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A Review of Some Fungi Infection in African Fish *Saprolegniasis*, Dermal Mycoses; *Branchiomyces infections*, Systemic Mycoses and Dermocystidium

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Abstract: *Saprolegniasis*, *Dermal Mycoses*, *Branchiomyces infections*, Systemic Mycoses and *Dermocystidium* are some fungi infection in African fish reviewed to educate fish culturist and both private and public sector fish culturist on some fungal fish diseases likely encountered in culture fisheries management. The description, taxonomy, diagnosis, life cycle, biology, Pathology, epizootiology and control of saprolegnia and other phycomycete infections (dermal mycoses); branchiomyces infections, systemic mycoses and dermocystidium-like organisms and some fungi infection reviewed. These fungal infections can cause drastic fish mortality and need proper management in culture fisheries.

Key words: *Branchiomyces infections*, *Dermocystidium*, *Dermal mycoses*, *Saprolegniasis*, *Systemic mycoses*

INTRODUCTION

Fungi are heterotrophs and are mostly saprophytes. Parasitic fungi are facultative saprophytes as most saprophytic fungi are facultative parasites. However, this depends on the prevailing conditions. Fungal organisms vary in structure from species- to -species, genus-to-genus and family-to-family. This difference in structure is important in fungi taxonomy. For instance, yeast occurs in single cells. Hyphae fungi are many filamentous cells joined together. Dimorphic fungi occur in the two forms. Filamentous fungi can branch in all direction and entangle to form a mycelium. Each filamentous cell is separable from others by septa (septic hyphae). The “non septic hyphae” have no distinct separation form each other.

Fungi can reproduce sexually or asexually. Sexually reproduction is through, the union of two adjacent cell gamete. Asexual is by fragmentation, binary fission or budding, spore are the primary units of transmission in forms of reproduction. Four species of fungi are known to cause disease in fishes. Poor environmental condition, malnutrition and other disease can facilitate fungal epidemic.

Saprolegnia is often used as a collective name for phycomycete fungi of several genera (such as Saprolegnia, Achyla, Aphanomyces and Dictyuchus) predominantly of the order Saprolegniales (family Saprolegniaceae) (Neish and Van As, 1980; Chien, 1981). Lesions may have mixed infections. While characteristics of the sporangium formation and zoospore release are important for determination of saprolegnian genera, details of the reproductive organs, the oogonia and the antheridia are important criteria for specific differentiation. Mycelia recovered from fish contain, however, only asexual reproductive organs (oval elongated sporangia which containbiflagellated zoopores). Saprolegniaceae may be cultured on any nutritive agar medium plate with the addition of antibiotics (such as Sabouraud's agar). A specific methodology for isolation of Saprolegnia was devised by Willoughby and Pickering (1977). All these cultures yield only asexual generations. Sexual generations may be obtained only through specialized culture methods (over hemp seeds - Neish and Van As, 1980).

Branchiomyces are only known from hyphal stages in the gills. Branchiomyces is readily isolated and grown on routine agar media (with antibiotics). Its appearance in culture is similar to its appearance in the gills. Peduzzi (1973) obtained gemmae on hemp seeds and was able to show antigenic similarity and morphological affinities with Saprolegniaceae.

Ichthyophonus hoferi usually occurs in various fish from the sea, but only exceptionally in fish from fresh waters. Aspergillomycosis and *Paecilomyces marquandii* infections have been reported from cultured tilapia (*Oreochromis* spp.) Records from Africa and African fish are as follows: *Ichthyophonus* were identified from *Mugil cephalus*, Kowi lagoon (brackish water), southeastern Cape, South Africa (Paperna, 1986) and aquarium held *Hemichromis bimaculatus* (Chauvier, 1979); *Aspergillomycosis* from farmed *Sarotherodon* spp.

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in Mombasa,Kenya (Olufemi *et al.*, 1983) and *Paecilomyces* mycosis in red tilapia hybrids from Arizona, USA (Lightner *et al.*, 1988a).

