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Marine finfish pathology : specific problems and research in the French West Indies

D. GALLET DE SAINT AURIN, J.C. RAYMOND, V. VIANAS

IFREMER, Pointe Fort, 97231 LE ROBERT, Martinique

Abstract — The finfish species reared in marine intensive conditions in the French Caribbean Islands are imported (European SeaBass, American Red Drum and *Oreochromis* Hybrids) or local (Carangids and Lutjanids) species. The European SeaBass (*Dicentrarchus labrax*) is susceptible, during its first year of life, to a contagious disease characterized by whirling and hyperexcitability, associated with severe lesions of the brain, viral particles being pointed out in the damaged nervous cells. The only way to contend with this disease is to undertake preventive actions (excellent sanitary management and reinforced diet with vitamins and immunostimulants). The red Drum (*Sciaenops ocellatus*), in its hatchery phase, is highly susceptible to parasitism (*Amyloodinium ocellatum* and *Microsporidia*-like organisms) that adapted treatments are able to control. In its nursery and grow-out phases, nutritional deficiencies can induce vertebral column spontaneous fractures, making necessary extemporaneous vitaminic additions to the diet. This technique allows to prevent as well the Scale Loss-Blindness-Melanism Syndrome of Lutjanids, characterized by melanism and blindness with retina degeneration. The *Oreochromis* Hybrids, the Carangid Palometa (*Trachinotus goodei*) and the Lutjanid Yellowtail Snapper (*Ocyurus chrysurus*) are susceptible to parasitism by the Monogenean Fluke *Neobenedenia melleni*, and, for the latter species, by the Ciliate Protozoa *Brooklynella hostilis* and *Cryptocaryon irritans*. Depending on the parasite, the fish species and the rearing condition (tank or cage), different preventive and curative treatments have been carried out. All these primary diseases create favourable conditions for bacterial contaminations, which were always found secondary.

INTRODUCTION

The Finfish species reared in marine intensive conditions in the French Caribbean Islands are both :

- imported ones, such as European SeaBass (*Dicentrarchus labrax*), American Red Drum (*Sciaenops ocellatus*), and « Florida » *Oreochromis* Hybrid (*O. mossambicus* o X *O. hornorum* o)

- and local ones, such as Carangid Palometa (*Trachinotus goodei*) and Lutjanid Yellowtail Snapper (*Ocyurus chrysurus*).

The first three ones are reared with a purpose of production, the two others are in an experimental phase. Rearing technologies, which have been adapted from an initial pattern, are similar. Consequently, the diseases encountered are in keeping with these technologies as well as with the animals themselves.

The IFREMER Laboratory of Pathology has a very practical goal, which is to prevent the rearings from disease; this requires the three following steps :

- first, identifying the outstanding pathological risks for each rearing stage of each species;
- second, finding out or adapting specific curative and preventive treatments to each problem;
- and third, inserting the preventive techniques into the rearing technology itself, in a routine way.

At the present state of knowledge, the Outstanding Primary Diseases for each rearing stage of each species are given in Table I. It is obvious that all these primary diseases create favourable conditions for bacterial contaminations, which were always found secondary.

Tab. I. — Outstanding diseases in the F.W.I. rearings

Species	European Seabass	Red Drum	Florida Hybrid	Carangids	Lutjanids
Diseases					
Whirlingh	H N G				
Parasitosis	B	H	G	B N G	N
Environmental < nutritional > Diseases		N			B G

- B Breeders storing
- H Hatchery stage
- N Nursery stage
- G Grow-out stage

THE WHIRLING DISEASE OF THE EUROPEAN SEABASS (*Dicentrarchus Labrax*)

A/ Epizootics since 1983

In the summer of 1983, appeared in the cages of European SeaBass a new disease which seemed to be linked to the temperature rising over thirty degrees centigrade (Picollier, pers. com.). It was called « Summer

Disease ». It was an apparently contagious disease which affected only young of the year animals and led them to death (morbidity = mortality).

