner et al., 2020, 2021). The case is made that preserving a healthy river ecosystem with summer-cool water temperatures plays an important role in controlling the infectious disease (Oredalen et al., 2021; for lakes see: Sobocinski et al., 2018). Under ideal environmental conditions, that is, oxygen-rich mountainous water, close to the source and with summer water temperatures lower than 15°C, *T. bryosalmonae* infections do not result in PKD (Kristmundsson et al., 2010; Ros et al., 2021; Wahli et al., 2002). Apart from the intrinsic value of preserving aquatic ecosystems, such measures will benefit future generations both economically and recreationally. The review places emphasis on PKD and focusses on research on *T. bryosalmonae* in the context of current wild salmonid population losses and conservation. In addition, the impacts on salmonid aquaculture are briefly discussed. Throughout the review, the terms infect and its derivates are used but these are meant to include infest sensu Bush et al. (1997).

2 | THE PARASITE'S LIFE CYCLE AND PROLIFERATIVE KIDNEY DISEASE

Tetracapsuloides bryosalmonae is a member of the class Malacosporea of the subphylum Myxozoa. All myxozoans (i.e. Myxosporea and Malacosporea) are endoparasites with complex life cycles involving an invertebrate and a vertebrate host, which in the case of Malacosporea are a bryozoan and a teleost species (Holzer et al., 2018). Both morphologic and molecular evidence supports that myxozoans have evolved from a free-living member of the phylum Cnidaria (Bartošová et al., 2009; Fiala & Bartošová, 2010). Different from the Myxosporea, which are very species rich, in Malacosporea, only five species in two genera, that is, *Buddenbrockia* and *Tetracapsuloides*, have been named with limited morphological variation, while molecular methods indicate that many species have not been described yet (Fiala et al., 2015). All five described species are parasites of a broad range of Holarctic freshwater bryozoans (Anderson et al., 1999; Gay et al., 2001; Longshaw et al., 1999; Okamura & Wood, 2002), and they show specificity for both bryozoan and fish hosts (Henderson & Okamura, 2004). The greatest morphological difference between malacosporean species has been found in the proliferation phase in the bryozoan host. *Buddenbrockia* sp. and at least one *Tetracapsuloides* sp. produce a free-moving vermiform stage by which they can move between bryozoan zooids (Patra et al., 2017). Other *Tetracapsuloides* sp. including *T. bryosalmonae* are capable of amoeboid movement between zooids but proliferate in a sack-like form that may contain thousands of spores (Canning et al., 1999; McGurk et al., 2006). Within-species variation has been shown by phylogenetic analysis based on the polymorphic internal transcribed spacer 1 (ITS-1) region, with evidence for an American and a European clade of *T. bryosalmonae* (Henderson & Okamura, 2004). To understand the genetic variation within these clades in relation to occurrence of PKD, a wider part of the genome and a more diverse geographical dataset should be analysed.

The life cycle of *T. bryosalmonae* (Figure 2) is interwoven with the life histories of its bryozoan and salmonid host. The most abundant bryozoan hosts for *T. bryosalmonae* in Europe are *Fredericella sultana* and *Plumatella emarginata* (Hartikainen et al., 2014; Longshaw & Feist, 2000; Okamura & Wood, 2002; Ros et al., 2021). Apart from these species, *T. bryosalmonae* has also been detected in bryozoans like *Cristatella mucedo* and *Pectinatella magnifica* that can be found in cold water lakes and rivers in North America (Hartikainen et al., 2014). Taking these bryozoans as the starting point for the life cycle, parasitic cells have been found encapsulated (Hartikainen et al., 2013) in germinative stages (statoblasts) that bryozoans produce not only to spread but also to survive through the winter (Brown, 1933). These stages, which may contain the parasite, disperse downstream by flow, but may be brought upstream or even to other rivers in hanging water in feathers or hairs of passing birds or mammals (Okamura & Wood, 2002; Wood & Okamura, 2005). *Fredericella* sp. produce statoblasts, which are denser than water and remain in the neighbourhood of the original colony or within dying structures attached to the substrate. It is also possible that parasites within bryozoan colonies might survive through mild winter conditions.

In salmonid habitat, rising water temperatures in spring provide the conditions for bryozoan colonies to grow earlier, which, for example, is at 10–15°C for central European *F. sultana* and *P. emarginata* (Raddum & Johnsen, 1983; Vohmann et al., 2009). This also provides the conditions for the parasite to proliferate from covert to presaccular overt stages in the metacoel of the bryozoan zooids (Fontes et al., 2017; Tops et al., 2009). Microscopic studies have shown cell division and amoeboid movements of the parasite by which they may distribute throughout the bryozoan colony (Feist et al., 2015; Morris & Adams, 2006a). Further spread of parasites during this period might be due to the activity of insects and vertebrates, through which infected fragments of bryozoans get detached from the colony and these may start new colonies elsewhere (Morris & Adams, 2006b). When water temperature surpasses 8–10°C, sporogonic cells mature in a spore sack containing the malacospores (Figure 3j) (Gay et al., 2001; Tops et al., 2006). In this so-called overt stage of the cycle, spore sacks can be seen free floating in the metacoel, and the growth of colonies of bryozoans may show phenotypical anomalies (Hartikainen et al., 2013).

Horizontal spread, that is, spread within one host colony, has been shown only between bryozoans through the presaccular amoeboid stages and through fragmentation of infected bryozoans (Morris & Adams, 2006a; Morris & Adams, 2006b). Such presaccular infections in bryozoans have been found commonly at lower temperatures in

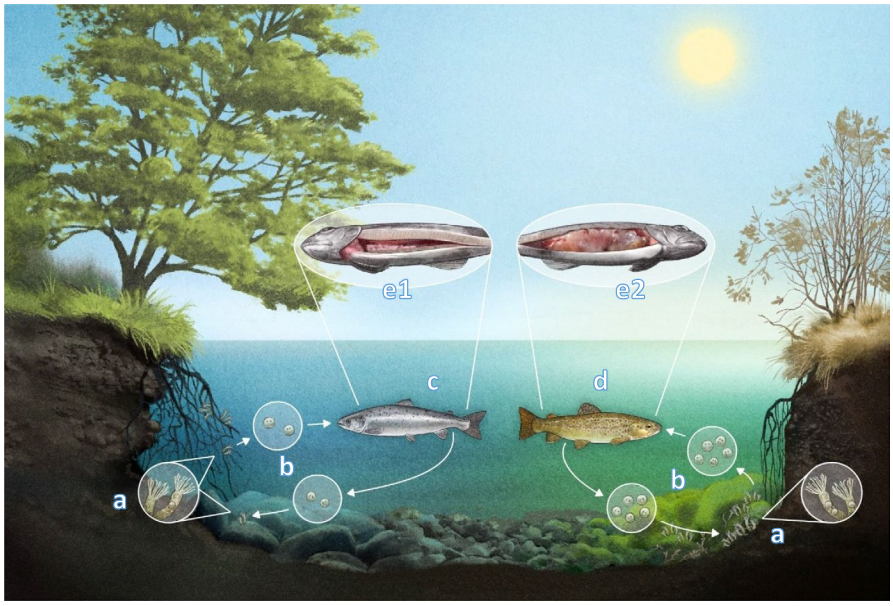

FIGURE 2 Life cycle of *Tetracapsuloides bryosalmonae* and the effect of temperature on the expression of proliferative kidney disease. The parasite has two hosts. (a) Bryozoans are primary hosts. Naturally, they grow in colonies at places that are not overgrown with algae like under and between stones, boulders and in shaded parts on tree roots. Sunshine-increased temperature increases primary production which supports the development of larger bryozoan colonies that may boost production of infectious malacospores for salmonids. (b) This process can be reversed by providing riparian strips that provide shade. Different salmonids like (c) Atlantic salmon and (d) brown trout can be infected with the parasite, and development of PKD is strongly species and temperature dependent. The spores enter via mucus-rich tissue, mainly the gills, and proliferate in the fish kidney. Species show different temperature-dependent immune responses to the parasitic infection. In general, at low water temperatures, infected kidneys do not differ in appearance from healthy kidneys (e1). However, higher temperatures for extended times result in (e2) more severe kidney hyperplasia and the disintegration of haematopoietic tissue may result in higher mortality in warmer water with lower oxygen levels. After sporogenesis, the parasite produces fish malacospores that are excreted via urine

small bryozoan colonies that are not producing statoblasts (Fontes, Hartikainen, Williams, et al., 2017). Fish malacospores shed by infected salmonids may kick-start as well as augment infections and maintain the parasite in bryozoan colonies in head waters. In Central Europe, *F. sultana* (Figure 3g,h) and *Plumatella* sp. (Figure 3i) are widely distributed in the rhithral river environment and overlap in range with *T. bryosalmonae* infections of brown trout (*Salmo trutta*; Carraro et al., 2017; Fontes et al., 2017; Ros et al., 2021; Waldner et al., 2020). These bryozoan species can be found patchily distributed often between or under large rock slabs or boulders (Figure 3e) and between structures such as tree roots (Figure 3f; Wood & Okamura, 2005). Elevated head waters in mountainous areas with fast flowing water (Figure 3d) seem less favourable for bryozoans (Ros et al., 2021; Waldner et al., 2020). This could be explained by a limited period at higher elevations with temperatures that favour bryozoan growth (Carraro et al., 2017).

2.1 | Transmission to salmonids

Mature spore sacks (Figure 3j) eventually leave the zooid and burst to release the malacospores (Sudhagar et al., 2019). These waterborne malacospores must infect a salmonid species to further complete the life cycle (see table in: Sudhagar et al., 2019). Transmission of parasites to fish has been studied in rainbow trout and brown trout and, in general, the patterns are similar. Malacospores are activated by fish mucus to fire their polar filaments. When they attach to the gills, the sporoplast leaves the spore and penetrates the tissue using amoeboid movements (Grabner & El-Matbouli, 2010). The parasite has been found to penetrate gills both in rainbow trout (Grabner & El-Matbouli, 2010; Longshaw et al., 2002; Morris et al., 2000) and in brown trout (Holzer et al., 2005; Schmidt-Posthaus et al., 2013). Additionally, parasites have been shown in mucus cells in the skin of rainbow trout which might thus act as an additional portal of entry for *T. bryosalmonae* for this species and likely also for other salmonids (Longshaw et al., 2002).

