Vol. 15: 41–50, 1993 | DISEASES OF AQUATIC ORGANISMS Dis. aquat. Org. | Published February 2

Pathological and histopathological studies of the swimbladder of eels *Anguilla anguilla* infected by *Anguillicola crassus* (Nematoda: Dracunculoidea)

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ABSTRACT: The swimbladder lesions produced by *Anguillicola crassus* (Nematoda) infection, causing mass mortality among eels in Lake Balaton (Hungary) were studied by histological methods. In the initial phase of infection, no severe changes developed in the swimbladder wall despite the presence and intensive blood-sucking activity of worms that filled the lumen of the swimbladder. After disruption of the worms and primarily because of repeated reinfection by larvae, however, the wall of the swimbladder markedly thickened and showed degenerative, inflammatory and proliferative changes. Acute processes were characterized by epithelial hyperplasia and hyperaemia of the swimbladder wall. In cases of chronic swimbladder inflammation, oedema and hyperplasia of tissues of the tunica propria, submucosa and serosa could be observed, as well as granulomatoid infiltration by mononuclear cells and fibrinoid degeneration around the larvae.

INTRODUCTION

Anguillicola nematodes are common parasites of eels in the Pacific and Indian Oceans. To date, 5 species are known (Moravec & Taraschewski 1988), 2 of which infect European eel *Anguilla anguilla*. These species were brought into Europe by ill-considered introduction of Pacific eel species at the end of the 1970's. Paggi et al. (1982) were the first to describe an *Anguillicola* infection in Italy. They identified the parasites found as *A. australiensis* Johnston & Mawson, 1940, but Moravec & Taraschewski (1988) described this as a new species, *A. novaezelandiae*. The other introduced species, *A. crassus* Kuwahara et al. 1974, was recorded in Western Europe (Neumann 1985, van Banning et al. 1985, Peters & Hartmann 1986). The presence of this parasite in Hungary was first demonstrated in 1990 (Székely et al. 1991). The parasite, which was identified as *A. crassus*, caused mass mortality in Lake Balaton (Hungary) in summer 1991 (Molnár et al. 1991).

Since the introduction of *Anguillicola* spp. to Europe, numerous papers have been published on the occurrence and development of *Anguillicola* infection. However, very few have been concerned with the pathology of this nematodosis. The first report on its pathological effect was written in Japan by Yamaguti (1935), who observed that *A. globiceps* caused considerable thickening of the swimbladder wall in heavy infections. Studying the parasitic infections of introduced European eels in Japan, Egusa (1979) found that pond-cultured eels were heavily infected with *A. crassus*, and various pathological changes were produced in their swimbladders. Similarly, thickening of the swimbladder wall was found in *Anguillicola*-infected eels in Europe (van Willigen & Dekker 1989, Kamstra 1990, van Banning & Haenen 1990). Mortality due to *Anguillicola* infection has been reported by Sarti et al. (1985), Hartmann (1987), Liewes & Schaminee-Main (1987), Mellergaard (1988), Boon et al. (1989) and Molnár et al. (1991).

Few data are available on histopathological changes caused by *Anguillicola* species. Only van Banning & Haenen (1990) described histological changes in the swimbladder wall after natural infection with *A. crassus*, and Haenen et al. (1989) studied the pathological changes developing in the swimbladder wall after experimental infection with 3rd stage larvae.

Pathological and histological changes of the swimbladder, due to heavy infection by larval and adult stages of *Anguillicola crassus*, in eels from Lake Balaton are reported in this paper.

MATERIALS AND METHODS

Eels were collected by electrofishery from 5 sites in the 3 main regions of Lake Balaton, namely from the eutrophic western part which showed the most severe *Anguillicola* infection (Keszthely, Badacsony), from the midregion (Tihany), and from the oligotrophic eastern basin (Csopak, Balatonalmádi). The eels were immediately sent live to the laboratory and stored in aquaria until used for detailed investigations.

More than 600 eels measuring 20 to 86 cm in length were examined between 15 July and 31 December 1991. Some of them were processed by routine diagnostic methods but the majority were subjected to parasitological examinations. A total of 285 eel specimens were examined for bacteriological infection, and in some cases samples were also sent for toxicological and virological examinations.

Parasitological examination was aimed at determining the infection rate of the swimbladder and organs by adult and larval stages of *Anguillicola crassus*. Worms found in the lumen of the swimbladder were collected in saline solution and fixed in 10 % buffered formalin. Larval stages were collected only from the swimbladder walls; specimens occurring in other organs were only recorded. After evaluating the changes found in the inner organs, parts of the entire swimbladder (and occasionally the liver and the gut) were fixed in Bouin's solution or 10 % buffered formalin. Some of the fixed organs were sectioned in frozen state, while other materials were embedded in paraffin wax, cut into 4 µm thick sections which were then stained with haematoxylin and eosin. Connective tissue fibres and smooth muscle elements were studied in sections stained by van Gieson's and Farkas-Mallory's technique, while materials deposited among the connective tissue fibres were studied by Weigert's fibrin stain, periodic acid-Schiff (PAS) reaction, and polarization microscopy. The wall thickness of intact and pathologically changed swimbladders was measured with an ocular micrometer.

RESULTS

From 13 July until the end of August 1991, massive eel mortality occurred in Lake Balaton. The majority of deaths were observed in the western basin of the lake. The eels that died during that period weighed a total of 250 tons (Molnár et al. 1991). Although dead eels covered the surface of the lake in large masses, no mortality occurred among the numerous other fish species living in Lake Balaton. No moribund eels or eels showing clinical signs of disease could be observed. At the same time, numerous eel specimens caught by electrofishery failed to survive proper and expert transportation procedures. Besides paleness of the liver, only swimbladder lesions were found in the eels that died during transport. The majority of eels collected from the western basin of Lake Balaton, where the highest number of deaths occurred, showed substantial thickening of the swimbladder wall. Only about half of the pathologically altered swimbladders contained *Anguillicola crassus* worms in their lumen. In that area, the average number of worms found in the infected eel specimens was 30 in July and as many as 23 even in September. In some eels, 73 worms were found. In the less severely infected eastern basin, the prevalence of infection was nearly 100 % but was much less intense (6.3 worms per infected eel on the average) than in the western basin, and no thickening of the swimbladder wall was observed during the summer. (The dynamics of infection will be the subject of a forthcoming paper.) Under a stereomicroscope, from 1 to 230 3rd and 4th stage larvae were counted in the swimbladder wall. In some eels, migrating larvae were found in the intestinal wall and on serous membranes of the abdominal cavity.