During the summer of 1984 (June to October), the total mortality due to this disease reached eighty percent of the yearling livestock.

For the summer of 1985, preventive measures (Gallet de St Aurin, 1985) probably allowed to minimize the incidence of the disease, which was however active (ten percent mortality from June to October).

In the early 1986, a very similar syndrome affected the fingerlings in the nursery and the larvae in the hatchery, each cohort being affected younger than the previous one (the last one to be affected was only 30 days old); furthermore, the survivors of this « Whirling Disease » did not suffer « Summer Disease » in cages afterwards.

B/ Symptoms

The main symptoms of both « Summer Disease » and « Whirling Disease » are the following :

- progressive darkening, beginning on the caudal part of the fish
- nervous troubles, alternating with stages of remission : total loss of equilibrium; apparent blindness; hyperexcitability, in particular to noise or changes in the light intensity; and sudden and violent muscular contractions.

In all cases, death occurs within one week after the beginning of the nervous symptoms.

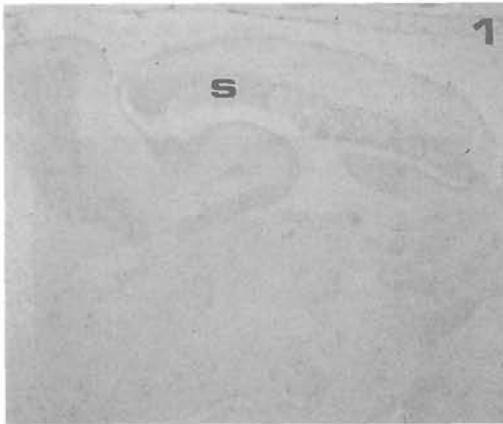


Figure 1. — Longitudinal section of the brain of a 10g diseased fish, showing spongiosis (S) in the optic tectum granular layer (HES \times 40).

C/ Lesions

Histological studies showed, in addition to muscular degeneration (Gallet de St Aurin et al., in press), specific lesions in the brain of diseased fish :

- a Spongiosis, which in the cage fish is limited to the optic tectum granular layer (Figure 1); in the fingerlings and larvae, this

spongiosis can become so extensive that the whole nervous tissue (including the spinal cord and the retina, which is a part of the mesencephalon) is affected (Figure 2).



Figure 2. — Longitudinal section of the anterior part of the brain of a 45 days old diseased larva (HES \times 100).

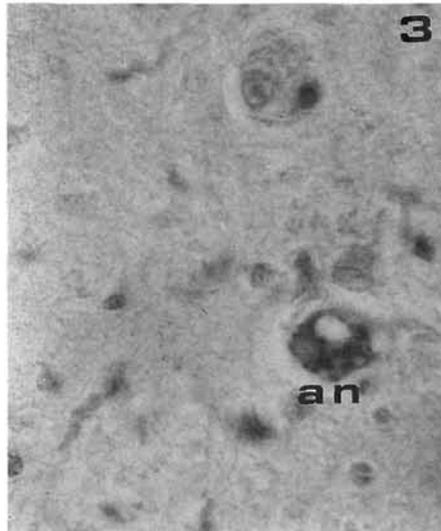


Figure 3. — Vacuolated and inclusion-bodies containing cytoplasm of an abnormal neuron (a n) surrounded by normal ones (HES \times 1000).

- the presence of abnormal neurons in different parts of the brain (optic tectum, tegmentum, cerebellum, vagal lobes, medulla oblongata and spinal cord) : they are enlarged cells with cytoplasmic vacuolation and presence of basophilic round-shaped inclusion-bodies (Figure 3). The vacuoles are sometimes very extensive (Figure 4). The younger the affected fish is, the more extensive and destructive the lesions are.