Once the sporoplast has entered the fish host, it must migrate through the bloodstream and leave at the right place to enter the kidney interstitium in order to complete sporogenesis. This taxis mechanism to find the kidney (Bettge et al., 2009; Kent & Hedrick, 1986) must be highly efficient as experimental exposure with a single or few malacospores have been shown to result in PKD in rainbow trout (McGurk et al., 2006). In other myxozoans, a high level of specialization in the route to the target tissue has been shown (Gómez et al., 2014). The taxis mechanisms of *T. bryosalmonae* to leave circulation and enter the kidney have not been elucidated yet. The parasite has been found to attach to the blood vessel wall where a thrombus is formed with macrophages, lymphocytes

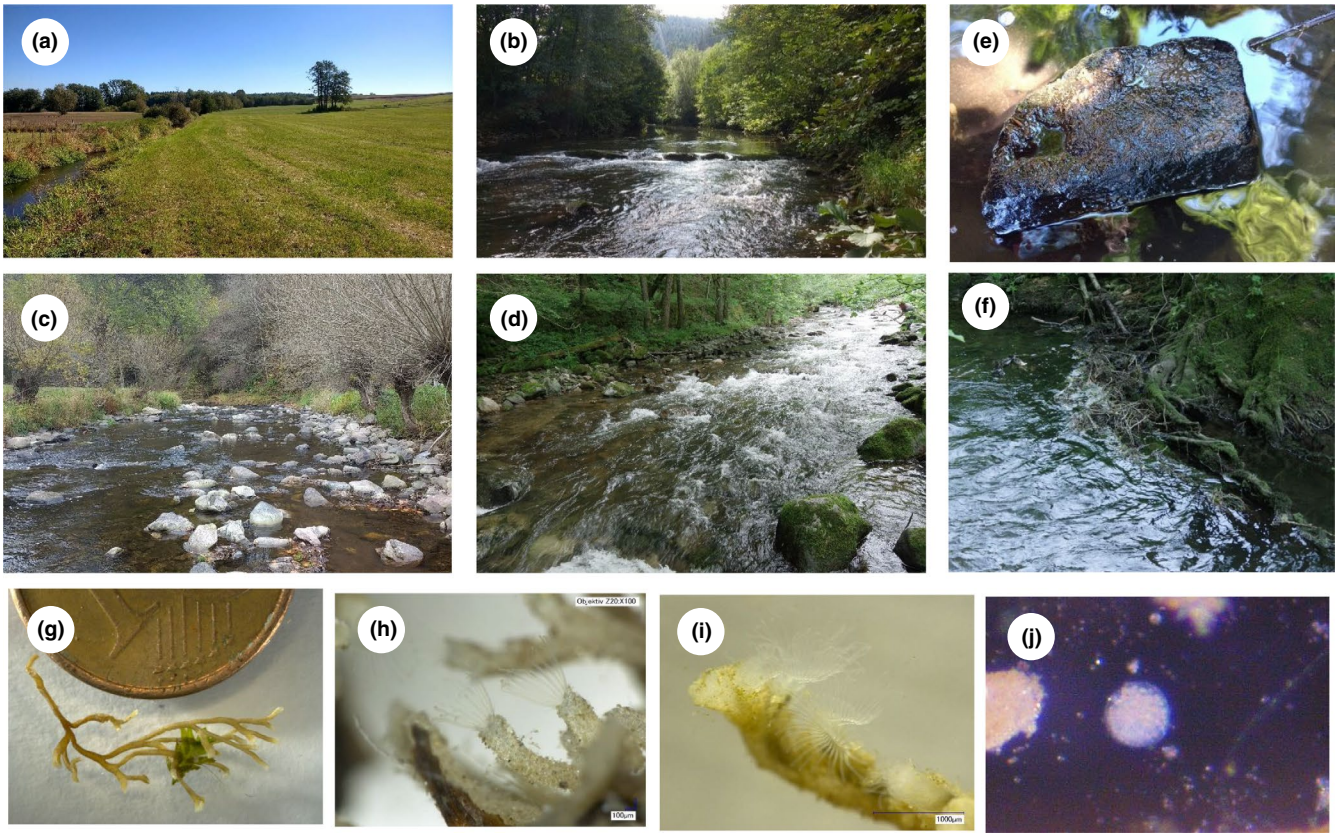

FIGURE 3 (a-d) are examples of streams where salmonids can be found in high numbers; (a-c) have populations of the bryozoan *Fredericella sultana* (g and h) and *Tetracapsuloides bryosalmonae* ((j) shows a spore sack); in (b and c) also colonies of *Plumatella* sp. (i) can be found. (a) has large stretches that miss riparian buffers. (b-d) have riparian buffers. Bryozoan populations and PKD are found in highest numbers in (a). Bryozoans are often found in shady places under and between rocks and stones (e) or between tree roots (f)

and erythrocytes, and which probably helps the parasites to stay on place at the vascular wall through which it can migrate into the interstitial tissue (Schmidt-Posthaus, 2016).

2.2 | Proliferation, sporogony and shedding of spores

Once in the interstitium, the pre-sporogonic phase of development starts in which parasitic cells multiply and then differentiate (proliferation phase, Figure 4). Sporogenesis (Feist et al., 2015; Morris & Adams, 2008) starts with the formation of a cell doublet in which a primary cell contains a secondary cell within it. Sometimes the secondary cell multiplies up to a maximum of three secondary cells within the primary cell. In a further stage, a pseudoplasmodium is formed through engulfment of one secondary cell by another one (S-T doublet). Such pseudoplasmodia have been recognized in brown trout, rainbow trout and Arctic charr (*Salvelinus alpinus*; Morris & Adams, 2008). In the final stage of sporogony, the secondary cell of the pseudoplasmodia undergoes two divisions to produce four valvogenic cells (Morris & Adams, 2008). The S-T doublet transforms into two capsulogenic cells (the polar capsules) and a sporoplasmogenic cell. All this still takes place within the primary cell. The following stage of sporogony takes place after the pseudoplasmodia leaves the interstitium to enter a renal tubule (Morris & Adams, 2008). The mature fish malacospore moves from the distal tubulus to the collecting ducts and is then shed with urine (Hedrick et al., 2004; Tops et al., 2004).

At experimental temperatures of 12 or 15°C, sporogenesis can be completed in 7–8 weeks after parasite infection (Figure 4), which is well in the range of summer temperatures measured in central European rivers where salmonids can be found (Rubin et al., 2019). Fish start shedding fish malacospores (measured as parasite DNA in the water) from this period onwards (Strepparava et al., 2018, 2020). Shedding then likely results in a balance among presporogonic multiplication, maturation and fish malacospores leaving the kidney, resulting in a plateau phase with lymphocytic hyperplasia of the kidney (PKD), parasite intensity and shedding remaining relatively stable for a period of about 17 weeks (Strepparava et al., 2018, 2020). After this period, kidney hyperplasia and parasite intensity clearly decrease, but shedding still continues (Figure 4). Translated to the field, this implies that fish that get infected in May–June will shed fish malacospores in July–August when bryozoan populations are at their maximum density (Fontes, Hartikainen, Holland, et al., 2017; Fontes, Hartikainen, Williams, et al., 2017). The fish that survive the winter may still shed fish malacospores and infect the new growing

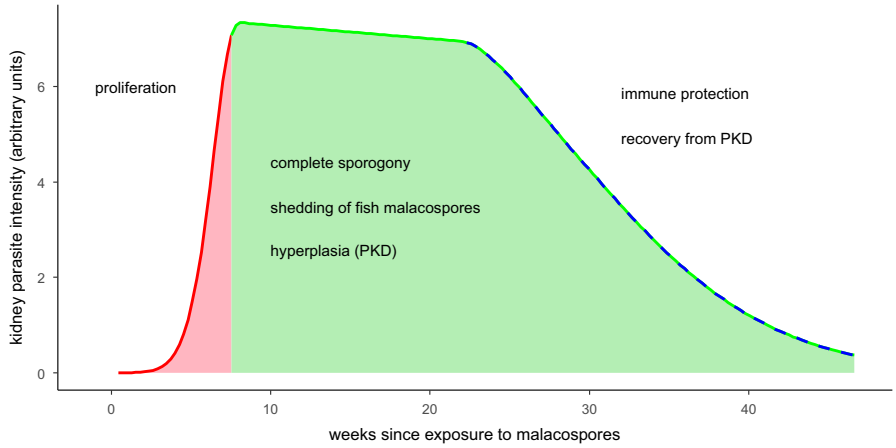

FIGURE 4 Temporal pattern of kidney changes after exposure to *Tetracapsuloides bryosalmonae* malacospores based on studies on brown trout (Strepparava et al., 2018, 2020). (1) Proliferation phase (red): the parasite population shows exponential growth in the kidney, but also spleen and liver may be colonized. Downregulation of innate cellular defences (macrophage activity) and dysregulation of adaptive immunity (B-cell markers and cytokines) have been reported in this phase and might later result in lymphocytic hyperplasia at higher ambient temperatures (Bailey, Holland, et al., 2020). Spore density and temperature affect the speed of the proliferation but not so much the number of parasites reached in the peak of this phase. (2) Plateau phase (green): sporogony completes and spores migrate through tubuli to be shed with urine as fish malacospores. At higher temperatures, kidneys may show severe hyperplasia, that is, PKD, resulting in loss of haematopoietic tissue; (3) 'Recovery phase' (blue hatched): parasite numbers decrease in the kidney, and the kidney show signs of recovery. Some parasite infections may persist and fish may remain shedding fish malacospores for years after the infection (Soliman et al., 2018). Phagocytosis and markers of adaptive immunity are upregulated and fish obtain resistance against new infections and the development of PKD

bryozoan populations in spring. In fact, some experimental brown trout were found to shed fish malacospores 5 years post-exposure that could still infect bryozoans (Soliman et al., 2018).

