Parasitic Diseases of Trout and Their Controls

in Sustainable Development of Aquculture: Platyhelminthes

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Abstract

Aquaculture is an important food-producing sector. It provides much needed protein, employment, income and livelihoods support to many people in the world and this is especially true in most developing countries. A significant challenge to the expansion of aquaculture production is the outbreak of disease. Potential economic losses from disease outbreaks are significant, and can affect the survival of the industry. Fish diseases were caused by enfectious agents as parasite, bacteria, virus and fungus. Gyrodactylus spp., Tetraonchus spp., Discootyle spp., Cyathocephalsus spp., Proteocephalus spp., Eubothrium spp., Diphyllobothrium spp., Trianophorus spp., Crepidostomum spp., Phyllodistomum spp., Sanguinicola spp., Diplostomum spp., Tylodelphys spp., Apatemon spp. are observed in rainbow trout. Gyrodactylus salaris caused the mortality up to 95 % in salmon population in some rivers in Norvay. So, G. salaris is a disease between compulsory notifiable diseases in EU. However, such a pathogen monogenean enfestations have not been appeared in Turkey, First record of Crepidostomum farionis was carried out in rainbow trout in Elazığ from Turkey. The present work aim to the parasitic diseases of freshwater trout caused by phylum

Plathyhelmintes, 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, platyhelminthes, diagnosis, treatment

1.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 2012).

Type of fish	2006	2007	2008	2009	2010
Inland water Trout	56 026	58 433	65 928	75 657	78 165
Marine Trout	1 633	2 740	2 721	5 229	7 079
Total	57 659	61 173	68 649	80 886	85 244

Table.1. Aquaculture production of Turkey (Ton) (TUIK 2012)

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.

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.

The Plathelminthes typically are slightly dorso-ventrally flatted, witth a few exceptions hermophroditic worms without an anus and some of them without a mouth and intestine. Their surface layer is a tegument which is made of a syncytium. This Phylum comprise monogeneans, digeneans and cestodes

Monogeneans are flatworms with representatives in freshwater, brackish and marine habitatts. The most of species are ectoparasitic and they all have a direct life cycle. A few species have adopted an endoparasitic life. They have a characteristic posterior attachment organ called an opisthaptor. This organ is equipped with sclerotiinized structures as large hooks, clamps and marginal hooklets. Also the fore part of the parasite has adhesive pads. Using these adhesive structures he flatworm can move in a leech-like manner on the host.

2. Tetraonchosis:

2.1. Aetiology: Tetraonchus alaskensis, T. awakurai. T. oncorhynchi

The lenght of the T. awakurai is 650-1080 μm and width is 250-415 μm (Ogawa and Egusa 1978).

2.2. Epizootiology: Tha adult parasite attaches to the gill epithelium with its two pairs of anchors and their associated marginal hooklets, then produce eggs. These are liberated to the aquatic enviroment where they develop and hatch. The ciliated larva called oncomiracidium seeks the its host, attaches, sheds the ciliated cells and moves to the gill filaments. Depending on the ambient temperature the post larva develops to the adult stage on the gills (Buchmann and Bresciani 2001). Oncomiracidia hatch from embryonated eggs and transmission occurs when the temperature is above 10 °C (Ogawa and Egusa 1985).

2.3.Pathogenicity: Heavy infections mat elicit inflamatorry reactions in the gills, hyperplasia of epithelial and mucous cella and haemorrhages.

2.4. Symtomps: Parasite cause hyperventilation, lethargy and mortality.

2.5. Diagnosis: Detection of the parasite on the gills of the host. The parasite has four eye spots, four large hooks in the opisthahaptor.

2.6. Prophylaxis: Quarantine measures, mechanical filtration of fish tank water to remove oncomiracidia.

2.7. Treatment: Formalin, mebendazole and praziquantel may have effect (Buchmann and Bresciani 2001).

3. Gyrodactylosis:

3.1. Aetiology: Gyrodactylus salmonis, G. salaris, G. derjavini, G. truttae, G. colemanensis, Gyrodactylus elegans, G. cobitis. G. salmonis measures 0.5 mm in length and 0.15 mm in width. The hamuli are stout and 53-65 um long.

