Treatment Trials Of Parasites Of Sea Bass (Dicentrarchus labrax) and Sea Bream (Sparus Aurata) in Turkey

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Abstract: With over 8300 km of coastline and 25 million square hectares of useably sea, Turkey has particularly bright future in aquaculture. Interest has centred on two major species sea bream, sea bass, Those are most favourable have been the Aegean and Western Mediterranean coasts. Sea bass and sea bream pruducts have reached to 75,000 tons in Turkey. The gradually increase of this production of fish resulted in serious pathological problems in all countries where intensive aquaculture is practiced. Thus, focus has been placed on fish diseases in these enterprises and their economic and ecological impact. Especially, parasitical diseases have become increasingly visible during the latest decades in connection with the development of aquacultural industries troughout the world. In this study, various studies were carried out in different time about parasites of cultured gilthead sea bream (*Sparus aurata* L.) and sea bass (*Dicentrarchus labrax* L.) in Turkey and their treatment were investigated. Different species such as *Trichodina* spp., *Costia* spp., *Amyloodinium ocellatum*, *Furnestinia echeneis, Microcotyle chrysophrii, Diplectanum aequans, Caligus minimus, Lernanthropus kroyeri* and *Ceratothoa oestroides* were reported on the gills of sea bream and sea bass in these studies. In this review, the parasites observed on sea bass and sea bream, and their epizootiology, clinical signs, pathogenicity of the parasites and their treatment were given, separately.

Keywords: Sea bass, sea bream, parasite, diagnosis, control, treatment

Introduction

Turkey is a country of which three sides have been surrounded by the seas. Its coastline is 8333 km and 25 million square hectares of useable sea. There is a great aquaculture potential in Turkey. Therefore, Turkey is a most important aquaculture producer in the Mediterranean. The Gilthead sea bream (*Sparus aurata*) and sea bass (*Dicentrarchus labrax*) are the main cultured fish specieses in the Mediterranean area. Recently, it is shown in Table.1 that sea bass and sea bream pruducts have reached to 80,940 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.

Vibriosis, pasteurellosis and tenacibaculosis are serious threatening bacterial infections of sea bass and sea bream. The most important parasites for cultured sea bass and/or sea bream are *Trichodina* spp., *Ichthyobodo* spp., *Amyloodinium ocellatum*, *Furnestinia echeneis, Microcotyle chrysophrii, Diplectanum aequans, Caligus*

minimus, Lernanthropus kroyeri and *Meinertia oestroides*. This research presents the individual parasies types producing probles 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.

Type of fish	**2004**	**2005**	**2006**	**2007**	**2008**
Inland water					
Trout	43 432	48 033	56 026	58 433	65 928
Carp	683	571	668	600	629
Marine water					
Trout	1 650	1 249	1 633	2 740	2 721
Sea bream	20 435	27 634	28 463	33 500	31 670
Sea bass	26 297	37 290	38 408	41 900	49 270
Mussel	1 513	1 500	1 545	1 100	1 772
Prawn	-	-	-	-	-
Other	-	2 000	2 200	1 600	196
Total	**94 010**	**118 277**	**128 943**	**139 873**	**152 186**

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

1. *Trichodina* Spp.

Trichodinid protozoans are cosmopolitan aquatic parasites, common on gills and skin of fish in both the freshwater and marine environments. Trichodinids are peritrich ciliates (order Mobilina, family Trichodinidae) that glide on the surface of the fish. They normally feed on bacteria and mucus and are often considered as ectocommensal nuisances rather than true parasites.

1.1. Aethiology: *Trichodina* spp. are a group of dorsal-ventrally flattened oval ciliated protozoan parasites of marine and freshwater species of finfish. The diameter of the ciliate is mostly about 50 to 100 µm. A readily distinguishable characteristic of these organisms is the presence of a prominent denticular or "tooth-like" internal cytoskeleton ring. There are four additional genera of trichodinids (*Trichodina, Trichodinella, Paratrichodina, Tripartiellea, Hemitrichodina*) which are similar in description and life cycle.

