An Exotic Nematode Parasite of the American Eel

By Ann M. Barse and David H. Secor

ABSTRACT
We investigated reports from commercial fishers of parasitized American eels *(Anguilla rostrata)* in the Patuxent River (located in the mid-Chesapeake Bay) and discovered that some eels were infected with an exotic swim bladder nematode, *Anguillicola crassus*. Here, we (1) describe the dispersal of this blood-feeding nematode, endemic to the Japanese eel *(A. japonica)*, in Europe and North America; (2) discuss what is known of the effects of this parasite on eels; (3) summarize current knowledge of the life cycle and identification of this parasite; and (4) present new data on the range of *A. crassus* in the eastern United States. We examined the swim bladders of 329 American eels from 4 sites in the mid- and upper Chesapeake Bay as well as 150 eels from 4 sites in the Hudson River in New York. Prevalence ranged from 10% to 29% in the Chesapeake Bay and from 0% to 12% in the Hudson River. Maximum intensity of infection was 24 for Chesapeake Bay eels and 3 for Hudson River eels. We urge our U.S and Canadian colleagues to examine American eels for *A. crassus* to assess the dispersal of this parasite in eastern North America.

**An important consequence of deliberate or accidental introductions of exotic species is dispersal of the parasites carried with them.** Typically, indigenous species are more susceptible to disease caused by introduced parasite species than are the host species that transport pathogens into a new habitat, presumably because native species lack adaptations for resistance that are acquired only after long periods of parasite-host coevolution. In the Hudson River and Chesapeake Bay, we have discovered one such exotic parasite (*Anguillicola crassus*) a nematode that infects swim bladders of *anguillid* species of eels.

Prior to 1980, *A. crassus* occurred only in east Asian countries, infecting the native Japanese eel (*Anguilla japonica*) and the introduced European eel (*A. anguilla*) (Moravec 1992). In the early 1980s, *A. crassus* infections were noted in European eel populations. Scientists presumed the introduction was due to shipment of Japanese eels from Asia to aquaculture facilities in Germany, where the nematode first was observed in 1982 (Koie 1991). Since then, *A. crassus* also has been recorded in the Netherlands, Belgium, Spain, Portugal, France, England, Denmark, Italy, Greece, the former Yugoslavia, Austria, the Czech Republic, Hungary, Poland, Sweden, Estonia, Russia, and Egypt (Höglund and Thomas 1992; Nagasawa et al. 1994).

In February 1995, Fries et al. (1996) reported that the Texas Parks and Wildlife Department found *A. crassus* in the swim bladders of 8 of 23 eels from a Texas aquaculture facility, although the identity of the host species was not confirmed. Subsequently, these authors examined swim bladders of 30 wild American eels (*Anguilla rostrata*) collected from Winyah Bay, South Carolina, and several Texas rivers for the presence of *A. crassus*. Only a single Winyay Bay eel was infected, representing the first confirmed record of *A. crassus* for this *anguillid* host.

In spring 1997, local watermen alerted us that American eels in the Patuxent River (a tributary in the mid-Chesapeake Bay) contained numerous “worms,” which we identified as *A. crassus*. Subsequent sampling revealed that American eels infected with *A. crassus* were present in several Chesapeake Bay and Hudson River localities (Table 1). Because juvenile stages of *A. crassus* live in the swim bladder wall, and we enumerated only those worms living in the swim bladder lumen, these data underestimate the true prevalence (% of eels infected) and intensity (number of nematodes per eel) of infection. We suspect that this nematode parasite has dispersed widely among North American Atlantic coast rivers and estuaries.

**A. crassus** Life Cycle

The life cycle of *A. crassus* (Figure 1) includes the eel definitive host, a crustacean intermediate host (required for development), and small fish as paratenic (= transport) hosts (De Charleroy et al. 1990; Moravec et al. 1994). Only eels of the genus *Anguilla* can serve as final hosts (where the parasite reaches sexual maturity) of all nematodes in the genus *Anguillicola* (Nagasawa et al. 1994).
al. 1994). The final hosts for *A. crassus* are the Japanese, European, and American eels.

Copulation between adult male and female *A. crassus* occurs in the swim bladder of eels. *Anguillicola crassus* is ovoviviparous, i.e., fertilized eggs develop in the uteri of females and, at the time of oviposition, contain motile, second-stage juveniles. Eggs are laid in the host swim bladder, which frequently contains tens of thousands of eggs (De Charleroy et al. 1990). (Fecundity of individual worms was not reported.) Moravec et al. (1994) estimated the prepatent period to be approximately three months, and the patent period is not longer than one month. Females degenerate soon after egg deposition (Moravec et al. 1994).

