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Review

The pathogenic helminth parasites of eels

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Abstract

Although 63 and 55 species of helminths have been reported from each species of Atlantic eel and from 29 to 19 for each species of Pacific eel only the monogeneans *Pseudodactylogyrus bini* and *P. anguillae* and the nematode *Anguillicola crassus*, originally specific to species of Pacific eels, can be considered serious pathogens. None of the three are normally pathogenic to their preferred natural eel host species in the wild. *Pseudodactylogyrus* spp. only cause serious local gill damage when present on a host in large numbers under optimal conditions that facilitate transmission. This is the case in eel aquaculture, where infections can be controlled by drugs. *Anguillicola crassus* is only pathogenic to *Anguilla anguilla* and *A. rostrata* when Atlantic eels are introduced to the far east or when the parasites have been introduced to Europe. Here the parasite life cycle differs in that *A. crassus* can infect a wide range of intermediate hosts, employ paratenic hosts and survive as larvae for months in the swimbladder wall. This makes it an excellent colonizer. Its major pathogenic effects on eels result from haemorrhaging in, and thickening of, the swimbladder wall. It reduces the oxygen concentration in the swimbladder, reducing its ability to function as a hydrostatic organ, and increases the stress response of eels. In shallow lakes at warm temperatures this can result in mass mortalities. It is also feared that the parasite affects the ability of eels to migrate to the Sargasso Sea and so contributes to the decline in eel populations. Control by drug treatment is possible in culture, but not in the wild.

Keywords: *Anguilla* spp., *Anguillicola crassus*, aquaculture, introductions, pathogenicity, *Pseudodactylogyrus* spp.

Introduction

Eels, *Anguilla* spp., are widespread throughout the freshwaters and coastal lagoons of the North Atlantic and the Pacific seaboards, and they are important natural components of the fish fauna in both lakes and rivers. They are also an important economic resource. They are farmed in ponds throughout much of their range in Europe and East Asia; they are stocked into lakes in central Europe; elvers are caught along the eastern Atlantic seaboard for such stocking; Atlantic eels have been translocated for stocking purposes to East Asia and Pacific eels have been introduced into Europe; and adult Australian eels are flown regularly to Hong Kong markets. They are thus the basis of several economically important fisheries.

Despite their importance, little is known about their parasites by comparison with the extensive information available on the parasites of salmonids in both the wild and in farms or of species of carp. Indeed, it is only because the translocations of eels have of necessity involved translocations of their parasites and some species of these have caused problems in aquaculture that interest in the parasite fauna of eels has been stimulated. However, the reality is that most of our information on eel parasites relates to the two species of Atlantic eels, *Anguilla anguilla* (L.) and *A. rostrata* (Lesueur). Information on parasites of Pacific eels is incomplete

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for some species and non-existent for others, and it is still not possible to compile a check list of parasites of *A. japonica* Temminck & Schlegel despite this being the subject of extensive aquaculture.

The aim of this review is therefore to focus on those species of helminth parasite that have been identified as potential or actual pathogens of eels. It then goes on to evaluate the damage they can cause to individual eels, to eel populations and to eel stocks in the wild and in aquaculture and to consider whether treatments are effective, commercially viable and ecologically acceptable.

Helminth parasites of eels – a perspective

Representatives of all groups of helminths can be found in eels, but the species list is longer for the Atlantic eels (Table 1). This may reflect the number of studies undertaken on each species rather than any fundamental difference in species richness between Atlantic and Pacific eels. There is little information available on the helminths of many species of Pacific eels including *A. australis* Richardson and *A. dieffenbachii* Gray and information on parasites of *A. japonica*, one of the best studied species, is incomplete. Although the numbers of species recorded from Pacific eels is lower than those from Atlantic eels, the parasite fauna of individual *A. reinhardtii* Steindachner, is on average richer than that of *A. anguilla* and *A. rostrata* (Kennedy 1995; Marcogliese & Cone 1998).

Overall, only a very small number, maximum four for any species of eel, of helminth species have been identified as real or potential pathogens. This represents a very small proportion of the total parasite fauna, between 5% and 14%. As far as is known, all the remaining species are harmless, or only cause very local tissue damage, for example acanthocephalans in attaching to the intestinal wall. Many species of *Gyrodactylus* are known to cause serious damage to their fish hosts but there is no convincing evidence that *G. nipponensis* (on Japanese eels) or *G. anguillae* (on Atlantic eels) are important pathogens of eels even though individual host densities may be high (Malmberg 1970; Huu-Yun, I-Hsiung & Guang-Hsiung 1984). Indeed, the range and local abundance of both these species have declined substantially in recent years, probably as a direct consequence of inter-specific competition with *Pseudodactylogyrus* species (Huu-Yun *et al.* 1984; Kennedy & Di Cave 1998).

The pathogenic species can be divided into two groups, those that have been confirmed as pathogens and those that are suspected of being capable of being pathogenic. *Philometroides anguillae*, for example, is a large nematode that is found in the pericardium and heart muscle of wild *A. reinhardtii*. Up to 10 specimens have been recorded from a single eel (Kennedy 1995), and it seems impossible that the heart of such an infected eel would be undamaged and able to function normally. Species of *Eustrongylides* are found regularly in Pacific eels,

Table 1 Number of species of helminth parasites of eels (*Anguilla* spp.) and identity of those reported as actually or potentially pathogenic

North America		Europe	Australia	New Zealand	
A. rostrata		*A. anguilla*	*A. reinhardtii*	*A. australis*	*A. dieffenbachii*
Mo	3	4	4	0	0
Di	20	16	10	8	6
Ce	5	6	2	1	1
Ne	16	21	12	7	11
An	19	8	1	2	1
Σ sp.	63	55	29	19	19
Pa. Mo	*P. anguillae* *P. bini*	*P. anguillae* *P. bini*	*P. anguillae*		
Di	*S. tenuae*				
Ne	*A. crassus* *Eustrongylides*[a]	*A. crassus*	*Eustrongylides*[a] *Ph. anguillae*[a]	*Eustrongylides*[a]	
References	Hoffman (1999)	Borgsteede, Haenen, De Bree & Lisitsina (1999)	Kennedy (1995)	Hewitt & Hine (1972)	

Mo, Monogenea; Di, Digenea; Ce, Cestoda; Ne, Nematoda; Ac, Acanthocephala; Σsp., total number of species; Pa, identity of pathogenic species. Sources as indicated, but subsequently updated where appropriate.

[a]Suspected.

at intensities of up to 26 per eel (Kennedy 1995), but are uncommon in Atlantic eels, which appear to be accidental hosts for them. They may not be pathogenic as such, but large cysts in the body cavity are unsightly and may put off customers, particularly if the larval nematodes, which are bright red and can be several centimetre long, have emerged from the cysts *post mortem*.

The only digenean reported as being pathogenic is *Stephanostomum tenuae*. Metacercariae of this species have been reported from the pericardium of elvers of *A. rostrata* by Oliveira & Campbell (1998). Up to 92% of the elvers can be infected in a locality. The pericardium becomes inflamed, the thorax may be distended and swimming of an infected eel may be impeded.

The remaining species, *Pseudodactylogyrus anguillae*, *P. bini* and *Anguillicola crassus* are specialists of eels. It is considered by many authorities that *Pseudodactylogyrus* spp. were originally parasites of Pacific eels that have been introduced into Europe and America through the eel trade (Buchmann, Mellergaard & Køie 1987a; Buchmann 1993; Hayward, Iwashita, Crane & Ogawa 2001). However, this view has been challenged by Nie & Kennedy (1991), Marcogliese & Cone (1993) and Cone & Marcogliese (1995). These authors have suggested that the presence of *P. anguillae* in regions to which eels have not been introduced, e.g. maritime Canada, southwest England and the western coast of Ireland may indicate that this species is a natural parasite of American and European eels. It is not an introduced species, but may have been present but undetected in Atlantic eels for some time. It does not appear to be pathogenic to wild Atlantic eels, or to native Australian eels (Kennedy 1995), supporting the view that this species has had a long history of co-evolution with its eel hosts. The question of the origin of *P. anguillae* remains open, but there is widespread agreement that *P. bini* has been introduced to Europe and North America with the eel trade.

There is no doubt, however, that *A. crassus* has recently been introduced into Europe (Kennedy & Fitch 1990) and America (Fries, Williams & Johnson 1996; Barse & Secor 1999; Moser, Patrick & Crutchfield 2001) from East Asia, where its normal host appears to be *A. japonica*. It is not pathogenic to this host, but only to the two species of Atlantic eel. The remainder of this review will therefore focus on these three species of helminth.

Pseudodactylogyrus species

Biology

The two species of *Pseudodactylogyrus* are very similar in appearance and can be very difficult to separate. Their life cycles are similar and typical of those of most Monogenea: shed eggs hatch to produce oncomiracidia, which are short lived and need to find an eel before they can develop further and into an adult. They both parasitize the gills of eels and can co-occur on the same eel. The distribution of the two species on the gills does differ, and even though they may overlap, each has a preferred site with *P. bini* preferring the anterior gill arches and *P. anguillae* the posterior ones (Buchmann 1988, 1993; Rodrigues & Saraiva 1996). They differ slightly in their ecology, in that *P. bini* is less able to tolerate salinity than *P. anguillae*, which can reproduce in waters of salinity up to 20‰ although not in sea water itself. *Pseudodactylogyrus bini* needs higher temperatures for reproduction: it cannot develop at 10 °C but at 25 °–34 °C it produces more eggs than *P. anguillae*. *Pseudodactylogyrus bini* overwinters as eggs, whereas *P. anguillae* may overwinter on gills and is generally the commoner species (Køie 1991). There is a positive correlation between the body length of an eel and the parasite intensity: small eels may harbour up to 200+ parasites such that the whole surface of the gills is covered by them, whereas infection levels in larger eels may exceed 1000+ (Huu-Yun *et al.* 1984; Buchmann 1989). The species exhibit pronounced seasonal cycles in abundance and prevalence, with levels peaking in late summer and reaching a nadir over winter (Nie & Kennedy 1991). The optimum temperature for hatching and development is around 20 °–25 °C, at which generation time may be only 10–12 days, and this is also a favoured temperature for eel culture ponds (Køie 1991; Buchmann 1993).

