27

Parasitic Diseases of Fish

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INTRODUCTION

The word 'parasite' comes from the Greek word's '*para*' meaning 'beside' and '*sitos*' meaning 'food'. The organisms known as parasites prey on and, in turn, injure their hosts after getting food and shelter from them. The parasites live in obligate association and get benefits such as nutrition at the host's expense, mostly without killing them. They use the energy that is otherwise required by the host for growth, development, maintenance, and reproduction and ultimately affect hosts survival (Overstreet 2021).

Fish Parasites

Fish parasites belongs to various classes and comprised of protozoans, pentastomes, trematodes, turbellarians, nematodes, cestodes, leeches, acanthocephalans, monogeneans, isopods, copepods, crustaceans, and lice. Their life cycles range from simple, needing no intermediate host, to complex and indirect, requiring one or more intermediate hosts. Fish can act as primary, paratenic, or intermediate host in the life cycle of parasites. Taking the life cycles of the identified parasites under inspection is very important for effective treatment. For instance, merely the theronts that is free-swimming developing life stage of the ciliated ectoparasite, *Ichthyophthirius multifiliis* are targeted at and affected by chemical treatments (Hoffman 1999, Roberts et al. 2001).

Diagnosis of Parasites in Fish

All infected fish must be tested or diagnosed using appropriate data which comprises an explanation of the fish's background from the owner of the fish, an assessment of water quality, an inspection of clinical indications, a physical test, an analysis of wet-mount cytology of skin scrapes, biopsy of gills and gathering the fecal samples (Reavill and Roberts 2007).

No specific indications of parasitic diseases in fish are seen but a group of symptoms may be observed. The general signs of parasitic infection include flashing behavior (scratching of body on the bottom of the tank or pond), sluggishness, skin bruises along with loss of scale, sores, formation of mucus, fast opercular motions, gasping, decrease in body weight, osmoregulatory disturbances, and morbidities (Roberts et al. 2007). External parasites may be seen clearly on gill cytology preparation and wet-mount skin of tranquillized fish. Internal infestations of parasites can be diagnosed by creating a wet mount of fresh fecal samples, gross visualization of the parasite at the outlet, evaluation of blood smears, histopathology, and necropsy inspection (Roberts et al. 2001, Roberts et al. 2007).

Protozoa

Ciliated Protozoans

White Spot Illness

Ichthyophthirius (multifiliis, sometimes known as "white spot illness" or "ich," is a parasitic disease that influences the fish living in freshwater across the world (Hoffman 1999, Baker et al. 2007, Noga 1996). The fish without scales i.e., catfish, is specifically in danger, because this parasite can live in a variety of temperatures and hosts. The systems that show overcrowding and bad status of water, causes more tension and decreased immune functioning in fish, which in turns raises the fatality rate. *I. multifiliis* can cause acute disease which may lead to 100% death rate (Noga 1996, Hadfield et al. 2007). The marine complement is *Cryptocaryon irritans*, both similar clinical symptoms (Roberts et al. 2009).

Life Cycle

The two parasites have a direct life cycle characterized by a free-swimming infective stage (theront) which is sensitive to the treatment. The feeding stage is enclosed within a sac called Trophonts like white nodes. These Trophonts burst out from the epithelium and turn into encysted tomonts having outer sticky capsules that attach to lifeless substrate in the environment, including gravel stones, nets, plants, and many more (Baker et al. 2007, Longshaw and Feist 2001). These tomonts split, generating tomites that breach the nodule's wall

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to release moving and disease-carrying theronts. The disease-causing theronts takes 48 hours to locate a new host at 25°C (Stoskopf et al. 1993, Noga 1996, Longshaw and Feist 2001). The thereont crosses the epithelium after obtaining a host and transforms into a ciliated trophont. *Ichthyophthirius* transmission is through the aerosol scattering of infective stage (Wooster et al. 2003). Ich has a temperature-dependent life cycle. At 25°C, it seems to last in 3 to 6 days, while at 15°C, it lasts about 10 days. At temperatures between 15 and 25°C, disease occurrence is most prevalent. Compared to *Ichthyophthirius*, *Cryptocaryon* has a longer life cycle, hence needed prolonged therapy (Roberts et al.2009).

Clinical Signs

Clinical signs include white, raised nodules up to 1mm (0.5mm for Cryptocaryon) on the skin and gills (Fig. 2), flashing, formation of mucus, sluggishness, shortness of breath, secondary bacterial or fungal diseases, and osmoregulatory disturbances due to the epithelial and gill damage. Upon examination of gills under the microscope, hyperplasia, more mucus, and tissue damage may be noticed (Reavill and Roberts, 2007).

Diagnosis

A wet-mount cytology of the skin or gills is inspected to confirm the diagnosis. Ich is a large sized parasite that is entirely covered in cilia, moves slowly and comprises of a nucleus that has the shape of alphabet C- or horseshoe (Fig. 3) (Noga 1996).

Chilodonella

Chilodonella is the condensed, ciliated parasite with a heart- or onion-shaped morphology. Striations that are evident on the parasite's length confirmed the existence of cilia. *Chilodonella* can flourish in brackish water and an array of temperatures. Its marine equivalent name is *Brooklynella hostilis*, which was found in the Brooklyn Aquarium. Both parasites can cause extreme tissue damage and serious sickness (Stoskopf et al. 1993; Noga 1996; Baker et al. 2007).

Clinical Signs

Clenched fins, mottled skin, enhanced formation of mucus, secondary skin ulcers, proliferation and merging of the lamellae, respiratory instability (gapping, piping, opercular flaring, augmented gilling), hypertrophy, and high fatality rates are the clinical indications (Palmeiro et al. 2009). Brooklynelliosis is a fetal disease that is cause by the ciliated protozoan *Brooklynella hostilis.* The afflicted fish use things to scratch their bodies. This parasite harms the skin and causes skin bleeding due to its adherence to the skin and gills (Fig. 4) (Cruz-Lacierda et al. 2004).

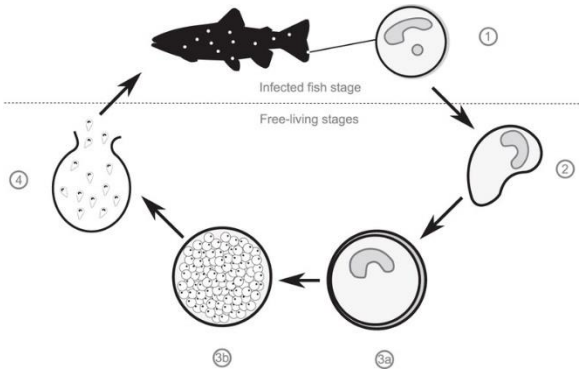

Fig. 1: Life cycle of the endoparasite *Ichthyophthirius multifiliis*

Fig. 2: White spot disease due to the protozoan *Ichthyophthirius multifiliis*

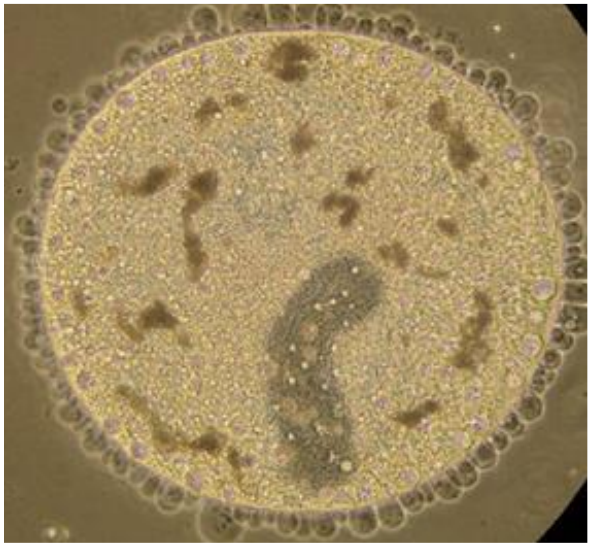

Fig. 3: *Ichthyophthirius multifiliis* (a wet-mount observation)

