

Viral, bacterial and fungal diseases of Siluroidei, cultured for human consumption

Jan H. Boon and Elbertus A. Huisman

Department of Fish Culture and Fisheries, Wageningen Institute of Animal Sciences,
Wageningen Agricultural University, P.O. Box 338, 6700 A.H. Wageningen, The Netherlands.

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Abstract

Based on an extensive literature search, the present overview discusses the economic relevant diseases of Siluroidei, cultured for human consumption. One herpes virus and two irido viruses play a significant role for channel catfish (*Ictalurus punctatus*), sheatfish (*Silurus glanis*) and yellow bullhead (*I. melas*) culture, respectively. There is only one fish species-specific bacterial disease which has an economic impact in the catfish industry: *Edwardsiella ictaluri*. Secondary infections with opportunistic bacteria and fungi are of interest in all catfish species cultured both under intensive and extensive circumstances. Etiology, pathology, prevention and treatment are shortly discussed for all diseases. Also some diseases of unknown etiology, which occur in African (*Clarias gariepinus*) and Asian (*C. batrachus*) catfish, are described.

Keywords: Siluroidei, diseases, viruses, bacteria, fungi, treatment, prevention.

Les maladies virales, bactériennes et fongiques des poissons-chats d'élevage.

Résumé

Cette synthèse, établie d'après une importante recherche bibliographique, fait le point sur les maladies des Siluroidei, cultivés pour la consommation humaine, ayant des conséquences économiques notables. Un virus de l'herpès et deux irido-virus jouent un rôle significatif dans les élevages de poisson-chat américain (*Ictalurus punctatus*), de poisson-chat européen (*Silurus glanis*) et d'*Ictalurus melas*.

Il n'y a qu'une maladie bactérienne spécifique qui a un impact économique dans l'industrie du poisson-chat: *Edwardsiella ictaluri*. Des infections secondaires dues à des bactéries et des champignons sont présentes chez toutes les espèces cultivées en élevage de type intensif ou non.

L'étiologie, la pathologie, la prévention et le traitement sont brièvement discutés pour chacune des maladies. Quelques maladies dont l'étiologie est inconnue, observées chez le poisson-chat africain (*Clarias gariepinus*) et chez le poisson-chat asiatique (*C. batrachus*) sont également décrites.

Mots-clés : Poissons-chats, maladies, virus, bactérie, champignon, traitement, prévention.

INTRODUCTION

Fish cultured for consumption often suffer diseases, both from infectious and non-infectious origins (Durborow *et al.*, 1991). Most attention is paid to infectious pathology, but it is questionable if this is justified because many diseases have a pathophysiological background related to environmental

stressors (Snieszko, 1974; MacMillan and Tucker, 1985; Hanson and Grizzle, 1985; Plumb and Olah, 1984; Fedoruk, 1981). Examples of such stressors might be (1) husbandry factors like crowding (Ventura and Grizzle, 1987), capture and hauling, (2) water-related problems like toxicants (Faisal *et al.*, 1988; Tucker *et al.*, 1984), temperature and oxygen extremes (Plumb and Olah, 1984; Walters and Plumb, 1980),

and rapid environmental changes (Ciembor *et al.*, 1995), (3) social interactions (Wise *et al.*, 1993), and (4) transport (Blazer, 1992). Stress results in a higher susceptibility for diseases, especially infectious ones (Ellis, 1981). The prevalence of such diseases depends on the interaction between fish, pathogen and environment (Snieszko, 1974).

In infectious fish diseases carrier state animals can play an important role. Carrier state fish are asymptomatic animals which harbour the pathogens without any clinical symptoms. They start spreading the pathogen mostly under unpredictable circumstances, which can result in a sudden onset of a disease in an obviously healthy population. Carrier state fish are difficult to detect because usually they are present in a low number in a population, thereby hiding the pathogen and hampering its isolation. Also in diseases of Siluriformes carrier state fish can play a significant role (Klesius, 1992*b*; Mqolomba and Plumb, 1992).

There are fish diseases of which the etiology is not exactly known (Boon *et al.*, 1987*b*). Micro-organisms might be involved in these respective disease processes. However, nearly always, opportunistic micro-organisms are found in these (chronically) diseased fish. In some cases, it is not (yet) possible to experimentally induce the pathology of such diseases, and consequently the relationship between disease and micro-organism(s) have still to be proven.

From all viruses, known to be infectious for finfish, only a few are related to diseases of cultured Siluriformes: Ictalurid herpes virus (Wolf, 1988), Sheatfish iridovirus (Ahne *et al.*, 1989) and Catfish iridovirus. Two other viral infections diagnosed in Siluriformes, Catfish reovirus (Hedrick *et al.*, 1984) and Sheatfish herpes virus (Békési *et al.*, 1981) proved so far not pathogenic for these fish species (table 1).

Only one bacterial pathogen has a specific pathogenicity for one of the Siluriformes: *Edwardsiella ictaluri* (Blazer *et al.*, 1985). However, Morand (unpublished results) isolated *E. tarda* from European catfish suffering a septicaemia with haemorrhagic barbels and fins.

