Parasitic Diseases of Trout and Their Controls in Sustaınable Development of Aquculture: Crustaceans

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Abstract: Fin fish the primary source of production for humans in many part of world and this is especially true in most developing countries. Aquaculture is one of the increasingly developing industry. But fish diseases have become increasingly visible during the latest decades in connection with the development of aquacultural industries troughout the world. Diseases problem including hazards caused by parasitic organisms are the main threat to further increase of the industry. The salmon louse *Lepeophtheirus salmonis* is now the main problem in cage-cultured salmon in the marine environment in Scandinavian countries. In recent years however, *Argulus* spp. have been reported to cause problems in UK stillwater trout fisheries. A survey of such fisheries found 29% of them suffered from problem infections by the parasite in the year 2000. *Argulus* spp. were perceived to cause economic losses in infected fisheries through a reduction in the number of anglers due to reduced aesthetic appeal and catchability of fish. *Lernaea* spp. in the eyes of trout cause blindness. The present work aim to the parasitic diseases of freshwater trout, how they are transmitted, which effects they have on trouts, how they could be diagnosed, and how they could be controlled and treated.

Keywords: Trout, disease, parasite, diagnosis, control, treatment

Introduction

Turkey has rich inland water sources, about 200 natural lakes, about 750 artificial lakes or ponds, about 193 reservoirs, 33 rivers and streams of 177.714 km length and 8.333 km of coastal strips. Aquaculture sector in Turkey is new when compared with European countries. The first fish farm was established as a rainbow trout farm in 1970s. The following years, new fish farms have been established year by year. The main fish species cultured in Turkey are Carp (*Cyprinus carpio*), Rainbow trout (*Oncorhynchus mykiss*), Atlantic salmon (*Salmo salar*), Gilthead sea bream (*Sparus aurata*), European sea bass (*Dicentrarchus labrax*), Bluefin tuna (*Thunnus thynnus*), Black sea turbot (*Psetta maxima*), Mediterranean mussel (*Mytilus galloprovincialis*) and Shrimp (*Penaeidae* spp). Aquaculture production of Turkey has grown steadily over the years from 5782 tonnes in 1990 to 63 000 tonnes in 1999 and to 152 186 tonnes in 2008. Recently, it is shown in Table.1 that trout pruducts have reached to 68,649 tons in Turkey (TUIK, 2009).

The intensification of aquaculture and globalization of the seafood trade have led to remarkable development in the aqauculture industry. The industry has been plagued with disease problems caused by viral, bacterial, fungal and parasitic pathogens. In recent years, disease outbreaks are becoming more frequent in he aquaculture and associated morbidity and mortality have caused substantial economic losses. Health problems have two fiscal consequences on the industry: loss of productivity due to animal mortality and morbidity, and loss of trade due to food safety issues. Thus, disease is undoubtedly one of the major constraints to production, profitability and sustainability of the aquaculture industry. Parasites in fish have become increasingly visible during the latest decades in connection with the development of aquacultural industries in the world. Thus, focus has been placed on parasitic infections in these enterprises and their economic and ecological impact.

Type of fish	2004	2005	2006	2007	2008
Inland water					
Trout	43 432	48 033	56 026	58 433	65 928
Marine water					
Trout	1 650	1 249	1 633	2 740	2 721
Total	**45 082**	**49 282**	**57 659**	**61 173**	**68 649**

Table.1. Aquaculture production of Turkey (TUIK, 2009)

Diseases problem caused by parasitic organisms are the main threat to further increase of the industry. There are various parasites caused the diseases on the trout. This study consist of crustaceans parasites. This research presents the individual parasites types producing problems in sea bream and sea bass. Each section is presented with 1. aetiyology, the parasitic organism responsible for the disease, 2. epizootiology, the transmission of the diseases and life cycle of the parasite, 3. pathogenicity, how the parasite produces diseases in the fish, 4. symptoms, clinical signs of the diseases, 5. diagnose, how the infection can be identificated, 6. treatment, how the infection can be controlled.

1. Lernaea spp.

Lernaeosis is caused by parasitic copepods *(Lernaea* spp.) that infect many freshwater fish important to aquacutture and recreation. Lernaea spp or anchor worms are common pests in cyprinids and to a lesse extent of salmonids and other fish. Epizootics in cultured fish are often associated with high mortality. The paraistes also cause problems in man-made lakes (Anon, 1980; Berry et al., 1991) and in commercial aquaria (Shariff et al., 1986; Dempster et al., 1988). The parasite particularly pathogenic to small fish because of their relatively large size.

