ParaFishControl

GUIDE 2 – FISH FARMER'S GUIDE TO COMBATING PARASITIC INFECTIONS IN TURBOT AQUACULTURE

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A Series of ParaFishControl Guides to Combating Fish Parasite Infections in Aquaculture. **Guide 2**

ParaFishControl

> “The experimenter who does not know what he is looking for will not understand what he finds.”
>
> *Claude Bernard*

Intensive culture of turbot, *Scophthalmus maximus*, is a successful and well consolidated industry in Western Europe and, more recently, in Chile and China. Modern turbot aquaculture is usually carried out in flow-through, land-based facilities with a high level of process control technology. Even in successful turbot farms, a few parasitic diseases can have a devastating influence on the production of this species. This guide focuses on the two main parasitic diseases that affect turbot culture, providing clear, practical information and advice for fish farmers.

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Introduction

The turbot, *Scophthalmus maximus* (Linnaeus, 1758), is a flatfish species belonging to the family Scophthalmidae, order Pleuronectiformes. This species is distributed throughout the Northeast Atlantic, the Baltic Sea, the Mediterranean Sea and the Black Sea. It has a disc-shaped body with marked asymmetry in juveniles and adults, with both eyes occurring on the left side of the head. Although the species lacks scales, its head and body are covered by numerous bony protuberances or tubercles. The dorsal side is grey–brownish in colour and has dark spots, although the colouration can vary greatly depending on the background. The ventral side is whitish in colour. Turbot are carnivorous fish and can reach up to 1 m in length and up to 25 kg body weight. The flesh is greatly sought-after and has high commercial value.

Turbot aquaculture began in the 1970s in Scotland and was then introduced to other European countries (Source: FAO). Turbot aquaculture has since expanded to Chile and China. In 2017, the total turbot production in Europe was 11,571 MT, with Spain being the main producer (8,546 MT). The world turbot production in the same year was estimated to be 59,616 MT (Source: APROMAR, 2018). Cultured turbot larvae are fed microalgae for the first 3-5 days, followed by rotifers (*Brachionus plicatilis*) and then by *Artemia* and conventional weaning diets. The high mortality of larvae is a serious problem during the hatchery period. Juveniles (aged 2-3 months) are raised in a nursery until they reach about 20-30 g in body weight. Fish are then transported to on-growing facilities, where they can be reared in square or circular onshore tanks, with open or recirculating seawater systems, or in submerged, flat-bottomed cages.

The **ParaFishControl** project has provided a strong basis for future success of European aquaculture by investigating and developing new diagnostic and control systems for parasitic infections. In this guide, fish farmers can find background information on the two main parasitic diseases that affect farming of turbot in European countries. Here the farmer can find valuable information on clinical signs, identification of the pathogen, biology of the parasite, the life cycle and recommendations for control. This guide does not provide comprehensive details but may serve as an easily comprehended and necessary support during the daily handling and management of these fish. The guidance provided for individual parasites reflects the current state of knowledge for these pathogens and has been informed, in part, by research conducted through the **ParaFishControl** project.

Guides are provided for the two parasites that generate the highest mortality rates in turbot, the ciliate *Philasterides dicentrarchi*, which causes scuticociliatosis, and the myxozoan *Enteromyxum scophthalmi*, the cause of enteromyxosis. For each parasite, we provide a background on its biology, an examination of key risks for infection and disease progression, and up-to-date guidance for the management and control of the parasite. Since the expression of parasitic disease can be affected by many different factors and is therefore very much site, stock and environment dependent, instigation of any of the suggested control measures should be accomplished with the assistance and guidance of suitable fish health professionals, including veterinary practitioners and farm based health and welfare professionals. These guides also provide details for expert contacts within Europe who may be consulted for further support.

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1. Fish farmer's guide to combating *Philasterides dicentrarchi* infections in turbot

Introduction

Figure 1. *Philasterides dicentrarchi.* Photo : J. Manuel Leiro, University of Santiago de Compostela.

