British Journal of Pharmacology and Toxicology 2(5): 213-226, 2011
ISSN: 2044-2467

Submitted: July 26, 2011 Accepted: September 25, 2011 Published: November 25, 2011

A Review of Some Basic Parasite Diseases in Culture Fisheries Flagellids, Dinoflagellides and Ichthyophthriasis, Ichtyobodiasis, Coccidiosis Trichodiniasis, Heminthiasis, Hirudinea Infestation, Crustacean Parsite and Ciliates

J.F.N. Abowei, O.F. Briyai and S.E. Bassey
Department of Biological Science, Faculty of Science, Niger Delta University,
Wilberforce Island, Bayelsa State, Nigeria

Abstract: A review some basic parasite diseases in fish: flagellids, dinoflagellides and ichthyophthriasis in African fish was carried out to educate fish culturist and the private sector on some challenges faced in culture fisheries. Some common parasite diseases: Ichtyobodiasis, Coccidiosis, Ichtyopthiariasis, Trichodiniasis, Heminthiasis, Crustacean parasite, Hirudinea infestation, Flagellates and Ciliates, Taxonomy and diagnosis, Life cycle and biology, Epizootiology, pathology, control, Infections with parasite dinoflagellids and Ichthyophthiriasis are some parasite infections in fish discussed.

Key words: African fish, biology, causative agents, epizootiology, life cycle, pathology and control, some parasite infections, taxonomy and diagnosis

INTRODUCTION

Parasite infection in fish refers to a diseased condition in fish resulting from organism living in or on thre fish (Bassey, 2011). The relationship between the fish and parasite is referred to parasitism. In this relationship, one benefits while the other suffers. The host suffers and the parasite benefits. Various types of host exist. The parasite reaches its adulthood in or on the definitive or final host. Secondary (intermediate) host harbors parasite but hinders its development until it passes to a definitive host. A temporary host harbors the parasite briefly. Parasites from temporary host become independent soonest. Reservoir host serves as a source of parasite for other host. Protozoan and Metazoans are examples of fish parasites. Protozoan is a unicellular organism while metazoans are multicelluar organisms (Bassey, 2011).

The ubiquitous ectoprotozoans are cosmopolitan or trans-continentally dispersed via translocation of their cultured fish hosts (carp and tilapia in particular) (Ichthyobodo necator, Cryptobia branchialis, Chilodonella hexasticha,C.piscicola, Trichodina acuta, T. heterodentata and T. pediculus, Trichodinella epizootica, - Hoffman, 1978; Basson *et al*., 1983; Van and Basson, 1987, 1989; Natividad et al., 1986; Shaharom-Harrison and Abdullah, 1988; Abaladejo and Arthur, 1989; Bondad-Reantaso and Arthur, 1989; Basson and Van, 1993).

Distribution of the more specialised host-specific species follows that of their hosts, but may also be more restricted, sometimes to only one or a few watersheds. There is evidence for the presence of Ichthyobodo, Chilodonella, and in particular trichodinids and sessilians in a number of water systems in tropical Africa (Lake Volta and East African lake systems - Fryer, 1961; Paperna, 1968; Paperna and Thurston, 1968; Fryer and Iles, 1972), but taxonomic data are limited to only a few locations (Kazubski and El-Tantawy, 1986; El-Tantawy and Kazubski, 1986). Southern Africa and the Zambezi river system. Data are also available from Israeli fish (Basson *et al*., 1983; Basson and Van, 1987; Van and Basson, 1989, 1992).

Integumental ectoprotozoan genera are readily differentiated (Kabata, 1985), while diagnosis of species is difficult and often requires special staining. Most ectoparasitic forms are readily detected in direct microscopic examination of skin and gill scrapings from live (or freshly killed) fish. Flagellates may be further detected in air dried, methanol fixed, Giemsa stained smears. Smears containing ciliates should be air dried, fixed in Bouin for 20 min., destained in 70% ethanols, brought to water, stained in a haematoxylin stain and mounted after dehydration. Trichodinids for specific differentiation should be impregnated with silver. Air dried smears should be placed in 2% silver nitrate for 7-9 min. in the dark, rinsed in water and exposed to the sun or UV for 5-10 min.

Some common parasite diseases: Ichtyobodiasis, Coccidiosis, Ichtyopthiariasis, Trichodiniasis, Heminthiasis, Crustacean parasite, Hirudinea infestation, Flagellates and Ciliates, Taxonomy and diagnosis, Life cycle and biology, Epizootiology, pathology, control, Infections with parasite dinoflagellids and

Corresponding Author: J.F.N. Abowei, Department of Biological Science, Faculty of Science, Niger Delta University, Wilberforce Island, Bayelsa State, Nigeria

Ichthyophthiriasis x are are reviewed to educate fish farmers and the public sector to challenges faced in culture fisheries.

COCCIDIOSIS

This refers to infestation of the coccid organisms. *Emeria* and *Haplozoon* species. The sporocyst and cocyst stages are the only stages outside the host. Emeria species infest the intestinal mucosa and epithelium, causing white blisters in the intestinal wall and enlarged intestine. Haplozoon species on the other hand is a blood borne parasite infesting the blood cells.

The presence of oocyst in faecal caste and intestinal smear are diagnosis of the disease. Observation of blood smear under the microscope can reveal haplosan oocyst in the erythrocytes and leucocytes. Severity of the condition depends on the load of the infective organisms in the infected organs, and the extents of damage. Cases of active coccidiosis are rare but severe condition can be controlled with the coccidiositamprol and furapol used for higher vertebrate.

Ichtyopthiariasis: Another name for this disease is white spot disease or "ich". The disease is common with cold and warm water fish species. The causative agent, *Ichtyophthinus multifilis* is the largest protozoan parasite found on fish. Matured trophozoites are *oval* to round in shape and 0.5 to 1.0 mm at the longest axis. The trophozoite dislodges from host's body as a trophont uniformly ciliated with macro and micronucleus. The trophont produces 250 -1000 tomites (Infective host) within itself. The tromites then invade the skin and the gills of a fish host forming white spots.

Heavy infestation results in restlessness, emaciation, body irritation and gasping. Morbidity can be up to 100%, while mortality only occurs with extensive damage to the gills and the skin. Presence of white spots on the skin, gills and identification of the protozoan parasite in the scraping of the white spot observed under the microscope confirms the condition.

Most protozoacide disinfectants can destroy tomites and other non intra-dermal stages, but cannot affect the intra dermal stages, because disinfectants cannot penetrate the white spot. However, a temperature of 32ºC can weaken or kill the intra dermal stages. All other control methods such as quarantine, maintenance of hygienic environment and good water quality are also important.

Trichodiniasis: This is trichodina infection. The etiologic agent is trichodina species, a uniformly ciliated saucer shaped protozoan parasite. The organisms infect the skin, gills and cause irritation on the tissues. The disease manifests in the form of restlessness, loss of appetite and gasping (Dyspnoea) in heavy infestation of the gills. The condition can be diagnosed based on the clinical signs and the identification of the protozoan parasites on the skin scrapings and gill arches mounted on glass slides, viewed under the microscope. Most protozoacide disinfectants can eliminate trichodina, but prevention with maintenance of good water quality, avoiding overcrowding and good hygienic practices is best.

Heminthiasis: This is an infection due to parasite worms; Examples of such worms are Trematoda (Monogenea and digenea), Cestodes and Nematodes. The latter requires intermediate hosts; hence the life cycle is complex. Monogenea trematodes include the families, Dactylogyridae and Gyrodactylidae. These worms are found on the skin or gills of lower aquatic vertebrates. Only a few of these worms are viviparous. All others are oviparous. The specially adapted structures haptor and opishaptor aid their attachment to the host. Hooks and suckers are responsible for the damage on the host. These create portals of entry for opportunistic pathogens. This can occur only in heavy infestation. Extensive damage can be done to gills in dactylogyrus infestation. The damage on the gill can cause fish to gasp. Gyrodactytus infestation results in the development of whitish or grayish spots on the skin of infected host.

Observation of trematodal worms on skin scraping on gill tissue under the microscope is good aid in diagnosis. Some species of monogenea trematodes can be observed without magnification, because of their size.

Formalin and potassium permanganate are good disinfectant in controlling the monogena trematodes. Chemotherapy in the control of the infestation without good health management cannot be effective. Fry, fingerlings and adult fish can be treated with formalin at a dose of 25-50 ppm applied to pond. The concentration level of 0.12 ppm Bromex ^{R} or 0.25 ppm, Dipterex^{R} (Dylox ^{R}masotea ^{R}) is, suitable for pond treatment. Pond banks can also be disinfected with quicklime or calcium cyanamide. Catfish is also an intermediate host for Trematodian matecercariae. They are particularly present in high number in the connective tissue around the brain and muscular tissues.