Dispersal

The two species are considered to have been disseminated widely throughout Europe by movements of eels for stocking and aquaculture purposes. If both have been introduced from East Asia with imported *A. japonica*, as is believed by some authorities (e.g. Køie 1991), the introduction appears to be independent of that of *A. crassus* to Europe and America as they were detected before *A. crassus* on both continents and in natural

localities where there had been no stocking of eels (Nie & Kennedy 1991; Cone & Marcogliese 1995), and there are no reports of damage to their eel hosts. Both species have been reported from the USA, where they are regarded as recent invaders spread by the eel trade and natural eel movements (Hayward *et al.* 2001). They are widespread throughout Europe, and they are very easily disseminated from farm-to-farm with eels themselves (Mo, Håstein & Lunder 1988; Mo & Sterud 1998). In Scandinavia, *P. anguillae* is considered a plague in Norwegian eel farms by Mo *et al.* (1988), who believe that it may have been introduced to the country by fishing boats from other Baltic countries or by natural eel movements around the Baltic Sea.

Pathology

The parasites appear to cause few, if any, problems to wild eels. They may cause tissue damage and impaired respiration, and signs of stress (Køie 1991), but they do not appear to cause direct mortality or to have any effect on eel migration. They are, however, considered to be a serious problem and to cause economic losses in eel farms, where intensity levels can be very high, in Taiwan where they infect *A. japonica* (Huu-Yun *et al.* 1984), in Europe where they infect *A. anguilla* (Buchmann *et al.* 1987a; Mo *et al.* 1988) and in China where they infect introduced *A. anguilla* (Zhang 1995). In Japan, they infect *A. anguilla* elvers introduced into farms, where heavy infections in summer lead to infected individuals stopping feeding and exhibiting abnormal behaviour before death (Egusa 1979). Both species feed on mucus and gill epithelia and blood (Buchmann, Køie & Prentø 1987b) and their hamuli cause bleeding and damage (*P. anguillae*) or provoke a host reaction (*P. bini*) (Køie 1991). There is very little evidence of resistance to re-infection and there is only a very weak immune response (Buchmann & Bjerregaard 1990; Buchmann 1993). High infection levels lead to hyperplasia of gill tissues and fusion of secondary lamellae, and in some cases to hyperanaemia. Eels infected with several hundred or more individuals swim lethargically at the surface and seek areas of reduced water current (Køie 1991). If untreated, host death may result. The effects of both species on *A. rostrata* in North America, where they are regarded as serious pests that retard production in eel rearing farms and which may lead to morbidity and mortality, appear to be very similar to those on *A. anguilla* in Europe (Hayward *et al.* 2001).

Control

Pseudodactylogyrus infections are a major problem in fish farms where heated water is re-cycled. In some cases, it may be possible to reduce transmission rates and so infection levels by non-chemical methods such as changing water temperature, flow rates and pH (Barker & Cone 2000). However, in farm conditions chemical methods of treatment are generally more appropriate and effective. Formalin was the initially recommended treatment, but the levels at which is was effective were unpredictable and it is now considered a dangerous chemical. Praziquantal has been shown to be very effective against the parasites (Buchmann, Székély & Bjerregaard 1990) as has mebendazole (Székely & Molnár 1987), although drug resistance may develop (Buchmann & Bjerregaard 1990; Buchmann 1993).

Anguillicola species

In Pacific eels

Five species of *Anguillicola* are currently known (Table 2). All five are native and specific to species of Pacific eels (Table 2), to which they are so well adapted that they cause no damage. Their basic life cycles, in so far as they are known, are very similar (Kennedy 1994a; Moravec, Di Cave, Orecchia & Paggi 1994a; Nagasawa, Kim & Hirose 1994; Taraschewski, Boomker, Knopf & Moravec 2005). Adults are found in the swimbladder of a species of Pacific eel, and feed on blood. Eggs are released into the swimbladder and contain the second larval stage encased in the sheath of the first stage larva. The larvae may hatch in the swimbladder, or not until the eggs are passed out through the pneumatic duct of the eel and into water via the intestine. The free-living larvae attach to stones or any other hard surface, where they undulate in response to tactile or other similar stimuli: this is considered to make them more attractive to feeding copepods. Larvae are ingested by one or more species of cyclopoid copepods and pass into the haemocoel, where they moult to the third larval stage. Infected copepods are eaten by eels, when the larvae pass rapidly out of the intestine and migrate through the intestinal wall (Wang & Zhao 1980) and the wall of the

Table 2 Distributions and hosts of species of *Anguillicola*

Species	Distribution	Hosts			Source
		Intermediate	Paratenic	Definitive	
A. crassus	Japan, China, Korea, Taiwan	Two spp. of cyclopoid copepod	None	*A. japonica*, *A. anguilla*[a]	Nagasawa *et al.* (1994)
	Europe[a]	Nine spp. of cyclopoid copepod, ostracod	20[b] spp. of fish, snail, amphibia	*A. anguilla*	Thomas & Ollevier (1992), Moravec & Konecny (1994), Szekely (1994), Moravec (1996)
	N. America	Copepod[b]	Unknown	*A. rostrata*	Moser *et al.* (2001)
A. globiceps	Japan, China	Six spp. of cyclopoid copepod	None	*A. japonica*	Nagasawa *et al.* (1994)
A. australiensis	Australia	Copepod[b]	None	*A. reinhardtii*	Kennedy (1994c)
A. novaezelandiae[c]	New Zealand, Australia	Copepod[b]	None	*A. australis*, *A. diffenbachii*	Moravec & Taraschewski (1988)
	Italy[a,d]	Copepod	None	*A. anguillae*	Moravec *et al.* (1994a)
A. papernai	S. Africa	Copepod[b]	Unknown	*A. mozambica*	Moravec & Taraschewski (1988), Taraschewski *et al.* (2005)

[a]Introduced.
[b]Presumed.
[c]Originally, e.g. Hewitt & Hine (1972) and Rid (1973), identified as *A. australiensis* but following the taxonomic revision of Moravec & Taraschewski (1988) it was recognized as a new species *A. novaezelandiae* and was subsequently also reported by Moravec & Rohde (1992) from Australia in the eel *A. australis*. It has also been reported from *A. diffenbachii* in New Zealand, but this is possibly a misidentification for *A. australiensis* which is otherwise considered to be a native of Australia and specific to *A. reinhardtii* (Moravec & Rohde 1992).
[d]Originally reported as *A. australiensis* from Lake Bracciano by Paggi *et al.* (1982), having been introduced with *A. australis*. It was subsequently confirmed as *A. novaezelandiae* by Moravec *et al.* (1994a).

swimbladder into the lumen, where they moult to the fourth larval stage and then to the adult. If delayed in their passage through the swimbladder wall, the larvae are destroyed (Kennedy 1994a).

As far as is known, none of the Pacific species can employ paratenic hosts in their life cycle (Puquin & Yuru 1980; Wang & Zhao 1980; Nagasawa *et al.* 1994) and none are pathogenic to their natural host (Hine & Boustead 1974; Wang & Zhao 1980; Kennedy 1994a; Taraschewski *et al.* 2005). *Anguillicola globiceps* appears to occur only in wild *A. japonica* and may cause some thickening of the swimbladder wall, and it is more tolerant of cold temperatures than *A. crassus* (Nagasawa *et al.* 1994). In Japan, *A. crassus* is found in both wild and farmed *A. japonica*, but although intensities are higher in cultured eels it does not appear to cause any damage to wild or cultured eels (Münderle, Taraschewski, Klar, Chang, Shiao, Shen, He, Lin & Tzeng 2006). However, when *A. anguilla* was introduced into Japanese aquaculture ponds to increase stock production, it was found that both prevalence and intensity of infection were higher in this introduced species than in *A. japonica*, and furthermore that the parasite was pathogenic to, and caused mortality in, *A. anguilla* (Egusa 1979; Egusa & Hirose 1983). Subsequent experiments (Knopf & Mahnke 2004) have confirmed that recoveries of *A. crassus* are higher, 33.2%, in *A. anguilla* than in *A. japonica*, 13.8%, in which development of the parasite is slower, adults are smaller and a greater proportion of larvae are encapsulated in the swimbladder wall and die. Clearly, *A. japonica* (Nielsen 1999) mounts more effective immune responses to *A. crassus* than does *A. anguilla* and apparently infrapopulations of *A. crassus* in Japanese eels are regulated by the defence system of this host (Knopf & Mahnke 2004; Münderle *et al.* 2006). Despite Egusa's warnings about translocating eels from Japan to western Europe, *A. crassus* first appeared in Europe around 1980 and has subsequently spread (Peters & Hartmann 1986; Taraschewski, Moravec, Lamah & Anders 1987; Moravec & Taraschewski 1988; Kennedy & Fitch 1990) through the eel trade throughout the continent and into North Africa and subsequently to North America (Barse & Secor 1999; Moser *et al.* 2001).