2.3 | Host specificity and susceptibility

Transmission studies in Europe showed that the parasite is able to conclude its life cycle in the abundant native brown trout (Grabner & El-Matbouli, 2008; Morris & Adams, 2006c). The Arctic charr is also a likely candidate for completing the parasite life cycle (Bucke et al., 1991; Hedrick et al., 1993) as Svavarsdottir et al. (2021) found mature fish malacospores in kidney tubuli in Arctic charr in Iceland and Mo and Jørgensen (2017) found *T. bryosalmonae* in Arctic charr in one of the northernmost rivers in Norway and the European mainland. Studies in Europe with non-native brook (*Salvelinus fontinalis*) trout and rainbow trout have returned variable results. Earlier studies in the UK reported that the brook trout is not susceptible to *T. bryosalmonae* (Seagrave et al., 1981), while an Austrian transmission study found the parasite to complete its life cycle in hatchery procured brook trout (Grabner & El-Matbouli, 2008). Here, it would be interesting to study whether this is related to PKD susceptibility of the native Arctic charr, as hybrids between this species and brook trout are frequently detected in hatcheries in the Austrian study area (El-Matbouli & Grabner, 2007; Gross et al., 2004). Some indication was also found for completion of the life cycle of the parasite in rainbow trout as several studies have detected spores of the parasite or its DNA in urine (Bettge et al., 2009; Hedrick et al., 2004). However, an Austrian transmission study for this species did not find evidence for shedding of fish malacospores, and no bryozoans got infected with the parasite after exposure to the water in which the infected rainbow trout had swum (Grabner & El-Matbouli, 2008). Furthermore, aquaculture farms in the UK with infected rainbow trout are not producing infective fish malacospores (Bucke et al., 1991). It thus remains unclear whether American salmonid species may also be involved in the life cycle of *T. bryosalmonae* in Europe, but *Oncorhynchus* species likely play a role on the American continent (*Oncorhynchus clarki*: Hedrick et al., 1993). Although it is not yet clear whether shedding of fish malacospores is parasite strain dependent (Morris et al., 1997), the data suggest that American *T. bryosalmonae* strains adapted to American salmonid species and European *T. bryosalmonae* strains adapted to European salmonid species (Hedrick et al., 1993).

European transmission experiments with the European grayling (*Thymallus thymallus*) could not detect shedding of fish malacospores infecting bryozoans (Grabner & El-Matbouli, 2008). Independent of whether salmonids as the grayling and the rainbow trout might be dead end hosts for (the European strain of) *T. bryosalmonae*, the parasite does still infect these salmonids and cause disease. Native species that have been found susceptible to *T. bryosalmonae* infections in the wild are for Europe: brown trout (*Salmo trutta*: Wahli et al., 2002), Atlantic salmon (*Salmo salar*: Feist et al., 2002; Lauringson et al., 2021; Sterud et al., 2007), Arctic charr (*Salvelinus alpinus*: Kristmundsson et al., 2010; Mo & Jørgensen, 2017; Svavarsdottir et al., 2021), European grayling (*Thymallus thymallus*: Feist & Bucke, 1993; Vasemägi et al., 2017; Wahli et al., 2002) and European whitefish (*Coregonus lavaretus*: Sobocinski et al., 2018); and for North America: rainbow trout (*Oncorhynchus mykiss*: Hutchins et al., 2018b; Ruggeri et al., 2020), Chinook salmon (*Oncorhynchus tshawytscha*: Hutchins et al., 2018b), Cutthroat trout (Henderson &

Okamura, 2004; *Oncorhynchus clarkii*: Macconnell & Peterson, 1992), pink salmon (*Oncorhynchus gorbuscha*: Braden et al., 2010), brook trout, Arctic charr (*Salvelinus alpinus*: Brown et al., 1991) and, for example, mountain whitefish in the Yellowstone River (*Prosopium williamsoni*: Hutchins et al., 2018b).

The question remains whether transmission in Europe is restricted to brown trout and brook trout or whether related *Salmo* and *Salvelinus* species might play a role as well. Due to its large geographical reach, anadromous Atlantic salmon could be a very important species in the distribution dynamics of *T. bryosalmonae* and they are highly susceptible to infection (Ellis et al., 1985; Lauringson et al., 2021; Mo & Jørgensen, 2017; Sterud et al., 2007). Salmon parr has been found positive to the parasite in 70.3% of sampled rivers in Norway, including rivers in the upper North where they are the only salmonid that can spread the parasite (Mo & Jørgensen, 2017). There are some indications that the high prevalence of PKD in Atlantic salmon strains from Norway (Sterud et al., 2007) is not matched by the prevalence of Atlantic salmon in central Europe (Germany, Switzerland) and the Baltic (Estonia) (Lauringson et al., 2021; Thomas Wahli & Zopfi, 2016b). Here, it would be interesting to study whether differences in mucus-facilitated spore penetration success might play a role (Grabner & El-Matbouli, 2010) as there is large strain variation in Atlantic salmon in mucus composition and/or gill goblet numbers (Persson et al., 2021). Although mature fish malacospores have been found in the kidney of Atlantic salmon (Svavarsdottir et al., 2021), parasite transmission studies showing shedding of the parasite and successful infection of bryozoans have so far not been carried out for this species.

2.4 | Temporal pattern of proliferative kidney disease and recovery

Tetracapsuloides bryosalmonae may infect salmonids and reproduce in their kidneys without causing disease (Figure 2: e1). Without molecular or histological examinations, such fish are indistinguishable from non-infected fish, and they do not show a gross hyperplasia of the kidney (Figure 2: e2) that is characteristic of clinical pathology of PKD (Bruneaux et al., 2017; Clifton-Hadley et al., 1987). Both in non-clinical infections and in fish developing PKD, the parasites strongly increase in numbers in 1 to 2 months after reaching the kidney (Figure 4), and this leads to detectable levels as early as a few days after infection when using molecular techniques (Strepparava et al., 2020). In milder forms of PKD, the infiltration of the kidney interstitium with mononuclear cells is limited to one or few granulomatous inflammatory nodules that normally occur in the posterior kidney, but in severe clinical cases the inflammation extends over the whole kidney and may result in a clearly visibly distended belly (Bettge, Segner, et al., 2009; Schmidt-Posthaus et al., 2015). Together with the hypoxia induced due to thrombus formation, more severe inflammation results in necrosis of the haematopoietic tissue. Also, the spleen is affected by the disease, being clearly enlarged (splenomegaly) in fish with PKD (Chilmonczyk et al., 2002; Feist et al., 2001). Although both kidney and spleen harvest high parasite counts, also small numbers of parasites can be found in the liver (Palikova et al., 2017), intestine, gills and even other tissues, like cerebral tissue. In advanced PKD, the diseased fish become anaemic showing pale gills, low vigour and exophthalmos (Bruneaux et al., 2017; Palikova et al., 2017).

The period after the clinical phase of PKD (Figure 4: blue hatched period) is probably least understood. In this period, kidneys from sampled rainbow and brown trout harvest decreasing numbers of pseudoplasmodia and show clear evidence of regeneration of interstitium and tubuli (Clifton-Hadley et al., 1987; Schmidt-Posthaus et al., 2012; Schmidt-Posthaus et al., 2013). The recovery in these surviving fish appeared to be independent of temperature regime during the clinical phase both in laboratory conditions (Clifton-Hadley et al., 1987; Schmidt-Posthaus et al., 2012; Strepparava et al., 2018) and in the field (Schmidt-Posthaus et al., 2013). Some evidence has been presented that recovery can commence when parasite numbers, and thus the intensity of the parasite–host tissue interaction, fall under a certain threshold (Schmidt-Posthaus et al., 2012). This may also explain why in some river environments with high malacospore density and warm water temperatures, the recovery process might be impeded (Schmidt-Posthaus et al., 2013). As individuals cannot be followed through the disease process, it is not possible to know whether fish with severe hyperplasia will regenerate as well as fish with low-level hyperplasia. However, the fact that infected rainbow trout held at both 12 and 18°C regenerate completely indicates that even severe damage through PKD-related pathology that occurred at 18°C can be resolved when ambient conditions are optimal (Schmidt-Posthaus et al., 2012). Under less optimal conditions, like in polluted waters after wastewater treatments, such recovery might be hampered (Bailey, Rubin, et al., 2018; Rubin et al., 2019). When fish survive infection, they are thought to be protected to some extent against future parasitic infection. This might be by altering the tissue response and tolerating the parasites infecting the kidney and/or in preventing infections with *T. bryosalmonae* at the port of entry (gills and/or skin). In general, this fits with the observation that adult fish may test positive for *T. bryosalmonae* but do not show hyperplasia. It is still unknown whether salmonids are capable of producing acquired immunity via immunoglobulins (Bailey et al., 2020). However, current success in vaccination procedures (Faber et al., 2019) indicates that acquired immunity could play a role in the recovery and protective phase against PKD.

3 | THE RELATION BETWEEN TEMPERATURE AND PROLIFERATIVE KIDNEY DISEASE

3.1 | Temperature effects on disease outcome

Even before the life cycle of the parasite *T. bryosalmonae* was discovered, it was realized that keeping rainbow trout at lower temperatures inhibited the clinical development of PKD (Ferguson, 1981). This has since been confirmed in experiments with 0+ trout which in general report mild kidney hyperplasia and

pathological changes with low mortality at 9°C or 12°C, increased kidney pathology at water temperatures of 15°C and increased pathology with increased mortality at temperatures of 18°C or higher (Bettge, Segner, et al., 2009; Clifton-Hadley et al., 1986; Strepparava et al., 2018; Waldner et al., 2021). Although higher temperatures affect the PKD disease outcome, they have a lesser effect on the life cycle of the parasite. Conditions at 9°C might be adverse for the parasite to proliferate (Clifton-Hadley, Richards, et al., 1986). However, already at 12°C, parasites proliferate in brown trout kidney leading to fish malacospore shedding which is only slightly delayed from that at 15°C (Strepparava et al., 2018). Also, parasite proliferation (Figure 4 plateau phase) appears to reach similar copy numbers per tissue weight independent of temperature (Strepparava et al., 2018).

