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Anguillicolosis in the short-finned eel *Anguilla australis*: epidemiology and pathogenicity

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Abstract A sample of 189 short-finned eels (*Anguilla australis*) from four geographical areas in New Zealand were examined for the occurrence of the swimbladder nematode *Anguillicola* spp. *Anguillicola novaezelandiae* was found in three of the four localities. Overall, the prevalence was low (<12%) as was the intensity of infection (between 1 and 5 parasites per infected eel). In comparison with data from Europe on a similar host-parasite system involving congeneric species (*Anguilla anguilla-Anguillicola crassus*), it was concluded that the New Zealand short-finned eels do not greatly suffer from this infection. In particular, no effect of infection on body condition was detected. The damage caused by the parasite to the swimbladder was minimal, even in large silver eels about to undertake a long journey at great depth to reach their spawning grounds in the Pacific. A review of the available literature is presented to update the current knowledge of the distribution of *Anguillicola* infections in New Zealand.

Keywords *Anguilla australis*; infection; *Anguillicola novaezelandiae*; swimbladder degenerative index; body condition

INTRODUCTION

Anguillicolosis, i.e., the infection of eels by nematodes of the genus *Anguillicola*, is a problem of concern in many parts of the world. The 5 species currently recognised within the genus are known to infect 6 of the 15 anguillid eel species (Moravec & Taraschewski 1988; Lin et al. 2001). These nematode parasites use eels as definitive hosts and reproduce exclusively in their swimbladder. Eels become parasitised by ingesting infected prey, either planktonic crustaceans (intermediate hosts) or small fish (paratenic hosts) (Thomas & Ollevier 1992; Moravec et al. 1994). *Anguillicola* larvae grow and moult within the tissue layers of the swimbladder wall where they feed on cells (histotrophy). Pre-adult and adult forms inhabit the swimbladder lumen and actively feed on blood from the capillary system (haematophagy) (Polzer & Taraschewski 1993; Würtz & Taraschewski 2000).

The impact of anguillicolosis on eels has been thoroughly studied and appears to vary greatly according to the host and parasite species under consideration. Typically, as for many other host-parasite systems, severe pathological infections are observed with the translocation of parasite species into areas inhabited by novel potential host species (Kennedy 1994; Wakelin 1996). Because eels have a very high fishing and commercial value, numerous transcontinental importations have occurred for the purpose of restocking (Køie 1991). A typical example of such a biological invader is provided by the now widespread Asian nematode *Anguillicola crassus*. Whereas a low susceptibility to *A. crassus*

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is reported in its original host, the Japanese eel *A. japonica* (in terms of prevalence and histopathological damage, see Egusa 1979), severe symptoms have been documented in its new hosts, the European eel *Anguilla anguilla* and the American eel *Anguilla rostrata* (Van Banning & Haenen 1990; Molnár et al. 1993; Würtz & Taraschewski 2000; Barse et al. 2001). Damage is caused by the migration of larval stages through the different layers of the swimbladder wall (Molnár et al. 1993) and by the mechanical injuries resulting from the haematophagous activity of the adult lumen worms (Würtz & Taraschewski 2000). This results in inflammation, fibrosis, and local necrosis, leading occasionally to a total degeneration of the swimbladder. This damage can persist after the loss of the worms (Van Banning & Haenen 1990). Infection by *A. crassus* has been mentioned as a possible cause of successive mass eel mortalities on the European continent (Molnár et al. 1991; Barus & Moravec 1999).

