Dynamics of·Anguillicola crassus (Nematoda: Dracunculoidea) infection in eels of Lake Balaton, Hungary

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Abstract. Following the introduction of Anguillicola crassus into Lake Balaton, by 1991 the entire eel population became infected. At the same time, marked differences existed in the prevalence and intensity of infection between different areas of the lake. The highest prevalence values occurred in the eastern basin less densely populated with eels, while in the western basin a large proportion of the fish were free of infection. Helminth-free status accompanied by thickening of the swimbladder wall developed after intensive infections. In 1991, eel mortality could be observed only in the western basin. In 1992, the number of eels with swimbladders having a thickened wall but not containing helminths increased also in the central and eastern areas of the lake. Parallel to this, a mortality less expressed than the one in 1991 occurred in the central part of the lake. By 1993, a host-parasite equilibrium had become established in Lake Balaton.

Since Anguillicola crassus was introduced into Europe at the end of the 1980s, numerous papers have dealt with its prevalence (Paggi et al. 1982, Neumann 1985, Sarti et al. 1985, Peters and Hartmann 1986, Hartmann 1987, Taraschewski et al. 1987, Dupont and Petter 1988, Belpaire et al. 1989, Koops and Hartmann 1989, Kennedy and Fitch 1990, Køie 1991, Székely et al. 1991, Moravec 1992), life cycle (De Charleroy et al. 1990, Haenen and van Banning 1991, Höglund and Thomas 1992, Thomas and Ollevier 1992), seasonal occurrence (van Willigen and Dekker 1989), pathogenic effect exerted on the host (Mellergaard 1988, Boon et al. 1989, 1990a,b,c, Haenen et al. 1989, van Banning and Haenen 1990, Molnár et al. 1991, Möller et al. 1991, Sprengel and Lüchtenberg 1991, Höglund et al. 1992), the mechanical injuries caused by it in the swimbladder wall (Lieves and Schaminee-Main 1987, Kamstra 1990), and the histopathological changes induced by the parasite (Haenen et al. 1989, van Banning and Haenen 1990, Molnár et al. 1993). Although many of the above-listed authors have touched upon the dynamics of the parasite's occurrence, detailed data can be found only in the works of van Willigen and Dekker (1989), Thomas and Ollevier (1992), and Höglund and Andersson (1993).

The literature listed above indicates that anguillicolosis is a relatively well-studied parasitosis. The studies conducted by us were justified by the fact that in 1991 and 1992 A. crassus infection caused mass mortality among eels in Lake Balaton. This called for investigations to determine the epizootiological, ecological and environmental factors that had contributed to such a severe manifestation of the disease in Lake Balaton.

During our three-year investigations, we studied the prevalence and intensity of A. crassus infection as well as the correlation between helminth infection and the severity of swimbladder lesions.

MATERIALS AND METHODS

The period of study lasted from August 1991 to November 1993. Eels (size > 20 cm) were collected from three different habitats of the lake (Fig. 1), from the eutrophicated western basin (Keszthely, Badacsony), the less eutrophicated central region (Udvari, Tihany, Csopak), and from the eastern region of oligotrophic character. According to fishery data, the population density of eels is about nine times higher in the eutrophicated western basin than in the eastern one.

We collected a certain proportion of the eels (primarily in the central and eastern region) with our own electrofishery devices, while the majority of eels derived from the Keszthely region came from "fishermen's catch". For technical reasons, in the first half of 1992 we were unable to collect fish from the eastern basin. The size of fish derived from the Keszthely region usually exceeded 60 cm.

Full dissection of eels for anguillicolosis was initiated on 1 September 1991 and involved a total of 726 fish. Prior to that time, 91 eel specimens had been examined without studying larval infection. Some of the tables include the data of all eels examined, while others contain only the results of the full-scale studies.

As far as possible, within the fishing season the eels were collected from the different sampling places at one-month
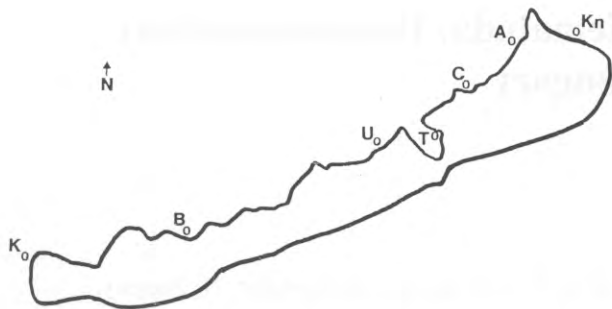
Fig. 1. Sampling places in Lake Balaton in the years 1991 through 1993. Sampling places: Western basin: K=Keszthely, B=Badacsony; Central basin: U=Udvari, T=Tihany, C=Csopak; Eastern basin: A=Almádi, Kn=Kenese.

intervals and transported to the laboratory alive. The fish kept in our aquaria were always examined within 3 days. As far as possible, at least 20 specimens were dissected on each occasion. Dissection was aimed primarily at studying the condition of the swimbladder; however, some specimens were subjected to full parasitological dissection. The fish were anaesthetized in MS 222 solution, then, after decapitation and opening the abdominal cavity, the swimbladder and intestines were removed. During the examination of the swimbladder, its size, wall thickness, number and size of adults present in its cavity, and the number of 3rd and 4th stage larvae found in the wall were recorded. In addition, the quantity of fluid present in the cavity and the number of parasitic nodules in the wall, as well as the degree of pigmentation, if any, were also put on file.

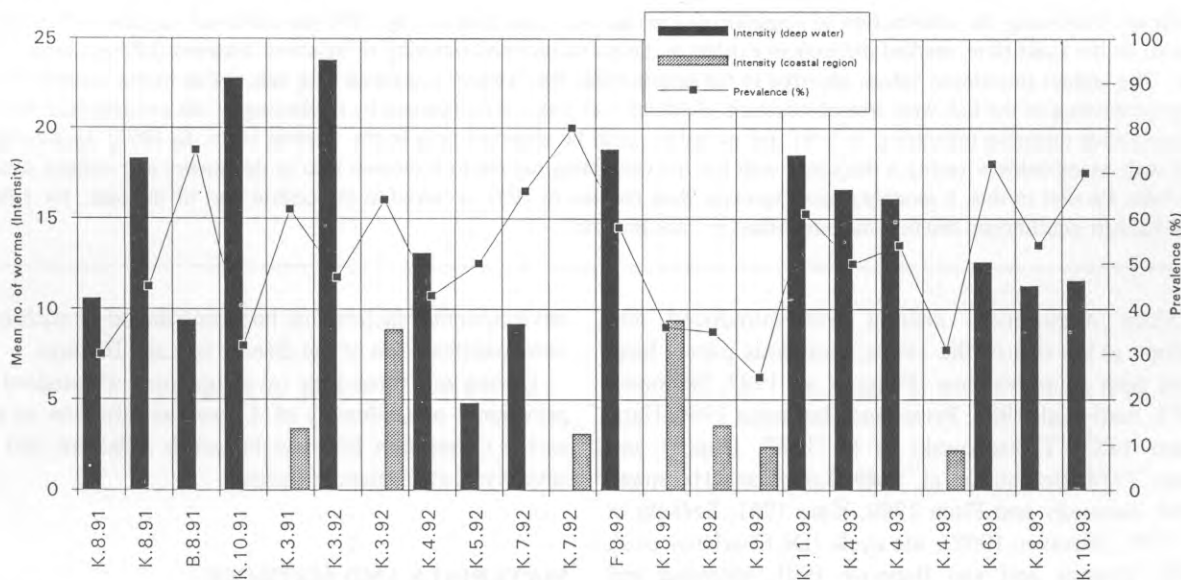During the dissections, a swimbladder wall less than 1 mm