Hirudinea infestation: Blood sucking leeches can be present in fresh water ponds. Leeches are referred to as predacious parasite, capable of surviving at 20ºC. This explains why they constitute problem for warm water fishes. Leeches suck host's blood, reducing their vitality by rendering them anemic. They damage host's skin and are creating portals of entry for opportunistic pathogens. They are known to transmit the protozoan parasite, Trypanozon and Trypanosomes. Leeches are easily recognized when attached to the skin or fins. In heavy infestations the attachment areas are hyperemic and hemorrhagic leeches collected from fish can be anaesthetized by adding menthol crystal to the water in the dish where the leeches are kept to ensure complete

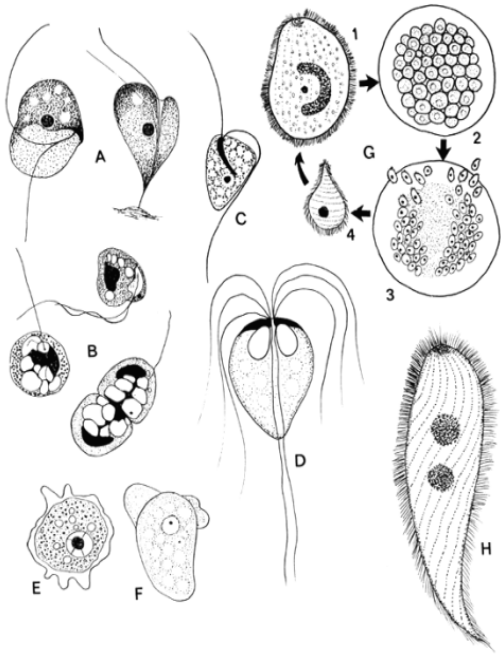

Fig. 1: Ectoparasites and intestinal protozoa

relaxation before fixation in 95% alcohol. Heavy infestation of leeches causes anemia and secondary bacteria infection of the ulcer on the fish skin. The application of 5 ppm cupric chloride ($CuCl_2$) and 25 ppt sodium chloride (NaCl) for 1 h can be effective in the treatment of hirudinea infestation.

Crustacean parasite: These include several species of copepods, branchiurans isopods, amphipods and barnacles. *Lernae* sp. and *Argulus* sp. (fish lice) are copepods and branchiura respectively. Both crustaceans are oviparous, laying eggs, which develop into nauplic, metanauplic and young crustacean. Subsequent molting results to adulthood. The first copepod stage infests the skin and gills and damages the tissues of these organs. Light infestation can cause much harm to fish host.

Hosts rub their bodies against the sides of the holding facilities to dislodge the irritating parasites. The skin, fin or gill secrets excess mucus. Erythematous or hemorrhagic lesions occur on infected parts of fish body. An area of attachment of the parasite becomes swollen.

Benzene hexachloride (Lindans (R) and Masoten (Dylox(R) can be applied indefinitely at 0.12 and 0.25 mg/L for the control of these parasites. the parasites. Identification of the organisms seems not difficult because they can be seen with the naked eye. This can be based on the morphology of the adult female Lernae species and any adult *Argulus* sp.

FLAGELLATES AND CILIATES

Host specific species are associated with a wide range of fish species from most families. Ubiquitous or opportunistic species (Ichthyobodo necator, Chilodonella spp., and some species of Trichodina, Ambyphrya and *Scopulata (Scyphidia)* are particularly common in juvenile cichlids and carp.

Figure I shows Ectoparasites and intestinal Protozoa: A. Ichthyobodo necator free (left) and attached (10-15 µm long axis). *B. Ichthyobodo* sp. from Aplocheilichthy Cryptobia (length 6-8 µm).D.*Hexamita* sp. from tilapia hybrid gut (7-12 µm). E. *Thecamoeba* (40 µm diam.) F.*Entameoba*(15µm diam.).G.Life cycle of *Ichthyophthirius multifiliis*:1. Trophont; 2. Dividing tomont; 3. End of division - tomites (theronts) escape from the cyst residues; 4. Tomite (theront). H.*Protoopalina* (150-350 µm long).

Plate 1 shows Ectoparasite Protozoa: a. *Ichthynecator* on gill arch of *Oreochromis aureus*, Israel. b. *l. necator* mouth lining of wild goldfish, Israel. c-e. Cryptinfections, Israc, on farmed goldfish;d.on *Hypophthalmichthys molitrix* (silver carp) gill rackers. f-m. *Chilodonellosis*: *O. aureus, Israel*; g. live *Chilodonella hexasticha*, *O. mossambicus*, S. Africa; h. *C. hexasticha*, silver impregnated, *O. mossambicus, S. Africa*; i.C. (= *cyprini O. mossambicus*, S. Africa (h,i. by courtesy of *L. Bassok–m*. Gill damage in *O. aureus*x*niloticus*, Israel: k,l, abrasion and desquamation (arrows- proliferating chloride cells) and m, epithelial hyperplasia.

Plate 2 continued ectoparasite protozoa: a. Scanning electron microscopic view of severe gill chilodonellosis in *O. aureus* x *niloticus* Israel. b. *Ambyphrya* sp. from *O. mossambicus* S. Africa (haematoxylin stained). c. same *Ambyphrya* sp. live. d. *Apiosoma* sp. live ex *O.mossambicus*. e. Stalked *Apiosoma* sp.*O. mossambicus*. f. Scanning electron microscopic view of *Scopulata* sp. *O. aureus* x *niloticus* fry, Israel. g. *Scopulata constricta* from *O. mossambicus*, S. Africa (haematoxylin stained) (courtesy of *L. Basson*). h. Enlarged view of live, stalked *Apiosoma, O. mossambicus*, S. Africa. i. *Scopulata* sp. live, from *O. mossambicus*, S. Africa. k. *Apiosoma piscicola* from carp skin (haematoxylin stained).

Plate 3 shows Ectoparasitic Protozoa: Trichodinids: a,b. *Trichodina heterodentata*: a, live, carp, Israel; silver impregnated, *O. aureus x niloticus, Israel*. c. Scanning electron microscopic (SEM) view of *T. reticulata* of goldfish. d,e. SEM view of *O. aureus* x *niloticus* gill infection with *Tripartiella cichlidarum*; f. same as d, in histology.

Plate 4 continued Ectoparasitic Protozoa: trichodinids: Silver impregnated trichodinids: a. *Trichodina compacta*

