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The Propagation of Cod *Gadus morhua* L.

# DISEASE PROBLEMS IN COD REARING

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## ABSTRACT

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Disease seems to be an inevitable part of fish rearing systems, and the first disease problems in the Austevoll cod rearing experiments are reported. Work on prophylactic treatment against vibriosis through vaccination has also been started.

## INTRODUCTION

The purpose of this preliminary report on disease conditions in the cod rearing experiments at Austevoll Aquaculture Station, is to emphasize that disease is one of the negative aspects of any fish rearing system and one that has great economic consequences. To hold a symposium on cod rearing without mention of disease would be to omit an important aspect that anybody getting involved in sea ranching or cage culture of cod will undoubtly be confronted with.

When the cod work was planned eventual disease problems do not seem to have been considered. The disease laboratory of the Institute of Marine Research is a small one, and cannot put much effort into new problems without some planning.

Therefore the major problem of fungal infections in egg and early larval stages was overlooked during the first few years of rearing experiments. The subsequent years, however, saw a greater involvement by the disease laboratory into the cod rearing work and some of the problems examined are presented here.

## SPAWNERS

Most of the work has been done on bacterial and parasitic infections.

The latter category has the most deleterious effect on the spawners which are selected from captured specimens of the local stock. The prospective spawners are kept in a 175 $m^3$ cage made of plastic coated nylon material and supplied with water of relatively constant temperature pumped up from depth. The water inlet is about midway down the cage creating a surface current that makes it possible to skim off the eggs.

Throughout 1982 no special problems were encountered in the spawners until last winter (-82/83) when they started to die. The gills of the dead fish were covered with a slimy layer and smears of the slime revealed large masses of a protozoan parasite of the genus *Trichodina*. Some of the spawners also had superficial skin lesions containing the same parasite. The overall mortality was about 20%, and Trichodinads were found on 18 out of 19 dead fish.

The genus *Trichodina* of the family Urceolariidae are peritrichous ciliates. They are rather large (40 up to 100 µm in diameter). Seen from below the parasite is ovoid with a ring of cilia at the circumference, seen from the side it is bell-shaped. On the ventral side there is a characteristic ring of saw-like hooks (Fig. 1). These features render it easily recognizable in smear preparations. The parasite seems to multiply excessively only on fish weakened by previous stress and is assumed to feed on epidermal tissues.

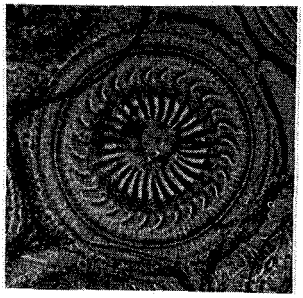

Fig. 1. *Trichodina* sp.

The affected cage was treated with formalin in calculated doses of 1:4 000. As the circulation in the cage was rather slow, particularly in the bottom layers, the formalin was added without stoppage of the water flow on the assumption that the exchange would be of sufficient duration for the chemical to take effect. Only one treatment was necessary to stop the mortality.

Trichodinads seem to be fairly common in the marine environment and most probably start to multiply and cause harm only after the fish is weakened by some sort of stress. Under rearing conditions prophylactic treatment with formalin may therefore be advisable.

## EGGS AND YOLK SAC LARVAE

In the initial years of rearing experiments all fish died in either the egg or yolk sac larva stage. At least a fungus, but probably also bacteria, was involved, although we were not able to cultivate the fungus.

In the present practice hatching is carried out in filtered and U.V.-treated water, and the eggs are treated prophylactically with an antibiotic and a fungicide prior to being placed into the hatching vessels.

To test the influence of heavy bacterial concentrations on the early larval stages, two experiments were conducted in April and May of this year.

In the first experiment four 10 l jars were half-filled with filtered and U.V.-treated seawater and placed in a flow through bath of seawater with a temperature of 6.5-6.9°C. An equal amount of 5 day old yolk sac larvae were put into each container. One jar was maintained as a control group and to the others 24 to 48 h cultures of *V. anguillarum*, isolated the previous year from moribund cod, were added. The concentrations used were approximately in ratios of 2:1, 1:1 and 1:10 of the amount of cells usual for challenge experiments.

For the second experiment a culture of *Flexibacter sp.*, isolated from cod with peduncle disease, was used. Again three different concentrations, prepared as in the above experiment but without any counting, were used to challenge the larvae. The temperature during these trials varied between 6.0 and 6.8°C.

