

Malacosporean parasites (Myxozoa, Malacosporea) of freshwater bryozoans (Bryozoa, Phylactolaemata): a review

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Abstract: Myxozoans belonging to the recently described class Malacosporea parasitise freshwater bryozoans during at least part of their life cycle. There are two malacosporeans described to date: *Buddenbrockia plumatellae* and *Tetracapsuloides bryosalmonae*, the causative agent of salmonid proliferative kidney disease (PKD). Almost nothing is known about the ecology of malacosporeans and their interactions with bryozoan hosts. Here we review recent advances in our knowledge of malacosporean biology, development and life cycles.

Key words: Malacosporeans, *Tetracapsuloides bryosalmonae*, *Buddenbrockia plumatellae*, Proliferative Kidney Disease.

Introduction

Freshwater bryozoans (Phylum Bryozoa, Class Phylactolaemata) are common, frequently abundant, sessile, colonial invertebrates. Phylactolaemates have a world-wide distribution and are found inhabiting both lotic and lentic environments of varying water quality (JOB 1976; WOOD 2001). Most species of freshwater bryozoans overwinter as dormant, encapsulated, yolk-filled buds, known as statoblasts. These seed-like structures serve as a refuge in both space and time, and are the primary means of bryozoan dispersal between waterbodies.

Despite their ubiquitous distribution and great abundance, freshwater bryozoans remain relatively poorly studied. However, the recent discovery that these organisms are hosts to several myxozoan parasites, one of which is the causative agent of an economically important fish disease, has led to greater research interest in these animals.

The phylum Myxozoa

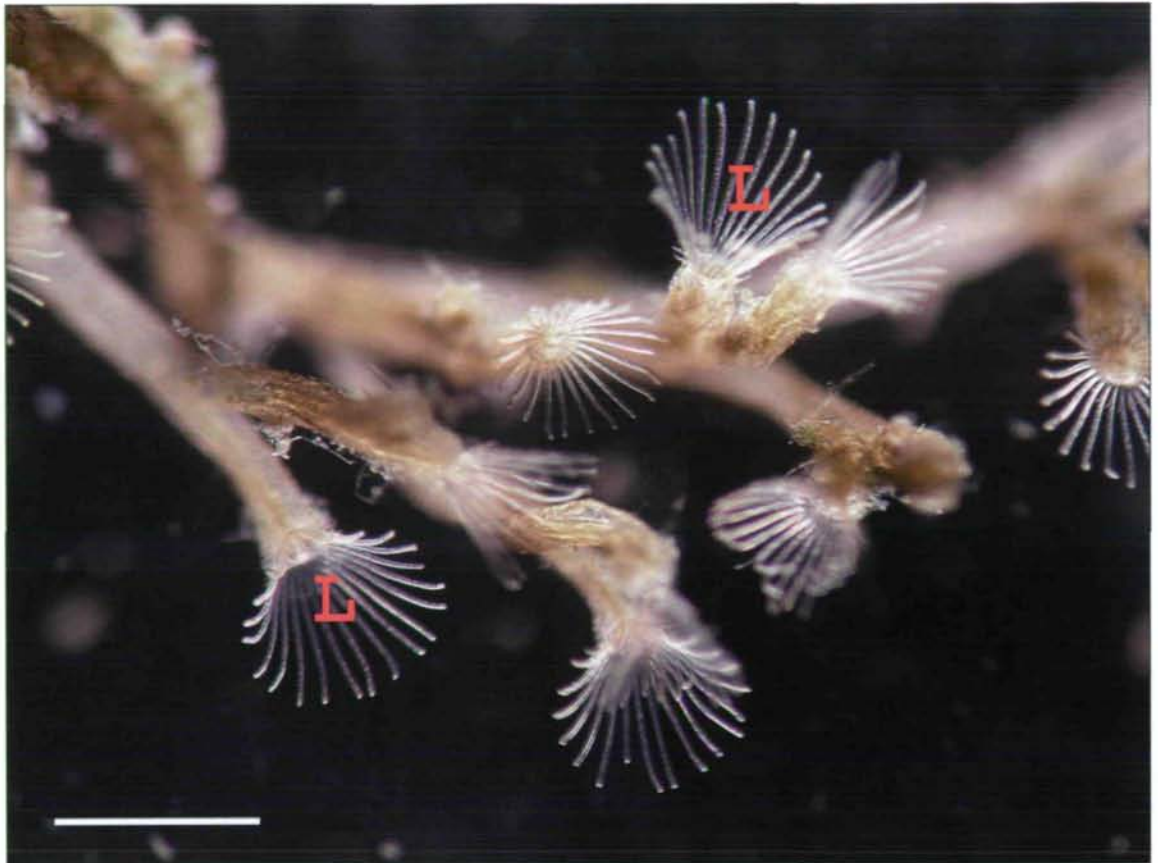
The phylum Myxozoa GRASSÉ 1970 is comprised of microscopic, multicellular, spore-forming endoparasites. Myxozoans are best known as parasites of poikilothermic

