Bull. Eur. Ass. Fish Pathol. 11 (6), 219, 1991

CRANIAL DEFORMITIES IN ATLANTIC SALMON ALEVINS

By Rodger, H.D. and Murphy, T.M.

Physical anomalies in juvenile fish stocks are an occasional observation and have been reported by various authors (Gemmill, 1912, Roberts, 1989). The following study presents the findings from an investigation into physical abnormalities and mortalities in stocks of farmed Atlantic salmon (*Salmo salar*) alevins during March and April 1991. The pathology observed is described and the possible causes of the deformity discussed.

Four freshwater hatcheries in Ireland reported that certain batches of fry were exhibiting lethargic behaviour with significant mortalities. Samples of fish were examined from two of the affected hatcheries and the gross physical appearance was that of a swollen cranium and exophthalmos (Fig. 1).

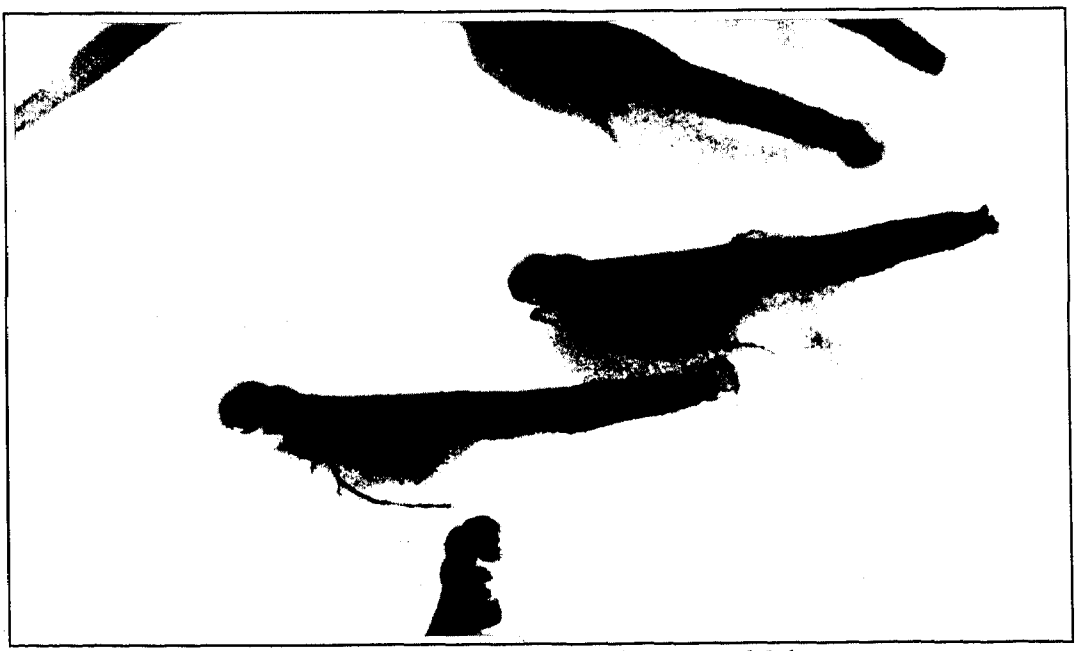

Fig. 1. Atlantic salmon yolk-sac fry exhibiting swelling of the cranium and exophthalmos.

Also present was a varying degree of elongation of the yolk-sac. Otherwise the external appearance was normal. Mortalities and deformities in all four hatcheries were limited to one particular stock origin of fish and ranged from 10 to 20% stock losses. The age of the affected fish ranged from two to four weeks from hatching.

Samples of whole alevins were processed for virology from two of the affected hatcheries. Under standard isolation procedures, using chinook salmon embryo (CHSE-214) rainbow trout gonad (RTG-2) and Atlantic salmon (AS) cell lines, no cytopathic effects were seen and the alevins were deemed negative for pathogenic viruses. Samples from the same two hatcheries were taken for histological examination and revealed that the cranial swelling was due to an increase in the space between the meninges and the optic lobe (Fig. 2). In some cases haemorrhage was obvious in the sub-arachnoid space or in the granular layer of the optic lobes. Necrotic cells were obvious in the

meninges and on the outer surface of the molecular layer of the optic lobe (the stratum fibrosum marginale), as shown in Figure 3. These changes are consistent with a build up of excessive cerebrospinal fluid or hydrocephalus.

Fig. 2. Section through the cranium of yolk-sac fry revealing increased space between the meninges (arrow) and the optic lobe (O). A focal haemorrhagic area (FH) can be observed adjacent to the stratum fibrosum marginale (molecular layer) of the optic lobe (haematoxylin and eosin x 200).