Other species have also been introduced into Europe. *Anguillicola novaezelandiae* [originally identified as *A. australiensis* by Paggi, Orecchia, Minervini & Mattiucci (1982)] was introduced into Lake Bracciano in Italy in 1975 by imported *A. australis*. It survived there for several years,

reaching prevalence levels of 80% and intensities of up to 27, but caused no damage to the native *A. anguilla* and, as far as is known, never escaped from this isolated habitat to other lakes. In recent years, its numbers have declined following the introduction of *A. crassus* into the lake in 1993 (Moravec *et al.* 1994a). It has also been confirmed experimentally that *A. novaezelandiae* can infect *A. anguilla* (Knopf & Mahnke 2004). It seems likely that *A. globiceps* must also have been introduced to Europe with Pacific eels, but there are no reports of its having established there. The absence of any pathogenic effects of these two species on *A. anguilla* is not readily explicable.

Since, therefore, only *A. crassus* appears to be an important pathogen of Atlantic eels, the remainder of this section will focus on this species.

Anguillicola crassus in Atlantic eels

Changes in life cycle

Accidental introduction of *A. crassus* into Europe and its subsequent success and rapid spread throughout the continent are associated with significant differences in its life cycle. In the first place, it shows much wider specificity to its first intermediate host as it is able to infect several species of freshwater cyclopoid copepods (Table 2) as well as estuarine copepods such as *Eurytemora affinis*. This enables it to complete its life cycle in freshwater lentic and lotic habitats, as well as in estuaries and saline lagoons.

Secondly, it employs paratenic hosts in the life cycle. At least 20 species of freshwater fish as well as snails, amphibians and insects have been identified as paratenic hosts for the third larval stage (Thomas & Ollevier 1992; Moravec & Konecny 1994; Székely 1994; Moravec 1996; Moravec & Škoriková 1998). Not all are of similar suitability: in some species only a few larvae survive as the host reacts against them, whereas in others, especially species of Perciformes, larvae appear to develop to a fourth larval stage (Pazooki & Székely 1994; Székely, Pazooki & Molnár 1996). Thus, young eels may become infected by ingesting infected copepods and older and larger eels by ingesting infected fish. Thirdly, when the larvae reach the swimbladder wall, they do not move rapidly through it as they do in Pacific eels but remain in the wall where they moult to the fourth larval stage. They may remain there for some time before moving into the lumen of the swimbladder and moulting to the adult stage.

Biology and ecology

At first sight, *A. crassus* does not appear to immediately possess many of the attributes of a good colonizer as set out in Kennedy (1994b). These are good dispersal ability, a high reproductive potential and asexual reproduction or hermaphroditism. In respect of parasites, species with direct life cycles and wide host specificity are likely to be more successful invaders, and success is likely to be improved if biotic and abiotic conditions are similar in the source locality and the locality being invaded, and if there are vacant niches in the invaded community. The species does not have a particularly high reproductive potential, the sexes are separate and it has an indirect life cycle. However, it is *r*-selected, it has wide specificity to intermediate and paratenic hosts and although it is very specific to *A. anguilla* as definitive host, the eel itself is very versatile, adaptable and widespread in its distribution. Conditions throughout much of Europe must also be similar to those in Japan and China. Furthermore, the swimbladder of the eel represented a vacant niche for the parasite.

Any limitations in natural dispersal ability were more than compensated for by the anthropochore movements of eels around the continent and across the seas as a consequence of the eel trade (Kennedy & Fitch 1990).

Studies on the life cycle of *A. crassus* in Europe by Haenen, Grisez, De Charleroy, Belpaire & Ollevier (1989); De Charleroy, Grisez, Thomas, Belpaire & Ollevier (1990); Kennedy & Fitch (1990); Höglund & Thomas (1992); Thomas & Ollevier (1992, 1993); Haenen, van Banning & Dekker (1994); Haenen, van Wijngaarden, van der Heijden, Höglund, Cornelissen, van Leengoed, Borgsteede, van Haenen & Muiswinkel (1996) and Moravec, Di Cave, Orecchia & Paggi (1993, 1994b) amongst others have shown that eggs of *A. crassus* do not hatch below 10 °C and the rate of hatching increases up to 25 °C–30 °C. Hatching rate is also related to salinity, but the percentage of eggs hatched, and survivorship and infectivity of the second larval stage declines as salinity increases (Kirk, Kennedy & Lewis 2000a). The majority of second stage larvae attached to the substrate within 2 or 3 days, and they may then survive for up to 8 months at 7 °C or 5 months at 24 °C.

Survivorship and infectivity decrease exponentially with time (Kennedy & Fitch 1990) and with increasing salinity, even though the parasite can survive in some saline lagoons (Di Cave, Berrilli, De Liberato, Orecchia & Kennedy 2001). The whole life cycle is temperature related and is retarded at low temperatures. Third stage larvae may survive 4 months at 4 °C in copepods, although they may become unable to infect eels, and adult mortality increases over 4 months at the same temperature (Knopf, Würtz, Sures & Taraschewski 1998). Inside paratenic hosts third stage larvae can survive for longer periods and remain infective (Székely 1996) The preference for warmer temperatures shown by *A. crassus* may explain why it is uncommon in, or absent from, the more northern boreal regions (Höglund & Thomas 1992; Thomas & Ollevier 1992, 1993; Knopf *et al.* 1998), where it may be restricted to thermal effluents (Höglund, Andersson, Wickström & Reizenstein 1992). The whole cycle can be completed in 90 days at suitable temperatures, but will normally take longer, at least 4 months (Haenen *et al.* 1989).

Glass eels (Nimeth, Zwerger, Würtz, Salvenmoser & Pelster 2004) and elvers are susceptible to infection, and eels become vulnerable to infection as soon as they commence feeding in rivers or estuaries. Infection can continue throughout life, and in general infection levels tend to be higher in older and larger eels. Eels can lose infections, but they are not immune to re-infection. There is no relationship between primary and secondary infections (Haenen *et al.* 1996) and higher doses of infection will normally produce more severe clinical signs. It was initially thought that there was no antibody response to the parasites (Békési, Hormok & Székely 1997) but later studies by Sures & Knopf (2004a) have shown using ELISA that the body wall of the parasite is a good antigen and significant levels of antibodies can be detected in the blood after 61 days. The response is suppressed by the initial rise in cortisol levels in all eels due to handling stress, which assists parasite establishment. In contrast to the situation in *A. japonica* where the host and *A. crassus* have co-evolved over a long period and the eel is able to mount an effective immune response to the parasite (Nielsen 1999; Münderle *et al.* 2006), *A. anguilla* is an immunologically naive host and unable to mount an effective immune reponse against this parasite (see Taraschewski 2006 for further discussion of this issue). Knopf, Naser, Van der Heijden & Taraschewski (2000a,b) have also reported a humoral response. Nielsen (1999) has shown that the antibody response of *A. japonica* to *A. crassus* is higher than that of *A. anguilla*, which suggests that an immune response may be involved in specificity if not in control of numbers.

Under natural conditions, infection levels in eels as measured by prevalence and abundance of *A. crassus* appear in several localities to increase and then reach an asymptote and level out over time. Ashworth, Kennedy & Blanc (1996) and Ashworth & Kennedy (1999) have identified three density-dependent regulatory processes that may be responsible for this situation. They have shown that there is significant parasite induced copepod mortality such that 50% of uninfected copepods survive for 30 days post-infection, whereas the equivalent survival time for infected copepods is only 12 days. Moreover, mortality of infected copepods is also density-dependent, and so heavily infected copepods are taken out of the system. In the eel, the intensity of gravid females per infrapopulation remains constant over time and is independent of the overall infrapopulation density, so that the proportion of gravid females declines as adult numbers increase. They also suggested that the presence of adult males and females in the swimbladder could inhibit the movement of fourth stage larvae from the swimbladder wall into the lumen and larvae were arrested in development in a density dependent manner. The mechanisms responsible for these regulatory processes on *A. crassus* infection levels in the adult eel have not been identified more precisely than this, but their existence does appear to be capable of limiting the parasite population size in infections in the wild.

Kennedy & Fitch (1990) determined that adult parasites could survive in *A. anguilla* for up to 4 weeks when the eel was kept in 100% sea water. Survivorship also declined in coastal lagoons of increasing salinity (Di Cave *et al.* 2001). Kirk *et al.* (2000a) and Kirk, Lewis & Kennedy (2000b) showed that some adults could survive and continue to produce eggs in eels in 50% and 100% sea water for up to 6 months, but around 10% of the adult parasites were damaged following exposure to high salinity. Kirk, Morritt, Lewis & Kennedy (2002) showed that the parasites are osmoconformers, achieving this by feeding on eel blood, but around 20% of the parasites could not tolerate the osmotic stress of living in eels in 100% sea water but died and disintegrated. It is thus possible that parasites of

freshwater origin can survive in eels in coastal lagoons and estuaries as well as during the eel's migration to the Sargasso Sea. The life cycle could also be completed in waters of enhanced (up to 50%) salinity by using estuarine copepods such as *Eurytemora affinis* as an intermediate host, but it was considered unlikely (Kirk *et al.* 2002) that the parasites could transmit in sea water, as most marine copepods were of the wrong size to serve as intermediate hosts. The ability of the parasite to survive in eels in the Baltic Sea (Höglund & Thomas 1992; Reimer, Hildebrand, Scharberth & Walter 1994) could thus be due to transmission there or to the survival of freshwater infections.