According to the literature, the New Zealand short-finned eel *Anguilla australis* Richardson, 1841, is only infected by the native *Anguillicola novaezelandiae* Moravec & Taraschewski, 1988 (Hine et al. 2000). Based on previous studies, the spread of *A. novaezelandiae* and its pathogenicity appear limited, but most previous investigations consist of parasite checklists or systematic descriptions and thus nothing is known of the impact of *Anguillicola* infection on the health state of the swimbladder or the body condition of New Zealand eels (Brunsdon 1956; Rid 1973; Hine 1978; Boustead 1982; Moravec & Taraschewski 1988; Hine et al. 2000). Interestingly, *A. novaezelandiae* was introduced into a lake in Italy, but did not spread throughout Europe and finally was replaced by the Asian species *A. crassus* which also invaded the lake (Moravec et al. 1994). Considering the colonising attributes of *A. crassus*, the possibility exists that other, more pathogenic, *Anguillicola* species infect eels in New Zealand.

As part of a broader study aiming to resolve the co-phylogenies of *Anguillicola-Anguilla* throughout the world, we obtained short-finned eel samples from different localities in New Zealand. Here, we used these animals to: (1) check the taxonomic status of *Anguillicola* specimens found in New Zealand; (2) determine the prevalence and intensity of *Anguillicola* spp. infections in New Zealand eels; (3) assess the impact of these parasites on the swimbladder of eels, using the swimbladder degenerative index (SDI) developed for the European eel *A. anguilla* by one of us (Lefebvre et al. 2002a); (4) assess the potential impact of infection on eel physiology and metabolism by comparing the body condition of infected and uninfected eels; and (5) update the current knowledge on the geographical spread of this infection in New Zealand.

MATERIALS AND METHODS

Freshly caught eels *A. australis* were obtained in late 2003 from professional fishers in four localities throughout New Zealand (see details and date of collection in Table 1). They were transported alive to the laboratory where they were anaesthetised immediately and killed with a lethal dose of benzocaine (3 mg litre^{-1}). The total length (L) was recorded to the nearest mm, and the somatic weight (W, eviscerated carcass weight, i.e., without alimentary tract and gonads) was recorded to the nearest 1 mg. The sex was determined macroscopically by examining the shape and size of the gonads (Beentjes & Chisnall 1998). For those eels classified visually as maturing "silver" individuals (dark lateral line, silver coloration, well developed gonads, see Pankhurst 1982), the vertical (Dv) and horizontal (Dh) eye diameters were measured with callipers to the nearest 0.1 mm. Based on the relationship between the size of the eyes and L, the Pankhurst's ocular index for silvering was calculated as follows: $((Dv + Dh)/4)^2 \times (\pi/L)$. Only eels that met the threshold index value of ≥6.5 were considered as silver individuals (Pankhurst 1982).

The swimbladder of each eel was removed and examined macroscopically for the presence of nematodes. The number of lumen worms (pre-adults and adults within the swimbladder) was recorded. All worms were identified to species according to the key of Moravec & Taraschewski (1988). Throughout the study, the parasitic descriptors used were those defined by Bush et al. (1997). Prevalence is the percentage of infected eels in a sample, and mean intensity is the average number of worms per infected eel.

The health of the eel swimbladder was assessed by using the swimbladder degenerative index (SDI, see Lefebvre et al. 2002a). Assessment was based on macroscopically-visible alterations in the swimbladder. Three criteria were used, each one being coded by the values 0, 1, or 2 (increasing degradation). The first criterion focused on the transparency-opacity of the swimbladder wall. The value 0 was assigned to normal looking swimbladders (transparent-yellowish coloration, see

Clarke & Witkomb 1980). Total opacity (when no reading is possible throughout the swimbladder wall) was assigned a value of 2, and all intermediate examples a value of 1. The second criterion was the presence of pigmentation and exudate instead of gas in the swimbladder lumen (dead worms, erythrocytes, decaying swimbladder tissue, eggs, and L2 stage of *Anguillicola* spp.). Value 2 was assigned to swimbladders that exhibited both pigmentation and exudate, value 1 to those that showed either pigmentation or exudates, and value 0 for no pigmentation and no exudate. The last criterion concerned the thickness of the swimbladder wall, estimated visually. Value 0 was assigned to thin walled-swimbladders (<1 mm). Value 2 was assigned to severely affected swimbladders with little if any lumen left (more than 3 mm thick wall), and value 1 to all other examples. The cumulative index (i.e., SDI) thus can range from 0 when no pathological signs of infection are observed to 6 in examples of extremely damaged swimbladder.