Other pathogenic bacteria in Siluriformes are opportunistic bacteria: *Flexibacter columnaris*, *Aeromonas hydrophila* and *Pseudomonas* sp. Sometimes a relatively rare bacterial species is isolated for instance *Pasteurella piscicida* from *Silurus glanis* (Farkas and Olah, 1984). The latter bacteria does not seem of significant economical impact for the catfish culture.

As usual in fish culture, fungal infections in catfish species are nearly all opportunistic infections after primary lesions.

Treatment of infectious diseases

Although the best treatment of diseases is an effective prevention, treatment by drugs is regularly needed. Such treatment has to be supported by environmental optimization. If the use of drugs is not effective, like in viral diseases, other methods have been used *e.g.* isolation and/or eradication of infected populations. Large enough fish can be slaughtered for consumption if no hazards for consumers exist. For a proper treatment of bacterial fish diseases drugs must be available. Unfortunately, knowledge about correct use of antimicrobial drugs in fish culture is scarce. Research to support the registration of promising therapeutic agents is urgently needed (Meyer, 1991). Nearly no drug dose levels used in fish culture practice are based on scientific research. However, in the USA and The Netherlands pharmacokinetic studies of different drugs have been done for channel- (*Ictalurus punctatus*) and African catfish (*Clarias gariepinus*) respectively (Stehly and Plakas, 1993; Van der Heijden *et al.*, 1994). Beside pharmacokinetic characteristics, results of drug sensitivity tests of the respective bacterial strains are needed for a proper treatment of bacterial infections. Large differences exist between countries concerning the legal possibilities of drug use. For instance in Canada, only one antibiotic (Oxytetracycline) is registered and approved for use in fish medicine, while in the Federal Republic of Germany only Trimethoprim/Sulphadiazine is

Table 1. – Pathogenic viral infections in Siluriformes.

Infection	Natural Host	Geogr. Distr.	Source tissue	Disease	Reference
CCV	<i>Ictalurus punctatus</i>	USA, Centr. Am.	Fry/juveniles	Yes	Hedrick <i>et al.</i> , 1984
EHNV	<i>Silurus glanis</i>	Europe	Systemic	Yes	Ahne <i>et al.</i> , 1989
	<i>Ictalurus melas</i>	Europe	Systemic	Yes	Pozet <i>et al.</i> , 1992
CRV	<i>Ictalurus punctatus</i>	Centr. USA	Systemic	No	Fijan <i>et al.</i> , 1970
SHV	<i>Silurus glanis</i>	Europe	Integument	No	Békési <i>et al.</i> , 1981

CCV = Channel Catfish Virus

EHNV = Epizootic Haematopoietic Necrosis Virus

CRV = Catfish Reo Virus

SHV = Sheatfish Herpes Virus

(According to Hedrick and Hedrick, 1993)

fully licensed (Bernoth, 1992). In contrast, in The Netherlands no drug is approved for use in fish medicine. Therefore, in the latter country drug use in fish disease treatment is always off-label, with all the risks for the veterinarian who prescribed the drug. Because of unscientific and illegal use of chemotherapeutants drug-resistant bacteria have been developed in many parts of the world. In this context Depaola (1995) reported tetracycline resistance by bacteria in response to oxytetracycline-contaminated catfish feed. To achieve really effective treatments more attention has to be paid to (1) proper diagnosis, (2) "preventive treatment", (3) treatment period, and (4) dosage rates for effective disease control (Wellborn and Schwedler, 1980).

Prevention of infectious diseases

Prevention of outbreaks of infectious diseases have to be supported by avoiding unnecessary stress. When this is not possible, stress will induce infectious disease outbreaks if the pathogen is present in the fish population. Mostly these diseases are treated with antimicrobial drugs. Because treatment of infections with drugs may lead to (1) immunosuppression (Van der Heijden *et al.*, 1992) and (2) development of bacterial resistance, other techniques to attack fish diseases have to be applied. The latter can be supported by using high feed quality. Quantity and quality of food and feed are determining factors for fish health (Fracalossi and Lovell, 1994) as measured by factors indicating specific (Henken *et al.*, 1987) and a-specific resistance (Schippers *et al.*, 1994). For channel catfish it has been proven that fish health is influenced by the diet. Vitamin C and dietary lipid can manipulate disease resistance (Duncan and Lovell, 1994; Sheldon and Blazer, 1991). Immunomodulators, such as glucan, can be helpful too in potentiating the immune system (Chen and Ainsworth, 1992).

Fish can be immunized using dead or alive micro-organisms, or different kinds of treated antigens from the micro-organisms. For administration of vaccines different methods are used (1) injection, (2) orally by feed, (3) immersion and (4) spray (Newman, 1993). The method used depends on fish age and fish value, as well as on the fish farmers technical knowledge. However, it must be emphasized that health management problems should not be masked by using indiscriminately vaccination strategies. Such problems must be solved first, whereafter vaccination might not be needed any more.

Moreover it is stressed that the key for disease control in tropical countries, where drug and vaccine use is generally limited and impractical, lies in the prevention of stress conditions through adequate farm management practices (Fedoruk, 1981).