Philasterides dicentrarchi is a free-living scuticociliate found in seawater habitats, with a worldwide distribution. This ciliate (about 25 x 45 µm) is fusiform, with a pointed anterior and rounded posterior end, a prominent caudal cilium and 10-15 (usually 12-14) somatic kineties (Figures 1 and 2). It has a micro- and a macro-nucleus, the latter mainly located in the middle-anterior part, several digestive vacuoles of variable size and a contractive vacuole located at the posterior end. This ciliate feeds on smaller organisms such as bacteria or microalgae, and also on organic matter present in sea water and surfaces. Under favourable conditions, it can behave as a facultative parasite infecting fish and causing high mortalities. Many fish species kept in captivity have been affected by scuticociliatosis caused by *P. dicentrarchi*, including farmed and aquarium fish. However, the highest economic losses are reported in turbot and other farmed flatfish (olive flounder, *Paralichthys olivaceus* and fine flounder, *Paralichthys adspersus*). Virulent parasites mainly enter the fish through skin or gill lesions and then proliferate in the blood and internal organs causing a systemic infection. *P. dicentrarchi* can be found in the open sea and continuously enter fish farms, reaching seawater channels / pipes or tanks. Overall, the ciliates are more abundant in tanks, probably due to the presence of a higher amount of organic matter.

Biological life cycle

P. dicentrarchi can occur as two morphologically distinct stages, a microstome active form and a smaller non-feeding form called a tomite. Microstomes can feed on bacteria or organic particles and after reaching a certain size can divide asexually. When food is scarce and compatible partners are present, microstome forms may undergo conjugation, interchanging genetic material. Microstomes can transform into tomites in response to starvation.

Figure 2. Micrographs of *Philasterides dicentrarchi* under bright field microscopy (A) and Nomarski microscopy (B). The cilia (red arrows) and caudal cilium (black arrows) are shown. Macronucleus (M). Bar = 25 µm. Photo: Jesús Lamas, University of Santiago de Compostela.

Seasonality

Scuticociliatosis tends to occur in spring, summer and autumn months, when seawater temperature is highest (especially above 18 ºC).

Age / mean weight susceptibility

Smaller fish, especially those with immature innate immune systems, are more susceptible to infection than older fish.

Risk predisposing factors

Important risk factors include inadequate hygiene of tanks (leading to high levels of organic matter and bacteria in the water), size of fish (smaller fish are more susceptible), high densities of fish (increasing the presence of skin lesions and generating stress in fish) and high seawater temperatures.

A) What clinical signs should alarm me?

External signs

External clinical signs of turbot affected by scuticociliatosis usually include changes in skin colouration, with fish either showing discolouration or darkening of the skin, and swimming disturbances. Some affected fish may have skin ulcers (mainly at the opercular level), exophthalmia and a distended abdominal cavity (Figure 3).

Internal signs

P. dicentrarchi can invade most organs, including the brain and the heart, resulting in systemic infection. Organs and tissues show haemorrhages and necrosis in the areas where the parasite has proliferated.

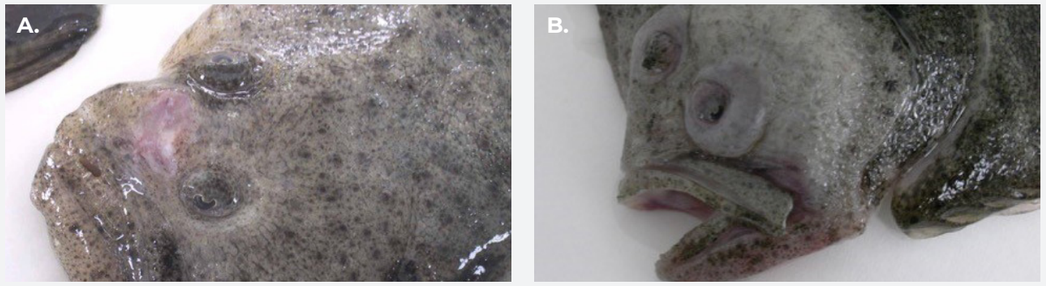

Figure 3. Naturally infected turbot showing characteristic signs of scuticociliatosis: A) skin ulcer in the naso-olfactory area and B) periorbital inflammation. Photos: C. Zarza, Skretting-ARC.

B) How to detect the parasite at farm level

1. Monitoring plan (what to measure and how often) and trigger level for action

Special care should be taken during summer months. *P. dicentrarchi* can easily be obtained from infected fish and identified by light microscopy. If exophthalmia and repetitive swimming movements occur, samples of periorbital and / or cerebral tissue should be examined. Slides containing squash preparations of fresh tissue / organs, or a drop of ascitic fluid can be observed under a bright field microscope by closing the iris diaphragm of the condenser, or under phase contrast or Nomarski microscopy (Figure 2). Ciliates are mobile and much larger than fish cells, and therefore easy to identify in the preparations. Water or sediment samples can be taken to evaluate parasite load by qPCR.