No bacteria could be isolated from the organs and swimbladder lumen of freshly caught eels. However, the swimbladders of eels that arrived at the laboratory after long transportation or were kept there in overcrowded aquaria with a water temperature of more than 25 °C yielded a mixed bacterial flora consisting of *Aeromonas* and *Vibrio* species. In severe cases, these eels showed erythroderma typical of red disease as well.

In a less advanced stage of infection, severe swimbladder changes could not be observed even if the eel was infected by 20 to 50 worms, filling up to one-half the lumen of the swimbladder. In such cases, damage of the thin, transparent swimbladder was indicated by blood-filled mucosal blood vessels and a moderate enlargement of the gas glands in addition to the presence of worms. By stereomicroscopy, the walls of these swimbladders were found to contain from 1 to 20 3rd and 4th stage larvae.

In more advanced infection, the swimbladder wall became opaque and the serous membranes and the mucosa contained capillary haemorrhages. The lumen of the swimbladder was filled by numerous live worms; however, dead worms also occurred and released a fluid containing decomposing red blood cells into the swimbladder lumen. In some cases, the swimbladder

lumen was up to one-half or one-third, full with a turbid, blood-stained fluid containing dead worms and their tissue debris. Eggs and 2nd stage larvae were seen attached to the mucosal surface. In that phase, the lumen of the pneumatic duct, often markedly dilated and reddened because of the dilatation of its blood vessels, frequently held worms which were exiting the swimbladder or a scarlet-coloured fluid containing decomposing red blood cells and worm detritus which had oozed there from the swimbladder. The gas glands, normally reddish, glass pinhead sized paired organs situated ventrally in the swimbladder wall, had become markedly enlarged and hyperaemic.

At the stage of chronic parasitic swimbladder inflammation, gross inspection revealed that the lumen of the swimbladder had already become markedly narrower, and its wall more compact and stomach-like to the touch. In such cases, the swimbladder wall was substantially thickened, reaching 4 to 5 mm in diameter. It had become completely opaque and its lumen had narrowed. Upon incision, the outer part, comprising loose connective tissue and serous membrane, easily separated from the inner part including the muscle layer and the mucous membrane. In such cases, worms were only rarely found in the lumen but the mucous membrane was mostly hyperaemic. In addition to the swimbladder lesions, inflammation of the pneumatic duct was also typical.

Histological examinations

As described by Dorn (1961), the thin (maximum 0.5 mm thick) wall of the worm-free swimbladder consisted of 4 distinct layers (Fig. 1). The innermost layer facing the lumen was the mucosa; this was followed by a muscular layer, then a submucosa consisting of loose connective tissue, and covered by the serous membrane. Also, depending on the distention of the swimbladder at the time of fixation, the cuboidal epithelial cells of the mucosa may have been flattened or high cuboidal in shape. The tunica propria was very narrow, finely enmeshed with narrow capillaries, and contained only a few connective tissue elements. The narrow muscular layer consisted of smooth muscles running diagonally. The cell-deficient loose connective tissue of the submucosa contained a network of parallel-running collagen fibres. The serosa (tunica externa) covered the submucosa as a thin layer richer in cells. Its matrix was constituted by a split-up connective tissue more compact than that of the submucosa.

The walls of swimbladders containing worms but free from gross lesions were thin (Fig. 2); only the dilated and blood-filled mucosal vessels and the increased amount of supporting connective tissue elements of the tunica propria were striking. As a result of the latter, the internal wall of the swimbladder formed slight folds (Fig. 3). The tunica propria was infiltrated by mononuclear cells (lymphocytes, histiocytes and plasma cells). At the same time, the epithelium was free from conspicuous injuries despite the obvious blood-sucking activity of blood-filled worms found in the lumen. The muscle layer was characterized by blood-filled capillaries, but the presence of larvae could not be established. The submucosa contained cross-sections of 3rd and 4th stage larvae, around which no host response could be observed, only tissue injuries caused by larval migration.

The opacity of slightly thickened, worm-containing swimbladders was occasionally caused only by exudate and masses of eggs and larvae that attached to the internal surface of the swimbladder. In these cases, the most characteristic lesions were the repletion of mucosal blood vessels and the slight thickening of the mucous membrane (Fig. 4). The capillaries of the muscle layer were also more noticeable, and red blood cells