3.2. Epizotiyology: They are vivipourus hermophroditic parasites. The female parasite give birth to an almost fully developed daughter which already has a growing embryo in its uterus. The parasites attcches to thebody surface, fins, buccal cavity. At a farm nets and buckets are potential vehicles of transmission. Water-borne transmission occurs in spring when melting ice frequently causes flooding between holding units. Shipment of fish from other facilities are also potential sources of infeciton and so is the water supply of the farm.

3.3. Pathogenicity: Due to penetration of the epithelium by the marginal hooklets and the feeding activitiy the fish may suffer from osmoregulatory distress. Opportunistic infections may arise in the injured epidermis.

3.4. Symtomps: Heavily infected fish with parasite are cachexic, lethargic and overall dark in clour (Cone and Cusack 1988).

3.5. Diagnosis: Microscopic examination of slides with the parasite shows the approximately 0.5 mm platyhelminth without eyespot but with two large ventral hamuli, connected by a dorsal and ventral bar and 16 marginal hooklets. The shape and size of the hamuli and hooklets are species characteristic.

3.6. Prophylaxis: Quarantine measures. Strict prevention of the introduction of specific gyrodactylids may be necesssary in farms with susceptible fish species.

3.7. Treatment: 4.5-5 % solution of NaCl for 1.5 to 2.5 min and then transfer immediately to fresh runnig water. A second bath may be necessary after several days (Cone 1991). Triclabendazole and nitroscanate are effective at dosages 40 g/kg of feed for 10 d and 0.6 g/kg of feed for 1 d, respectively (Tojo and Santamarina 1988). Formalin, copper sulphate, hydrogen peroxide, sodium per carbonate, mebendazole, praziquantel may use.

4. Discocotylosis:

4.1. Aetiology: Discocotyle sagittata, D. ohridana.

D. sagittata is up to 12 mm length, 3-4 mm width. The rectangular opisthaptor is equipped with four pairs of clamps for attachment to gill.

4.2. Epizotiyology: Sanguinivorous polyopisthocotylean monogenean occur on the gills of freshwater salmonid fishes, with an extensive geographical distribution in the Northern Hemisphere. It occurs at low intensities in the wild (Valtonen et al. 1990). Eggs are produced by the harmaphroditic worm. The egg embryonate and hatch. The larva seek the host and develops into the adult stage.

4.3. Pathogenicity: The parasite feed on host blood and may cause anameia. Inflammatory reactions may impair respiration. Heavy infection by D. sagittata can result in deleterious effects on the host, especially in the context of fish farms. Rainbow trout reared in the Isle of Man have been shown to harbourhigh parasite burdens (>1000/fish) and exibit high mortality rates due toparasite-induced anaemia (Gannicott 1997).

4.4. Symtomps: Anameia, morbiditiy, hyperventilation.

4.5. Diagnosis: Detection of the up to 12 mm long worm on the gills of the host. They are charecterized by the clamps on the opisthaptor.

4.6. Prophylaxis: Quarantine measures should be applicated.

4.7. Treatment: Praziquantel (Rubio-Godoy and Tinsley 2004)

Digeneans are heteroxenous and their adult stages are parasitic in vertebrates. They need more than one host in their life cycles. Molluscs serve as first intermediate host. Bivalves are also the fisrt intermediate host of Fellodistomatidae, Gorgoderidae (Phyllodistomum) and Allocreadiidae (Crepidostomum). Adult digeneans usually have a dorsoventrally flattened oval body with a smooth, spiny or corrugated surface, a sucker around the anteroventral mouth and an additional ventral sucker, or acetabulum. Both suckers are used for attachment and locomotion (Paperna 2006).

5. Crepidostomosis:

5.1. Aetiology: Crepidistomum farionis, C. metoecus

5.2. Epizotiyology: The first intermediate hosts in the life history of this trematode are Pisidium amnicum and Sphaerium corneum. Although the latter is unusual, the second intermediate host is the larva of the mayfly, Ephemera danica. There are two generations of rediae, the first gives rise to daughter rediae, which in turn produce cercariae. Final host is trout (Brown, 2009).

5.3. Pathogenicity. The parasites cause swelling of the intestine. Inflammation of the intestinal wall may cause malabsorption. Parasite reduce the hemoglobin and hematocrit levels in the fish (Klein et al. 1969).

5.4. Symtomps: Inflammatory state of intestinal wall, haemorrhages.

5.5. Diagnosis: Detection of parasite in the intestine. The charecteristic cephalic lobes and the two suckers are found in both species.