2. Recommendations for the submission of samples to be diagnosed

Tissue or ascitic fluid samples containing the parasite can be added to a few drops of sterile sea water diluted in distilled water 1:1 (v/v) at room temperature and sent refrigerated to the laboratory as soon as possible. For a more accurate identification, samples may be frozen at -20 °C or fixed in ethanol 70% and sent to the laboratory to be analysed by molecular methods.

3. Contact laboratories

- Laboratory of Parasitology, Institute of Research on Chemical and Biological Analysis (IIAQBUS), Rúa de Constantino Candeira, 15705 Santiago de Compostela, Spain.
- Fish Parasites Diagnostic Service, Institute of Aquaculture Torre de la Sal (IATS-CSIC), 12595 Castellón, Spain.

C) Action plan after diagnosis

1. Prevention and farm management

Since *P. dicentrarchi* feeds on bacteria or organic matter derived from fish food or from dead animals, it is crucial to keep tanks as clean as possible. It is also important not to overcrowd the tanks or overfeed the fish. Farming of turbot larvae in hatcheries in ciliate-free seawater is recommended. Finally, to generate protection against *P. dicentrarchi* , vaccination of fish with an autovaccine (i.e. one that contains the ciliate serotypes present in the fish farm system as antigen) is also recommended.

2. Treatment

Since *P. dicentrarchi* causes systemic infection, the best method to decrease the spread of the disease is to reduce the amount of ciliates in seawater, especially the most virulent ones. Prompt removal of dead fish, moribund fish or any fish suspected of being infected from the tanks is recommended. Reducing the concentration of ciliates in the tanks as far as possible is also advised, following the methods indicated in the prevention section. No chemical treatments are available against scuticociliatosis.

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2. Fish farmer's guide to combating *Enteromyxum scophthalmi* infections in turbot

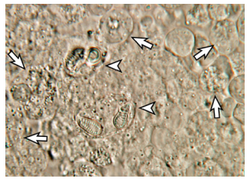


Figure 3. Fresh smear of intestinal tissue containing developmental stages (arrows) and immature spores of *Enteromyxum scophthalmi* (arrowheads) Photo: O. Palenzuela, IATS-CSIC.

Introduction

Enteromyxum scophthalmi (Figure 3) is a parasite causing a serious disease in turbot, namely the sunken-head syndrome, or turbot enteromyxosis. This parasite is closely related to another species, *Enteromyxum leei*, a common parasite of gilthead sea bream and other fish. The development of *E. scophthalmi* in the intestinal epithelium of turbot, and the inflammatory response that it triggers, usually cause a severe subacute catarrhal enteritis leading to disruption and failure of the intestinal barrier, severe dehydration, cachexia, and death. The infection can have a devastating impact, as it usually results in heavy losses (up to 100 % mortality) which can affect entire farms.

Biological life cycle

Enteromyxum scophthalmi belongs to the myxozoans, a group of parasites related to cnidarians. Their life cycle generally involves two alternating hosts: a fish and an aquatic annelid. However, *Enteromyxum* spp. have a unique ability to be transmitted directly from fish to fish (Figure 4). The catarrhal response triggered by the parasite causes shedding of epithelial tissue and mucous casts containing developmental stages of the parasites, which can survive for short periods of time in the water until they reach and infect other fish. This direct transmission has been reproduced experimentally by cohabitation, waterborne contamination, and by eating infected material, and these mechanisms facilitate quick contagion of the disease in farmed stocks. The infection has a long prepatent period, during which infected animals can release large numbers of parasites and multiply the infection exponentially, until affecting the entire stock.

The developmental stages or trophozoites are the ones responsible for the contagion. Like other myxozoans, the development of *E. scophthalmi* in the fish culminates with the development of spores, but these are not infectious for fish. Indeed, spores are usually scarce in turbot and present only during terminal stages of the infection. It is presumed that these spores can reach an invertebrate host, possibly a polychaete, although this is yet unknown for this species. In the invertebrates, other myxozoans undergo sexual reproduction and develop into a different type of spore (actinospores) that is the stage infective for fish. This natural cycle likely sustains *E. scophthalmi* populations in the wild, using unknown hosts, whereas the epizootic episodes in intensive farms occur exclusively due to the direct transmission and exponential amplification of parasite numbers favoured by the high fish biomass concentration.