vertebrates, primarily freshwater and marine teleosts (LOM 1984), but are also recorded as parasites of amphibians (e.g. UPTON et al. 1992; MCALLISTER et al. 1995), reptiles (see LOM 1990), ducks (LOWENSTINE et al. 2002) and moles (FRIEDRICH et al. 2000). They have even been recorded as persisting within immuno-compromised humans (MONCADA et al. 2001), possibly through ingestion of infected fish. Annelid worms are known to act as hosts in the life cycle of some myxozoans (KENT et al. 2001).

There are currently approximately 1350 described myxozoan species, in 55 genera (KENT et al. 2001). Some of these are the causative agents of economically important fish diseases, however, the majority of species exert innocuous effects on their hosts (LOM 1990). With the global expansion of marine and freshwater fish farming many myxozoan diseases have gained prominence (MOSER & KENT 1994), although the effect of myxozoan diseases on wild fish remains largely unknown.

Myxozoans have been long classed as protozoans, although several independent attempts have been made to reclassify them as metazoans (Štolk 1899; Emery 1909; Ikeda 1912; Weil 1938; cited in CANNING &

Fig. 1: Portion of a colony of *Fredericella sultana*, showing open lophophores (L) of individual zooids; scale bar = 500 μ m.



OKAMURA 2004). These calls for reclassification were primarily based on the multicellular nature of the group and phylogenetic affinities. Although myxozoans were accorded their status as phylum by GRASSÉ 1970, relatively recent publications continue to classify them as protists (BRUSCA & BRUSCA 1990; GILBERT & GRANATH 2003). Compelling evidence that myxozoans should be officially reclassified as a metazoan phylum now exists, again on the basis of the multicellularity of their spores, as well as molecular phylogenetic studies (e.g. SMOTHERS et al. 1994; SIDDALL et al. 1995; KENT et al. 2001, SCHLEGEL et al. 1996; ZRZAVÝ 2001, ZRZAVÝ & HYPŠA 2003; OKAMURA & CANNING 2003, CANNING & OKAMURA 2004). While the metazoan nature of myxozoans is now widely accepted, their phylogenetic status within the Metazoa has been strongly debated (see discussion in section on *Budendbrockia plumatellae*).

The long-standing taxonomic dispute regarding the phylum Myxozoa has not merely been limited to higher-level phylogenetic affinities. Prior to 1994, two classes existed within the group: Myxosporia BUTSCHLI

1881 and Actinosporia NOBLE 1980. These classes were based on differing spore morphology. When actinosporians infecting oligochaete annelids were identified as stages in the life cycle of the myxosporian *Myxobolus cerebralis* (MARKIW & WOLF 1983), the class Actinosporia was suppressed leaving only one class (the Myxosporia) within the phylum (KENT et al. 1994a). To date about 25 life cycles of myxozoans have been resolved and these involve infection of annelid worms and fish hosts (listed in KENT et al. 2001).

The discovery and description of a myxozoan parasite infecting freshwater bryozoans in 1996 (CANNING et al. 1996; OKAMURA 1996) led CANNING et al. (2000) to propose a new class – the Malacosporia – within the phylum Myxozoa. This class incorporates myxozoan parasites which include freshwater bryozoans in at least part of their life cycle, but to date no complete malacosporian life cycle has been resolved.

The class Malacosporia

There is a great paucity of data on malacosporian biology and ecology. One member

of the Malacosporea, *Tetracapsuloides bryosalmonae*, is now recognised as the causative agent of proliferative kidney disease (PKD) in salmonid fish (ANDERSON et al. 1999a, b; CANNING et al. 1999; FEIST et al. 2001).

Malacosporeans form an ancient clade of myxozoan parasites, which on the basis of 18S rDNA sequences, appear to have diverged early in the evolution of the Myxozoa (ANDERSON et al. 1999a, b; KENT et al. 1998, 2001). Two species of malacosporean have so far been described. One is *Buddenbrockia plumatellae* SCHRÖDER 1910 (formerly *Tetracapsula bryozoides*; cp. CANNING et al. 2002) and the other is *Tetracapsuloides bryosalmonae* (formerly *Tetracapsula bryosalmonae*; cp. CANNING et al. 2002). Despite the relatively recent description of the class, myxozoans appear to have been observed in several early studies of phylactolaemate bryozoans. Thus, malacosporeans appear to be figured in various illustrations (e.g. ALLMAN 1856; COOKE 1906).