1.1. Aetiyology: In the genus of *Lernae* of Lernaeidae family, there are over 40 species. The taxonomy is based on the shape of the anchors. *L. cyprinacea* is specific to *Carassius species*. *L. cruciata*, a parasite of Lepomis, Ambloplites, Micropterus and Morone spp. in North America (Kabata, 1988**).** *L. elegans* is found on a range of fishes. *L. cyprinacea* was found on rainbow tout (Berry et al., 1991). The adult female *L. cyprinacea* has a small semispherical cephalothorax which contains the mouth. Behind it is a well-developed holdfast normally consisting of a birfurcate dorsal process and a simple ventral process. The elongate neck and trunk carrythe four pairs of legs of the premetamorphosed female. The abdomen is short (Lester and Roubal, 1995).

1.2. Epizootiology: *L. cyprinacea* requires only one host to complete its life cycle. It has three free-living naupliar stages and five copepodid stages which are usally on the gill and are relatively immobile although they are not permanently attached. The male is fre-moving and the post-metamorphosed female attached. At 27 ºC the larval stages take 12 to 17 days to develop into adult male or premetamorphosed females. Adult male die within 24 hours. Females are fertilized and either attack the same host or swim to another host short (Lester and Roubal, 1995).

Trout in the United States are infected by *L. cyprinacea,* whereas *L. esocina* and *L. minima* are of concern in salmonid fisheries in Europe (Kabata, 1970; Hoffman, 1973). The disease is enzootic in several Utah reservoirs (USA) where fingerling rainbow trout (7 cm long) are stocked, and the fisheryis managed to maximize the return of stocked fish to the angler. On the premise that stocking the best-performing rainbow trout strain might be an economical means of improving the fishery,

Anchorworms were first detected in midsummer each year and were most abundant in the fall. The mean parasite intensity was highest in October 1982 (19 anchorworms per fish); in other years, maximum density was 7 to 9. The dorsal and caudal areas of the fish were the most heavily parasitized.

1.3. Pathogenicity: Fish less than 20 mm long may be killed by the parasites if vital organs are penetrated by the anchors (Khalifa and Post, 1976). Uzmann and Rayner (1958) reported that “heavy” infections of *L. cyprinacea* caused death of yearling rainbow trout *(Oncorhynchus mykiss).* Copepods may open routes for secondary infection; however, quantitative data on these secondary effects are scant (Hoffman, 1976; Dempster et at., 1988).

Moreover, the attached copepods may make the fish undesirable to anglers, thus affecting fishery management goals.

The histological response to parasite attachment included an infectious granuloma similar to that reported in other fish hosts. Bacteria were not found in the kidneys of fish before stocking, but afterward bacteria that were presumptively identified as belonging to the genus *Aeromonas,* were found in the kidneys of up to 45% of the parasitized fish. Most (94%) anglers noticed the anchorworms, but few (8%) discarded parasitized fish. Some 28% used special cleaning techniques to prepare fish but 49% did nothing special to clean them. Lernaeosis probably had little effect on the fishery management goals for the reservoir.
fishing, *Oncorhynchus mykiss,* copepoda.

The mechanical destruction of epidermis and dermis caused by grazing copepodids stimulates inflammation (Shields and Tidd, 1974). When tha parasites abundant on the gills, this presumably precipitates distress trough oedema and slowed blood circulation in the lamellae. Host mortality caused by adult females is generally the result of the physical destruction of tissues. They also induce prssure necrosis and may secrete a histolytic and digestive anzyme (Shields and Goode, 1978). Small fish (4-6 cm) suffer more than large fish because the head of the parasite often enters the body cavity and penetrates the liver, or brain, spinal cord, or other vital organs.

1.4. Symptoms: The bases of the dorsal and anal fins were common attachment sites. Ulcers are commonly observed with and without an attached parasite. The loss of scales and skin resulted in circular ulcers that averaged about 1.4 mm (diameter). Sometimes ulcerations are not observed around some parasites.