5.6. Prophylaxis: Removal of intermediate host.

5.7. Treatment: Praziquantel is liable to have effect when used in feed oral treatmenta.

Articles

6. Phyllodistomosis:

6.1. Aetiology: Phyllodistomum simile

6.2. Epizotiyology: Brown trout, salmon parr. Large numbers of Phyllodistomum simile were found in the urinary bladder of the trout from the River Teify. There was no evidence of a seasonal variation in the occurrence of P. simile in trout aged two years or more.Trout aged four or more years were more heavily infested than young trout and there was no indication of the development of a total immunity with increase in the age of fishes. The life history of P. simile was elucidated under experimental conditions by feeding the trout precocious metacercariae present in daughter sporocysts obtained from the gill lamellae and epibranchial cavity of the freshwater bi-valve, Sphaerium corneum (Thomas 1958).

6.3. Pathogenicity: Extension of urethrae.

6.4. Symtomps: Not clear.

6.5. Diagnosis: Detection of the parasites in the urethrae or bladder.

6.6. Prophylaxis: Removal intermediate hosts.

6.7. Treatment: Praziquantel may have effect (Buchmann and Bresciani 2001).

7. Nanophyetosis:

7.1. Aetiology: Nanophyetus salmincola is a digenic trematode, which means that it is an unsegmented worm that is flattened dorsoventrally. Adult worms alternate shape from "a sphere to a long blunt rod. The worms are 0.8 to 1.1mm long and 0.3 to 0.5 mm wide and are hermaphroditic, having both male and female reproductive organs in the same organism. (Bennington and Pratt 1960).

7.2. Epizzotiyology: The life cycle of the N. salmincola requires three hosts. The first intermediate host is an Oxytrema silicula stream snail. The second intermediate host is a salmonid fish, though some non-salmonid fishes also play a role. Lastly, the definitive host is most commonly a canid, though many other mammals are also definitive hosts, including humans. Transmission of N. salmincola to the definitive host occurs upon ingestion of parasite-infected fish (Bennington and Pratt 1960).

7.3.Pathogenicity: Cercariae invading fish fry may kill the fish probably due toimpairment of the osmoregulatory system. The parasite can transmit bacterial infection (Neorickettsia helminthoeca) to the dog.

7.4. Symtomps: Lethargy, morbidity, mortality of fish.

7.5. Diagnosis: Detection of metaacercariae in fish tissue. These are pyriform, unspined, large oral sucker of same size as ventral sucker, short oesaphagus.

7.6. Prophylaxis: Removal of intermediate snail hosts.

7.7. Treatment: Praziquantel may have effect in the early stage of infection but this has not been documented (Buchmann and Bresciani 2001).

8. Sanguinicolosis:

8.1. Aetiology: Sanguinicola alsea, S. davisi, S. fontinalis, S. idahoensis, S. klamathensis. Sanguinicola fontinalis body very flat, elongate when fixed relaxed, colourless, translucent, 0.90 mm long, 0.21 mm wide after hot fixation, staining and mounting (Hoffman et al. 1985).

8.2. Epizotiyology: The adult parasite lives blood vessels of the fish. Eggs are produced wich hatch to release the first larval stage called the miracidum.. These larvae from the eggs pass out of the gills into the pond water. In the pond miracidia infect the intermediate host as snails of the genus Oxytrema or Fluminicola. In the intermediate host sporocysts are developed and release cercariae which are infective for the fish. Then parasite penetrate into the blood vessel and develop there.

8.3. Pathogenicity: The release of eggs and penetrating miracidia mat elicit pathological reactions in the gill and blood vessels.

8.4. Symtomps: Branchitis (diseased gill), swollaen kidney, mortality, morbidity, lowered haematocrit.

8.5. Diagnosis: Detection of parasite (length 0.5-0.9 mm) in the blood vessels (branchial, renal, cardiac vessels). The adultfluke is lanceolate withour suckers and a narrow oesophaagus. Eggs are thin-shelled.

8.6. Prophylaxis: Removal of snails from ponds and the environment. Filtration of pond water to clear infective cercariae.

8.7. Treatment: Praziquantel may have a effect but this has not been documented (Buchmann and Bresciani 2001).

9. Diplostomosis:

9.1. Aetiology: Larvae (metacercariae) of Diplostomum spathaceum (Diplostomatidae, Digenea). Body length of cercariae is 160-260 μm and the bifurcated tail stem is as long as the body. The established metacercariae are flattened, about 400 μm in length, and possess two suckers and an attachment organ (Hoglund 1999).