There are several distinctive diagnostic features of malacosporeans infecting bryozoan hosts, which indicate that they are not actinosporean stages of the class Myxosporrea (CANNING et al. 2000). The most prominent attribute is the lack of hardened spore valves, a characteristic which led to the naming of the class (CANNING et al. 2000). In addition, spore development occurs within closed sacs or hollow 'worms' encasing spores (CANNING et al. 2000). Since sexual reproduction of malacosporeans has been shown to occur in the bryozoan phase of malacosporean life cycles, bryozoans are considered as the true (definitive) hosts of these enigmatic parasites (CANNING et al. 2000). For a comprehensive review see CANNING & OKAMURA (2004). Although there are obvious similarities between the two malacosporean species, they differ in their spore development, ecology and possibly also their life histories. Therefore, they will be discussed separately in the following sections.

Buddenbrockia plumatellae

The first record of *Buddenbrockia plumatellae* is that by DU MORTIER & VAN BENEDEEN (1850), reporting worm-like (vermiform) creatures, about 0.1 mm long and filled with

Tab. 1: Species of bryozoans and fish, which have been identified as hosts to species of malacosporean.

<i>Buddenbrockia plumatellae</i>	<i>Tetracapsuloides bryosalmonae</i>	
Bryozoan hosts	Bryozoan hosts	Fish hosts
<i>Lophopodella carterii</i>	<i>Pectinatella magnifica</i>	<i>Oncorhynchus mykiss</i>
<i>Hyalinella punctata</i>	<i>Fredericella sultana</i>	<i>Salmo trutta</i>
<i>Stolella evelinae</i>	<i>Plumatella rugosa</i>	<i>Salmo salar</i>
<i>Plumatella repens</i>	<i>Plumatella emarginata</i>	<i>Oncorhynchus tshawytscha</i>
<i>Cristatella mucedo*</i>	<i>Cristatella mucedo</i>	<i>Oncorhynchus kisutch</i>
<i>Plumatella fungosa</i>		<i>Oncorhynchus clarki</i>
		<i>Rutilus rutilus</i>
		<i>Salvelinus alpinus</i>
		<i>Thymallus thymallus</i>
		<i>Esox lucius</i>
* sac-like stages only		

cells, moving in the coelom of a bryozoan in Belgium. Studies conducted on bryozoans infected with *B. plumatellae* in Germany led SCHRÖDER (1910) to name the species. He initially proposed that these 'worms' were mesozoans, but later revised his opinion suggesting a nematode affinity, due to the presence of the four blocks of longitudinal muscles characteristic of this species (SCHRÖDER 1912). The phylogenetic placement of *Buddenbrockia* remained obscure, and, until recently, it had never been assigned to an animal phylum, nor had a monotypic phylum been erected for it (MONTEIRO et al. 2002). In fact, it was included as one of the 'five enigmatic taxa' by NIELSEN (2001) in his text on animal evolution.

Since the early studies conducted by SCHRÖDER, *B. plumatellae* has been encountered parasitising several bryozoan genera (Tab. 1) across a broad geographic distribution (OKAMURA et al. 2002). In spite of this, the vermiform parasite is still rarely encountered. As such, little is known regarding the ecology and life cycle of this malacosporean.

There has been a resurgence in interest in *B. plumatellae* in recent years. Molecular and ultrastructural evidence suggests that *B. plumatellae* occurs as both worm-like and sac-like stages within bryozoan hosts as is shown in Figure 2 (MONTEIRO et al. 2002; OKAMURA et al. 2002). Both stages produce infective spores, but the longitudinal muscles are absent in sacs (OKAMURA et al. 2002; CANNING et al. 2002). The sac-like stages were initially described as *Tetracapsula bryozoides* (CANNING et al. 1996), but 18S rDNA sequences and ultrastructural similarities, including spore morphogenesis and polar capsule structure, resulted in the sac-like *T. bryozoides* being placed in synonymy

