# Parasitic Diseases And Their Controls In Sustainable Development Of Aquculture Of Bluefin Tuna (Thunnus Thynnus)

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## Abstract

In the last decades Turkish and Mediterranean mariculture has focused its production mainly on two species, gilthead sea bream (Sparus aurata L.) and sea bass (Dicentrarchus labrax L.).

However, due to its high commercial value bluefin tuna (Thunnus thynnus) has been considered as an alternative aquaculture target. The culture of bluefin tuna has started by private sector in 2001 in Turkey. The catched fishes are fed by frozen herring, sardina, mackerel between May and June and after fattened, they are sold. Total feeding time in Turkey 4 to 10 months like other countries which are culturing bluefin tuna. As an developing sector in Turkey and in the world, blue fin tuna farming require to further studies on larvae production, feed investigations and diseases occur on the fish. According to studies conducted to date, 28 species were found in Pylums Ciliphora, Myxozoa, Platyhelmintes, Aschelmintes and Arthropoda. The present work aim to reveal diseases of bluefish tuna caused by parasites, how they are transmitted, which effects they have on tuna fish, how they could be diagnosed, and how they could be controlled and treated.

***Keywords:*** Bluefin tuna, disease, parasite, diagnosis, treatment.

## 1.INTRODUCTION

Aquaculture is one of the fastest growing industries in Turkey having grown in volume by over 20 percent for the past ten years (Table 1). Production of the three major species, namely rainbow trout (Oncorhynchus mykiss), seabass (Dicentrarchus labrax) and seabream (Sparus aurata) increased rapidly during the 1990s, with efforts having been given to the development of new species, such as the Black Sea turbot (Scophthalmus maeoticus) and some Mediterranean species such as sharpsnout seabream (Diplodus puntazzo), common seabream (Pagrus pagrus), common dentex (Dentex dentex) and groupers (Epinephelus spp.). Atlantic bluefin tuna (Thunnus thynnus) fattening, which started at the turn of the millennium has been the latest development in terms of species diversity.

| Species | Farm number | Rate (%) | Capacity (tonnes) | Rate (%) |
|---|---|---|---|---|
| Seabass/seabream | 309 | 87 | 99 869 | 74 |
| Trout | 5 | 1 | 820 | 0.6 |
| Shell | 4 | 1 | 1 925 | 1.4 |
| Tuna | 8 | 2 | 7 440 | 5.5 |
| Trout & seabass | 14 | 4 | 9 613 | 7.2 |
| Seabass & bream & new species | 13 | 4 | 9 529 | 7.1 |
| Trout | 1 | 0 | 4 800 | 3.6 |
| Seabass & bream & trout | 1 | 0 | 100 | 0.1 |
| Seabass | 1 | 0 | 25 | 0 |
| Total | 356 | 100 | 134 121 | 100 |

Table 1. Number of marine fish farm, capacities and production in 2009.
(http://www.fao.org/fishery/countrysector/naso_turkey/en)

Tuna aquaculture is presently in its early stages of development and will likely continue to expand, but with this expansion, parasitic disease will become increasingly important. Thus, focus has been placed on parasitic infections in these enterprises and their economic and ecological impact. Diseases problems including parasitic organisms are the

main threat to further increas of the industry. There are various parasites caused the diseases on tuna fish. This research presents the individual parasites types producing problems in tuna fish. 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.

## 2. Coccidiosis

### 2.1. Aetiology. Goussia auxidis

Mature oocysts (18-28 µm diameter) occur in the splenic pulp and in melanomacrophage centres in the spleen and liver, and in the peribiliary connective tissues of the liver. The oocyst has a thin membranous wall, and each contains four sporocysts but no oocyst residuum. Sporocysts (length 8-12 x 6-7 µm) have a thickened wall with a faint dehiscence suture occasionally visible (Jones 1990).

