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Infection of eel *Anguilla anguilla* (L.) and smelt *Osmerus eperlanus* (L.) with *Anguillicola crassus* (Nematoda, Dracunculoidea) in the Netherlands from 1986 to 1992

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Abstract

Dutch wild eels [*Anguilla anguilla* (L.)] and smelt [*Osmerus eperlanus* (L.)] from freshwater and saltwater areas in the Netherlands were collected from 1986 to 1992 and their swimbladders were examined for *Anguillicola crassus* (Nematoda, Dracunculoidea) and for parasite-related pathological changes. Throughout the 6-year sampling period, young eels (up to 17 cm) showed severe pathological changes due to the parasite. The prevalence of infection in larger eels (23–34 cm) showed the highest prevalence between 1987 and 1988, and the highest intensity (i.e. number of parasites per infected fish) between 1988–1989. After 1989 the prevalence of the parasite decreased, and the lesions became less severe. Larger eels (23–34 cm) from the Waddenzee (salt water), which is close to the IJsselmeer, showed a high prevalence of the parasite from 1987 to 1990, although the intensity of infection decreased from 1987 onwards, as did the percentage of fibrotic swimbladders after 1988. Smelt, which is a paratenic host for larvae of *A. crassus* and which is a prey for large eels, showed a sharp decrease in prevalence of the parasite shortly after 1988. Thereafter the prevalence stayed rather constant at about 40% of the smelt population. No pathological changes were found in the smelt.

Keywords: *Anguillicola*; European eel; Pathobiology; Smelt; Netherlands

1. Introduction

The parasitic nematode, *Anguillicola crassus* (Moravec and Taraschewski, 1988), originally described as *A. crassa* (Kuwahara et al., 1974), resides in the swimbladder of the

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eel, *Anguilla anguilla*. It was introduced into Europe in the early 1980s (Neumann, 1985; Van Banning et al., 1985), and has since then spread rapidly among European eel populations (Peters and Hartmann, 1986; Canestri-Trotti, 1987; Køie, 1987; Taraschewski et al., 1987; Dupont and Petter, 1988; Hellström et al., 1988; Belpaire et al., 1989; Dekker and van Willigen, 1989; Koops and Hartmann, 1989; Van Willigen and Dekker, 1989; Kennedy and Fitch, 1990; Möller et al., 1991; Székely et al., 1991; Cruz et al., 1992; Moravec, 1992).

Immediately after the parasite was introduced into Europe, both farmed and wild eel showed high intensities of infection and severe swimbladder lesions (Neumann, 1985; Dekker and van Willigen, 1988; Van Banning and Haenen, 1990; Køie, 1991; Molnár et al., 1991, 1993; Cruz et al., 1992; Thomas and Ollevier, 1992; Molnár et al., 1993).

After the parasite was found in Dutch wild eels, an investigation was conducted from 1986 to 1992 in the freshwater lakes IJsselmeer and Ketelmeer, and in the sluice-connected waters Markermeer and Waddenzee (from 1987 to 1990) (Fig. 1). This article reports the findings of the investigation, the purpose of which was to determine trends in the presence of the parasite in eels of different sizes as well as its pathological effects. Smelt (*Osmerus eperlanus*), which are preyed upon by eel and which act as paratenic host for *A. crassus* larvae (Haenen and van Banning, 1990, 1991), were also sampled.

2. Materials and methods

Fig. 1 shows the sites at which fish were caught for the study.

Sampling and examination of small eels from the IJsselmeer

Small eels (up to 17 cm) were caught with an electrified fine meshed beam trawl in spring (May–June) and autumn (October–November) around the site Wagenpad, located in the western part of the IJsselmeer. The eels were taken alive to the laboratory to be examined. The eels were anaesthetized with metomidate (20 mg/l) or 2-phenoxyethanol (0.2–0.5 ml/l), and measured. The swimbladders were dissected and examined with a light microscope for *A. crassus* (number and stages of development). A few fish, depending on the sample sizes, were transversally cut, and the pieces were fixed in 10% buffered formalin, embedded in wax, sectioned transversely at 4 μm, and stained with haematoxylin and eosin (H&E) for histopathological examination.