9.2. Epizotiyology: The life cycle involves pulmonate snails as first intermediate hosts, fish as second intermediate hosts, and piscivorous birds as final hosts harbouring the adult worms. Fish are infected with cercariae. The adult parasite in the small intestine of a fish eating bird releases eggs. These pass with the host faeces to the aquatic environment where they hatch and release free swimming miracidia. These infect the first intermediate host, a pulmonate snail, Lymnaea pereger or L. stagnalis. In the snails a new larval stage, the sporocyst, develops. Tail bearing larvae, cercariae, in turn appear in the sporocysts and are liberated to the water where tthey seek a fish host. When attached to the skin the tail is lost and the invasion process is initiated. Following penetration of the fish skin, the larva migrates through the lymphatic ducts to the final locality (the eye) in the fish. In the eye the larva develops into the metacercarial stage (Buchmann and Bresciani 2001).

9.3. Pathogenicity: The free-living feeding metacercariae lie in the cortex of the lens. Rupture of the lens and proliferating lens epithelium are sometimes observed at the site of parasite entry (Hoglund 1999). Cercarial penetration of skin may compromise osmoregulation (Buchmann and Bresciani 2001).

9.4. Symtomps: Infection may result in cataract formation within the lens, leading to partial or total blindness. Starvation may cause a significant reduction in condition, or even death. After penetration of the cercariae, lesions may form on the gills, body surface, and fins, which can cause death, especially in young fish, at high infection rates (Hoglund, 1999).

9.5. Diagnosis: Detection of the metacercaria in the lens of the fish.

9.6. Prophylaxis: Removal of intermediate snail hosts. Preventing access of final host (fish eating birds) because they release parasite eggs. Mechanical filtration of pond water may reduce density of cercariae. Increrase of water flow in ponds may flush away the positevely phototactic cercariae.

9.7. Treatment: Praziquantel (330 mg/kg) has been used orally in feed (Buchmann and Bresciani 2001).

10. Apatemonosis:

10.1. Aetiology: Apatemon gracilis

10.2. Epizotiyology: Adult trematodes live in birds. First intermediate host is a freswater snail, the trout is the second intermediate with infective metracercariae. When the fish-eating birds eats the fish it will result in maturation of tehe metacercaria into the adult stage in the intestine of the bird.

10.3.Pathogenicity: The presence of metaccercariae in host tissue produce inflammatory reactions which may affect physiological function of the organ. Trout infection with Apatemon gracilis resulted in fibrogranulomatosis of the epicardium and failure in in-vitro pumping performance (Tort et al. 1987, Watson et al. 1992).

10.4. Symtomps: Heavy infections associated witth lethargic fish. Heart function may decrease.

10.5. Diagnosis: Detection of metacercaria in the fish tissue.

10.6. Prophylaxis: Eradication of intermediate host snails in the culture system and surroundeings. Prevention of bird entry.

10.7. Treatment: Anthelmintic such as praziquantel may have effect (Buchmann and Bresciani 2001).

11. Tylodelphysosis:

11.1. Aetiology: Tylodelphys clavata, metacercariae; body lenght is 0.28-0.69 mm, width is 0.8-0.21 mm. Diameter of oral sucker is 0.025–0.047 x 0.021–0.039 mm, ventral sucker is 0.016–0.029 x 0.020-0.029 mm. Brandes's organ 0.048-0.079 x 0.021–0.045 mm (Karatoy and Soylu 2006).

11.2. Epizotiyology: The adult parasite in the small intestine of a fish-eating bird sheds parasite eggs. these are delivered with the host faeces to the aquatic environment where they hatch and release free awimminng miracida. The first intermediate host, a pulmonate snail,become infected by penetration. In the snails a new larval stage, the sporocyst, develops. Tail-bearing larvae, cercariae, in turn appear in the sporocyst and are liberated to the water where they seek a fish host. When attached to the skinthe tail is lost and the invading process is initiated. Following penetration of the fish skin, the larva migrates in the lymphatic ducts to the final locality (the eye) in the fish. In the eye the larva develops into the metacercarial stage.

11.3. Pathogenicity: The movements and excretion from the metracercaria may cause disturbances of the eye function.

11.4. Symtomps: Normally metacercariae do not cause serious disease. However, penetration of cercaria into small fish ma elicit mortality.