A second important aspect of this temperature effect is that the outcome is different between species. In European studies of PKD, temperature effects on disease outcome have been found more accentuated for the native brown trout in comparison to the introduced rainbow trout (Bailey, Rubin, et al., 2018). This may still be the case in adult wild fish, as both species were found to be infected with *T. bryosalmonae* at the age of 1 year, whereas only brown trout showed clinical signs of PKD together with other pathology (Arndt et al., 2019). This cannot be explained by an effect of the parasite strain alone as also different native species have been found to respond differently to *T. bryosalmonae*. European grayling and Atlantic salmon have been reported to be less susceptible to PKD than brown trout when kept at similar temperatures (Wahli & Zopfi, 2016a, 2016b). Also, PKD disease prevalence in these species have been found low in comparison to disease prevalence in brown trout at similar ambient temperatures (Ros et al., 2021), and in experimental settings (Grabner & El-Matbouli, 2009; Schmidt-Posthaus et al., 2017). Still in rivers with temperatures that often rise above 20°C, like the Danube in Southern Germany (Ros et al., 2021) and several rivers in Finland (Vasemägi et al., 2017), grayling has been found to be highly susceptible to *T. bryosalmonae* with parasite infection prevalence as high or even higher than that of brown trout in the area. For fry of Atlantic salmon, high PKD-related mortalities have been reported in a river in central Norway during a period in which temperatures stayed for nearly a month above the normal mean summer temperatures of 15°C (Sterud et al., 2007). Also, in Northern America, several salmonid species including the mountain whitefish (*Prosopium williamsoni*) have been found susceptible to PKD (Hutchins et al., 2021).

3.2 | Temperature effects on immunity and tolerance

Studies into proximate mechanisms of temperature effects on PKD in trout are important for understanding how fish may adapt to the current changes in their environment. One major point of focus is how trout may 'tolerate' infections with *T. bryosalmonae* at low temperatures, whereas they develop severe inflammatory responses leading to PKD at higher temperatures, as discussed in the former section. In infected rainbow trout, it has been shown that some kidney damage can be prevented by suppressing the inflammatory response with cortisol implants, which, however, resulted in much higher numbers of extrasporogonic parasite stages (Kent & Hedrick, 1987). Higher numbers of extrasporogonic stages in cortisol-treated fish suggest they benefit from decreased immune response. Interestingly, extrasporogonic stages can be found engulfed by host macrophages (Morris & Adams, 2008) but do not show indications of being degraded. It is possible that *T. bryosalmonae* may use this as an evasion strategy to render itself invisible to further immune vigilance. This has been shown for some bacteria and parasites which manipulate the internal signalling and transport of host macrophages to survive in the cytoplasm or self-produced vacuoles (Schmid-Hempel, 2009). Such evasion strategies might explain how *T. bryosalmonae* is able to proliferate in the kidney for several months and may still complete sporogenesis years after infection (Soliman et al., 2018; Strepparava et al., 2018).

Recently, much research has focussed on mechanisms of host immunity during clinical PKD, elucidating host immunity and parasite evasion strategies that have been expertly reviewed (Bailey et al., 2020; Sitjà-Bobadilla et al., 2015). Most of these studies have focussed on kidney responses to PKD in rainbow trout. The immune system is an elaborate suite of physiological mechanisms that play a role in surveillance, detection, suppression and clearance of aberrant cell activity and pathogens. In order to successfully exploit a host, *T. bryosalmonae* has to breach the epithelial barrier, survive chemical barriers and evade innate and acquired cellular immunity. The results of these studies show that *T. bryosalmonae* infection-related pathology is characterized by a decrease in myeloid cells (Bailey et al., 2017; Chilmonczyk et al., 2002), and an imbalance in the differentiation patterns of the T-helper-like response with dysregulation of B-cell antibody responses (Bailey, Holland, et al., 2020; Bailey et al., 2017; Gorgoglione et al., 2019; Holland et al., 2003). This immune phenotype does not fit a targeted immune response to the parasites, and T-helper cell differentiation rather resembles aspects of a lymphoproliferative autoimmune disease (Bailey, Holland, et al., 2020). This together with the long-lasting survival of *T. bryosalmonae* in kidney tissue suggest that the parasite plays an active role in the deficient immune phenotype by suppressing cytokines that are pro-inflammatory (Bailey, Holland, et al., 2020; Bailey et al., 2017; Gorgoglione et al., 2019; Holland et al., 2003), and by provoking polyclonal Ig activation in order to dilute the effective antibody response of B cells (Bailey, Segner, et al., 2020).

3.3 | Temperature effects on co-evolutionary processes

It is still little understood how at low ambient temperatures the host response to *T. bryosalmonae* prevents tissue damage, whereas at

higher temperatures inflammation results in PKD with necrosis of the haematopoietic tissue, ultimately leading to hypoxia and death (Schmidt-Posthaus et al., 2013; Strepparava et al., 2018). In general, there seems to be no clear benefit of advanced PKD to the parasite as tissue damage cumulating in mortality would also decrease the release of viable fish malacospores. Thus, it would be possible that under normal circumstances in summer-cool rivers, the parasites are able to evade or prevent host immune responses, whereas this is disturbed at higher temperatures (Bailey, Segner, et al., 2020). On the host side, it seems reasonable to deduce that it would be beneficial to prevent the gross tissue damage that occurs with the massive granulomatous inflammation shown in advanced PKD. Recent progress in comparative transcriptomics (Ahmad, Debes, Pukk, et al., 2021; Faber et al., 2021) and genetic profiling of immunity (Bailey, Holland, et al., 2020) is now starting to reveal the extent to which such adaptations have evolved in both parasite and its hosts (Ahmad, Debes, Nousiainen, et al., 2021). In general, such adaptions are expected to result in evolution for low virulence of the parasitic infection and this would fit the hypothesis that proliferation of *T. bryosalmonae* in the kidney does not result in gross pathology at water temperature conditions that prevailed in salmonid habitat before anthropogenic global warming (Borgwardt et al., 2020; Bruneaux et al., 2017; Lauringson et al., 2021; Strepparava et al., 2018; Wahli et al., 2002). Additionally, it is expected that adaptations fluctuate between local populations, dependent on gene flow and species interactions (Nadeau & Urban, 2019). Geographically isolated host–parasite populations might harbour different isomorphs of the parasites to which, in turn, hosts might respond differently. If so, this would result in geographically different parasite–host interactions, at the level of different continents, different watersheds or even different rivers of the same watershed.

The amount of gene flow between populations is important for assessing the geographic scale on which local adaptations may develop (Johnson et al., 2020). Gene flow should be expected through migration of infected fish between rivers, through release of infected statoblasts and through dislocation of infected bryozoans (Abd-Elfattah, Fontes, et al., 2014; Abd-Elfattah, Kumar et al., 2014). A role of birds in parasite dispersal has been postulated either through ingestion or through infected statoblasts that stick to feathers (Abd-Elfattah, Fontes, et al., 2014; Hallett et al., 2015; Henderson & Okamura, 2004). However, a recent study did not support that consumption of fish infected with *T. bryosalmonae* could result in excretion of viable malacospores (Schmidt-Posthaus et al., 2020). Thus, dispersal of the parasite might be expected to be limited to the displacement of bryozoan and salmonid hosts, and therefore restricted to the scale of a river system (Carraro et al., 2018).

Selection leading to co-evolution and local adaptation may ultimately explain differences within and between salmonid species in susceptibility to infections with *T. bryosalmonae* and/or in PKD development. To successfully exploit their hosts, parasites are in a continuous arms race to evade host resistance mechanisms (Agrawal, 2001; Holzer et al., 2018). The most stable outcomes appear to be those where parasites have relatively low virulence, as this lowers the selection pressure on the host resistance mechanisms allowing momentum in the evolution of evasion mechanisms in the parasite (Paterson et al., 2010; van Baalen, 1998). Natural variation in susceptibility to PKD development, which could be interpreted as variation in the virulence of *T. bryosalmonae*, has been detected for several species. This was shown for brown trout in rivers in Estonia, in which variation was found between brown trout strains in the reaction, expressed as differences in parasite intensity and clinical response (kidney hyperplasia, haematocrit values, condition; Debes et al., 2017). In concordance, surviving or not surviving a season with PKD has been shown to be correlated with differential gene expression and this would thus result in natural selection for the underlying genes (Ahmad, Debes, Nousiainen, et al., 2021). When temperatures rise, tolerant or more immunocompetent brown trout strains would benefit and spread in the population. As the selection effect is dependent on the local temperature conditions, this could then lead to locally adapted brown trout populations (Jensen et al., 2008). Geographically distinct populations of Atlantic salmon can show either high PKD-related mortality (Norway: Sterud et al., 2007) or a benign clinical response to the parasite (Estonia: Lauringson et al., 2021; Switzerland: Wahli & Zopfi, 2016b). Finally, variation in the response or susceptibility to infections with *T. bryosalmonae* has also been detected in aquaculture facilities between strains of rainbow trout (Grabner & El-Matbouli, 2009; Syrová et al., 2020) leading to attempts to breed a PKD-resistant line (Butterfield, 2008).

4 | PROLIFERATIVE KIDNEY DISEASE, POPULATION LOSS AND CLIMATE CHANGE

4.1 | Climate change and proliferative kidney disease related to population loss

Proliferative kidney disease is generally viewed as an emerging disease in response to climate change that leads to mortality at increased water temperatures (Okamura et al., 2011; Waldner et al., 2021). The disease is currently implicated as one of the key factors causing decline in brown trout populations of central and southern Europe (Arndt et al., 2019; Burkhardt-Holm & Zehnder, 2018; Dash & Vasemägi, 2014; Gorgoglione et al., 2016; Ros et al., 2021; Skovgaard & Buchmann, 2012; Wahli et al., 2008). It is difficult to estimate mortality due to PKD, as mortality is influenced by a combination of confounding stressors (Hutchins et al., 2019). However, even in cases in which PKD is not the direct cause of death, the disease may still increase vulnerability to other causes that lead to mortality such as: (1) anaemia through destruction of haematopoietic tissue leading to decreased oxygen absorbability (Bruneaux et al., 2017; Hoffmann & Lommel, 1984; Palikova et al., 2017) and therefore to lowered oxygen levels caused by elevated water temperatures (Demars & Manson, 2013); (2) PKD-induced anaemia leading to failure of glomerular filtration (Clifton-Hadley et al., 1987), which may render the diseased fish more vulnerable to the effects of pollution (Bailey, Rubin, et al., 2018;

Rubin et al., 2019; Schmidt et al., 1999) and (3) hyperplasia resulting in lower physiological performance (Bruneaux et al., 2017), which might expose infected fish to higher predation (Borsuk et al., 2006; Feist & Bucke, 1993).