Fig. 2. Prevalence and intensity of *Anguillicola crassus* infection in the western basin of Lake Balaton from August 1991 to October 1993. Initials of the places (K=Keszthely, B=Badacsony, F=Fonyód) and dates of collection.
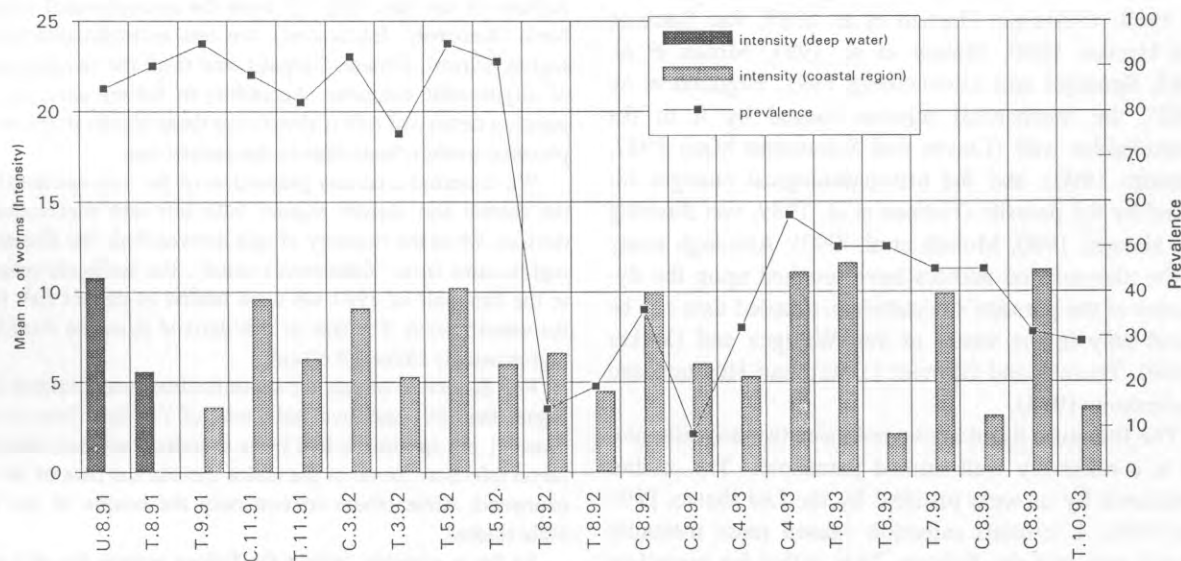

Fig. 3. Prevalence and intensity of *Anguillicola crassus* infection in the central basin of Lake Balaton from August 1991 to October 1993. Initials of the places (T=Tihany, C=Csopak, U=Udvari) and dates (month, year) of collection.
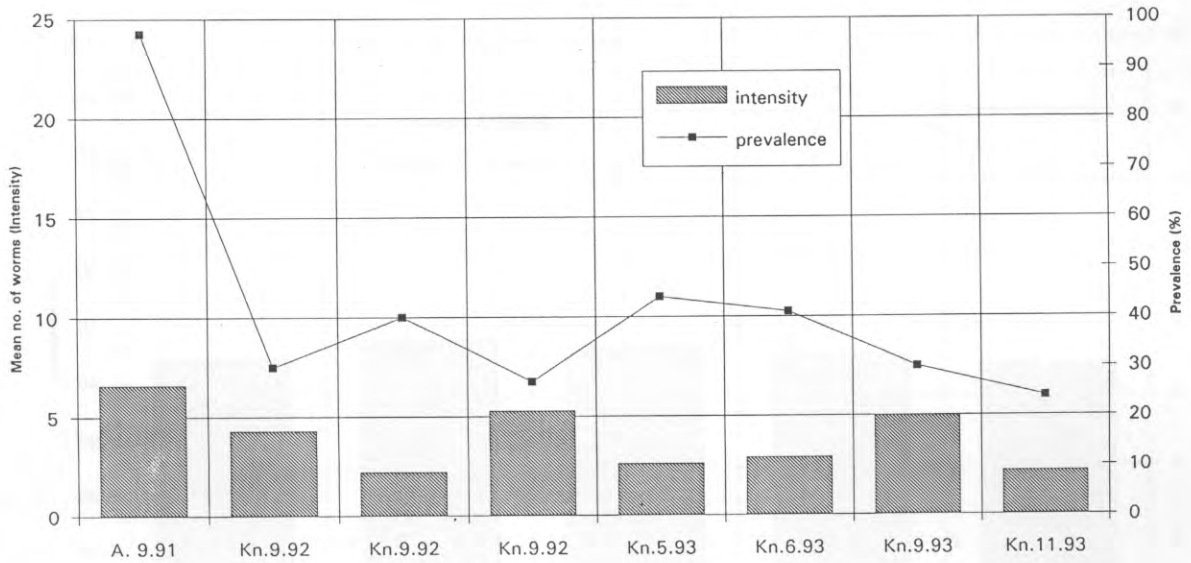
Fig. 4. Prevalence and intensity of *Anguillicola crassus* infection in the eastern basin of Lake Balaton from September 1991 to November 1993. Initials of the places (A=Almádi, Kn=Kenese) and dates (month, year) of collection.
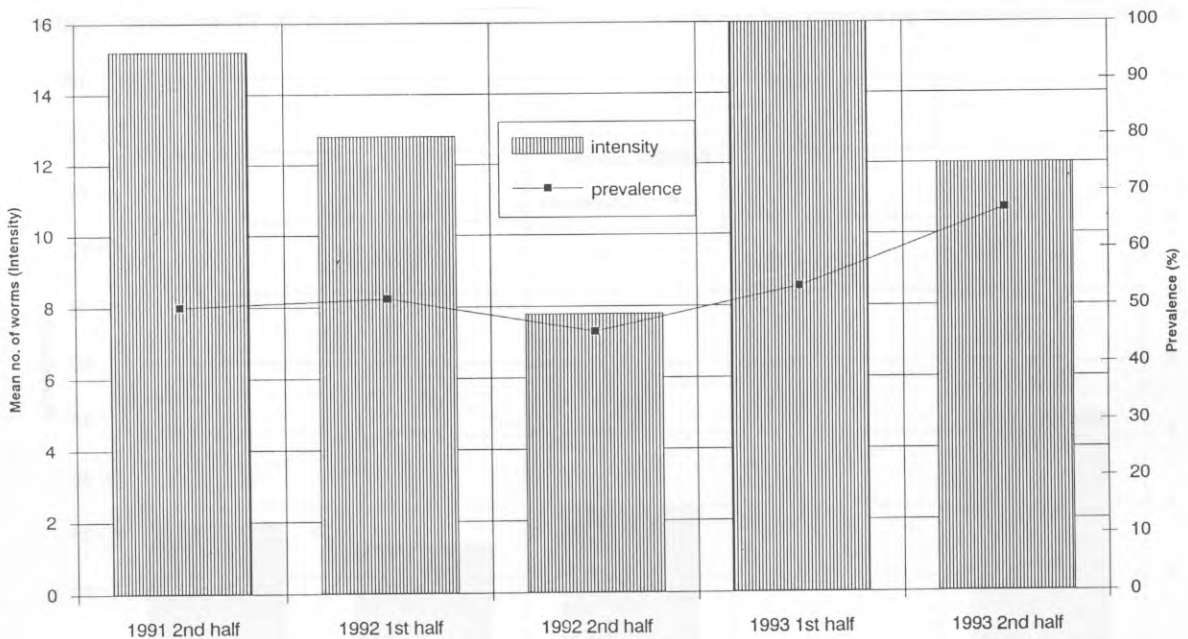