Electron Microscopy studies were performed on the brain of seventy days old healthy and diseased fingerlings. Healthy fish showed normal neurons (Figure 5). On diseased fish, the studies showed a modified shape of the affected neuronal cells and their inside nucleus, and the presence of round-shaped osmiophilic inclusion-bodies (one micrometer average diameter) in the cytoplasm (Figure 6).

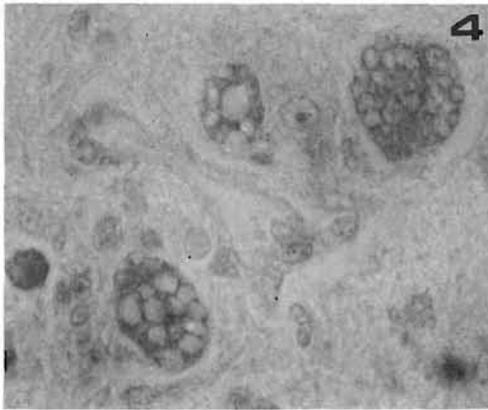


Figure 4. — Extensive vacuolation of enlarged affected neurons (HES \times 1000).



Figure 5. — Normal neurons. G : \times 5900.

At a higher magnification, the inclusion-bodies appear to be limited by a unit-membrane and to be filled with icosahedral particles (mean

diameter of the envelope : 35 ± 5 nm) (Figure 7). These particles sometimes have a very dense pseudo-cristalline array, and were identified as viral units. In some cases, the unit-membrane was interrupted, and the particles were spread into the neuronal cytoplasm (Figure 8).

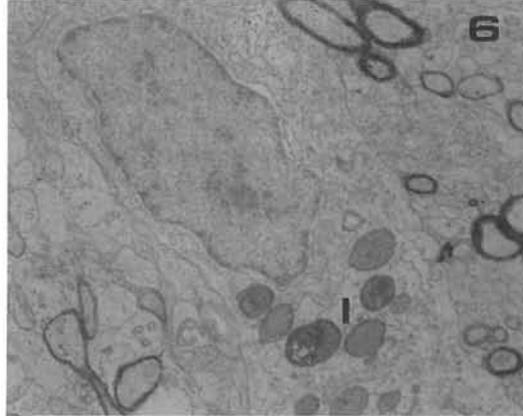


Figure 6. — Affected neurons : modified shape of the cell and the internal nucleus, and round-shaped osmiophilic inclusion-bodies (I) in the cytoplasm. G : $\times 5900$.

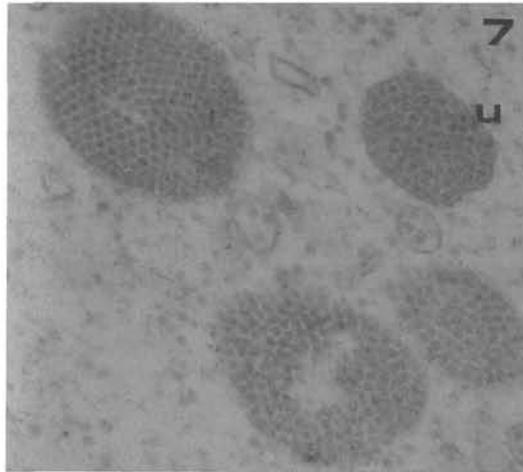


Figure 7. — Higher magnification of inclusion-bodies, showing the unit-membrane (U), and the inside icosahedral viral particles (external diameter : 35 nm). G : $\times 67000$.

D/ Discussion

At that state of knowledge, some questions are to be discussed :

1) Are the viral particles the aetiological agents of the « Whirling Disease » ?

Among the results (Gallet de St Aurin *et al.*, in press), it was found that :

- the cell-culture trials always failed
- thus far, the disease has not been experimentally reproduced.

However, the presence of viral particles in the cytoplasm of strongly affected neurons of fish showing nervous troubles, and their absence in the brain of apparently healthy fish, brings a quasi-certainty about the role of these particles in the morbid process. Also the following facts :

- progressive contagion,
- higher and higher virulence (fish affected younger and younger; nervous tissue lesions more and more extensive), come in aid of an infectious aetiology.