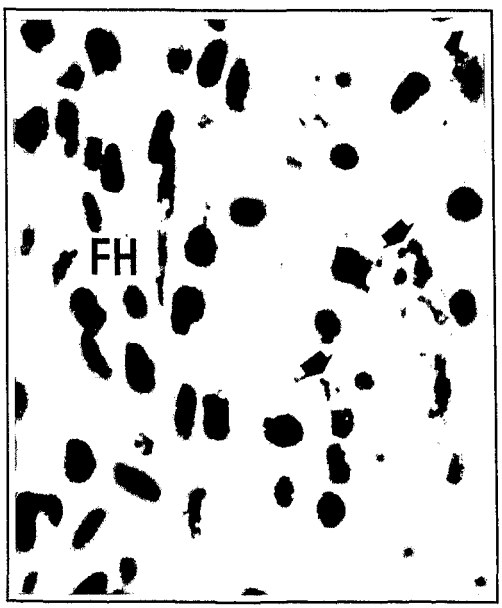

Fig. 3. Section through the stratum fibrosum marginale of the optic lobe showing nectrotic cells (arrow) below a focal haemorrhage (FH)(haematoxylin and eosin x 1000).

There was no evidence of inflammatory exudate, tumours or parasites, all of which can give rise to obstruction of fluid drainage. In view of the number of stock affected, the age of the stock and the fact that four seperate hatcheries experienced similar problems, the hydrocephalus can be considered congenital.

In experiments, viral diseases have been shown to induce hydrocephalus in juvenile terrestrial animals (Margolis and Kilham, 1969) and cranial distension and mortalities in pike fry (*Esox lucius*) have been observed associated with infections with pike fry rhabdovirus (Bootsma, 1971; de Kinkelin *et al.*, 1973). In this case study there were no viral isolations under standard procedures.

Hydrocephalus has been associated with several experimentally induced nutritional deficiencies in pregnant laboratory rodents (Jones and Hunt, 1983) but no references can be found linking these in fish. Haemorrhage and degeneration of specific nuclei of the periventricular areas of the brain of Atlantic herring (*Clupea harengus*) were described by Baxter *et al.*, (1974) after experimentally feeding a diet deficient in thiamine. The fish in this study were experiencing the pathologies prior to the feeding of solid food, so it is considered unlikely that the deformities have a nutritional basis.

Ferguson (1989) records gross lesions, similar to those in this study, in rainbow trout (*Oncorhynchus mykiss*) and coho salmon (*Oncorhynchus kisutch*) fry which were also unrelated to any virus. The suspect cause of the lesions was a teratogenic agent, such as malachite green. In this study, malachite green was used in the affected hatcheries for the control of fungus on eggs. However despite all eggs being treated, only one strain of fish appeared affected by the deformity, indicating that the role of the dye in inducing this problem was remote. However the possibility that the one strain of fish were particularly susceptible to the chemical cannot be excluded.

Inherited defects usually due to autosomal recessive traits can also give rise to congenital hydrocephalus in cattle (Greene *et al.*

1974). This must be considered as a possibility in this case study, where specific cross fertilizations may have given rise to this phenomenon.

Summary

Four salmon hatcheries experienced stock losses in a particular strain of fish. Clinically they presented as lethargic alevins with cranial swelling and exophthalmos. Histology indicated an increase in the space between the menginges and the optic lobes, with associated haemorrhage and focal necrosis. There was no evidence for an infectious agent on standard virological isolation. The basis for the mortalities is suggested as a congenital hydrocephalus.

Acknowledgements

We wish to acknowledge Ms. Margaret Ruttledge and Ms. Catherine Hickey for technical assistance in histological and virological processing and preparation.

References

Blaxter, J.H.S., Roberts, R.J., Balbontin, F. and McQueen, A. (1974). B-group vitamin deficency in cultured herring. Aquaculture, *3*, 387-394.

Bootsma, R. (1971). Hydrocephalus and red-disease in pike fry *Esox lucius*L.. Journal of Fish Biology, *3*, 417-419.

de Kinkelin, P., Galimard, B. and Bootsma, R. (9173). Isolation and identification of the causative agent of „red-disease" of pike (*Esox lucius*L., 1766). Nature, *241*, 465-7.

Ferguson, H.W. (1989). Systematic Pathology of Fish. Iowa State University Press, Iowa.

Gemmill, J.F. (1912). The Teratology of Fishes. Maclehose, Glasgow.

Greene, H.J., Leipold, H.W. and Hibbs, C.M. (1974). Bovine congenital defects. Variations of internal hydrocephalus. Cornell Veterinarian, *64*, 596-616.

Jones, T.C. and Hunt, R.D. (1983). Veterinary Pathology. Lea and Febiger, Philidelphia, 5TH edition.

Margolis, G. and Kilham, L. (1969). Hydrocephalus in hamsters, ferrets, rats and mice following inoculations with *Reovirus* type 1. II. Pathologic studies. Laboratory Investigation, *21*, 189-198.

Roberts, R.J. (1989). Fish Pathology. Baillere Tindall, London, 2ND edition.

Author's address

National Diagnostic Centre, BioResearch Ireland, University College, Galway, ireland.