Fig. 5. Half-year summaries of the intensity and prevalence of *Anguillicola crassus* infection in the Keszthely (west) region.

thick was considered thin even if it exhibited signs of a passed-off infection (smoke-like opacity, parasitic nodules, pigmentation, minor haemorrhages, etc.). Swimbladders with a 1–3 mm thick wall were assigned to the category of "moderate severe" lesions, while eel specimens showing a swimbladder wall thickness exceeding 3 mm were considered severely affected.

After opening the swimbladder, the adults were placed in saline solution and counted in a Petri dish. The thickness of the swimbladder wall was evaluated after cutting open the swimbladder and comparing its wall thickness to glass slides of different thickness. If the swimbladder wall was thin, the larvae present in it were counted under a stereomicroscope,

while thicker, less transparent walls were examined either under stereomicroscope after fixing them between two glass slides, or with a light microscope in 100-fold magnification.

In addition to the swimbladder, the gut was inspected in all cases and the number of parasitic nodules caused by *Anguillicola* larvae was recorded.

RESULTS

All the 726 eel specimens collected from different areas of Lake Balaton and dissected in the laboratory
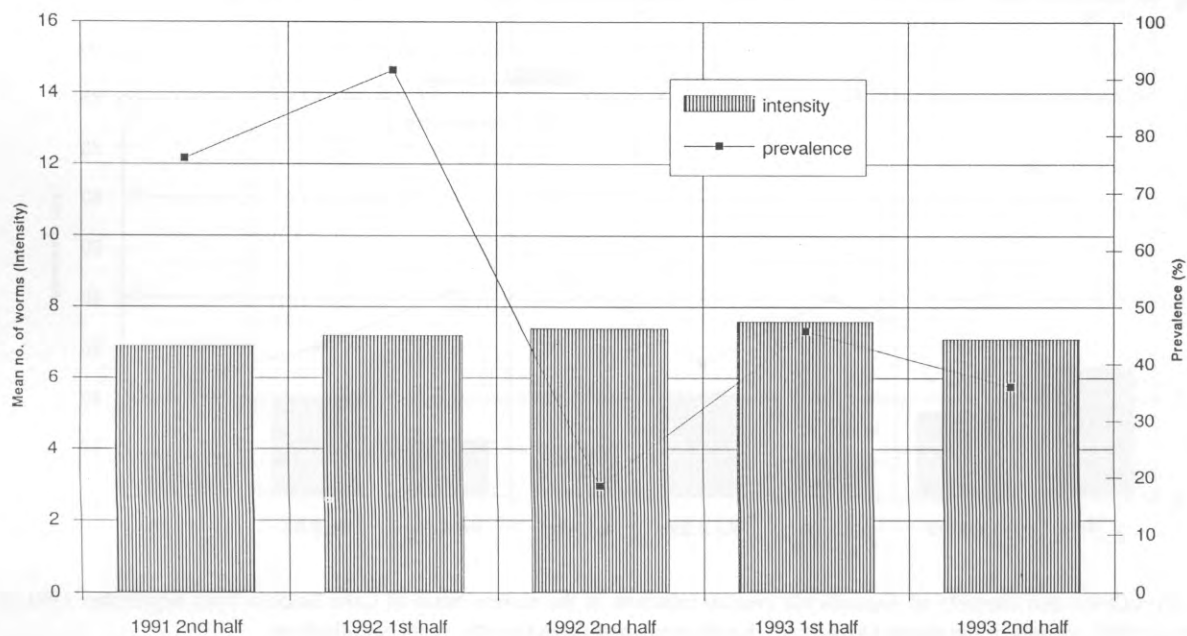
Fig. 6. Half-year summaries of the intensity and prevalence of *Anguillicola crassus* infection in the Tihany (central) region.
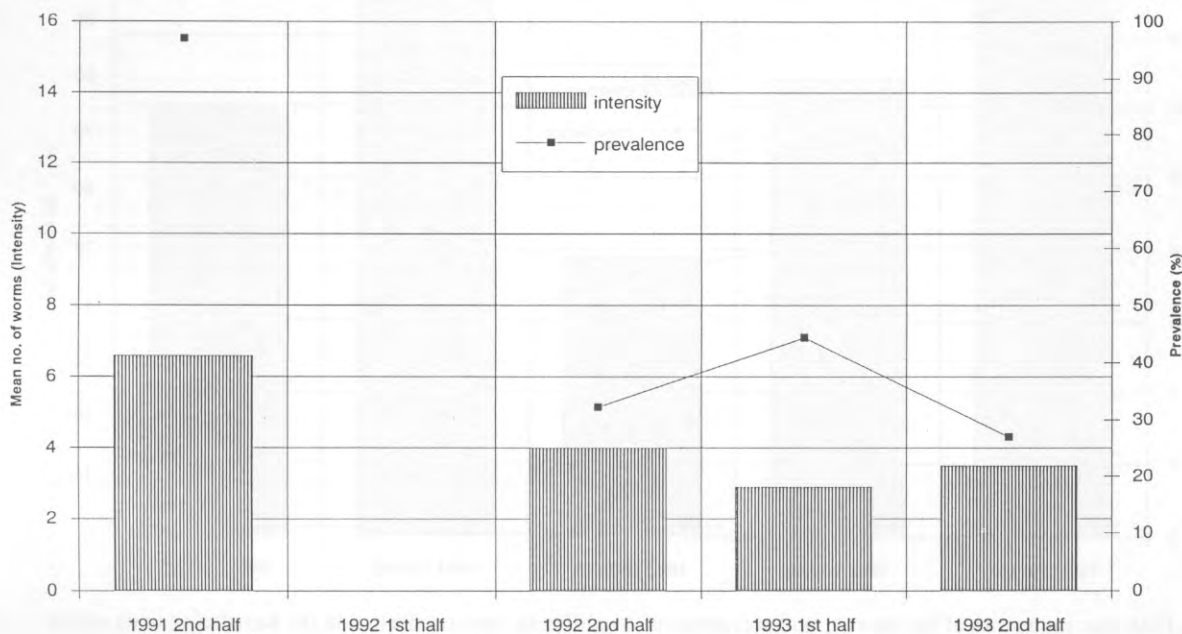