Diagnosis

The examination of the wet mount prepared from skin and gills enables the parasite identification. On wet-mounting, *Chilodonella* demonstrates a gliding or circling motion (Longshaw and Feist 2001; Weber and Govett 2009).

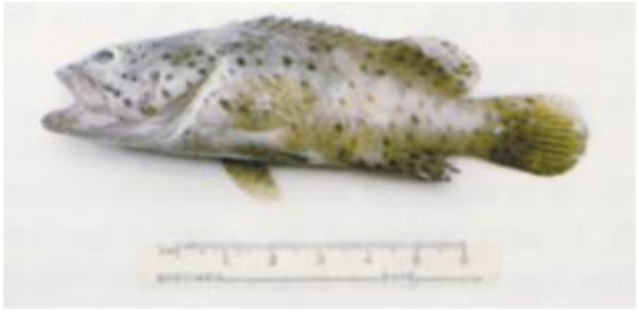

Fig. 4: Brooklynelliosis in *Epinephelus tauvina* showing excessive disruption and bleeding skin

Fig. 5: Chilodonelladiasis

Tetrahymena and *Uronema*

Tetrahymena which is parasitic species of freshwater and *Uronema* which is the marine species, are ciliated parasites that are the causative agents of visible gill and skin lacerations and systematic infections internally (Stoskopf et al. 1993; Noga 1996; Longshaw and Feist 2001; Weber and Govett 2009).

Clinical Signs

Tiny white spots on the skin, sloughing, skin contusions, malformations in the gills, and atrophy are all indications of infection (Fig. 5). Fish that suffer from systemic illnesses might exhibit nonspecific symptoms including anorexia nervosa and sluggish behaviour. After the onset of the infection, the fish may die instantly (Stoskopf et al. 1993; Noga 1996).

Tetrahymena, sometimes referred to as "guppy killer" or "guppy sickness," is a pathogen that primarily affects cichlids, guppies, and other livebearers. It has also been reported that this parasite lives in aquatic organic waste (Stoskopf et al. 1993; Noga 1996).

The *Tetrahymena's* clinical signs are similar to *Uronema* infection. *Tetrahymena* infection can also cause muscular edema and periocular lacerations. Due to the intimate relationship between the skin and the cornea, keratitis may also be caused by these protozoa and other parasites (*Cryptocaryon*, *Ichthyophthirius*, *Henneguya*, and *Glugea*) (Williams and Whitaker 1997).

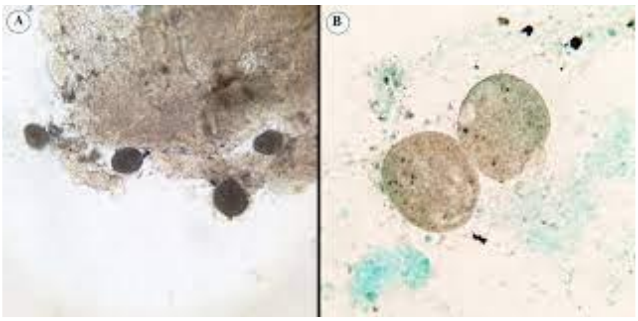

Fig. 6: *Cryptocaryon irritans*

Fig. 7: White spots on body surface of fish infected with *Cryptocaryon irritans*

Diagnosis

Wet-mount examination or immunohistochemistry of the skin and gill tissue are used to find parasites. In the event of deep or systemic infestations, immunohistochemistry of the affected organ or tissue will be required (Palmeiro et al. 2009).

Cryptocaryonosis

As sick fish exhibit few to many whitish or grey dots on their outer surface and gills, cryptocaryonosis is sometimes known as "white spot sickness." Cryptocaryonosis is brought on by an attack of *Cryptocaryon irritans* (Fig. 6) (Nagasawa and Cruz-Lacierda 2004).

Clinical Signs

Whitish or gray marks appear on the body and gills (Fig. 7). Anorexia nervosa, lethargy with abnormal swimming pattern, dark body, bleeding, and protruded eyes are the indications of infection. Excess mucus is formed and fish scratches its body with objects (Nagasawa and Cruz-Lacierda 2004).

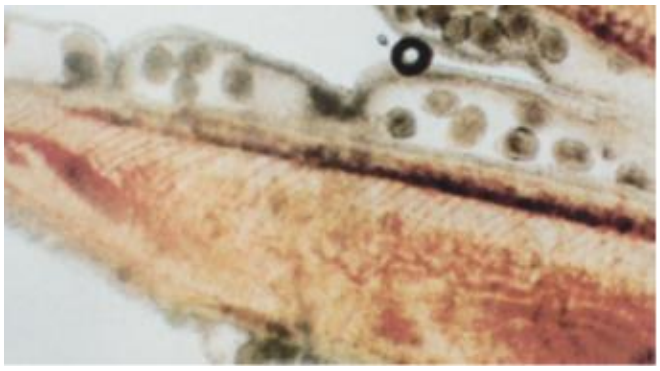

Fig. 8: *Cryptocaryon irritans* on gills of *Cromileptes altivelis*. Fresh mount

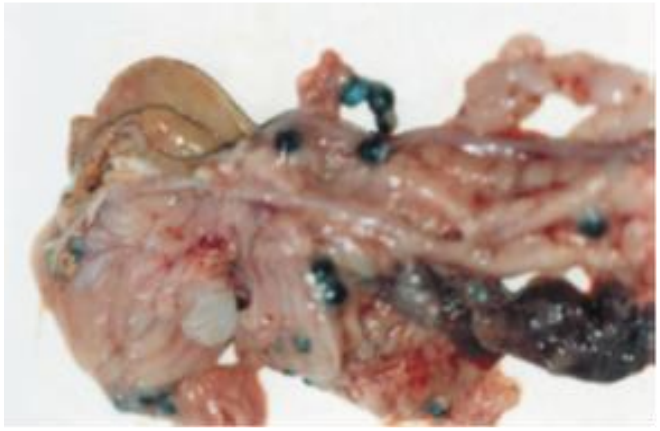

Fig. 9: Brownish-black cysts (arrows) on parenchyma of digestive organs of *Epinephelus tauvina*

Diagnosis

Under a microscope, spherical parasites moving inside the host and mucus on the surface of the body could be seen (Fig. 8) (Nagasawa and Cruz-Lacierda 2004).