Larger eels from the IJsselmeer and Markermeer (freshwater)

Larger eels (23–34 cm) were caught from the IJsselmeer and Markermeer with an electrified fine-meshed beam trawl at different sites in spring and autumn from 1986 to 1992. In 1987 no samples were collected from the Markermeer. The swimbladders of all eels were examined macroscopically for the number of *A. crassus* and for lesions, but were not examined histopathologically.

Larger eels from the Waddenzee (salt water)

Larger eels (23–34 cm) were caught from the Waddenzee from 1987 to 1990 between mid-September and mid-November. Fyke nets were left in place for 3–5 days per catch along the coastline, close to Kornwerderzand sluice. This site is located at the eastern end

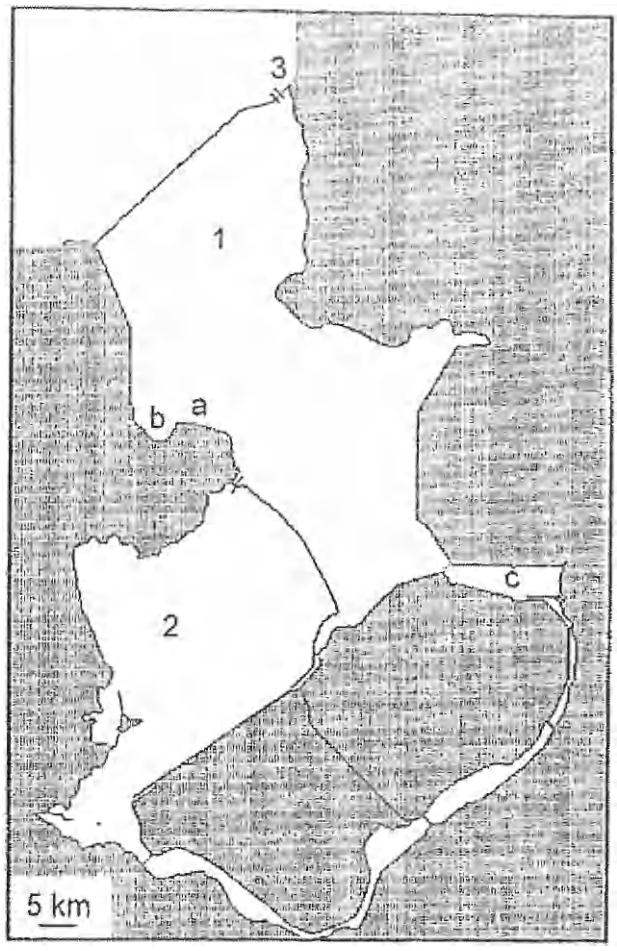

Fig. 1. Sampling sites of eels and smelt in the Netherlands. 1 = IJsselmeer, 2 = Markermeer, 3 = Waddenzee, a = Wagenpad, b = Medemblik, c = Ketelmeer.

of the Afsluitdijk, a dyke which separates the Waddenzee from the IJsselmeer. A random sample of about 50 eels per catch was taken alive to the laboratory. The eels were anaesthetized as described earlier, measured, and examined in the same way as large eels taken from the IJsselmeer and Markermeer.

Smelt

In the spring and autumn of 1988, 1989, 1991 and 1992, we also collected smelt (*Osmerus eperlanus*) from different sites in the IJsselmeer, in the same way described for the small eels. The smelt died immediately after they were caught. They were put into a plastic bag, and transported under cool conditions to the laboratory, where they were stored at 4°C. The next morning 50 fish were randomly selected from each group, measured, and necropsied. A fresh tissue preparation was made of each swimbladder and was examined under a light microscope for the number and stages of development of *A. crassus* specimens.

The Generalized Linear Model (McCullagh and Nelder, 1989) was used to analyze statistically the prevalence and intensity of infection per year in the large freshwater eels. For smelt, analysis of variance (Genstat 5 Committee, 1987) was used to analyze statistically the prevalence and intensity of infection per year and site, and the length of the fish in relation to the intensity of the infection.