Fig. 7. Half-year summaries of the intensity and prevalence of *Anguillicola crassus* infection in the Kenese (east) region.

showed signs of a currently existing or previous infection by *A. crassus*. Of them, 367 eels were infected by adults parasitic in the swimbladder lumen, while the remaining 359 fish were free from such forms. As 3rd and 4th stage larvae were found in the swimbladder wall of 379 eels, and such larvae were sometimes present also in fish free from adults parasitizing the swimbladder lumen, the number of eels free from both larvae and adult stages was 224. The fish free from both adults and larvae also showed signs of a passed-off infection, and

it was precisely this group which yielded the specimens exhibiting the most severe pathological signs and having swimbladders with a markedly thickened wall. At the same time, a slight (less than 1 mm) wall thickening, smoke-like opacity, minor haemorrhages, parasitic nodules and pigmentation were observed also in the swimbladder of some eel specimens which had a thin-walled swimbladder.

As regards the prevalence and intensity of infection by adult *A. crassus*, major differences occurred among
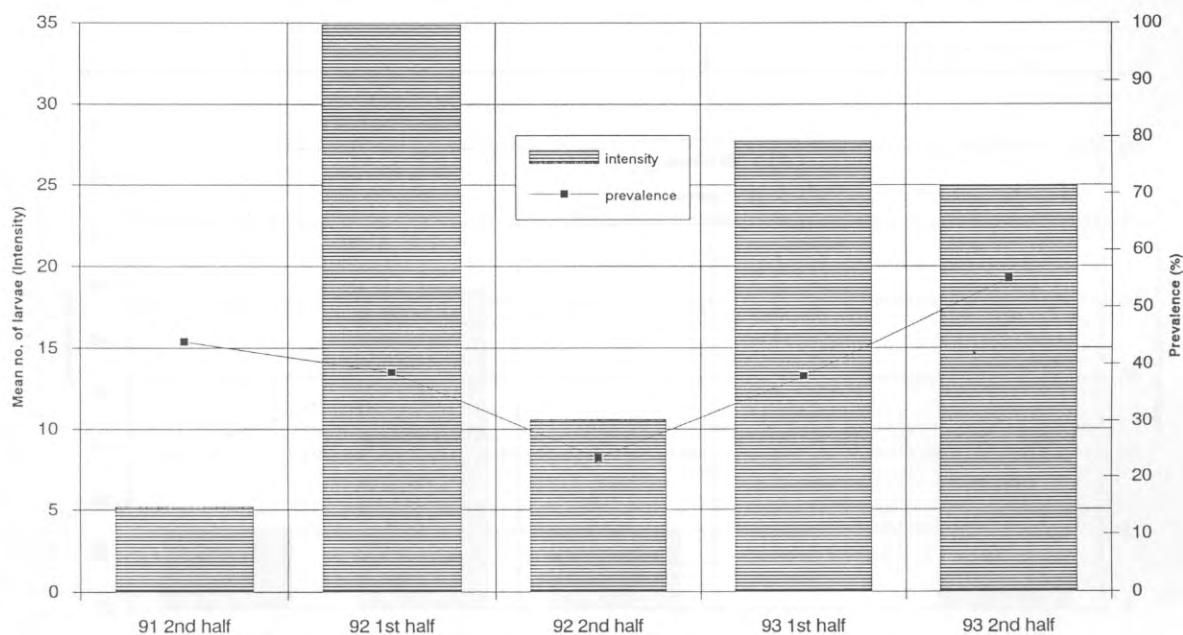
Fig. 8. Half-year summaries of the intensity and prevalence of *A. crassus* larval infection of eels in the Keszthely (west) region.
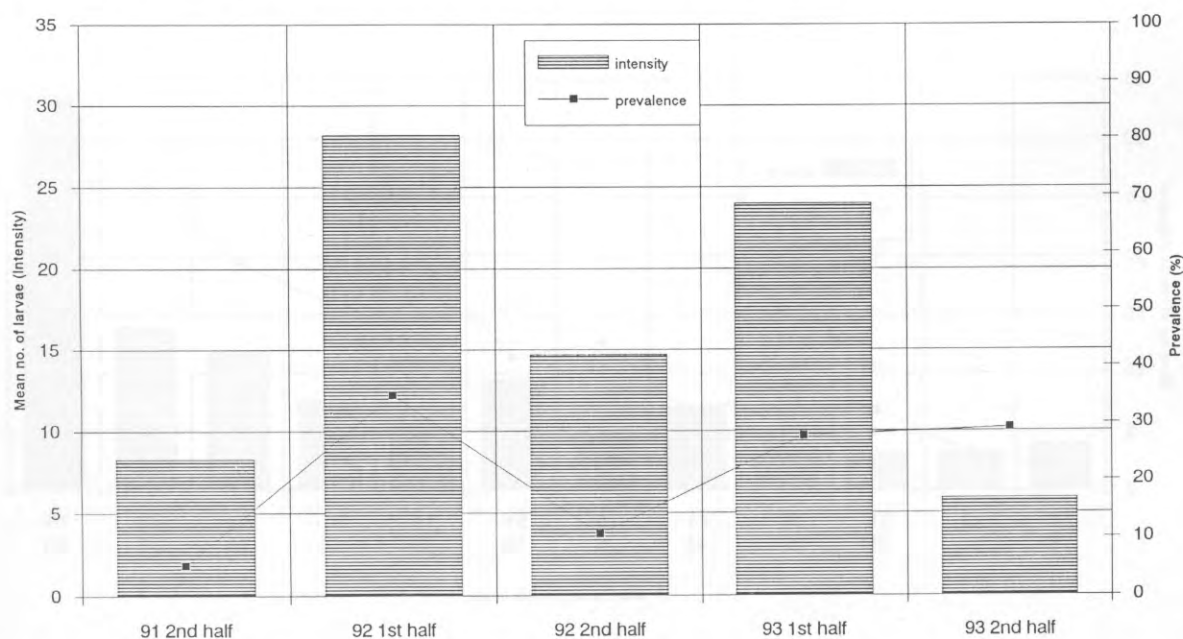