Prevention Techniques

Fish should be treated with 0.5 ppm copper sulphate (CuSO4) for 5-7 days with vigorous aeration while being maintained in freshwater. Every day, freshwater that's being used for treatment needs to be replaced (Nagasawa and Cruz-Lacierda 2004).

Microsporidiosis

Microsporidiosis is brought on by a microsporidian infection of fish. Microsporidia are protozoa and endoparasites that have been detected in China and India, including *Epinephelus tauvina* and *Epinephelus* species. Spores in the form of pear are housed in minute nodes that sprout on the sick tissue (Nagasawa and Cruz-Lacierda 2004).

Clinical Symptoms

Fish with illness have enlarged bellies. Various-sized brown to black nodules might be detected in internal organs and adipose tissue (Fig. 9) (Cruz-Lacierda et al. 2004).

2.1.2- Sedentary or Sessile Ciliates

Koi, catfish, and goldfish are among the fish raised in ponds that commonly reveal sedentary or sessile ciliates in water that is rich in organic trash and dissolved solids (Stoskopf et al. 1993, Noga 1996). In addition of being primary invaders on skin ulcers, several parasites can cause epithelial damage in some species of pet fish. *Epistylis* (previously known as *Heteropolaria*), *Capriniana piscium* (previously called *Trichophyra*), *Apiosoma* (previously known as *Glossatella*), and *Ambiphyra* (called *Scyphidia* in past) are among the species that are often sighted (Noga 1996).
Epistylis leads to white, fluffy bruises on the borders of the fins and tail opercula, mouth and throat. Due to their similar indications, these bruises may be mistaken for fungus or columnaris sickness. *Capriniana* prefers gill tissue in particular and causes severe respiratory impairment in sick fish through mechanical obstruction (Noga 1996; Longshaw and Feist 2001; Reavill and Roberts 2007).

Diagnosis

The methods adopted for detection of sessile ciliate infestations are wet-mount cytometry (Fig. 10) and immunohistochemistry of infested tissues (Noga 1996).

Trichodina and *Trichodinella*

Trichodina and *Trichodinella* species are two prominent ciliated parasites that may be encountered on aquarium fish kept in both freshwater and saltwater. Although some of these parasites will parasitize the urinary bladder or oviduct, most of these parasite strains have a unique propensity for illness in skin and gill epithelium. Malnutrition, overpopulation, excessive organic litter in the water, and recent poor state of water are the factors that are frequently linked to these parasites. The parasites are usually seen among pool fish like goldfish and koi (Stoskopf et al. 1993; Baker et al. 2007; Weber and Govett 2009).

Life Cycle

Like in many other protozoan parasites, the life cycle is direct, and reproduction takes place by binary fission. Fomites and live plants added to ponds and tanks result in the introduction of *Trichodinids* into water (Noga 1996; Baker et al. 2007; Reavill and Roberts 2007; Weber and Govett 2009).

Fig. 10: *Apiosoma spp.* (Wet-mount examination).

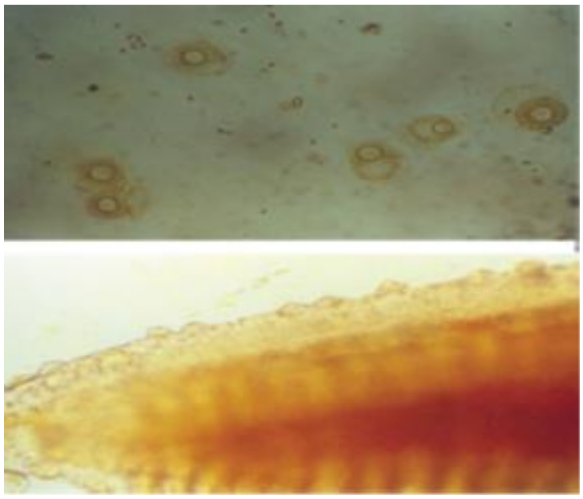

Fig. 11: *Trichodina* sp. from *Epinephelus coioides*: a) On body surface b) On gill filaments.

Clinical Signs

Flashing, murky skin because of increased secretion of mucus, dermatological haematuria, frayed fins and tail, sluggishness, and persistent fatality rates are all common manifestations of extreme branchial infections. The parasite has been portrayed as a "scrubbing bubble" or flying saucer (Fig. 11) (Noga 1996; Baker et al. 2007; Reavill and Roberts 2007; Weber and Govett 2009).

Flagellated Protozoans

Amyloodinium ocellatum and *Piscioodinium*

In marine and freshwater tropical fish, parasitic dinoflagellates (*Amyloodinium (A.) ocellatum* and *Piscioodinium*) can be encountered. These two parasites resemble *Ichthyophthirius* in terms of their life cycles, outward characteristics, and reactivity to temperature. Only the dinospore that is free-living is impacted by therapy. Elasmobranchs and teleosts can also get sick from *A. ocellatum* (Noga 1996). It has been seen to be transmitted up to three meters in active airflow systems, like *Ichthyophthirius*, through aerosol scattering of water drops. (Roberts-Thomson et al. 2006).

Clinical Signs

The epidermis and gills are the more likely or inclined sites for invasion, and a large infection can result in edoema, enlargement, infection-related redness, bleeding, issues with osmoregulatory function, and necrosis in the gill filaments. It is also referred as Amyloodioniosis, which is caused by *A. ocellatum*. Other pathological changes, in combination with respiratory disruption, can be seen as a darkish, gold look on the skin. That's why disease is also called so the named as "velvet sickness," "gold dust illness," and "rust disease" (Fig. 12) (Reavill and Roberts 2007).

Diagnosis

The process of identifying the disease from its symptoms is done by wet-mount cytometry or immunohistochemistry of the skin and gills (Fig. 13) (Baker et al. 2007).

Ichthyobodo

It was previously referred as *Costia* and is a microscopic, flagellated parasite of freshwater fish that is found worldwide in a diverse range of species. It is not larger than the red blood cell. The parasite may survive in a wide temperature range of 2–30°C (Reavill and Roberts 2007).

Clinical Signs

Acute lung trouble, lethargy, sadness, flashes, anorexia, epithelial inflammation and excessive mucus secretion are few of the clinical symptoms leading to fatalities. Death may occur prior to any clinical symptoms (Reavill and Roberts 2007).

Diagnosis

The diagnosis is made based on wet-mount cytometry. The organism's motion has been compared to that of a candle that is "twitching" or to uncontrolled spirals (Palmeiro et al. 2009).