1.2. Epizootiology: Trichodinids reproduce by simple binary fission under conditions that are usually optimal for the host fish. Most species are host specific and presumably spread from fish to fish by incidental contact between susceptible host fish, as well as through contact with the organism in the water column. Transmission is direct, from fish to fish. Within 8 to 10 h's of the host's death, trichodinids leave the host but, depending on the temperature, may survive for several days in the water (Lom, 1995).

1.3. Patogenicity: While small numbers of these organisms on a fish generally do not cause much of a health problem, large numbers can cause moderate to serious pathology and ultimately, death of fish. Small fish and fry are especially susceptible, and mortality can occur quickly if undiagnosed (Toksen, 2004). *Trichodina* spp. cause irritation by feeding on the epithelial layer of cells covering the surface of the gills and skin of the fish. This can result in hyperplasia (proliferation) of the epithelial cells, clubbing of the gill filaments and even fussion of the gill filaments. This affects the ability of the gills to maintain optimal respiratory and excretory activities, and the ability of the skin to maintain proper homeostatic osmoregulatory properties. Massive infestations of these parasites on fish can also directly result in superficial to deep ulcerative skin lesions which then allow for secondary bacterial and fungal infections to develop at the affected site (Lom 1995). *Trichodina spp* can cause extensive fish mortality in an aquaculture system. The ability of this parasite to quickly multiply under certain environmental conditions or when the fish are stressed by other factors makes early detection of this parasite a high priority in an aquaculture facility. Once diagnosed, an appropriate treatment or management response is essential to prevent rapid loss of fish stocks (Samartin-Duran et al., 1991).

1.4. Symptoms: Heavily infected fish may have a greyish-blue coat, which is formed by excessively secreted mucus and peeled epithelia. The fins may be frayed (Lom, 1995).

1.5. Diagnose : The following measurements and counts are of primary diagnostic value; diameter of the adhesiv disc, diameter of the denticulate ring, number and size of denticles. The diameter of the horseshape macronucleus and position of the micronucleus in relation to the macronucleus is also of diagnostic value.

1.6. Treatment: There are several methods by which *Trichodina* spp. may be controlled in the aquaculture of foodfish. These include chemical treatments, freshwater baths, and flushing. UV is generally considered ineffective due to the high dosage rates required to kill the organism.

A formalin bath of 170-250 ppm for 60 minutes is applied effectively (Toksen, 2004). However, experience has shown that a single formalin bath may not completely remove all of the parasites from fish, especially marine fish, and long term or periodic treatments may be needed to keep this parasite under control. Therefore a continuous bath of 25 ppm formalin is also approved for use on foodfish.

Another common method for controlling *Trichodina* spp. on marine finfish is to utilize periodic fresh water dips. Though stressful on fish due to increased handling and the osmotic stress, this method can be very effective in reducing the overall number of parasites on fish. This is an effective method for treating individual fish such as broodstock, but may not be a viable option in a production facility due to the logistics associated with handling and treating large numbers of fish (Brown and Markus, 1998).

Flushing of production systems (i.e., the removal of system water prior to treatment) is another means of reducing infestation levels of *Trichodina* spp. This method may be effective by physically removing any dislodged parasites in the water column from the system.

2. *Ichthyobodo* Spp.

Ichthyobodo necator (former *Costia necator*) is a common parasite that infects a wide range of freshwater fish species. The parasite is found on the skin and gills of fish, most commonly attaching to the edges of the gills. Infected fish have a disease called ichthyobodosis. The first observation of *Ichthyobodo* spp. infection in cultured seabream in Turkey carried out by Toksen. Fifty to sixty percent of mortality was observed in a farm of gilthead seabream (*Sparus aurata L.*) (1g) which were trasferred from Yumurtalık, South-East Mediterranean Sea to Kokar Bay, Western Coast of Aegean Sea (Toksen, 2000).

2.1. Aetiyoloy: Free swimming form is ovoid to spherical and measures 5-18 µm. It has two flagella, one of them longer than the other. It uses flagella for motility and to attach to the host fish (Lom, 1995; Toksen, 2000).