INFECTIOUS CATFISH DISEASES

Viral diseases

Three pathogenic viruses for Siluriformes species have been isolated so far: one herpes virus and two irido viruses.

The herpes virus is pathogenic for *I. punctatus* (Wolf, 1988). The two irido viruses are related to the amphibian virus frog virus-3 and the Epizootic Haematopoietic Necrosis Virus (EHNV) of redfin perch and rainbow trout, and are pathogenic for *S. glanis* and *I. melas*, respectively (Hetrick and Hedrick, 1993).

Channel catfish virus disease

- *Virus* - Channel catfish virus disease (CCVD) is caused by an acute infection of young-of-the year *I. punctatus* by the Ictalurid Herpesvirus 1 (CCV), one out of a group of at least 17 distinct viral agents isolated from or observed in fish (Hetrick and Hedrick, 1993). The enveloped virus particles are between 175-200 nm, ether sensitive, loose infectivity in glycerol, do not haemagglutinate and require the envelope for infectivity (Wolf, 1988). The virus survived for only two days at 25°C in pond water while at 40°C the virus can survive one month under the same conditions (Plumb, 1988). Cell cultures mostly used for CCV are the ictalurid lines BB and CCO. All cultures show syncytium development, followed by pyknosis and lysis (Wolf, 1988). The optimal temperature is between 25-30°C.

- *Clinical symptoms* - The onset of the disease is sudden and characterized by haemorrhages. Often the disease results in a high mortality (Hetrick and Hedrick, 1993). Clinical signs are not specific. Beside decreased appetite, mortality, changed behaviour (head-high and/or hanging posture and convulsive swimming in spirals) and haemorrhages, a swollen abdomen and exophthalmia were observed in clinical CCV infections (Wolf, 1988).

- *Pathology* - At post mortem examination an intraperitoneal yellowish/reddish fluid is found. The visceral mass seems to be anaemic and the intestinal tract is only filled with yellowish mucoid material (Wolf, 1988). Histologically, CCVD is characterized by severe haemorrhagics with generally edema and marked necrotic changes in kidneys, liver, gastro-intestinal tract, spleen, musculature, neural and pancreatic tissue (Plumb *et al.*, 1974). Occasionally, intracytoplasmatic eosinophilic inclusions will be observed in the liver cells (Major *et al.*, 1975).

- *Diagnosis* - CCVD diagnosis can be confirmed by viral isolation and identification and/or serologically. Isolation is easy from victims or fingerlings taken during a CCVD outbreak or at the most a few days afterwards. A positive culture is characterized

by syncytia. Identification is obtained by a serum neutralization test or a fluorescent antibody technique.

- **Immunity** - Passive immunity have been observed by Hedrick and McDowell (1987). However, there is a large variability which is influenced by temperature changes (Hedrick *et al.*, 1987).

Plumb and Chappell (1978) and Plumb *et al.* (1985) showed that blue catfish (*I. furcatus*), yellow bullhead (*I. natalis*), brown bullhead (*I. nebulosus*), and the European catfish (*S. glanis*), respectively, are more or less resistant to CCV infection, depending the route of infection. Boon *et al.* (1988) proved that the African catfish (*C. gariepinus*) and the Asian catfish (*C. batrachus*) are resistant too to CCV. Therefore, CCV shows a high degree of host specificity, similar to mammalian herpes viruses (Leong and Fryer, 1993).

Carrier state animals can be a problem because of latent infections in survivors. Leucocytes may be a reservoir in those fish (Chinchar *et al.*, 1993).

- **Treatment** - Since CCVD is a viral disease no treatment is available. Only in case of secondary infections by opportunistic bacteria, these can be treated by antimicrobial drugs.

Most extreme treatment is total destruction of the infected and diseased population. Thereafter, precautions have to be taken with respect to obtain a CCV free population, for instance selection of CCV free channel catfish farms for the providing of CCV free fry.

- **Prevention** - The most effective prevention is avoiding the infection by buying CCV free fry or fingerlings from channel catfish farms with CCV-free broodstock. Therefore, certification of such farms can be useful in the prevention of CCVD. Furthermore, use of more resistant hybrids is possible in enzootic areas (Plumb and Chappell, 1978).

Vaccination seems to become of great importance (Plumb, 1988) if the problems with carrier state fish and related certification difficulties (Leong and Fryer, 1993) have been solved.

Catfish Iridovirus disease

- **Virus** - Catfish iridovirus has been isolated for the first time from young yellow bullhead (*I. melas*) undergoing an acute haemorrhagic syndrome. Virions of 150-160 nm have only be found in cytoplasm and has a shape identical to EHN. They are susceptible to chloroform and 5-iodo-2-desoxyuridine (Pozet *et al.*, 1992), the latter indicating that the virus contain lipids in the envelope and a DNA genome.

There is a similarity with other iridoviruses isolated from sheatfish and redfin perch. This suggests that the host range is greater than the species from which the virus is isolated (Hedrick *et al.*, 1992).