The failure of experimental reproduction of the disease indicates that probably other factors are necessary for its clinical manifestation; some authors showed that concomitant factors were involved in the expression of viral diseases (Schwedler and Plumb, 1982; Wolf, 1984).

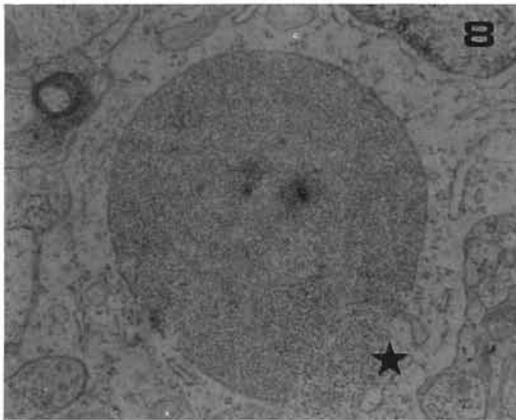


Figure 8. — Inclusion-body with interrupted unit-membrane and spreading of the virions into the neuronal cytoplasm (*). G : $\times 12500$.

2) Are the « Whirling Disease » of larvae and fingerlings, and the « Summer Disease » of cage fish, the same syndrome ?

These diseases have strong similarities in their epidemiology and symptoms, and above all induce identical histological lesions in the brain of the affected individuals.

Another argument in favour of the hypothesis of the uniqueness of these diseases is the fact that the cohorts previously affected by « Whirling » were not susceptible to « Summer Disease » some weeks later; as if these fish had developed an immunity resistance against this disease.

An Electron Microscopy study could not be performed on neurons of « Summer Diseased » fish. Only this would have brought the proof of the uniqueness.

3) What do such lesions look like ?

In Slow Virus Encephalitis such as KURU or CREUTZFELDT-JAKOB diseases in human beings, or SCRAPIE in sheeps, the lesional pattern is alike the above described one (Gajdusek, 1967 ; Carrier *et al.*, 1973); though the aetiological agents of these diseases are prions, which are not complete virions.

Nevertheless, the SeaBass Disease could be useful as an experimental model for the study of these Encephalitis (Mikol, pers. com.).

4) In a practical point of view, what was possible to do to contend with this disease ?

- the shore-side buildings (hatchery and nursery) were closed for several months.
- for the cage-rearing stage :
- new areas were looked for (lower temperatures, longer distance to the shore-side)
- preventive actions were undertaken before and during the critical summer period (from early May to October), consisting in a daily survey, an excellent sanitary management and reinforced diet with tonic and stimulant chemicals, such as :

Vitamin C : 100 mg/kg, 3 days a week

Vitamin E : 40 mg/kg, 3 days a month

Fish autolysate : 600 mg/kg, once a week

Levamisole : 100 mg/kg, twice a month

As seen above, these actions, undertaken in 1985, probably allowed to greatly reduce the mortalities during the summer period.

PARASITICAL DISEASES

The second kind of Outstanding Primary Diseases, and the most frequent, is Parasitism. It affects all the species at one or several rearing stages (Table 1). The tropical intensive mariculture conditions are highly favourable to direct external parasitosis, caused by Protozoa or Monogenean Flukes (Table 2, Figure 9).

A/ Parasitosis by *Neobenedenia melleni*

Its very wide repartition area and the various fish species it attacks (Jahn and Kuhn, 1932; Nigrelli and Breder, 1934; Nigrelli, 1935; Loyau, 1985; Conroy, pers. com.) make this parasite a real danger for many tropical fish cultures. In Martinique, among the reared species, only the red Drums have never been found with the parasite on them; the most susceptible are the Florida Hybrids.