3. Results

Small eels from the IJsselmeer

Table 1 presents quantitative data on the infection of small eels with *A. crassus*. The prevalence of the infection varied between 42 and 91% over the years, with no apparent trend. Swimbladders showed the following lesions: haemorrhages (0–70% of the eels), congestion of blood vessels (14–60% of the eels), and pigmentations (3–30% of the eels), also without a trend.

The percentage of eels with fibrotic swimbladders varied between 45 and 90%. The small-sized eels showed severe lesions of the swimbladder, caused by the parasite, such as haemorrhages, congestion of blood vessels and thickening of the swimbladder. As early as 1987 (Fig. 2), parasites were found encapsulated in the fibrotic swimbladder wall, which was often connected to the intestine by fibrotic tissue. Mostly L2 and L3 larvae were found in these complexes, as well as dead pre-adults and adults. Tunnels showing the migration pathway of L3 larvae were clearly visible in the fibrotic tissue. The kidney was also sometimes covered with a fibrotic layer, in which *A. crassus* L2 and L3 larvae could be found. Often many lymphocyte-like cells were seen in the fibrotic swimbladder wall.

A. crassus was found only in eels larger than 8 cm (we checked 12 eels of 6–8 cm).

Larger eels (23–34 cm) from the IJsselmeer and Markermeer

The mean yearly prevalence (%) of *A. crassus* in the eels is shown in Fig. 3. For the IJsselmeer the peak years were 1987 and 1988 with a maximum of 97.2% ($n=216$) in 1987. For the Markermeer a similar trend was seen, with a maximum in 1988 of 96.4% ($n=85$). No data were recorded in 1987, however.

Table 1
Anguillicola crassus infections in small-sized eels (< 18 cm) from the IJsselmeer in the period 1987–1992 (macroscopic and microscopic examinations)

Date	No. of eels	Length range (cm)	Site[a]	Prevalence of infection (%)	Fibrotic swimbladders (%)
June '87	18	7–15	Wag	56	44
Oct. '87	8	9–16	Med	88	75
June '88	16	6–15	Med	81	69
Oct. '88	10	9–15	Ket	90	90
June '89	19	7–16	IJs	42	53
Oct. '89	16	9–16	IJs	69	56
Sept. '90	17	8–16	IJs	65	47
May '91	11	9–16	Wag	73	46
Oct. '91	35	9–16	Wag	91	60
May '92	47	6–16	Wag	81	70
Nov. '92	29	9–17	Wag	86	90

[a] Wag = Wagenpad; Med = Medemblik; Ket = Ketelmeer; IJs = central IJsselmeer.