Dermal and gill infections are found in various fish including carp, eels and salmonids. Visceral granulomatous infections occur in Goldfish (Landsberg and Paperna, 1992), carp (Kovac-Gayer *et al.*, 1986), *Oreochromis* hybrids (tilapia) (Paperna, 1986), and salmonids (Hedrick *et al.*, 1989). The disease is yet known in Africa. Gill *Dermocystidium* occurs in eels, and visceral granulomatous infections in goldfish and tilapia farmed in Israel. A review of some fungi infection in African fish will educate fish culturist and both private and public sector fish culturist on some fungal fish diseases likely encountered in culture fisheries management. The description, taxonomy, diagnosis, Life cycle, biology, Pathology, Epizootiology and Control of Saprolegnia and other Phycomycete infections (Dermal Mycoses); Branchiomyces infections, Systemic Mycoses and Dermocystidium-like organisms and some fungi infection reviewed for the needed education.

Saprolegnia and other phycomycete infections (dermal mycoses):

***Saprolegniasis*:** This is a fungi disease of fishes and fish eggs. Saprolognia, Achlya sp. and Idctyuchus sp. belonging to the family, Saprolegniaceae, are responsible for the disease. These organisms are commonly referred to as "water moulds." Water moulds can be found in the brackish water with salinity level of 28 parts per thousand, but occur primarily in fresh water. Microscopic examination can distinguish each other. Malnutrition, presence of toxic substances in water, damages on skin, fins or gill and stress can create room for the secondary invasion of fish tissue by water moulds. Dead fish eggs are good growth medium for the fungi organisms. Fungi mycelia mass extends from one dead fish egg to another thus suffocating and subsequently killing them. This process continues, increasing the zoospores and infestation of fish eggs.

Presence of fluff, cottony growth on fish wound or dead fish eggs and microscopic examination of the cottony growth, stained with lacto phenol cotton blue ormethylene blue, are enough for presumptive diagnosis. Isolation and identification of fungi organism is based on the microscopic structure of hyphae, sporangium zoospores and biochemical test. Removal or correction of primary cause and environmental condition, combined with the use of disinfectant are, effective control measures. The following are the disinfectants used in the control of fish and fish egg infestation:

- Malachite green at 5 mg/L for one hour
- 5% sodium chloride (common salt) at 1-2 mg/L
- Combination of 100 mg/L of formalin and 2.5 mg/L of malachite green for 1 h

Removal of infested eggs from the troughs or incubation gutter is also important in the control of the disease. Potentially in all freshwater fishes, Incubated eggs are readily infected. Piscine dermal phycomycetes are universal. Infection in Africa has been recorded from wild and cultured fish in South Africa, Uganda and Israel.

Description, taxonomy and diagnosis: Skin infection is easily detected by the appearance of patchy or extensive cottonwool cover -- the fungus mycelia, emerging usually from an haemmorhagic skin lesion. Microscopic examination reveals hyphae, giving rise to sporangia. The genus *Saprolegnia* has oblong sporangia and is also recognized by its branched, non-septate multinucleated mycelium. The released zoospores typically swim away. In the genus *Achyla* the spores encyst at the mouth of the sporangium where they form a hollow ball. Encystment at the mouth of the sporangium also occurs in Aphanomyces (Neish and Van As, 1980; Chien, 1981).

Taxonomic study of phycomycete skin fungi of African and Near East fish has never been attempted. The generic and species compositions of skin fungi in tropical waters may differ from that known in temperate and cold water regions. Sampling for saprolegniaceae in freshwaters in Thailand yielded *Achlya* and *Aphanomyces* but not *Saprolegnia* (Willoughby and Lilley, 1992).

Plate 1 shows Fungal infections: a. Saprolegnial hyphae and sporangia, skin of *Oreochromis aureus* × *niloticus*, Israel. b. *Branchiomyces* infection in carp gills, Israel (by courtesy of S. Sarig). c. *Ichthyophonus* bodies in gill tissue of *Mugil cephalus*, S. Africa. d. *Ichthyophonus* granuloma in spleen of *M. cephalus*, S. Africa. e-k. Visceral granuloma caused by *Dermocystidium*-like organisms in goldfish, Israel. e, Giemsa stained organisms in a smear and f, g, live, viewed by Nomarski microscopy. h. Transmission electron microscopic view.