As it is easy to treat and to prevent by hyposalinity (freshwater dips), this parasitosis is really dangerous only in cages. The infestation is favoured by a high density of fish, a small mesh size or a low quality food such as long-stocked pellets (Soletchnik *et al.*, 1988b). Also a previous

disease will favour its occurrence (Raymond, 1988). Two peaks of high infestation were revealed : March to May and August to October (Loyau, 1985).

Neobenedenia melleni is a Plathelminthus from Monogenea class and Capsalidae family. The adult is egg-shaped and can reach 5 mm in length. The parasitological cycle lasts 3 to 6 days at 29°C (Loyau, 1985).

Tab. 2. — Parasitological risks in the F.W.I. rearing conditions

Rearing conditions	Effects on pathogens	Expected parasitosis
Water	Effective vector	
High and stable temperatures	Rapid proliferation	
Some are weakened	Permanent source	Direct (Monoxene)
Many animals	Easy contagion	Ecto parasitosis
Organic matter	Supports for free stages	Protozoa
Closed or semi-closed systems	Increased efficiency	Monogenean flukes

REARING CONDITION	CAGE	TANK		HATCHERY	
	<i>Neobenedenia melleni</i>	<i>Brooklynella hostilis</i>	<i>Cryptocaryon irritans</i>	<i>Amyloodinium ocellatum</i>	<i>Microsporidia</i> - like
PARASITE					
	adult 0.3-5 mm	70 x 40 µ	50-450 x 30-50 µ	30-70 x 150 µ	3-6 granulations in cyst (200-500µ)
HOST(s)	FLORIDA HYBRID LUTJANIDS EUR. SEABASS CARANGIDS	LUTJANIDS EUR. SEABASS	LUTJANIDS EUR. SEABASS CARANGIDS	RED DRUM	

Figure 9. — Parasites affecting the rearings.

The first symptom is an abnormal behaviour of fishes. They rub against net (« flashing ») and jump out of the water. Anorexia and hyperproduction of skin mucus are also observed. Then appear haemorrhagic ulcers on the sides, fin rot (mainly on anal and caudal fins), thickening and opacification of the eye cornea. At this stage, it is easy to see many parasites on eyes, skin and fins. Ulcers are very quickly invaded by bacteria (*Vibrio*, *Aeromonas*, *Pseudomonas*) and mortality increases.

Prevention in cages is based on avoidance of favouring factors, by limiting the density of fish, providing a good quality food and sanitary management. These measures are able to keep the parasitological infestation at a low level, on the less susceptible species (Soletchnik et al., 1988b). They are not sufficient for Florida Hybrids, for which an early treatment, before anorexia, is possible with Trichlorfon (Dylox) in the food (50 mg/kg of fish, four times at 3 days intervals), each month during the critical periods (Loyau, 1985).

B/ Parasitosis by Ciliate Protozoa

The most dangerous parasites encountered in tank rearings, other than in the Red Drum Hatchery, are the Ciliate Protozoa *Brooklynella hostilis*, and above all *Cryptocaryon irritans* (Loyau, 1985; Gallet de St Aurin et al., 1986). This last parasite has a worldwide repartition area, affecting a large number of species of marine aquaria and cultures (Brown, 1951; de Graaf, 1962; Nigrelli & Ruggieri, 1966; Wilkie and Gordin, 1969; Sindermann, 1977; Herwig, 1978; Violetta, 1980; Hignette, 1981; Huff and Burns, 1981; Paperna, 1983; Loyau, 1985; Weppe, pers. comm.).

B. hostilis was previously reported from aquarian fish (Lom & Nigrelli, 1970).

In Martinique, Lutjanids and European SeaBass did suffer high infestations by *B. hostilis*. Also *C. irritans* affected Lutjanids, Carangids and European SeaBass in tanks. The infestations could occur all the year round, but they were more frequent on the summer time (Loyau, 1985).

Both parasites are localized on gills, causing respiratory troubles; but *C. irritans* also invades skin, and lives embedded under the host's epithelium (Colorni, 1985), that makes it much more destructive and also harder to reach by chemicals.