2.2. Epizootiology: The method of transmission of infection is unknown. 98 % (140 of 143) of albacore, T. alalunga, and an individual yellowfin tuna, T. albacares, from the South Pacific were found be infected (Jones 1990).

2.3. Pathogenicity: Oocysts occurred in both the liver and spleen where they produced minimal host response. Host response appears to be minimal with slight proliferation of fibroblasts around the oocyst in some fish. The spores are positively associated with melanomacrophage centres but the degree of association varies from fish to fish (Jones 1990).

2.4. Symptoms: No clinical signs have been reported.

2.5. Treatment and Prophylaxis: Not required.

## 3. Uronemosis

3.1. Aetiology: Uronema nigricans

Fixed and stained parasite is variable in size ranging from 19.8-34 µm in length by 7.1-20 µm in width. They contained distinctive somatic ciliature consisting of 12-14 longitudinal kineties arranged in meridional rows (Munday et al. 1997).

3.2. Epizootiology: Until recently, infections of fish with Uronema sp. had been mainly reported in aquarium species in which they were invasive and resulted in severe tissue damage and subsequent mortalities (Cheung et al. 1980, Bassleer 1983). More recently the potential for scuticociliatids to be a problem in mariculture has been highlighted by reports of

serious disease of larval and juvenile fish in Japan and Spain attributed to infection with Uronema sp. (Yoshinaga and Nakazoe 1993, Dykova and Figueras 1994) and of mature sea bass in the Mediterranean, where parasite was described as Philasterides dicentrarchi (Dragesco et al. 1995). In Australia mortalities of southern bluefin tuna Thunnus maccoyii in growout seacages have also been reported with the causative agent being identified as Uronema nigricans (Munday et al. 1997). Epizootiological factors which may be implicated in the initiation of the disease in SBT are water temperature and host immune status. The "swimmer" syndrome has not occurred when water temperatures have exceeded 18°C. SBT maintain a body temperature of about 24°C, even in much colder water, so it is possible that

the parasite, which would be expected to have an optimum growth temperature of about 25°C (Parker 1976), would be preferentially attracted to the fish under conditions of relatively low water temperatures (< 18°C).

3.3. Pathogenicity: U. nigricans initially parasitizes the olfactory rosette at which stage the host mounts a vigorous inflammatory response. If the host response is inadequate, the ciliate then invades branches of the olfactory nerve present in the axis of the olfactory rosette. Even though there is still some host response to invasion of the olfactory nerve the migration of U. nigricans is probably then inexorable, ending in invasion of the brain, which causes locomotor dysfunction and, ultimately, death. Generalised infections leading to severe mortalities in larval marine fish were reported (Munday 1996).

3.4. Symptoms: Typically, affected tuna fish came to the surface, turned light blue and swam vigorously around the cage. Eventually, the fish ceased compulsive swimming and exhibited short bursts of forward motion with their heads out of water, followed by periods of sinking, before once again coming to the surface and then repeating the process. Finally, the fish sank and died at the bottom of the netpen (Munday et al. 1997).

3.5. Diagnosis: Presumptive diagnosis of the disease in SBT can be made by examining wet preparations of CSF and brain. However, although a fluorescent antibody test has been developed for cultured and environmental organisms (Watts 1995), this is not suitable for clinical material because of autofluorescence of host tissues. Definitive diagnosis can be made by microscopic examination of histological slides of nervous tissues. As clinical pathology only reflects general stress and perturbed osmoregulation, it is of no specific diagnostic value.

3.6. Treatment: The most effective concentrations of formalin were 100 and 200 ppm where total cell lysis occurred after 120 and 60 min respectively. Hydrogen peroxide was lethal to the ciliate at all concentrations (Crosbie and Munday, 1999).

## 4. Kudoasis

4.1. Aetiology: Kudoa prunusi, K. crumena

Typical Kudoa crumena spores measuring 7.5 x 9.9 µm and 4.4-5.6 x 7.8-10.0 µm. Average spore size of Kudoa prunusi is 9.63 µm in width and 7.50 µm in length (Meng et al., 2011).