Plate 2 shows a continuation of Fungal infections: i-j, histological sections of granulomata in the kidney, C, necrotic core, E, epitheloid and fibroblast envelope, p, parasites at the periphery of the lesion, arrows, single and multiple parasite bodies. I, m. Granuloma with *Dermocystidium*-like organisms in livers of *Oreochromis aurea* × *niloticus*, Israel (Fig. e, f, g, photographed by Landsberg and Paperna (1992).

Continued:

Life cycle and biology: Saprolegnia and other phycomycete fungi reproduce asexually by production of zoospores in the sporangia. Released biflagellated zoospores settle and produce new hyphae. Sexual reproduction occurs only under special circumstances, and has never been observed in parasitic forms. In the sexual generation, in the formed oogonia, 1-20 eggs develop. Antheridia developing on adjacent hyphae penetrate into

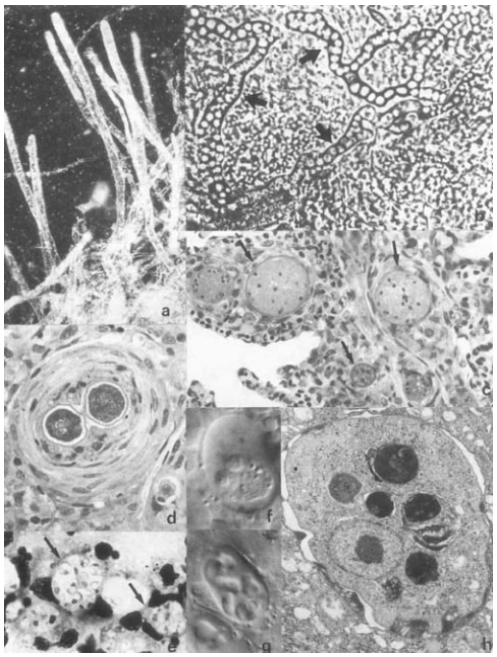

Plate 1 : Fungal infections

the oogonia and fertilise the eggs. The fertilised zygotes develop into resting spores. In some species of *Saprolegnia* antheridia are absent and eggs develop into parthenospores. The germinating spore undergoes meiotic division followed by several mitotic divisions and sends out an unbranched hypha which turns into a sporangium, which contains zoospores (Neish and Van As, 1980).

Pathology: Saprolegnian fungi are opportunistic facultative parasites. They are necrotrophs when they grow on living sources and saprotrophs when they derive their nutrition from non-living sources. *Saprolegnia* often acts as a 'wound parasite' and handling fish often predisposes them to infection. However, there is good evidence to suggest that saprolegnian fungi can act as primary invaders, in physiologically debilitated (example - decline in mucus production) and immunologically compromised fish (in "stress" situations) (Willoughby, 1978; Neish and Van As, 1980).

The fungus usually establishes itself focally, invading the *stratum spongiosum* of the dermis and then extends laterally over the epidermis, eroding it as it spreads. In severe and prolonged infection, mycelia will penetrate beneath the dermal layer into the muscles and in very small fish will reach the inner organs. Saprolegnian infection extends less commonly to the gill integument. In young fish, infection is often confined to the posterior half of the body and consequently the caudal fin is lost and the caudal peduncule vertebrae become exposed. Hyphae induce extensive necrosis in the tissues they invade. Inflammatory response also occurs around damaged

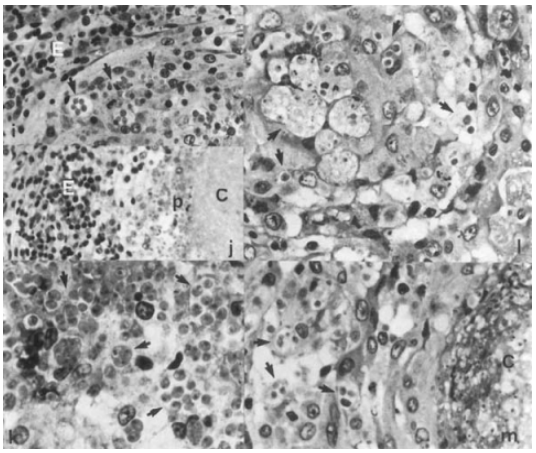

Plate 2: Fungal infections

tissues; oedema, haemorrhaging and cellular infiltration is intense, particularly where secondary bacterial contamination follows (Willoughby, 1978; Neish and Van As, 1980; Chien, 1981).