11.5. Diagnosis: Detection of metacercaria in the vitreous humour of the fish.

11.6. Prophylaxis: Removal of intermaediaate snail hosts. Preventing access of final fish-eating hosts releasing parasite eggs. Mechanical filtration of pond water may reduce density of cercariae. Increase of water flow in ponds may flush away the positively phototactic cercariae.

11.7. Treatment: Praziquantel (330 mg/kg) may be used orally in feed (Buchmann and Bresciani 2001).

The cestoda or tapeworms are exclusively endoparasitc and hermoaphroditic with no asexual propagation in the intermediate host. Cestodes are oviparaous and have complicated life cycles which in many cases include free-swimming ciliated larvae called coracidia. Other cestode eggs infectt the host by hatching after ingestion. They have no mouth nor intestine. They feed by absorbing of substances through their tegument.

12. Cyathocephalosis

12.1. Aetiology: Cyathocephalus truncatus

12.2. Epizootiyology: Hexacanth embryos of C. truncatus were found to develop optimally in eggs cultured at between 15 °C and 20 °C for about 25 days. Gammarus pulex became infected only by swallowing egg capsules a containing hexacanth embryos fed to them. The young developing embryo grows over a period of about 10 weeks to the infective procercoid stage in the body cavity of the amphipod. In fish, the tapeworm forms an attachment in the distal end of a pyloric caecum 3 days after infection and matures in 8-10 days with production of eggs. By the 15th day the attachment to host tissue has become so firm that it is impossible to separate the worm from it. The tapeworm's hold on the caecal wall is probably achieved by the sucking effect of the funnel-shaped scolex supplemented by the spike-like microtriches of the inner scolex surface (Okaka 1984).

12.3. Pathogenicity: In heavily infected fish, the entire region of the pyloric caeca becomes fused into a single mass which may additionally fuse with the abdominal body wall immediately adjacent to it. In some cases, tapeworms were seen to have penetrated through the caecal wall and were attached directly to the muscles of the abdominal wall with the strobila still within the caecum. In other cases the whole tapeworm had moved through the perforated distal tip of the caecum and was found free in the body cavity. Worms could also be discharged into the intestinal tract with the detached distal, end of the caecum still held in the soolex as described previously. Caeca with perforated tips are common in heavily infected fish.

12.4. Symtomps: Heavily infected fish are often pale in colour, appearing very anaemic and showed blister-like pustules on the skin of the abdomen. Light infections do not elicit symptoms but mass mortalities in association with heavy infections have beeen described (Okaka 1984).

12.5.Diagnosis: Unsegmented cestode with a maximum length of 5 cm. The scolex is cup-shaped.

12.6. Prophylaxis: Removal of intermaediate snail hosts.

12.7. Treatment: Praziquantel, mebendazole or albendazole may have effect due to their anticestodal activity (Buchmann and Bresciani 2001).

13. Proteocephalosis

13.1. Aetiology: Procephalus longicollis

13.2. Epizootiyology: The adult hermaphroditic cestode produce eggs which are liberated to the water. Following hatching the larava (coracidium) will be ingested by a copepode which in turn becomes infected with the procercoid stage. When the fish eats the copepod this

crustacean will get deigested whereas the cestode larva develops into the adult stage in the fish intestine. Adult cestodes are usually located in the anterior part of the intestine or caeca, while juveniles may be found throughout the intestine (Scholz 1999). Parasite abundance is highest in the period mid June - mid July

13.3. Pathogenicity: Mainly due to absorption of nutritive substances in the host intestine. Inflammation of intestinalepitthelium due to cestode attachment.

13.4. Symtomps: It has been suggested that infections with P. longicollis reduce the growth of rainbow trout (Priemer and Goltz 1986, Engelhardt et al. 1988).

13.5. Diagnosis: Detection in intestine of cestodes with four suckers.

13.6. Prophylaxis: Removal of intermaediate copepod hosts.

13.7. Treatment: Praziquantel (Buchmann and Bresciani 2001).

14. Eubothriosis

14.1. Aetiology: Eubothrium crassum E. salvelini

14.2. Epizootiyology: Transmission of E. crassum occurs when fish ingest infected copepods, and the first site of E. crassum is either the stomach or intestine, where digestion of the copepod happens. Growth only commences when a plerocerciform worm moves into a caecum, and larger worms are more likely to be found in anterior caeca, the preferred site of E. crassum (Kennedy 1996). The increase in prevalence and abundance of E. crassum occur from late March to mid May.