Effects on the eel host

Histopathological effects

Since eels of all sizes can be infected, it could be predicted that the effects of the parasite on its host will relate to the number of parasites present and the size of the eel. The most evident visual effects can be observed on the swimbladder. In glass eels, the labyrinth of the gas gland is reduced and resting oxygen consumption is increased (Nimeth *et al.* 2004). In small, < 17 cm long, and young eels, the signs of infection include dilation and congestion of blood vessels, haemorrhages, inflammation and thickening and fibrosis of the swimbladder wall (Haenen *et al.* 1989, 1994; Molnár, Székely & Baska 1991; Molnár, Baska, Csaba, Glávits, Székely & Perényi 1993; Molnár, Székely & Perényi 1994; Molnár 1994). There may be an increase in the spleen mass (Lefebvre, Mounaix, Poizat & Crivelli 2004). In older eels and at higher infection intensities, the swimbladder wall may be thickened up to 10× normal; the mucosal epithelium becomes hyperplastic and the propria may be filled with dilated blood vessels. Granulation occurs around the larvae. The lumen of the swimbladder may become filled with a cloudy fluid containing degenerated larvae, eggs and pieces of decayed adults. The swimbladder wall continues to thicken, often up to as much as 3–4 mm, and becomes haemorrhagic: the effects relate to the intensity of the infection (Molnár *et al.* 1993; Haenen *et al.* 1996; Würtz & Taraschewski 2000). Similar effects are evident in *A. rostrata* (Barse & Secor 1999). These effects are summarized in Table 3.

Table 3 A summary of the effects of *Anguillicola crassus* on *Anguilla anguilla*

(a) Histopathological effects on the swim bladder[a]
- Fibrosis and thickening of the swimbladder wall
- Dilation of blood vessels
- Inflammatory and oedematous lesions in mucosa and submucosa
- Development of haemorrhages
- Infiltration of inflammatory cells and formation of granulation tissue

(b) Physiological effects[b]
- Dysfunction of the swimbladder due to impairment of the gas gland and deposition mechanisms
- Reduction of the contribution of oxygen to the swimbladder by 36–63%
- Reduction of swimming ability of eels
- Reduced ability of the swimbladder to function as a hydrostatic organ during migrations
- Increased stress responses in eels
- Reduced toleration of eels to unfavourable environmental conditions

(c) Population effects[c]
- Causes infected eels to be selectively captured
- Causes mass mortality of eels in ponds in summer
- May affect ability of adults to migrate to Sargasso Sea for spawning

[a]*Sources*: Taraschewski *et al.* (1987); Molnár *et al.* (1993); Würtz & Taraschewski (2000).
[b]*Sources*: Sprengel & Lüchtenberg (1991); Würtz *et al.* (1996); Kelly *et al.* (2000); Sures, Knopf & Kloas (2001); Gollock *et al.* (2005a,b).
[c]*Sources*: Molnár *et al.* (1991); Bálint, Ferenczy, Kátai, Kiss, Krécsei, Kufcsák, Láng, Polyhos, Szabó, Szegletes & Németsók (1997); Kirk *et al.* (2000b, 2002); Palíková & Navrátil (2001); Gollock *et al.* (2004); Sures & Knopf (2004b).

Physiological effects

There are several other, albeit less visible, effects of *A. crassus* on *A. anguilla*. Boon, Lokin, Ceusters & Ollevier (1989) initially detected no significant difference in haematocrit and plasma proteins in infected eels but later studies (Boon, Augustijn, Cannaerts, Lokin, Machiels & Ollevier 1990a; Boon, Cannaerts, Augustijn, Machiels, De Charleroy & Ollevier 1990b), using more rigorous controls, found a significant difference. Baruš, Kráčmar, Tenora & Prokeš (1998) and Baruš, Tenora, Kráčmar & Dvořáček (1999b) demonstrated lower levels of methionene and aspartic acid in the muscles of infected eels, and significantly lower levels of muscle Ca, P, Fe and Mn. Scholz & Zerbst-Boroffka (1994) have determined that *A. crassus* is iso-osmotic with the eel body fluids, but that there are ionic differences in eel chloride levels in sea water composition. These ionic and osmotic changes impose stress on the parasites which are ionic and osmotic conformers.

Molnár (1993) demonstrated that when uninfected and infected eels were deprived of oxygen, the severely infected eels died first. The impact of the oxygen shortage was temperature dependent and the effect on individual eels related more closely to the thickening of the swimbladder wall rather than to the number of parasites present.

Infected eels had an increased demand for oxygen, but the presence of *A. crassus* impaired the functioning of the swimbladder and this in turn could result in eel death.

Würtz, Taraschewski & Pelster (1996) were able to demonstrate that there was in fact a significant correlation between the oxygen concentration in the swimbladder and the level of *A. crassus* infection. The contribution of oxygen to the swimbladder gas was reduced by between 36% and 60% in naturally infected eels, and this also related to the changes in the swimbladder wall. Overall, the presence of parasites impeded the function of the swimbladder as a buoyancy and hydrostatic organ by impairing the functioning of the gas gland.

Sprengel & Lüchtenberg (1991) showed experimentally that there was a decrease in swimming performance by infected eels, which could be as severe as a 19% reduction in maximum swimming speed. Münderle, Sures & Taraschewski (2004), however, failed to demonstrate an adverse effect of *A. crassus* on the swimming activity of elvers of *A. anguilla*. Any change in swimming performance of infected eels would explain infected why they are easier to catch (Gollock, Kennedy, Quabius & Brown 2004) and why they were more easily sucked into power station intakes (Thomas & Ollevier 1992).

Kelly, Kennedy & Brown (2000) could find no significant differences in stress hormones or metabolic hormones or osmoregulatory status of infected eels and concluded that eels were able to adapt to chronic infection levels. However, Gollock *et al.* (2004) found that infected eels were more stressed under aquaculture and when netted and when exposed to air. They found that the cortisol response did not differ between infected and uninfected eels but plasma glucose levels were higher in infected eels and glucose metabolism and utilization was increased, i.e. stress of infection elevated glucose turnover. In a later study, Gollock, Kennedy & Brown (2005a) showed that acute temperature alone had little effect as an eel stressor, but under such conditions there was a lag in glucose metabolism in infected eels and there was no significant increase in haemoglobin levels when compared with the responses of uninfected eels as both groups showed a significant increase in haemoglobin. Gollock, Kennedy & Brown (2005b) then went on to demonstrate that infected eels exhibited a more pronounced stress response to hypoxia than uninfected individuals.

Despite these effects upon the stress responses of infected eels, several workers have found that the length/weight relationship does not differ between infected and uninfected eels and that any difference in weight between infected and uninfected eels is not significant even if, paradoxically, the eels are suffering mortality due to the parasite (Barus & Prokeš 1996). Koops & Hartmann (1989) also found no difference in condition factor between infected and uninfected eels or a relationship between condition factor and parasite intensity, whereas Möller, Holst, Lüchtenberg & Petersen (1991) reported a higher condition factor in infected eels but no change in the hepatosomatic index. Køie (1991) could find no evidence of lack of appetite in infected eels or difference in condition factor, but confirmed a greater mortality of infected eels during storage or transport due to stress and possibly to secondary bacterial infections.

Population effects

The experimental evidence that the functioning of the swimbladder is impaired in infected eels and that they are more susceptible to stress and to human activities such as netting (Gollock *et al.* 2004) and more likely to suffer mortality during transport (Køie 1991) suggests that infected eels may respond differently to conditions in natural populations. It would seem very likely, for example, that infected eels would be more susceptible to natural avian predators. There is no direct evidence that this is the case, and such evidence would be very difficult to obtain, but even the reduction in swimming performance and speed reported by Sprengel & Lüchtenberg (1991) must surely affect their escape response to predators.

There is also direct evidence that *A. crassus* can cause host mortalities in eel farms. The early reports of *A. crassus* in *A. anguilla* in Japanese eel farms (Egusa & Hirose 1983; Nagasawa *et al.* 1994) showed clearly that the parasite was capable of causing severe mortality of infected eels. Eventually, the mortalities in *A. anguilla* in Japanese eel farms

due to the parasite were largely responsible for the abandonment of cultivation of *A. anguilla* in Japan (Taraschewski, Renner & Mehlhorn 1988). Following the introduction of the parasite to Europe, Mellergaard (1988) reported increased mortality of infected eels in a Danish eel farm in which 65% of the eels were infected and also made reference to a Dutch eel farm, where mortality rates due to the parasite ranged from 15% to 65%. Køie (1991) noted particularly that only eel farms using sea water were parasite free.

Eel mortalities in lakes in central Europe where eels are stocked to form the basis of fisheries also suggest that *A. crassus* plays a role in these mortalities. The best documented of these mortalities took place in Lake Balaton in Hungary (Molnár *et al.* 1991). The mass mortality of eels took place during summer 1991, with an estimated loss of 250 t of eels in the western basin. Losses were lower in 1992, when only 40 t were lost as conditions improved in the western basin of the lake and the infection spread to the eastern basin. No other species of fish was involved. Initially, it was suggested that *A. crassus* alone might have been the cause. Infection levels were very high in the lake at that time and virtually all eels were infected, with 30–50 adults per eel and up to 200 larvae. Eel population densities were also very high. The effects of the parasites on eel swimbladders were typical, with eels showing swimbladder walls haemorrhagic and thickened up to 10 times normal, and the contents of the swimbladder filled with fluid, eggs and decaying and live adults. This, together with the known ability of the parasite to cause mortalities in eel farms, made this not an unreasonable suggestion. Subsequently, following a detailed examination of physico-chemical conditions in the lake, opinion on the role of the parasite changed. It was clear that water temperature levels in the lake were unusually high during that summer whilst oxygen levels were correspondingly very low. These conditions caused stress to the eels, and it now seems more likely that the combined effects of this stress together with that caused by *A. crassus*, and especially the dysfunctioning of the swimbladder and gas gland, were the causes of the mortality (Molnár *et al.* 1991, 1994) as both blood loss and direct effects on the swimbladder wall were eliminated as potential causative agents.