A *B. hostilis* infestation can easily be treated by a 200 ppt Formalin 15 mn bath; complete eradication is obtained by repeating this treatment 3 times at 48h intervals. On the other hand, the only efficient treatment used against *C. irritans* was a 12 days continuous exposure to Tris buffered Copper Sulfate, at a rate of 0.15 mg of Copper ion Cu^{++} per litre. Obviously this is a very heavy constraint.

So a quarantine process has been developed, to be used whenever new fish are to be put in tanks. Based on reported studies on the biology and attempts to control *C. irritans* (Nigrelli and Ruggieri, 1966; Canella, 1972; Blasiola, 1978; Herwig, 1978; Cheung et al., 1979; Hignette, 1981; Huff and Burns, 1981; Colorni, 1985) and local observations (Loyau, 1985), this 12 days quarantine has to be done in a special tank which had been previously disinfected and dried out, and which receives filtered water under 5 micrometers; at days 1, 5, 8, and 11, the fish are subjected to a 200 ppt Formalin 30 mn exposure, followed by a 5 mn freshwater dip and an antiseptic (50 g/m³ Furaladone Chlohydrate, or 2 ppt Quaternary Ammonia) 30 mn bath. Transfer occurs at day 13 to the rearing tank receiving filtered water under 5 micrometers; at day 14, a last antiseptic bath allows to avoid bacterial infection.

C/ Parasitosis in red Drum Hatchery

The red Drum larvae were affected by the Dinoflagellate *Amyloodinium ocellatum* and by what we tentatively identified as a Microsporidia-like organism.

A. ocellatum has a very wide repartition area and affects a large number of fish species (Blasiola, 1978; Overstreet, 1978; Lawler, 1979; Cheung et al., 1981; Baticados and Quintio, 1984; Barbaro and Francescon, 1985). The red Drum is noticed as a susceptible species by American researchers (Johnson, 1987; Arnold, pers.comm.; Roberts, pers.com.).

In Martinique, the European SeaBass breeders had suffered an *A. ocellatum* infestation (Gallet de St Aurin, 1985).

The eggs of red Drum were imported from the U.S.A., and it was not determined whether the parasites were imported as well, or were local strains. The larvae were affected as early as day 18 post hatching.

The Microsporidia-like organisms were found on 20 days old larvae (S.I.M., 1988).

The clinical manifestations of both parasitosis are identical : invading gills, skin and fins, they induce respiratory troubles by themselves and by the thick layer of mucus secreted by the host in response to their irritating presence.

According to reported studies on the biology and control trials on these parasites (Overstreet and Whatley, 1975; Bulla and Cheng, 1976 and 1977; Lawler, 1977b; Overstreet, 1978; Johnson, 1984; Paperna, 1984a and 1984b; Colorni, pers. com.) and to local observations (Gallet de St Aurin, 1985; S.I.M., 1988), attempts to treat these parasitosis in red Drum hatchery were carried out, after conclusive in vitro tests. But, due to the weakness of so young larvae and the difficulty to handle them, these treatments induce mortalities by themselves, which is not satisfactory.

A strict sanitary prevention remains the best way to avoid troubles. In particular, to avoid entering of parasites, the water in which the eggs are shipped needs to be filtered under one micrometre before shipment. Also the rearing water and the live preys should be under control. To avoid proliferation, a limited density of larvae (under 5 individuals per litre) and a good water quality maintenance are necessary. To avoid spreading of any parasites which could have entered the hatchery, time partitioning between batches, space partitioning between tanks (each one having its own equipment, which is disinfected after each use), a daily sharp observation of the animals, and in case of disease in one tank, the early discard of this tank, are necessary measures.

ENVIRONMENTO-NUTRITIONAL DISEASES

The third group of Outstanding Primary Diseases is the environmental (including nutritional) one. These diseases affected in Martinique mainly the red Drum in nursery and the Lutjanids in grow-out cages (Table 1).