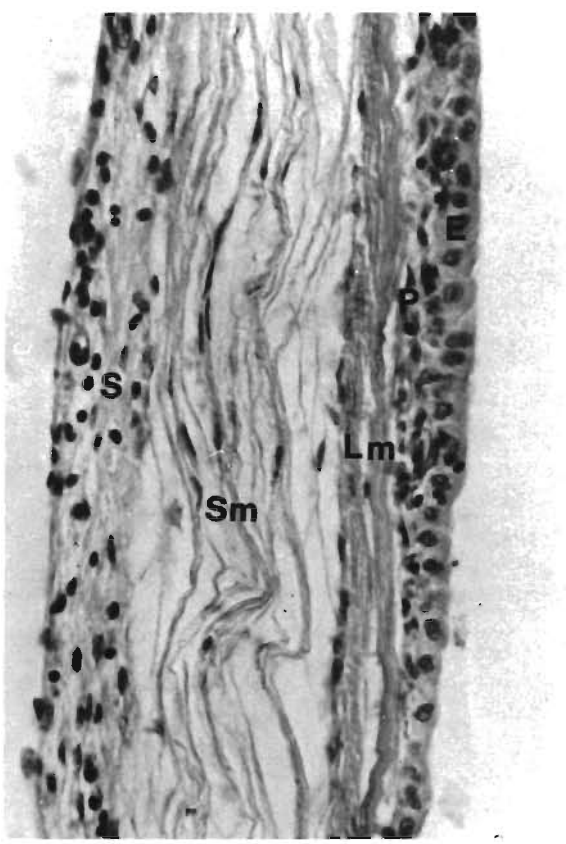

Fig. 1. *Anguilla anguilla*. Cross-section of the wall on an intact swimbladder. Under the simple cuboidal epithelium (E) note a very thin tunica propria (P) and a layer formed by some muscle cells (Lm). The submucosa (Sm) is constituted by loose connective tissue, and covered by the more compact serosa (S) towards the abdominal cavity. Haematoxylin and eosin, 500×

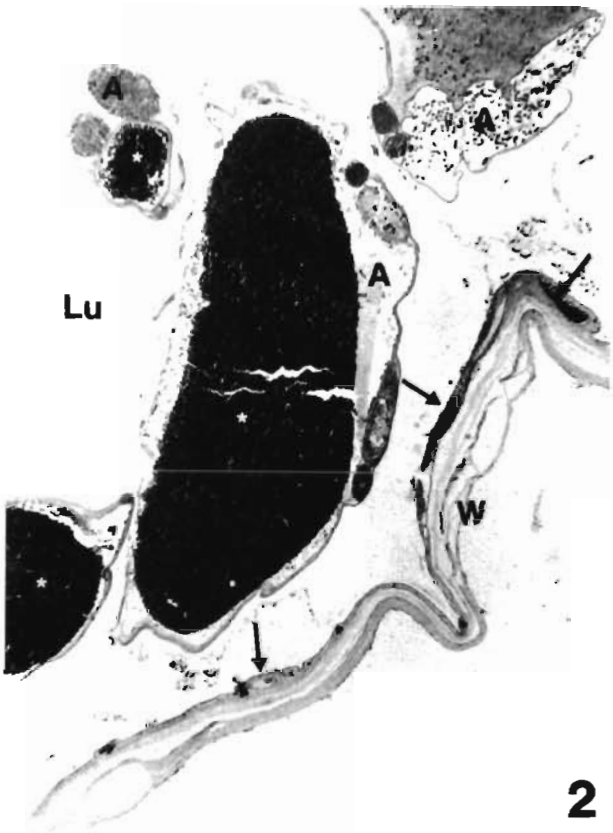

Fig. 2. *Anguilla anguilla* infected with *Anguillicola crassus*. Cross-section of a swimbladder infected by adult worms (A). The swimbladder wall (W) is only slightly thickened, the large blood vessels running in the tunica propria (arrows) are markedly dilated and filled with blood. The alimentary tract of worms present in the swimbladder lumen (Lu) contains large quantities of digested red blood cells (*). Haematoxylin and eosin, 50×

were seen freely among the muscle cells. The loose connective tissue of the submucosa was only slightly thicker than normal. The 3rd and 4th stage larvae situated in the connective tissue were not surrounded by a cellular host reaction. In cases when many, sometimes more than 100, larvae were found in the swimbladder wall under a dissecting microscope, dozens of larvae were also demonstrable in the loose connective tissue of the submucosa in histological preparations. At such times the connective tissue showed serous infiltration, and both the connective and the muscle tissue contained masses of red and white blood cells outside the blood path. However, no signs of host reaction were seen around these larvae either (Fig. 5). Along with the thickening of the swimbladder wall, the capillaries of the considerably enlarged gas glands became markedly dilated and were filled with blood cells.

In the case of the 1 to 1.5 mm thick swimbladders, hyperplasia of the mucous membrane was the most conspicuous finding. The epithelial layer covered a submucosa often showing polypous proliferation abundantly enmeshed with a connective tissue matrix, and containing dilated blood vessels and mononuclear cells as well as eosinophilic granulocytes. In the muscle layer, the amount of connective tissue elements increased; these elements were seen along the dilated blood vessels. In the submucosa, a large volume of exudate accumulated, which extended to large areas and was seen among the connective tissue cells in small lacunae subdivided by internal lamellae (Figs. 5 & 6). Around some of the larvae a slight proliferation of granulation tissue could already be observed (Fig. 6). In some cases, the connective tissue and the muscle layer contained red blood cells and mononuclear cells that had left the blood path. Parallel with the changes of the submucosa, the serosa also markedly thickened, its normally compact, split-up matrix gradually became loose, with serum accumulating among the fibres. The

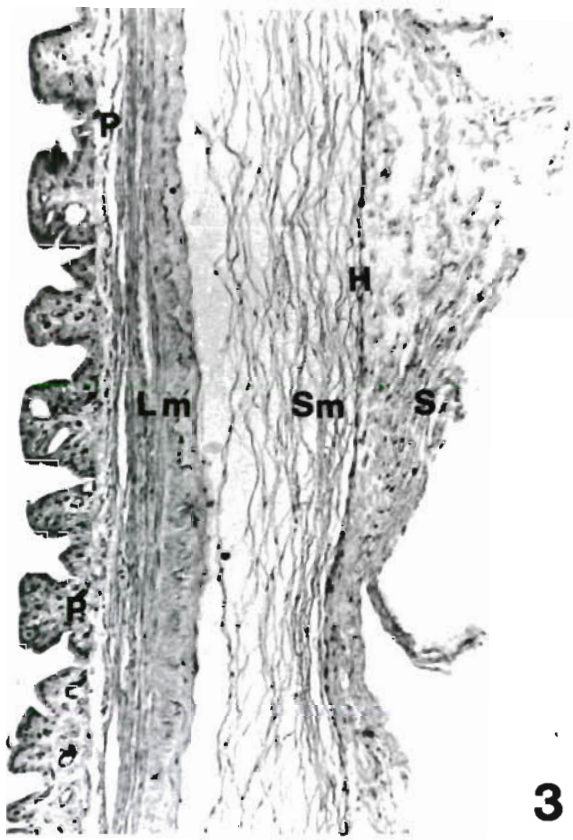

Fig. 3. *Anguilla anguilla* infected with *Anguillicola crassus*. Histological section of a slightly thickened swimbladder wall. The epithelium makes slight folds, while the tunica propria (P), the muscle layer (Lm) and the connective tissue layers (Sm: submucosa; S: serosa) have widened. At the border of the serosa and submucosa note a cell layer (H) with nuclei showing bright staining with haematoxylin. Haematoxylin and eosin, 100×