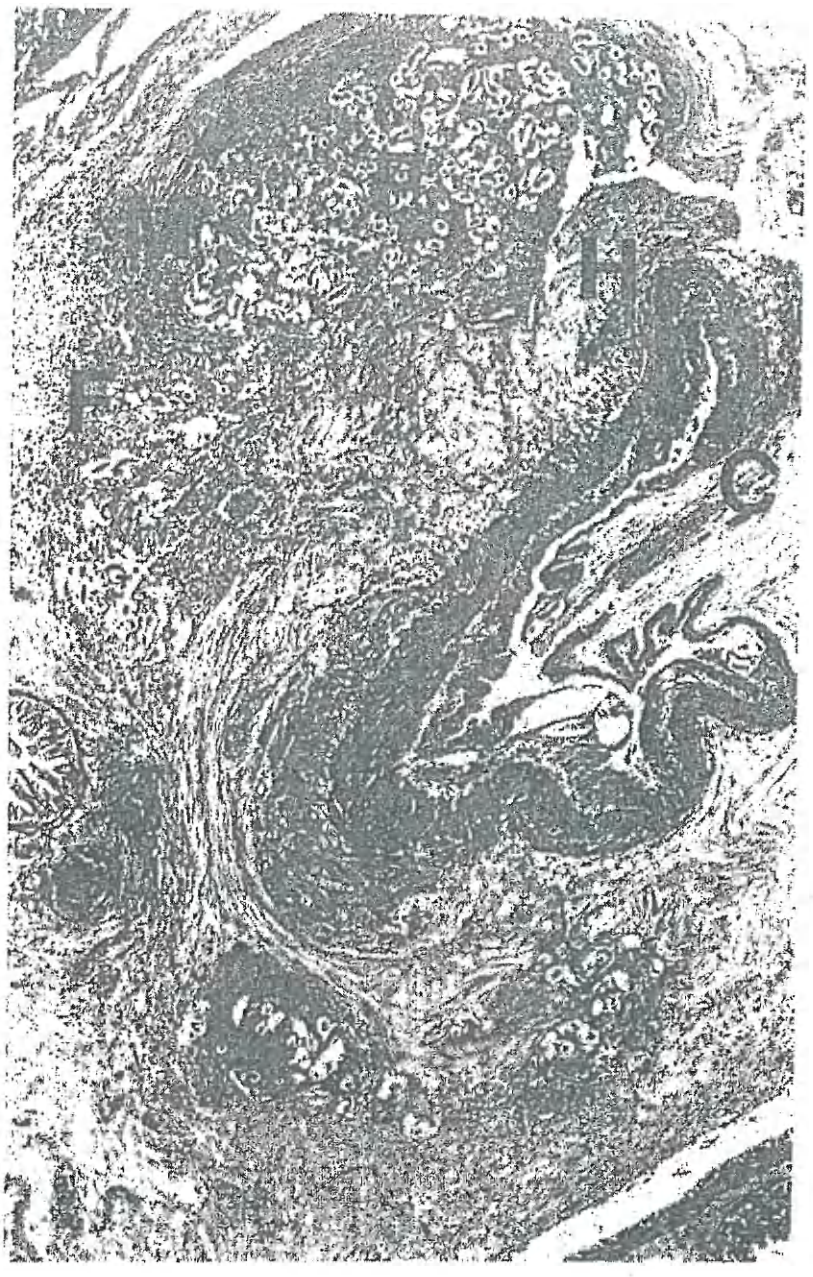

Fig. 2. Cross-section of the swimbladder of European eel (IJsselmeer, 1987; 14 cm) with a chronic infection of *Anguillicola crassus*. Severe fibrosis (F) of the swimbladder wall with many encapsulated L1 and L2 larvae of the parasite (P) surrounded by mononuclear phagocytes (M), congestion of blood vessels (C) and haemorrhages (H) (40×).

The intensity of infection (i.e., mean number of *A. crassus* per infected eel) is also shown in Fig. 3. The mean intensities were 10.3 parasites per infected eel for both the IJsselmeer and the Markermeer.

From 1987 onwards fibrotic swimbladders were found in 3–20% of the eels from the IJsselmeer. Fibrotic swimbladders were not detected until 1989 onwards in eels from the Markermeer (2–36%).

Larger eels (23–34 cm) from the Waddenzee

Table 2 shows the mean prevalence and intensity of *A. crassus* infections from 1987 to 1990. Although the prevalence of infection was high and it was fairly constant during the sampling period (September to November), no change was observed. In contrast, the