In the last two decades, an increasing number of studies have reported population declines in salmonids (Switzerland: Burkhardt-Holm & Zehnder, 2018; Iceland: Kristmundsson et al., 2010; Germany: Ros et al., 2021; Austria: Waldner et al., 2020) and fish kills (Germany: Arndt et al., 2019; USA: Hutchins et al., 2018a; Norwegen: Sterud et al., 2007) that are concurrent with high prevalence of PKD. Primarily young (YOY/parr) but also to some extent adult fish might be affected by PKD (Arndt et al., 2019; Bailey et al., 2019; Hoffmann & Dangschat, 1981). For example, the density of YOY brown trout was found to be more than 90% lower in rivers with PKD than in rivers without PKD (Schager et al., 2007), and this could be attributed to high PKD-related mortality of YOY, beside other natural causes of death in this age class (Schubiger, 2003; Waldner et al., 2021), while differences in water quality between the rivers must have played a role as well (Burkhardt-Holm & Scheurer, 2007; Rubin et al., 2019; Schmidt et al., 1999)

4.2 | Estimates of PKD-related mortality in young of the year (YOY)

There have been some tentative estimates of mortality effects of *T. bryosalmonae* infections on wild populations of salmonids. Most data have been collected for brown trout in Europe. Under conditions of oxygen-rich river water with summer temperatures close to or higher than 15°C, an additional mortality of 15% was measured in YOY of brown trout with *T. bryosalmonae* infection prevalence of about 70% (Schmidt-Posthaus et al., 2017; Schmidt-Posthaus et al., 2015). In two Swiss rivers in which *T. bryosalmonae* infections were detected and with river temperatures that measured often above 15°C, a loss of approximately 50% of YOY brown trout was reported from summer to autumn (Ahmad, Debes, Nousiainen, et al., 2021; Borsuk et al., 2006; Vatland & Caudron, 2015). Not known is whether survivors of *T. bryosalmonae* infections have lower chances surviving while recovering from PKD during winter than fish not infected with the parasite (Carraro et al., 2017).

In general, river stretches have limited carrying capacity (food, hiding places) for top predators like salmonids, resulting in density dependency regulation of population sizes (Baer & Brinker, 2008). Mark and recapture studies have also shown that young trout show site fidelity higher than 80% over a period of 3 months (Vatland & Caudron, 2015). Thus, disappearance of YOY from a river stretch is a likely proxy of mortality in those fish (see also Ahmad, Debes, Nousiainen, et al., 2021). A year with higher YOY mortality would result in less recruitment to 1st year trout but in turn these would face less competition for food and thus an increase in their chance of survival. Density dependency may thus partly compensate for the loss in recruitment due to increased mortality through PKD (Borsuk et al., 2006). Still the recent, more extreme summer temperature conditions are suspected to severely disrupt recruitment in trout (Carraro et al., 2017; Ros et al., 2021). The combination of high water temperatures and PKD might cascade mortality via a loss of recruitment to a level that apparently cannot be compensated for by density-dependent mechanisms. This has been proposed for the decrease in population density in two rivers in Germany, the Wutach and the Gutach. Fisheries management of these rivers is based on natural production of local brown trout accompanied with low fishing pressure (Baer & Brinker, 2010). In the decade after cessation of stocking, in 2000, populations stabilized, but recently the brown trout has seen a 50% decline in all age classes, and this decline was found to be strongest in river stretches in which PKD was found, and especially in hot years recruitment from YOY to 1st year fish seemed disrupted (Ros et al., 2021). Supporting PKD as causative factor, this decline was related with increased hyperplasia (Ros et al., 2021).

Little PKD-related mortality data are available for other species than brown trout. The authors are not aware of studies on wild-living American salmonids like for the *Oncorhynchus* species. Atlantic salmon populations have decreased sharply, with only Northern European countries reporting healthy populations (but see for recent declines: Forseth et al., 2017; WWF, 2001). Anthropogenic factors for this decline are chemical pollution (Plum & Schulte-Wülwer-Leidig, 2014; Villamayor-Tomas et al., 2014), epizootics originating from fish farms (Costello, 2009) and the disruption of their anadromous migration patterns through river fragmentation and power-generating water works (Marschall et al., 2011). The combination of climate change and PKD (Ellis et al., 1985; Forseth et al., 2017) is an increasing factor of concern for the fragile restoration of salmon populations (Bölscher et al., 2013). Infections with *T. bryosalmonae* have been found in 70% of sampled Norwegian rivers with Atlantic salmon (Mo & Jørgensen, 2017), although prevalence of parasite infections in Atlantic salmon populations of sampled rivers in England and Wales (Feist et al., 2002) and Denmark (Skovgaard & Buchmann, 2012) is low. An increase of 5°C above normal summer temperatures in a Norwegian river in 2002–04 resulted in an estimated additional 85% mortality of fry that showed advanced symptoms of PKD (Sterud et al., 2007). In this fish kill, older parr were also affected but it was reported that their mortality was more difficult to estimate. This may partly be due to parr in comparison to fry being in downstream more turbulent parts of the river (washing out of moribund fish), and probably due to increased predation and scavenging on affected fish in such parts of the river (Feist et al., 2002; Sterud et al., 2007). Such factors may result in underestimation of the impact of PKD on fish mortality.

4.3 | Proliferative kidney disease-related mortality in adult fishes

The situation in relation to PKD is still unclear and complex for adult populations of salmonids. The hypothesis is that fish that survived *T. bryosalmonae* infections are mostly immune protected against new

infections. Nevertheless, in a recent study, adult brown trout in a population in Bavaria were still found be infected with *T. bryosalmonae* with many fish showing kidney hyperplasia (Arndt et al., 2019). Adult salmonids perform many energetically challenging traits like migration and reproduction. Considering that PKD may affect salmonid physiology of YOY brown trout through impairing aerobic performance and thermal tolerance (Bruneaux et al., 2017), more information would be needed to evaluate whether and how *T. bryosalmonae* would impact salmonids that have life histories with outward migration of smolt (Mo et al., 2011), and whether this might play a role in the dwindling numbers of adults returning to the spawning grounds (Foott et al., 2007; Hedrick et al., 1984; Kent et al., 1995; Mo et al., 2011).

5 | MANAGEMENT OPTIONS FOR MITIGATING PKD IN SALMONIDS

5.1 | Monitoring the occurrence of hosts and parasite in river systems and PKD in salmonid populations

Salmonid population sizes are strongly fluctuating, whereby traits like predation, available food items and young survival are affected by weather conditions like excessive rainfall, harsh winter conditions or high water temperatures during summer (Baer & Brinker, 2010; Crozier et al., 2008; Huusko et al., 2013; Lobón-Cerviá & Mortensen, 2005; Shuter et al., 2012). As weather conditions shift considerably from year to year, it is only when populations are monitored over extended time spans that changes in population sizes and the effect of their diseases will become evident and traceable (Ros et al., 2021; Wahli et al., 2002). To account for this year-to-year variation, climatologists calculate their 'climate normals' as 30-year averages (World Meteorological Organization, 2018). However, with the speed by which the environment is changing through climate change (Sherwood et al., 2020), comparisons of data collected over such long periods would not capture the actual conditions of the populations under management. Thus, parallel to evaluating the long-term averages, it is now advised by the WMO to inform about climate effects with publications using interim comparisons and correlative tendencies calculated over 10-year periods (World Meteorological Organization, 2018).

Thus, climate change-related reductions in local salmonid population sizes could in principle be detected based on a 20-year dataset if the population under study over that period is not strongly influenced by other stressors (e.g. chemical stressors), and rigorous standards are followed that allow for geographical and temporal comparisons and to correct for catch per unit of effort (Ros et al., 2021). As collection of long-term datasets is costly and labour intensive (Baer & Brinker, 2010; Burkhardt-Holm et al., 2002), it is expedient to analyse the generally available records of fishing societies and fisheries that report their catches. Although it is difficult to correct such data for catch per unit of effort due to variability in capture methods and report accuracy (but see Grilli et al., 2021), these data have been used to detect which populations might be under pressure of environmental change, especially when changes have been vast and therefore difficult to miss, and in the case when fish mortalities occur (Arndt et al., 2019; Hutchins et al., 2019; Schneider, 2011; Unfer et al., 2015). This then provides a valid starting point to set up monitoring of PKD as one method to search for possible causes of a decline.

To detect the occurrence of *T. bryosalmonae* and the severity of PKD in salmonids, both microscopic and molecular methods have been applied. Microscopic determination of the parasitic (PKX) cells in the kidney is done from histologic preparations which require tissue that has been collected from freshly killed fish. Kidneys should be directly prepared and formalin fixed (10% neutral buffered formalin: Clifton-Hadley et al., 1987; Schmidt-Posthaus et al., 2012; Schmidt-Posthaus et al., 2015) to prevent artefacts through cells autolysing prior to fixation (George et al., 2016). For molecular detection of *T. bryosalmonae*, a part of the fresh kidneys (20 mg of kidney is sufficient to extract DNA using regular tissue kits) should be preserved in 96% absolute ethanol (Mo et al., 2011), oversaturated salt solution (Gorgoglione et al., 2013) and/or kept frozen at −80°C prior to extraction and qPCR analysis (Bettge, Segner, et al., 2009; Seidlova et al., 2021; Strepparava et al., 2018). qPCR analysis has even been able to detect the parasite in samples that were taken from already dead fish collected from rivers (Hutchins et al., 2021). Post hoc Sanger sequencing of some of the qPCR-positive samples is important to confirm detection of parasitic DNA and may give additional information on the infection strain (Henderson & Okamura, 2004; Hutchins et al., 2021).