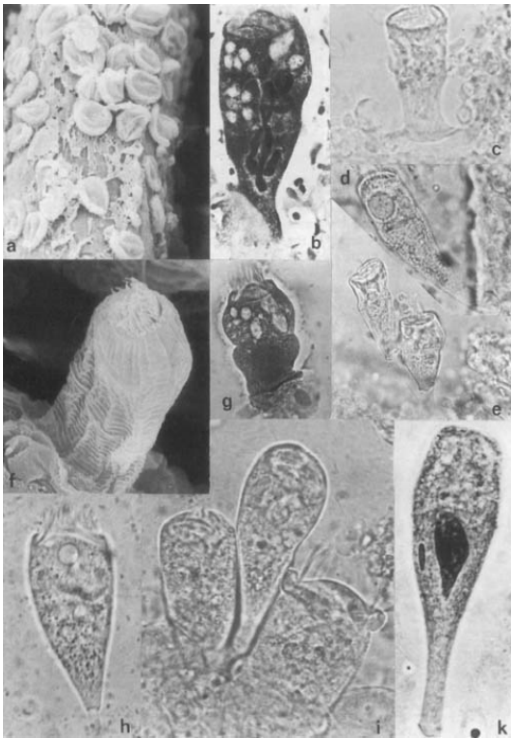

Plate 1: Ectoparasite protozoa

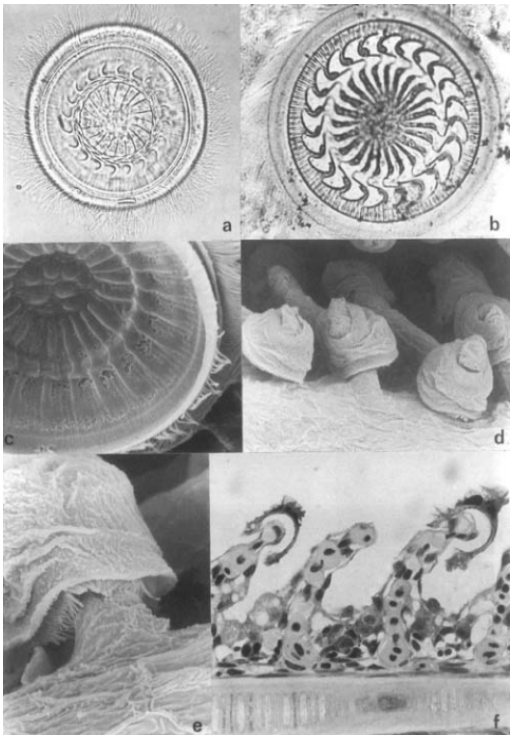

Plate 2: Continued ectoparasite protozoa

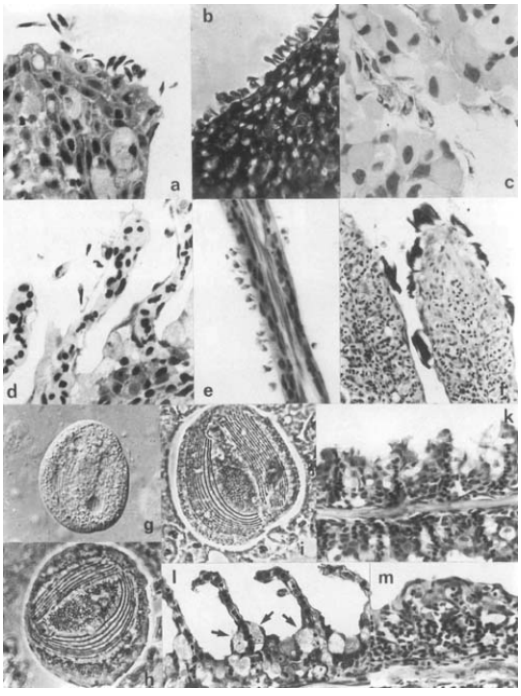

Plate 3: Ectoparasite protozoa. Trichodinid

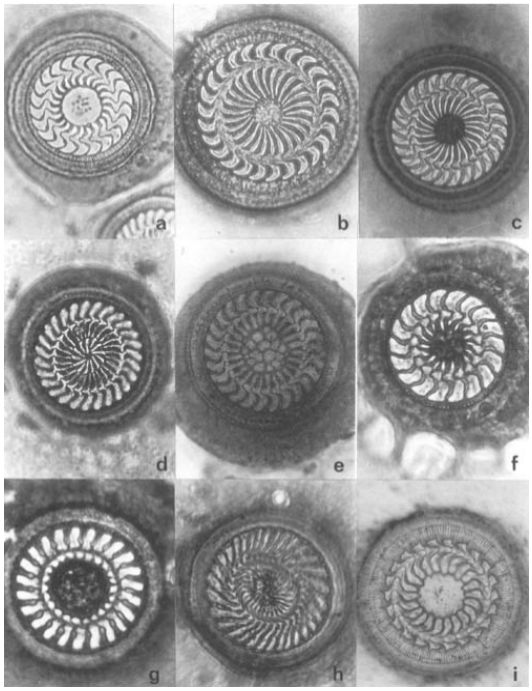

Plate 4: Ectoparasite trichodinid

(prev. *acuta*), *O. mossambicus*, S. Africa. b. *T. migala* (prev. *pediculus*), *O. mossambicus*, S. Africa. c. *T. mutabilis*, *Barbus paludinosus*, S. Africa. d. *T. centrostrigata O. mossambicus*, S. Africa. e. *T. reticulata*, farmed goldfish, Israel. f. *T. minuta*, *O. mossambicus*, S. Africa. g. *Trichodinella epizootica*, Carp, S. Africa. h. *Tripartiella cichlidarum*, *O. aurea*, Israel. i. *Hemitrichodina robusta Marcusenius macrolepidotus* (by courtesy of *L. Basson*). Flagellates [Mastigophora, Kinetoplastida]:

Cryptobia: Free, spindle shaped, 10-30×3-5 μm in size (*C. branchialis*), or pyriform when attached to the integument, with two flagellae, one wholly or partly adjunct to the body, kinetoplast rod-shaped or round.

Ichthyobodo: Free, 13-26×2-7 μm in size, or attached to the integument; with four flagellae.

Ciliates [Ciliophora]: Chilodonella rounded to oval, cytostome distinct, macronucleus round and cilia on the concave ventral surface are arranged in several concave parallel rows:

***C. hexastichas*ize:** 30-65×20-50 μm, with 6-8 ciliary lines on each side.

***C. piscicola* (syn: *C. cyprini*):** size 33-100×24-60 μm, with more than 10 ciliary lines on each side.

Trichodinacup: shaped, 20-100 μm in diameter withconcentric rows of cilia and a crown of denticles. The denticle shape is a distinct taxonomic feature; for differential specific diagnosis of African spp. (Basson *et al*., 1983; Basson and Van, 1987; Van and Basson, 1989, 1992).

Small trichodinids, predominantly from the gills, are bell shaped (*Tripartiella* and *Paratrichodina*) and often settle on the tips of the gill lamellae (Basson and Van, 1989, for generic division of Trichodinidae). In *Trichodinella*, the ray (the inner extension of the denticle) (Van and Basson, 1989), is totally reduced, and in the other two genera is delicate or rudimentary (Kazubski and El-Tantawy, 1986; Basson and Van, 1987).

Genera of sessile peritrichs are differentiated by their macronuclei and scopula (attachment leg) (Viljoen and Van, 1983, 1985): *Scopulata* (*Scyphidia*) round macronucleus and wide scopula;

Apiosoma (*Glossatella*) pyriform nucleus, small scopula; *Ambyphrya* Ribbon shaped macronucleus and wide scopula. Stalked sessile peritrichs - Heteropolaria with elongate body and curled macronucleus (Foissner *et al.*, 1985); *Epistilis* cup-shaped with horseshoe-shaped macronucleus (Viljoen and Van, 1983). Some *Apiosoma* also develop on stalks. Suctoria: (*Trichophyra* and other genera) - cilia lacking, variable numbers of tentacles arise from the rounded body.

Life cycle and biology: Most ectoprotozoans, flagellates as well as ciliates have simple life histories. Species of *Cryptobia* are ectoparasites as well as intestinal and vascular parasites. It has been shown that an ectoparasitic phase occurs in two vascular species (Woo, 1987). Both ectoparasitic flagellates, *I. necator* and *Cryptobia* spp., occur either free swimming or attached to the integument, the former through a cytoplasmic protrusion (Schubert, 1968) and the latter by attachment with the flagellum (Lom, 1980). Reproduction is usually by binary fission. Conjugation is sometimes observed in ciliates. Sessile species also bud and give birth to a free swimming mobile generation, reminiscent of mobile peritrichs, which settle on suitable substrates (fish). The sessile suctorians reproduce by internal and external budding, the detached buds are ciliated. As the buds become attached to a new location on the piscine integument, cilia are shed and tentacles appear (Hoffman, 1978). Spores or other forms of waiting stages are unknown; the suggested existence of waiting stages, such as encysted forms of *Chilodonella hexasticha* in the gills (Rowland *et al*., 1991) or free cysts (Bauer *et al*., 1969), has to be confirmed.

Water temperatures do not seem to be an important parameter, in spite of reports of low temperatures being more optimal for reproduction of *Chilodonella piscicola* and some trichodinids (Bauer *et al*., 1969). Massive infections with *I. necator*, both species of *Chilodonella* and the ubiquitous trichodinids and sessile species, occur in low (12-17ºC) and high (25-30ºC) ambient temperatures in southern Africa and Israel. Most freshwater ectoparasitic protozoans disappear in ponds with increased salinities (above 2000 ppm chlorinity), only *I. necator* and some *Cryptobia* are tolerant and become the predominant parasites in fish of such ponds. There are also halophilic species of *Ambyphrya* and *Scyphidia* which infect fish (grey mullet) in estuaries.

A number of ciliates (species of *Tetrahymena*, *Ophryoglena*, *Glaucoma*, *Colpidium* and others (Hoffman, 1978) are facultative parasites, or opportunists which will colonise fish in special circumstances, most often when fish are stressed or traumatised (Hoffman, 1978). All others mentioned above are obligatory parasites which will apparently survive for only a limited time outside their hosts. Non-parasitic sessile peritrichs are different species from those colonising living organisms. Trichodinids and sessile species found on aquatic invertebrates comprise different species from those infecting fish (Van and Basson, 1987; Viljoen and Van, 1983, 1985). There are, however, a few documented exceptions: *T. pediculus* being reported from both hydra and fish, and *T. diaptomi* a parasite of a calanoid copepod, which temporarily invaded hatchery grown fry of *Clarias gariepinus* (Basson *et al*., 1983; Basson and Van, 1991).

There are several degrees of adaptation of trichodinids to their piscine hosts: ubiquitous species, of an opportunistic nature, which are always found on the fish skin but never on the gills (*T. pediculus* and *T. acuta*); other ubiquitous species occur both on gills and skin (*T. heterodentata*); additional, seemingly ubiquitous, widespread species appear to have a variable degree of predilection for one fish family or another (cichlids or cyprinids). Among the latter, trichodinids with seemingly related morphological characteristics (e.g., *pediculus*-like, *acuta*-like and *nigra*-like), in different geographical regions, demonstrate definite affinities to a particular group of hosts and may in fact comprise diverse species (Van and Basson, 1989). Host specific trichodinids, are all, with only a few exceptions, gill parasites: *T. centrostrigata* and great numbers of small trichodinids mainly species of *Tripartiella*, are associated with Cichlidae; *T. reticulata* occurs mainly in goldfish, *T. kazubski* has been found in South African *Barbus* spp. and *T. nobilis* and *T. kupermani* mainly in asian carp (Basson *et al*., 1983; Van and Basson, 1987, 1989; Abaladejo and Arthur, 1989).

Pathology: Ectoparasitic protozoa are variable in their effect on their hosts. Pathological effects are density dependent, when both the size of the parasite population and the nature of the tissue responses are modulated by the physiological (clinical) condition of the fish. Hostile environments (stressful conditions) compromise the fishes' capacity to counteract infection.

Ichthyobodo necator attaches itself to epithelial cells and through an inserted protrusion consumes their contents (Schubert, 1968), whereas *Chilodonella* spp. browse the epithelial surface (Paperna and Van, 1983). Histopathological changes in the integument following infection by *Chilodonella* spp. and *I. necator* are an outcome of two counteracting cellular processes - hyperplasia of the epithelial cells, including mucus cells and chloride cells, versus a progressive cellular destruction. Cellular destruction primarily occurs due to direct action of the parasites, and later by enhanced abrasion of the peripheral cells after the depletion of mucus forming cells. The production of mucus cells is limited. Accelerated mucus cell production stimulated by the infection apparently exhausts resources for mucus production, and the infected fish become “dry”. Some parasites seem to yield cytotoxins or proteolytic enzymes which could be the cause of spongiosis, which affects both the proliferated and unchanged epithelial layer (Robertson *et al.*, 1981; Paperna and Van, 1983). Secondary cellular damage due to degeneration, necrosis and desquamation results in the degradation and disintegration of the epithelial layer.