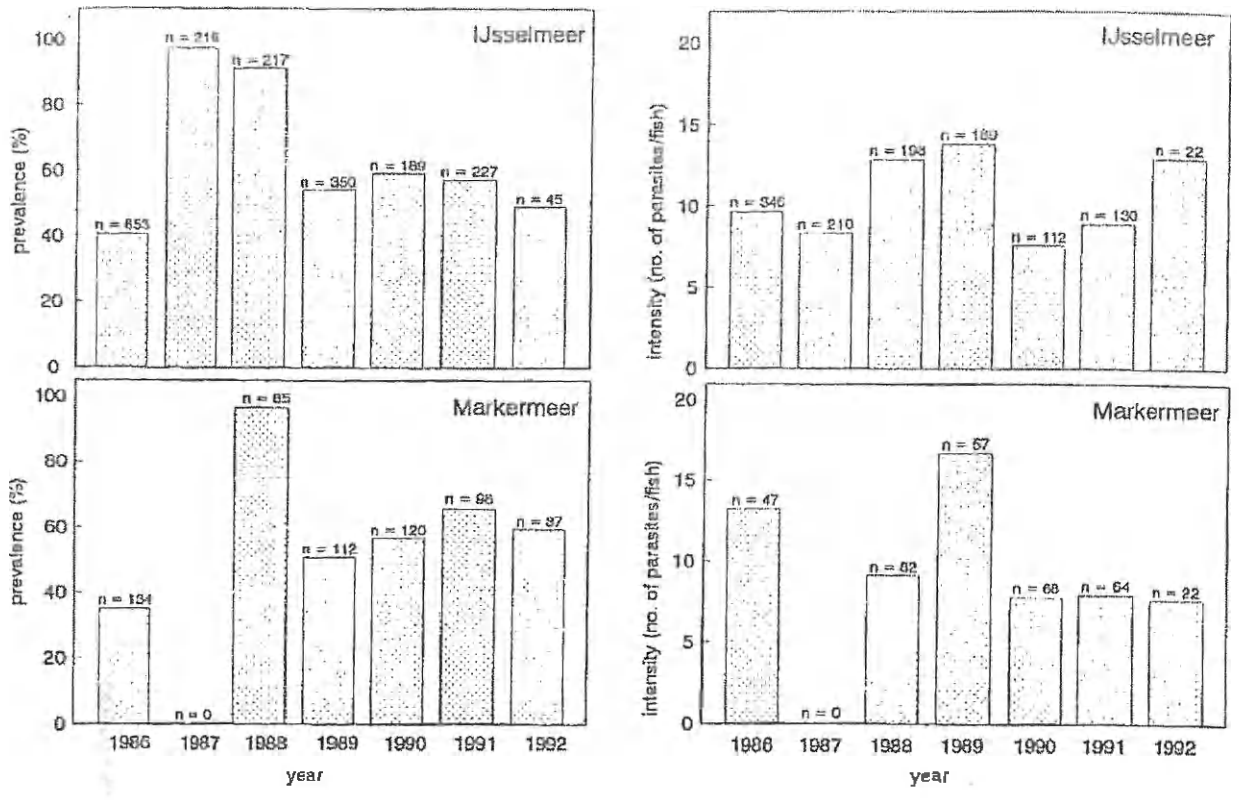

Fig. 3. Prevalence and intensity of *Anguillicola crassus* infections in larger eels (23–34 cm) in the Netherlands (IJsselmeer and Markermeer) in 1986–1992. n = number of eels analyzed.

intensity of infection decreased during the study. The mean percentage of eels with fibrotic swimbladders peaked in 1988 at 24.5% (range: 10–33% per sample catch) and decreased thereafter.

Smelt from the IJsselmeer

Table 3 shows the percentage and intensity of *A. crassus* infections in smelt from 1988 to 1992. Smelt from the Ketelmeer showed a high prevalence of infection (88%) in 1988, but this figure is based on only one sample catch. No trend in the prevalences or intensity was observed in smelt caught at the Wagenpad site from 1991 to 1992.

Most infected smelt contained L3 larvae of *A. crassus* and sometimes L4 larvae were found. Dead or encapsulated L3/L4 larvae were found only rarely in the swimbladder

Table 2

Anguillicola crassus infections in larger eels (23–34 cm) from the Waddenzee between 1987 and 1990: mean prevalence (%) and intensity (number of parasites per infected eel)

Year	No. of eels	Prevalence (%)	Range[a] (%)	Intensity[b]	Range[a]
1987	378	86	80–96	7.7	6.0–8.6
1988	500	85	77–96	6.0	4.5–9.0
1989	399	87	82–94	4.8	4.2–5.9
1990	291	90	86–94	4.8	4.0–5.5

[a] Per group of about 50 eels.

[b] Intensity = mean number of parasites per infected eel.

Table 3
Anguillicola crassus infections in smelt from different sites of the IJsselmeer between 1988 and 1992

Date	Site	No. of smelt examined	Prevalence of infection (%)	Intensity[a]
19 Oct '88	Ketelmeer	24	88	not recorded
18 Oct '89	Kornwerderzand	25	48	2.16
30 May '91	Wagenpad	50	18	1.88
30 May '91	Staverse Geul	50	42	1.42
28 Oct '91	Wagenpad	50	38	1.42
18 May '92	Wagenpad	50	38	1.21
18 May '92	Kreupel	50	30	1.20
11 Nov '92	Wagenpad	50	42	1.52

[a] Mean no. of *A. crassus* larvae per infected fish.

lumen. No other stages of the parasite were found in the swimbladders, and swimbladders showed no pathological changes.