When the focus lies on the spread of the parasite rather than on the disease, new highly sensitive environmental DNA (eDNA) protocols are developed (Hutchins et al., 2018a) that can be applied to detect *T. bryosalmonae* and its bryozoan hosts through genetic material from environmental samples such as water. Further research is ongoing using eDNA to monitor the parasite in the field (Fontes, Hartikainen, Holland, et al., 2017). Furthermore, this approach is non-invasive and thus could be applied to monitor the presence and dynamics of the parasite in fragile salmonid populations. A particularly interesting related method is to extract DNA from fish urine (Hedrick et al., 2004; Seidlova et al., 2021). Although this low invasive sample method would fail to detect *T. bryosalmonae* in urine from fish with early infection stages, it would allow to study within-individual disease dynamics and its consequences as resampling of individuals is possible. This non-lethal sampling is particularly promising in combination with upcoming molecular methods to link individual variation in *T. bryosalmonae* infection, survival and PKD status with gene expression (Ahmad, Debes, Nousiainen, et al., 2021).

Although *T. bryosalmonae* infections may be detected in some fish throughout the year, the best period for monitoring PKD is at the end of the warm summer period when ambient temperatures start to drop under 15°C again (in central Europe, the end of summer–early autumn period is at August/September). As due to the high infection success of the parasite (McGurk et al., 2006),

prevalence of *T. bryosalmonae* is usually in the range of 80% to 100% in affected regions (Debes et al., 2017; Ros et al., 2021; Wahli et al., 2002; Wahli et al., 2007), a minimum 10 sampled YOY fish would reliably detect the presence of the parasite (at $N = 10$ the power to detect 1 or more positive fish is higher than 0.9 for a prevalence of 20% or higher). For a more detailed description of severity of PKD and tissue damage, a larger sample of up to 25 fish would be preferable (Carraro et al., 2017; Ros et al., 2021). Macroscopic examination gives a direct estimate of kidney hyperplasia that together with molecular proof of the presence of the parasite informs about the clinical severity of the disease. Histology is necessary to quantitatively and qualitatively evaluate tissue damage. Due to the recent discovery of PKD in natural populations (Wahli et al., 2002), there are no long-lasting time series of PKD yet. Such time series are necessary to evaluate the spread of the disease, its severity and mortality, in relation to changes in environmental conditions.

5.2 | Giving local adaptation a chance

As outlined in sections 2 and 3, genetics clearly plays a role in the susceptibility to *T. bryosalmonae* and in the development of PKD. Brown trout, rainbow trout, grayling, arctic charr and salmon all share a common salmonid ancestry, but they have very different temperature-related PKD outcomes, and are distinctly different in their susceptibility to the transmission of the parasite (see Section 2.3). These species also have different growth and lethal temperature tolerances. For example, YOY grayling can be found at the lower border of the rhithral zone and have been shown to tolerate temperatures about 5°C higher than those tolerated by brown trout (Schubert, 2010). The concordantly different temperature-dependent PKD disease outcomes in different species might thus be the result of the selection pressures on the parasite–host interactions that result from their prevailing thermal environments. There is also increasing evidence that genetic factors play a role in PKD severity of different strains within populations of the same species (Debes et al., 2017; Syrová et al., 2020) and in survival from PKD (Ahmad, Debes, Nousiainen, et al., 2021). Thus, it is important to, at least, decelerate climate change so that natural selection can result in local (climate) adaptations to the parasite and less severe disease outcomes. This would make a strong case for restoring river systems to such a degree that salmonid populations have the possibility to freely pass through the river system (preferably by providing effective up- and downstream fish ladders or by removal of barriers) to be able to perform avoidance migrations and to be able to reach spawning grounds to reproduce naturally and foster such adaptations (Schmidt-Posthaus et al., 2021). Here, a decision should be made whether the beneficial effect of the measure outweighs the potential risk of spreading disease to upstream populations. Such risks in the case of *T. bryosalmonae* will be insignificant when environmental conditions in upstream sections are not favourable for PKD to develop (Schmidt-Posthaus et al., 2021).

Stocking is known to disrupt local co-adaptations, thus cessation or restricting stocking actions should generally be considered when natural reproduction is still intact (Araki et al., 2008; Baer & Brinker, 2008; Carvalho, 1993; Pinter et al., 2019). Fortunately, as adults return to their natal stream for reproduction, the selection for locally adapted survival traits is thought to be relatively strong in salmonid populations (Araki et al., 2008; Carvalho, 1993). The success of cessation of stocking for returning self-reproducing locally adapted salmonid populations is currently being evaluated in a limited number of European streams, one of which is the Wutach, which is a tributary of the Rhine in southern Germany. The results for the Wutach were encouraging, as cessation of stocking in 2000 did not initially lead to a reduction in the trout stocks or local fishery yields (Baer & Brinker, 2010). However, trout numbers have been halved in the last 10 years, an effect that was strongly related to the ongoing rise in temperatures and severity of PKD in this river (Ros et al., 2021). This leads to the question as to whether evolution of local adaptations to counteract temperature-driven PKD (Ahmad, Debes, Nousiainen, et al., 2021) may be fast enough to walk in step with the acceleration of current climate change (Fox et al., 2019). Such an acceleration might result in a mismatch in life history traits that have been locally adapted to a phenomena that is affected by climatic conditions, as found in reproductive mistiming to food availability in temperate zone songbirds (Visser et al., 2004). Therefore, with ambient temperatures playing a dominant role in PKD as is suggested from laboratory experiments (Waldner et al., 2021), then intervention by development of resistant strains and stocking methods might be the only option to render salmonid ecosystems climate resilient under current climate change prognoses (see Section 5.5).

5.3 | Limiting parasite spread

Some studies have reported high variation in parasite load and/or disease severity between individuals within a population (Bruneaux et al., 2017; Dash & Vasemägi, 2014; Debes et al., 2017; Lauringson et al., 2021), and some salmonid habitats were found to be free of the parasite (Feist et al., 2002; Ros et al., 2021; Wahli et al., 2008). As the distribution of the parasite is tightly related to that of its primary bryozoan hosts (Feist et al., 2002; Ros et al., 2021; Wahli et al., 2008), this heterogeneous variation is likely caused by the patchy distribution of bryozoans that are infected with the parasite (Dash & Vasemägi, 2014; Fontes, Hartikainen, Holland, et al., 2017). Understanding the factors that limit growth and distribution of bryozoans may therefore contribute to management of PKD (Carraro et al., 2017; Hartikainen et al., 2009). Some factors that play a role in reducing bryozoan populations are turbulent water flow and low temperatures through summer as both prevent bryozoan populations from establishing and/or growing (Ahmad, Debes, Nousiainen, et al., 2021; Raddum & Johnsen, 1983; Ros et al., 2021; Wahli et al., 2008). Also algae play an important role as these are an important food source for bryozoans (Raddum & Johnsen, 1983; Vohmann et al., 2009). Algae growth may be lowered through decreasing

eutrophication (Hartikainen et al., 2009) and decreasing direct sunlight by providing shading, and this may thus limit *T. bryosalmonae* spore production (Figure 2). Finally, a winter period with close to freezing water temperatures (<5°C) dies out most bryozoan colonies which delays the parasite cycle in the next warm season (Raddum & Johnsen, 1983; Vohmann et al., 2009). This provides an additional reason to decrease thermal pollution as water temperatures elevated to about 10°C may allow bryozoans and *T. bryosalmonae* to survive through winter (Raddum & Johnsen, 1983), which would result in early spore release in the next warm season.

Furthermore, the increase in leisure and sports activities must be taken into account as possible cause for the spread of parasite spores that may travel with infected bryozoans (statoblasts) via wet gear and boats within and between rivers and watersheds (Lamborn & Smith, 2019; Jones et al., 2015). Salmonid populations (and probably the bryozoan populations as well) might be particularly susceptible to the parasite when they come into contact for the first time. When such a salmonid population is stressed as a result of increased water temperatures, low water or wastewater pollution, infections with the parasite could lead to severe PKD-associated pathology, resulting in massive fish kills (Gorgoglione et al., 2016; Hutchins et al., 2019; Lamborn & Smith, 2019; Unfer et al., 2015). For example, during a recent fish kill that was attributed to PKD, a large section of a river in the USA was precautionary closed for the public out of fear of further spread of *T. bryosalmonae* (Hutchins et al., 2019). As a precautionary measure and to prevent costly closures of rivers (Hutchins et al., 2021), it is advisable to raise awareness about protection measures and cleaning wet gear to limit the spread of parasites when travelling along and between waters.

5.4 | Reducing effects of climate change on rivers

River temperatures are buffered in comparison to atmospheric conditions (air temperature) as a function of the capacity of the running water to store heat (flow, volume of water, etc.), topography of the river (orientation, shading, etc.) and groundwater input (Caissie, 2006). Still, on average, water temperatures seem to follow the ongoing warming trend in ambient temperatures as a result of climate change (Caissie, 2006; Masson-Delmotte et al., 2019). In central Europe, water temperatures have increased by 1 to 2°C compared to the long-term average (Daufresne & Boët, 2007; Kędra, 2020; Michel et al., 2020; Webb & Nobilis, 2007), and a further 1 to 3°C could be expected in the 21st century (Masson-Delmotte et al., 2019). Following a likely climate change trajectory as already set by humanity (ranging between RCP 4.5 and RCP 8.5, Masson-Delmotte et al., 2019; Sherwood et al., 2020; Wunderling et al., 2021), it should be considered that by mid-century many of the current salmonid habitats, and even those at higher elevations, are likely to end up unsuitable for salmonids (Almodóvar et al., 2012; Ros et al., 2021). Therefore, management of the river climate regime will be necessary to restore or maintain suitable PKD-free rhithral habitats for salmonids (Figure 5).