2.2. Epizootiology: Both free swimming and parasitic stages multiply by longitudinal binary fission. The parasit is not host spesific. Malnourished and young fish are more secerely affected then healthy adults (Robertson, 1985; Toksen, 2000).

2.3. Pathogenicitiy: These parasites do not cause distinctive lesions on the fish but do block the flow of oxygen when heavily loaded on the gills. As with most protozoa, environmental degradation and crowded conditions cause them to become more damaging. However, prevention measures such as reducing stocking densities and lowering feeding rates may make fish production unprofitable. But stocking and feeding rates should be kept reasonable. Contact a qualified aquaculture or fisheries scientist for advice on proper stocking densities for the fish species you are raising skin and fins. The base of the stalk attaches to a hard, calcified surface such as scales and fin rays or spines. *Ichthyobodo* occurs on the skin and gills (Lom, 1995).

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2.4. Symptoms: Ichthyobodosis causes damage to the gills and skin of fish. Infected fish can lose condition, become emaciated and be very lethargic. These symptoms can be seen in fish with only a light infection. The attachment and feeding of *Ichthyobodo necator* causes severe damage to skin and gill cells. Hyperplasia can occur within the gills, reducing respiratory efficiency. The gills may also swell with fluid, and fish often die as they are unable to control the movement of water in and out of their bodies. The parasite also causes irritation and infected fish produce excess mucus (Lom, 1995).

2.5. Treatments: Formalin is used against to *Ichthyobodo* spp. effectiveliy (Toksen, 2000). Bithionol (25 ppm for 3 h or 2 consecutive days) is very effective in eliminating the paraiste from rainbow trout (Tojo et al., 1994).

3. Amyloodinium Ocellatum

3.1. *Amyloodinium ocellatum* is an important and the most common dinoflagellate that infects the gills and skin of both marine and brackish water fishes (Lauckner, 1984). A similar organism, *Oodinium* spp., is found in freshwater fish. The disease caused by these organisms has been referred to as "velvet," "rust" and "gold dust disease" because of the shiny sheen the parasite imparts to heavily infected fish.

3.2. *Amyloodinium* spp. can cause great losses of aquarium fish or fish held in high-density culture systems and has caused serious problems in public aquaria, aquaculture facilities and home aquaria (Montgomery-Brock et al, 2001). If allowed to become established in high-density recirculating systems, it can be difficult to control. For example, cultured red drum have been shown to be extremely susceptible to this infection. *Amyloodinium* infects a wide variety of fish and has been reported to occur in more than 100 species in North America. In Turkey, first *Amyloodinium* enfestation was observed on cultured 15-20 g of sea bass in pond with 100% mortality (Cagirgan and Toksen, 1996).

3.3. Aetiyology: The trophont is pear-shaped to ovoid and up to 350 um long. An osmophilic ring encircle the basal region, and an attachment plate bearing numerous filiform rhizoids exista through the break in the theca. Divisions withina common cyst wall produce up to 256 dinosporea. Dinospores are 8-13.5 µm long by 10-12.5 µm wide (Lom, 1995).

3.4. Symptoms: Often, the first indication of an amyloodinium infection is dead or dying fish. Amyloodinium should always be considered as a possible cause of mortality when a disease outbreak involving marine or brackish water fish occurs. Behavioral signs may include a decrease in or complete lack of feeding activity, flashing (rubbing against objects in the tank or on the bottom substrate) and coughing (backflushing water across the gills). The skin of heavily infected fish may have a dull gold or brown sheen. Closer examination of the skin may reveal scale loss and patchy accumulation of mucus (Reed and Francis-Floyd, 1994). Diseased fish shows sluggishness and asphyxia symptoms with darkened pigmentation of the skin and V shaped loss by the reason of necrosis of tail. The gill is pale and haemorrhagic in infected fish. Extensive necrotic areas are observed in macroscopycally on the gill (Cagirgan and Toksen, 1996).