Statistical analysis

According to the Generalized Linear Model analysis, there was no significant difference ($P > 0.05$) between the prevalence (logistic model) or the intensity (loglinear model) of the infection in larger eels (23–34 cm) from the IJsselmeer and the Markermeer. The residuals around the prevalence were considered to be distributed binomially, and the intensity was considered to be distributed according to the Poisson model. To test if there were yearly effects, the data on eels taken from the two waters were pooled (Table 4) for an analysis with *t*-values of pairwise differences between years. The prevalence of *A. crassus* in larger eels in 1987 and 1988 did not differ significantly, but was significantly higher in 1987 and 1988 than in 1986 and 1989–1992. The intensity of *A. crassus* was highest in 1988 and 1989, with no significant difference between the two years. The intensity measured in this period was significantly higher than in 1986–1987 and 1990–1992 ($P < 0.05$).

Table 4
Estimated mean prevalence and intensity of *A. crassus* infection in large eels (23–34 cm) from the IJsselmeer and Markermeer between 1986 and 1992

Year	Prevalence (%)	Intensity (no. of parasites/infected eel)
1986	39.8 (a)[a]	6.78 (a)
1987	97.2 (b)	6.59 (a)
1988	92.7 (b)	6.93 (b)
1989	53.2 (c)	7.14 (b)
1990	58.2 (c)	6.49 (a)
1991	59.7 (c)	6.62 (a)
1992	53.7 (c,a)	6.80 (a)

The estimated mean prevalences were based on logistic regression, and the intensities on loglinear regression.
[a] a, b and c are significant different groups ($P < 0.05$).

Wagenpad, which was sampled several times, did not differ significantly from the other sites in the IJsselmeer for the prevalence or intensity of the infection in smelt. Also, there was no correlation between the number of *A. crassus* larvae per swimbladder and the length of the smelt. No significant differences in prevalence and intensity was found from 1988 to 1992 ($P > 0.05$).

4. Discussion

In small eels, severe lesions such as those described by Van Banning and Haenen (1990) and Molnár et al. (1993) were seen during the whole period. Chronic swimbladder inflammation (Molnár et al., 1993) was often found, with the encapsulated remains of dead adult worms within the heavily fibrotic tissue.

The fact that no trend in the infection of small eels was observed means that the infectivity of the parasite for small eels did not change. Moreover, the fact that no trend in the infection-related lesions of small eels was seen makes us conclude that *A. crassus* did not evolve to become less pathogenic for the eels. Every year new elvers arrive in the IJsselmeer (Dekker et al., 1992) and get their first infection with *A. crassus*. Our results show that the small eels have remained susceptible to the parasite in the last few years. Therefore, it is unlikely to be a case of genetic selection of less susceptible eels. This selection might occur in the long term.

The explanation for the decrease in infection in larger eels might be an immunological one. The development of a humoral and non-specific immunological response against *A. crassus* in eels has been suggested by Van Willigen and Dekker (1989), Höglund et al. (1992) and Molnár et al. (1993). Buchmann et al. (1991) demonstrated a humoral response of eels to the parasite. Whether the immunological response is protective is not known. Protective immunity is known to occur in some fish species towards protozoan parasites, e.g. against *Ichthyophthirius multifiliis* in carp (Houghton, 1987). Metazoan parasites such as *Diphyllobothrium dendriticum* elicit a non-specific and a humoral immune response in rainbow trout (Sharp et al., 1992). However, in metazoan parasitic infections of fish, the precise role of the immune response has yet to be determined (Woo, 1992). This aspect should receive more attention in future studies.

The *A. crassus* infection developed rapidly towards high prevalences, both in IJsselmeer and Markermeer, and the Waddenzee. In 1985, IJsselmeer eels had a prevalence of *A. crassus* infection of only 28–45%, the Waddenzee eels (near Harlingen) only 2% (Van Banning et al., 1985). By 1986, prevalences were as high as 37.5–50% for the IJsselmeer and Markermeer and 0.5–25% for the Waddenzee (Dekker and van Willigen, 1989). In 1987, thickened swimbladders without a lumen, so-called collapsed swimbladders, were first detected in infected eels from the IJsselmeer (Dekker and van Willigen, 1988). This condition indicates the chronic form of infection (Molnár et al., 1993).