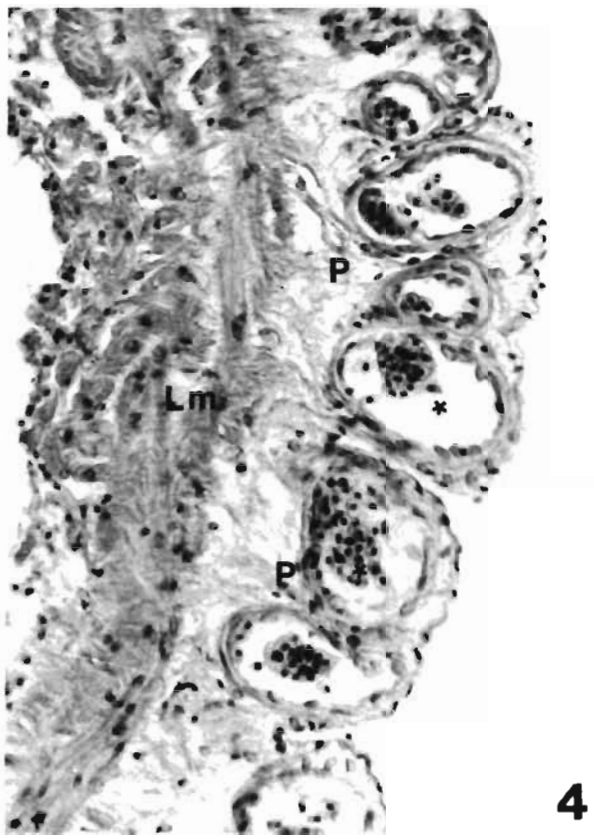

Fig. 4. *Anguilla anguilla* infected with *Anguillicola crassus*. Markedly dilated blood vessels (*) in the submucosa. Both the tunica propria (P) and the muscle layer (Lm) have thickened. Haematoxylin and eosin, 250×

matrix of the originally cell-deficient serosa became rich in young connective tissue cells and white blood cells. A layer of cells with nuclei staining brightly with haematoxylin appeared at the border of the submucosa and the serosa (Figs. 3, 5 & 9).

The 2.0 to 5.0 mm thickening of the swimbladder wall was caused mainly by the substantially increased volume of the submucosa; however, in these eels the mucosa and the serosa are also much thicker than usual (Table 1). In these specimens, the mucous membrane had deep folds (Fig. 7), covered by an epithelium showing hyperplasia and, here and there, metaplasia. The folds contained dilated vessels of thickened walls (Fig. 7). The connective tissue elements of the tunica propria multiplied, and the tissue contained dilated capillaries and red blood cells that had left the blood path. Occasionally, haemosiderin granules indicative of earlier haemorrhages, and eosinophilic granulocytes could also be detected. The muscle layer had become loose, the muscle fibres drew away from one another and were separated by dilated capillaries. In the fibrous connective tissue of the submucosa, the proliferation of a granulation tissue rich in blood vessels, consisting of fibroblasts, fibrocytes and connective tissue fibres, and containing mononuclear cells and eosinophilic granulocytes could be seen. Here and there, signs of fibrinoid degeneration (separation of fibrinoid material among the collagenic fibres, as well as swelling and degeneration of the fibres) could also

Table 1 *Anguilla anguilla* infected by *Anguillicola crassus*. Thickness of different layers of the swimbladder wall (μm) in infection-free eels and in eels affected with chronic anguillicolosis

	Control	Infected
Epithelium	20–30	260–320
Mucosa	90–100	900–1000
Submucosa	160–200	2000–3100
Serosa	90–100	1100–1900
Swimbladder wall, total	350–400	4000–6000

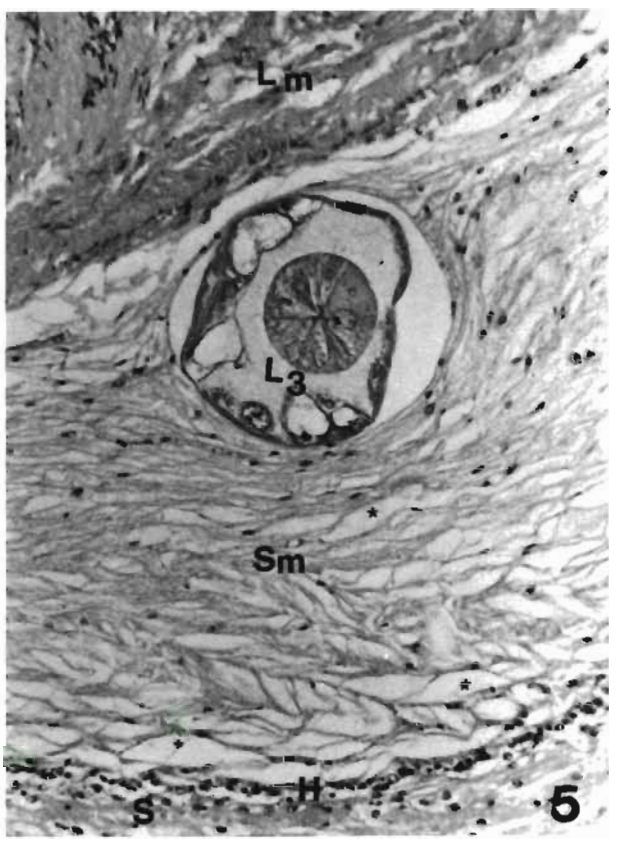

Fig. 5. *Anguilla anguilla* infected with *Anguillicola crassus*. Cross-section of a 3rd stage larva (L_3) in the submucosa (Sm), without signs of a cellular host reaction around it. The fibres of the submucosa have become loose and the lacunae thus formed (*) are filled with serum. Lm: muscle layer; S: serosa; H: layer rich in cells between the serosa and the submucosa. Haematoxylin and eosin, 250×