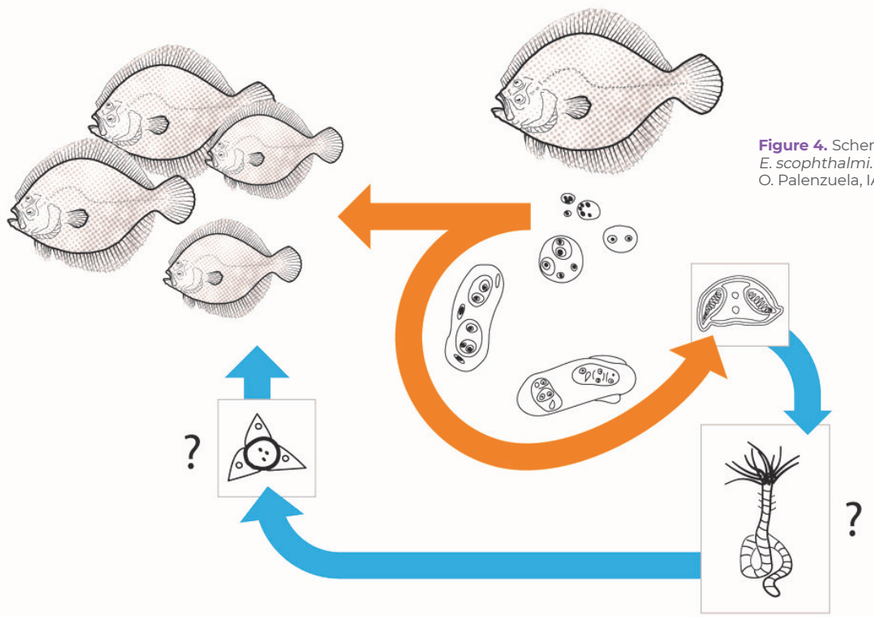


Figure 4. Schematic life cycle of *E. scophthalmi*. Illustration: O. Palenzuela, IATS-CSIC.

Seasonality

Water temperature is a critical factor affecting the onset of enteromyxosis of turbot, as it greatly determines the parasite's multiplication and subsequent development of clinical signs, as well as the transmission rate. Outbreaks usually occur at temperatures between 14 and 20 °C. In experimental infection studies at 18 °C, mortality begins approximately 5-6 weeks after exposure and can reach 100 % in approximately 12 weeks. The parasite's development slows down at lower temperatures and it is practically arrested below 11-12ºC. However, the dynamics of epidemic episodes in farm settings are complex because the emergence of clinical signs and mortality is affected by additional host and environmental factors, as well as by the infective pressure in the system. In a new batch of fish introduced in enzootic on-growing facilities, a relatively long pre-patent period (>3 months) is characteristic, during which the parasite load increases exponentially before it reaches a level detectable by routine performance indicators and health checks.

From a practical perspective, this means that parasite outbreaks in a farm are usually only noticed after a fish stock has already been resident in an enzootic farm for a few months at temperatures over 14-15ºC. Thus, stocks introduced in spring (April) start displaying clinical signs in the next autumn or winter, whereas fish introduced in autumn will display them the next spring and summer. The development of clinical signs and the mortality will slow down in the cold season, but they don't disappear completely in most farms and they will usually relapse as the temperature rises.

Age / mean weight susceptibility

All sizes and age classes of turbot are susceptible to the infection. However, in farm conditions, the disease tends to be first noticeable with mortality and clinical signs in the largest stock, which usually accumulates the longest exposure time. During the outbreaks, intermediate size classes (100-300g) usually display the highest prevalence and intensity of infection. Because younger turbot are most often maintained in nursery facilities with some degree of water filtration, lower age and size classes (< 100g) are usually the least affected as these stocks will not get epidemic levels of infection until a few months after they are exposed to contaminated raw water in on-growing facilities. Senegalese sole (*Solea senegalensis*) is susceptible to the infection, but it does not usually display serious clinical signs or mortality. It is very likely that other fish hosts can harbour the parasite in the wild and act as vectors, with variable susceptibility, as in the related species *E. leei*.