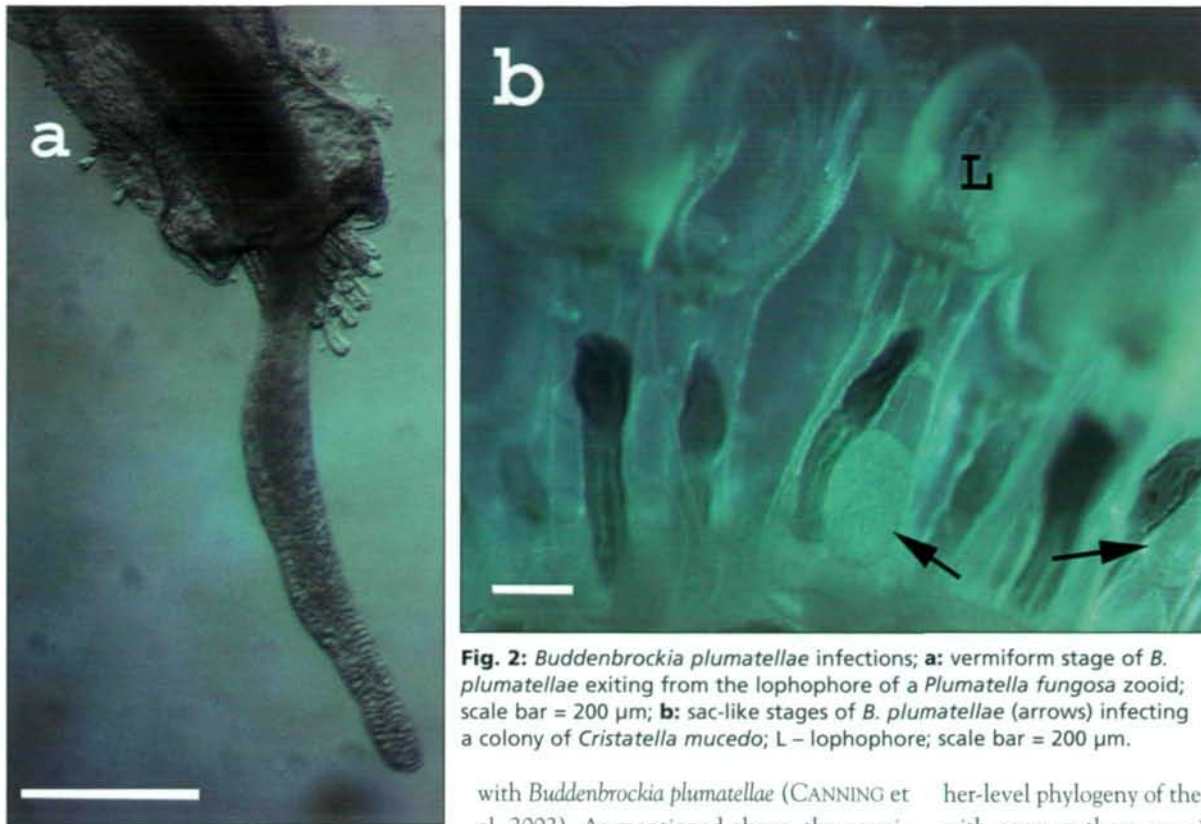


Fig. 2: *Buddenbrockia plumatellae* infections; **a:** vermiform stage of *B. plumatellae* exiting from the lophophore of a *Plumatella fungosa* zooid; scale bar = 200 μ m; **b:** sac-like stages of *B. plumatellae* (arrows) infecting a colony of *Cristatella mucedo*; L – lophophore; scale bar = 200 μ m.

with *Buddenbrockia plumatellae* (CANNING et al. 2002). As mentioned above, the vermiform stage of *B. plumatellae* has been found infecting several bryozoan species (CANNING et al. 2002; MONTEIRO et al. 2002), but it is interesting to note that the sac-like stages have only been observed parasitizing one bryozoan host – *Cristatella mucedo*.

The discovery that *B. plumatellae* is a myxozoan, has generated debate over the phylogenetic affinities of the Myxozoa. Its body plan with four longitudinal muscle blocks and a lack of gut lends support to a bilaterian placement of this phylum. Loss of the gut is a feature of other lower metazoan organisms which have become parasitic, such as the Rhombozoa, Orthonectida and some species of Nematoda (OKAMURA & CANNING 2003). A triploblastic nature for myxozoans was previously suggested on the basis of 18S rDNA phylogenies (e.g. SMOTHERS et al. 1994; HANELT et al. 1996; SCHLEGEL et al. 1996; KIM et al. 1999) and the presence of central class Hox genes (ANDERSON et al. 1998). Support is therefore mounting for placing the Myxozoa within the Bilateria, with myxozoans showing an extreme secondary reduction in body plan, due to their parasitic life-style (OKAMURA et al. 2002). However, the debate over the high-

er-level phylogeny of the Myxozoa persists, with some authors remaining steadfast in the belief that myxozoans are degenerate cnidarians (SIDALL et al. 1995; ZRZAVÝ et al. 1998; KENT et al. 2001).