MYXOZOA

Myxosporea (myxosporidiosis)

There are several families and subspecies in the class Myxosporea belonging to the phylum Myxozoa and the majority of which are fish parasites. Some types are well-known freshwater fish infections. Myxosporea infections in

Fig. 12: Adult Siamese fighting fish (*Betta splendens*) with velvet disease

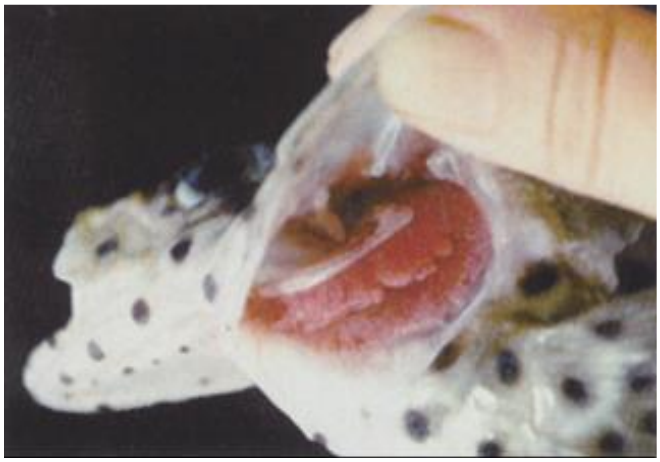

Fig. 13: *Cromileptes altivelis* having yellow gills due to *Amyloodinium ocellatum*

farm marine fish have been encountered more often in recent years. One or more disease spreading sporoplasms, one or more closures, and one or more bipolar capsules seem to have an internal polar filament helix. Whirling disease, PKD, sphaerosporosis, and ceratomyxosis are the four deadliest illnesses that affect freshwater fish. Whirling disease is caused by *Myxobolus cerebralis* (Alvarez-Pellitero and Sitjá-Bobodilla 1993).

Life Cycle

It was proven 18 years ago that the myxosporean's life cycle involves an intermediate oligochaete host. This information has made it easier to take care of the environment, such as using ceramic or plastic pools or tanks and regularly sanitizing them to stop the growth of oligochaetes and the subsequent spread of illness. Consideration of the finite effectiveness of current therapies like fumagillin and toltrazuril for myxosporea and other species is very crucial (Alvarez-Pellitero 2004).

Clinical Signs

Pathological changes include spine bending, darkening of the hind portion of the body and irregular swirl swimming. The vulnerability of illness is variable depending on the species, but all salmonid species may be diseased (Fig. 14) (Alvarez-Pellitero 2004).

Diagnosis

The histological examination of the skull cartilage or their enzymatic digestion proceeded by a microscopical study of the characteristic spores serves as the cornerstone for the diagnosis. Additionally, a PCR test can also be performed (Alvarez-Pellitero 2004).

Proliferative kidney disease (PKD)

Tetracapsuloides bryosalmonae, originally referred as PKX, has recently been recognized as the causal culprit. Although this myxosporean generates spores in a bryozoan host, but phases of this parasite that are without spores are found in the kidneys of several salmonid fish. A death rate of 30-50% occurs because this highly disease-spreading parasite can cause harsh sickness in rainbow trout (Canning et al. 1999).

Clinical Indications

Visible clinical symptoms are belly enlargement, hyperpigmentation and bulging eyes (Fig. 15). Internal indications comprise the fact that one can see enlarged kidneys and in more severe instances, cirrhosis. Immunohistochemistry of the kidney reveals interstitial proliferation together with tubular degeneration and persistent systemic inflammatory interstitial nephropathy (Fig. 16). This parasite also has the side effects of poor dietary metabolism and depressed immune system (Canning et al. 1999).

Diagnosis

The macroscopical identification is based on the complete observation of increased size of kidney. Confirmation is attained by seeing the parasitic stages in histological sections or squash preparations by skilled examiners (Canning et al. 1999).

Spaherospora renicola

Massive populations of *Spaherospora (renicola* are seen in intense cultivation of cyprinids, primarily *Cyprinus carpio*. While spores and their sporognic states are found in the renal tubules, prolific phases can travel via the circulation of blood and inflame the swim bladder (Sitja-Bobadilla and Alvarez-Pellitero 1992).

Fig. 14: Whirling disease

Fig. 15: Polycystic kidney disease

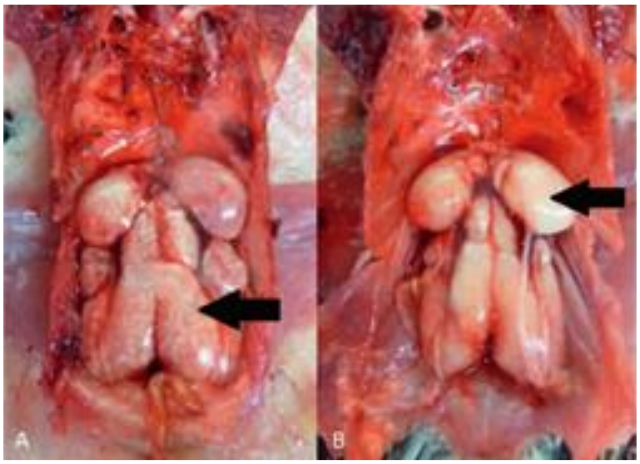

Fig. 16: Polycystic kidney disease in Rainbow lorikeets

Clinical Signs

While spores and their spore forming stages are found in the renal tubules, prolific phases can travel via the circulation of blood and inflame the swim bladder. The parasite *S. renicola* might be very dangerous. It causes ballooning, degeneration, and epithelial deterioration in the renal tubules, which compromises functional status of kidney (Fig. 17). Junior carps have swim bladder soreness as their swim bladder phases mature. Fish can also exhibit certain pathological symptoms, such as abnormal movements and swimming in ring patterns (Alvarez-Pellitero 2004).

Ceratomyxa shasta

On the west coast of North America, *Ceratomyxa shasta* is a significant disease-causing agent that has led to significant losses in salmonid communities, both in the wild and in captivity (Alvarez-Pellitero 2004).

Life Cycle

This Myxosporean's life cycle has been shown to involve an intermediate host that is a polychaete. For diagnosis, a PCR test can be performed (Alvarez-Pellitero 2004).

Clinical Signs

The main organ infected is the intestine, where parasites can be detected in the epithelium, causing tissue damage, hypertrophy, and lymphatic invasion. Severe complications of the illness result in the transmission of the parasites to certain other organs, anorexia, sluggishness, abdominal enlargement, ascites, and bulging eyes in the fish. According to fish species, there may be considerable fatality rates since vulnerability varies (Alvarez-Pellitero 2004).

Enteromyxum spp.

Two species of this genus infect the digestive tract of ill fish and are of pathological concern for marine fish with significant economic value. The myxosporean originally referred as *Myxidium leei* but because of phenotypic and genetic research, it was renamed as *Enteromyxum leei*. It produces the most important myxosporidiosis of cultivated sparids in the Mediterranean Sea, that is now called enteromyxiosis. The vulnerability extent of the fish is significantly broad: seabass, mullets, *Sciaenops ocellatus* and several marine aquarium fish, related to 25 species are infected (Branson et al. 1999).

Clinical Signs

Malnourishment and fatality are the two main outcomes of this parasite's assault on the gastrointestinal system, which causes severe enteritis with permanent repercussions. Therefore, the most extreme degree of slenderness, referred as "knife-fish," fundamentally constitutes the clinical manifestations. Some stock losses, particularly in *Diplodus puntazzo*might reach as high as 80% (Alvarez-Pellitero 2004).