Similar mass mortalities have been reported from other water bodies which have been stocked with eels, for example, in the Vranov Reservoir in the Czech Republic (Baruš, Moravec & Prokeš 1999a). Here there was a loss of some 3–5 t in 1994, and again the mortalities occurred in summer when water temperatures were high and water oxygen levels low and eel densities were high. Again, no other fish species were involved. It is likely therefore that here also it was the combined effects of environmental and parasite induced stresses that caused the mortalities. The ideal conditions for such epizootics were stated by Baruš & Prokeš (1996) as being high eel density in a closed, shallow lake, where densities of copepods and paratenic host species were high, thus facilitating a rapid increase of the parasite population levels. Additionally, however, there needed to be high water temperatures and/or stress factors. These conditions were similar and were met in both Lake Balaton and Vranov Reservoir in particular years.

Conditions in the shallow, productive lakes of central Europe in which eels are stocked for commercial fisheries can clearly from time to time result in eel mortalities, but these are not regular or inevitable occurrences. In other years, apparently similar physico-chemical conditions did not result in eel mortalities in Lake Balaton (Bálint, Ferenczy, Kátai, Kiss, Kráczer, Kufcsák, Láng, Polyhos, Szabó, Szegletes & Nemcsók 1997). Schabuss, Kennedy, Koneeny, Grillitsch, Reckendorfer, Schiemer & Herzig (2005) have noted a similarity in physical and chemical conditions between Neusiedler See in Austria and Lake Balaton and both lakes support a stocked eel fishery. Infection levels with *A. crassus* were also high in Neusiedler See, but there was no occurrence of mass mortalities there over a period of many years.

Convincing evidence of damage to natural, i.e. unstocked, eel populations in lakes and rivers is lacking. There are no reports of natural mortalities of eels in rivers or lakes that are not subjected to eel stocking, but in which prevalence levels of *A. crassus* may be high. Possibly in these natural habitats, the density dependent regulatory processes identified by Ashworth & Kennedy (1999) operate to keep the parasite population below the levels at which mortality might occur and/or eel densities are never as high as in stocked lakes. It is also, of course, possible that parasite induced eel mortalities do occur but have never been detected or identified as such. Again, it would be very difficult indeed to do so.

Since the first appearance of *A. crassus* in Europe, concern has been expressed about possible effects of

the parasite upon the migration of adult eels back to the Sargasso Sea to spawn. As knowledge of the effects of the parasite upon its eel host has increased, so has this concern. Yellow eels undergo stress in their movements from fresh water to the sea and in their transition to silver eels. The silver eels themselves use their swimbladder as a hydrostatic organ in the course of their migration to the Sargasso. Knowledge of the effects of *A. crassus* on the gas gland and oxygen concentration in the swimbladder suggests very strongly that its ability to function as a hydrostatic organ will be impaired in infected eels and this must surely affect their vertical movements on migration. The ability of the parasite to survive for several months in eels in sea water (Kirk *et al.* 2000b, 2002) means also that it could be stressing the eels directly throughout the whole period of their marine migrations. It is, however, very difficult to see how these suggestions could be tested directly by experiment (Taraschewski 2006).

It was thought at one time that the decline in population levels of *A. anguilla* throughout Europe during the 1980s might be directly related to the spread and increase of *A. crassus* over the same period. A decline in eel populations and elver runs has been well documented throughout the continent (Moriarty & Dekker 1997), and several factors including overfishing of elvers and adults and global warming have been considered to be wholly or partially responsible. The correlation between the increase in *A. crassus* infection levels and decrease in host population levels might suggest a causal relationship, but doubt was thrown upon this suggestion when it was realized that a similar decline in magnitude of recruitment (98%) was taking place simultaneously in the *A. rostrata* population in North America at a time before *A. crassus* had spread to that continent. This decline was also blamed on overfishing and pollution. However, it has been suggested that the coincidence in timing of the declines on both sides of the Atlantic implies an Atlantic-wide cause, e.g. changes in climate or Gulf Stream (Castonguay, Hodson, Moriarty, Drinkwater & Jessop 1994). Nevertheless, it is very hard to believe that *A. crassus* is not at least partially responsible for, or does not contribute to, the decline in eel populations and many workers believe that this is in fact the case (Køie 1991; Sures & Knopf 2004b). The scenario envisaged is similar to that in respect of the cause of eel deaths in Lake Balaton, where the parasite acts together with other stressors to cause eel mortality.

Control

In view of the rapid spread of *A. crassus* throughout Europe and its proven abilities as an excellent colonizer, involving even a trans-Atlantic crossing, it is too late to attempt to limit its distribution by limiting imports or restricting transfers of elvers. The parasite's preference for warmer waters will limit its spread northwards in Europe and America so that its range will never be completely congruent with those of *A. rostrata* and *A. anguilla*, but there can be no doubt that in warmer conditions its range will coincide with those of both eel species and it is here to stay. Its ability to infect glass eels and elvers not only assists its spread throughout natural waters but also makes it very difficult to obtain elvers free of the parasite for stocking purposes.

Control measures are clearly impracticable in natural water bodies, and indeed may be unnecessary if *A. crassus* populations are regulated in a density dependent manner at levels below which they cause mortality. In natural lakes such as Lake Balaton which supported eel fisheries through stocking, control was impracticable and stocking ceased in 1991 as a direct consequence of the high eel mortality. If stocking densities could ever have been reduced to a level at which the fishery remained commercial but the eel population did not rise to a density at which the eels were stressed, then mortalities would have been less likely to occur.

Even in eel ponds, control of the parasite levels may be difficult. Attempts to reduce the densities of copepods that can serve as intermediate hosts may not be very successful. Increasing the flow of water through the eel ponds (Egusa & Hirose 1983) may well not be practicable, and is at best only likely to reduce copepod densities and not eliminate them. Using chemicals to try and eliminate copepods is not considered to be very effective (Egusa & Hirose 1983) and is moreover not environmentally acceptable as the effluent from the ponds would end up contaminating natural water bodies. Eels from farms in which water salinity is higher tend to be free of the parasite, or to harbour lower levels (Køie 1991).

Drug treatment can be used in control (Hartmann 1987; Geets, Liewes & Ollevier 1992). A number of nematocidal drugs have been tried, but the most effective are levamisole and metrifonate in freshwater baths (Taraschewski *et al.* 1988). There is no drug specific reaction by the parasite:

muscle cells and intestinal walls are damaged, leading to cytoplasmic disorder and vacuolation and ultimately to death of the parasite. Levamisole appears to be the better of the two drugs as the cumulative lethal dose ratio is more favourable than with metrifonate (Taraschewski *et al.* 1988). Adult parasites in eels treated by bathing in the drug disintegrate, although the larvae in the swimbladder wall are not affected. Ashworth & Blanc (1997) have compared the effectiveness of levamisole, which they also consider to be the best drug, when administered by intravenous injection and oral feeding. Following injection, the drug was distributed rapidly to tissues outside the blood, but total clearance was high and elimination rather fast. Absorption of the drug was slower when administered orally, but retention was longer.

Conclusions

It is evident that the specific helminth parasites of eels have undergone a long period of co-adaptation with their preferred host species. Many of the helminth species reported from eels are generalists and none of these cause any disease problems. The three species that can cause serious disease problems are all specialists of eels and when co-existing with their preferred definitive host at normal densities in natural habitats throughout their normal range they give no cause for major concern. It is only when their normal host is cultured intensively and/or their range has been extended by introductions and they have been given the opportunity to infect a species of eel other than their preferred host and which is not adapted to them that problems arise. *Pseudodactylogyrus anguillae* and *P. bini*, for example, have not been reported as causing other than local gill damage to eels in the wild. They appear only to cause eel mortality when densities of eels are high and they are kept under optimum temperature conditions, i.e. in aquaculture, and the resulting increase in transmission rates allows them to attain infrapopulation densities that can seriously damage or even kill their hosts. Problems of disease here arise principally from the increase in parasite numbers, and both their normal host and other species of eel can be affected. Being ectoparasites, they are more susceptible to control measures.

Anguillicola crassus, however, poses very different problems in kind and degree. This species, and apparently all the other species of *Anguillicola*, are never pathogenic to their preferred eel species in the wild or in culture. It seems likely that *A. crassus* is specific to *A. japonica* with which it has co-evolved. Disease problems only arise when *A. crassus* is exposed to a species of eel to which it is not adapted, i.e. to *A. anguilla* or *A. rostrata*, when imported to East Asia or after introduction of the parasite into Europe and America. The impact of *A. crassus* on Atlantic eels is not just a reflection of a large parasite being enclosed in a small swimbladder, or merely a reflection of numbers of parasites in a single host. Its effects on its eel host are of a different nature to those of the monogeneans: it affects the swimbladder structurally and physiologically in its ability to function as a hydrostatic organ; it affects many blood parameters; it causes considerable stress to its eel host and it can result in both individual and mass eel mortality. Its greatest damage is done when eels are cultured, in farms or shallow lakes, but it can and does affect eels in the wild also.