Water discharge and groundwater input buffer the effects of ambient climatic conditions on river temperatures (Caissie, 2006; Dawson & Kern-Hansen, 1979; Garcia de Leaniz, 2008; Larned et al., 2010; Mack et al., 2019; Palmer et al., 2009). These factors are increasingly threatened through water abstraction for households, forestry and agriculture, especially during hot periods (Larned et al., 2010). Heat exchange with the ambient climate is further increased through impediment of the flow of the river through dams and weirs (Caissie, 2006; Larned et al., 2010). Temperatures of water below dams have been shown to increase with more than 5°C as discharged water often originates from the hotter surface (eplimnion) of the reservoir (Zaidel et al., 2021). As a mitigating measure, especially in the case of deeper reservoirs, structures could be installed that would discharge water from the deep water (hypolimnion) which then could cool downstream waters (Sinokrot et al., 1995). There is a growing awareness that the consequences for riverine ecosystems and fisheries should be taken into account in projects using water for agricultural, household and energy production. Recycling of water and the implementation of alternative energy sources may alleviate the pressure on river ecosystems (Figure 5). This should also include measures to restore lateral and longitudinal connectivity within streams and between streams and tributaries (Garcia de Leaniz, 2008; Larned et al., 2010; Palmer et al., 2009; Schmidt-Posthaus et al., 2021). Loss of salmonid habitat due to increased temperatures at lower elevations may then be compensated for by improving the opportunity for salmonids to migrate and reach river sections at higher elevations where they may still find cool and PKD-free suitable habitat (Borgwardt et al., 2020; Ros et al., 2021; Wahli et al., 2008). As this restores the original migration patterns of salmonid populations, it would not affect the natural distribution of *T. bryosalmonae*.

For small rivers, providing riparian vegetation (Figure 3a vs. Figure 3b,c; Figure 5) is an important mitigating factor in decreasing PKD. Shading from such strips may change daytime temperature maxima with a decrease of 3 to 8°C (Caissie, 2006; Dugdale et al., 2019). In general, a 12-m-wide riparian buffer with tall and dense trees may provide up to 80% shading, where trees on the north (sun) side of a stream are most efficient (DeWalle, 2010). Although the effect strongly increases with the width of riparian buffers, planting a single line of trees may block 20 to 40% of sunshine (DeWalle, 2010). Moreover, riparian strips may act as a buffer zone for drainage of pollution and organic compounds from human activity such as agriculture, and this might have a positive effect on fish health via controlling primary production and thereby the food for bryozoans (Hartikainen et al., 2009; Raddum & Johnsen, 1983). Providing riparian strips might become an increasingly important mitigating measure because many countries have already mandatory and voluntarily regulations that could be further implemented (Dworak et al., 2009). In practice, the fact that many rivers have undergone severe modifications (deforestation and implementation of waterworks, dams and weirs) may be seen as a benefit as this offers a large potential for corrective management to improve river climate regimes and connectivity (Carvalho et al., 2019; Szałkiewicz et al., 2018).

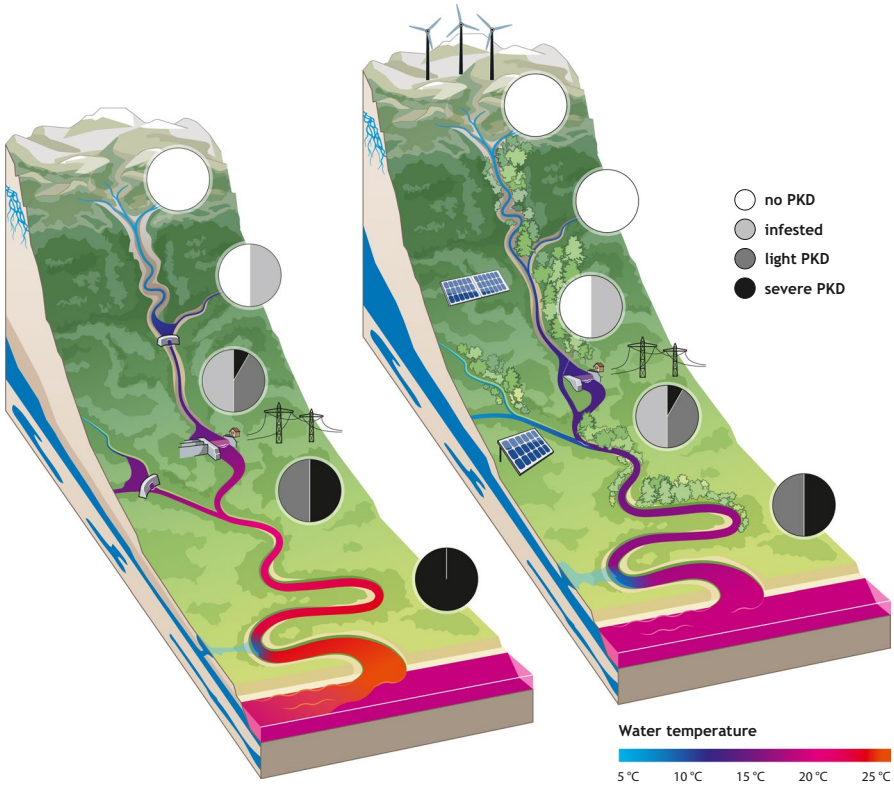

FIGURE 5 Active river management can mitigate climate change effects of PKD on salmonids. Panel left: Many rivers are sensitive to changes in climate due to canalization, intensive water management for human use (agriculture and cities) or weirs and barriers that slow down water flow. Panel right: To prepare for a warmer climate, in addition to the necessity to invest in alternative energy sources (including biomass, wind, solar and water plants), measures are necessary that have to make rivers more resilient. For example, riparian shading may be increased which may be facilitated by existing regulations that protect a margin around rivers from agricultural or other use. Natural water flow may be re-achieved by removing barriers and weirs. Conservation of groundwater is important especially in period of extreme heat as this effectively cools down the river and provides a buffer for water flow during periods of drought. Restoration of lateral and horizontal connectivity allows fish to reach cooler stretches of the river and reach spawning grounds. Finally, flood plains may protect river ecosystems against extreme peaks in water flow. The pie graphs provide a pictorial representation of the severity of PKD at the nearby river section (see legend)

5.5 | Responding to PKD in a 'worst case scenario'

There is a genuine concern that humanity might not be able to curb greenhouse gas emissions enough to prevent river climates from undergoing severe shifts through global warming (Lenton et al., 2019). Out of precautionary principle, it is therefore necessary to develop measures that mitigate the adverse effects of temperature-dependent PKD on salmonid populations. It has been estimated, for example, for central European brown trout populations (Figure 1) that the combined effects of climate change and PKD may result in a loss of more than 80% of their current suitable areas (Ros et al., 2021). With limited possibilities to migrate and taking into account that many spawning grounds might be lost within streams, it is not unlikely that many salmonid populations might collapse in areas where they currently are common. In fact, and as discussed above, many PKD affected European salmonid populations of running waters already are in strong decline (Ros et al., 2021; Wahli et al., 2002; Waldner et al., 2020). To be better prepared for the even tougher future challenges for salmonids, there is therefore an urgent need to carry out research into mechanisms underlying the large phenotypic variation in susceptibility to *T. bryosalmonae* between and within species.

Stocking rivers and streams with salmonid brood is commonplace practice in many European and American salmonid populations (Arlinghaus et al., 2002; Cucherousset et al., 2021; Fraser, 2008). The method has persisted even though a growing body of research publications warn for possible negative consequences of stocking on the loss of locally adapted genetic strains and point out that stocked fingerlings have lower survival than natural brood stock (Baer et al., 2007; Baer & Rösch, 2008; Derry et al., 2019; Fraser, 2008; Harbicht et al., 2014). However, when spawning grounds in a river system are lost or non-reachable or when adverse environmental conditions decimate the population, stocking remains the method of choice and often the only option left to maintain or restore a salmonid population (Fraser, 2008). For example, in an extreme case, such as the total disappearance of Atlantic salmon through pollution of the central European River Rhine system, sanitation measures together with restocking have proved a successful strategy (Monnerjahn, 2011). With the current understanding of the importance to maintain genetic biodiversity and local adaptations

in populations, current attitudes have shifted towards using local strains (Baer & Brinker, 2008). However, relying only on local strains for stocking might not be sufficient when young fish undergo massive mortality through high temperature and PKD-related mortality. Therefore, there is a need to further modify the stocking methods to correct for widespread losses in salmonid populations.

When climate change effects are great enough to compromise reproduction or recruitment of locally adapted salmonid strains, an important option would be to develop brood stock that is resistant to *T. bryosalmonae* infections. This can be done by exposing local salmonid strains to the local parasite strains; by developing a vaccination programme against PKD and by artificial selection of strains of the local salmonid species that are more tolerant to the parasites and to the more extreme climate.

5.5.1 | Proliferative kidney disease-resistant stocks

In order to decrease PKD mortality, it has been proposed to delay stocking till late in the season (Syrová et al., 2020). The rationale for this delay is that late in the season, *T. bryosalmonae* malacospores should still be abundant enough to infect the stocked fish, but that parasite proliferation in the kidney will not develop into severe PKD as temperatures will soon drop during the autumn/winter period (Ferguson & Ball, 1979; Foott & Hedrick, 1987). Such fish will then be more resistant to parasitic infection in the next 'PKD-season'. Although the method has been successfully deployed in aquaculture systems (Clifton-Hadley, Richards, et al., 1986; Ferguson, 1981; Schlotfeldt, 1983), the success of this method on stocking survival in the field has not been proven (Syrová et al., 2020). An additional problem is that the method is particularly sensitive to the timing of stocking. Stocking early in the PKD season (i.e. spring to summer) leaves fish vulnerable to develop severe PKD before the winter starts, while late in the season malacospore numbers decrease leaving the possibility that a large part of the stock fails exposure and thus do not develop immunity or tolerance to the parasite. These issues are additive to common problems in stocking campaigns that may decrease survival of salmonids after stocking and should be addressed to turn the tool successful for the survival of local salmonid populations (Baer & Brinker, 2008; Fraser, 2008; Kennedy et al., 1984).