- **Clinical symptoms** - The disease is characterized by a high mortality (up to 100%) and haemorrhages in the pectoral and pelvic girdles and in the fins (Pozet *et al.*, 1992).

- **Pathology** - Post mortem examination reveals haemorrhages in the viscera and in the abdominal cavity. Histopathologically, degenerative changes like acute necrosis of the haematopoietic tissues (kidney and spleen) and edema of the liver can be observed (Pozet *et al.*, 1992).

- **Diagnosis** - The virus can be detected by culture on EPC monolayers.

- **Immunity, treatment and prevention** - Concerning immunity, treatment and prevention no reports have been published so far. General rules with respect to viral fish diseases might be taken into account.

Sheatfish Iridovirus disease

- **Virus** - The virus is extremely pathogenic for sheatfish (*S. glanis*) fry (Ahne *et al.*, 1990). The shape has an icosahedral symmetry with a diameter from 125-135 nm and exists only in cytoplasm. The virus is sensitive to chloroform and is inhibited by 5-iodo-2-desoxyuridine (Ahne *et al.*, 1989).

- **Clinical symptoms** - Sheatfish virus disease is a systemic one with a high mortality (up to 100 %) and evokes spiralic swimming and petechial haemorrhages in the skin and, occasionally, in the eyes and barbels (Ogawa *et al.*, 1990).

- **Pathology** - Post mortem examination reveals necrosis of the haematopoietic tissues (kidney and spleen) without a marked host inflammatory response. This is also the case for EHN in redfin perch (Langdon and Humphrey, 1987).

- **Diagnosis** - The virus can be isolated from sheatfish using monolayers of several fish cell lines (BF-2, RTG-2 and FHM) at temperatures between 20-30°C. Cytopathogenic effect is characterized by cell rounding, presence of cytoplasmic inclusions and eventual lysis.

- **Immunity, Treatment and Prevention** - See catfish iridovirus disease.

BACTERIAL DISEASES

Fish-species-specific: Diseases of channel catfish

Edwardsiella ictaluri disease

- **Bacteria** - *E. ictaluri* is a gram-negative rod that survives in catfish and pond bottom mud (Newton *et al.*, 1989). Infection with the micro-organism can cause an acute or chronic disease. The bacteria measuring $0.75 \times 2.5 \mu\text{m}$ at 26°C or 5 to $7 \mu\text{m}$ at 37°C (Plumb and Vinitnantharat, 1989) is weakly motile with peritrichous flagella and cytochrome oxidase negative. Optimum growth temperature is between 25 and 30°C. On Brain-Heart Infusion (BHI) agar, colonies will be 2 mm in diameter and smooth, circular and slightly convex (Hawke, 1979). There seems to be a high homogeneity between the different strains according to the study of Newton and Triche (1993) who characterized the lipopolysaccharides of the outer

membrane of 40 *E. ictaluri* strains isolated in the USA.

Although *E. ictaluri* seemed to be Ictalurid specific, other non-ictalurid fish species like European catfish, *S. glanis* (Plumb and Hilge, 1986), walking catfish, *C. batrachus* (Kasornchandra *et al.*, 1986), and rainbow trout, *Oncorhynchus mykiss* (Baxa and Hedrick, 1989) can be infected too.

• *Clinical symptoms* - *E. ictaluri* disease of channel catfish manifests itself in two clinical entities *e.g.* (1) an acute gastro-intestinal septicemia followed by mortality, and (2) a chronic disease characterized by a "hole-in-the-head" lesion which may proceed to septicemia too (Shotts and Blazer, 1986). The mortality is temperature dependent; 25°C seems to be the optimal temperature for mortality of channel catfish after exposure to *E. ictaluri* (Baxa *et al.*, 1992). Diseased fish are listless, hanging at the surface tail down, horizontally spiralling, rapid spinning or display other erratic swimming behaviour (Blazer *et al.*, 1985; Hawke, 1979). On the skin, white spots and circular slightly-raised epithelial lesions can be observed over the entire body. Petechial haemorrhages are found around the mouth, at the fin base and on the ventral and lateral body sides, as well a swollen abdomen, exophthalmia, and pale swollen gills. In chronic diseased fish an open lesion is found caudomedially on the frontal bones of the skull (Blazer *et al.*, 1985; Hawke, 1979).

• *Pathology* - Post mortem examination reveals symptoms related to an acute septicemia such as (1) blood tinged or yellow ascitic fluid, (2) petechial haemorrhages in liver, serosa, mesenteries, intestine, and adipose tissue, (3) macropapular white or red spots in the liver, (4) dark red and edematous spleen and hypertrophied kidney (Hawke, 1979), and (5) granulomatous inflammation of the olfactory bulb and the telencephalon.

Furthermore, *E. ictaluri* produces chondroitinase which may play a role in the pathophysiology of the chronic disease inducing cartilage degradation (Waltman *et al.*, 1986).

• *Diagnosis* - Beside routine culture procedures on BHI agar, an ELISA for detecting serum antibodies was developed by Klesius and co-workers (1991) which is a reliable tool for diagnosing and monitoring of *E. ictaluri* infections.