4.2. Epizootiyology: K. crumena was reported in a yellowfin tuna (Kent et al. 2001). The infections described by Langdon (1990) were in southern bluefin tuna caught off southwestern Western Australia when the fish would have been 1-3 years of age and feeding on cephalopods, crustaceans and salps (Kailola et al. 1993). Infections have also been reported in southern bluefin tuna in South Australia (Rough 2000) and wild fish caught off the New South Wales coast where the prevalence of about 1% affected fish was a cause of commercial loss. The prevalence of K. crumena in albacore was reported as 5%. Kudoa sp. infection of northern and southern bluefin tuna producing lesions in the flesh.

4.3. Pathogenicity: The infection in southern bluefin tuna produces white cysts 1-10 mm in diameter which are apparently in the muscle (Rough 2000) although Langdon (1990) produced evidence to suggest that most, if not all, cysts in southern bluefin tuna were in peripheral nerves, especially the intercostal nerves. Histologically the cysts are found to consist of numerous Kudoa spores surrounded by a fibrous capsule. Similar lesions because of an unidentified Kudoa sp. have been reported in southern bluefin tuna and Langdon (1990) suggested that the parasite could be K. nova, but as it does not produce myoliquefaction, this seems unlikely.

4.4. Symptoms. None reported.

4.5. Diagnosis. Kudoa sp. can be seen in wet preparations or histological sections. Numerous plasmodia were localized not only in the cavity of the optic tectum but also in the tissue of cerebellum. Host inflammatory response and gliosis were occasionally found around plasmodia (Meng et al. 2011).

4.6. Treatment and Prophylaxis. Neither treatment nor prevention are practicable.

**5. Hexacapsulosis**

5.1. Aetiology: Hexacapsula neothunni

Spores measure 6.2-11.0 µm and have six shell valves each containing one polar capsule (Lom and Dykova 1992). Kudoa nova spores measure 5.3-6.5 x 8.5-9.8 µm and have four shell valves each containing one polar capsule.

5.2. Epizotiology: Most myxosporeans, for which life cycles are known, have a two-host cycle with the myxosporean in a fish and an actinosporean in an invertebrate (Kent et al. 2001). As most juvenile tuna consume invertebrates such as squid and crustaceans (Kailola et al. 1993), it is conceivable that these prey species could be alternative hosts.

5.3. Pathogenicity: The postmortem liquefaction of the muscle caused by the release of proteases from the parasites that is the most dramatic result of the infection (Ogawa 1996). Histologically, the myxosporean spores are found aggregated in the cystic structures and usually produce minimal host response.

5.4. Symptoms: The parasites produce no clinical signs, and, while with heavy infections cysts may be visible in the musculature

5.5. Diagnosis: Typical myxosporean spores can be easily found in wet preparations or histological sections of affected muscles.

5.6. Treatment and Prophylaxis. Neither treatment nor prevention is practicable.

**6. Cardicolosis**

6.1. Aetiology: Cardicola ahi, Cardicola forsteri

Adult flukes are dorsoventrally compressed and 100-150 µm in width, with marginal tegumental spines, parenchymous body cavity, and indistinct reproductive and digestive tracts (Cribb et al. 2000).

6.2 Epizootiyology: C. ahi has been reported from yellowfin and bigeye tunas (Smith 1997). C. forsteri (Cribb et al. 2000) occurs in southern bluefin tuna. The intermediate hosts of Cardicola ahi and C. forsteri are not known. It is also not known if teleosts, other than Thunnus spp., act as final hosts. Colquitt et al. (2001) reported that the prevalence and severity of the infection increased with the time that southern bluefin tuna were held in captivity suggesting that the life cycle was maintained in the vicinity of the cages. Additionally, tuna farmers have reported that infections tend to be more severe at new cage sites suggesting that the parasite may also have a deleterious effect on the intermediate host.