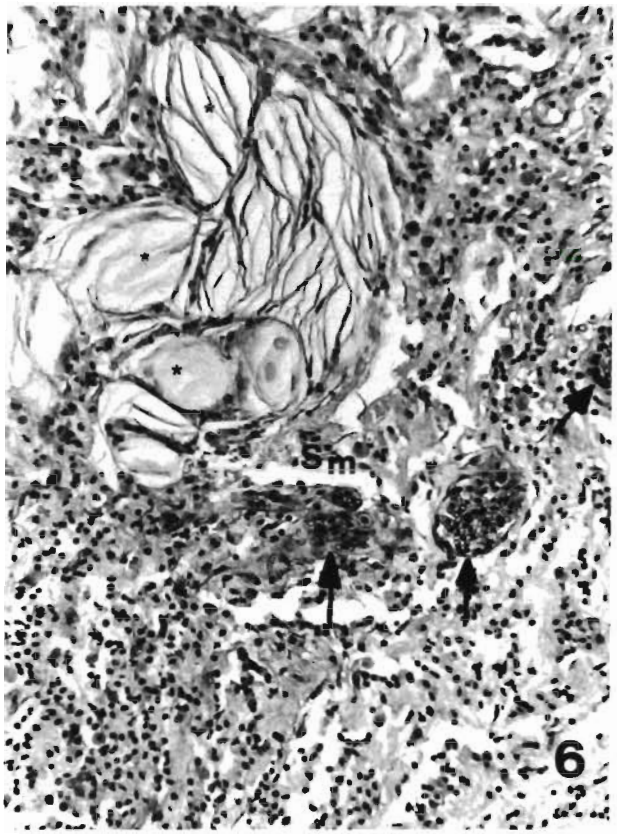

Fig. 6. *Anguilla anguilla* infected with *Anguillicola crassus*. Histological picture of inflammatory-oedematous lesions developing in the submucosa (Sm) as a result of larval migration. As a result of serum accumulation, the connective tissue fibres have drawn away from each other and formed extensive cisternae (*). The submucosa is markedly infiltrated by granulation tissue elements and mononuclear cells. In the tissues, note dilated blood vessels (arrows) and red blood cells outside the blood path. Deposition of a fibrinoid-type substance on and among the connective tissue fibres (fibrinoid degeneration) can be observed. Haematoxylin and eosin, 250×

be observed (Fig. 6), while in other places the separation of anisotropic crystals showing bundle- or nest-like arrangement was seen. The tissue contained numerous cross-sections of 3rd and 4th stage larvae, some of which were situated in intercellular mechanical injuries without any host reaction. Other larvae were surrounded by a granulation tissue, containing numerous mononuclear cells (Fig. 8), which was usually narrower at the worm's head part than around its body (in the direction in which the worm progresses). Some larvae were aggregated into, and kept confined in, well-isolated foci by the granulation tissue (Fig. 9). In an eel specimen infected with more than 200 migrating 3rd stage larvae, numerous larvae embedded in granulation tissue, surrounded by inflammatory cells including also eosinophilic granulocytes, were found – in addition to the swimbladder – on the peritoneum and in the intestinal wall (Fig. 10). Besides the damage done to the mucosa and submucosa, pronounced lesions were observed in the serosa. The space formed in places where the markedly thickened serosa was torn to shreds was filled with serum containing large numbers of intact and degenerated red blood cells and mononuclear cells. In that case, foci surrounded by a firm connective tissue capsule were left in the intestinal wall and on the serosae following the necrosis of larvae migrating in the tissues. A substance containing liquefied host cell debris and necrotic larvae, and showing intensive staining with eosin, was found in the middle of these foci. In some cases, when mucosal injury was also observed, from the lumen of the swimbladder 2nd stage larvae had penetrated the tunica propria and the submucosa. Around these larvae the intensive formation of granulation tissue was detectable (Fig. 11).

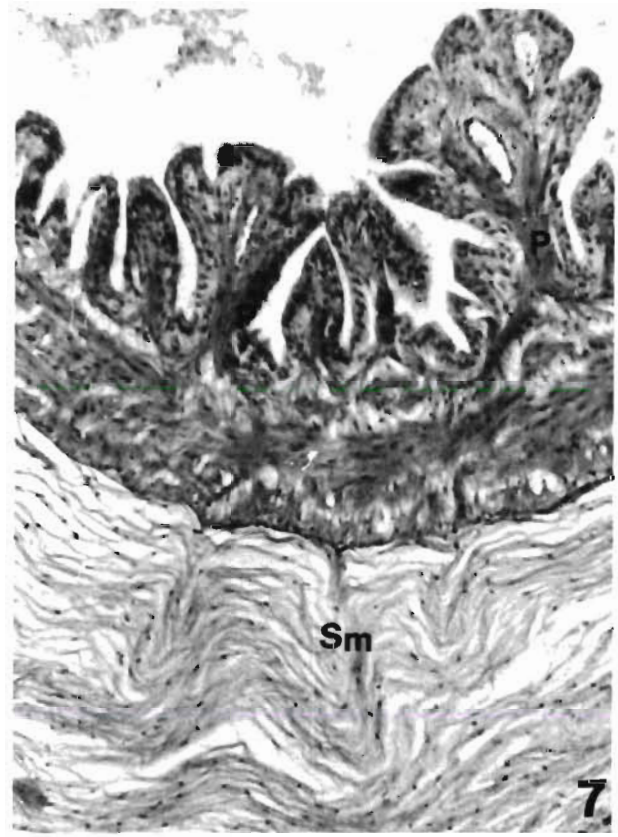

Fig. 7 *Anguilla anguilla* infected with *Anguillicola crassus*. Detail of a markedly hyperplastic swimbladder wall. The epithelium shows papillomatous changes: due to its substantially increased connective tissue matrix, the tunica propria (P) shows branch-like ramifications and is covered by a single epithelial layer composed of markedly elongated columnar epithelial cells. The submucosa (Sm) has widened and its fibres loosened. Haematoxylin and eosin, 250×