Cryptobia attachment through the flagellum does not induce any pathological or even ultrastructural cellular damage (Lom, 1980), contrary to reports of morbidities associated with this parasite (Woo, 1987). Although there are a number of reports on poor condition and mortalities, particularly of fry, coinciding with massive infestation of trichodinids, *Trichodinella epizootica* in particular (Lom, 1973), and the sessilians *Apiosoma*, *Ambyphrya* and *Scopulata* (Fijan, 1961; Meyer, 1970; Paperna, 1984b,1980) Lightner *et al.*, 1988), histopathological changes in events of massive infections by these ectoprotozoans are hardly evident, if occurring at all (Fitzgerald *et al.*, 1982; Paperna, 1985). *Trichodinella epizootica* in carp (Lom, 1973) and *Tripartiella cichlidarum* in Paperna (1980) cichlids cause some erosion of the gill epithelium. However, food vacuoles of trichodinids revealed bacteria rather than sloughed cells.

Ultrastructural observation on attached *Apiosoma* did not reveal any interference with the host cell serving as substrate (Lom and Corliss, 1968; Lom, 1973; Fitzgerald *et al.*, 1982) or peripheral tissue response. Thus, mortalities following massive colonisation of gills by sessilians (Fijan, 1961) could result from the dense cover of sessilians disrupting gas exchange through the respiratory epithelium. The only exception among these infections are the colonies of the stalked sessilia *Heteropolaria (Epistilis)* which cause lesions (“red sore”) at the stalk attachment to the fish skin, these inflamed haemorrhagic lesions are also contaminated with the bacterium *Aeromonas hydrophila* (Esch *et al.*, 1976; Miller and Chapman, 1976). Reported localised infection above the opercular bone (in cultured tilapia in Israel) resulted in aggravation of the lesion into a wide (6 mm in diam.) perforation of the bone. Suctorians (*Trichophyra* spp.) in certain instances cause cytological damage to the gill lamellae cells in direct contact with the parasites and subsequent hyperplasia and haemorrhages of the gill tissue (Heckmann and Carroll, 1985).

Epizootiology: The course of infection by ectoparasitic protozoans is determined either individually or by the interaction of the following factors:

- Mobility of the fish
- The fish's capacity to activate its defence systems
- Reduced mobility facilitates parasite colonisation as well as moderating loss through detachment
- Drift from the integumental surface

Defence mechanisms other than epithelial hyperplasia, and specific immune responses to integumental ectoparasites have not yet been studied except in *I. multifiliis*, although spontaneous recovery from infection has been frequently observed. Juvenile fish and fish under stress and at below optimum ambient temperature have both limited mobility and apparently immunological incompatibility, being either naive or immunosuppressed (Sniezko, 1964; Avtalion, 1981).

Heavy infections by ectoparasitic protozoans are mainly found in young fish (less than one year old) when overcrowded and confined to restricted habitats, and under stress conditions. In these circumstances opportunistic and ubiquitous species are involved. Infections otherwise, in grown-up fish, are very low and host-specific species predominate.

Cichlids fry, as soon as they were weaned from parental care, and sometimes before, became heavily infected by trichodinids and sessilians of the genera *Ambyphrya* and *Scopulata*. Infestation reached its climax level in fish 10-12 mm long. Such infections occur in natural habitats (lakes), man-made impoundments, as well as in hatchery installations (Fryer, 1961; Paperna, 1984c). Heavy infections, however, were not found in all the breeding habitats of the investigated lake system. Conditions for infestation varied with habitat and ambient conditions and were positively related to the abundance of fry schools.

Level of infection in the fry sharply declined as fish gained in size. The decline in infection also coincided with changes in parasite species composition, the ubiquitous, generalists and opportunists: *T. pediculus T.migala, T. acuta, T. compacta, T. heterodentata,* and species of *Ambyphrya* and *Scopularia* being gradually replaced by species specific to cichlids: *Tripartiella* spp. *Trichodina centrostrigata* and species of *Apiosoma* (Basson *et al.*, 1983; Kazubski and El-Tantawy, 1986; El-Tantawy and Kazubski, 1986; Basson and Van, 1987; Van and Basson, 1989).

Heavy infections with ubiquitous trichodinids (*T.pediculus*) and sessile peritrichs (mainly *Scopulata* spp.) also occur in carp fry in hatcheries and nursery ponds, and likewise as fish grow, are replaced by more specialised species such as *T. nigra, T. mutabilis* and *Apiosoma* spp. (Basson *et al.*, 1983; Shaharom-Harrison and Abdullah, 1988; Abaladejo and Arthur, 1989). Heavy infections (by trichodinids and sessile species) accumulate in fish - small spp. of *Barbus, Alestes,* cyprinodontids and juvenile cichlids and *Clarias* spp. crowded into residual pools in rivers drying-out during the dry season. In larger water bodies in Africa, infections with both trichodines and sessilians in fish other than fry may be common but low (Paperna, 1968; Viljoen and Van, 1985; Kazubski, 1986). Low temperature stress plays an important role in epizootic outbreaks of ectoprotozoan infections in cichlid fish outside the limits of the tropical environment and of populations introduced to non-tropical countries such as the southern USA.

Heavy infections by skin and gill protozoa, predominantly of *Chilodonella* spp., are a frequent occurrence in overwintering stocks of cultured tilapia hybrids (*Oreochromis aureus x niloticus*) in Israel, and *O.mossambicus* in ponds and dam reservoirs in southern Africa (Du Plessis, 1952; Oldewage and Van, 1987; Paperna, and Van1983; Paperna, 1984b).

In small ponds (1 hectare) fish are not spared even in relatively mild winters, with minimum temperatures above 13ºC. Fish in lakes and large reservoirs on the other hand, become severely affected only in extremely cold winters, with temperatures declining to 10ºC and below. Mortalities often occur from the cumulative effects of ectoprotozoans, dermal saprolegniases and systemic bacterial diseases, all mediated by the stress of low temperature. In addition to temperature stress, overwintering tilapia in ponds are often stressed by overcrowded stocking and inadequate feeding. Intermittence of higher and lower ambient temperatures, characteristic of the Mediterranean type winters, increases the unpredictability of food demand by fish and thus complicates feeding schedules.

Infestation levels rise by late fall, with increased abundance of trichodinids and *C. hexasticha*. Fish succumbing in early winter were predominantly hyperinfected by *C. hexasticha*. Late winter and early spring mass mortalities (even when temperatures were already rising above 15ºC) were associated with *C. piscicola* hyperinfections. *C. piscicola* is abundant in carp in some ponds already by early winter; however, it will only infect tilapia at the end of the cold season when they become compromised by prolonged stress.

Ichthyobodo necator hyperinfections are morbid to cichlids as well as to fish of other families. Mortalities occur in fish overcrowded in holding tanks, ponds and in both warm and cold water conditions. Natural infection was also revealed in *Aplocheilichthys gambiae* from a pool in Ghana. In Israel *Cryptobia* spp. occasionally swarm the gills of tilapia, goldfish and silver carp and also, in the latter, in low saline waters (8-10 ppt salt) but data from Africa are lacking.

O. mossambicus appears to be more tolerant to low temperatures in water of higher salinities, and also where most ectoparasites are excluded except *l. necator*. Members of the genus *Tilapia*, in Israel (*T. zillii*) and in southern Africa (*T. rendalli* and *T. sparmanii*) are also less affected in freshwaters by low temperatures and are rarely heavily parasitised.

Few instances of mortalities coincided with heavy infections, concomitantly or exclusively, by trichodinids, sessilians (*Apiosoma*), *Chilodonella* spp. and *I. necator*, in overwintering carp, but occurred more often in relation to other stress factors such as high levels of overcrowding or high nitrite concentrations (Fijan, 1961; Sarig, 1971). Heavy infections by *Chilodonella* spp. seems to have an excluding effect on other integumental protozoans. Otherwise, skin and gill ectoparasites coexist, and are even synergistic, with metazoan ectoparasites (*Gyrodactylus* and *Argulus*) and skin lesions (epithelioma), (Paperna and Kohn, 1964; Sarig, 1971; Paperna, 1985). Mass mortalities of farmed *Clarias gariepinus* (in the Central African Republic) were associated with mass infestation by *Chilodonella hexasticha*. *Epistilis* infections, including red sore and

opercular perforations only occur sporadically with no particular link to overwintering.

Control: Treatment with formalin is still the only effective means to control massive ectoparasitic infections in all warm water cultured fish species. In Israeli fish farms, ponds are sprayed with formalin up to concentrations of 25 or 40 ppm (of the 37% commercial product) (Sarig, 1971; Lahav and Sarig, 1972). Efficacy of formalin treatments is affected by ambient temperatures, water quality, including salinities and parasites treated. Product quality is variable, and is particularly affected by storage, resulting in accumulation of polymerised (paraformaldehyde) sediment. Trichodinids were readily eradicated with treatment by 25 ppm, while elimination of *Chilodonella* was achieved after treatment with 40-50 ppm. Van *et al.* (1984) also demonstrated differential efficacy with the type of fish treated, e.g., 25 ppm per 24 h was effective in cleaning infected carp, while with tilapia fry it has been achieved with 45 ppm per 24 h.