• *Immunity* - Despite the general belief among animal production scientists that there is no relationship between specific plasma antibody levels and protection against the respective specific infections it can be calculated from the data given by Klesius (1992a) that in channel catfish a significant relationship between antibody level against *E. ictaluri* and protection against the related disease exists ($r=0.70$, $p<0.01$). This was also observed by Vinitnantharat and Plumb (1993) and it agrees with Klesius's statement that acquired immunity is important in resistance against *E. ictaluri* infection of channel catfish, thereby

making this species an excellent candidate for vaccine development. But, although vaccines against *E. ictaluri* have been developed, none of them are used on a large scale because of the difficulty in finding the adequate opportune time in the life cycle of the channel catfish for immunization. Chen and Ainsworth (1992) found that administration of glucan greatly reduced *I. punctatus* mortality from experimental infection with *E. ictaluri*. These results indicate that glucan potentially could be utilized prophylactically as an immunomodulator in channel catfish.

Although the problem of carrier state fish needs more research (Thune *et al.*, 1993), there are indications that carrier state fish can play a role in the epizootiology of the disease (Mqolomba and Plumb, 1992; Thune and Johnson, 1992). This carrier state can be induced by use of an antimicrobial drug to prevent enteric septicemia of channel catfish (Klesius, 1992b).

• *Treatment* - Different antimicrobial drugs which are generally used in animal husbandry can be used for treatment of Edwardsiellosis of channel catfish. Mostly, medication is given by medicated feed. Sarafloxacin proved to be effective against Edwardsiellosis. Plumb and Vinitnantharat (1990) observed a significant reduction of channel catfish mortality by *E. ictaluri* of $\pm 75\%$ using 10-14 mg of this drug/kg fish body weight/day for 5 days. Also a potentiated sulfonamide (Romet-30 super (TM)) seemed to be effective against *E. ictaluri*. In four field trials Plumb *et al.* (1987) observed a mortality reduction of $\pm 60\%$ using 50 mg Romet/kg fish body weight/day during five days. Beside an increased survival rate, an improved weight gain was observed also after treatment of Edwardsiellosis with Sarafin- and Romet-medicated feeds (Johnson *et al.*, 1992; Johnson *et al.*, 1993).

Special attention has to be given to sensitivity tests of the *E. ictaluri* strains isolated from disease outbreaks, since shifts in sensitivity for drugs occur. This provides the opportunity to change the drug of first choice, if necessary.

• *Prevention* - Most important preventive measure is to avoid contamination of a fish farm by strict control of fish to be stocked for clinical and subclinical outbreaks and for the presence of carrier state animals. Therefore, a screening program has to be developed using techniques which make it possible to screen large numbers of fish in a short period.

Optimal fish condition is important too. Durve and Lovell (1982) and Li and Lovell (1985) observed a relationship between dietary vitamin C content and resistance against *E. ictaluri* infections. In future immunomodulators which potentiate the immune defence mechanism can be used prophylactically on a large scale to increase fish resistance.

Diseases of *Silurus glanis*

Red Head Disease

• **Bacteria** - The causative bacteria are gram-negative, oxidase-positive, curved and motile rods and identified as *Vibrio* sp. Blood agar cultured colonies are round convex, surrounded by a zone of total haemolysis. The *Vibrio* sp. related to diseases in sheatfish can also grow in freshwater outside the host (Farkas and Malik, 1986).

• **Clinical symptoms** - Farkas and Malik (1986) isolated a *Vibrio* sp. from *S. glanis* fry during an epizootic. The disease was characterized by red spots in the head region. Therefore they named this disease "Red Head" disease.

• **Pathology** - Histological examination reveals an infiltration of brain tissue by erythrocytes. The brain is edemic and exudate with bacteria is formed. In acute cases the meninges and epidermis ruptures with fatal results (Farkas and Malik, 1986). This pathology differs from other infections with *Vibrio* sp. in other fish species which more likely a septicemia induce (Thune *et al.*, 1993).

• **Diagnosis** - The diagnosis has to be made by clinical inspection focused on the head region and isolation and identification of the bacterial agent.

• **Treatment** - *Vibrio* sp. involved in the disease can be treated by antimicrobial drugs. Different antibiotics are suitable (Farkas and Malik, 1986).

• **Prevention** - Measures to prevent Red Head Disease are related to buying *Vibrio* free fry and avoiding contact with infected populations. Specific measures remain to study.

Diseases caused by opportunistic Bacteria

Motile Aeromonad Septicaemia

• **Bacteria** - Motile aeromonads are free living motile mesophilic bacteria, which are considered primary and secondary pathogens of aquatic organisms (Leblanc *et al.*, 1981). Diseases caused by motile aeromonads have been described in many fish species (Newman, 1982). Motile aeromonads can be divided into three different species, *e.g.* *A. hydrophila*, *A. sobria* and *A. caviae* (Popoff, 1984). The motile aeromonads are pathogenic for mammals (including man) and birds too (Davis II *et al.*, 1978; Shane and Gifford, 1985; Wohlgenut *et al.*, 1972). *A. hydrophila* finds access to the internal organs through the digestive tract or through the injured skin under crowded conditions and high temperatures (Ventura and Grizzle, 1987).