Tetracapsuloides bryosalmonae

PKD has been recognised as a serious salmonid fish disease since the early part of the 20th century, but the etiological agent had never been identified and had consequently been referred to as PKX ('X' implying organism unknown) (SEAGRAVE et al. 1980). The myxozoan traits of PKX were recognised by KENT & HEDRICK (1986). The discovery of an alternate worm host in the life cycle of the myxozoan *Myxobolus cerebralis* (MARKIW & WOLF 1983, WOLF & MARKIW 1984) prompted research to identify an invertebrate host for PKX (LONGSHAW & FEIST 2000, LONGSHAW & FEIST, unpublished data cited in FEIST et al. 2001; MORRIS D.J. et al. 1999). These searches were unsuccessful until molecular data identified freshwater bryozoans as hosts of PKX (ANDERSON et al. 1999a, b), which finally allowed the species to be described as *Tetracapsula bryosalmonae* (CANNING et al. 2000). Confirmation that *T. bryosalmonae* is the causative agent of PKD was subsequently

obtained by transmission studies (FEIST et al. 2001). Formerly named *Tetracapsula bryosalmonae*, this species was renamed when *T. bryozoides* was placed in synonymy with *Buddenbrockia plumatellae* (see MONTEIRO et al. 2002) and the new generic name of *Tetracapsuloides* was proposed for *Tetracapsula bryosalmonae* (see CANNING et al. 2002).

Most salmonid fish species and many species of freshwater bryozoan are susceptible to infection by *Tetracapsuloides bryosalmonae* (Tab. 1). Interestingly, pike (*Esox lucius*), which is not a salmonid fish species, is also capable of contracting PKD. However, pike are found within the same clade as salmonids (NELSON 1994; BERRA 2001).

While five species of bryozoan have been confirmed as hosts of *T. bryosalmonae* (Tab. 1) on the basis of 18S rDNA sequence information, only one species (*Fredericella sultana*; Fig. 1) has been confirmed through transmission studies (FEIST et al. 2001). It remains unclear whether all freshwater bryozoan species are susceptible to *T. bryosalmonae* infection. On the basis of a survey conducted in parts of Europe and North America, OKAMURA & WOOD (2002) concluded that members of the genera *Fredericella* and *Plumatella* are the most important hosts of *Tetracapsuloides bryosalmonae*, since they were most commonly associated with fish farms that sustained outbreaks of PKD.

Importance and distribution of proliferative kidney disease (PKD)

A comprehensive review of PKD was presented by HEDRICK et al. (1993). The disease was first recorded from Germany as 'Amöbeninfektion der Niere' by Plehn in 1924 (cited in HEDRICK et al. 1993). The distribution of PKD is limited to the northern hemisphere where it is found in many countries in Europe and mainly in the western states of North America (KENT & HEDRICK 1986). Due to the great economic losses to aquaculture industries, PKD has been identified as one of the most economically important diseases affecting cultured salmonids fisheries in Europe, including the U. K. (CLIFTON-HADLEY et al. 1984), and in