3.5. Epizootiology**:** Amyloodiniosis is limited to warm waters. The optimal temperature for tomont division and sporulation ranges from 23-27 ºC. Completion of tomont division is limited to 16-30 ºC (Paperna, 1984). Infections do not occur at less than 17 ºC. The minimum effective salinity varied from 1 to 20 ppt, depending upon the isolate (Paperna, 1984). Tomonts or infective dinospores can be introduced directly with incoming seawater, becoming a source of infection for fish in the system. Obviously, introducing fish infected with trophonts into a culture system will serve as a source of infection as soon as the throphonts detach and begin the reproductive process.

3.6. Diagnosis: The only sure way to diagnose an amyloodinium infestation is by identification of the parasite in infected tissue. Preparations of gill, fin and skin (scrapings of mucus and scales) can be examined with a light microscope. The trophont attaches to the tissue of the fish by means of an attachment plate, which may be visible with a light microscope. Trophonts are removed brushing the fish gently, followed by microscopic examination of the sediment, which contains detached parasites (Noga, 2000).

3.7. Treatment: The most commonly applied treatment for control of amyloodinium is copper. In marine recirculating systems, which do not contain invertebrates, copper is added to the system gradually over a period of several days until the free copper ion (Cu^{2+}) is at a concentration of 0.12-0.15 mg/l; this level is then maintained for up to 3 weeks (Cardeilhac and Whitaker, 1988). This standard procedure, observed for many years, is moderately effective but requires repeated testing of the copper concentration to ensure that amyloodinium is being controlled without killing fish. This treatment will kill all invertebrates present in the system and certain groups of fish.

Freshwater dips are effective in killing free-swimming stages of amyloodinium; however, since encysted stages are protected, a single freshwater dip is not an effective treatment. Decreasing the salinity in a system has been suggested as a method for controlling amyloodinium epizootics, but because the organism flourishes in brackish water, the effectiveness of this strategy is doubtful.

Given the lack of a safe, effective therapeutant for the control of amyloodinium, avoidance is an extremely important means of preventing outbreaks of this parasite. All incoming fish should be quarantined for a minimum of 3 weeks before being introduced into an existing system. Do not feed live or frozen food items

that may be infected with amyloodinium. Do not introduce water into a system that may be contaminated with amyloodinium dinospores without using effective filtration or sanitation procedures (Reed and Francis-Floyd, 1994).

4. Furnestinia Echeneis

The monogenean was found on the gill of sea bream *Chrysophrys aurata* by Wagener in 1857 and formerly named as *Dactylogyrus echeneis* Euzet ve Audouin (1959) renamed as *F. echeneis* (Oliver, 1969).

4.1. Aetiyology: *F. echeneis* is 560-890 μm in length, 140-230 μm in width in ovary level. Parasite has a haptor 190-270 μm in diameter and lamellar shaped squamodisc 180-220 μm in diameter in haptor.

4.2. Epizootiology: Infestation is successfully transmitted to naïve gilthead seabream by egg exposure. Parasite occure in all seasons of year but the number of parasite increase in spring (Revarsat et al., 1992). *Furnestia echeneis* caused high mortality in *Siganus auratus* (Paperna, 1978).

4.3. Symptoms: Infested fish showing severe signs of asphyxia due to necrosis on the gill and mass mucuos secretion. *Myxobacterium* spp. is found in necrotic lessions on the gill (Paperna et al., 1977).

4.4. Pathogenicity: No pathological signs are referred to *F. echeneis* infections, also with 50 specimens/gill arch infection intensity (Quaglio et al., 2007). But in heavily infestation shows hyperplasia of gill epithelium with thickening of lamellae up to fusion.The gills show diffused degeneration and necrosis in the filament epithelial tissue (Reversat et al., 1992). It has been reported to cause mortalities in natural sea bream in Red Sea and Acabe Bay (Paperna and Baudin Laurencin, 1979).

4.5. Treatment: Formalin bath 200 ppm 1 h is effective (Paperna et al., 1977; Toksen, 1999).

5. Microcotyle Chrysophrii

S. chrysophrii Euzet and Noisy 1981, originally called *Microcotyle chrysophrii* (van Beneden and Hesse 1863) (Microcotylidae: Polyopisthocotylea), is a common parasite of cultured Gilthead sea bream which has caused lethal epizootics in sea cages (Alvarez-Pellitero 2004).