None of the various concentrations of the two bacterial species seemed to have any effect. The larvae died the 18th day from starvation. The outcome of the vibrio experiment could be explained by the low temperature, whereas the temperature was within the range where the *Flexibacter* usually is harmful. The results may indicate that weakening of the larvae by some sort of stress is necessary to render them susceptible to the bacteria.

## VIBRIOSIS

Vibriosis is an infectious disease of marine fish generally caused by the bacterium *Vibrio anguillarum*, a small, comma-shaped, Gram-negative, motile rod. It was first, as the name indicates, described from eels in brackish water by Bergmann in 1909, and since then the disease has been described - with different symptoms - in a wide range of marine fish species. In 1911 Bergmann described it as an eye lesion

in cod. Later Bagge and Bagge (1956) described vibriosis as an ulcus disease in cod. In sea-water culture of rainbow trout (*Salmo gairdneri*) and Atlantic salmon (*Salmo salar*) the predominant symptoms of vibriosis are superficial skin lesions, hemorrhages and boils. In saithe (*Pollachius virens*) for which the disease reached epidemic proportions around the Norwegian west coast in 1975, the main symptoms are superficial skin lesions (Egidius et al., 1983).

In the juvenile cod at Austevoll the primary symptoms were lesions in the head and tail regions. The fish became pop-eyed and within a short time the eyes disappeared entirely leaving eyesockets with hemorrhages (Fig. 2). Initially the tail lesions appeared similar to tail-rot, but within short time most of the tail also disappeared. Hemorrhages could also be seen elsewhere on the fish, but the principal sites of damage were the head and tail.

Biochemically there is a slight difference in reactions between strains isolated from different fish species, placing the strains in two groups that differ in 7 out of the nearly 70 reactions routinely tested.

We also have been able to demonstrate (Egidius and Andersen, 1978) some host specificity in the pathogenicity of strains isolated from different fish species. For example strains isolated from saithe did not affect rainbow trout, while strains isolated from rainbow trout only had minor effects on saithe. In an experiment last year (unpublished) with rainbow trout challenged with a strain originating from cod, symptoms appeared similar to those in cod, but rather different from those usually seen in rainbow trout. The bacterial strains isolated from different fish species seem to give at least some serological cross agglutination.

In rainbow trout, vibriosis can, to a certain degree, be treated with antibiotics and chemotherapeutics. In this country only oxytetracycline and tribissen (trimethoprim and sulphadiazin) are licensed for use in fish. In *vitro* the cod strains are susceptible to both, but in practice treatment does not seem efficient. The medicines are distributed

through the food and so far the same dosages as for salmonids have been used. The medication thus consumed is probably not sufficient for the cod.

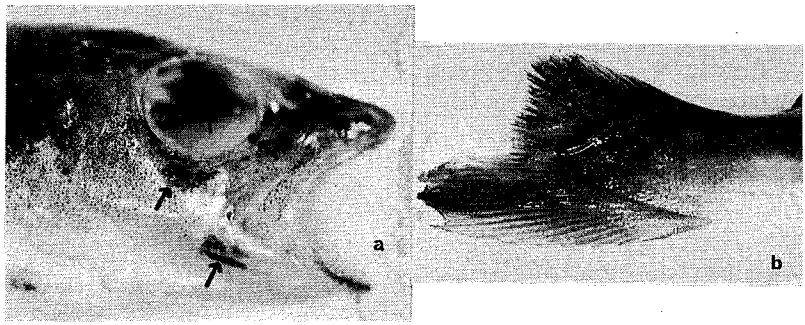


Fig. 2. Vibriosis in cod. a) Hemorrhages in head region, empty eye-socket also with hemorrhages. b) Typical tail lesion.

## VACCINATION

Vaccination against vibriosis has been shown to give reliable protection in rainbow trout and various vaccines are commercially available. As it became apparent that vibriosis would also be a problem in the rearing of cod, the feasibility of vaccination for this species was investigated.

For rainbow trout several vaccination methods have been described (Egidius et al., 1982). The easiest method - adding the vaccines to the food - is not yet economically feasible, and we therefore concentrated on bath vaccination which is, in our opinion, the least stressing of the methods available.