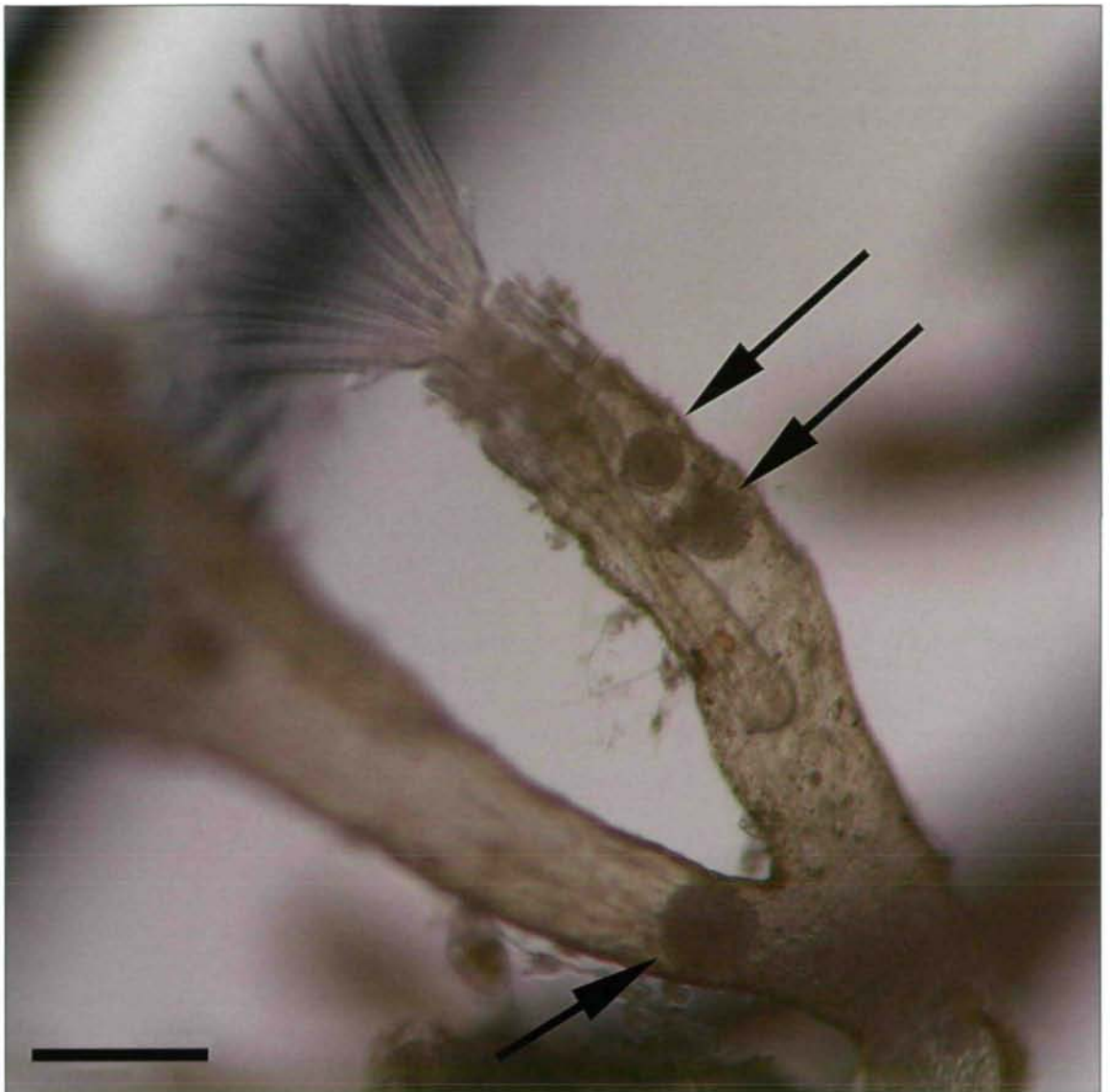
North America (KENT et al. 1994b). Economic costs to the U. K. aquaculture industry have been put at £1.8 million annually, as a result of stock losses, decreased food conversion rates and increased labour costs (MONTGOMERY 2000). In intensive farming situations, mortalities of up to 95-100 % of stock have been recorded (HEDRICK et al. 1993) and, even when mortality is low, morbidity can reach 100 % (CLIFTON-HADLEY et al. 1984). Wild salmonids are known to be susceptible to the disease, but there is a paucity of data on its impact on wild stocks (BUCKE et al. 1991; FEIST & BUCKE 1993, FEIST 1999, FEIST et al. 2002). It has been suggested that recent declines in brown trout populations in Switzerland may be due to PKD (WAHLI et al. 2002).

PKD primarily affects underyearlings (KENT & HEDRICK 1985) and an acquired immunity follows infection (FERGUSON & BALL 1979; FOOT & HEDRICK 1987). Natural outbreaks of the disease seldom occur at water temperatures below 15 °C (FERGUSON 1981), which lend a highly seasonal aspect to the disease (FERGUSON & BALL 1979). The seasonality is thought to relate to the immune system of fish, rather than the availability of infective spores in the water, since the presence of infective *T. bryosalmonae* spores capable of inducing PKD in rainbow trout has been shown to persist throughout the year (GAY et al. 2001). However, the biology of freshwater bryozoans is also characterised by extreme seasonal growth and proliferation (BUSHNELL 1966).

Malacosporean life cycles

The fact that phylactolaemate bryozoans are the source for the causative agent of PKD in salmonids is now well established (ANDERSON et al. 1999a, b; FEIST et al. 2001). However, whether stages that develop in and are released by fish are capable of infecting bryozoans has so far not been demonstrated. TOPS et al. (2004) undertook extensive transmission studies to elucidate the life cycle of malacosporeans. In particular, they investigated transmission of the parasite from salmonids to bryozoans. None of the 15 transmission trials were successful, indicating that there may be another host in the life cycle of malacosporean parasites.

Fig. 3: Sacs of *Tetracapsuloides bryosalmonae* (arrows) infecting a branch of *Fredericella sultana*; scale bar = 200 μ m.