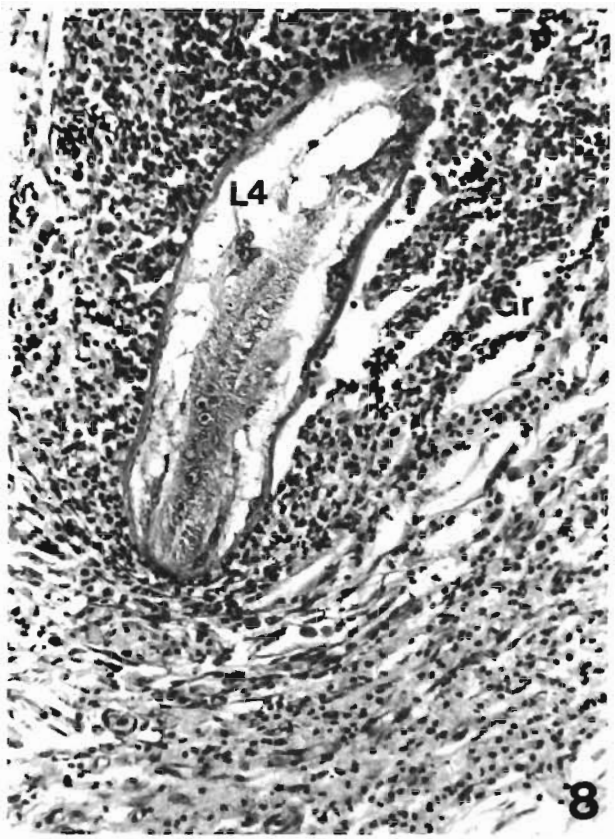

Fig. 8. *Anguilla anguilla* infected with *Anguillicola crassus*. Longitudinal section of the cephalic part of 4th stage larva (L_4) kept confined in the submucosa by granulation tissue and mononuclear cell infiltration. The granulation tissue (Gr) is thinner around the oral aperture than on the side of the larva. Haematoxylin and eosin, 250×

After the infection had ended, the wall of the swimbladder became transparent again and its thickness barely exceeded that typical of the preinfection state. No worms could be found in its lumen and no larvae were detectable in its wall. Previous infection was indicated only by yellowish pigment spots in the swimbladder wall, suggestive of earlier haemorrhages; these pigment spots could be histologically identified with the haemosiderin granules situated in the connective tissue. Less frequently, connective tissue foci containing necrotic larvae arranged like a string of pearls could be seen on the serosa of the already swimbladder or on the serosa covering the stomach (Fig. 12). Diffusely scattered melanomacrophages, or those situated in small islets, could often be observed in sections made from the wall of healed swimbladders; however, they accumulated in higher numbers around lymph vessels running near the gas glands.

DISCUSSION

Since the introduction of *Anguillicola crassus* to Europe, anguillicolosis has been a major problem in numerous countries. Despite the unquestionably demonstrated adverse effect of *A. crassus* infection, pathogenicity resulting in mass mortality of fish has not been reported in the literature so far, apart from the brief report of Molnár et al. (1991) dealing with the present case. This is obviously due to the fact that in the less intensive eel populations of cooler Western European waters, an infection similar in intensity to that observed by us could not develop. In the eel population of Lake Balaton, an *A. crassus* infection of unprecedentedly high prevalence and intensity occurred. This infection produced such severe pathomorphological changes in the swimbladder wall that they resulted in complete disfunction of the organ.

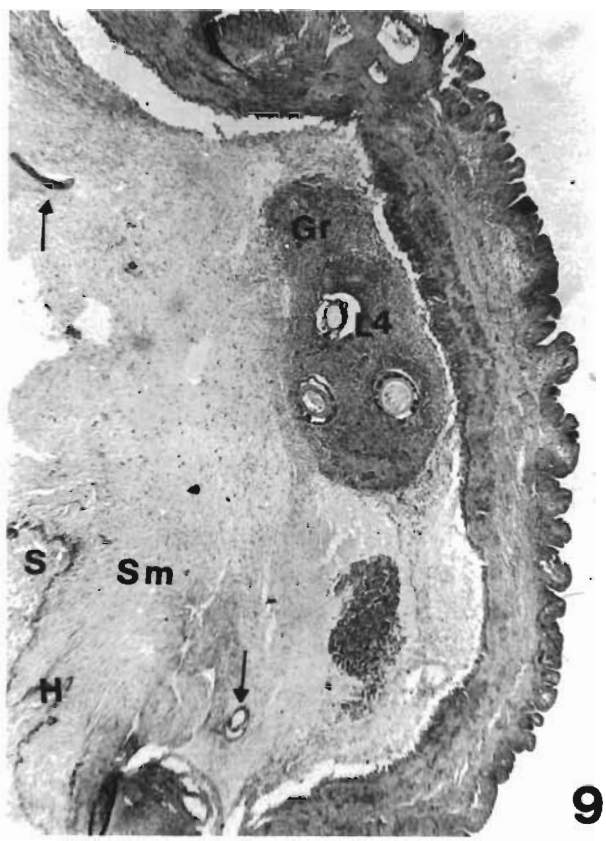

Fig. 9. *Anguilla anguilla* infected with *Anguillicola crassus*. Histological picture of a swimbladder severely infected by *Anguillicola* larvae (at low magnification). The expressly thickened mucosa casts folds; under it, in the extremely thickened submucosa the cross-sections of a 4th stage larva (L_4) enclosed in granulation tissue (Gr) can be seen. In the surrounding tissues free from mononuclear cells note the cross-sections of 3rd stage larvae (arrows) H: border of the serosa (S) and submucosa (Sm). Haematoxylin and eosin, 33×