A/ The Scale Loss- Blindness-Melanism Syndrome

1) Description of the disease

This disease affected the Lutjanids, and at a lesser extent the European SeaBass; it appeared after a few months of cage rearing and feeding on pellets (Gallet de St Aurin *et al.*, 1986). The affected fish were found lonely at the water surface, dark coloured, very slow moving, and emaciated. Scales were lost in patches on the fish sides, the lower lip was ulcerated, and from the anus was often issuing green-whitish faeces (Raymond, 1988). Fish were dying mostly by secondary bacterial infection.

2) Lesions

Necropsy of the dying fish showed a few unspecific signs, and mainly the absence of mesenteric fat (Raymond, 1988).

Histological studies were performed on slightly affected fish :

- no lesions were found in the integument; only the melanocytes were expanded and numerous
- in some cases, a lysis of the intestinal mucosa was observed
- a retina degeneration was evidenced for all the observed diseased fish (Figure 10); more obvious in the central retina area, this degeneration affects all the layers internal to the pigmentary epithelium : these layers become thinner and thinner, and merge together, making the thickness of the retina about one third of the normal value; the photoreceptor cells, mainly the Rod-cells, are altered and become scarce (Raymond, 1988).

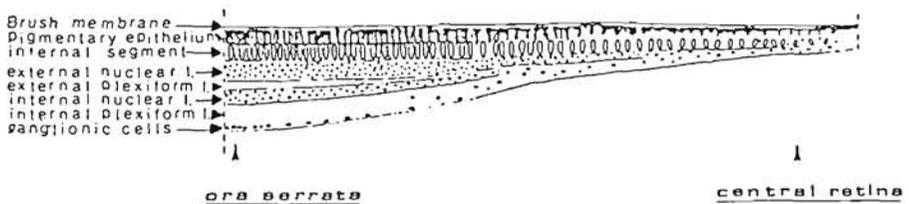


Figure 10. — Retina degeneration.

3) Pathogenic Scheme

All the experimental findings (Raymond, 1988) go to prove that the pathogenic scheme is the following :

The initial retina degeneration (which is the only specific lesion) induces blindness, and then melanism by humoral route. But also starvation, which itself explains a biliary retention which is responsible for the lysis of intestinal mucosa (observed in some cases), and emaciation and lack of mesenteric fat. Blindness explains as well the lower lip ulceration (which is the consequence of knocking against the net), and the poor

mobility. Parasites and Bacteria find very favourable conditions and induce secondarily the scale losses, and new ulcerative lesions.

4) Proposed Aetiology

According to the literature (Lawill *et al.*, 1977; Lanum, 1978; Penn, 1985), such a retina degeneration is probably due to a phototraumatism by high light intensity, the fish being reared in shallow cages in clear water.

This type of phototraumatism is greatly favoured by the high temperature, and the oxidizing of membrane lipids (Farnsworth & Dratz, 1976; Weigand and Giusto, 1983; Bazan *et al.*, 1984), which occurs for example when the given diet is Vitamin E and/or Vitamin C deficient, as both Vitamins act as membrane anti-oxidative (Ashley *et al.*, 1975; Farnsworth and Dratz, 1976; Amemiyat, 1981; Joel *et al.*, 1984; Organisciak, 1985; Cowey, 1986).

B/ Spontaneous Vertebral Column Fractures

1) Description of the Disease

The red Drum and the European SeaBass in their nursery stage were affected by a disease which induced low but constant mortality (one to two percent of the livestock per day). It appeared generally 15 to 20 days after weaning on pellets. The affected juveniles were seen motionless in an oblique posture, dark coloured in the caudal half part of the body, and sometimes showing a lateral curvature (Figure 11). When forced to swim away, they could only move the anterior fins, which suggests paralysis.

