

MASS MORTALITY OF EEL IN LAKE BALATON DUE TO *Anguillicola crassus* INFECTION

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In mid-July, 1991 an extensive eel kill occurred in Lake Balaton, Hungary. Thousands of dead eel floated on the surface of the water and an estimated 250 tonnes of eel succumbed within a month. No mortalities of other fish species in the Lake were observed. Dead and moribund eel measuring 60-80 cm in length were examined and their swimbladders were found to contain 30-50 specimens of adult nematode *A. crassus*. The lumina was filled with a cloudy fluid containing thousands of *Anguillicola* larvae, each bound by a membranous egg shell. Besides living adults, remnants of dead nematodes were found in the swimbladder and, in some cases, the ductus pneumaticus was filled both with living worms and parasite detritus. At this stage, the swimbladder wall was still transparent but had a red coloration caused by dilation of the capillaries. In some eel the swimbladder wall was thickened and haemorrhagic. By the end of August, the mortality dropped, but deaths still occurred. By September, only a small percent of the moribund eels harboured adult *Anguillicola* in the lumen of the swimbladder but in infected eel the swimbladder wall had thickened to 3 to 4 mm. The wall was haemorrhagic, and the lumen filled with a fluid containing remnants of dead worms. Among eel specimens caught by electroshocking, those with thickened swimbladders did not survive the stress of transfer to the laboratory. Those, however, with transparent, thin-walled swimbladders and containing fewer than 20 (10 to 20) *Anguillicola* specimens usually survived transportation. Within both the thickened and transparent thin-walled swimbladders, 3rd and 4th stage larvae could easily be detected in the wall. In addition to the swimbladder infection, encapsulated third stage larvae were found in the wall of the gut and on the abdominal serosa.

No inflammation or other signs indicating pathological changes in other organs were found. From these data we conclude that the extensive and selective fish mortalities in Lake Balaton were caused by *Anguillicola crassus*.

Anguillicola crassus is regarded as a moderately pathogenic parasite in natural waters and mortalities were, until now, recorded only in intensive rearing systems (Møllergaard, 1988; Hartmann, 1987).

Massive fish-kills caused by parasites in natural waters are rare. In natural systems, parasites characteristically cause periodic and sporadic fish-kills. However, in the case of introduction of a parasite to a new biotope, morbidity and sometimes mass mortality may occur. Such cases have been reported for *Nitschia sturionis*, a monogenean introduced into Lake Aral (Petrushevski and Schulman, 1958) and the introduction of *Gyrodactylus salaris* (Monogenea) into Norwegian waters (Johnsen and Jensen, 1986, 1988).

Anguillicola crassus was first noted in Lake Balaton in 1990 by Székely *et al.* (1991) who reported heavy infections in eel. They did not observe dead fish in the Lake, but found high mortality among collected, transported, and stored (stressed) specimens. It seems that over the past year, the numbers of parasites have increased to a level where they were capable of causing death.

In our opinion the following factors may have contributed to the rapid increase in parasite numbers and fatal result of the disease: (1) The introduction of *Anguillicola crassus* to a susceptible population of eel has occurred only recently. (2) The population density of the eel is very high because of difficulties in harvesting the fish in recent years. (3) Lake Balaton is a shallow lake, where the water temperature in summer may go up to

28°C. (4) Some portions of the lake are eutrophic, which resulted in the increase of the mass of copepodids, the intermediate hosts of *Anguillicola* species.

Summary

About 250 tonnes of eel in Lake Balaton, Hungary, died within a month because of serious *Anguillicola crassus* infection in the summer of 1991. Diseased fish were infected by 30 to 50 specimens of adult nematodes in the lumen of the swimbladder wall. In cases of heavy infection the swimbladder showed dilation of capillaries, haemorrhages and thickening of the wall. In more chronic cases, the haemorrhagic wall thickened to 3 – 4 mm and the swimbladder was filled by a fluid containing remnants of dead worms.

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