The vaccine for cod was produced in the same way as the rainbow trout vaccine. A *Vibrio anguillarum* strain isolated from cod dying of vibriosis was used. The pathogenicity was proved by challenge and the bacterium was re-isolated from the dead fish. For laboratory production, the bacterium is

grown in tryptone/glucose/yeast broth for 48 h, after which the culture is killed by adding 0.3% formalin and then digested with trypsin (0.25 g/l). For larger scale production the bacterium is grown in a fermentor. The suspension of cell fragments obtained is used as vaccine. For the 1982 experiments the vaccine was produced in our laboratory, whereas the 1983 vaccine is being produced on a larger scale at the University of Tromsø.

For vaccination the water flow in the tanks involved is substituted by oxygenation, the water level is lowered and the vaccine is added. The fish swim in this bath for two hours, and thereafter the water flow is returned. For rainbow trout the vaccine dose was 0.5 $l/m^3$ of water at a temperature above $8^{o}C$. For cod we used equal and larger doses at a temperature of $8\text{-}10^{o}C$.

In 1982 unfortunately only about 200 cod fry were available for experimentation. The fry were transferred to the Institute in June and kept in 200 l experimental tanks with U.V.-treated seawater. In a first experiment 50 fish were vaccinated and 50 kept for control. Approximately the same vaccine dose as for rainbow trout was used. The effect of this vaccination was very low. In a second experiment, far higher vaccine doses, together with a chemical facilating penetration, were applied to 41 fish. Only 16 fish were available as controls. Four weeks later these two groups were challenged for one hour with a 24 h culture of the parent bacterium. Just prior to challenge, the water temperature was raised $3^{o}C$ and maintained for the rest of the experimental time.

After two weeks (our regular test period for vaccination work on rainbow trout) mortality in the controls was 43% while in the vaccinated group it was approximately 10%. The bacterium used for challenge could be re-isolated in almost pure culture from the fish that died. These promising results will be followed up this year.

For practical application in the pond rearing of cod, vaccination can only be applied prior to release, that means

at a very early larval stage. In May 1983 investigations into the earliest possible stage for vaccination were begun using bath-vaccination techniques and four batches of larvae. The first batch was vaccinated 5 days after hatching, the second batch 10 days later and the third batch 26 days after hatching. For the fourth batch also 26 days after hatching, a double dose of vaccine was used. The water temperature during this period was over 8.3°C. Unfortunately, the over all mortality of fry hatched in May was very high, and to few of the larva in the experiment survived to make the challenge experiment worth while. This will have to be repeated with fry from the earliest egg batches next year.

As we suspect that vaccination at the very early stages necessary for the pond release, can be useless, experiments on oral vaccination also has been started.

## OTHER PROBLEMS

A parasite does not necessarily cause a disease, but a large infestation may make the product less appetizing for consumption. The nematode parasites of codfish are well known, and may be present in great numbers, but they seem rather harmless depending on species and stage. This year, samples of cod fry from the Austevoll pond revealed *Thynascaris aduncus* in the intestine, a condition which at least should be carefully observed. Also black spot (metacercariae of trematodes) was observed in fish from the pond in 1982.

To conclude, it appears likely that culture of a new fish species will encounter the diseases and parasites that are present in the freeliving populations of that species. But due to rearing conditions, the diseases may have greater persistence and far-reaching consequences.

REFERENCES

Bergman, A., 1909. Die rote Beulenkrankheit des Aals. Ber. K. bayer. biol. Vers. stn. Munchen, 2: 10-54.

Bergmann, A.M., 1911. Eine ansteckende Augenkrankheit, Keratomalacie, bei Dorschen an der Südküste Schwedens. Centralbl. Bakt. Abt. Orig. 62: 200-212.

Bagge, J. and Bagge, O., 1956. *Vibrio anguillarum* som årsag til ulcus-sygdom hos torsk (*Gadus callarias*, Linné). Nord. Vet.-Med., 8: 481-492.

Egidius, E. and Andersen, K., 1978. Host-specific pathogenicity of strains of *Vibrio anguillarum* isolated from rainbow trout *Salmo gairdneri* Richardson and saithe *Pollachius virens*. J. Fish Diseases, 1: 45-50.

Egidius, E., Andersen, K., Clausen, E. and Raa, J., 1982. Bath vaccination against vibriosis. Devel. Comp. Immunology, Suppl. 2: 193-196.

Egidius, E. Braaten, B., Andersen, K. and Lohne Gokstad, S., 1983. Vibriosis in saithe (*Pollachius virens*) populations off the Norwegian coast. Rapp. P.-v. Réun. Cons. int. Explor. Mer, 182: 103-105.

SECTION VI

# *Management of farmed and natural stocks*

CHAIRMAN

*T. Smith*