Motile aeromonads are gram-negative, non-acid-fast, rod-shaped, and motile by generally monotrichous polar flagella. The cells are usually not capsulated. They are aerobic and facultative anaerobic (Post, 1987) and can be isolated from all types of freshwater and brackish water habitats (Thune *et al.*, 1993). The pathological effects of *A. hydrophila* are mainly related

to extra cellular products (Thune *et al.*, 1993) of the S-layer of the bacteria (Ford and Thune, 1991) which are responsible for the cytolytic and fibrolytic capacities of *A. hydrophila*, destroying host defence systems and contributing to the invasive power of the bacteria (Bach *et al.*, 1978).

Supriyadi and MacLean (1986) reported that *Aeromonas* sp., especially *A. hydrophila*, cause secondary infections of *Clarias* sp. kept under intensive conditions. There are indications that *C. batrachus* is more susceptible to infections with *A. hydrophila* than common carp (*Cyprinus carpio*) and giant gourami (*Osphronemus gourami*).

• **Clinical Symptoms** - Clinical signs depend on the form of the disease but are a-specific in all cases. The acute form is characterized by exophthalmia and a distended abdomen. The chronic form shows more deep dermal ulcers with haemorrhages and inflammation (Post, 1987; Thune *et al.*, 1993).

• **Pathology** - At post mortem the acute form is characterized by edema, haemorrhages and diffuse necrosis (Thune *et al.*, 1993). The name Motile Aeromonad Septicemia (MAS) is originated from this septicemic picture. The chronic form is characterized by local necrosis in the epidermis and musculature and by petechia on the serosa and in the muscles (Thune *et al.*, 1993). Fish with MAS have a severe branchitis as indicated by leucocytic infiltration and dilation of the gill's central venous sinus. In addition the nuclei in the branchial epithelium are enlarged (Grizzle and Kiryu, 1993).

• **Diagnosis** - For a proper diagnosis different tests are available. The bacteria can easily be cultured and identified but more sophisticated diagnostic techniques are also possible. From these various tests the slide agglutination procedure proved effective under field conditions. However, the fluorescent antibody technique is more sensitive than other procedures and can be performed in less time (Eurell *et al.*, 1978).

• **Immunity** - Both cellular and humeral immune responses after primary infection have been described for rainbow trout and carp (Post, 1966 and Baba *et al.*, 1988), suggesting the same for the Siluriformes. Despite the availability of vaccines against MAS vaccine use is not common because of the small geographical area of efficacy due to occurrence of specific strains in small areas (Schachte in Newman, 1993).

• **Treatment** - In clinical cases antibiotic treatments are indicated. Because of changing sensitivity patterns of the aeromonads this should be done according to results of sensitivity tests of the isolated aeromonad strains.

Some antimicrobial drugs used in human medicine have been used for treatment of bacterial septicemia in *C. batrachus*. Saha *et al.* (1990) fed catfish supplementary feed mixed with Trimethoprim and Sulphamethoxazole (Bactrim) for 10 days. They found

the drug effective, both as treatment and as prevention of the disease.

- *Prevention* - The best prevention is to avoid stressful periods. Stress factors advance clinical bacterial infection as proven by Faisal *et al.* (1988). They found in *C. gariiepinus* stressed by a molluscicide (Bayluscide) a depletion of haematopoietic tissue, an increased susceptibility to pathogens, and an appearance of granulae with bacteria.

Cytophaga (Flexibacter) *columnaris* disease

- *Bacteria* - *Cytophaga columnaris* is a gram-negative, rod- or filament shaped ("pine tree needles") bacteria, characterized by gliding motility. It is proteolytic, ferments sugars and is strict aerobe (Post, 1987). The bacteria causes explosive diseases of freshwater fish kept above 18°C. Often the infection becomes clinically when the fish is simultaneously infected with other micro-organisms (Marks *et al.*, 1980).

- *Clinical symptoms* - Infections with *Cytophaga* sp. are mostly secondary infections. Fish suffering environmental stress and /or injuries, are highly susceptible. Especially young stages (eggs, larvae and fry) are attacked, invaded and destroyed by the bacteria.

The most dominant clinical aspect of an infection with *Cytophaga columnaris* is local thickening of the mucus on the skin, followed within a short time by a cotton wool-like layer, covering the primary skin lesions. A-specific symptoms like exophthalmia, haemorrhages and hydrops of the abdomen can often be observed. Clinical signs depend on strain pathogenicity.

- *Pathology* - No specific post mortem characteristics are known. Sometimes pale internal organs are observed, possible related to toxins produced by the bacteria.

- *Diagnosis* - Diagnosis is made by combining the clinical aspects, especially the cotton-like layer, and analyzing results of the smears of the suspect skin spots.

- *Immunity* - Immunity is not a significant factor in the prevention and treatment of the disease. Mostly, affected fish, especially young ones, will die and recovering of the disease is time consuming. Therefore, fattening of a recovered (and diminished) population often prove unattractive.