The prevalence of *A. crassus* infection in the IJsselmeer rose to a peak of 97% in 1987, according to our results. In 1988 91% of the IJsselmeer eels and 97% of the Markermeer eels were still infected. After these years the prevalences of infection decreased. This tendency to decrease had already been suggested by Dekker and van Willigen (1988). However, the prevalence of infection continued to fluctuate: even in 1993 81% ($n = 116$

eels) of the 23–34 cm eels from the IJsselmeer and 84.6% ($n=65$) from the Markermeer were infected. However, the intensity of infection in these eels was lower than in earlier years: 5.06 ($n=94$) for the IJsselmeer eels and 4.40 ($n=55$) for the Markermeer eels. We suggest that these values will probably not stabilize for a few years.

The effects of annual variations of weather on the population dynamics of zooplankton (intermediate hosts, often copepods) and the role of different fish species acting as paratenic hosts and prey for eels have not been taken into account as possible causes of the infection fluctuations in this study. However, our study shows that possible fluctuations in the infection of copepods with *A. crassus* larvae did not give rise to changes in the infection of small eels and smelt (after 1988) during our investigation.

The larger eels from the Waddenzee in our study had been caught with fyke nets near the Kornwerderzand sluice. This sluice allows contact between salt water and fresh water from the IJsselmeer. Eels can be transferred between the two waters by the strong current every time the sluice opens, the water flowing mostly in the direction of the Waddenzee (Dekker, unpublished). The infected Waddenzee eels, which may originate from the IJsselmeer (Dekker and van Willigen, 1989) showed a higher prevalence of infection than the IJsselmeer eels. This could be the result of heavily infected eels swimming more slowly (Sprengel and Lüchtenberg, 1991) and being weaker because of decreased haematocrit and plasma proteins (Boon et al., 1990), and they are therefore less capable of swimming back against the strong current in the sluice. Also, quick transfer of eels between fresh water and salt water might induce stress and therefore immuno-suppression (Ellis, 1981).

The intensity of infection (number of parasites per infected eel) was high in both the IJsselmeer (7–14) per eel and Markermeer (7–13) per eel from 1986 to 1992. These numbers were not as high as those found by Molnár et al. (1991), who often detected 30–50 *A. crassus* per swimbladder in lake Balaton (Hungary), directly after the introduction of the infection. Dekker and van Willigen (1989) found 0.14% of the eels carrying more than 20 *A. crassus* in 1986 in the Netherlands (both salt and fresh water). In Belgium, Thomas and Ollevier (1992) found a mean intensity of *A. crassus* of 17 parasites in 1990–1991, higher than the rate found in the Netherlands in that period. They counted the parasites not only macroscopically, as we did, but also microscopically, which enabled them to detect larval stages, and thus more parasites. They also caught the eels in a different way, by using fyke nets and by harvesting eels trapped in intake screens of a power plant.

Prevalences or intensity of *A. crassus* infection in eels did not differ statistically in our study, in the IJsselmeer and Markermeer. These waters are separated by a dike and two sluices, which are opened more than 20 times a day. The Ketelmeer is directly connected with the IJsselmeer.

Many fish species are known as paratenic hosts (De Charleroy et al., 1990; Haenen and Van Banning, 1990; Thomas and Ollevier, 1992), of which some are known to be preyed upon by eels (De Nie, 1987). Smelt are preyed upon by eels and may transmit the L3 larvae of *A. crassus* to the eel (Haenen and Van Banning, 1990, 1991). In examining smelt, we sometimes also detected *L4* larvae, but no pre-adult or adult parasites. Many other fish species preyed upon by eels can be paratenic hosts. Large eels also consume infected copepods (Kennedy et al., 1992) and are thereby directly infected. These facts might explain the rapid development of the *A. crassus* infection in eels. Although dead or encapsulated larvae were sometimes found in the smelt, the parasite is not pathogenic to the smelt.

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