Swimbladder pathology during larval development of turbot (Scophthalmus maximus L.)

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Introduction
Studies by Bucke (1983), Cousin et al. (1986), Perez Benavente and Gatesoupe (1988), Nicolas et al. (1989), and Gatesoupe (1990) have shown that nutritional and infectious problems are the two main groups of causes of turbot (Scophthalmus maximus L.) larvae mass mortalities, although the specific pathogenic processes have not yet been explained. Histopathological studies (Cousin et al., 1986; Padros et al., 1993) carried out on turbot larvae showed that the main pathological features were observed in the digestive tract. Very few reports are available in the literature about other organs and systems. The swimbladder is known to be involved in pathological problems in other fish species (Johnson and Katavic, 1984; Bagarinao and Kungvankij, 1986; Chatain, 1987), but little is known about the role of the swimbladder in the pathology of turbot larvae. The aim of this paper is to describe the main alterations observed in turbot swimbladder related to development.

Materials and methods
Investigations were carried out on six different batches of turbot (S. maximus) larvae (totalling in number 1066) from a commercial hatchery (NW of Spain) throughout a period of 3 years. Larvae were reared in 7000-l tanks at a density of 7–12 larvae l⁻¹ in a flow-through system (T = 17.5 ± 2.5°C; salinity 33–36; dissolved O₂ = 95% saturation) and fed on enriched rotifers (Brachionus plicatilis) and Artemia. Every 2 to 5 d, larvae were taken randomly at different levels from the rearing tank and fixed in 10% buffered formalin after they were anaesthetized with MS-222 (Sandoz). Specimens were classified in developmental stages in accordance with Al-Maghzachi and Gibson (1984), and standard length and total length were measured. After fixation, specimens were embedded in 2-hydroxyethyl-glycol methacrylate, serially sectioned (2–3 μm) and stained with toluidine blue, haematoxylin-eosin, and Schiff periodic acid.

Results and discussion
Different rates of inflation and alterations (see Fig. 1) were observed in the six batches investigated, especially from stages 2 to 4. The structure of inflated swimbladders was similar to that described by Cousin and Baudin-Laurencin (1985). In contrast, non-inflated swimbladders could display a normal structure or a wide range of abnormalities. Normal non-inflated swimbladders were...
Figure 1. Percentage of inflation and abnormalities in the turbot larvae studied. INF = normal inflated swimbladders, NINF = normal non-inflated swimbladders, INV = non-inflated swimbladders invaded by bacteria, ABN = abnormal non-inflated swimbladders.

Figure 2. Turbot larvae. Bacterial aggregates (arrows) in the lumen of the swimbladder. G = gas gland, K = kidney tubules, N = notochord. Scale bar = 100 μm.
characterized by a lack of enlargement of the air chamber and the cubic shape of epithelial cells in the upper zone of the swimbladder. Frequently, large dilations of the pneumatic duct in the transitional zone between the bladder and the duct could also be seen. This expansion might be associated with a certain inability of the air bubbles to pass into the air chamber. Several authors (Spectorova and Doroshev, 1976; Al-Maghazachi and Gibson, 1984) have reported the relevance of the swimbladder inflation in the turbot. In contrast, our observations show that larvae without gas filling display neither special pathological problems nor any differences in development compared to larvae with inflated swimbladders. The fact that turbot swimbladder degenerates during stage 5 would suggest that its inflation during larval development might not have the same implications as in species such as the sea bass or the sea bream. Moreover, in our study, larvae with inflated swimbladders were usually seen on the surface rather than in the water column.

Abnormal swimbladders were characterized by hypertrophy of the gas gland and hyperplasia of the rete mirabile. Multiple capillaries surrounding the gas gland and abnormal disposition (cranial) of the rete mirabile were observed. These alterations in the swimbladder structure could interfere with the inflation process, although it can be argued that hyperplasia of the gas gland might be a proliferative response to the lack of inflation.

In some larvae, bacterial cells, free or in aggregates, were seen invading the swimbladder through the open pneumatic duct (Padros and Crespo, 1991). Microorganisms reached the air chamber (Fig. 2) and invaded the gas gland (Fig. 3) and neighbouring tissues, such as the kidney, producing intense necrosis. Occasionally, when the rete mirabile was also affected, microorganisms could be seen in the larval spleen. Bacterial cells observed in the live food and in the digestive tract, especially during stages 3 and 4, could be vehiculated to the swimbladder during the air-gulping process. It must be taken into account that the pneumatic duct can still be open until the end of stage 3. Bucke (1983) also described one case of bacterial invasion in turbot larvae, although the swimbladder was not reported to be involved.

Larvae with bacterial invasion of the swimbladder will probably not survive, particularly if we consider that the defensive system of the larvae is still immature at these stages.

We conclude that, although several swimbladder abnormalities preventing inflation could be present, inflation of the bladder might not be necessary for a correct development of the turbot, and that only swimbladder bacterial invasion seems to have a clear detrimental effect on the larvae.

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References