We chose to quantify body condition using the residual index (i.e., the residuals of the regression of W on L), because other indices (such as Fulton's condition index, see Bolger & Connolly 1989 for complete references) do not adequately control for variation in body length (Jakob et al. 1996). The effect of infection on body condition was examined by comparing infected and uninfected eels.

Means are given ±SE. All statistical tests were performed with the STATISTICA 6.0 package.

RESULTS

Data regarding the origin of the different samples and the length and weight of the 189 eels examined are summarised in Table 1. Overall, samples consisted mainly of large individuals, with a length ranging from 430 to 940 mm (mean of 588 ± 6 mm) and a weight ranging from 169 to 1725 g (mean of 409 ± 16 g). The sex ratio was highly female biased in every sample, ranging from 89% to 100%, with a mean value of 95% of females. Very few eels met the ocular criteria of Pankhurst (i.e., ≥6.5) to be classified as silver individuals (7/189 = 3.70%). Thus, most eels examined for the presence of *Anguillicola* parasites were large immature females in their inland growth phase (so-called yellow eels).

Anguillicola taxonomy and occurrence

All 19 (14 adults, 5 juveniles) nematodes collected from the swimbladders of the New Zealand short-finned eel *Anguilla australis* were identified as *Anguillicola novaezelandiae*. This species can be differentiated from the congeneric *A. australiensis* (the host of which, *Anguilla reinhardtii*, is now established in New Zealand) by examining the shape of the head. *A. novaezelandiae* has a slightly expanded head end whereas this part of the body is clearly bulbous in *A. australiensis*. In addition, *A. novaezelandiae* is characterised by a very small buccal capsule with c. 35 minute circumoral teeth. The 14 adult *A. novaezelandiae* specimens (fixed in 70% ethanol) available had the following measurements (in mm):

Male (4 specimens): length of body 5.34–22.29, maximum width 0.214–1.553. Length of slightly enlarged head end 0.074–0.100, its width 0.065–0.094; width of body at neck constriction 0.054–0.076. Buccal capsule 0.007–0.008 long and 0.021–0.024 wide. Length of oesophagus 0.500–0.752, its maximum width 0.069–0.257. Length ratio of oesophagus and body 1 : 10.7–29.6.

Female (10 specimens): length of body 3.71–26.72, maximum width 0.175–3.511. Length of slightly enlarged head end 0.084–0.126, its width 0.065–0.125; width of body at neck constriction

Table 1 Sampling localities, date of dissection, sample size, total length (L) and somatic weight (W) of short-finned eels (*Anguilla australis*), prevalence and mean intensity of infection by *Anguillicola novaezelandiae*, and health state of the swimbladder (SDI). Localities are given by New Zealand districts unless further geographical details were provided by local fishers.

Locality	Date	*N*	Mean L (mm)	Mean W (g)	Prevalence (%)	Mean intensity	Mean SDI score
Canterbury	10 Oct 2003	49	558±12	355±36	2.0	5.0	0.51±0.10
Canterbury	12 Dec 2003	47	555±11	374±31	10.6	1.8±0.3	0.85±0.15
Bay of Plenty	15 Oct 2003	45	653±11	520±37	4.4	1.0±0.0	0.38±0.09
Waikato (Te Awamutu)	12 Dec 2003	25	588±10	382±17	12.0	1.0±0.0	0.44±0.17
Waikato (Whangamarino)	12 Dec 2003	23	591±16	406±32	0.0	–	1.13±0.27
Total		189	588±6	409±16	5.8	1.7±0.4	0.63±0.07

0.054–0.096. Buccal capsule 0.006–0.008 long and 0.021–0.027 wide. Length of oesophagus 0.450–0.723, its maximum width 0.079–0.257. Length ratio of oesophagus and body 1 : 8.2–38.5.