Risk predisposing factors

Enteromyxosis has been described in all types of turbot farming facilities (RAS, raceways, concrete / PVC tanks and sea cages). However, the vast majority of turbot production is carried out in flow-through onshore tank-based farms. The single most important factor related to catastrophic outbreaks in these facilities is the water reuse or recirculation, as it facilitates the exponential multiplication of the parasite and its rapid transmission to the entire farm stock. Different degrees of water reuse can occur due to the farm design (e.g., semi-closed systems or raceways) and these should be completely avoided to prevent the infection. However, most importantly, recirculation of water containing parasites can occur behind the scenes due to the influence of water emissaries in the farm intake. This can be aggravated by poorly designed infrastructures, but also by local dynamics that are difficult to predict. Tidal effects and currents, limited water exchange (e.g., in lagoons or bays), or civil infrastructures altering the local conditions (breakwaters, piers, etc.) can become triggering factors for the emergence of enteromyxosis outbreaks.

The second most important factor is the temperature, as explained under the seasonality section. In general, facilities reaching temperatures above 18-20 ºC during substantial periods of time are at a higher risk of outbreaks and present a worse prognosis when cases appear.

Other risk and aggravating factors are related to the farm management policies, as they will affect the speed and degree of spreading of random spontaneous infections to epizootic levels. High biomass densities, poor water exchange, accumulation of faeces in the tanks, infrequent removal and / or inappropriate disposal of dead fish, poor hygiene and disinfection policies, lack of specific targeted diagnosis and contingency plans all contribute to an increased risk of parasite infection and spread.

A) What clinical signs should alarm me?

External signs

In turbot, clinical enteromyxosis is characterized by cachexia and mortality. This is usually preceded by nonspecific signs such as anorexia, weight loss, and lethargy. Severely affected fish usually display muscle atrophy, and the head and eyes appear “sunken” below conspicuous bony ridges between the eyes (Figure 5).

Internal signs

At the necropsy, the intestinal wall appears thinned, with frequent congestion and haemorrhages. Pale organs and presence of seromucous contents in the intestine and ascites are commonly observed (Figure 6).

Figure 5. External clinical signs of turbot enteromyxosis. Note the muscle atrophy, sunken eyes and conspicuous cranial bony ridges. Photo: O. Palenzuela, CSIC.

Figure 6. Internal signs of turbot enteromyxosis. Note the congestive and haemorrhagic intestine. Photo: C. Zarza, Skretting ARC.

B) How to detect the parasite at farm level

1. Monitoring plan (what to measure and how often) and trigger level for action

In a farm without a history of enteromyxosis, the disease typically emerges for the first time in older age classes. Therefore, the mortalities in these stocks displaying clinical signs should be tested. The parasite is difficult to detect in fresh smears of the intestine examined under the microscope and, unless spores are present (Figure 3), diagnosis from these samples is very unreliable. The parasite can be confirmed with high sensitivity and specificity by qPCR from a small piece of tissue, preferably from the anterior intestine or pyloric caeca region. In farms at high risk or with a history of disease, monitoring of the infection by qPCR is recommended for all the batches, before the introduction to the farm and during the production cycle. A proactive targeted monitoring strategy is essential in order to promptly detect the infection and apply control measures before the parasite spreads to all the stocks.

2. Recommendations for the submission of samples to be diagnosed

Whole fresh fish can be sent on ice, but they should be delivered to the lab within 24 hours for microscopical examination. For histopathological work and qPCR, it is best to take the samples at farm level. qPCR samples can be taken as small pieces of intestine (i.e. < 1cm^2), preferably from the anterior portion, at the pyloric caeca level (behind the stomach) and fixed in 70 %-80 % ethanol. Non-lethal biopsies of the intestine mucosae can also be taken through the vent using clinical swabs, which should be placed in a preservative buffer. Samples for histopathological studies should be taken in 10 % neutral buffered formalin or other histology fixatives, as advised by the consulting laboratory.