14.3. Pathogenicity: The presence of the cup-shaped scolex in the tip of the pyloric caecum induces an extensive ball-shaped tissue reaction.

14.4. Symtomps: infection by E. crassum may have a negative effect on host condition factor.. Parasite cause direct mortalities among the heavily infected fish, and indirect mortalities by predation from reduced growth, poor swimming performance and aberrant behaviour. The negative growth effects highlighted are considered caused either by poor host nutrition due to demands of the parasite or from reduced food gathering capabilities (Smith 1973).

14.5. Diagnosis: Detection of cestodes in the pyloric caeca and intestine.

14.6. Prophylaxis: Removal of intermaediate copepos hosts. Filtration of water.

14.7. Treatment: Di-n-butyl tin oxide 80-160 mg/kg three days (Mitchell 1993)

15. Diphyllobothriosis

15.1. Aetiology: Diphyllobothrium is a genus of tapeworm which can cause Diphyllobothriasis in humans through consumption of raw or undercooked fish. The principal species causing diphyllobothriosis is Diphyllobothrium latum, known as the broad or fish tapeworm, or broad fish tapeworm. D. latum is a pseudophyllid cestode that infects fish and mammals. D. latum is native to Scandinavia, western Russia, and the Baltics, though it is now also present in North America, especially the Pacific Northwest. In Far East Russia, D. klebanovskii, having Pacific salmon as its second intermediate host, was identified (Muratov and Posokhov 1988).

15.2. Epizootiyology: Adult tapeworms may infect humans, canids, felines, bears, pinnipeds, and mustelids. Immature eggs are passed in feaces of the mammal host. After ingestion by a suitable freshwater crustacean such as a copepod (the first intermediate host), the coracidia develop into procercoid larvae. Following ingestion of the copepod by a suitable second intermediate host, typically a minnow or other small freshwater fish, the procercoid larvae are released from the crustacean and migrate into the fish's flesh where they develop into a plerocercoid larvae. The plerocercoid larvae are the infective stage for the definitive host.

15.3. Pathogenicity: Inflammatory reactions in the affected tissue.

15.4. Symtomps: Clinical signs of diphyllobothriasis are generally mild, and can include diarrhea, abdominal pain, vomiting, weight loss, fatigue, constipation and discomfort in human. Approximately four out of five cases are asymptomatic and may go many years without being detected (Scholz et al. 2009). In a small number of cases, this leads to severe vitamin B12 deficiency due to the parasite absorbing 80% or more of the host's B12 intake, and a megaloblastic anemia indistinguishable from pernicious anemia (David and William 2006).

15.5. Diagnosis: Detection of pleurocercoids in the fish viscera of flesh.

15.6. Prophylaxis: Contamination of the environment with worm eggs should be prevented.

15.7. Treatment: Praziquantel may have effect (Buchmann and Bresciani 2001)

16. Triaenophorosis

16.1. Aetiology: Triaenophorus crassus, T. nodulosus

16.2. Epizootiyology: Adults of T. crassus and T. nodulosus occur in the intestine of the pike, Esox lucius L. The first intermediate hosts of Triaenophorus spp. are cyclopoid copepods and in European waters Cyclops vicinus and C. strenuus are the most important first hosts of T. crassus (Kuperman 1973). Plerocercoids of T. crassus occur in the musculature of the fish host, and in wild populations of salmonids and coregonids may cause loss of quality in fish products. Infection first occurs in early June when the trout fry have been moved into the

cages (Bauer and Solomatova 1984).

16.3. Pathogenicity: Severe inflamatory reactions in muscles due to infectins with T. crassus. The plerocercoids may exert pressure on organs and thereby disturb normal physiological function.

16.4. Symtomps: Parasite cause letthargy and mortality of fingerlings.

16.5. Diagnosis: Detection of plerocercoids in the organs or muscles of the intermaediate host. The worms have four characteristic hooks shaped as three-toothed combs in the scolex.

16.6. Prophylaxis: Removal of intermaediate copepod hosts. Any pike in the surrondings should be removed. Water filtration prevent to contaminate of copepods.

16.7. Treatment: Anticestodal drugs such as praziquantel mat have effect (Buchmann and Bresciani 2001).

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