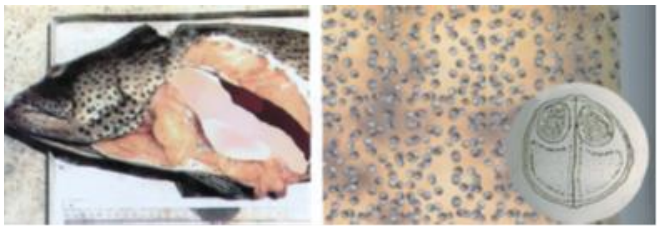

Fig. 17: Myxosporeans in kidney of *Epinephelus malabaricus*

Diagnosis

Immunohistochemical analysis of the intestine and recognition of parasitic phases are done for diagnosis of the parasite. So, the parasitic stages may also be observed in newly made smears by skilled examiner (Alvarez-Pellitero 2004).

E. scophtahlmi

Enteromyxum (E.) scophthalmi is the parasite belongs to the genus *Enteromyxum* and is mainly detected in turbot *Scophthalmus maximus*. *E. scophtahlmi* is a significant parasite for turbot farms because it can cause 100% tank or population mortality, which has a negative influence on the economy **(**Redondo et al. 2002).

Clinical Signs

Anorexia nervosa, caquexia, droopy eyelids and a distinctive pronounced bony hump on the head are the exterior pathological symptoms of disease. At site of tissue damage, further findings include the accumulation of fluids in the colon, intestinal bleeding, and internal organ pallor **(**Redondo et al. 2002).

Diagnosis

The recognition of the parasite is specifically done by microscopic examination of fresh smears and histopathology. The use of PCR technique is limited (Alvarez-Pellitero 2004).

CESTODA

Biology and Taxonomy

Tapeworms are the endoparasites which are found globally. The body of mature cestodes is flat made up of sticky scolex at the apex, the part capable of growing called neck, and the strobilus having different number of androgynous proglottids (Barber and Huntingford 1995; Barber et al. 1995).

Life Cycle

The life cycle of cestodes always need a certain host and one or more intermediate hosts. Fish may be an intermediate host for variable larval stages of parasites or as a main host. When fish is secondary intermediate host the larva of various tapeworms may have various tissue tropisms, but when fish is the main host, the cestodes that attain the mature state produced eggs in the gut of fish (Barber and Huntingford 1995; Barber et al. 2008) (Fig. 18).

Clinical Signs

Clinical indications extend from no symptoms to sluggishness, persistent loss of appetite, decreased weight, long term intestinal swelling, intestinal blockage, and harsh damage of mucosa. The traditional zoonotic infections caused by fish tapeworms are diplogonoporiasis and diphyllobothriasis, also called as 'tapeworm pernicious anemia'. Diphyllobothriasis is a situation which involves megaloblastic, macrocytic anemia along with thrombocytopenia and leukopenia due to lack of vitamin B12. This shortage is a consequence of more need of vitamin B12 in the ATP formation reactions in *Diphyllobothrium latum* and *D. dendriticum*. It is also demonstrated in the larval tapeworms having the capacity to produce anaphylactic reactions in animals that feed on contaminated fish meat. Similarly, the hypersensitivity reactions in humans have also been suggested (Paladini et al. 2017).

Diagnosis

Cestodes may be separated from the fish, cleaned and washed in water and then fixation in formalin or70–99% ethanol is done. At this stage, the cestodes can be kept preserved for a long time. To examine the main properties of internal structure of proglottids and for attaining a better perception of any disease caused by the parasite, histology of the mature tapeworms is beneficial. Identification can also be done by visual examination and wet mount preparation from feces (Paladini et al. 2017).

Treatment

To treat the infection praziquantel is given orally at 50 mg/kg for one dose, or 5 to 12 gm/kg of feed every 24h for 2 to 3 days. Treatment should be provided in an isolated tank so that the eggs of died cestodes may not scatter in the tank (Paladini et al. 2017).

ACANTHOCEPHALA

Biology

Acanthocephalans are distinguished by an invertible proboscis that is differently equipped with a sequence of hooks, the number and arrangement of which have phylogenetical significance. They are also described as "thorny headed" or "spiny headed" worms. A junctional skin

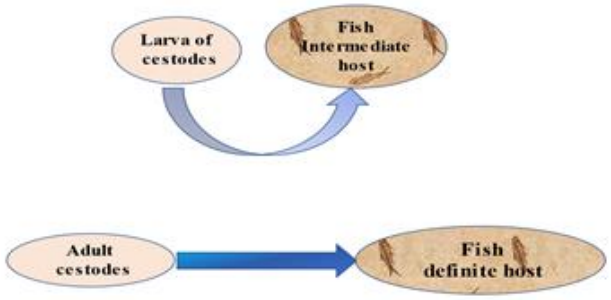

Fig. 18: Life cycle of cestodes

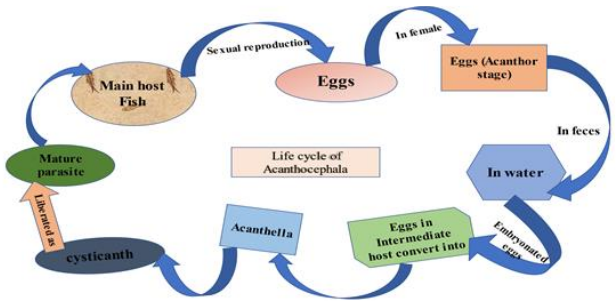

Fig. 19: Life cycle of *Acanthocephala*

serves as the body's boundary and because acanthocephalans are without a digestive membrane, they obtain their nutrition through the cuticular surface. They are hermaphroditic; males have different numbers of testicles, a copulatory bursa and "cement glands" that are responsible for closing the female's uterus after fertilization, in accordance with the species (Kennedy 2006).

Life Cycle

The mature parasite specifically dwells the gut of the main host, in which they undergo sexual reproduction. In the female the impregnated eggs grow to the acanthor stage and then eggs are secreted in the feces of the host in water. When taken in by intermediate host, the embryonated eggs emerge into an acanthella that afterwards enclose in a cyst in the tissue of host till a larval cystacanth is liberated (Fig. 19). Fish may behave like a paratenic host, for species like *Acanthocephalus anguillae*. Fish can also act as main host for some species like *Pomphorhynchus laevis* in trout, *A. anguillae* in European eels, *Anguilla anguilla*, and *A. lucii* in northern pike, but other piscine species may become a postcyclic host. For instance, parasites are capable to live in the predatory host if they ingest a host infected with mature parasite (Kennedy 1999).

Significant Pathogens within the Group

Considerable pathogens in this group include *Acanthocephalus* spp., *Bolbosoma* spp., *Echinorhynchus* spp. and *Pomphorhynchus* spp. (Paladini et al. 2017).

Clinical Signs

The clinical effect of the parasites does not depend on the number of parasites in the fish instead on the number of parasites to body size of the eel. The surface area of gills enhances significantly with the increasing body length (Hughes 1966) and the area of adherence also enhances with the size of host (Buchmann et al. 1989b). So, there will be serious effect of only few parasites in glass eels and young fish that will result in no troublesome in large sized eels. More infestations cause the eels to become sluggish and anorectic. The primary symptom is reduced feeding process and the obvious mark of gill-disease is that the fish look for the surface of water because diseased gills get in less oxygen. When reach the utmost point eel rotate its upper side down and finally die (Woo and Buchmann 2012).