MORRIS D.C. et al. (2002) and MORRIS D.J. et al. (2002) reported possible transmission of *T. bryosalmonae* and *Buddenbrockia plumatellae* from fish to bryozoans. The studies were uncontrolled. Their results may have reflected previous development of the parasite in bryozoans in the field or PCR amplification of residual DNA that had adhered to bryozoans or of undetected, infected invertebrates. It is, however, known that PKD is not transmitted from fish to fish (FERGUSON & BALL 1979; D'SILVA et al. 1984).

Certain observations have been cited as evidence that salmonids may be aberrant hosts for *Tetracapsuloides bryosalmonae*. These include the severe inflammatory response of fish kidneys, as well as apparently incomplete spore development within salmonid

hosts (MACCONNELL et al. 1989). However, previous to the description of the Class Malacosporea (CANNING et al. 2000), researchers expected final spore development to terminate in stages with hardened valves consistent with myxosporeans. The possibility that fish are not aberrant hosts is supported by the release of apparently functional malacosporean-like spores in fish urine (HEDRICK et al. 2004). However, the presence of *T. bryosalmonae* has been confirmed from bryozoan populations collected from sites devoid of salmonids (OKAMURA et al. 2001). This suggests that fish may, at best, be facultative hosts. It also suggests that malacosporeans may be able to exploit the clonal growth of bryozoans and remain as cryptic latent infections in bryozoan populations.

Thus far no vertebrate host has been identified for *Buddenbrockia plumatellae*. Although, organisms with distinctly malacosporean features have been described in pillar cells and endothelial cells of common carp, *Cyprinus carpio* (VORONIN 1993, VORONIN & CHERNYSHEVA 1993), the precise nature of these stages is yet to be identified.

Development of malacosporean parasites infecting bryozoan hosts

The development of sacs and spores of both malacosporean species in bryozoans have been studied through ultrastructure (CANNING et al. 1996, 2000, 2002; OKAMURA et al. 2002). Mature infections manifest themselves as spore-filled sacs. The sac-like forms tumble within the coelom of the bryozoans, moving with the coelomic fluid. The vermiform stages of *Buddenbrockia* are capable of independent movement due to the possession of 4 longitudinal muscle groups. Recently, there has been an increase in our knowledge of the early development of malacosporeans infecting their invertebrate hosts.

Molecular, histological and ultrastructural investigations have revealed that cryptic stages in the body wall are a feature of malacosporean infections of bryozoans (CANNING et al. 2002; TOPS & OKAMURA 2003). Cryptic stages have similarly been identified from myxosporean parasites infecting tubificid worms (*Tubifex tubifex*). The discovery of these stages suggests that infections of bryozoans may be able to persist within bryozoan populations as cryptic, latent infections, only proliferating into spore-filled sacs when the conditions are appropriate.

The inclusion of a latent period in the life cycle of malacosporeans could play a similar role to the inclusion of dormant, resistant stages in other pathogens. Such resistant stages include occlusion bodies in the case of some baculoviruses (BURDEN et al. 2003) and hardened spores as in the myxosporean stages of myxozoans (CANNING & OKAMURA 2004). These stages allow for the long-term persistence of pathogens by providing environmental reservoirs of infection (BURDEN et al. 2003). Perhaps long-term cryptic infec-

tions of malacosporeans in bryozoan hosts allow for a long-term persistence of the parasites within the environment.

The long-term persistence of infections could explain many aspects of the *Tetracapsuloides bryosalmonae* life cycle, especially when infecting *Fredericella sultana* (see Fig. 3). This species of bryozoan can overwinter as live colonies and may thus provide a winter refuge for cryptic parasitic stages. Notably, a survey of bryozoan species associated with water courses enzootic for PKD over a wide geographic range, identified *F. sultana* as one of the most commonly found species of bryozoan to be collected in PKD areas (OKAMURA & WOOD 2002). Overwintering in the widely available live *F. sultana* colonies would not only allow the persistence of infections through unfavourable periods, but also provide an explanation for the persistence of annual outbreaks of PKD infections in consecutive years at some fish farms. Long-term cryptic infections in *F. sultana* would also provide a means of dispersal for the malacosporean. *Fredericella sultana* colonies become brittle as they grow and branches detach, drift downstream and reattach elsewhere (WOOD 1973). This fragmentation strategy could allow for cryptic malacosporean stages to be transported to new habitats within their host.