DINOFLAGELLIDS AND ICHTHYOPHTHIRIASIS INFECTIONS

Parasite dinoflagellids: Parasitic dinoflagellids,the marine *Amyloodinium ocellatum* and the freshwater *Piscinoodinium pillulare* and *P. limneticum*, are not discriminatory in their choice of piscine hosts and have been implicated in mass mortalities of tropical marine and freshwater aquarium fish (Jacobs, 1946; Paperna, 1980). *A. ocellatum* has infected sea water acclimatised *Oreochromis mossambicus* and *Aphanius dispar* in inland salt pans; some strains of this parasite survive in salinities as low as 10 ppt. *P. pillulare* infection in 14 species of tropical ornamental freshwater fish of diverse families as well as in carp and crucian carp. Epizootic infections and mortalities were recently reported in farmed cyprinid fish in Malaysia, including grass carp (*Ctenopharyngodon idella*), bighead (*Aristichthys nobilis*), *Leptobarbus hoevenii* and *Puntius gonionotus* (Shaharom-Harrison *et al.*, 1990). The presence of *P. pillulare* has never been established in Africa, but this ubiquitous parasite may eventually be found. If introduced with culture seed, it is likely to become established.

Diagnosis: Trophonts, when reaching the final stage of growth, are visible to the naked eye (80-100 μm diameter) as white spots (similar to that seen in ichthyophthiriasis) and turn dark blue when exposed to Lugol's-iodine. They are oval with a smooth wall and with inner aggregates of globules. In Malaysian fish, clinical signs of *P. pillulare* infection comprise both a rust-coloured appearance of the skin, indicating the presence of the parasite trophonts (20-75×14-50 μm), and a dense covering of mucus (Shaharom-Harrison *et al.*, 1990).

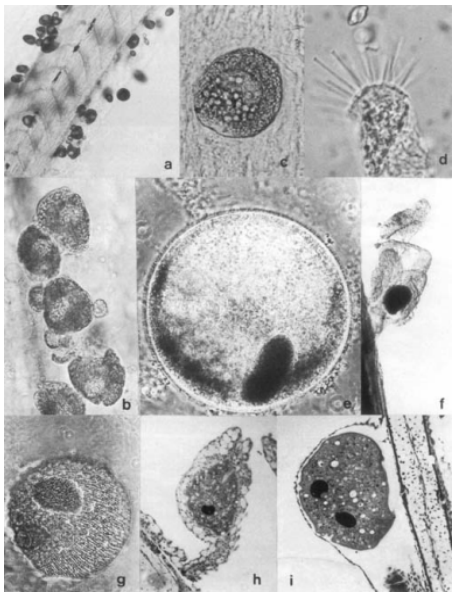

Plate 5: Dinoflagellids and ichthyophthiriasis

Plate 5 shows Dinoflagellids and Ichthyophthyriasis: a. *Piscinoodinium* sp. on *Colisa lisa* fry (Photo A.Diamant). b. Enlarged view of a. c. *Amyloodinium ocellatum* on farmed *Sparus aurata* fry, Red Sea. d. *Trichophyra* sp. (Suctoria) from *Morone saxatilis*, USA. e-i. *Ichthyophthirius multifiliis*: e, live trophont, carp, Israel (by courtesy of S. Sarig); f, benign infection on eel's (*Anguilla anguilla*) skin, live; g, Silver impregnated, *Oreochromis mossambicus*, SA; h, histology of same infection as f; i, benign infection carp.

Life cycle and biology: The life cycle of the dinoflagellid fish parasite is comprised of a parasitic feeding stage (trophont) which attaches to integumentary epithelial cells, and an encysted dividing stage (tomont) which is detached from the host. The trophonts of *P. pillulare* derive an essential part of their nutrition from photosynthesis. Trophonts dislodged at any time during their trophic stage will transform into a dividing tomont. Divisions yield a motile infective stage (dinospore) which attaches to a new host. There are several detailed studies of *A. ocellatum* (Paperna, 1984a), but comparable detailed data on the freshwater fish dinoflagellids are lacking. Data on *P. limneticum* growth and division (Jacobs, 1946), suggests that parasites reach a "maturation" prior to detachment. *P. pillulare* trophonts on the gills, at 23-25ºC, develop from dinospore to detached tomont in three to four days. The tomont then completes division to the dinospore stage within 50-70 h. At 15-17ºC, the process of division is lengthened to 11 days.

Pathology and epizootiology: In larval fish, infections were limited to the skin, whereas in large fish the highest

parasite densities occurred on the gill filaments and in the buccal-pharyngeal integument. Fish recovering from the epizootic infestation through a gradual decrease in infection, could not be reinfected (Paperna, 1980). *A. ocellatum* is attached to and feeds from the host epithelial cell by means of rhizoids, which penetrate the host cell. The consumed cell gradually degenerates and collapses. Damage to infected cells leads to focal erosion of the epithelium. Prolonged infection exhausts a generation of mucus cells and leads to accelerated desquamation. Proliferation of the epithelium causes obliteration of the gill lamellae, while the inner strata of the epithelium become spongious and in some cases undergo complete lysis (Paperna, 1980).

Attachment and penetration organelles of *P. pillulare* differ from those seen in *A. ocellatum*, in that the host cell is penetrated by nail-like extensions. However, damage to the host cell is similar (Lom and Schubert, 1983). Significant histopathological changes are only seen in the gills, where most of the infection occurs, namely a massive proliferation of the branchial epithelium which causes fusion of the lamellae by a confluent cellular mass (Shaharom-Harrison *et al.*, 1990). *Piscinoodinium* infection in Malaysia initially occurred among ornamental fish, but it spread eventually to pond farmed local and exotic cyprinids, causing mortality in fry of *Puntius gonionotus* in particular, although clinical signs were also apparent in a wider range of cyprinid fish species (Shaharom-Harrison *et al.*, 1990).

Control: A. ocellatum is controlled by continuous application of copper sulphate, 0.75 ppm into infected tanks. A further option is a mixture of 5-hydrate copper sulphate with citric acid monohydrate, to yield 0.15 ppm copper ion concentration in the water (Kabata, 1985). The same methodology will apparently effectively control *Piscinoodinium* infection, although concentrations should be adjusted to the freshwater medium and the fish targeted for treatment. In freshwater with a pH below 7.0 (in tropical aquaculture), concentrations above 0.3 ppm may be lethal to fish (e.g. *Puntius gonionotus* fry).

ICHTHYOPHTHIRIASIS

Most species of freshwater fish are susceptible, although some may be more so than others. The world wide distribution of *I. multifiliis* (Hoffman, 1978) has apparently been facilitated by the widespread translocation of cultured and ornamental fish. The presence of this parasite in autochthonous fish, in remote areas of the world including, southern Venezuela (Ventura and Paperna, 1985) and Northern Transvaal in South Africa (Paperna, 1985) may suggest, however, that many populations, particularly those in the tropics, are comprised of a mix of autochthonous and introduced

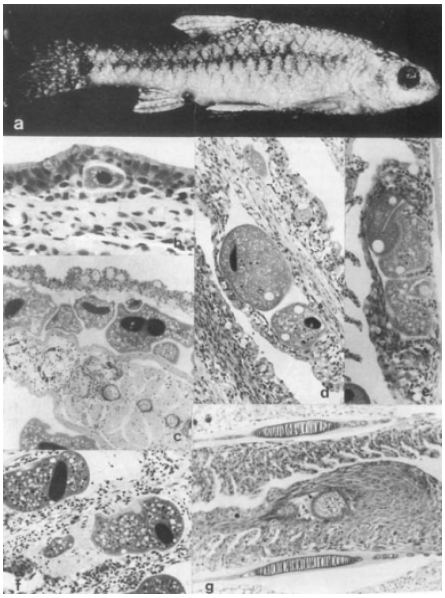

Plate 6: Ichthyophthiriasis

parasites. Data available from Africa are limited to Southern Africa (in cichlids, carp, *Barbus* spp., trout and eels - Du Plessis, 1952; Lombard, 1968; Van *et al*., 1984) and Uganda (on native *Barbus amphigramma* and exotic *Lebistes reticulatus* from small streams at Paperna 1972). It is very common in Israel, in both farmed (Sarig, 1971; Hines and Spira, 1973a) and wild fish including cichlids (Ventura and Paperna, 1985).

Diagnosis: Gross signs - white spots on the skin and the gills, which under microscopic examination reveal (in skin and gill scrapings) uniformly ciliated organisms with a small cytostome, which may reach up to 1 mm in diameter. Staining with either haematoxylins or Giemsa (after adequate fixation, in such as Bouin) reveals a large crescent-shaped macronucleus and small micronucleus. *Ichthyophthirius multifiliis* is a monotypic genus of hymenostomatid ciliates.