Epizootiology: Saprolegnian fungi are ubiquitous components of aquatic habitats. The circumstances by which these fungi are capable of invading fish are not fully understood. Fish succumb to infection either under circumstances which damage their skin, most commonly handling, netting and other manipulations associated with farming practices, or when predisposed by environmental stressors. Often both conditions occur together (Sarig, 1971; Roth, 1972; Neish and Van As, 1980). It has been suggested that skin wounds caused by ectoparasites, notably argulids and lernaeaids, facilitate initial invasions of the fungus (Oldewage and Van As, 1987).

Massive mortalities, due to saprolegnial dermal mycoses of pond reared tilapia and of cichlids stocked in artificial lakes (dam reservoirs), commonly occur during winter months in the non-tropical parts of Africa (in the Transvaal highveld and Cape regions of South Africa), in the Near East (Israel), and in introduced tilapia in USA where water temperatures decline below 15ºC (Sarig, 1971; Paperna, 1984; Oldewage and Van As, 1987; Lightner *et al*., 1988b). Infections and losses usually involve *Oreochromis* spp. which are more vulnerable to low water temperatures, while the more cold tolerant *Tilapia* spp. (*T. zillii, T. sparmanii*) are only exceptionally affected. Economic loss to tilapia farming is considerable, particularly in the colder winters when 50-80% of the overwintering stocks become infected and die, including market sized (300-500 g) fish. In such years, L. Kinneret *Oreochromis* cichlids also succumb to dermal mycoses.

Integumental wound aetiology is the background for saprolegniases frequently affecting warmwater farmed eels, resulting from aggressive behaviour due to

overcrowding. Additional predisposing factors are inadequate nutrition and poor water quality (Eugusa, 1965; Chien, 1981). Saprolegniasis initiated by skin abrasions is also a cause for losses among scaled farmed fish (grey mullets, silver and grass carp) following netting (Sarig, 1971). Heavy losses, due to dermal mycoses, occur in the process of acclimatisation to freshwater of grey mullet fry collected from coastal waters for stocking in freshwater ponds. Dermal mycoses do not occur in fish farmed in salinities exceeding 1 ppt. Eggs are badly damaged by *Saprolegnia* when infected during artificial incubation in cold water (salmonids) as well as in warm water (cyprinids, cichlids and clariids). Invasion is promoted by existing necrotic substances such as unfertilised and damaged eggs.

Control: Malachite green oxalate (zinc-free) treatment is still the most commonly practised remedy for dermal mycoses (Alderman and Polglase, 1984). It is applied to water in holding tanks at a dose of 0.1-0.2 ppm for 1 h, or by continuous flow, to yield a final concentration of 0.05-0.075 ppm for several days. Earth ponds stocked with carp and tilapia are treated with 0.15 ppm (Sarig, 1971) and the recommended concentration for heavily eutrophic eel ponds is 0.2-1.0 ppm (Rickards, 1978). A preventive treatment is recommended immediately after handling or netting.

Fish of various species differ in their susceptibility to Malachite green. The tolerance limits of carp, tilapia, grey mullets and trout are around 1.1 ppm/6 h. Tolerance to the drug changes with age; applied doses are toxic to young (smaller than 100 mm) American and African catfish (Hoffman, 1970).

Tolerance to treatment declines at higher temperatures and also depends on the water conditions and the physiological state of the fish. It is therefore recommended to perform toxicity tests before larger scale treatment is applied. A safer alternative to this treatment is the use of saline water (above 0.1% NaCl), for one to several days, if it is tolerated by the fish and applicable to the conditions of the farming system. Treatment of eggs: a ten seconds dip in 66 ppm (trout) to 1500 ppm Malachite green (*Ictalurus punctatus*); one hour dip in 0.1 ppm to 2.2-5.0 ppm (trout) or by maintaining such concentrations in flowing water for several days.