A promising variant of the 'late season stocking' method would be to carry out controlled malacospore exposure during raising of the stock. Bryozoans infected with *T. bryosalmonae* may be obtained from the local sites where the stock will be released. By mechanically fracturing their tissue, the malacospores are released and will be infectious for a short period (Strepparava et al., 2018). At temperatures that are kept below 15°C, brown trout exposed with such a parasite preparation will show proliferation of the parasite in the kidney, build up resistance, but will not develop PKD (Bailey et al., 2021; Strepparava et al., 2020). Such a 'pre-exposure' method could maintain local varieties of the salmonids when used as stock and should be carried out with the local parasite strain as resistant fish may continue to carry and spread *T. bryosalmonae* (Abd-Elfattah, Kumar, et al., 2014; Soliman et al., 2018; Strepparava et al., 2018). The method would produce resistant stock that can be released, independent of ambient malacospore levels, when stocking conditions are favourable, for example, during periods with no expected significant rain events. Also, it would be possible to maintain the resistant stock over winter and release them in the spring when food availability and thereby the chances for the brood to survive increase. These methods are currently being tested in laboratory and field trials (A. Ros and A. Brinker, pers. communication). They could facilitate further adaptation of these strains to changing climate conditions. More importantly, they would permit locally adapted strains to survive the negative population effects of PKD until local and/or global methods to mitigate climate change are successfully implemented.

An alternative method to achieve a PKD-resistant stock would be through the development of a vaccine against *T. bryosalmonae*. Although such a vaccine is not currently available, recent advance has been made in its development (Faber et al., 2019). Once available, vaccinations could be administered to brood captured from upstream breeding grounds adjacent to PKD risk areas, or to cultured salmonids that will later be released in the wild. Both vaccinated and pre-exposure-treated fish would also benefit when dispersing to downstream areas where they would be exposed to higher levels of malacospores. Migrating species like salmon and the lake trout form of the brown trout could especially benefit from such campaigns, as both migrate from cold mountainous headwaters through lower rhithral and summer warm potamal streams where PKD is often abundant (Ros et al., 2021; Wahli et al., 2008).

5.5.2 | Temperature resilient stocks

A different avenue for developing PKD-resistant stocks is artificial selection of strains that would tolerate or are resistant to *T. bryosalmonae* at higher temperatures than the current strains. Variations in susceptibility or tolerance to *T. bryosalmonae* have been found between families in a natural population of brown trout (Debes et al., 2017) and this could be a starting point for selection programmes. Research using artificial selection of rainbow trout strains has shown potential in terms of developing PKD-resistant strains (Butterfield, 2007) but has not yet led to a reviewed scientific publication. Furthermore, although variation between aquaculture strains of rainbow trout exists, the average level of PKD resistance of different rainbow trout strains did not show significant differences (El-Matbouli et al., 2009; Grabner & El-Matbouli, 2009). It would also be important to study how PKD resistance is achieved in populations that live already in warmer climates at their thermal limits. However, possible target populations of Northern African or Mediterranean trout species and lineages do currently inhabit isolated mountainous head waters and these are under threat of extinction through stocking with non-native rainbow trout and climate change (Dauwalter et al., 2020; Lobón-Cerviá & Mortensen, 2005). Furthermore, no data exist on whether such native 'southern' salmonid populations

are factually in contact with *T. bryosalmonae* and more resilient to PKD than 'northern' salmonids. The most southern findings of PKD are from rainbow and brown trout farms in the North of Italy (*Salmo trutta marmoratus*: Beraldo et al., 2006; *Oncorhynchus mykiss*: Henderson & Okamura, 2004; Tops et al., 2005) in Europe and in Sacramento, California, in Northern America (*Oncorhynchus mykiss*: Hedrick et al., 2004). These farms do report mortality associated with PKD (Beraldo et al., 2006; Henderson & Okamura, 2004). So, until now, no clear picture can be drawn about which native PKD-resistant trout populations could be candidates to show climate change-related migration (northwards or to higher altitudes) or could be assisted in this migration.

5.6 | Management and prevention of proliferative kidney disease in aquaculture

Early descriptions of kidney hyperplasia suggest that PKD has been a returning and deadly disease in aquaculture at least since the beginning of the last century (Plehn, 1924; Schäperclaus, 1954). The disease became a problem for rainbow trout farming at the end of the century where after a large research effort the parasitic cause and possible cures were finally discovered (Clifton-Hadley, Richards, et al., 1986; Feist, 2002; Quigley & McArdle, 1998; Schlotfeldt, 1983). As management options for fish farms with PKD outbreaks most of the following solutions were accurately proposed by Clifton-Hadley et al. (1984):

1. To date there are no licensed and safe chemical treatments against PKD. A positive effect of treatment with salt water on PKD-related mortality was found in 0+ salmon (O'Hara, 1985) and rainbow trout (Enevova et al., 2018). Nevertheless, this effect was not found in Chinook salmon (Hedrick & Aronstien, 1987), and the salt treatment might result in additional stress to the fish. The best options to date, and in general, remains to prevent or decrease the chance of parasitic infections in general by cleaning or replacing water filters and pipes, and to treat inflowing water with ozone or UV (Hendrick et al., 1986; Summerfelt et al., 2008). This situation might, however, change as recent transcriptome studies have identified promising molecular targets (i.e. endoglycoceramidase, legumain-like protease, carbonic anhydrase 2 and pancreatic lipase-related protein 2) that can be used for developing future drugs against *T. bryosalmonae* infections (Ahmad, Debes, Pukk, et al., 2021; Faber et al., 2021).
2. The main solution for farms with mortalities related to PKD has been to raise the fingerlings using cool spring or mountain water (<15°C; De Kinkelin & Loriot, 2001). If larger ponds cannot be provided with cool water, trout should be transferred later in the season so they still get exposed to the parasite but can build resistance in the subsequent cold winter conditions (Clifton-Hadley, Richards, et al., 1986; Ferguson, 1981; Schlotfeldt, 1983). In such a setup, it is essential to monitor malacospore availability in the water (eDNA) and sample fish in autumn to assure that they get infected with the parasite. If exposure fails, the fish will be susceptible to PKD in the next season.
3. When trout acquire the disease, it is beneficial to avoid stressful operations while providing cool, clean and oxygen-rich water. Trout with PKD have been shown to be sensitive to additional stressors like pollution (Bailey, Rubin, et al., 2018; El-Matbouli & Hoffmann, 2002; Rubin et al., 2019) and respiratory distress (see Clifton-Hadley et al., 1984). Furthermore, restricting the diet has been found to decrease mortality in PKD diseased trout (Ferguson & Ball, 1979), which might be related to a positive effect on water quality.
4. Recent developments in transcriptomics greatly increase the possibilities to find specific physiological disease pathways that can be used in developing drugs and vaccines against PKD (Ahmad, Debes, Pukk, et al., 2021; Faber et al., 2019; Faber et al., 2021). Also, the route of treatment should be further developed as vaccination might be costly and impractical for production farms when the fish are fingerlings, still they could be very useful when protecting valuable brood stock against PKD in aquaculture or in the field.
5. Setting up legal frameworks to control or monitor outbreaks of PKD will help to gather information on disease distribution, severity and impact. For example, in the Nordic European countries and Switzerland, trout farms must report PKD outbreaks to the veterinary office (Burkhardt-Holm & Zehnder, 2018; https://infosm.blv.admin.ch/info; Midtlyng et al., 2000).

6 | CONCLUSIONS

The discovery of *T. bryosalmonae* and the high costs that PKD incurred on rainbow trout aquaculture at the end of the 20th century stimulated initial research about the causes of the spread of the parasite. Based on subsequent discoveries of its role in recent declines in salmonid populations together with temperature dependence on the disease outcome, the cause of the perceived increase in PKD was postulated to be a recent change in environmental factors and in particular anthropogenic climate change. The mountainous rhithral streams and rivers where salmonids are naturally abundant are typically cool environments with water temperatures remaining mostly below 15°C in summer. At these low temperatures, *T. bryosalmonae* parasites are able to complete sporogenesis without resulting in severe damage to the kidney causing minimal virulence and mortality to their host. Both experimental and correlational field data support that an increase in water temperatures higher than 15°C will result in severe clinical PKD in American and European salmonids of a wide range of salmonid families like *Oncorhynchus*, *Salmo*, *Salvelinus*, *Prosopium* and *Thymallus*, and may increase fish malacospore production in the brown trout. This severe PKD is characterized by inflammation and necrosis of haematopoietic renal tissue and is followed by high mortalities (Waldner et al., 2021). For salmonids, this means

that disease outcomes, that is, PKD, will get worse in the coming decades without significant measures in place to curb climate change, and to decrease the impact of periods of severe heat and drought on river climate regimes. In the context of the already elevated threats to freshwater ecosystems (Tickner et al., 2020), controlling the virulence of *T. bryosalmonae* must be a priority for safeguarding valuable salmonid stocks. At least under today's climate conditions, salmonids could live with the parasite and its disease without significant negative impacts on local stocks, as long as cool, pristine and oxygen-rich stretches can be maintained in headwater streams and rivers. In the absence of such stretches, PKD will become a significant driver of salmonid population decrease and action is necessary.

Depending on the species of interest and the local conditions, the following measures should be considered:

1. In general, salmonids can live with *T. bryosalmonae* when they have access to cooler waters that remain under 15°C for most of the year (depending on life stage and salmonid species). This is relevant both for the river environment and for aquaculture facilities. Furthermore, cooler and less eutrophic waters limit bryozoan growth and thereby parasite spore production that are infective to salmonids.
2. Connectivity between different sections of rivers and streams should be maintained and when necessary restored, so that suitable habitat at higher elevations is accessible as potential future refuge, both in terms of climate resilience and exposure to PKD.
3. Headwaters with salmonid populations that are *T. bryosalmonae* free should be protected as these will be especially important for spawning ground and survival of young salmonids.
4. Measures should focus on making rivers more resilient to climate change by providing more riparian shading, lowering runoff depositions and maintaining cold water refuges.
5. The prognoses for habitat suitability underline that such conservation efforts to protect and restore salmonid populations, including not only the salmon but also currently abundant species like brown trout and grayling, should include an assessment of regional climate change.
6. As there are no curative treatment options yet, research should reveal mechanisms that favour local stock integrity and resistance to PKD.

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CONFLICT OF INTEREST

The authors have no conflict of interest to declare.

DATA AVAILABILITY STATEMENT

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