Plate 6 shows a continuation of Ichthyophthiriasis: a. heavily infected *Barbus amphigramma*, from Uganda; b. Early invasion of the pharyngeal mucosa of young eel, experimental. c. Heavy infection in skin of *Clarias lazera* fry, Israel; d, Gill infection of *Oreochromis mossambicus*, Transvaal, South Africa. e. Parasites displacing gill lamellae of carp, Transvaal, South Africa. f. Cytolysis in gill infection of *Haplochromis flavii-josephi*, Lake Kinneret, Israel. g. Infection resulting in proliferation of the gill epithelium of *Apistogramma* sp. from South Venezuela.

Life history and biology: Trophonts (feeding stages) develop within the integumentary epithelium, always

above the basal membrane (Ventura and Paperna, 1985; Ewing and Kocan, 1992). By maturity, which is reached in 2 days at ambient temperatures of 25-28ºC (3-4 days at 21-24ºC), the parasite evacuates the host tissue and settles within 2-6 h on a substrate in the water to form a cyst-encapsulated tomont (dividing stage). Parasites evicted from the tissue before the scheduled time for their spontaneous departure, fail to develop into tomonts and eventually die (Ewing and Kocan, 1992; Paperna, 1985). Within the cyst, tomonts undergo successive binary fissions with a resulting yield of 250-2000 tomites (infective, free swimming stages), which after release will seek a suitable host. The division of tomonts into tomites, in ambient temperatures of 25-28ºC, is completed within 15-20 h (Bauer, 1959; Meyer, 1969; Hoffman, 1978).

Invasion of tomites (teronts) (30-45 μm long) into the host integument is facilitated by the excretion of a sticky substance from subpellicular crystalline organelles named mucocysts. Active penetration causes focal necrosis of the epithelial cells. It has been suggested that hyaluronidase and other enzymes may be produced by the penetrating parasite (Ewing *et al*., 1985). In the absence of a suitable host, tomites will lose their infective potential within 24 h at 24-28ºC (Ewing and Kocan, 1992). Higher temperatures hasten trophont maturation and tomont division, but at lower temperatures, slower development allows the growth of larger trophonts (0.8-1.0 mm in 5-10ºC vs 0.5-0.7 mm in 20-24ºC), yielding tomonts with higher numbers of tomite progeny (Ewing *et al*., 1986). In lower ambient temperatures the survival of the tomites is prolonged, thus, allowing more time to locate a host. Low temperatures do not interrupt propagation; a full cycle is completed at 20ºC in 3-5 days, at 15ºC in 7-14 days and at 10ºC in 21-35 days (Bauer, 1959; Meyer, 1969). Data on the effect of other environmental parameters is less conclusive, although it has been suggested that dissolved oxygen levels below 1 mg/L affect parasite reproduction (Bauer, 1959).

Pathology: Ichthyophthiriasis is fatal to fish of all sizes. Chronic infection will cause serious damage to the skin, fin and gills; corneal infection impairs vision (Hines and Spira, 1973a, 1974a). The infective stage invades the integumentary epithelium and becomes established in the basal layer of the epithelium just above the basal membrane. Cellular damage in low to moderate infections remains restricted to the infected site. In addition to the damage caused to epithelial cells by the feeding and expanding parasites, in heavy infections mass exodus of parasites from the epithelial layer, having completed their scheduled growth, causes its erosion and detachment from the basal membrane. In some infections, parasites cause widespread lysis of the inner layer of the epithelium. Prolonged infection also induces epithelial proliferation and haemorrhagic inflammation, causing the integument to become severely disintegrated (Hines and Spira, 1974a; Ventura and Paperna, 1985; Ewing *et al*., 1986). Hines and Spira (1973b, 1974a,b) haematological and clinical data from heavily infected fish reveal evident physiological dysfunction resulting apparently from both direct pathological damage induced by the parasite and as a by-product of the stress response.

Epizootiology: Ichthyophthirius multifiliis is one of the most common, troublesome and difficult to control of fish pathogens. Epizootic infections have been reported in cold water salmonid farms (also in Africa, Du Plessis, 1952; Lombard, 1968) and warm water farmed carp, eels, *Clarias gariepinus* and *Ictalurus punctatus* (channel catfish) (Meyer, 1970; Sarig, 1971; Hines and Spira, 1973a; Hines, 1975; Jackson, 1978; Khalifa *et al*., 1983). Fish may maintain low, subclinical (enzootic) infection, while encysted tomonts may persist in the habitat. Enzootic infections in native fish have been found in *Lebistes reticulatus* in Uganda (Paperna, 1972), in cichlids and cyprinids native to the Lake Kinneret system in Israel, in glass eels, cyprinids and cichlids in native habitats of South Africa (Jackson, 1978; Van *et al*., 1984), and in a variety of native fish in the southern United States (Allison and Kelly, 1963).

Transition from nonclinical enzootic to epizootic clinical infection is usually stress-mediated, prompted by adverse growth conditions such as overcrowding, poor feeding and excess nitrogenous waste. Epizootic infection, however, never occurs in overwintering tilapia or *Clarias gariepinus* in Israel, or southern Africa, but rather, coincides with the warming of the water in early spring when fish are still kept in overcrowded conditions after winter storage. In South Africa, 6.4% of wild glass eels in the Southern Cape are infected. Via these, fish infection has been introduced into eel nurseries where elvers, especially those not completely acclimated, succumbed to severe infestations (Hines, 1975; Jackson, 1978).

Spontaneous recovery has been observed in both natural infections in natural habitats and in holding facilities, and even in experimental infections in aquaria (Paperna, 1972; Lahav and Sarig, 1973). The potential for spontaneous recovery varied with fish species. Infection in scaled fish, notably cichlids, regressed faster than in smooth skinned fish (eels, *Clarias* spp. and other siluriforms, mirror and leather carp) (Paperna, 1972). After recovery, fish were refractory to reinfection or retained a merely subclinical chronic infection (Hines and Spira, 1974c; Wahli and Meier, 1985; Paperna, 1985). The observed interspecific variation in susceptibility to infection could, however, also result from differential compatibility of various fish species to man-made habitats and variable vulnerability to stress.

Spontaneous recovery from infection and resistance to reinfection of recovered fish indicate that fish are capable of developing defence mechanisms against *I.multifiliis* (Hines and Spira, 1974c). Spontaneous

recovery observed in carp at temperatures as low as 10ºC (Lahav and Sarig, 1973) implies some protective responses other than via humoral antibody production, which becomes suppressed in carp below 12ºC (Avtalion, 1981). Hines and Spira (1974c) demonstrated immobilisation of free swimming tomites with sera taken from carp after their recovery from infection. The infective stages were also shown to be unable to penetrate the skin of resistant carp. Immobilisation tests with trophonts showed that in infected trout anti-parasitic activity of the mucus increases quickly after infection and decreases soon after the infection has disappeared.These results could have simulated a The anti-parasitic activity of the serum, in the same fish, increases slowly but remains at a higher level for at least 7 months (Wahli and Meier, 1985). Fish immunised with *Tetrahymena* spp. developed a resistance to a challenge of *lchthyophthirius* infection (Goven *et al.*, 1981).

Carp were immunised following controlled exposure to the infective tomite (teront) stage, and survived challenges with high infective doses, but lost protection after being immunosupressed by the administration of corticosteroids. These results could have simulated a stress mediated situation. Levels of humoral antibodies in immunosuppressed fish, however, remained the same as in the immunised group, which further confirms the involvement of other than humoral type immune systems in the protection processes against *l. multifiliis* infections (Houghton and Matthews, 1990). Immunisation with killed vaccines gave less satisfactory results, although better protection was obtained through intraperitoneal inoculation of live teronts (Burkart *et al.*, 1990). results could have simulated a the latter method, passively transferred a protective immunity to their fry (Subasinghe and Sommerville, 1989). Additionally to immunity passed from mothers via eggs, demonstrable by antibodies in the soluble extracts of fry tissues, a protective immunity was acquired directly from the parent mouth during the brooding period (Sin *et al.*, 1994).

Control: Both trophonts, localised beneath the epithelial layer of the integument, and the encysted tomonts, attached to substrates in the aquatic habitat, are resistant to practically all externally applied usable antiparasitic agents. Infection can be effectively controlled only by destruction or elimination of the free dividing tomonts or the tomites they release. In warm water systems (24-28ºC), three to four daily transfers of fish to clean tanks will effectively reduce infection, while enabling the fish to develop tolerance to reinfections. Tomonts can be effectively removed from large circulating tanks by repeated brushing with vacuum suction. Spontaneous recovery and transition into a refractory state will be further promoted by management techniques which alleviate stressing conditions (improving water flow, accelerating aeration and reducing stocking densities). Chemical parasiticides will be effective only through continuous or repeated daily application. Of the many listed (Meyer, 1969), the only cost-effective remedy for large scale farming systems is Malachite green at a dose of 0.05 ppm for continuous application (3-4 days) or up to 0.15 ppm (depending on the specific fish tolerance which varies with species - siluriforms are particularly susceptible). Formalin will dislodge some of the trophonts and is often applied mixed with Malachite green (50 ppm with 0.05 ppm) (Sarig, 1971).

Systemic therapy seems to be the only means of effective control. Elimination of tissue trophozoites was reported in several species of ornamental aquarium fish fed medicated food (Tetra, MA 100/50) containing Malachite green in a non-water soluble formulation for 4 days (neither drug concentration in the food, nor daily rations are given; Schmahl *et al.*, 1992). For use in commercial food-fish culture for human consumption, the cost efficiency of Malachite medicated feed formulations and their toxicity to humans must be considered.