5.1. Aetiyology**:** The parasite belong to genus *Microcotyle* (Microcotylidae, Polyopisthocotylea) comprising 17 recognized species in European waters. Monogenean is 3-5 mm in length, 0.5-0.7 mm in width in ovary level (Euzet et Noisy, 1979).

5.2. Epizootiology: *Sparicotyle chrysophrii* is successfully transmitted to naïve gilthead seabream by egg exposure and cohabitation with parasitized fish (Sitjà-Bobadilla and Alvarez-Pellitero**,** 2009). Parasite occure in all seasons of year (Reversat et al., 1992).

5.3. Pathogenicity: *S. chrysophrii* shows a high pathogenicity at low infection intensity (8 parasites/gill arch) with gross lesions such as gill and systemic anaemia already noticeable at necropsy. In this case histology shows severe hyperplasia of gill epithelium with thickening of lamellae up to fusion, and heavy sloughing off of the epithelial cells. Moreover the gills show diffused degeneration and necrosis in the residual epithelial tissue. The hematophagous attitude of *S. chrysophrii* is evident for the presence of several erythrocytes in the parasite gut (Quaglio et al., 2007; Reversat et al., 1992). It has been reported to produce mortalities in farmed fish (Alvarez-Pellitero, 2004), and it is frequently found in mixed infections with other parasites and bacterial infections (Padros and Crespo, 1995).

5.4. Symptoms: Infested fish swim near the water surface, showing severe signs of anemia as lethargy, emaciation, anoreksi and excessive mucus production (Padros and Crespo, 1995).

5.5. Treatment: Formalin bath of 250 ppm for 60 minutes is applied effectively (Toksen, 1999).

6. Diplectanum Aequans

Diplectanum aequans (Wagener, 1857) Diesing, 1858 is a common parasite of both wild and cultured European sea bass *D. aequans* is considered to be potentially harmful in intensive sea bass farming (Gonzalez-Lanza et al., 1991; Toksen, 1999).

6.1. Aetiyology: Monogenean parasite is 650-1.700 µm. in length and 260-500 µm. in width in ovary level. There is a haptor in the posterior end of body. The diameter of haptor is 0.11-0.30 µm. and has a squamodisc (180 µm. in diameter), two pairs of hamuli and 14 marginal hooks (Oliver, 1980; Toksen, 1999). The adult of *D. aeqauans* is observed on the gills of sea bass (Cecchini et al., 1991; Toksen, 1999) but larval stages of parasites can be also observed on the skin. parazitin genç evrelerine deride de rastlanılmaktadır (Cognetti, et al., 1992; Gonzales et al., 1991).

6.2. Epizootiology: The life span of *D. aequans* at 20°C is estimated to be 30 days. The parasites are oviparous and produce the eggs on the gill of sea bass. The diameter of egg is 59.14±6.96 µm. The parasite has 5 stages in ith life cycle; larval stage (oncomiracidium), post larval stage, 2. post larval stage, intermedier stage and adult stage (Silan and Maillard, 1989). The adult parasite is exhibiting hermaphroditism. The contamination is occured bye means of eggs between hosts.

6.3. Symptoms and Pathogenicity: *D. aequans* attaches to the gill lamellae and cause hyperplasia of the epithelium and mucuos cells, with resulting deformation and fusion of the secondary lamellae. Heavily infected fish exibit lethargy, anorexia and asphyxia symptoms (Oliver, 1977; Toksen, 1999).

6.4. Diagnosis: *D. aequans* is easiliy distinguished on the basis of the shape and size of the haptor, hamuli and hooks on the haptor, and male copulatory organ of adult parasite (Silan and Maillard, 1989; Lambert et Maillard, 1974).

6.5. Treatment: Rafoxanid bath of 6 ppm for 48 hours is applied effectively (Cognetti et al., 1992), trichlorfon bath in dose 0.15 ppm for 2 days is effective (Cognetti et al, 1991). Formalin has not good effect against *D. aequans* affect the parasite (Toksen, 1999).