Branchiomyces infections:

Brachiomycosis: This fungi disease of fish is characterized by necrosis of the gill tissue (gill rot). Two fungal organisms are associated with this disease: *Brachiomyces sauguins* and *Brachiomyces demigrans*. Infection is primarily on the gill filaments, lamellar capillaries and other tissues of the gills. They both produce branched, non-septate hyphae. The damage on the gill tissue can cause respiratory distress on fish. Infected fishes are weak and lethargic. Gills appear bright red in acute condition. Parts of gill become white or brown as the disease progresses, depending on the stage of necrosis. Chronic cases cannot be easily noticed because of the slow development of symptoms.

Gills necrosis and respiratory distress can be used in tentative diagnosis. Observation of fungal hyphae and spores in a quash preparation of infected tissue, examined with a microscope can also aid diagnosis.

Careful examination of the infected sites: Gill filaments, lamellar capillaries or gills tissues can indicate the *Brachiomyces* spp. involved. This can be observed in the histopathological studies of the infected gill tissue. A permanent bath in 0.1 mg/L malachite green or 0.3 mg/L for 12 h and 15 mg/L formalin (continuous bath) can be used in the treatment of the disease. Strict sanitation, routine disinfection and prompt disposal of dead fish are important in the disease control.

The fungi are reported in various fish species, notably common carp, American catfish and eels. In Africa and the Near East, infection has been reported thus far only in farmed carp. Branchiomycosis has been reported in farmed common carp in Transvaal, South Africa, and in Israel (Sarig, 1971).

Taxonomy, description and diagnosis: Infection is confined to the gills. Infected areas become necrotic, brownish-grey. Microscopic examination will reveal branched nonseptate hyphae containing numerous spores. Two species were recognized, *B. sanguinis* the causative agent of carp branchiomycosis and *B. demigrans* causing gill infections in tench and pike. Growth of the former species is confined to the vascular system while the latter expands to extra vascular tissues (Neish and Van As, 1980).

Life history and biology: Branchiomyces in carp gills is usually localised in the blood vessels, the efferent branchial vessels and the capillaries, producing branched coenocytic hyphae capable of producing aplanospores by endogenous cleavage (Neish and Van As, 1980). In eels branchiomycosis hyphae and spores spread to visceral organs (Chien *et al*., 1978). Infection is probably by spores liberated from the necrotic tissue, but the exact route by which fish contract infection is unknown.

Pathology: Infection in the blood vessels of the gill causes blockage, haemostasis and thromboses which consequently cause extensive necrosis of the gill filaments. Areas of the gill filaments turn brown, due to haemorrhages and thromboses, and grey as a result of ischemia. The process is fast and is accompanied by proliferation of the gill epithelium with resulting adhesions of the filaments (Richards, 1978; Neish and

Van As, 1980). In eels, lesions containing hyphae and spores occur in the epicardium and the spleen (Chien *et al*., 1978).

Epizootiology: Branchiomycosis occurs in eutrophic ponds with a high load of organic matter, ponds fertilized by organic manure, and water temperatures above 20ºC. During the hot season, when ambient water temperatures are above 25ºC, infection may spread to most fish in the pond and cause heavy mortalities.

Control: Recommended treatments for infected fish are, application of 0.3 ppm Malachite green per 24 h, 1.2 ppm copper sulphate into the pond or as a quick dip (10-30 min.) at 100 ppm, or a dip in 3-5% NaCI. However, the efficacy of such treatments is not well established. As a prophylactic treatment, it has been recommended to treat earth ponds prior to stocking as a measure for water quality, with 150-200 kg/ha Calcium oxide (quick lime) or 8 to 12 kg/ha Copper sulphate for 0.5 and 1 m deep ponds respectively (Schaperclaus, 1954; Sarig, 1971).