It is easy to be wise after the event and say that it should not have been introduced into Europe. As early as 1979, Egusa warned that *A. crassus* could cause serious damage to European eels if it was ever introduced to that continent. Despite this warning, it would not have been possible at that time to predict the changes in the life cycle of the parasite that have taken place after its introduction and which have made it such an effective invader and successful colonizer. No-one could have known that it would broaden its specificity to its intermediate host or that it had the ability to use paratenic hosts, any more than it could have been predicted that larvae would spend long periods of time in the walls of the swimbladder rather than pass rapidly through to the lumen. Moreover, it was never realistic to expect that eels would not be transported from one continent to another to meet a shortfall in demand given the economic value of the eel trade.

Anguillicola crassus is also unusual in that it causes damage to wild eels as well as to farmed ones. It may turn out that in the long-term it is the damage to wild eels that will prove to be the most important. Many authors believe that *A. crassus*, through its effects on the swimbladder as a hydrostatic organ, must be having an effect on the ability of adult eels to return to the Sargasso Sea. A decline in elver runs was already apparent before the full impact of *A. crassus* was evident but it is very unlikely that the parasite is not acting as an additional factor in the decline of eel populations. Thus, as well as causing serious economic damage to eel aquaculture in the short-term, the parasite may be having a long-term economic effect

on eel densities. Since declining eel populations are also going to have a knock-on effect on the natural fish communities in rivers and lakes, the parasite may also cause severe and as yet unpredictable biological and ecological problems. Even though controllable in aquaculture by drugs, the parasite cannot be eliminated from wild eel populations. It is here to stay as a permanent component of the European and American parasite fauna. *Anguillicola crassus* thus exemplifies in a unique way the dangers of introducing parasites into new ecosystems: a parasite may be able to realize a potential in its genome that was never previously even suspected and by so doing turn out to have the ability to seriously influence not only its new host populations, but whole ecosystems.

References

Ashworth S.T. & Blanc G. (1997) *Anguillicola crassus*, un colonisateur agressif récemment introduit dans les stocks européans d'anguilles. *Bulletin Francais de Pêche et Pisciculture* 1997, 345–342.

Ashworth S.T. & Kennedy C.R. (1999) Density-dependent effects on *Anguillicola crassus* (Nematoda) within its European eel definitive host. *Parasitology* **118**, 289–296.

Ashworth S.T., Kennedy C.R. & Blanc G. (1996) Density-dependent effects of *Anguillicola crassus* (Nematoda) within and on its copepod intermediate hosts. *Parasitology* **113**, 303–309.

Bálint T., Perenczy J., Kárai F., Kiss I., Kráczer L., Kufcsák O., Láng G., Polyhos C., Szabó I., Szegletes T. & Nemcsók J. (1997) Similarities and differences between the massive eel (*Anguilla anguilla* L.) devastations that occurred in Lake Balaton in 1991 and 1995. *Ecotoxicology and Environmental Safety* **37**, 17–23.

Barker D.E. & Cone D.K. (2000) Occurrence of *Ergasilus celestis* (Copepoda) and *Pseudodactylogyrus anguillae* (Monogenea) on American eel, *Anguilla rostrata*, in relation to stream flow, pH and temperature and recommendations for controlling their transmission among captive eels. *Aquaculture* **186**, 261–274.

Barse A.M. & Secor D.H. (1999) An exotic nematode parasite of the American eel. *Non-Native Species Management* **24**, 6–10.

Barus V. & Prokeš M. (1996) Length-weight relations of uninfected and infected eels (*Anguilla anguilla*) by *Anguillicola crassus* (Nematoda). *Folia Zoologica* **45**, 183–189.

Barus V., Kráčmar S., Tenora F. & Prokeš M. (1998) Amino acid spectrum of uninfected and infected eels (*Anguilla anguilla*) by *Anguillicola crassus* (Nematoda). *Folia Zoologica* **47**, 231–234.

Barus V., Moravec F. & Prokeš M. (1999a) Anguillicolosis of the European eel (*Anguilla anguilla*) in the Czech Republic. *Czechoslovak Journal of Animal Science* **44**, 423–431.

Barus V., Tenora F., Kráčmar S. & Dvořáček J. (1999b) Contents of several inorganic substances in European eel infected and uninfected by *Anguillicola crassus* (Nematoda). *Diseases of Aquatic Organisms* **37**, 135–137.

Békési L., Hormok S. & Székely Cs. (1997) Attempts to analyse *Anguillicola crassus* infection and the humoral host response in eels (*Anguilla anguilla*) of Lake Balaton. *Acta Veterinaria Hungarica* **45**, 439–445.

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 effects 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 effects of different infection levels with infective larvae of *Anguillicola crassus* on haematological parameters of European eel (*Anguilla anguilla*). *Aquaculture* **87**, 243–253.

Borgsteede F.H.M., Haenen O.L.M., De Bree J. & Lisitsina O.I. (1999) Parasitic infections of the European eel (*Anguilla anguilla* L.) in the Netherlands. *Helminthologia* **36**, 251–260.

Buchmann K. (1988) Spatial distribution of *Pseudodactylogyrus anguillae* and *P. bini* (Monogenea) on the gills of the European eel, *Anguilla anguilla*. *Journal of Fish Biology* **32**, 801–802.

Buchmann K. (1989) Relationship between host size of *Anguilla anguilla* and the infection level of the monogeneans *Pseudodactylogyrus* spp. *Journal of Fish Biology* **35**, 599–601.

Buchmann K. (1993) Epidémiologie et contrôle des infections à *Pseudodactylogyrus* dans les elevages intensifs d'anguilles: données actuelles. *Bulletin Francais Peche Pisciculture* **328**, 66–73.

Buchmann K. & Bjerregaard J. (1990) Mebendazole treatment of pseudodactylogyrosis in an intensive eel culture system. *Aquaculture* **86**, 139–153.

Buchmann K., Mellergaard S. & Køie M. (1987a) *Pseudodactylogyrus* infections in eels: a review. *Diseases of Aquatic Organisms* **3**, 51–57.

Buchmann K., Køie M. & Prento P. (1987b) The nutrition of the gill parasitic monogenean *Pseudodactylogyrus anguillae*. *Parasitology Research* **73**, 532–537.

Buchmann K., Székély Cs. & Bjerregaard J. (1990) Treatment of *Pseudodactylogyrus* infestations of *Anguilla anguilla*. II Trials with bunamidine, praziquantal and levamisole. *Bulletin of the European Association of Fish Pathologists* **10**, 18–20.

Castonguay M., Hodson P.V., Moriarty C., Drinkwater K.F. & Jessop B.M. (1994) Is there a role of ocean environment in American and European eel decline? *Fisheries Oceanography* **3**, 197–203.

Cone D. & Marcogliese D.J. (1995) *Pseudodactylogyrus anguillae* on *Anguilla rostrata* in Nova Scotia: an endemic or an introduction? *Journal of Fish Biology* **47**, 177–178.

De Charleroy D., Grisez L., Thomas K., Belpaire C. & Ollevier F. (1990) The life cycle of *Anguillicola crassus*. *Diseases of Aquatic Organisms* **8**, 77–84.

Di Cave D., Berrilli F., De Liberato C., Orecchia P. & Kennedy C.R. (2001) Helminth communities in eels *Anguilla anguilla* from Adriatic coastal lagoons in Italy. *Journal of Helminthology* **75**, 7–13.

Egusa S. (1979) Notes on the culture of the European eel (*Anguilla anguilla* L.) in Japanese eel farming ponds. *Rapports et*

Proces-Verbaux des Réunions. Conseil International pour l'Exploration de la Mer 174, 51–58.

Egusa S. & Hirose H. (1983) Anguillicolosis of eels. In: *Fish Pathology (Infectious Diseases and Parasitic Diseases)* (ed. by S. Egusa), pp. 308–310. Koseisha Koseikaku, Tokyo.

Fries L.T., Williams D.J. & Johnson S.K. (1996) Occurrence of *Anguillicola crassus*, an exotic parasitic swimbladder nematode of eels, in south-western United States. *Transactions of the American Fisheries Society* 125, 794–797.

Geets A., Liewes E.W. & Ollevier F. (1992) Efficacy of some anthelmintics against the swimbladder nematode *Anguillicola crassus* of eel *Anguilla anguilla* under saltwater conditions. *Diseases of Aquatic Organisms* 13, 123–128.

Gollock M.J., Kennedy C.R., Quabius E.S. & Brown J.A., (2004) The effect of parasitism of European eels with the nematode, *Anguillicola crassus* on the impact of netting and aerial exposure. *Aquaculture* 233, 45–54.

Gollock M.J., Kennedy C.R. & Brown J.A. (2005a) Physiological responses to acute temperature increase in European eels *Anguilla anguilla* infected with *Anguillicola crassus*. *Diseases of Aquatic Organisms* 64, 223–228.

Gollock M.J., Kennedy C.R. & Brown J.A. (2005b) European eels, *Anguilla anguilla* (L.), infected with *Anguillicola crassus* exhibit a more pronounced stress response to severe hypoxia than uninfected eels. *Journal of Fish Diseases* 28, 429–436.

Haenen O.L.M., Grisez L., De Charleroy D., Belpaire C. & Ollevier F. (1989) Experimentally induced infections of the European eel *Anguilla anguilla* with *Anguillicola crassus* (Nematoda, Dracunculoidea) and subsequent migration of larvae. *Diseases of Aquatic Organisms* 7, 97–101.

Haenen O.L.M., van Banning P. & Dekker W. (1994) Infection of eel *Anguilla anguilla* (L.) and smelt *Osmerus eperlanus* (L.) with *Anguillicola crassus* (Nematoda, Dracunculoidea) in the Netherlands from 1986 to 1992. *Aquaculture* 126, 219–229.

