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NOTE

Swimbladder abnormality in farmed Atlantic salmon *Salmo salar*

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ABSTRACT: Malformation of the swimbladder of farmed Atlantic salmon *Salmo salar* L. is an increasing problem in Norwegian aquaculture. Affected fish exhibit a characteristic behaviour suggesting that balance and buoyancy are altered. In affected fish, the swimbladder is shortened and dilated and the pneumatic duct opens at the normal point in the oesophagus, but it runs caudally along the ventral side of the swimbladder and enters the caudal pole of the organ. This is in contrast to normal fish, where the pneumatic duct is very short and enters the cranioventral part of the swimbladder. This malformation of the pneumatic duct may lead to altered filling of the swimbladder, which in turn may cause the abnormal shape and size of the organ.

KEY WORDS: Malformation · Swimbladder · Pneumatic duct · Norway · *Salmo salar*

Malformations occur quite frequently in farmed fish and are of concern because of increased mortality, impaired growth and problems during processing. Malformations commonly observed in farmed Atlantic salmon *Salmo salar* L. include spine and jaw deformities and absence of the transverse septum (Kaada & Hopp 1995).

During the winter 1994/95, increasing numbers of fish with aberrant behaviour were noticed in several smolt farms in northern Norway. The fish exhibited apparently normal behaviour until they reached approximately 40 g, whereafter aberrant behaviour was observed. Affected fish were easily discerned because they swam more energetically than normal fish, exposing their shining flanks and white bellies. Some even swam upside down or in an almost vertical position. Affected fish congregated near the edges of the tanks. When frightened, the fish often exhibited a normal swimming pattern for some time, then resumed their characteristic side-swimming patterns. Affected fish have been termed 'side swimmers' by the fish farmers. According to field observations, affected fish apparently had normal reflexes and appetite, and were no smaller than their siblings.

Other abnormalities are frequently reported concurrent with swimbladder malformation. These include underdeveloped ventricle, a severely reduced number of pyloric caecae and situs inversus of the liver.

In this paper, we describe a malformation of the swimbladder of farmed Atlantic salmon and discuss its patho-anatomical features and possible causes.

Materials and methods. Necropsy was performed on 11 1-yr-old smolts recently transferred to sea-cages which exhibited the previously observed behaviour associated with swimbladder abnormality. Fish with typical abnormal behaviour were euthanised with a blow to the head, the gill arteries severed and specimens were shipped on ice to the Norwegian College of Veterinary Medicine, Oslo. The fish weighed from 60 to 80 g.

During necropsy, the swimbladder and pneumatic duct were exposed by dissection. Particular care was taken not to puncture the swimbladder or sever the pneumatic duct. The anatomy of the swimbladder was altered in all fish, and 3 typical specimens with malformed swimbladders were chosen for casting this organ. Casts were prepared with epoxy solution (Biodur® E20, Biodur® hardener E2, Biodur® red colouring AC 50; Biodur™ Products, Heidelberg, Germany) diluted 20% with methyl ethyl ketone. The mixed solution was either injected directly into the swimbladder through the body wall or infused via the pneumatic duct using a catheter. For infusion, the oesophagus was opened and the opening of the pneumatic duct located. After infusion, the fish were incubated at 60°C for 3 d to ensure thorough hardening of the epoxy solution. Following hardening, the fish were gently boiled in water until tissue could be removed from around the cast.

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Two smolts of similar size from an unaffected farm were included as controls and casts prepared using the same procedure.

Results. At necropsy, all fish were in normal condition. There were no external signs of disease and all fish examined had moderate amounts of food in the gastrointestinal tract indicating normal feeding.

Five of the fish had a profound shortening of the swimbladder, while the remaining fish exhibited moderate shortening. The lengths of the malformed swimbladders ranged from 32 to 35 mm, while those of the 2 normal control fish measured 60 mm. The organ was dilated and confined to the anterior portion of the peritoneal cavity. Other abdominal organs (ventricle, pyloric caecae, liver) were pressed ventrocaudally by the misplaced swimbladder (Fig. 1). By careful dissection, it was possible to follow the pneumatic duct from its opening in the oesophagus via the ventro-lateral surface of the swimbladder to the posterior end of the swimbladder where it entered the organ. The course of the pneumatic duct and the shortening of the swimbladder were confirmed in the epoxy casts (Figs. 2 & 3). The rest of the gastrointestinal tract appeared normal.