Eggs with second-stage juveniles leave the swim bladder via the pneumatic duct, enter the digestive tract, and are released into the water with host feces. Hatching occurs either in the eel digestive tract or shortly after being released into the water (Moravec et al. 1993). Under experimental conditions free-living, second-stage juveniles aggregate in clumps of 10–30 individuals, attach to the substratum by their caudal extremity, and move actively, which presumably increases their probability of predation by intermediate hosts (Kennedy and Fitch 1990; Moravec et al. 1993). Eels exposed directly to second-stage juveniles do not become infected, illustrating the necessity of an intermediate host (Kennedy and Fitch 1990). Moravec and Konecny (1994) noted that at least 11 species of copepods (primarily cyclopoids) and 2 species of ostracods can successfully carry *A. crassus* from second-stage juveniles to infective third-stage juveniles. Kennedy and Fitch (1990) also (accidently) infected a single specimen of a brackish water copepod, *Eurytemora affinis*, an important forage item for North American estuarine fishes.

When eaten by a suitable crustacean intermediate host, the second-stage juveniles penetrate through the wall of the crustacean host's digestive tract to the hemocoel; there, they grow and then molt to third-stage juveniles after 10–12 days (De Charleroy et al. 1990). Third-stage juveniles are infective to eel hosts. However, if infected crustaceans are consumed by small fish, third-stage juveniles can survive either as third-stage juveniles or develop into fourth-stage juveniles. Experimental infections have shown that third-stage juveniles can survive in paratenic hosts for at least two months (Moravec and Konecny 1994). Thus, eels become infected by eating infected intermediate hosts or paratenic fish hosts (De Charleroy et al. 1990).

 Apparently, no studies have been done to identify paratenic hosts of *A. crassus* in East Asia (Nagasawa et al. 1994); however, in Europe Moravec and Konecny (1994) list 23 species of fishes in 9 families that are known to have contained *A. crassus* third-stage juveniles. In Lake Balaton, Hungary, 19 of 20 fish species were found infected with *A. crassus* third-stage juveniles, and 16 of those had a prevalence of at least 50% (Székely 1994). (The fish species that was not infected with *A. crassus* was represented by only one individual in the sample.) Recent experiments have indicated that besides fish paratenic hosts, certain aquatic snails; juveniles of Trichoptera, Odonata, and Megaloptera; and amphibians also can serve as paratenic hosts for *A. crassus* (F. Moravec, Institute of Parasitology, Academy of Sciences of the Czech Republic, pers. comm.).

The rate of development of *A. crassus* in European eels is variable. In experimental infections Haenen et al. (1991) found that third-stage juveniles reach the swim bladder wall within 17 h post-infection (p.i.) by penetrating the intestinal wall and migrating through the body cavity. Fourth-stage juveniles were observed three months p.i., still within the swim bladder wall. At this stage juveniles begin feeding on eel erythrocytes. Preadults with developing gonads were found in the swim bladder lumen four months after infection. Moravec et al. (1994) found fourth-stage juveniles in the swim bladder wall only 23 days p.i. and preadults 31 days p.i. However, *A. crassus* females with eggs containing juveniles were not observed until 98 days p.i. Haenen and van Banning (1991) found adult *A. crassus* and

<table>
<thead>
<tr>
<th>Locality</th>
<th>N</th>
<th>P</th>
<th>Intensity</th>
<th>Mean</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chesapeake Bay</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sassafras River</td>
<td>100</td>
<td>24%</td>
<td>3.1</td>
<td>1–13</td>
<td></td>
</tr>
<tr>
<td>Wye River</td>
<td>160</td>
<td>29%</td>
<td>5.8</td>
<td>1–24</td>
<td></td>
</tr>
<tr>
<td>Crab Alley</td>
<td>21</td>
<td>24%</td>
<td>7.6</td>
<td>2–18</td>
<td></td>
</tr>
<tr>
<td>Patuxent River</td>
<td>48</td>
<td>10%</td>
<td>6.2</td>
<td>1–15</td>
<td></td>
</tr>
<tr>
<td>Hudson River</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Athens</td>
<td>50</td>
<td>0</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>Kingston</td>
<td>25</td>
<td>4%</td>
<td>1.0</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Newburgh</td>
<td>50</td>
<td>12%</td>
<td>1.7</td>
<td>1–3</td>
<td></td>
</tr>
<tr>
<td>Haverstraw</td>
<td>25</td>
<td>0</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
</tr>
</tbody>
</table>

Table 1 summarizes the prevalence (% of eels infected) and intensity (number of worms per eel) of *Anguillicola crassus* in American eels (*Anguilla rostrata*) collected from four Chesapeake Bay and four Hudson River localities. (N = number of eels examined.)