Overall, prevalence of infection was rather low, ranging from 0% (in the Whangamarino swamp, Waikato) to 12% (in the Te Awamutu ponds, Waikato). The majority of infected eels harboured a single lumen worm, and the maximum was five worms in one eel from the Canterbury area (see Table 1).

Health state of the eel swimbladder

The highest individual SDI score was 4, but most fish presented little if any sign of degradation in their swimbladder. The proportion of fish with SDI scores of 0, 1, 2, 3, and 4 was 56.6%, 30.7%, 8.5%, 1.6%, and 2.6%, respectively. In the most severe examples, the transparency of the swimbladder was reduced and/or the thickness of the swimbladder wall was increased, but exudates and detritus of dead worms were never observed to fill the swimbladder lumen. No significant difference was detected between the seven silver eels and the others with respect to the health state of their swimbladder (1.14 ± 0.26 versus 0.61 ± 0.07; *t*-test for independent samples: $t = -1.53$, d.f. = 187, $P = 0.13$).

Among samples, the mean SDI score ranged from 0.38 (Bay of Plenty area) to 1.13 (Waikato area, Whangamarino swamp) (see Table 1). Overall, the mean SDI score was 0.63 ± 0.07. The mean SDI score of infected eels (1.36 ± 0.43) was significantly higher than the SDI score of uninfected ones (0.58 ± 0.07) (Mann-Whitney *U* test: *z* adjusted = –2.22, $P < 0.05$); thus, the swimbladder of infected eels was more likely to show signs of deterioration than that of uninfected eels. However, among infected fish only, there was no significant relationship between the intensity of infection and the SDI score (Spearman Rank test: $R = -0.09$, $N = 11$, $P = 0.80$).

Body condition of eels

Infected eels were smaller than uninfected ones, though the difference was not statistically significant (for L: 540 ± 18 mm versus 591 ± 6 mm, *t*-test for independent samples: $t = 1.95$, d.f. = 187, $P = 0.053$; for W: 313 ± 26 g versus 415 ± 17 g, $t = 1.47$, d.f. = 187, $P = 0.14$).

No significant difference in the residuals of the regression W versus L was detected between infected and uninfected eels (*t*-test for independent samples: $t = -0.82$, d.f. = 187, $P = 0.42$).

DISCUSSION

The short-finned eel *A. australis* in New Zealand appears to be infected only by one species of swimbladder parasite, the indigenous *A. novaezelandiae*. As far as is presently known, no specimen of *A. australiensis* has ever been recorded in New Zealand, though its host, the Australian speckled eel *Anguilla reinhardtii*, has become established in New Zealand in the last 10 years (McDowall et al. 1998). Similarly, the Asian *A. crassus*, a worldwide coloniser of various eel species, has still not been found in New Zealand waters. Though occurring at low prevalence in general, infection by *A.*

Table 2 Review of *Anguillicola novaezelandiae* distribution and prevalence in the short-finned eel (*Anguilla australis*) in New Zealand. In early publications (Rid 1973; Hine 1978), this parasite was originally reported as *Anguillicola australiensis*, before Moravec & Taraschewski (1988) introduced and described the new species *A. novaezelandiae*. (NI and SI refer to North and South Island of New Zealand, respectively.)