CONCLUSION

Fish culturist and the private sector need to know some common parasite diseases: Ichtyobodiasis, Coccidiosis, Ichtyopthiariasis, Trichodiniasis, Heminthiasis, Crustacean parasite, Hirudinea infestation, Flagellates and Ciliates, Taxonomy and diagnosis, Life cycle and biology, Epizootiology, pathology, control, Infections with parasite dinoflagellids and Ichthyophthiriasis as challenges faced in culture fisheries.

REFERENCES

Abaladejo, J.D. and J.R. Arthur, 1989. Some trichodinids (Protozoa: Ciliophora: Pertrichida) from freshwater fishes imported into the Philippines. Asian Fisheries Sci., 3: 1-25.

Allison, R., and K.D. Kelly, 1963. An epizooticof *Ichthyophthirius multifiliis* in a river fish population. Prog. Fish Cult., 25: 149-150.

Avtalion, R., 1981. Environmental control of the immune response in fish. CRC Crit. Rev. Environ. Control., 11: 163-188.

Bassey, S.E., 2011. A Concise Dictionary of Parasitology. 1st Edn., Zetus Concepts, Port Harcourt, pp: 115, ISBN: 978-2954-40-3.

Basson, L. and J.D. Van, 1987. Trichodinid (Ciliophora; Peritricha) gill parasites of freshwater fish in South Africa. Sys. Parasitol., 9: 143-151.

Basson, L. and J.G. Van, 1989. Differential diagnosis of the genera in the family Trichodinidae (Ciliophora: Peritrichida) with description of a new genus ectoparasitic on freshwater fish from southern Africa. Sys. Parasitol., 13: 153-160.

Basson, L. and J.G. Van, 1991. Trichodinids (Ciliophora: Peritrichia) from a calanoid copepod and catfish from South Africa and notes on host specificity Sys. Parasitol., 18: 147-158.

Basson, L. and J.G. Van, 1993. First record of European trichodinids (Ciliophora: Peritrichida), *Trichodina acuta* Lom, 1961 and *T. reticulata* Hirschmann ET Partsch, 1955 in South Africa. Act. Protozool. 32: 101-105.

Basson, L., J.G. Van and I. Paperna, 1983. Trichodinid parasites of cichlids and cyprinid fishes of South Africa and Israel. Sys. Parasitol., 5: 245-257.

Bauer, O.N., 1959. The ecology of freshwater fish. *Inves. Gosud.* Nauch-Issled. Inst. Ozer. Rech. Ryb. Khoz, 49: 5-206 (In Russian, English transl. Israel Prog. Sci. Trans. cat. No. 622, 1962, 3-215).

Bauer, O.N., V.A. Musselius and Y.A. Strelkov, 1969. Diseases" Moskva. In English: Israel Program for Scientific Translations, Jerusalem, 1973.

Bondad-Reantaso, M.G. and J.R. Arthur, 1989. Trichodines (Protozoa: Ciliophora: Peritrichida) of Nile tilapia (*Oreochromis niloticus*) in the Philippines. Asian Fisheries Sci., 3: 27-44. Entebbe 37: 128-130.

Burkart, M.A., T.G. Clark and H.W. Dickerson, 1990. Immunisation of channel catfish, *Ictalurus punctatus* Rafinesque, against *Ichthyophthirius multifiliis* (Fouquet): killed versus live vaccines. J. Fish Dis., 13: 401-410.

Du Plessis, S.S., 1952. Fish diseases in Transvaal C.S.A. Symp. Hydrobiol. Inland Fish Entebbe, 37: 128-130.

El-Tantawy, S.A.M. and S.L. Kazubski, 1986. The trichodinid ciliates from fish, *Tilapia nilotica* from the nile delta. Act. Protozool., 25: 439-444.

Esch, G.H., T.C. Hazen, R.V. Dimock and J.W. Gibbons, 1976. Thermal effluent and the epizootiology of the ciliate *Epistilis* and the bacterium *Aeromonas* in association with centrachid fish. Trans. Am. Microsc. Soc., 95: 687-693.

Ewing, M.S. and K.M. Kocan, 1992. Invasion and development strategies of *Ichthyophthirius multifiliis*, a parasitic ciliate of fish. Parasitol. Today, 8: 204-208.

Ewing, M.S., K.M. Kocan and S.A. Ewing, 1985. *Ichthyophthirius multifiliis* (Ciliophora) invasion of gill epithelium. J. Protozool., 32: 305-310.

Ewing, M.S., M.E. Lynn and S.A. Ewing, 1986. Critical periods in development of *Ichthyophthirius multifiliis* (Ciliophora) populations. J. Protozool., 33: 388-391.

Fijan, N., 1961. Massive invasion of carp fry (*Cyprinus carpio* L.) by protozoan of the genus *Glossatella* (Croatian text, English summary). Vet. Arth. Zagreb, 32: 30-33.

Fitzgerald, M.E., B.A. Simco and L.B. Coons, 1982. Ultrastructure of the peritrich ciliate *Ambyphrya ameiuri* and its attachment to the gills of the catfish *Ictalurus punctatus*. J. Protozool., 29: 213-217.

Fryer, G., 1961. Observation on the biology of the cichlid fish *Tilapia variabilis* Boulenger in the northern waters of Lake Victoria and the Victoria Nile. Rev. Zool. Bot. Afr., 64: 1-33.

Fryer, G. and T.D. Iles, 1972. The Cichlid Fishes of the Great Lakes of Africa. T.F.H. Publications, Neptune City, N.J.

Foissner, W., G.L. Hoffman and A.J. Mitchell, 1985. *Heteropolaria colisarum* Foissner & Schubert, 1977 (Protozoa: Epistylididae) of North American freshwater fishes. J. Fish Dis., 8: 145-160.

Goven, B.A., D.I. Dawe and J.B. Gratzeck, 1981. Protection of channel catfish (*Ictalurus punctatus*) against *Ichthyophthirius multifiliis* (Fouquet) by immunisation with varying doses of *Tetrahymena pyriformis* (Lwoff) cilia. Aquaculture, 23: 269-273.

Heckmann, R.A. and T. Carroll, 1985. Host-parasite studies of *Trichophyra* infesting cutthroat trout (*Salmo clarki*) and longnose suckers (*Catastomus catastomus*) from Yellowstone lake Wyoming. Great Basin Nat., 45: 255-265.

Hines R.S. and D.T. Spira, 1973a. Ichthyophthiriasis in the mirror carp *Cyprinus carpio* L.I. Course of infection. J. Fish Biol., 5: 385-392.

Hines, R.S. and D.T. Spira, 1973b. Ichthyophthiriasis in the mirror carp. II. Leucocyte response. J. Fish Biol., 5: 527-534.

Hines, R.S and D.T. Spira., 1974a. Ichthyophthiriasis in the mirror carp *Cyprinus carpio* (L.). III. pathology. J. Fish Biol., 6: 189-196.

Hines, R.S. and D.T. Spira., 1974b. Ichthyophthiriasis in the mirror carp *Cyprinus carpio* (L.). IV Physiological dysfunction. J. Fish Biol., 6: 365-371.

Hines, R.S. and D.T. Spira, 1974c. Ichthyophthiriasis in the mirror carp *Cyprinus carpio* (L.). V. Acquired immunity. J. Fish Biol., 6: 373-378.

Hoffman, G.L., 1978. Ciliates of Freshwater Fishes. In: Kreier, J.P., (Ed.), Parasitic Protozoa, II., pp: 584-632.

Houghton, G. and R.A. Matthews, 1990. Immunosuppression in juvenile carp, *Cyprinus carpio* L. the effect of the corticosteroid triamcinolone acetonide and hydrocortisone 21-hemisuccinate (cortisol) on acquired immunity and the humoral antibody response to *Ichthyophthirius multifiliis* Fouquet. J. Fish Dis., 13: 269-280.

Jackson, P.B.N., 1978. Health and disease in intensive aquaculture. J.S.A. Vet. Ass., 49: 57-59.

Jacobs, D.L., 1946. A new dinoflagellate from freshwater fish. Trans. Am. Microscop. Soc., 65: 1-77.

Kabata, Z., 1985. Parasites and Diseases of Fish Cultured in the Tropics. Taylor & Francis, London and Philadelphia.

Kazubski, S.L., 1986. The trichodinid ciliates from fish, *Tilapia* sp. from Lake Victoria (Kenya) and description of *Trichodina equatorialis* nom. nov. Act. Protozool., 25: 445-448.

Kazubski, S.L. and S.A.M. El-Tantawy, 1986. The ciliate *Paratrichodina africana* sp. n. (Peritricha, Trichodinidae) from Tilapia fish (Cichlidae) from Africa. Act. Protozoologica, 25: 433-438.

Khalifa, K.A., K.M. Al-Khayat and F.H. Al-Riab, 1983. Ichthyophthiriasis in farmed fish in Iraq. J. Wild life Dis., 19: 145.

Lahav, M. and S. Sarig, 1972. Control of unicellular parasite fauna. Act. Parasitol. Pol., 22: 49-66.

Lahav, M. and M. Sarig, 1973. Observation of laboratory infection of carp by *Ichthyophthirius* multifiliis Foquet Bull. Fish Cult. Israel-Bamidgeh, 25: 3-9.