Fig. 9. Half-year summaries of the intensity and prevalence of *A. crassus* larval infection of eels in the Tihany (central) region.

the three different habitats of Lake Balaton, i.e. the western region (the Keszthely Basin), the central area (Tihany region) and the eastern area (Kenese region) (Figs. 2, 3 and 4). This difference was the most expressed in the year 1991, and by 1993 it had markedly decreased.

From 1991 to the end of 1993, the prevalence of infection varied between 25 and 80% in the western basin (Fig. 2), and no appreciable difference could be demonstrated between the first and last months of the year

(Fig. 5). Around Tihany (in the central region), the prevalence of infection by adult helminths varied between 75 and 95% in 1991 and in the first half of 1992, but it dropped to 8–36% in the second half of 1992 and did not exceed 57% in 1993 either (Fig. 3). Similar trends could be observed in the eastern region, where the prevalence of infection was 97% in 1991 and declined to 24–44% subsequently (Fig. 4). In these habitats, half-year summaries of prevalence indicated a similar tendency (Figs. 6 and 7).
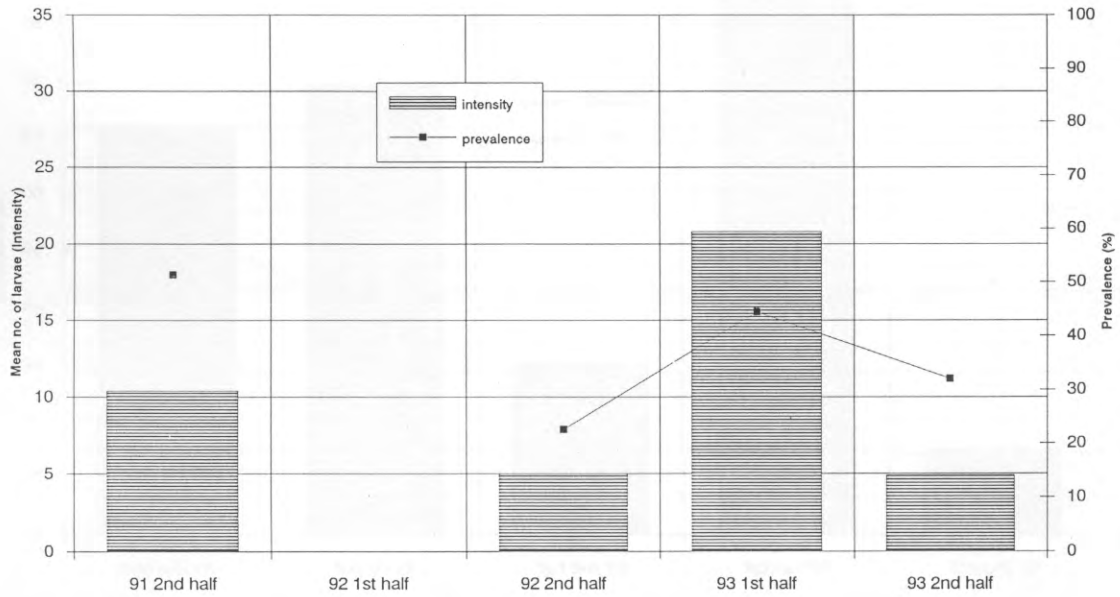
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Fig. 10. Half-year summaries of the intensity and prevalence of *A. crassus* larval infection of eels in the Kenese (east) region.
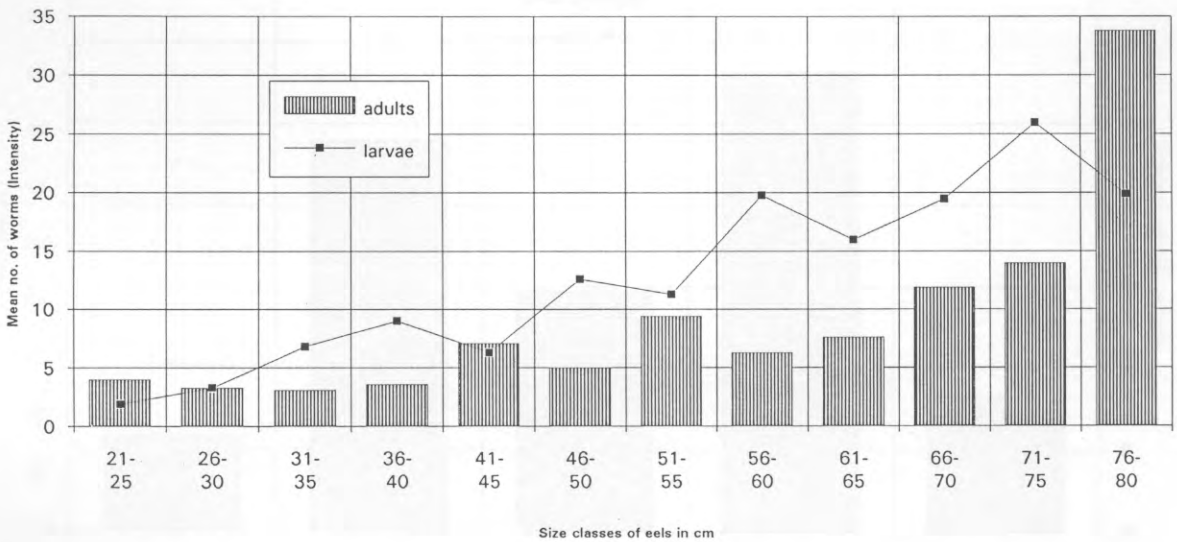

Fig. 11. Mean intensity of *Anguillicola crassus* infection in eels of different size, collected in Lake Balaton from September 1991 to November 1993. Size classes of eels in cm.