Haenen O.L.M., van Wijngaarden T.A.M., van der Heijden M.H.T., Höglund J., Cornelissen J.B.J.W., van Leengoed L.A.M.G., Borgsteede F.H.M., van Haenen O.L.M. & Muiswinkel W.B. (1996) Effects of experimental infections with different doses of *Anguillicola crassus* (Nematoda, Dracunculoidea) on European eel (*Anguilla anguilla*). *Aquaculture* 141, 41–57.

Hartmann S. (1987) Schwimmblasenwürmer beim aal. *Fischer und Teichwirt* 1, 2–3.

Hayward C.J., Iwashita M., Crane J.S. & Ogawa K. (2001) First report of the invasive eel pest *Pseudodactylogyrus bini* in North America and in wild American eels. *Diseases of Aquatic Organisms* 44, 53–60.

Hewitt G.C. & Hine P.M. (1972) Checklist of parasites of New Zealand fishes and of their hosts. *New Zealand Journal of Marine and Freshwater Research* 6, 69–114.

Hine P.M. & Boustead N.C. (1974) A guide to disease in eel farms. *Fisheries Research Division Occasional Publication* 6, 1–26.

Hoffman G.L. (1999) *Parasites of North American Freshwater Fishes*, 2nd edn. Cornell University Press, Ithaca, NY.

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. *Diseases of Aquatic Organisms* 13, 175–180.

Höglund J., Andersson J., Wickström H. & Reizenstein M. (1992) The distribution of *Anguillicola* in Sweden and its association with thermal discharge areas. *Irish Fisheries Investigations Series A (Freshwater)* 36, 143–150.

Hsu-Yun C., I-Hsiung L. & Guang-Hsiung K. (1984) Study of the parasites on the gill of cultured eel in Taiwan. *COA Fisheries Series No. 10. Fish Disease Research* 6, 24–33.

Kole M. (1991) Swimbladder nematodes (*Anguillicola* spp.) and gill monogeneans (*Pseudodactylogyrus* spp.) parasitic on the European eel (*Anguilla anguilla*). *Journal de la Conseil International pour Exploration de la Mer* 47, 391–398.

Kelly C.E., Kennedy C.R. & Brown J.A. (2000) Physiological status of wild European eels (*Anguilla anguilla*) infected with the parasitic nematode, *Anguillicola crassus*. *Parasitology* 120, 195–202.

Kennedy C.R. (1994a) The distribution and abundance of the nematode *Anguillicola australiensis* in eels *Anguilla reinhardtii* in Queensland, Australia. *Folia Parasitologica* 41, 279–285.

Kennedy C.R. (1994b) The ecology of introductions. In: *Parasitic Diseases of Fish* (ed. by A.W. Pike & J.W. Lewis), pp. 189–208. Samara Publishing Ltd, Tresaith.

Kennedy C.R. (1995) Richness and diversity of macroparasite communities in tropical eels *Anguilla reinhardtii* in Queensland, Australia. *Parasitology* 111, 233–245.

Kennedy C.R. & Di Cave D. (1998) *Gyrodactylus anguillae* (Monogenea): the story of an appearance and a disappearance. *Folia Parasitologica* 45, 77–78.

Kennedy C.R. & Fitch D.J. (1990) Colonisation, larval survival and epidemiology of the nematode *Anguillicola crassus*, parasitic in the eel *Anguilla anguilla*, in Britain. *Journal of Fish Biology* 36, 117–131.

Kirk R.S., Kennedy C.R. & Lewis J.W. (2000a) Effect of salinity on hatching, survival and infectivity of *Anguillicola crassus* (Nemaroda: Dracunculoidea) larvae. *Diseases of Aquatic Organisms* 40, 211–218.

Kirk R.S., Lewis J.W. & Kennedy C.R. (2000b) Survival and transmission of *Anguillicola crassus* Kuwahara, Niimi & Itagaki, 1974 (Nematoda) in seawater eels. *Parasitology* 120, 289–295.

Kirk R.S., Morritt D., Lewis J.W. & Kennedy C.R. (2002) The osmotic relationship of the swimbladder nematode *Anguillicola crassus* with seawater eels. *Parasitology* 124, 339–347.

Knopf K. & Mahnke M. (2004) Differences in susceptibility of the European eel (*Anguilla anguilla*) and the Japanese eel (*Anguilla japonica*) to the swimbladder nematode *Anguillicola crassus*. *Parasitology* 129, 491–496.

Knopf K., Würtz J., Sures B. & Taraschewski H. (1998) Impact of low water temperature on the development of *Anguillicola crassus* in the final host *Anguilla anguilla*. *Diseases of Aquatic Organisms* 33, 142–149.

Knopf K., Naser K., Van der Heijden M.H.T. & Taraschewski H. (2000a) Humoral immune response of European eel *Anguilla anguilla* experimentally infected with *Anguillicola crassus*. *Diseases of Aquatic Organisms* 42, 61–69.

Knopf K., Naser K., Van der Heijden M.H.T. & Taraschewski H. (2000b) Evaluation of an ELISA and immunoblotting for

studying the humoral immune response in *Anguillicola crassus* infected European eel (*Anguilla anguilla*). *Diseases of Aquatic Organisms* 43, 39–48.

Koops H. & Hartmann F. (1989) Infection of eels from different regions with *Anguillicola*. *Journal of Applied Ichthyology* 1, 41–45.

Lefebvre F., Mounaix B., Poizat G. & Crivelli A.J. (2004) Impacts of the swimbladder nematode *Anguillicola crassus* on *Anguilla anguilla* variations in liver and spleen masses. *Journal of Fish Biology* 64, 435–447.

Malmberg G. (1970) The excretory systems and the marginal hooks as a basis for the systematics of *Gyrodactylus* (Trematoda: Monogenea). *Arkiv for Zoologi* 23, 1–235.

Marcogliese D.J. & Cone D.K. (1993) What metazoan parasites tell us about the evolution of American and European eels. *Evolution* 47, 1632–1635.

Marcogliese D.J. & Cone D.K. (1998) Comparison of richness and diversity of macroparasite communities among eels from Nova Scotia, the United Kingdom and Australia. *Parasitology* 116, 73–83.

Mellergaard S. (1988) Alens svommeblaereorm *Anguillicola* – en ny parasit i den europaeiske alebestand. *Norges Aquakultur* 4, 50–54.

Mo T.A. & Sterud E. (1998) First observations of *Pseudodactylogyrus* spp. and other parasites in wild eel (*Anguilla anguilla* L.) in Norway. *Bulletin of the Scandinavian Society for Parasitology* 8, 1–5.

Mo T.A., Håstein T. & Lunder T. (1988) *Pseudodactylogyrus* en problemparasitt i åleoppdrett. *Norsk Fiskeoppdrett* 19, 30–31.

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*. *Diseases of Aquatic Organisms* 11, 193–199.

Molnár K. (1993) Effects of decreased oxygen content on eels (*Anguilla anguilla*) infected by *Anguillicola crassus* (Nematoda: Dracunculoidea). *Acta Veterinaria Hungarica* 41, 349–360.

Molnár K. (1994) Formation of parasitic nodules in the swimbladder and intestinal walls of the eel *Anguilla anguilla* due to infections with larval stages of *Anguillicola crassus*. *Diseases of Aquatic Organisms* 20, 163–170.

Molnár K., Székely Cs. & Baska F. (1991) Mass mortality of eel in Lake Balaton due to *Anguillicola crassus* infection. *Bulletin of the European Association of Fish Pathologists* 11, 211–212.

Molnár K., Baska F., Csaba G., Glávitis R., Székely C. & Perényi M. (1993) Pathological and histopathological studies of the swimbladder of eels *Anguilla anguilla* infected by *Anguillicola crassus* (Nematoda: Dracunculoidea). *Diseases of Aquatic Organisms* 15, 41–50.

Molnár K., Székely Cs. & Perényi M. (1994) Dynamics of *Anguillicola crassus* (Nematoda: Dracunculoidea) infection in eels of Lake Balaton, Hungary. *Folia Parasitologica* 41, 193–202.

Moravec F. (1996) Aquatic invertebrates (snails) as new paratenic hosts of *Anguillicola crassus* (Nematoda: Dracunculoidea) and the role of paratenic hosts in the life cycle of this parasite. *Diseases of Aquatic Organisms* 27, 237–239.

Moravec F. & Konecny R. (1994) Some new data on the intermediate and paratenic hosts of the nematode *Anguillicola crassus* Kuwahara, Niimi et Itagaki, 1974 (Dracunculoidea), a swimbladder parasite of eels. *Folia Parasitologica* 41, 65–70.

Moravec F. & Rohde K. (1992) Three species of nematodes of the superfamily Dracunculoidea from Australian fishes. *Acta Societa Zoologica Bohemoslovakia* 56, 187–195.

Moravec F. & Škoríková B. (1998) Amphibians and larvae of aquatic insects as new paratenic hosts of *Anguillicola crassus* (Nematoda: Dracunculoidea), a swimbladder parasite of eels. *Diseases of Aquatic Organisms* 34, 217–222.

Moravec F. & Taraschewski H. (1988) Revision of the genus *Anguillicola* Yamaguti, 1935 (Nematoda: Anguillicolidae) of the swimbladder of eels, including descriptions of two new species, *A. novaezelandiae* sp.n. and *A. papernai* sp.n. *Folia Parasitologica* 35, 125–146.

Moravec F., Di Cave D., Orecchia P. & Paggi L. (1993) Studies on the development of *Anguillicola crassus* Kuwahara, Niimi et Itagaki, 1974 (Nematoda: Dracunculoidea) in the intermediate host. *Folia Parasitologica* 40, 39–48.