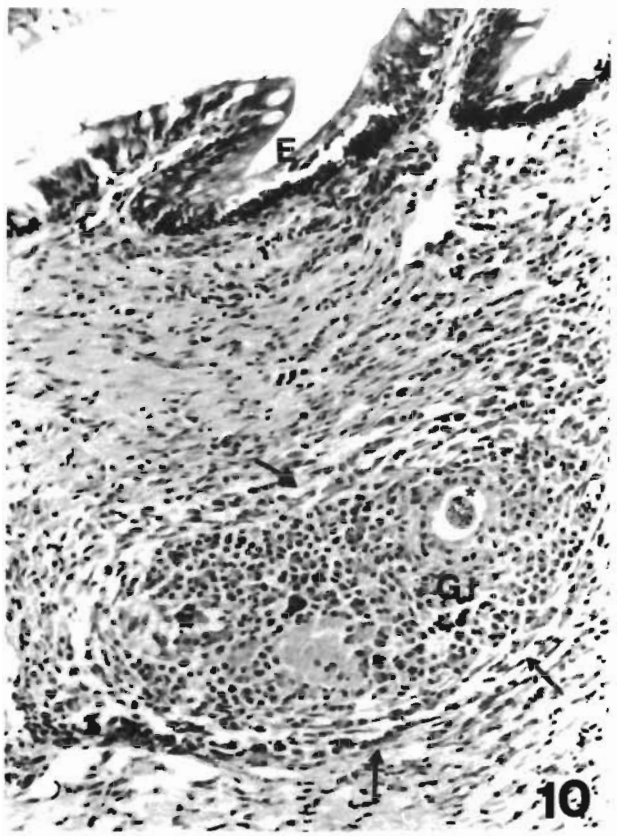

Fig. 10. *Anguilla anguilla* infected with *Anguillicola crassus*. Focal inflammatory reaction with eosinophilic cell infiltration, caused by migrating larvae, in the intestinal wall of an eel. The peripherally located 3rd stage larva (⋆) is surrounded by granulation tissue (Gr) containing many eosinophilic granulocytes. In the marginal part of the focus, young connective tissue cells form a capsule around the reaction (arrows). E: intestinal epithelium. Haematoxylin and eosin, 250×

Since the first report of Yamaguti (1935), several authors have reported the most characteristic sign of infection, i.e. thickening of the swimbladder wall (van Willigen & Dekker 1989, Kamstra 1990, van Banning & Haenen 1990). During our investigations, we also observed similar lesions as the most severe damage. As reported by van Banning & Haenen (1990), the thickening of the swimbladder wall and the associated pronounced histopathological lesions develop only at a later stage of infection. The results of our investigations failed to give essential proof of whether these lesions could be attributed to immunopathological processes resulting from the prolonged presence of the worms, to toxic products released from the dead worms, or rather to continuous reinfection by larvae. In our view, each of these processes, though to an extent varying by individual, may have played a role in the production of the pathomorphological lesions observed.

As opposed to chronic lesions of the swimbladder accompanied by thickening of its wall, which we stated to be the principal cause of the mortalities reported, it seems that infection does not lead directly to death even if the eels are colonized by large numbers of worms. A newly established infection does not cause pronounced lesions in the swimbladder wall despite regular blood sucking by the parasites. At the same time, from the studies of Sprengel & Lüchtenberg (1991) it is known that the presence of even a few worms is sufficient to markedly reduce the swimming speed of the eels, i.e. this presence is not neutral to the organism. These findings seem to be contradicted by the results of Möller et al. (1991) who failed to observe any difference in the body condition and liver somatic index of eels showing infection of varying severity. However, these findings should only be accepted with reservations, as among uninfected eels no distinction

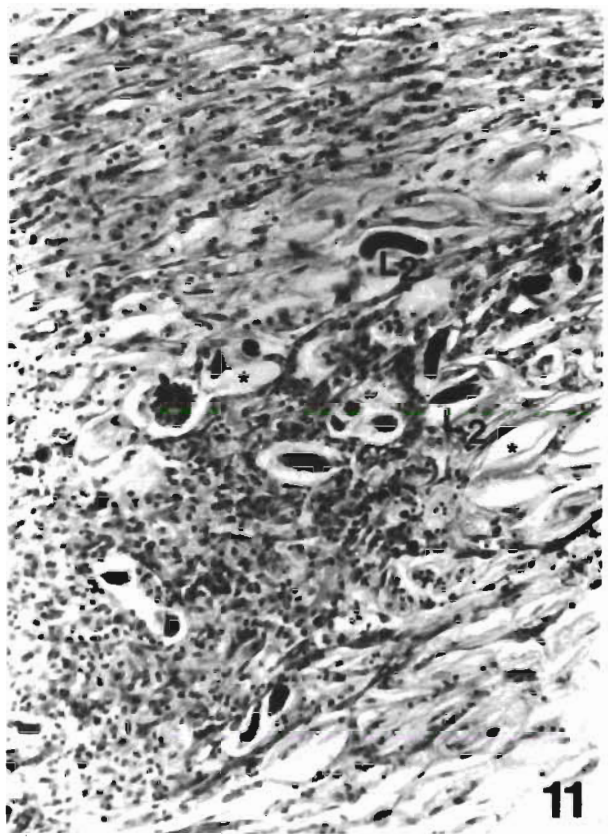

Fig. 11 *Anguilla anguilla* infected with *Anguillicola crassus*. Histological picture of the cellular reaction mounted against 2nd stage larvae (L_2) that penetrated deep into the tissues through gaps in the swimbladder epithelium. Larvae more intensely stained with haematoxylin are surrounded by granulation tissue containing numerous mononuclear cells and macrophages. Lacunae filled with exudate (⋆) can be observed in the vicinity of the tissue reaction. Haematoxylin and eosin, 250×