As regards the intensity of infection, the highest value was measured in the western basin (Fig. 2) while the lowest occurred in the eastern basin (Fig. 4). However, if only fish of smaller size (20–40 cm), collected by us, are taken into account, no marked difference can be found among the three regions. No distinct correlation could be established as regards larval infection; however, the count of larvae tended to increase in the spring months, while in the second half of the year prevalence and intensity decreased (Figs. 8–10). The only exception to this was the prevalence of infection found in the western region in the second half of 1993. A positive correlation existed between infection intensity and fish size. Bigger fish harboured substantially higher numbers of adults and larvae than did smaller ones (Fig. 11). Especially sharp differences could be observed in the class of swimbladder thickenings depending on the size and place of origin of the fish
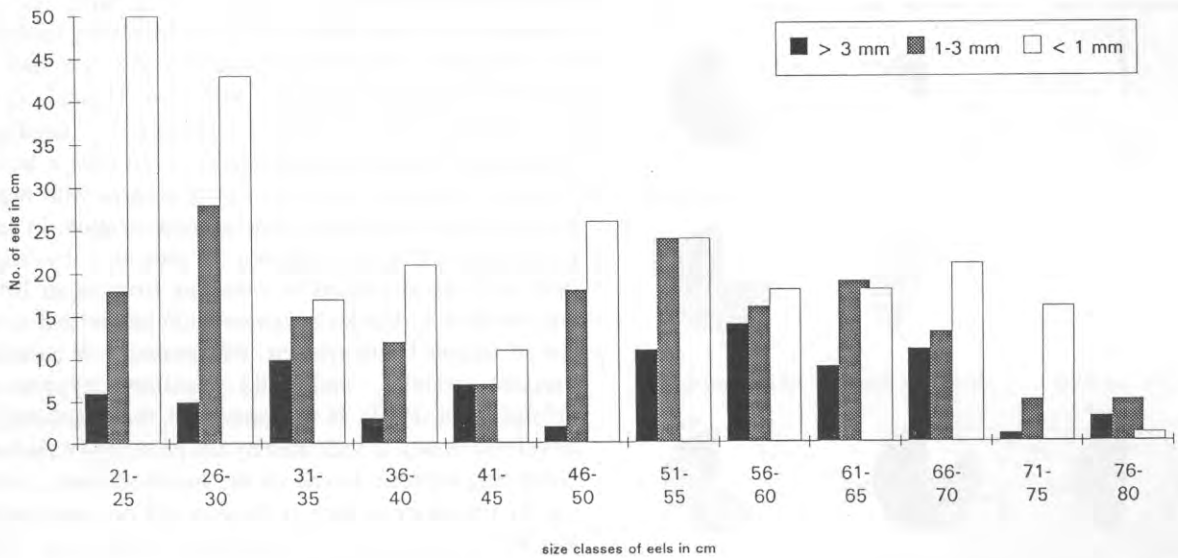
Fig. 12. Swimbladder changes induced by anguillicolosis in eels of different size of Lake Balaton (1991–1993). Initials of the thickness of swimbladder wall (>3 mm, 1–3 mm, <1 mm).

(Figs. 12–13). Among bigger eels, the ratio of specimens having a thickened swimbladder wall was much higher as related to the total number of fish specimens in the group. In many of these fish the normally 0.3–0.5 mm thick swimbladder wall had reached a thickness of 3–6 mm. While among eels less than 50 cm long the specimens having a thin swimbladder wall usually outnumbered those with a thickened swimbladder wall (Fig. 12), among eels longer than 50 cm the number of fish with a severely or moderately thickened swimbladder wall were much higher than that of fish with thin swimbladder walls in all but one group. A characteristic correlation was found in the different regions in the number of swimbladder lesions summarized half-yearly (Fig. 13). In 1991, the majority of eels caught in the Keszthely Bay had swimbladders having a markedly thickened wall (57%) and only 34% had thin swimbladder walls. Concurrently, in the Tihany region 84%, while in the Kenese region 97% of the eels had thin-walled swimbladders. In 1992 the number of eels with thick-walled swimbladders markedly decreased (28%), while at Tihany it reached the value that had been obtained for the Keszthely region in the preceding year (58%). In agreement with this finding, in 1991 eel mortality was observed at Keszthely (250 tons) while in 1992 at Tihany (40 tons). In the Kenese region, the ratio of eels with thickened swimbladder walls rose in 1992 but failed to exceed 27% even in 1993. In 1993, in the Tihany region a moderate improvement occurred, whereas the Keszthely region displayed substantial improvement in pathological changes of swimbladders.

DISCUSSION

The results presented here indicate that at the time of study anguillicolosis was a parasitosis widespread in the entire area of Lake Balaton and affected practically all eel specimens longer than 20 cm. At the same time, the results also demonstrate regional differences in the type of infection in early stages of the study (in 1991). The obtained prevalence and intensity values allow us to conclude that in the western part of Lake Balaton the disease occurred earlier than in the eastern part, in which it became widespread roughly at the time of starting this survey. By 1991, a host-parasite equilibrium seems to have set in the Keszthely Bay region, which was characterized by a high degree of parasitic infection as well as a transient freedom from parasites as a result of the host reaction. Although a host-parasite relationship is spoken of also in that case, this equilibrium was characterized by parasite dominance which manifested itself in massive eel mortality in the Keszthely Bay region. We assume that the parasite first emerged in the western part of Lake Balaton, in the region which lies close to the only eel-rearing farm of Hungary. Rapid multiplication of the parasite in that habitat was potentiated by the high eel density due to eutrophication of the region. Infection seems to have gradually spread from the west towards the east. This is indicated by the fact that prior to the onset of our study an infection of 100% prevalence had developed in the Kenese region, but no host response leading to swimbladder wall thickening had yet been mounted. In the Tihany region, lying west of the Kenese region, the process had
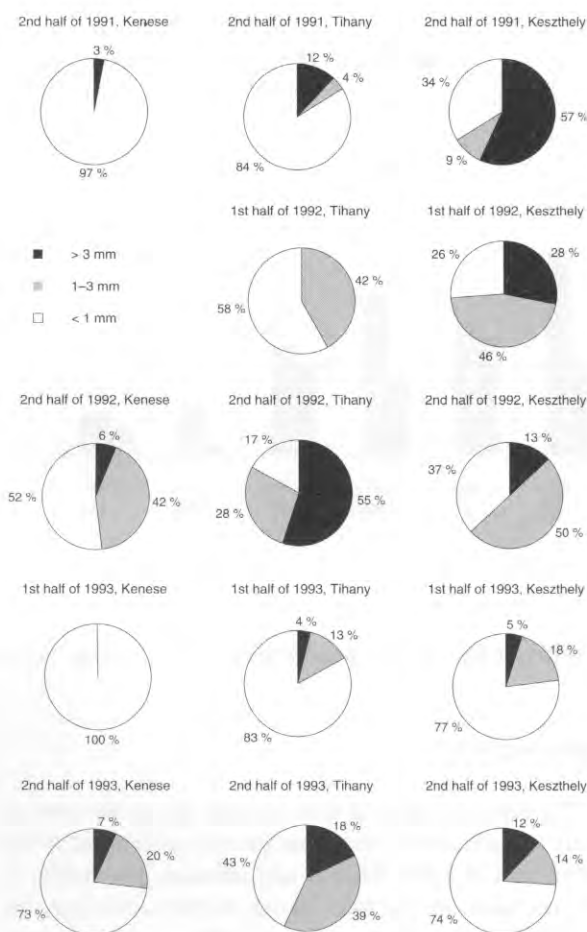
2nd half of 1991, Kenese 3 % 97 %