Discussion. In the physostomatous Salmonidae the pneumatic duct is very short and wide and the swimbladder itself is of a simple type, i.e. not divided into chambers as in cyprinids. The swimbladder of the Atlantic salmon begins its development as a mass of mesodermal cells dorso-laterally on the posterior foregut when the embryo is approximately 7 mm long and 7 wk old (Hoar 1937). A lumen does not appear until the embryo is approximately 14 mm long and 18 wk old, when the epithelium of the foregut evaginates into the anterior end of the solid mass. This evagination occurs late in embryological life and continues further caudally and retroperitoneally (Rauther 1937, Harder 1975). The cavity expands caudally until it fills the entire mesodermal cell mass at hatching (21 mm and 25 wk), at which time the connection between the swimbladder and the gut has been reduced to a tube-like pneumatic duct. Gas starts to appear in the swimbladder when the yolk sac is almost spent (38 to 39 mm and 30 wk). The bladder extends the length of the body cavity at approximately 33 wk, when the fry are 31 to 32 mm long. The swimbladder has reached its fully developed proportions at 33 to 34 mm and approximately 35 wk.

In physoclistous fish, a patent pneumatic duct is present in embryonic life and for a short period after hatching, but is resorbed after the fish have inflated the organ by gulping air at the surface. If the fish are denied access to the surface during this period, inflation of the swimbladder will not take place, causing retarded development of the organ (Chatain 1989, Summerfeldt 1991, Kitajima et al. 1994). While present, the duct is long and runs along the ventral side of the main body of the swimbladder, entering the organ near the caudal pole in a manner similar to that described here in the salmon with swimbladder abnormality.

In the salmon with swimbladder malformation, the pneumatic duct is dramatically elongated and may be compressed between the bladder and body wall or other organs, creating a functionally physoclistous state where passage of gas is impossible.

Fig. 1. *Salmo salar*. Severely shortened and dilated swimbladder in Atlantic salmon smolt

Fig. 2. *Salmo salar*. Epoxy cast of malformed swimbladder *in situ* after hardening. The needle is inserted through the orifice of the pneumatic duct and the duct's course can be followed caudally on the ventral side of the swim-bladder

Fig. 3. *Salmo salar*. Abnormal (top) and normal swimbladder from Atlantic salmon smolts of similar size. Match = 42 mm

While the present investigation cannot illuminate the cause of the condition, several factors (i.e. hereditary, alimentary, physical and environmental factors) have been associated with retarded development or malformation of the swimbladder in other species. Leino et al. (1990) produced retarded swimbladder development in fathead minnow *Pimephales promelas* by exposing them to water with low pH and salinity. Correct temperature and light are important for normal swimbladder inflation in larval sea bass *Dicentrarchus labrax* (Johnson & Katavic 1984). Paperna (1978) considered swimbladder deformities to be associated with hereditary factors in sea bream *Sparus aurata*. Reduced levels of dietary polyunsaturated fatty acids also adversely affect survival and swimbladder development in sea bream larvae (Kitajima et al. 1994).

Another condition, similar clinically to the malformation described herein is 'swimbladder stress syndrome' (SBSS), where overinflation of the swimbladder results in alteration of buoyancy and equilibrium (Ferguson 1989). This condition is closely linked to high levels of catecholamines and is the result of simple overinflation with no observable anatomical alterations (Kolbeinshavn & Wallace 1985).

A possible explanation of the observed swimbladder malformation in farmed Atlantic salmon could be that the polarity of the swimbladder anlage is reversed early in embryonic life and consequently grows in the 'wrong' direction, i.e. cranially, pulling the pneumatic duct along with it. The abnormality could also represent an atavistic character, a reversion to a hypothetical prototype of teleost swimbladder (Harder 1975).

Marty et al. (1995) suggested that the topography of the developing foregut in walleye *Stizostedion vitreum*, in particular the relationship between the pneumatic duct and the bile duct in the undifferentiated foregut, is the underlying reason for the limited time-window for initial swimbladder inflammation in this physoclistous species. After differentiation of the foregut into stomach and intestine, inflation of the swimbladder by ingestion of air can no longer take place, due to lack of surfactant from the bile. If such a connection exists in physoclistous fish, it is likely that factors that influence the development of the foregut would also affect the development of the swimbladder in physostomatous species.

Although other abnormalities were not seen in the gastrointestinal tract of the sampled fish, underdeveloped ventricle, hypoplastic pyloric caecae and aberrant location of the liver are frequently reported by fish farmers to occur concurrently with swimbladder abnormalities. As these organs also develop from the foregut, a link between this abnormality and swimbladder malformation is possible. The reduced number of pyloric caecae reported in some affected fish cannot be explained by pressure atrophy from the overinflated and dislocated swimbladder and points to the possibility of a more generalized malformation of the foregut and its adnexa. The swimbladder of Atlantic salmon starts its development before hatching and becomes filled with gas around first feeding. As the swimbladder reaches its fully developed proportions at 33 to 34 mm, possible causes of the type of malformation described in this study should primarily be sought in the preceding time-period.

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