The fish farms that provide uninterrupted flow of water in the tanks and biofilters, the diseased eels in them are incapable to be at their upper position in tanks and flow with water streams. This cause capturing of affected eels at the outlet (Buchmann et al. 1988b). More mucus is produced due to the hyperplasia of mucous cells which causes bashing or cudgeling of basic gill filaments and attachment of gill lamellae with each other and with neighboring filaments. Bleeding can also appear due to feeding of parasite and injection of hooks telangiectasis are found in highly affected eels (Woo and Buchmann 2012).

Diagnosis

The gathered parasites are particularly freed from the tissues of the host. These parasites are adhered to the tissue of host by piercing needles. After removing from the tissue of host, these are preserved in 70–95% ethanol for morphological and molecular-based studies. Brown et al. (1986) gave an explained method of collection, fixation, preservation, and examination of acanthocephalan. Alcohol-fixed specimens are cleansed with glycerol or stained with Mayer's acid carmine, for studying internal anatomy of these helminths. SEM may help in mapping and analyzing the framework of the proboscis and spines on body, but histology works for exploring host–parasite relationships and the pathogenicity (Paladini et al. 2017, Austin and Newaj-Fyzul et al. 2017).

TREMATODA (DIGENEA)

Morphology

These endoparasites belongs to the phylum platyhelminths and have complicated life cycles. All of them are androgynous besides some types living in blood (*Schistosomatidae*) and some tissue attackers found in marine fishes (*Didymozoidae*). They are known as "flatworms", but all species are not dorso-ventrally flattened (Thatcher 2006).

Life Cycle

The mature trematodes live in the digestive tract, blood circulation or hypodermic connective tissue of vertebrates. The whole process of mating and egg production take place in the hosts. The eggs are taken to the outside with host's feces or urine which burst in water after a short time. The primary larval phase of trematodes is miracidium that is ciliated and floats looking for a proper species of snail. After attaining snail, the miracidium enters into the body wall with the aid of its frontal penetration glands and reach the hepatopancreas. There it turns into a sac-like sporocyst after removing its cilia. The third stage of larva called rediae is formed in sporocyst, that ruptures in the snail's digestive gland. Cercariae are then produced from rediae that liberate from birth pores and leave the snail for finding an intermediate host or make encyst on vegetation. The tail of cercariae is removed during encystation and the resultant body is now a metacercaria. Sometimes, the main host is inhabited by cercariae directly. Species infecting the blood (*Sanguinicolidae*) and tissue forms (*Didymozoidae*) follow this direct way, while other fish trematodes get into the host in the form of metacercariae (Fig. 20) (Thatcher 2006).

Diseases

1. **"Black-spot disease"** is the disorder produced when cercariae attack the skin and form encystation there. This encystation is viewable to naked eye when host fish accumulates pigment cells around. The metacercariae in the skin does not destroy the health of the fish. Sometimes these black spots are too much that they turn the fish unlikeable to the consumer (Thatcher 2006).
2. **"Yellow-spot disease"** is the resembling situation. The metarcercariae of the family *Clinostomidae* cause this disease because of their yellow color (Thatcher 2006).
3. **"Eye fluke disease"** is the disorder due to the larval trematodes across the world. The larvae are observed moving around and in the eye of infected fish. No usual controversial reaction occurs, but the worms interrupt the sight of fish. Fish can become blind and is preyed by piscivorous birds (Ashton et al. 1969). Thatcher (2006) discovered that in Amazonian fish (*Chaetobranchus semifasciatus*), larval trematodes can cause branchial carcinoma.

Prevention and Treatment

Snails and plants in the environment of fish must be removed for a good and healthy aquarium.
There is no feasible treatment for encysted metacercaria. The mature trematodes may be vanished from the intestinal tracts of fish by using Di-N-Butyl Tin Oxide that is combined with the ratio of 0.3 % with respect to the body mass and weight and is given for one to five days. (Thatcher 2006).

Monogenetic Trematodes

The parasitic flatworms or flukes called monogenetic trematodes often reside on the skin of their fish hosts. The loose end browsing behavior of mouth and puncture of their adhesion organ both harm the host. They are known to be a significant fish disease in aquaculture. *Gyrodactylus* "skin flukes" and *Dactylogyrus* "Gill fluke" are considered as the most prevalent members of this group (Ernst et al. 2002; Ogawa 2002; Grau et al. 2003).

NEMATODES

Nematodes or roundworms are endoparasites having unsegmented bodies. The mature nematodes are visible to the naked eye. Nematodes affect various species of fish including *Epinephelu coioides, E. malabaricus, Cromileptes altivelis* and *Plectropomus leopardus* mainly prevalent in Indonesia, Malaysia, and Thailand. Common disease-causing agents of nematods are *Philometra* sp., *Anisakis* sp. and *Raphidascaris* sp. They can infect the growing or fingerling stages (Nagasawa and Cruz-Lacierda 2004).

Clinical symptoms

Dark red or black roundworms (without segments) are adherent to the parenchyma tissue of the digestive organs, muscles, fins, branchial chamber, and gonads of the ill fish (Fig. 21). Highly infected fish has faded and lean body (Nagasawa and Cruz-Lacierda 2004).
Parasite disturbs feeding which causes less growth and body becomes lean. Muscular destruction of infected gonads causes sterility (Nagasawa and Cruz-Lacierda 2004).

Transmission

Fish is considered as the main host for nematode parasites. Mature nematode produces egg, which burst into free-swimming larva, that is ingested by an invertebrate intermediate host (Nagasawa and Cruz-Lacierda 2004).

Diagnosis

The parasites are examined by microscopic examinations. The parasites are seen by operating the affected tissues. A mature *Philometra* sp. may grow up to 20 cm in length (Nagasawa and Cruz-Lacierda 2004).

COPEPODS

The copepods are ectoparasites of skin having sectioned shelly bodies (clearly divided bodies protected by shells) with segmented appendages. Copepods affect various species of fish including *coioides, E. fuscoguttatus, E. malabaricus,*

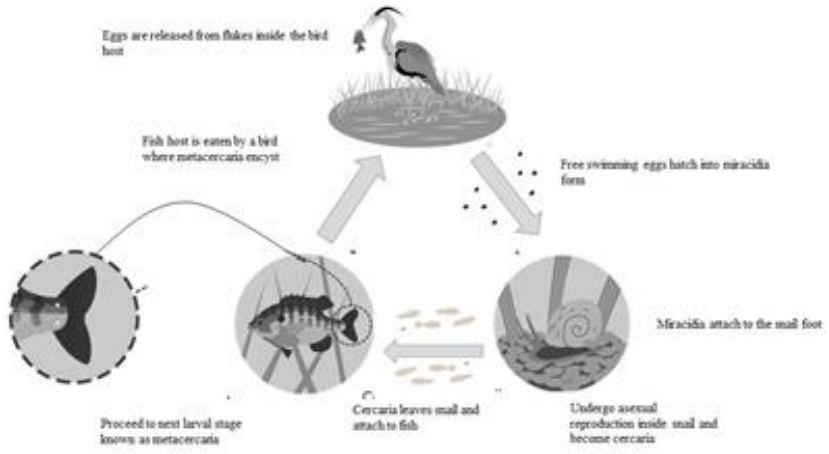

Fig. 20: Life cycle of the parasitic fluke *Clinostomum marginatum*, the yellow grub