The parasite cause disruption and necrosis of gill epithelium and large numbers of larvae on the gills cause the death of fish. The embedded anchors are surrounded by fibrous granulation tissue, and there are considerable leucocytic response below the dermis. Myofibril degeneration and hemorrhage are noted in most sections, but these reactions were not extensive. Bacteria were occasionally seen in histological sections. Fish were initially free of bacteria in the kidney but bacteria appeared in the kidney when parasites appeared on the skin. Bacteria were presumptively identified as a species of the genus *Aeromonas.* The number of ulcers declined and the number of scars increased in the fall. Scars were whitish, scaleless pockets that could not be penetrated by gentle probing with a blunt instrument. Microscopic examination revealed epidermal regeneration. Dermal collagen bridged the wound below the basal epidermal tissue, but was thinner than that in the adjacent stratum compactum layer. The amount of granulation tissue under scars was less than that observed in lesions. Melanocytes were scattered throughout the fibrotic tissue but were not abundant. Muscle bundles under most scars appeared normal (Berry et al., 1991).

1.5. Diagnosis: Adult female can be seen macroscopically; copepodids require the use of a dissecting microscope.

1.6. Treatment: To eliminate of the parasite requires treatment over several weeks to break the life cycle at the larval stage because embedded females are difficult to kill. To treatment, Organophosphate insecticides, particularly trichlorfon are commonly employed. Trichlorphon at 0.25 ppm will kill the copepodid stage but not the nauplii or adults. Bromexs at 0.12-0.15 ppm kils both nauplii and copepodids (Sarig, 1971). NaCl at 20-40 ppm eradicate *L. cyprinacea*. Dimilin at 0.03 ppm eradicate parasite (Hoffman and Lester, 1987).

2. Caligus rogercressyi

Caligus Müller, 1785 is the largest genus of parasitic copepods, containing more than 250 species. In recent years, epizootics of sea lice *Caligus elongatus* and *Lepeophtheirus salmonis*, a common marine ectoparasitic copepod of salmonids, have resulted in heavy losses and millions of dollars worth of damage to aquaculture throughout the northern hemisphere (Pike, 1989). *L. salmonis* feed on mucus, skin and blood of salmonids causing osmoregulatory stress and death in extreme cases (Grimnes & Jakobsen 1996, Björn & Finstad 1997).

2.1. Aetiyology: *Caligus rogercressyi* (originally identified as *C. flexispina*) is the most common species and *Caligus teres* is occasionally found on farmed trout in Chile. Adult caligids usally show sexual dimorphisim; the female is usally larger then thr male. The average length of adult females is 4.96 mm, (range: 4.46–5.49 mm) (Boxshall and Bravo, 2000).

2.2. Epizootiology: The parasite was transmitted to the farmed fish by the native rock cod *Eleginops maclovinus* and *Odonthestes regia* (Carvajal et al. 1998). *Caligus flexispina* have a life cycle consisting of the following stages: two nauplius, one infectious copepodid, four attached chalimus stages, one pre-adult and the adult.

The life cycle of *C. rogercresseyi* has eight development stages, three planktonic and five parasitic. The planktonic stages comprise two nauplius and a copepodid, the infective stage. The copepodid settles on the host, holding on with its hooked pair of antennae. During moulting, the copepodid extrudes its frontal filament to attach itself permanently to the fish. The parasite moults into the four different chalimus stages always attached by a frontal filament. The size of the parasite increases at each stage. The planktonic stage begins with the first nauplius whose average length was 425 µm that moults in the second nauplius with an average length of 463 µm and then to the copepodid of 658 µm in average length. The parasitic stage begins with first chalimus up to fourth chalimus ending in the adult females and males (Gonzalez and Carvajal, 2003).

2.3. Pathogenicity: *Caligus rogercresseyi* Boxshall and Bravo (2000) is the dominant sea lice parasite affecting the salmon and trout industry in Southern Chile. At the beginning of salmon production in the country, Reyes and Bravo (1983) reported the presence of *Caligus teres* Wilson (1905) in coho salmon (*Oncorhynchus kisutch* Walbaum). Another species, initially identified as *Caligus flexispina*, but now identified as a new species, *C. rogercresseyi* by Boxshall and Bravo (2000) was also reported in 1992 (Gonzalez and Carvajal, 1994) on rainbow trout *Oncorhynchus mykiss* (Walbaum). In Chile, *Caligus* has become a threat for the farmers, producing economic losses of US$ 0.30/kg due to the costs of delousing the fish in the processing plant and the slower growth of the fish, because of the presence of the copepods which stress them (Sievers et al., 1996). The excessive use of expensive drugs as bath or oral treatments to control the parasite is producing economic impact as well as pollution in coastal areas. Because this parasite causes less impact in farmed fish than microbial diseases (BKD), few studies on this Chilean parasitosis exist (Reyes and Bravo, 1983; Gonzalez and Carvajal, 1994). With the exception of the naupliar stages, sea lice feed on host mucus, skin and blood. Sea lice have been reported to reduced productivity and cause disease outbreaks in a variety of farmed fish species (Pike, 1989; Berland, 1993; Grimnes and Jakobsen, 1996).