7. Caligus Minimus

Caligus sp. or 'sea lice' are common copepod parasites in the family Caligidae, infesting a wide range of fish species in the coastal zones and cultured fish.

7.1. Aetiyology: *Caligus minimus* is seen in the mouth cavity and on the gill of sea bass in Mediterranean Sea, Adriyatic Sea and Atlantic ocean. Adult parasites show sexual dimorphism, the female is larger then the male. The female 3-5.5 mm in length, the male parasite is 8 mm in length with 4. legs (Radujkovic and Raibaut, 1989).

7.2. Epizootiology: Caligid copepods have direct life cycle, consisting of a free-living planktonic I. nauplii stage, II. nauplii stage, copepodid stage, I-VI chalimus stages, pre-adult stage and adult stage, and last 17 days at 22-24°C after hatchig (Hallett and Rroubal, 1995). The intensity of copepod infestation generally increases after rainfall and late spring and decline in winter and summer due to the lack of recruitment and parasite death. This is a major problem in cage cultured fishes (Jithendran et al., 2008).

7.3. Clinical Sings and Pathogenicity: The main lesions are observed on the skin of the head region, the buccal cavity, the palate, the tongue and the base of the gill arch (Ragiasa et al., 2004). The integument where parasites are located showed ulceration of the epidermis with marked inflammatory of the dermis as a result of the attachment and feeding activity of the parasites. The attachment is achieved by means of second pair of the antennae which were inserted into the host epidermal tissue. A marked reactive epidermal hyperplasia is observed at those areas as well as at the periphery of ulcerated lesions. Many epidermal cells around the damaged area show signs of necrosis, the vacuolar degeneration of basal cells was prominent and epidermis is also characterized by diffuse areas of spongiosis. In many cases, increased fibroplasia and spongiosis is noticed within dermal collagenous connective tissue (Ragiasa et al., 2004).

7.4. Treatment: Trichlorfon bath of 300 ppm at 20 minutes (Pike, 1989), dichlorvos 1 ppm 1 h (Branson, 1996), hydrogen peroxide 1500 ppm 20 minutes (Branson, 1996; Hodneland et al, 1993) and freshwater bath (Landsberg et al., 1991) are effective.

8. Ceratothoa Oestroides

Ceratothoa oestroides (Cymothoidae) is a ubiquitous fish parasite. It has been reported in 6 different fish families, Sparidae, Carangidae, Clupeidae, Maenidae, Scorpaenidea and Mugilidae, and has been most frequently isolated from the bogue bream *Boops boops* and sea bream *Sparus aurata* (Sparidae) (Charfi-Cheikhrouha et al., 2000; Toksen, 1999).

8.1. Aetiyology: The body of parasite is dorso ventrally flattened and is lacking a carapace. The isopod thorax consists of 7 free segments with 7 pairs of thoracic legs. As a result of the well-sheltered environment of the buccal cavity, species that establish there have evolved a thinner cuticular mineralisation and the pleopods of the three last pairs have transformed into respiratory organs. Paired eyes consist of numerous eyelets. On its ventral side, between the swimming legs, the female bears a brood pouch or "marsupium", shielded by special plates, called "oostegites", to carry the eggs and the larvae for some time after hatching.

8.2. Epizootiology: Female *C. oestroides* bear embryonated eggs in the brood pouch that develop first into stage I pullus, and then into pulli II and III (with rudimentary periopods of VII pairs), and finally into pullus IV, at which stage postlarval evolution begins (Mladineo, 2002). As a protandric hermaphrodite, the parasite passes through different developmental stages: male puberty, prolonged male puberty, transitory stage, female puberty and finally prolonged female puberty (Trilles 1969). During the male puberty stage, the parasite loses its swimming capacity and, once settled in the buccal cavity of a fish, it is incapable of active migration to another host. This fact is important in the epizootic evaluation of the route of infection. After settlement in the host, the parasite begins hematophagic nourishment, which comprises alternating cyclic periods of blood-sucking and blood absorption by the intestine (Trilles, 1969). As a consequence of its sedentary life in the wellsheltered buccal cavity, the parasite has evolved some structural changes, e.g. a thinner cuticle, the last 3 pairs of pleopods transformed into respiratory organs and a thinner-walled incubation chamber (Trilles, 1969).