Locality	Date of investigation	Prevalence, % (infected/*N*)	Source
Waimakariri River (Canterbury, SI)	May 1970	4.9 (2/41)	Rid (1973)
Bockets Stream (Wairarapa, NI)	Dec 1972	rare	Hine (1978)
Lake Pukepuke (Manawatu, NI)	Dec 1972	6.3	Hine (1978)
Lake Ellesmere (Canterbury, SI)	Oct 1974 June 1976	rare	Hine (1978)
Rangitaiki River (Bay of Plenty, NI)	Jan 1975	72.7 (8/11)	Hine (1978)
Matahina Dam (Bay of Plenty, NI)	May 1975	–	Moravec and Taraschewski (1988)
Canterbury (SI)	Oct–Dec 2003	6.2 (6/96)	present study
Bay of Plenty (NI)	Oct 2003	4.4 (2/45)	present study
Te Awamutu ponds (Waikato, NI)	Dec 2003	12.0 (3/25)	present study

novaezelandiae seems widespread throughout the country (see Table 2 for review). It has been found on both North and South Island, in lakes and rivers, mainly close to the coast. Based on data from Rid (1973), Hine (1978), and the present study, *A. novaezelandiae* has been recorded in 6 of the 30 localities so far investigated (20%).

In Europe, infection of eels *Anguilla anguilla* by *Anguillicola crassus* is a major problem. The direct damage caused by the nematode (Molnár et al. 1993; Würtz & Taraschewski 2000) leads to the altered gas compositions in the lumen of infected European eels (Molnár 1993; Würtz et al. 1996). It remains uncertain, however, whether these alterations negatively affect the spawning migration of the infected eels and thus the reproductive success of *Anguilla anguilla*. Recent experiments conducted by Münderle et al. (2004) did not support the hypothesis that parasites reduce the migratory ability of eels (but see Sprengel & Lüchtenberg 1991). However, low oxygen concentrations in aquatic systems cause higher mortality in infected than uninfected eels (Molnár et al. 1991; Molnár 1993; Barus & Moravec 1999), and heavily-infected eels are more susceptible to secondary bacterial infections (Van Banning & Haenen 1990).

The *Anguilla-Anguillicola* host-parasite system in New Zealand appears to be very different from that observed in Europe, for at least three reasons. First, the prevalence of infection by *A. novaezelandiae* is very low. In this study, among four samples obtained from different geographical areas, the highest prevalence was 12% (Te Awamutu ponds, Waikato). In previous investigations, except for the Rangitaiki River (Bay of Plenty) with a reported prevalence of 73% (Hine 1978), all localities investigated thus far had prevalence values below 10% (see Table 2). Thus infection rates by *A. novaezelandiae* are low in comparison with those observed in Europe, where prevalence often reaches 60–90% a couple of years after the introduction of *A. crassus* in a water system (see for example Kennedy & Fitch 1990; Würtz et al. 1998; Sures & Streit 2001; Lefebvre et al. 2002b).

Second, the intensity of infection is also rather low in comparison to data recorded in Europe. From previous investigations in New Zealand, little information is available regarding the mean number of parasites per infected eel. In the present study, the mean intensity, based on 11 infected eels, ranged between one and two parasites. Such a low parasite load, in conjunction with the relatively small size of adult worms (6–15 mm in males, 10–31 mm in females, see Moravec & Taraschewski 1988), suggests a low parasite pressure exerted on eels, especially with respect to the blood losses caused by the haematophagous activities of the parasite.

Third, our data show that the damage caused by *A. novaezelandiae* to the swimbladder tissues was minimal. The mean SDI score of the 189 eels examined was 0.63 on a scale ranging from 0 to 6. A very small proportion of eels exhibited moderate degradation of their swimbladder (SDI score of 3 and 4: 8/189 = 4.2%), and thus most of the fish had healthy swimbladders. In Europe, the mean SDI index is generally higher than 2, and ⅓ of the population may be suffering from severe lesion and inflammation, sometimes leading to the total disappearance of the swimbladder lumen (Lefebvre et al. 2002a, 2004).

In conclusion, the short-finned eel does not seem to be greatly impaired by the presence of *A. novaezelandiae*. No impact on body condition can be detected and the swimbladders of all eels seem to be fully functional. This is particularly relevant for the silvering individuals that will have to undertake a long reproductive migration (more than 2000 km at great depth) to reach their spawning grounds in the Pacific (thought to be in the warm tropical seas between Fiji and Tahiti).

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