6.3. Pathogenicity: Multifocal, white to yellow lesions involving the gills of infected southern bluefin tuna are described. The lesions ranged in size from 2 to 12 mm and often extended in an arc across the gills. The lesions appeared to be the result of the fluke ova impacting in the

afferent filamentary arteries where they stimulated a host response. Cardiac lesions were also reported and noted many ova surrounded by granulomas. There was marked hypertrophy of the cardiac spongiosa, presumably because of increased resistance to blood passing through the partly occluded branchial vasculature (Colquitt et al. 2001). It may not be a coincidence that bigeye tuna, which are known to be infected with C. ahi, have a much more compact ventricular myocardium (74%) compared with northern bluefin tuna (30–50%) which have not been reported to be to be infected with blood flukes (Santer and Greer-Walker 1980, Smith 1997).

6.4. Symptoms: C. forsteri infections of cultured southern bluefin tuna lead to increased mucus on the gills and have been associated with signs of respiratory distress, lethargy and slightly increased mortality (Rough 2000).

6.5. Diagnosis: In southern bluefin tuna the gross lesions are characteristic enough to enable a presumptive diagnosis. Histopathology is even more diagnostic, but definitive diagnosis depends upon flushing the adults from the heart and identifying them.

5.6. Treatment and Prophylaxis: Prevention of blood fluke infections depends upon an understanding of the parasites life cycle and, therefore, is not possible at present.

## 7. Didymocystiasis

7.1. Aetiology: Didymocystis wedli

The membranous cysts on the gill filament are oval in shape and measure 3-6 mm in length by 1.5-2.8 mm in width, depending on the stage of development and contain two similar worms applied closely against each other with opposite extremities. Under higher magnification we can observe the porous surface of the cyst. The characteristic feature of the body shape of D. wedli, with two distinct parts, can be well observed by light and scanning electron microscopy. The forebody is elongate, cylindrical, and slender, measures 1-2.2 mm long by 00.1-0.3 mm wide. The tegument of ventral and dorsal surfaces of forebody is wrinkled cobblestone-like, without spines or papillae. At higher magnification the oral opening is seen in the retractil tip of the forebody. Sensory papillae were not observed around the oral opening. The large hindbody has two symmetrical rounded lobes on the anterior end, forming a groove, from which emerges the elongated forebody; the posterior third of the hindbody is curved ventrally forming a somewhat pointed tail. The hindbody measure 3.4-10.2 mm in length by 0.9-3.1mm largest widht. The dorsal surface is more prominent than the ventral, forming a cover with tegumental transversal striations. There are no spines or papillae on either the ventral or the dorsal tegumentary surface of the hindbbody, which presents the same wrinkled cobblestone-like appearance (Kohn et al. 2001)

## 8. Anisakisasis

8.1. Aetiology: Anisakis simplex and Hysterothylacium cornutum (Williams and Bunkley-Williams 1996).

8.2. Epizootiyology: The definitive hosts of these parasites are marine mammals. Many other species of fish can act as intermediate hosts.

8.3. Pathogenicity: The small third-stage larvae are found encapsulated in the peritoneal mesenteries and, sometimes, the liver. If the fish are not quickly eviscerated it is possible for

the larvae to migrate to the abdominal muscles. Anisakis sp. infections are of importance because they can potentially infect humans.

8.4. Symptoms: The infections are covert.

8.5. Diagnosis: The presence of tightly coiled, encapsulated larval nematodes in the mesenteries of tunas is suggestive of anisakid nematode infection, but definitive identification of the larvae can be difficult.

8.6. Treatment and Prophylaxis: Human infection can be prevented by rapid evisceration of the fish and/or cooking of the flesh. In most instances tuna destined for sashimi are eviscerated soon after capture. There is no practicable treatment.