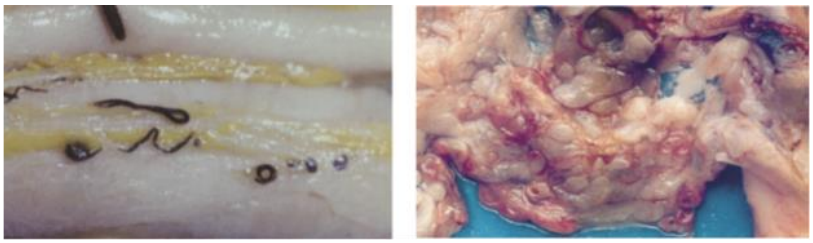

Fig. 21: Nematodes on tissue of digestive organ: a) *Plectropomus leopardus* b) *Epinephelus coioides*

Cromileptes altivelis and *Plectropomus leopardus*. Common pathogens of the group are *Caligus epidemicus*, *Caligus* sp. and *Lepeophtheirus* sp. They infect the small fingerling stages (Nagasawa and Cruz-Lacierda 2004).

Clinical Signs

These parasites are translucent and don't remain adhered to the body and fins of fish. They can be seen as white patches (Fig. 22). The affected places lack scales and have bleeding ulcers. Clumsy body, sluggishness, fish come to the surface to get oxygen, anorexia nervosa and excess mucus formation are the clinical indications. Highly infested fish may become lean (Nagasawa and Cruz-Lacierda 2004).

Prevention

Water should be changed to impede diseases. Freshwater washing of 10-15 minutes, or chemical washing by 150 ppm hydrogen peroxide for 30 mins should be established. Vigorous aeration should be given to the under-treatment fish (Nagasawa and Cruz-Lacierda 2004).

ISOPODS

Isopods have 10-50 mm sized body with short sections and two eyes. The parasite is seen in *Epinephelus (coioides* and *E. malabaricus. Rhexanella* sp. is seen in *E. coioides*. Isopods infect the fingerling stages of fish (Nagasawa and Cruz-Lacierda 2004).

Clinical Signs

The parasite adheres the body surface, mouth, nasal and opercular area (Fig. 23). Fish shows less opercular motion, becomes anorexic, body becomes thin, less growth, and it scratches its body with aquatic objects. The stress of parasites' body weight damage the fish tissue, skin layer and filaments of gill destroy. Small fish having high infestation die in 1-2 days (Nagasawa and Cruz-Lacierda 2004).

Prevention

Physically, parasite can be taken off and smashed. Washing by 200 ppm formalin for 30-60 mins is recommended. The aeration should be done and under treatment fish should remain in clean water (Nagasawa and Cruz-Lacierda 2004).

LEECHES

Leeches are ectoparasites having striped bodies, and pair of suckers that help in feeding and motion. The parasite causes sickness in *Epinephelus bleekeri*, *E. coioides*, *E. fuscoguttatus*, *E. lanceolatus*, *E. malabaricus* and *Cromileptes altivelis* etc. *Zeylanicobdella arugamensis* causes infection in *E. coioides*. Small growing fishes are heavily infested (Nagasawa and Cruz-Lacierda 2004).

Clinical Signs

The black and brownish parasites adhere in small blotches on infected locations like the body, fins, eyes, brachial and mouth spaces (Fig. 24). Diseased fish have ragged fins,

Fig. 22: Caligid copepods like white blotches on *Cromileptes altivelis*

Fig. 23: Isopod adhered on *Epinephelus coioides*

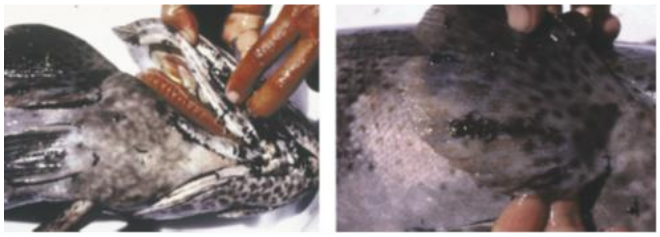

Fig. 24: *Zeylanicobdella arugamensis* on opercular space and pectoral fin of *Epinephelus coioides* broodstock

Fig. 25: Physical removal of leeches attached to *Epinephelus coioides* using a wet cloth

bleeding and irritation on adhering and feeding places of parasites, anorexia, anemia, sluggish and slow motion and fish come to surface for aeration. Highly infested fish exhibit high fatality rate (Nagasawa and Cruz-Lacierda 2004).

Life Cycle

Pre-disposing factors are poor maintenance of facilities and poor water state. Transmission is from one fish to other. Adult leeches release from fish and put their cocoons on rocks, shells or vegetation. A cocoon has one egg that burst into a young piscicolid leech, which then adheres to a host to become adult. After putting cocoons, adult leeches die (Nagasawa and Cruz-Lacierda 2004).

Prevention

From the water used for cultivation of fish, leeches can be eliminated by filtration. Physically, moist piece of cloth is used to clean blotches of the parasite (Fig. 25). Washing with formalin for an hour and powerful aeration will remove parasite. The post treatment fish are transmitted to clean water. Accessories used in cultivation must be cleaned with chlorine and placed in sunlight (Cruz-Lacierda et al. 2004).

1- Amoebae

Some naturally occurring amoebae have the potential to alter their behaviour and cause harm. Salmonid gill illness has been linked to several forms of amoeba (Nagasawa and Cruz-Lacierda 2004).

Amoebic Gill Disease (AGD)

AGD is a condition brought on by the commensal, free-living amoeba. A significant issue in marine salmon farming is *Paramoeba perurans*, which causes gill deterioration and death in infected fish. It has been viewed as the deadliest contagious disease and has become a critical challenge for sea-caged Atlantic salmon and rainbow trout in Tasmania (Roubal et al. 1989; Munday et al. 1990; Bryant et al. 1995; Findlay et al. 1995). There have also been reports of gill amoebic illnesses in fish apart from salmonids, such as European catfish (Dykova et al. 1998; Paniagua et al. 1998). AGD most frequently manifests at water temperatures between 10 and 20°C, while it can occasionally happen in temperatures above average. Raised, multifocal, white mucoid patches on the gills of sick fish are signs of severe disease and are sites of primary and secondary laminae epithelial proliferation. Desquamation of the epithelium, localized problems with blood flow, and increasing alterations symptomized by irritation before this step (Dykova et al. 1995; Adams and Nowak 2003). The gill respiratory surface area is reduced or destroyed because of all

the aforementioned alterations. Fish with AGD would experience severe cardiovascular abnormalities and acid-base imbalances that would lead to abrupt cardiac dysfunction and death (Powell et al. 2002).

Conclusion

There are numerous parasitic diseases around the world. Parasitic diseases are common in fish, and they can cost a lot of money to the fish farmer. As some pathogens are zoonotic in nature, so aqua farmers, fish technicians and processors must practice good hygiene. Many diseases can be avoided with proper management and vaccinations.