2.4. Symptoms: Salmon lice are caligid parasitic copepod species that have been reported to cause skin lesions in fish and losses in productivity in several farmed species in different parts of the world (Boxshall and Defaye, 1993).

2.5. Diagnosis: Tee large female parasites are usally visible to the dicerning eye on the gill, fins or body of fish or in the buccal and opercular cavities. The main characteristics used to identify the first chalimus of *Caligus rogercresseyi* is the cephalothorax with rounded posterior margins and frontal filament with a rounded base as defined by Kim (1993) and Piasecki and MacKinnon (1993) for other species of *Caligus*. The average length was 830 µm (range: 700–900) based on 21 specimens. The cephalothorax of the second chalimus has posterolateral corners. The frontal filament has two bases: a distal one corresponding to the first chalimus and a proximal one, newly formed and bilobed (Gonzalez and Carvajal, 1994).

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2.6. Treatment: Emamectin benzoate 50 mg/kg a day, for seven day (Stone et al., 1999). Ivermectin in feed, dosage of 0.05 mg kg-l, every third day for either 3 or 6 doses (Johnson and Margolis, 1993).

3. Ergasilus spp.

Ergasilid copepods damage the gills and cause commercially significant epizootics in cultured and wild fish. *Ergasilus sieboldi* affects tench, pike, whitefish, ell, flounder (Woo, 1995) and trout (Buchmann and Bresciani, 2001). Recenly, *E. labracis* killed large numbers of Atlantic salmon, *Salmo salar,* parr (85-125 mm long) over a 4 day period in New Brunswick, Canada (Hogans, 1989).

3.1. Aetiyology: Ergasilids copepods are less modified than fish-parasitic copepods and resemble free-living copepods in segmentation. Only fertilized females are parasitic; males and developmental stages live in the water column. The mature female of *Ergasilus sieboldi* is 1.5 mm long, Body, cyclopiform, has a cephalothorax formed from the cephalosome and the firt leg-bearing segment (Lester and Roubal, 1995).

3.2. Epizootiology: Adult females are external parasites, infesting the gills of a wide range of freshwater fishes and attaching to the gill filaments. Developmental stages are planktonic and occur in the water column of freshwater bodies. Parasites have unique lifecycle: eggs hatch into free-swimming naupliar phase. Only adult female is parasitic, seeking and attaching to a wide variety of freshwater fishes. Female attaches to gill filament of host, using clawed antennae that penetrate host gill epithelium. The life cycle of E. sieboldiconsist of six nauplii and five copepodid stages and adultr stage. It takes 22 days forth eggs of E. sieboldi to develop into free-living males and females at 17-20 ºC. After fertilization, the female attaches to a fish (Lester and Roubal, 1995).

3.3. Symptoms and Pathogenicity: There is extensive gill damage and severe haemorrhage with inflammation and exsanguination associated with the attachment and feeding of the parasite. Blood vessels in the gill filaments are blocked and this leads to atrophy of gill tips (Paperna and Zwerner, 1981). Gill damage resulted in loss of gill surface area for respiration and led to suffocatio, particularly at high water temperatures. Heavy infections cause death (Grabda, 1991; Buchmann and Bresciani, 2001).

3.4. Diagnosis: Detection of parasites attached to the fish gill.

3.5. Treatment: Trichlorfon bath of 0.15 ppm 6 h. , malathion at 0.2 ppm 6 h. are suggested (Kabata, 1985).

4. Argulus spp

Argulids have been recognized as pest of cultured trout in Europe (Kabata, 1985). They cause mortalities of fish in aquaria (Toksen, 2006), in lakes and estuarines and occasionally cause problem in sea caged salmonids (Stuart, 1990). The three best known species are *Argulus foliaceus, A. coregoni, A. japonicus*.