2nd half of 1991, Tihany 12 % 4 % 84 %

2nd half of 1991, Keszthely 34 % 57 % 9 %

■ > 3 mm
▨ 1–3 mm
□ < 1 mm

1st half of 1992, Tihany 42 % 58 %

1st half of 1992, Keszthely 26 % 28 % 46 %

2nd half of 1992, Kenese 6 % 52 % 42 %

2nd half of 1992, Tihany 17 % 55 % 28 %

2nd half of 1992, Keszthely 13 % 37 % 50 %

1st half of 1993, Kenese 100 %

1st half of 1993, Tihany 4 % 13 % 83 %

1st half of 1993, Keszthely 5 % 18 % 77 %

2nd half of 1993, Kenese 7 % 20 % 73 %

2nd half of 1993, Tihany 18 % 43 % 39 %

2nd half of 1993, Keszthely 12 % 14 % 74 %

Fig. 13. Swimbladder wall thickening in eels from three regions (Eastern /Kenese/, Central /Tihany/, Western /Keszthely/) of Lake Balaton between August 1991 and November 1993.

reached a somewhat more advanced stage, and some of the fish already exhibited a host reaction. However, pathological swimbladder wall thickening and the related mortality did not occur before 1992.

The results obtained by van Willigen and Dekker (1989), Kamstra (1990), van Banning and Haenen (1990), Thomas and Ollevier (1992) and Molnár et al. (1993) indicate that in intensively infected habitats a large number of eels can be found with swimbladders having a thickened (fibrotic) wall and containing no helminths. Molnár et al. (1993) pointed out that some of these specimens are already free from helminths, and suggested that the oedematic and hyperplastic changes result from an inflammatory reaction elicited by repeated larval invasion, which eventually leads to adult elimination and larval mortality. Thus, instead of indicating a lack of infection possibility, the adult-free condition represents a severe stage of anguillicolosis. This stage after demonstrates outcome of the infection, i.e. either during the host-parasite struggle the parasite

obtains the upperhand, leading to mortality, or the immunological resistance of the host increases, resulting in a temporary cessation of infection and in a state of resistance to permanent larval infection. The eel organism seems to be capable of restoring the condition necessary for swimbladder function even after a highly intensive infection, and even swimbladders with markedly thickened walls may undergo regeneration. In such cases the passed-off infection is indicated by slight opacity of the swimbladder, resulting from slight fibrosis, brownish or blackish pigmentation left behind at the site of former haemorrhages, the remnants of calcified parasitic nodules, and mild capillary hyperaemia (Molnár et al. 1993). In the latter case, the possibility of larval reinfection is indicated by the presence of nodules containing necrotic larvae on the intestinal wall. In adverse circumstances such as those which occurred in the Keszthely region in 1991 and near Tihany in 1992 (Molnár et al. 1991, 1993) intensive infection manifests itself in eel mortality. First, high temperature as a stress factor seems to be responsible for the mortality, as at the time of the mass mortality (in July and August of 1991 and 1992) unusually high water temperatures (exceeding 26°C for several days) were recorded. The population density of eels must also have played a role, as in the eastern basin, characterized by a lower eel population density, no deaths were observed. Fig. 13 demonstrates that in both western and central basin the mortality occurred in eel populations whose swimbladder was severely damaged. According to data recorded in 1993, in the years following the mass mortality the number of eel specimens with severely damaged swimbladder decreased in the above habitats as a result of the mortality, while in the eastern basin not affected by the mortality it did not increase substantially. This finding can obviously be explained by the increased resistance of the fish, as the number of larvae inducing infection did not undergo marked changes during the three-year study. The results of Székely (1994) have produced evidence that the infection of paratenic host fishes could provide opportunity for the development of new highly intensive infections. It seems that larval infection does not allow us to draw far-reaching conclusions. The published figures may perhaps indicate only that in the second half of the year the number of larvae tends to decrease while it usually increases in the first half. Similarly, Figs. 2–7 fail to demonstrate a correlation between eel mortality and the number of parasites found in the swimbladder lumen. Fig. 11 clearly shows that larger fish harbour more parasites while smaller fish less. This finding can only partially be explained by the fact that smaller fish consume less infected intermediate or paratenic hosts, as according to Székely (1994) the risk of helminth infection is very high in Lake Balaton. The number of adults is more

likely to be influenced by the host-parasite relationship which reaches an equilibrium after the colonization of smaller eels by less adults and that of bigger specimens by a larger number of parasites.

REFERENCES

BELPAIRE C., DE CHARLEROY D., THOMAS K., VAN DAMME P., OLLEVIER F. 1989: Effect of restocking on the distribution of the swimbladder nematode *Anguillicola crassus* in Flanders, Belgium. J. Appl. Ichthyol. 5: 151–153.

BOON J. H., LOKIN C. J. A., CEUSTERS R., OLLEVIER F. 1989: Some properties of the blood of European eel (*Anguilla anguilla*) and the possible relationship with *Anguillicola crassus* infestations. Aquaculture 76: 203–208.

BOON J. H., AUGUSTIJN H., CANNAERTS V. M. H., LOKIN C. J. A., MACHIELS M. A. M., OLLEVIER F. 1990a: The suitability of experimental inoculations with infective larvae of *Anguillicola crassus* and their effect on the growth and mortality of the European eel (*Anguilla anguilla*). Aquaculture 87: 111–120.

BOON J. H., CANNAERTS V. M. H., AUGUSTIJN H., MACHIELS M. A. M., DE CHARLEROY D., OLLEVIER F. 1990b: The effect of different infection levels with infective larvae of *Anguillicola crassus* on haematological parameters of European eel (*Anguilla anguilla*). Aquaculture 87: 243–253.