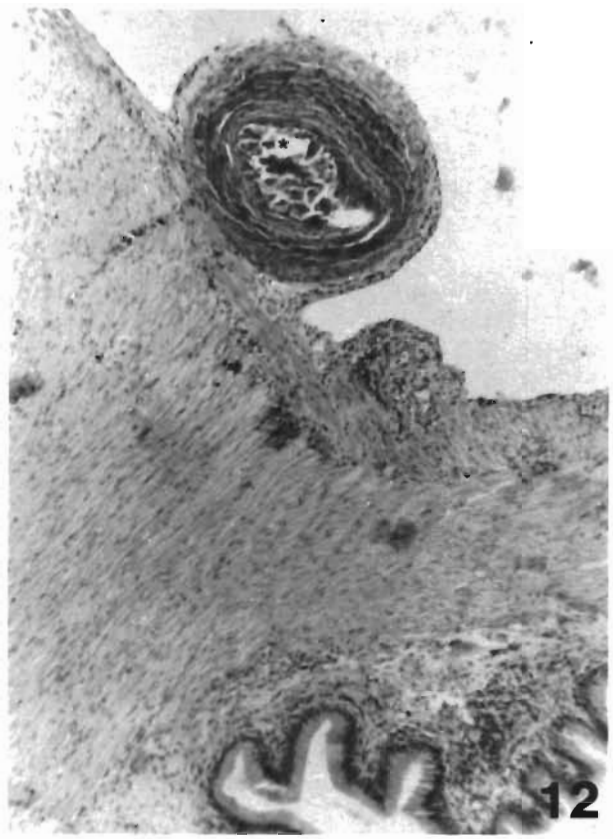

Fig. 12. *Anguilla anguilla* infected with *Anguillicola crassus*. Histological picture of a focus that developed on the intestinal serosa after the necrosis of migrating larvae. The focus is surrounded by a firm connective tissue capsule. Centrally, note the remnants of necrotized tissue and parasite elements (⋆) covered by a circularly arranged, multilayered connective tissue. Haematoxylin and eosin, 100×

was made between eels that had not yet become infected and those that had recovered from infection and possibly had a thickened swimbladder wall.

Our histological examinations indicate that, as first reaction to the colonization of worms, the blood vessels running in the tunica propria become dilated and passive congestive hyperaemia develops. At that stage, 3rd stage larvae that have penetrated the swimbladder are consistently situated in the loose connective tissue. It seems that these larvae do only mechanical damage to the swimbladder; the only traces of their migration are the microtraumas seen in the loose connective tissue. In more chronic cases, the connective tissue proliferates and its fibres increase in number and draw away from one other, with serum accumulating between them; as a result, the swimbladder wall thickens. In the case of more chronic or severe larval invasion, different inflammatory cells (lymphocytes, histiocytes and granulocytes) and red blood cells that have left the blood path appear among the connective tissue fibres; the blood cells may be associated with the active blood consumption of 4th stage larvae. In that case the host response manifests itself in lympho-histiocytic infiltration and the formation of granulation tissue. The masses of mononuclear cells grouped around the larvae can probably arrest the development of only a small number of larvae, as necrotized specimens were rarely seen among the encapsulated larvae. The possible release of worms from the granulation tissue is indicated by the observation of consistently fewer round cells near the cephalic end of the larvae than at other points of the body. At the same time, the possibility of successful host response is suggested by the findings in some eels that the intestinal wall contained completely encapsulated larvae and that the larvae had already disappeared from the swimbladders, which had markedly thickened walls but showed no signs of inflammation. The large lacunae situated in the loose connective tissue and filled with serum, the fibrinoid degeneration of the connective tissue fibres and the fibrinoid-type material deposited in their spaces suggest the possibility of a humoral host reaction (probably the formation of immune complexes). Interestingly, larvae were never demonstrable from the muscle tissue. Penetration of 4th stage larvae into the lumen must obviously be a short process.

While the changes occurring in the connective tissue are explained first by larval reinfection, the lesions observed in the mucous membrane can clearly be attributed to the worms living in the swimbladder lumen. Repeated blood sucking by live worms probably contributes to the development of some of the epithelial lesions, dilatation of the blood vessels of the tunica propria, proliferation of the connective tissue elements, and the appearance of blood cells. More severe pathomorphological lesions, including epithelial metaplasia, intensive inflammation of the mucosa, and the development of haemorrhages, can be explained rather by the toxic effect of larvae released from necrotized worms completing their life cycle and of half-digested blood elements. Liver injury observed as an accessory sign, manifesting itself in the pale colour of that organ also reported by Kamstra (1990), is attributed also to the toxic effect of substances (worm detritus and decomposing red blood cells) released from the disrupting worms, decomposing in the lumen and then absorbed. In intensive cases, bacterial complications cannot be ruled out, as bacteria can gain entry through the tissue detritus and blood-stained fluid stagnant in the swimbladder and flowing into the oesophagus through the pneumatic duct. We assume that severe cases, similar to those reported by Banning & Haenen (1990) in which 2nd stage larvae penetrated deep into the inflamed mucosa during a process considered 'unnatural', result from such bacterial complications.

Our results fit in well with what has been published in the special literature on anguillicolosis so far. *Anguillicola crassus*, a parasite living in a biological equilibrium with eels of the Pacific Ocean, becomes dominant in the new habitat, turning the scale (i.e. the host-parasite balance) in favour of the parasite. Intensive infections give rise to such pathological swimbladder lesions which indispose the fish for tolerating stress factors from the outside world. In our case, the unusually long-lasting high water temperature experienced in summer 1991 in Lake Balaton, one of Europe's shallowest lakes, must have contributed to the development of mass mortality among eels.

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Responsible Subject Editor: W. Körting, Hannover, Germany

Manuscript first received: June 22, 1992
Revised version accepted: August 28, 1992