Moravec F., Di Cave D., Orecchia P. & Paggi L. (1994a) Present occurrence of *Anguillicola novaezelandiae* (Nematoda: Dracunculoidea) in Europe and its development in the intermediate host. *Folia Parasitologica* 41, 203–208.

Moravec F., Di Cave D., Orecchia P. & Paggi L. (1994b) Experimental observations on the development of *Anguillicola crassus* (Nematoda: Dracunculoidea) in its definitive host, *Anguilla anguilla*, Pisces. *Folia Parasitologica* 41, 138–148.

Moriarty C. & Dekker W. (1997) Management of the European eel. *Fisheries Bulletin (Dublin)* 15, 1–110.

Moser M.L., Patrick W.S. & Crutchfield J.U. (2001) Infection of American eels, *Anguilla rostrata*, by the introduced nematode parasite, *Anguillicola crassus*, in North Carolina. *Copeia* 3, 848–853.

Münderle M., Sures B. & Taraschewski H. (2004) Influence of *Anguillicola crassus* (Nematoda) and *Ichthyophthirius multifiliis* (Ciliophora) on swimming activity of European eel *Anguilla anguilla*. *Diseases of Aquatic Organisms* 60, 133–139.

Münderle M., Taraschewski H., Klar B., Chang C.W., Shiao J.C., Shen K.N., He J.T., Lin S.H. & Tzeng W.N. (2006) Occurrence of *Anguillicola crassus* (Nematoda: Dracunculoidea) in Japnese eels *Anguilla japonica* from a river and an aquaculture unit in SW Taiwan. *Diseases of Aquatic Organisms* 71, 101–108.

Nagasawa K., Kim Y.-G. & Hirose H. (1994) *Anguillicola crassus* and *A. globiceps* (Nematoda: Dracunculoidea) parasitic in the swimbladder of eels (*Anguillicola japonica* and *A. anguilla*) in East Asia: a review. *Folia Parasitologica* 41, 127–137.

Nie P. & Kennedy C.R. (1991) Occurrence and seasonal dynamics of *Pseudodactylogyrus anguillae* (Yin & Sproston) (Monogenea) in eel *Anguilla anguilla* (L.) in England. *Journal of Fish Biology* 39, 897–900.

Nielsen M.E. (1999) An enhanced humoral immune response against the swimbladder nematode, *Anguillicola crassus*, in the Japanese eel, *Anguilla japonica*, compared with the European eel, *A. anguilla*. *Journal of Helminthology* 73, 227–232.

Nimeth K., Zwerger P., Würtz J., Salvenmoser W. & Pelster B. (2004) Infection of the glass-eel swimbladder with the nematode *Anguillicola crassus*. *Parasitology* 121, 75–83.

Oliveira K. & Campbell R.A. (1998) The occurrence and pathological effects of *Stephanostomum tenuae* (Digenea: Acanthocolpidae) metacercariae in elvers of the American eel. *Journal of Fish Biology* **53**, 690–692.

Paggi L., Orecchia P., Minervini R. & Marcheci S. (1982) Sulla comparsa de *Anguillicola australiensis* Johnston and Mawson, 1940 (Dracunculoidea: Anguillicolidae) in *Anguilla anguilla* del Lago di Bracciano. *Parasitologia* **24**, 139–144.

Paliková M. & Navrátil S. (2001) Occurrence of *Anguillicola crassus* in the water reservoir Koryčany (Czech Republic) and its influence on the health condition and haematological index of eels. *Acta Veterinaria Brno* **70**, 443–449.

Pazooki J. & Székely Cs. (1994) Survey of the paratenic hosts of *Anguillicola crassus* in Lake Velence, Hungary. *Acta Veterinaria Hungarica* **42**, 87–97.

Peters G. & Hartmann F. (1986) *Anguillicola*, a parasitic nematode of the swimbladder spreading among eel populations in Europe. *Diseases of Aquatic Organisms* **1**, 229–230.

Puquin W. & Yuru Z. (1980) Observations of the life history of *Anguillicola globiceps* (Nematoda: Anguillicolidae). *Acta Zoologica Sinica* **26**, 243–249.

Reimer L.W., Hildebrand A., Scharbeth D. & Walter U. (1994) *Anguillicola crassus* in the Baltic Sea: field data supporting transmission in brackish waters. *Diseases of Aquatic Organisms* **18**, 77–79.

Rid L.E. (1973) Helminth parasites of the long-finned eel, *Anguilla dieffenbachii*, and the short finned eel, *A. australis*. *Mauri Ora* **1**, 99–106.

Rodrigues A.A. & Saraiva A. (1996) Spatial distribution and seasonality of *Pseudodactylogyrus anguillae* and *P. bini* (Monogenea: Pseudodactylogyridae) on the gills of the European eel *Anguilla anguilla* from Portugal. *Bulletin of the European Association of Fish Pathologists* **16**, 85–88.

Schabuss M., Kennedy C.R., Koneeny R., Grillitsch B., Reckendorfer W., Schiemer F. & Herzig A. (2005) Dynamics and predicted decline of *Anguillicola crassus* infection in European eels, *Anguilla anguilla*, in Neusiedler See, Austria. *Journal of Helminthology* **79**, 159–167.

Scholz F. & Zerbst-Boroffka I. (1994) Osmo-und ionenegulation des aalparasiten *Anguillicola crassus* (Nematoda) und seines wirtes (*Anguilla anguilla*). *Zoologische Beitreagen* **35**, 103–117.

Sprengel G. & Lüchtenberg H. (1991) Infection by endoparasites reduces maximum swimming speed of European smelt *Osmerus eperlanus* and European eel *Anguilla anguilla*. *Diseases of Aquatic Organisms* **11**, 31–35.

Sures B. & Knopf K. (2004a) Individual and combined effects of cadmium and 3, 3′, 4, 4′, 5-pentachlorobiphenyl (PCB 126) on the humoral immune response in European eel (*Anguilla anguilla*) experimentally infected with larvae of *Anguillicola crassus* (Nematoda). *Parasitology* **128**, 1–10.

Sures B. & Knopf K. (2004b) Parasites as a threat to freshwater eels? *Science* **304**, 205–206.

Sures B., Knopf K. & Kloas W. (2001) Induction of stress by the swimbladder nematode *Anguillicola crassus* in European eels, *Anguilla anguilla*, after repeated experimental infection. *Parasitology* **123**, 179–184.

Székely Cs. (1994) Paratenic hosts for the parasitic nematode *Anguillicola crassus* in Lake Balaton, Hungary. *Diseases of Aquatic Organisms* **18**, 11–20.

Székely Cs. (1996) Experimental studies on the infectivity of *Anguillicola crassus* third-stage larvae (Nematoda) from paratenic hosts. *Folia Parasitologica* **43**, 305–311.

Székely Cs. & Molnár K. (1987) Mebendazole is an efficacious drug against pseudodactylogyrosis in the European eel (*Anguilla anguilla*). *Journal of Applied Ichthyology* **4**, 183–186.

Székely Cs., Pazooki J. & Molnár K. (1996) Host reaction in paratenic fish hosts against 3rd stage larvae of *Anguillicola crassus*. *Diseases of Aquatic Organisms* **26**, 173–180.

Taraschewski H. (2006) Hosts and parasites as aliens. *Journal of Helminthology* **80**, 99–128.

Taraschewski H., Moravec F., Lamah T. & Anders K. (1987) Distribution and morphology of two helminths recently introduced into European eel populations: *Anguillicola crassus* (Nematoda, Dracunculoidae) and *Paratenuisentis ambiguus* (Acanthocephala: Tenuisentidae). *Diseases of Aquatic Organisms* **3**, 167–176.

Taraschewski H., Renner C. & Mehlhorn H. (1988) Treatment of fish parasites 3. Effects of levamisole HCl, metrifonate, fenbenzadole, mebenzadole, and invermectin on *Anguillicola crassus* (nematodes) pathogenic in the air bladder of eels. *Parasitology Research* **74**, 281–289.

Taraschewski H., Boomker J., Knopf K. & Moravec F. (2005) Studies on the morphology and ecology of *Anguillicola papernai* (Nematoda: Anguillicolidae) parasitising the swimbladder of African longfin eel *Anguilla mozambica*, and on the helminth community in this eel. *Diseases of Aquatic Organisms* **62**, 185–195.

Thomas K. & Ollevier F. (1992) Paratenic hosts of the swimbladder nematode *Anguillicola crassus*. *Diseases of Aquatic Organisms* **13**, 165–174.

Thomas K. & Ollevier F. (1993) Hatching, survival, activity and penetration efficiency of second-stage larvae of *Anguillicola crassus* (Nematoda). *Parasitology* **107**, 211–217.

Wang P. & Zhao Y. (1980) Observations on the life history of *Anguillicola globiceps* (Nematoda: Anguillicolaidae). *Acta Zoologica Sinica* **26**, 243–249.

Würtz J. & Taraschewski H. (2000) Histopathological changes in the swimbladder wall of the European eel *Anguilla anguilla* due to infection with *Anguillicola crassus*. *Diseases of Aquatic Organisms* **39**, 121–134.

Würtz J., Taraschewski H. & Pelster B. (1996) Changes in the gas composition of the swimbladder of the European eel (*Anguilla anguilla*) infected with *Anguillicola crassus* (Nematoda). *Parasitology* **112**, 233–238.

Zhang B. (1995) Studies on parasitic diseases in European eel (*Anguilla anguilla*) farms in Guangdong, China. In: *Proceedings of the 10th Anniversary of the Founding of China Parasitological Society, Society of Parasitology of China*, pp. 108–112.

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