Systemic mycoses:

Ichtyophonus: This condition is characterized by deformation of the vertebral column (scoliosis) and granulomatosus lesions on the skin of the affected fish (sand paper effect). Ichtyophonushfferi causes it. The infectious stage is a cyst found in the tissues of infected fish. Transmission is through contact with faecal materials of infected host or by consumption of infected carcass. Internally, the viscera (Kidney, liver, spleen, spinal cord, ventricles of the heart and the brain) can be swollen, with presence of white or gray necrotic foci. Damage to brain and spinal cord results in lateral or dorsoventral curvature of the vertebral column (scoliosis or lordosis).

Clinical signs, presence of cyst in squash preparation of affected organs and lesions observed at autopsy, can aid diagnosis. Isolation and identification of fungus can give definitive diagnosis. No therapeutic agent is effective. Stop using dead fish to feed healthy ones. Routine sanitation of hatchery facilities is important.

Description, taxonomy and diagnosis: Systemic mycoses can be readily recognised by the extensive granuloma they induce. The encapsulated tissue spores of *Ichthyophonus* are visible with the naked eye or at low magnification (×50). Hyphae of other fungi can be detected (often in the core of the granulomata) only microscopically. *Paecilomyces* also produces characteristic chains of oval conidia within the lesion in the tissue (Lightner *et al.*, 1988a). The taxonomic position of *Ichthyophonus* is still unknown. Spores germinate hyphae in the host tissue post-mortem and in a similar manner on any culture medium. Neither in-vitro stages (Okamoto *et al.*, 1985) nor ultrastructural studies (Paperna, 1986) provide clues to the fungal taxonomic affiliations. *Aspergillus flavus* and *A. niger* (Ascomycetes) and *Paecilomyces marquandii* (Moniliaceae) were identified from *in vitro* cultured isolates from visceral lesions (Olufemi *et al.*, 1983; Lightner *et al.*, 1988a).

Life history and biology: Data on the life history of *Ichthyophonus* suggest the existence of parasitic stages in the fish and free non-parasitic forms. Fish, however, are readily infected through feeding on infected tissues. In live fish, small mononucleate spores develop into multinucleate spores, but germinate only after fish death. On culture media various developmental forms occur: amoeboidal, plasmoidal and hyphal bodies of several patterns. Morphotypes and sequences of their occurrence vary with the culture medium (Thioglycolate or MEM) and ambient pH (Okamoto *et al.*, 1985). The other systemic fungi occur in tissues as hyphae and proliferate through asexual division (Olufemi *et al.*, 1983); *Paecilomyces* also yielded conidia (Lightner *et al.*, 1988a).

Pathology: Fungal infections of tissues invariably induce chronic inflammation, resulting in granuloma formation with characteristic epitheloid, and later encasement in fibrouscollagenous capsule. Dense infection leads to formation of large continuous necrotic lesions which, in *Paecilomyces* infections, can expand beyond the limits of the organ where it primarily developed. Affected organs, kidney and spleen, may become enlarged.

The fungal body can be identified in the cellular or the necrotic core. In *Ichthyophonus* infections, all connective tissues are eventually invaded, including that of the gill filaments. In *Paecilomyces* infection of red tilapia, cottony patches of areal hyphae also occurred on the peritoneum wall near the kidneys (Olufemi *et al.*, 1983; Paperna, 1986; Lightner *et al.*, 1988a). Heavy infection is debilitating to the fish and often results in death. *A. flavus, A. niger* and some species of *Paecilomyces* are occasional pathogens of birds and mammals.

Epizootiology: Ichthyophonus infections in migratory euryhaline fish can be carried into inland waters, where they may be further disseminated via predation and necrophagy. Instances of infection in cultured freshwater fish (trout) and aquarium held *Hemichromis bimaculatus* appear to be the result of feeding on the contaminated flesh of marine fish (Dorier and Degrange, 1961). The routes by which, cultured tilapia (*Oreochromis* spp.) contracted systemic mycosis is unknown. Fish were successfully infected when injected intraperitoneally with

material from isolates of *Aspergillus* spp., with *A. flavus* being the more pathogenic. The original route of infection was suggested to occur via feeds; examined food stuff on the farm were, however, negative (Olufemi *et al.*, 1983). *A. flavus* when infecting feeds yields Aflotoxin B1, a causative agent of hepatoma in trout (Ghittino, 1976).