4.1. Aetiyology: The genus *Argulus* spp (Crustacea: Branchiura), or fish louse, are 5-10 mm in size. The general body-form of *Argulus* is a dorso-ventrally flattened and covered by a large chitinous carapace. The body can be divided into 3 distinct regions: cephalothorax; thorax and abdomen (Soulsby 1982; Fryer, 1982; Lester and Roubal, 1995).

4.2. Epizootiology: *Argulus* spp. have direct life cycle (Mikheev et al., 2001). The female is clasped by the smaller male and his genital opening is brougth into contact with the female spermatheca where the female deposits the sperm. The female then lay eggs on different objects in the water and they are fertilized at the time of deposition. Just after hatching the larva isable to parasitize the host. The first stage larvae which has a resemblance to adults but without suckers. Following 5-8 moults the adult stage is reached. The development is highly temperature dependent. The parasite feeds by puncturing host cells with its ventral stylet and sus-bsequently suckink up extracellulary digested cell debris (Buchmann and Bresciani, 2001).

4.3. Pathogenicity: The direct effects of this parasite on its host depend on the number of parasites and the size of the host. One or two parasites on a small fish cause the local damage on the epithelia. Several parasites have a very significant effect.

4.4. Clinical Signs: Fish with heavy infestations become lethargic, can cease feeding and show a general deterioration in their condition as reduced pigmentation, fins drooping (Lester and Roubal 1995). As well as the damage and stress caused by *Argulus* itself, one of the main worries for fish producers is the associated secondary infestations that can result from infestations with parasite. Several studies have examined the role of parasites as vectors for other diseases such as Aeromoniasis or Pseudomoniasis (Bauer 1991, Richards 1977).

4.5. Diagnosis: Argulus spp. can be seen scuttling over the surface of the fish with the naked eye.

4.6. Treatment: Emamectin benzoate 50 µg/kg for a period of 7 d. (Hakalahti et al., 2004), potassium permanganate (2-5 mg/lt, bath) or dimilin (0.01 mg/lt, bath) (Öge 2002), pyrethrum (20-100 ppm for 10-20 minutes); malathion (0.25 ppm for 6 hours); dipterex (100 ppm for 1 hour); trichlorfon (0.25 ppm for several hours); quinine hydrochloride (13.5 ppm for several days); atebrine (10 ppm for sevaral days) (Kabata 1985); DTHP (2.5 ppm 1 hour) (Puffer and Beal 1981), DDVP (1 ppm 1 h.) (Toksen, 2006) are effective againt to *Argulus* spp.

5. Salmincola spp.

Members of genus *Salmincola*, especially, Salmincola californiensis are potential threat to salmonids farmed freshwater (Woo, 1995). *Salmincola edwardsii* is found mainly in cold northern climates. It has a holarctic distribution and infects only fish of the genis *Salvelinus*. *Salmincola edwardsii* is limited to the locations that *Salvelinus* exists. A few places in which *Salmincola edwardsii* have been reported are northern North America, Britain, and Scandinavia. Common places for *Salmincola edwardsii* to exist are in rivers with free access to the sea (Black, 1982; Black et al., 1983).

5.1. Aetiyology: *Salmincola edwardsii* is a relatively small organism. The average length of an adult male is 718.6 µm. The females are considerably larger averaging around 2055.8 µm. Size of the organism may be directly related to the site of attachment on the host fish. The gills of the fish offer protection and a more nutritious food supply; this location may support larger and more rapid development. The larvae are smaller than the adults approximately 512.4 µm. The adult female has an impermeable cuticle. *Salmincola edwardsii* body structure is modified away from the ancestral copepod. In the adult all external signs of segmentation have disappeared. Adult females are permanently anchored to the host's flesh. They are attached by a structure called a "bulla". The bulla is nonliving. It is formed from head and maxillary gland secretions. This structure is the anchor that holds the female onto the host. The females of this family, Lernaeopodidae, are attached almost completely outside the host, which is different than most other parasitic copepod families. *Salmincola edwardsii* have huge maxillae that are fused to the bulla. Located and functioning anteriorly *Salmincola edwardsii* has modified maxillipeds for a grasping structure. The swimming legs and abdomen that is seen in most copepods is absent or vestigial in adult female *Salmincola edwardsii* (Conley and Curtis, 1992; Conley and Curtis, 1994; Roberts and Janovy, 2000).