3. Contact laboratories

- Fish Parasites Diagnostic Service, Institute of Aquaculture Torre de la Sal (IATS-CSIC), 12595 Castellón, Spain.
- Fish Pathology Diagnostic Service, Autonomous University of Barcelona. School of Veterinary, Campus UAB, Barcelona, Spain.
- Laboratory of Parasitology, Institute of Research on Chemical and Biological Analysis (IIAQBUS), Rúa de Constantino Candeira, 15705 Santiago de Compostela, Spain.

C) Prevention and action plan

1. Prevention

In land-based flow-through systems, treatment of inflow water with micro-filtration (e.g. 20-40 µm drum-filters), ozone and / or UV light reduces the risk of introduction of parasite stages in the farm. However, while these measures are effective to contain sporadic entry of infective stages from the wild, they cannot completely shield a farm against continuous infective pressure due to recirculation from an infected farm outlet. Intake water treatment measures must be adopted in conjunction with targeted surveillance focusing in the stocks at higher risk (i.e. older age classes), and when signs compatible with enteromyxosis appear. Serial raceways and other water reuse systems are inherently vulnerable to enteromyxosis and should be avoided: stocking the younger fish upstream in these facilities would be a better practice. Cage-based facilities should be placed in areas of moderate current (avoiding tidal currents) and cold water. In all systems, but particularly in RAS-based facilities, quarantine and PCR checkout of new stocks before introduction in the main system is of paramount importance.

Spatial containment of the tanks, age-segregation and a growth system based on multiple rearing units may contribute to avoiding cross-contamination of the stocks, should isolated cases emerge. In general, the infective stages responsible for the spread of the infection are relatively labile and do not survive standard hygienic routines. Prompt removal and examination of dead fish, and disinfection of all the shared material used for maintenance and handling must be reinforced.

2. Farm Management

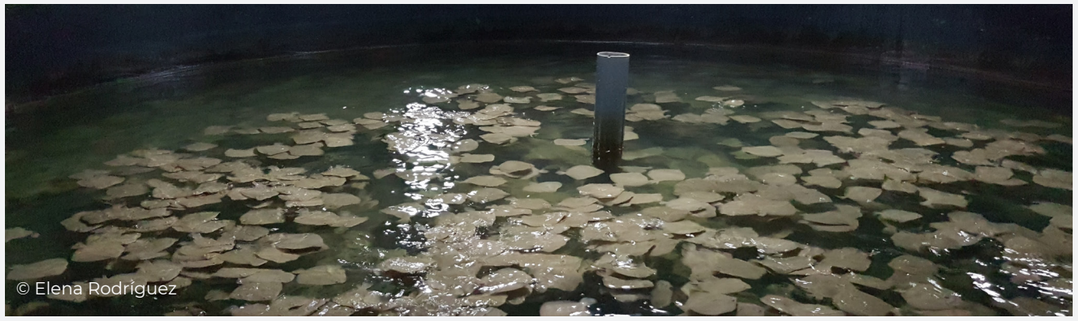
If cases of *E. scophthalmi* are confirmed, the affected tank(s) must be immediately isolated and culled. If the production cycle is based on stock splitting, additional tanks from the same cohort should be flagged, quarantined, and tested if they shared a tank in the previous 4-8 weeks. If the infection is spread among different stocks, the prognosis is poor, and depopulation of the entire farm must be considered. This should be followed by a careful auditing of the plant management and infrastructures, particularly the water intake and discharge, and the adoption of corrective measures.

3. Treatment

There are currently no registered prescription treatments effective against *E. scophthalmi* Some mixtures of antibiotic and anticoccidial compounds, such as Amprolium + Salinomycin, or Robenidine + Sulfonamides, were proved to be partially effective in experimental trials. The treatments delayed the onset of disease, mitigated the intensity of infection, the severity of associated intestine lesions, and the mortality. Natural extracts added in some infeed nutraceutical solutions (Skretting: SHIELD) showed a similar effect. However, none of these products could stop the progress of the infection and, eventually, all fish reached 100% prevalence.

4. Management of co-infections

Turbot with enteromyxosis could be affected by other pathogenic parasites such as the systemic ciliate *P. dicentrarchi* or the gill amoeba *Neoparamoeba perurans*. In general, due to the poor prognosis of animals with enteromyxosis and their role in the further spreading of the disease, farmers should focus on addressing this infection as a point of maximum priority.



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Other ParaFishControl Resources

1. Integrated Pest Management Strategies for *Philasterides dicentrarchi*: bit.ly/2RSh5xJ

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