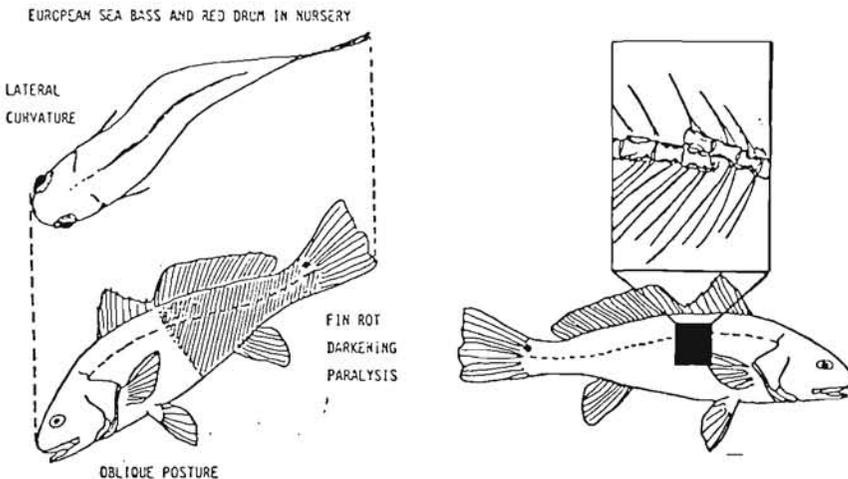


Figure 11. — Symptoms and lesions of « vertebral column spontaneous fractures ».

2) Lesions

Necropsy and radiography of the affected fish showed a fracture of the vertebral column, located between vertebrae six and twelve (Figure 11).

Radiography performed on apparently healthy fish from the same batch showed, in 12 percent of the cases, a scoliosis or a lordosis (unpublished data).

3) Proposed Aetiology

Scoliosis and lordosis in fish are assigned to various causes (Hoffman and Dunbar, 1961; Ashley *et al.*, 1975; Kloppel and Post, 1975; Overstreet, 1978; Halver, 1980; From *et al.*, 1985). Between them, a Vitamin C deficiency is known to induce a deficient synthesis of the connective tissue, and then squelettal deformities and frailty (Koenig, 1984). In our case, during the fast growing nursery period, a spontaneous fracture will occur in the maximal bending zone of the body of some fish, when swimming.

C/ Prevention of Scale Loss-Blindness-Melanism Syndrome and Spontaneous Vertebral Column Fractures

Both above mentioned diseases, so different in their clinical manifestations, have in common a nutritional aetiological factor, which is a Vitamin C deficiency. The use of commercial pellets available in the French West Indies, even without any storage, seems to be inadequate for supplying the fish vitaminic needs (Koenig, 1984); high temperature and moisture greatly reduce the conservation time of vitamins, specially Vitamin C (Halver, 1980; Messenger, *pers. com.*); Vitamins C and E have synergical activities (Halver, 1980; Cowey, 1986; Cillard, 1987).

According to the literature and to the tests carried out in Martinique (Raymond, 1988; Gallet de St Aurin, unpublished data), the prevention of the above diseases can only be obtained by extemporaneously oil-coating the daily diet (on a seven days a week feeding basis) with the following amounts of Vitamins :

- during the nursery stage, beginning as early as the weaning time :
 - 100 mg Vitamin C per Kg of fish
 - 20 mg Vitamin E per Kg of fish
- during the grow-out stage :
 - 30 mg Vitamin C per Kg of fish
 - 2 mg Vitamin E per Kg of fish

CONCLUSION

All the above mentioned pathologies could be linked to a definite aetiology, which is often concealed by secondary pathogenic causes; thanks to an accurate diagnosis, they could be contended with, in a more or less successful way. On the other hand, in larval rearings, the results in terms of survival remain highly variable from one batch to another, suggesting the occurrence of « pathological events » which, due to the size of larvae and to the rapidity of the course of such events, remain too often misunderstood. The researches have now to be focused on this field.

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