5.2. Epizootiyology: *Salmincola edwardsii* is found in aquatic habitats. The first larval stage of *S. edwardsii* swims in the water to find a host. *Salmincola edwardsii* need to be where its host genus *Salvelinus* lives, which is in lakes, and rivers that usually have outlets to the sea. *Salmincola edwardsii* spends most of its time near the bottom of the water column prior to attaching to a host. (Poulin et al., 1990).

5.3. Pathogenicity: Salmonid fish, such as trout that are raised in commercial hatcheries are at high risk for heavy infestations of *S. edwardsii*. *Salmincola edwardsii* can cause infections and create serious health problems for fish in cage culture. Larger fish circulate more water over their gills and thus bring more *S. edwardsii* (also other copepodids) in contact with them. Ironically, in natural fish populations the occurrence and intensity of the infection of *S. edwardsii* are usually low and have little impact of the fish. The fish is only vulnerable for the period of time when *S. edwardsii* is in its infective, free-living stage looking for a host. If the timing of egg hatching and the release of the free-living stage can be timed, hatcheries will know when the fish can become infected and hopefully in the future control *S. edwardsii* infection. In times of high temperatures and low dissolved oxygen, fish with many *S. edwardsii* attached to their gills have an especially difficult time. Also as a result of high infestation, growth and sexual maturation is stunted. (Amundsen et al., 1997; Conley and Curtis, 1992; Poulin et al., 1989).

5.4. Clinical Signs: The parasitized fish were found to develop anemia, expressed by the reduction in red cell counts, hemoglobin concentrations and hematocrit values. This anemic condition is attributed to hemodilution of the blood, resulting from damage to the gill and skin epithelia, and, in turn, leading to an osmotic imbalance between the water and the internal fluids. In addition, the progressive reduction of the red cells in the circulating blood may be a result of the absorption of parasite metabolic secretions through the gills or the bulla. Such absorption seems likely because of the observed variations of the cells in the leucocytic system and the significant increase in lymphocytes, neutrophils and "granulocyte cells" in relation to infection time. Furthermore, the blood of the infected fish clotted faster than that of the non-infected fish. During the course of infection a marked increase was also observed in the number of thrombocytes. Parasitized fish were less able to cope with environmental stresses. A water temperature of 21°C was found to be the median lethal temperature o infected fish. The swimming ability of infected fish was also reduced. The parasitized fish reached 50% fatigue when they swam in water of a velocity of 65 cm/sec for only 250 min. The chance of survival for the infected fish in this high water velocity is only 6.6% over the period of 600 min. The ability of the infected fish to transfer from fresh water to salt water was also affected. Mortality of the infected fish increased during this transition and these fish, as indicated by the salinity preference test, also avoided high salinity, suggesting that they may not have been ready to migrate. The critical period of infection where marked differences were found in all the parameters was that period when the parasites reached maximum size and a second infection took place with copepodids hatched from the original group (Pawaputanon, 1980; Buchmann and Bresciani, 2001).

5.5. Treatment: Ivermectin is intubated with 0.2 mg/kg fish, orally. A second treatment is administered after a further 14 days (Roberts et al., 2004).

Conclusion:

Aquaculture is one of the increasingly developing industry. But fish diseases have become increasingly visible during the latest decades in connection with the development of aquacultural industries troughout the world. Diseases problem including hazards caused by parasitic organisms are the main threat to further increase of the industry. Sustainable development of aquaculture relies on disease prevention.

In summary, parasitic diseases are economically important parasites in frehwater aquaculture. Disease outbreaks and subsequent mortalities caused by parasite are now rare due to the development of a variety of effective treatments. However, large economic losses still occur as the result of reduced feed conversion and growth, indirect mortality, loss of product value, and treatment costs. Although it is well understood that parasites have a major impact on trout aquaculture, there are relatively few published reports of disease and/or disease treatments. There are no reports of economic costs associated with these infections. Husbandry practices as well as a variety of engineering, environmental, and biological factors can have an impact on the level of infection by parasitic copepods. However, the relative importance of these factors in controlling parasite abundance varies between sites. There is no evidence from field studies to support the suggestion that parasites can act as vectors for fish diseases. The aim of this paper is to present general overview of parasitic diseases occured on trout.

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