8.3 Symptoms and Pathogenicity: Heavy infestations of parasitic larvae may kill smaller fish when they first infect them seeking permanent attachment. Pulli II larvae and juveniles attack relatively younger fish, about 5g-20g of weight and cause considerable damage to the skin around the head, the eyes and the gill epithelium by injuring the gill lamellae. Their voracious haematophagy and the mechanical damage of their hooks lead to severe inflammation and necrosis of head, eye and gill tissues. The infested fish are usually apathetic and anorexic and may show respiratory distress. The haemorrhagic and necrotic head tissues are evident when observing the fish in their cage. When the sick fish are removed from the water, several isopod larvae may be seen in their buccal and gill cavities and/or on the skin near the opercula (Varvarigos, 2003; Mladineo, 2002; Toksen, 1999).

Injured tissues are frequently invaded by secondary bacterial pathogens, such as *Aeromonas spp., Tenacibaculum spp., Vibrio spp.* and this may lead to severe escalation of mortality. In young stocks, the cumulative mortality due to parasitism by the pulli II larvae may run as high as 15% even without any bacterial implications (Varvarigos, 2003).

The adult isopods are haematophagus and cause anaemia. The parasitised fish have significantly lower erythrocyte counts as well as haematocrit and haemoglobin values. The leukocyte counts are increased, obviating the host's immune response to the presence of the isopods. In addition, the established adult isopods can cause considerable damage to the mouth tissues with their biting and sucking mouth parts, or their copulation activity. Their large size (up to 6 cm in length) may cause atrophy of the tongue, dysplasia of teeth and slackening of the cartilagenous tissues leading to a "bag-shaped" lower jaw. Invariably, the presence of large adult parasites in the buccal cavity interferes with feeding, causes chronic stress and results in growth retardation and a predisposition to bacterial and/or endo-parasitic invasions (Varvarigos, 2003).

Isopod infestation is confirmed by gross observation of the parasites on the skin, mouth, or in the gill chamber of the fish. In addition, they often produce the lesions described above that characterise

8.4. Treatment: Cypermethrin and deltamethrin are effective in dose of 10 ppb for 60 minutes (Martinsen et al., 2001)

9. Lernanthropus Kroyeri

Lernanthropus is the most common genus of parasitic copepods. So far, more than 100 species isolated from gills of different marine teleosts have been described. Some species of
Lernanthropus are strictly host specific, but many are parasitic on several species of fish within one or several genera (Sharp et al., 2003).

9.1. Aetiyology: Female parasite body is elongate, 2,9 mm including fourth legs 3.7 mm in length (Toksen et al., 2008).

9.2. Symptoms and Pathogenicity: Fish infected with *L. kroyeri* spp. show signs of respiratory distress, enhanced mucus secretion, congestion, haemorrhages associated with the feeding activity of the parasite, primary gill lamella erosions and lethargy, dark coloured skin and surface swimming (Toksen, 2007). Histologically, erosion, desquamation and vacuolar degeneration occurred near the site of attachment. Lamellar fusion in the distal ends of the filaments was observed. Compression of gill tissue by the head and second antennae of female parasite resulted in erosion of the branchial lamellar epithelium and lacerate tissue. Second antennae and maxilliped of parasite has caused partial occlusion and ruptures in capillary (Toksen, 2007) .

9.3. Treatment: Emamectin benzoate of 100 µg kg-1 in feed is effective (Toksen, et al., 2006).

Conclusion

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. Toksen (2000; 2004) reported that Ichthbodosis and trichodiniasis caused fifty to sixty percent of mortality in different two farm of gilthead sea bream (*Sparus aurata L.*). Recently, almost 400,000 gilt head bream died in a single night on fish farms located in the southwestern province of Muğla's Güllük Gulf this week. But the reason of death could not determine. Sustainable development of aquaculture relies on disease prevention.

In summary, parasitic diseases are economically important parasites in marine 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 sea bream and sea bass 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 sea bass and sea bream.

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