Lightner, D., R. Redman, L. Mahoney, G. Dickerson and K. Fitzsimmons, 1988. Major Diseases Encountered in Controlled Environment Culture of Tilapias in Fresh Water and Brackish Water over a Three Year Period in Arizona. In: Pullin, R.S.V., T. Bhukaswan, K. Tonguthai and J.L. Maclean, (Eds.), The Second International Symposium on Tilapia in Aquaculture. ICLARM Conference Proceedings 15, Dep. Fish. Bangkok, Thailand, ICLARM Manila, Philippines, pp: 111-116.

Lom, J., 1973. The mode of attachment and relationship to the host of *Apiosoma piscicola* Blanchard and *Epistilis Iwoffi* Faure-Fremiet, ectocommensals of freshwater fish. Folia Parasitol. (October 18, 2011Praha), 20: 105-112.

Lom, J., 1980. *Cryptobia branchialis* Nie from fish gills: ultrastructural evidence of ectocommensal function. J. Fish Dis., 3: 427-436.

Lom, J. and J.D. Corliss, 1968. Observation on the fine structure of two species of the peritrich ciliate genus *Scyphidia* and on their mode of attachment to their host. Trans. Am. Microsc. Soc., 87: 493-509.

Lom, J. and G. Schubert, 1983. Ultrastructural study of *Piscinoodinium pillulare* (Schaperclaus, 1954) Lom, 1981 with special emphasis on its attachment to the fish host. J. Fish Dis., 6: 411-428.

Lombard, G.L., 1968. A survey of fish diseases and parasites encountered in Transvaal.The First Asian Fisheries Forum. Asian Fisheries Society, Ma News letter Limnol. Soc. S. Afr., 11: 23-29.

Meyer, F.P., 1969. Parasites of freshwater fishes; II. Protozoa 3. Ichthyophthirius Multifiliis Fish Dis. Leaflets, U.S. Dep. Int. Bur. Fish. Wild. Washington D.C., pp: 4.

Meyer, F.P., 1970. Seasonal fluctuations in the incidence of diseases on fish farms: A Symposium on Diseases of Fishes and Shellfish. Am. Fish. Soc. Sp. Publ., 5: 21-29.

Miller, R.W. and W.R. Chapman, 1976. Epistilis and Aeromonas hydrophila infections in fishes from North Carolina reservoirs. Prog. Fish Cult., 38: 165-168.

Natividad, J.M., M.G. Bondad-Reantaso and J.R. Arthur, 1986. Parasites of Nile tilapia (*Oreochromis niloticus*) in the Philippines. In: Maclean, L.J., L.B. Dizon and L.V. Hosillos, (Eds.), The First Asian Fisheries Forum. Asian Fisheries Society, Manila, Philippines, pp: 225-259.

Oldewage, W.H. and J.D. Van, 1987. Parasites and winter mortalities of *Oreochromis* mossambica. S. Afr. J. Wild. Res., 17: 7-12.

Paperna, I. and A. Kohn, 1964. Studies on the host-parasite relations between carp and; 24: 269-276.

Paperna, I., 1968. Ectoparasitic infections of fish of Volta lake, Ghana. Bull. Wild. Dis. Ass., 4: 135-137.

Paperna, I., 1972. *Ichthyophthirius multifiliis* (Ciliata, Holotrichia) in fish in Uganda. Prog. Fish. Cult., 34: 162-164.

Paperna, I., 1980. *Amyloodinium ocellatum* (Brown, 1931) (Dinoflagellida) infestations in cultured marine fish at Eilat, Red Sea: Epizootiology and pathology. J. Fish Dis., 3: 363-372.

Paperna I., 1984a. Reproductive cycle and tolerance to temperature and salinity of Amyloodinium ocellatum (Brown, 1931) (Dinoflagellida). Ann. Parasitol. Hum. Comp., 59: 7-30.

Paperna, I., 1984b. Chemical control of *Amyloodinium ocellatum* (Brown 1931) (Dinoflagellida) infection: *In vitro* tests and treatment trials with infected fishes. Aquaculture, 38: 1-18.

Paperna, I., 1984c. Winter Diseases of Cultured Tilapia. In: Agrigrup, (Ed.), Fish Diseases. Fourth COPRAQ Session. Editora ATP Madrid (Espanha), pp: 139147.

Paperna, I., 1985. Infectious Diseases of fish in Extreme Environment. Final Report to the Ministry for Science and Art of Lower Saxony, Germany-unpublished.

Paperna, I. and J.P. Thurston., 1968. Report on ectoparasitic infections of freshwater fish in Africa. Bull. Off. Int. Epizoot., 69: 1192-1206.

Paperna, I. and J.G. Van, 1983. Epizootiology and pathology of *Chilodonella hexasticha* (Kiernik, 1909) (Protozoa, Ciliata) infections in cultured cichlid fishes. J. Fish Biol., 23: 441-450.

Paperna, I., J.G. Van and L. Basson, 1984. Review of Diseases Affecting Cultured Cichlids. In: Fishelson, L. and Z. Yaron, (Eds.), International Symposium on Tilapia in Aquaculture, Proceedings. Tel Aviv University Press, pp: 174-184.

Robertson, D.A., R.J. Roberts and A.M. Bullock, 1981. Pathogenesis and autoradiographic studies of the epidermis of salmonids infested with *Ichthyobodo nacator* (Henneguy, 1883). J. Fish Dis., 4: 113-125.

Rowland, S.J., B.A. Ingram and F.B. Prokop, 1991. Suspected cysts of the protozoan parasite Chilodonella hexasticha. Bull. Eur. Ass. 11: 159-161.

Sarig, S., 1971. The Prevention and Treatment of Diseases of Warmwater Fish Under Subtropical Conditions, with Special Emphasis on Intensive Fish Farming. T.F.H Publications Inc., Jersey Citypp: 127.

Schmahl, G., H. Mehlhorn, H. Schmidt and G. Ritter, 1992. Treatment of fish parasites. 9. Effects of a medicated food containing malachite green on *Ichthyophthirius multifiliis* Fouquet, 1876 (Hymenostomatidae, Ciliophora) in ornamental fish. Parasitol. Res., 78: 183-192.

Schubert, G., 1968. The injurious effects of *Costia necatrix*. Bull. Off. Int. Epiz., 69: 1171-1178.

Shaharom-Harrison, F. and S.Z. Abdullah, 1988. Study of trichodinid ectoparasites from bighead carp *Aristichthys nobilis*, grass carp *Ctenopharyngodon idella* and lampam jawa *Puntius gonionotus* in peninsular Malaya. Trop. Biomed., 5: 131-137.

Shaharom-Harrison, F.M., I.G. Anderson, A.Z. Siti, N.A.M. Shazili, K.J. Ang and T.L. Azmi, 1990. Epizootics of Malaysian cultured freshwater pond fishes by *Piscinoodinium pillularae* (Schaperclaus, 1954) Lom, 1981. Aquaculture, 86: 127-138.

Sin, Y.M., K.H. Ling and T.J. Lam, 1994. Passive transfer of protective immunity against ichthyophthiriasis from vaccinated mother to fry in tilapias, *Oreochromis aureus*. Aquaculture, 120: 229-237.

Sniezko, S.F., 1964. The effect of environmental stress on outbreaks of infectious diseases of fishes. J. Fish. Biol., 6: 197-208.

Subasinghe, R.P and C. Sommerville, 1989. An experimental study into the possible protection of fry from *Ichthyophthirius multifiliis* (Fouquet) infections during mouth breeding of *Oreochromis mossambicus* (Peters). J. Fish Dis., 12: 143-149.

Van, J.G. and L. Basson, 1987. Host specificity of trichodinid ectoparasites of freshwater fish Parasitol Today, 3: 88-90.

Van, J.G. and L. Basson, 1989. A further contribution to the taxonomy of the trichodinidae (Ciliophora: Peritricha) and a review of the taxonomic status of some fish ectoparasitic trichodines. Sys. Parasitol., 14: 157-179.

Van, J.G., L. Basson and J. Theron, 1984. An experimental evaluation of the use of formalin to control trichodiniasis and other ectoparasitic protozoans on fry of *Cyprinus carpio* L. and *Oreochromis mossambicus* (Peters). S. Afr. J. Wild. Res., 14: 42-48.

Ventura, M.T and I. Paperna., 1985. Histopathology of *Ichthyophthirius multifiliis* infection in fishes. J. Fish Biol., 27: 185-203.

Viljoen, S. and J.G. Van, 1983. A taxonomic study of sessile peritrichians of a small impoundment with notes on their substrate preferences. J. Limnol. Soc. Sth. Afr., 9: 33-42.

Viljoen, S. and J.G. Van., 1985. Sessile peritrichs (Ciliophora: Peritricha) from freshwater fish in the Transvaal, South Africa. S. Afr. J. Zool., 20: 79-96.

Wahli, T. and W. Meier, 1985. Ichthyophthiriasis in Trout: Investigation of Natural Defence Mechanisms. In: Ellis A.E., (Ed.), Fish and Shellfish Pathology, Academic Press, London, pp: 347-353.

Woo, P.T.K., 1987. *Cryptobia* and cryptobiosis in fishes. Advan. Parasitol., 26: 199-237.