REFERENCES

Adams MB and Nowak BF, 2003. Amoebic gill disease (AGD): Sequential pathology in cultured Atlantic salmon (*Salmo salar* L.). Journal of Fish Diseases 26: 601-614.

Alvarez-Pellitero P and Sitjá-Bobodilla A, 1993. Pathology of Myxosporea in marine fish culture. Diseases of Aquatic Organisms 17: 229

Alvarez-Pellitero P, 2004. Report about fish parasitic diseases. Etudes et Recherches, Options Mediterranennes. CIHEAM/FAO, Zaragoza 2004: 103-130.

Ashton N et al., 1969. Trematode cataract in freshwater fish. Journal of Small Animal Practice 10: 471-478.

Austin B and Newaj-Fyzul A, 2017. Diagnosis and control of diseases of fish and shellfish, John Wiley and Sons, New York, USA.

Baker DG et al., 2007. Parasites of fishes. In: Baker DG, editor. Flynn's parasites of laboratory animals (2nd Ed.) Hoboken (NJ): Blackwell; pp: 69-116.

Barber I and Huntingford FA, 1995. The effect of *Schistocephalus solidus* (Cestoda: Pseudophyllidea) on the foraging and shoaling behaviour of three-spined sticklebacks, *Gasterosteus aculeatus*. Behaviour 132: 1223–1240.

Barber I et al., 1995. The effect of hunger and cestode parasitism on the shoaling decisions of small freshwater fish. Journal of Fish Biology 47: 524–536.

Barber I et al., 2008. Growth and energetics in the stickleback *Schistocephalus* host–parasite system: a review of experimental infection studies. Behaviour 145: 647-668.

Bishop TM et al., 2003. Aerobiological (airborne) dissemination of the fish pathogen *Ichthyophthirius multifiliis* and the implications in fish health management. In: Cheng Sheng L, Patricia OB, editors. Biosecurity in aquaculture production systems: exclusion of pathogens and other undesirables. Baton Rouge (LA): The World Aquaculture Society; pp: 51-64.

Branson E et al., 1999. Myxosporean infection causing intestinal disease in farmed turbot, Scophthalmus maximus (L.) (Teleostei: Scophthalmidae). Journal of Fish Disease 22: 395-399.

Brown AF et al., 1986. A key to the species of Acanthocephala parasitic in British freshwater fishes. Journal of Fish Biology 28: 327-334.

Bryant MS et al., 1995. Immunogenicity of amoebicantigens in rainbow trout *Onchorhynchus mykiss* (Walbaum). Journal of Fish Diseases 18: 9-19.

Buchmann K, 1988b. Feeding of *Pseudodactylogyrus bini* (Monogenea) from Anguilla anguilla. Bulletin of the European Association for Fish Pathologists 8: 79-81.

Buchmann K, 1989b. Relations between host-size of Anguilla anguilla and the infection level of the monogeneans *Pseudodactylogyrus spp*. Journal of Fish Biology 36: 599-601.

Canning E et al., 1999. Organism, the cause of Pkd in Salmonid Fish. Bulletin of the European Association of Fish Pathologists 19: 203.

Dykova I et al., 1995. Amoebic gill infection of turbot, *Scopthalmus maximus*. Folia Parasitologica 42: 91-96.

Hadfield C et al., 2007. Emergency and critical care of fish. Veterinary and Clinical Exotic Animals 10: 647–675.

Hoffman GL, 1999. Parasites of North American freshwater fishes, 2nd Ed., Ithaca (NY): Cornell University Press.

Hughes GM, 1966. The dimensions of fish gills in relation to their function. Journal of Experimental Biology 45: 179-195.

Kennedy CR, 1999. Post-cyclic transmission in *Pomphorhynchus laevis* (Acanthocephala). Folia Parasitologica 46: 111–116.

Kennedy CR, 2006. Ecology of the Acanthocephala, Cambridge University Press, Cambridge.

Longshaw M and Feist S, 2001. Parasitic diseases. In: Wildgoose WH, editor. BSAVA manual of ornamental fish health (2nd Ed.) Gloucester: BSAVA.

Munday BL et al., 1990. Paramoebic gill infection and associated pathology of Atlantic salmon, *Salmo salar*, and rainbow trout, *Salmo gairdneri*, in Tasmania. In: Perkins FO, Cheng TC, editors. Pathology in Marine Science: Academic Press, San Diego; pp: 215–222.

Nagasawa K and Cruz-Lacierda ER, 2004. Diseases of cultured groupers. Aquaculture Department, Southeast Asian Fisheries Development Center.

Noga EJ, 1996. Problems 10–42. In: Noga EJ, editor. Fish disease: diagnosis and treatment. St Louis (MO): Mosby; pp: 75-138.

Overstreet RM, 2021. Parasitic diseases of fishes and their relationship with toxicants and other environmental factors. In: Couch JA, Fournie JW, Menzer RE, editors. Pathobiology of marine and estuarine organisms: CRC press, Florida, USA; pp: 111-156.

Paladini G et al., 2017. Parasitic diseases in aquaculture: their biology, diagnosis and control. Diagnosis and Control of Diseases of Fish and Shellfish 2017: 37-107.

Paniagua E et al., 1998. Effects of temperature, salinity and incubation time on in vitro survival of an amoeba infecting the gills of turbot, *Scophthalmus maximus* L. Journal of Fish Diseases (United Kingdom).

Reavill D and Roberts H, 2007. Diagnostic cytology of fish. Veterinary Clinics: Exotic Animal Practice 10: 207-234.

Redondo MJ et al., 2002. Experimental transmission of Enteromyxum scophthalmi (Myxozoa), an enteric parasite of turbot Scophthalmus maximus. Journal of Parasitology 88: 482-488.

Roberts HE et al., 2009. Bacterial and parasitic diseases of pet fish. Veterinary clinics of North America: Exotic Animal Practice 12: 609-638.

Roberts-Thomson A et al., 2006. Aerosol dispersal of the fish pathogen, Amyloodinium ocellatum. Aquaculture 257(1-4): 118-123.

Roubal FR et al., 1989 Studies on cultured and gill attached Paramoeba sp. (Gymnamoeba: Paramoebidae) and the cytopathology of paramoebic gill disease in Atlantic salmon,

Salmo salar L., from Tasmania. Journal of Fish Diseases 12: 481-492.

Sitja-Bobadilla A and Alvarez-Pellitero P, 1992. Light and electron microscopic description of *Sphaerospora dicentrarchi* n. sp. (Myxosporea: Sphaerosporidae) from wild and cultured sea bass, *Dicentrarchus labrax* L. The Journal of Protozoology 39: 273-281.

Thatcher VE, 2006. Amazon fish parasites (Vol. 1), Pensoft Publishers, Sofia, Bulgaria.

Weber and Govett P, 2009. Parasitology and necropsy of fish. Compendium (Yardley, PA) 31(2): E12-E12.

Williams CR and Whitaker BR, 1997. The evaluation and treatment of common ocular disorders in teleosts. Journal of Exotic Pet Medicine 6: 160–169.

Woo PT and Buchmann K, 2012. Fish parasites: pathobiology and protection, Centre for Agriculture and Bioscience International (CABI)