Dermocystidium-like organisms: The genus name *Dermocystidium* has been applied to a variety of pathogenic organisms, of doubtful relationships, that infect aquatic animals: oyster pathogens, once regarded as a fungus, presently renamed *Perkinsus* and placed among the protozoans (Levin, 1978); *Dermocystidium* of skin and gills of fish (Olson *et al.*, 1991), with some affinities with fungi and the "*Dermocystidium*-like" organisms associated with systemic granuloma in fish (Hedrick *et al.*, 1989; Landsberg and Paperna, 1992).

Taxonomy description and diagnosis: Skin and gill infections form readily detected, white, either round (up to 1.1 mm diameter, in salmonids) or elongated cysts (up to 0.5×3 mm, in eels and carp). When mature, such cysts contain numerous 5-8 μm spores with a characteristic large vacuole (Cervinka *et al.*, 1974; Wooten and McVicar, 1982; Nitzan, 1990; Olson *et al.*, 1991). The granuloma-forming organisms from different fish seem to be related, and show some common ultrastructural features (Voelker *et al*., 1977; Kovac-Gayer *et al.*, 1986; Nash *et al*., 1989; Hedrick *et al.*, 1989) which suggest affinities with fungi. Voelker *et al.* (1977) and Lom and Dykova (1992), however, considered these organisms to be amoebae. Organisms associated with granulomata are best revealed through histological preparation, but may be detected both in wet mounts or air dried, methanol fixed, giemsa stained smears (Landsberg and Paperna, 1992).

Life history and biology: Spores released from mature cysts in salmonid gills, following incubation in water, transformed into zoospores with a single flagellum. Salmonids exposed to these zoospores were found to be infected with numerous cysts within 4 days. Eel cyst fine structure is similar to that reported by Olson *et al.* (1991) from salmonid infections.

The life history of the visceral pathogens and how fish become infected is unknown. Development in goldfish, carp and salmonids seems to be similar. In goldfishinfections,parasitesarelocated within macrophages and grow into multinuclear bodies which divide up into 10 unicellular offspring (3.5-5 μm in diameter) (Landsberg and Paperna, 1992). Parasites aggregated in the tissue on the periphery of thegranulomata.Degenerated infected macrophages predominated in late infections, while later parasites totally disappear from the periphery of the granulomatous lesions.

Pathology and epizootiology: Gill *Dermocystidium* infections are pathogenic, and cause mortalities in stocks of salmonids (Olson *et al.*, 1991), carp (Cervinka *et al.*, 1974) and eels (Wooten and McVicar, 1982; Molnar and Sovenyi, 1984). The large or numerous cysts cause pressure damage to the gills, focal necrosis and extensive epithelial proliferation which obstructs lamellar structure. Infection in farmed carp and eels may reach epizootic proportions.

Systemic infections in salmonids (Hedrick *et al.*, 1989), in goldfish (Landsberg and Paperna, 1992) and in tilapia were associated with intense granuloma. Lesions were mainly epitheloid, with a gradually increasing necrotic core. In goldfish, infection first developed in the kidneys, next in the spleen and later in other organs. Haemorrhagic dropsy often occurred and kidney and spleen became enlarged, particularly the kidney, which pressed, and sometimes also perforated the lateral abdominal body wall. In tilapia infection was located in the liver. Lesions persisted several months after the disappearance of the parasites. Infection occurred repeatedly in earth pond reared goldfish, active infection occurred in late fall and in winter. In tilapia infection was epizootic, and thus far, has been detected only once, in overwintering stock in March.

CONCLUSION

Adequate knowledge on the description, taxonomy, diagnosis, Life cycle, biology, Pathology, Epizootiology and Control of Saprolegnia and other Phycomycete infections (Dermal Mycoses); Branchiomyces infections, Systemic Mycoses and Dermocystidium-like organisms and some fungi infection inevitable in culture fisheries management and practices in Africa.

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