## 9. Copepodiasis

9.1. Aetiology: C. elongatus, Euryphros brachypterus, Penella filosa and Pseudocycnus appendiculatus

Overall length of female of Pseudocycnus appendiculatus is 13.7 mm-15.6 mm. Anterior quarter of ventral length bright red, rest of body yellowbrown, the red pigment persistent in alcohol for at least several months. Cephalothorax subovate, (1.2 mm-1.3 mm x 1.2 mm), greatest width a little posterior to midpaint. Second thoracic segment subovate, length/width (0.6 mm-0.8 mm x 1.1 mm). Third thoracic segment, length/width (0.5 mm-0.6 mm x 1.0 mm-1.2 mm). Fourth thoracic segment subovate, length/width (1.7 mm x 1.2 mm-1.3 mm). Trunk, excluding the anterior swollen portion, cylindrical, length more than 6 times width (6.6 mm-7.9 mm x 1.0 mm-1.1 mm), lateral margins parallel with 2 plate-like subsemicircular dorsal projections, % trunk width, length % width, carried laterally on posterior margin. Abdomen, length % width (0.6 mm-0.7 mm x 0.9 mm-l.0 mm), caudal laminae borne laterally on posterior margin of abdomen, length 12 times basal width (3.4 mm-3.8 mm x 0.3 mm-0.4 mm). Egg strings extending from posterior margin of trunk, beneath platelike projections dorsal to abdomen, present on only 1 specimen, as long as body, eggs uniserial, length 13.7 mm, posterior of egg strings empty of eggs in this individual (Hewitt 1969, Purivirojkul 2011).

9.2. Epizootiyology: As C. elongatus and Penella filosa have multiple hosts, tuna may become infected from a variety of sources. However, Euryphorus brachypterus is almost genus specific for Thunnus spp. (Williams and Bunkley-Williams 1996). In the case of C. elongatus infecting captive southern bluefin tuna, capture trauma and high stocking densities are believed to predispose to heavy infections (Rough et al. 1999).

9.3. Pathogenicity: Lesions because of the copepods are related to their grazing behaviour (C. elongatus) or the damage caused by their attachment to the host (Euryphorus brachypterus and Penella filosa). Opportunistic infections of the bacterium Aeromonas sp. have also been reported in association with louse-associated damage to tuna eyes (Munday et al. 2003).

9.4. Symptoms: C. elongatus grazes on the integument of southern bluefin tuna and may produce grazing trails including over ocular tissues. Damage to the eye results in keratitis, panopthalmitis, cataract formation and blindness, leading to significant production losses (Rough 2000, Munday et al. 2003). Very heavy infections of Euryphorus brachypterus have been reported in northern bluefin tuna in which the pseudobranch has been carpeted with the parasite leading to ulceration and bleeding (Rough et al. 1999). Similar, but less severe lesions may be present on the gills and skin. The very large copepod Penella filosa penetrates into the

muscles of a number of tuna species. It has been reported to cause the fish considerable discomfort (Williams and Bunkley-Williams 1996).

9.5. Diagnosis: Experts can make a presumptive diagnosis of these copepod infections based on the morphology of the parasites and the types of lesions induced by their activities. However, definitive diagnosis is only possible by a scientist skilled in identifying the parasites.

9.6. Prophylaxis: Trauma may predispose to C. elongatus infections then reduction of damage because of capture, towing and harvesting should simultaneously reduce the level of infestation/damage caused by this copepod. Additionally, as this parasite and Penella filosa are carried by other species of fish, it would be appropriate to keep other forms of aquaculture separate from tuna farms (Rough et al. 1999).

9.7. Treatment: Although a number of therapeutants are capable of killing copepod parasites (Lester and Roubal 1999) it is impracticable to use these agents under current tuna aquaculture conditions. In addition, at the present level of loss of production, such treatments would be uneconomical.

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