- *Treatment* - Different antimicrobial drugs can be used for treatment. Sensitivity tests have to be done to select a suitable drug. However, without changing the environment, antimicrobial treatment is a waste of money.

- *Prevention* - The best prevention is proper husbandry. In cases where stress cannot be avoided and the risk of an infection is high the use of antimicrobial drugs as a preventive measure is advised.

FUNGAL DISEASE

Saprolegnia sp.

- *Fungus* - The family of *Saprolegniaceae* ubiquitous in the world, primarily in freshwater. The fungi have long, branched non-septate hyphae and reproduce primarily asexually forming zoospores. The optimum growth temperature is between 15 and 30°C (Post, 1987).

- *Clinical symptoms* - Fungus diseased fish show fluffy cotton-like, white grey to grey brown structures on the skin, fins, gills and eyes. Affected eggs show the same (Post, 1987).

In *Clariidae* fungal infections often occur. During a survey in a Nigerian freshwater fish pond 24 fungal species belonging to 6 fungal genera were isolated of which the major part were *Saprolegnia* sp. *C. gariiepinus* had the highest amount of isolates. There were similarities between the isolates from the pond fish and those isolated from the hatchery from where the fish were obtained (Ogbonna and Alabi, 1991). Fungi are mostly secondary invaders. Firstly, some tissues with a decreased resistance against micro-organisms were infested. For instance, after Gas Bubble Disease with degeneration/necrosis of the peripheral fin tissue caused by air-embolism of small blood vessels and/or capillaries, clinical *Saprolegnia* infections appear very soon (Boon *et al.*, 1987a).

- *Pathology* - Xu Dhai and Rogers (1991) observed that epidermic cells in the lesions related to fungal infections were necrotic. After penetration of the dermis fibroblasts and collagen lamellae were damaged.

In channel catfish, Bly *et al.* (1992) observed a complete lack of bacteria and leucocytic infiltration around the site of fungal penetration during the winter period ("winterkill syndrome"). Therefore, they suggested that winterkill syndrome should be regarded as an immunodeficiency disease of fungal origin.

- *Diagnosis* - Clinical diagnosis is confirmed by examination of direct smears of affected spots. At low magnification characteristic fungal hyphae can be observed.

- *Treatment* - Saprolegniosis can be treated by using chemicals by bath, like malachite green (5 mg/L for one hour), or by dipping, like sodium chloride (5% for one or two minutes). However, in many countries it is forbidden to use malachite green for treatment of food fish diseases due to teratogenic properties of the drug (Post, 1987). Recently a negative effect of malachite green on the haematocrit of rainbow trout (*O. mykiss*) related to haemolysis within 12 hours post treatment was described (Tanck *et al.*, 1995). A same effect in other fish species is most likely.

- *Prevention* - Saprolegniosis is seldom a primary disease. Therefore, the best prevention is good husbandry practice avoiding mechanical damage and all other kinds of stress.

DISEASES OF UNKNOWN ETIOLOGY

Broken Head Disease

• *Clinical Symptoms* - In *C. gariepinus* skeletal deformities can sometimes be observed. Especially young growing fry, fed a vitamin C deficient diet, can develop impaired growth and spinal deformities, including lordosis and scoliosis (Boon, unpublished results; Mgbenka and Eya, 1991). These clinical signs are comparable to those related to vitamin C deficiency observed in other fish species (Andrews and Murai, 1975; Ashley *et al.*, 1975) and very clearly and easily to observe. However, in Central Africa and The Netherlands sometimes mature *C. gariepinus* suddenly stop feed intake, become lethargic and die with swollen weak tissue on both head sides. The disease is characterized by a passive movement of the head of approximately 90 degrees after a push on the nose. Therefore, the farmers give this disease the name of "Broken Head Disease" (Boon, unpublished results).

• *Etiology* - Some fish farmers suggested that microorganisms were involved in the disease, but results of bacteriological investigations and treatment with antimicrobial drugs were not consistent. So, whether there is a causative infectious agent remains unclear so far. In cases where treatment of the disease with antimicrobial drugs seems effective, this could be caused by the treatment being effective to the secondary bacterial infections. Vitamin C deficiency as an etiological factor of this disease can not be excluded too.

• *Pathology* - X-ray investigation of affected specimen revealed a fissura, localized between the bones of neuro- and splanchnocranium nearby the sutura between the two main parts of the bony head. This can be responsible for the abnormal passive movement.

• *Diagnosis* - Diagnosis are made by moving the splanchnocranium independently from the rest of the head and is confirmed by X-ray of the head region.

• *Treatment* - Because the unknown etiology a specific therapy is not available.

• *Prevention* - Adequate and proper husbandry techniques (including diets) seems to be relevant in prevention of the Broken Head Disease.

Ruptured Intestine Syndrome

• *Clinical Symptoms* - In fast growing young *C. gariepinus* a syndrome clinically characterized by (1) lethargic behaviour, (2) swollen abdomen, (3) discoloured abdominal skin, and (4) reddish anal area occurs. Morbidity and mortality range between 10-70% (Boon *et al.*, 1987b). Fish in the final stage of the disease show a rupture of the abdominal wall.