Since malacosporeans do not produce hardened, resistant spores there has been much speculation regarding what happens to the parasites during the winter months, since most bryozoan species regress and overwinter as dormant statoblasts. One possibility is that malacosporeans are capable of overwintering as cryptic stages in statoblasts (OKAMURA & WOOD 2002). This strategy would not only allow for the survival through periods which are not permissive for host growth, but would also provide a means of dispersal of the parasite, since statoblasts are the dispersal stage of bryozoans. There is a growing body of evidence that waterfowl act as dispersal vectors of bryozoan statoblasts, which lends credence to this possibility. Evidence includes ongoing gene flow amongst sites traversed by migratory waterfowl (FREELAND et al. 2000), the presence of intact statoblasts in waterfowl digestive tracts and faeces (FIGUEROLA et al.

2003) and the viability of some statoblasts, which hatched following ingestion and excretion by waterfowl (CHARALAMBIDOU et al. 2003). GILBERT & GRANATH (2003) have similarly speculated that the myxozoan *Myxobolus cerebralis* may undergo dispersal thorough the dormant cysts of *Tubifex tubifex*.

It can be predicted that the life history of bryozoans plays an important role in the maintenance and spread of cryptic malacosporean infections without the necessity of regular transmissions from any other hosts. Should malacosporeans possess the ability to proliferate as the host colony grew, they would be able to ensure that all regions of colonies became infected. In addition, *Fredericella sultana* itself overwinters as live colonies (WOOD 1973; RADDUM & JOHNSEN 1983), providing the parasite with a winter-refuge, thus allowing cryptic infections to be maintained year-round in this species (GAY et al. 2001). Finally, should cryptic stages infect dormant bryozoan statoblasts, infections could be passed to new colonies through hatching from statoblasts. All of these processes could promote long-term infections in bryozoan populations without the need for regular transmissions from any other hosts. This could explain the high prevalence of mature *T. bryosalmonae*-infections in bryozoan populations early in the growing season (LONGSHAW et al. 1999; Tops & Okamura unpubl. data).

If cryptic stages of *T. bryosalmonae* are capable of infecting bryozoan statoblasts, then the maintenance of infections in some sites may simply be explained by colonization through infected statoblasts and the subsequent spread of infection in the bryozoan population through proliferation of parasites within clonally reproducing hosts. Furthermore, waterfowl-mediated dispersal of possibly infected statoblasts, followed by the proliferation of parasitism in the bryozoan population provides one explanation for the presence of infected bryozoan populations in sites lacking salmonids (TOPS et al. 2004). It is interesting to note that statoblast production is reportedly reduced, but not precluded in bryozoans with overt malacosporean infections (OKAMURA 1996; MORRIS D.J. et al. 2002; Tops pers. obs.), but

whether it is reduced in bryozoans with cryptic infections is unknown.

Vermiform and sac stages

Molecular and ultrastructural evidence has suggested that sac-like and vermiform stages are common in the life cycle of *Buddenbrockia plumatellae* (MONTEIRO et al. 2002; OKAMURA et al. 2002). Nevertheless, sacs and worms have not been found to coexist in any host. In addition, although numerous bryozoan species are recorded as hosts to the vermiform stage, the sac stage has only been encountered in *Cristatella mucedo* (CANNING & OKAMURA 2004). It is possible that the life cycle of *Buddenbrockia plumatellae* entails obligate cycling between life history stages or that these different morphologies represent facultative stages, the expression of which is controlled by an unidentified cue. Alternatively, they may represent different but closely related species.

Observations have shown that narrow branching species of bryozoans are able to seal off infected portions of colonies, thus limiting the infection to a portion of the bryozoan colony (CANNING et al. 2002; MORRIS D.J. et al. 2002). Vermiform stages, capable of independent movement, may reduce the likelihood of becoming trapped and may promote exit from the host (CANNING et al. 2002, OKAMURA & CANNING 2003).

There has been speculation regarding the possible existence of a vermiform stage of *Tetracapsuloides bryosalmonae* (OKAMURA & CANNING 2003; TOPS et al. 2004). In a study assessing statoblasts collected from areas enzootic for PKD, TATICCHI et al. (2004) observed vermiform stages emerging from KOH treated statoblasts. They hypothesised that these could be worm-like stages of *T. bryosalmonae*, but the study lacked molecular and ultrastructural confirmation. To date there is no evidence for different morphotypes in *T. bryosalmonae*.

Conclusions and future work

As a result of ongoing debate regarding the phylogenetic affinities of the Myxozoa (CANNING et al. 2002; MONTEIRO et al.

2002; OKAMURA et al. 2002) there has been a resurgence of interest in the study of the genus *Buddenbrockia* (MORRIS D.J. et al. 2002; ZRZAVÝ 2001, ZRZAVÝ & HYPŠA 2003). In addition, PKD appears to be increasing in many places (WAHLI et al. 2002). In light of these basic and applied issues, we can expect further work on bryozoans and their parasites.

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