BOON J. H., ZUXU Y., BOOMS G. H. R. 1990c: Effects of *Anguillicola crassus* and *Trypanosoma granulosum* infection on peripheral blood cells of European eel (*Anguilla anguilla* L.). Bull. Eur. Ass. Fish Pathol. 10: 143–145.

DE CHARLEROY D., GRISEZ L., THOMAS K., BELPAIRE C., OLLEVIER F. 1990: The life cycle of *Anguillicola crassus*. Dis. Aquat. Org. 8: 77–84.

DUPONT F., PETTER A. J. 1988: Anguillicola, une épizootie plurispécifique en Europe. Apparition d'*Anguillicola crassa* (Nematoda, Anguillicolidae) chez l'Anguille européene (*Anguilla anguilla*) en Camargue, Sud de la France. Bull. Fr. Pêche Piscic. 308: 38–41.

HARTMANN S. 1987: Schwimblassenwürmer beim Aal. Fischer & Teichwirt 38: 2–3.

HAENEN O. L. M., GRISEZ L., DE CHARLEROY D., BELPAIRE C., OLLEVIER F. 1989: Experimentally induced infections of European eel *Anguilla anguilla* with *Anguillicola crassus* (Nematoda, Dracunculoidea) and subsequent migration of larvae. Dis. Aquat. Org. 7: 97–101.

HAENEN O. L. M., VAN BANNING P. 1991: Experimental transmission of *Anguillicola crassus* (Nematoda, Dracunculoidea) larvae from infected prey fish to the eel, *Anguilla anguilla*. Aquaculture 92: 115–119.

HÖGLUND J., ANDERSSON J. 1993: Prevalence and abundance of *Anguillicola crassus* in the European eel (*Anguilla anguilla*) at a thermal discharge site on the Swedish coast. J. Appl. Ichthyol. 9: 115–122.

HÖGLUND J., ANDERSSON J., HARDIG J. 1992: Haematological responses in the European eel, *Anguilla anguilla* L., to sublethal infestation by *Anguillicola crassus* in a thermal effluent of the Swedish Baltic. J. Fish Dis. 15: 507–514.

HÖGLUND J., THOMAS K. 1992: The black goby, *Gobius niger*, as a potential paratenic host for the parasitic nematode *Anguillicola crassus* in a thermal effluent of the Baltic. Dis. Aquat. Org. 13: 175–180.

KAMSTRA A. 1990:. Anguillicola in Dutch eelfarms; current state. Int. Revue ges. Hydrobiol. 75: 867–874.

LIEWES E. W., SCHAMINEE-MAIN S. 1987: Onderzoek naar de effecten van de parasiet *Anguillicola crassa* op de ontwikkeling van de paling (*Anguilla anguilla*) in een zout water palingmesterij. Internal Report, Texvis B.V., Den Burg Texel, The Netherlands.

MELLERGAARD S. 1988: Ålens svommeblaereorm *Anguillicola* – en ny parasit i den europaeiske ålebestand. Nordisk Aquakultur 4: 50–54.

MOLNÁR K., BASKA F., CSABA Gy., GLÁVITS R., SZÉKELY Cs. 1993: Pathological and histopathological studies of the swimbladder of eels *Anguilla anguilla* infected by *Anguillicola crassus* (Nematoda: Dracunculoidea). Dis. Aquat. Org. 15: 41–50.

MOLNÁR K., SZÉKELY Cs., BASKA F. 1991: Mass mortality of eel in Lake Balaton due to *Anguillicola crassus* infection. Bull. Eur. Ass. Fish Pathol. 11: 211–212.

MORAVEC F. 1992: Spreading of the nematode *Anguillicola crassus* (Dracunculoidea) among eel populations in Europe. Folia Parasitol. 39: 247–248.

MÖLLER H., HOLST S., LÜCHTENBERG H., PETERSEN F. 1991: Infection of eel *Anguilla anguilla* from the River Elbe estuary with two nematodes, *Anguillicola crassus* and *Pseudoterranova decipiens*. Dis. Aquat. Org. 11: 193–199.

NEUMANN W. 1985: Schwimmblasenparasit *Anguillicola* bei Aalen. Fischer & Teichwirt 36: 322.

PAGGI L., ORECCHIA P., MINERVINI R., MATTIUCCI S. 1982: Sulla comparsa di *Anguillicola australiensis* Johnston e Mawson, 1940 (Dracunculoidea: Anguillicolidae) in *Anguilla anguilla* del Lago di Bracciano. Parassitologia 24: 139–144.

PETERS G., HARTMANN F. 1986: *Anguillicola*, a parasitic nematode of the swim bladder spreading among eel populations in Europe. Dis. Aquat. Org. 1: 223–230.

SARTI M., GEORGETTI G., BRISINELLE W. 1985: A new problem for intensive eel rearing in Italy: *Anguillicola australiensis*. Proc. E.A.F.P. Congress, Montpellier, September 1985, p. 95.

SPRENGEL G., LÜCHTENBERG H. 1991: Infection by endoparasites reduces maximum swimming speed of Europaen smelt *Osmerus eperlanus* and European eel, *Anguilla anguilla*. Dis. Aquat. Org. 11: 31–35.

SZÉKELY Cs. 1994: Paratenic hosts for the parasitic nematode *Anguillicola crassus* in Lake Balaton, Hungary. Dis. Aquat. Org. 18: 11–20.

SZÉKELY Cs., LÁNG M., CSABA Gy. 1991: First occurrence of *Anguillicola crassus* in Hungary. Bull. Eur. Ass. Fish Pathol. 11: 162–163.

TARASCHEWSKI H., MORAVEC F., LAMAH T.,

ANDERS K. 1987: Distribution and morphology of two helminths recently introduced into European eel populations: *Anguillicola crassus* (Nematoda, Dracunculoidea) and *Paratenuisentis ambiguus* (Acanthocephala, Tenuisentidae). Dis. Aquat. Org. 3: 167–176.

THOMAS K., OLLEVIER F. 1992: Population biology of *Anguillicola crassus* in the final host *Anguilla anguilla*. Dis. Aquat. Org. 14: 163–170.

Van BANNING P, HAENEN O. L. M. 1990: Effects of the swimbladder nematode *Anguillicola crassus* in wild and farmed eel, *Anguilla anguilla*. In: F. O. Perkins, T. S. Cheng, (Eds), Pathology in Marine Science. Academic Press, New York, pp. 317–330.

Van WILLIGEN J., DEKKER W. 1989: 1988 update on *Anguillicola* in Dutch outdoor waters. EIFAC (FAO) Working Party on Eel. Oporto, Portugal, 29 May to 3 June, 1989, 8 pp.