• *Etiology* - The disease is diagnosed in The Netherlands in intensive cultured *C. gariepinus* (Boon *et al.*, 1987b). Similar observations, corresponding to equivalent clinical symptoms as in *C. gariepinus*, were

made for both *Heterobranchius longifilis* and *S. glanis* (Legendre and Proteau, respectively, unpublished results). The prevalence of the disease strongly depends on the feeding level during the weeks 2-5 post hatching (Schippers *et al.*, 1992; Hariati *et al.*, 1994) indicating that feed quantity and/or quality influenced the prevalence of the disease. An infectious etiological agent is not found (yet), but participation of microorganisms in the etiology of the disease cannot be excluded.

• *Pathology* - Post mortem examination reveals (1) a smelling haemorrhagic fluid present in the abdomen, and (2) a rupture in the mid-part of the gut. At the final stage of the disease the abdominal wall is partly vanished as a result of the intra-abdominal inflammation.

• *Diagnosis* - Clinical symptoms during weeks 5 to 10 weeks post-hatching are pathognomonic for the disease.

• *Treatment* - Because the disease outbreak becomes apparently only some time after the etiological moment, a therapy at the onset of the clinical signs is too late.

• *Prevention* - Occurrence of the disease seems feeding dependent. A maximum feeding level corresponding with optimal growth up to week 4 post hatching seems to prevent the disease (Hariati *et al.*, 1994).

Ulcerative disease

• *Clinical Symptoms* - In *Clarias* sp. cultured in Southeast Asia (Malaysia, Singapore, Philippines, Indonesia, Thailand, Burma and the Lao Public Democratic Republic) a disease is observed which is characterized by skin ulcerations and therefore named "Ulcerative disease" (Anonymous, 1986; Llobrera and Gacutan, 1987). Clinical signs are (1) sluggish behaviour, and (2) large eroding, red or white necrotic skin ulcers, particularly on the head, *e.g.* mandible and maxilla, and on the caudal peduncle regions.

• *Etiology* - From all Ulcerative Disease outbreaks, bacteria, especially acromonads, and fungi can be cultured. However, no consistent relationship between Ulcerative Disease and any (micro)biological agent has been found so far (Anonymous, 1986). Llobrera and Gacutan (1987) suggested that the presence of *A. hydrophila* in Asian catfish suffering Ulcerative Disease is secondary to some predisposing factor(s).

• *Pathology* - At post mortem most fish is emaciated while in severe cases characteristics of septicemia (inflammation of the intestine and kidney or abdominal wall) are observed. At histopathological examination (1) meningeal hyperaemia, (2) edema and occasional round cell infiltration or cuffing, focal degeneration of the cardiac musculature, and (3) very severe focal renal degeneration are found. In kidney and spleen large, round, eosinophilic cells can be observed. In these cells rhabdovirus-like particles can be demonstrated.

• *Diagnosis* - The disease is diagnosed by the presence of the ulcers combined with histopathological characteristics.

• *Treatment* - Because bacterial involvement is most likely, a treatment with a broad spectrum antimicrobial drug can be helpful to minimize the damage to catfish populations suffering from this disease.

• *Prevention* - No specific preventive measures are known. A proper husbandry system is the only one.

CONCLUSION

From the present review it is clear that in the culture of catfish problems with infectious diseases exist. However, compared to the culture of other fish species, for instance salmonids with a number of important viral and bacterial diseases, the number of pathogens related to catfish diseases with a significant economic impact is low. At the present state only one viral (by CCV) and one bacterial disease (by *E. ictaluri*) can be a threat for catfish industry. However, both diseases are diagnosed in North America. In Europe, Asia and Africa no comparable catfish-specific infectious diseases are diagnosed suggesting that they do not exist in those parts of the world. However, the latter is doubtful and may be related to the difference in scientific input in catfish disease research between especially the USA and the rest of the world as a reflection of the economic impact of the respective national catfish productions. That may also be the main reason for which the etiological agents of different *Clarias* sp. diseases are not described yet. A comparison can be made with the research in viral fish diseases, e.g. from the eighties onwards scientists paid more attention to those which resulted in a dramatically increased number of identified viruses and related diseases (Hedrick and Hedrick, 1993).

Moreover, it is known that in industrial fish production, like the channel catfish production in the USA, epidemics occur more easily compared to small scale catfish production like in other countries. Furthermore, it is suggested that for example *C. gariepinus* is very infectious-disease resistant. This implicates that if this fish species becomes diseased it is nearly always related to bad culture conditions. It is likely that this is the same with *C. batrachus*. This explains the involvement of mainly secondary infections in the diseases of those species.

With the development of catfish culture industry, it can be expected that the prevalence of viral, bacterial and fungal diseases will increase, followed by an increase in drug (ab)use and need for vaccination. If so, research in catfish diseases has to make a shift in near future into improving catfish health by optimization of husbandry systems and increasing the disease resistance of catfish. The latter also implies development of a better genetic capability of the fish to cope with pathogens.

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