![Figure 1](image_url) Figure 1 shows the life cycle of *Anguillicola crassus*.
second-stage juveniles in the swim bladders of eels only eight weeks post-infection. These data suggest that under laboratory conditions, the entire life cycle of *A. crassus* in American eels can last two to six months.

Temperature and season affect *A. crassus* life stage and life cycle duration (De Charleroy et al. 1989; Kim et al. 1989; Höglund and Thomas 1992). Hatching of second-stage juveniles, and thus recruitment of *A. crassus* to the intermediate host, is diminished in cool water. However, many field investigations have revealed little evidence of seasonality in *A. crassus* infections of European eels (e.g., Kennedy and Fitch 1990; Möller et al. 1991; Thomas and Ollevier 1992). Survival of third-stage juveniles in fish paratenic hosts for periods of at least two months (Moravec and Konecny 1994) may sustain recruitment of *A. crassus* to the eel host during colder months.

*A. crassus* infections can occur across a wide range of eel sizes in both freshwater and brackish water systems. De Charleroy et al. (1990) infected glass eels (mean length 6.4 cm) and elvers (mean length 9.2 cm) experimentally, and Thomas and Ollevier (1992) found high prevalence (>86%) and mean intensities (i.e., average number of worms per infected eel) (>10 worms per fish) among all size classes of wild eels between 9.5 cm and 96.5 cm. Taraschewski et al. (1987) suggested that transmission of *A. crassus* appears to be limited to fresh or low-salinity environments because prevalence decreased in the Elbe estuary (Germany) toward the open sea. However, several studies have found high prevalence and intensity of *A. crassus* in European eel populations in freshwater, brackish, and marine localities (e.g., Haenen et al. 1994; Reimer et al. 1994; Höglund and Thomas 1992, and Reimer et al. 1994) have identified several species of fish paratenic hosts of marine origin. Kennedy and Fitch (1990) demonstrated that there is no loss of viability among fourth-stage juveniles or adults of *A. crassus* in eels maintained in 100% sea water for up to 4 weeks. Juvenile survival decreased in salt water; however, even in 100% sea water, 50% of the juveniles survived for 10 days, long enough to be prey for copepods.

**Identification of Anguillicola spp.**

The five species in the genus *Anguillicola* (family *Anguillicolidae*) have been described by Moravec and Taraschewski (1988). General characters of the genus include a buccal capsule at the anterior end that is armed with a circumoral row of small teeth; a cuticle that may or may not be covered with minute spines; a short, wide esophagus; a dark intestine; and four rectal glands (three large and one small) at the posterior end. In addition, males lack spicules but have a prominent caudal process and six pairs of caudal papillae. Females have a vulva that opens on the tip of a conspicuous cone in the posterior part of the body and are ovoviviparous. Gravid females contain numerous eggs containing second-stage juveniles. All five species are parasites of the swim bladder of eels.
Taxonomic features separating *A. globiceps* from the remaining species include filiform body (long and thin), the esophagus, which is greatly expanded between the nerve ring and the buccal capsule, and a spinose cuticle (i.e., body surface covered with minute spines). The distribution of *A. globiceps* is restricted to the Far East (China and Japan).

*Anguillicola australiensis* can be distinguished by a filiform body, an aspinose cuticle, and a marked neck restriction anterior to the nerve ring, resulting in a head end that is inflated bulbously. *Anguillicola australiensis* is indigenous to Australia but also has been recorded in New Zealand and Italy, although the identity of the specimens from the latter two countries is in question.

Specimens of *A. crassus* can be identified by a fusiform, plump body, aspino-cutele, a large buccal capsule compared with other species, and the shape of the esophagus, which is expanded at its posterior half. More recently, Moravec et al. (1994) described additional features of smaller *A. crassus* adults recovered from small eels. Juvenile stages have been described by Blanc et al. (1992), and Moravec et al. (1993) described *A. crassus* juveniles and eggs.

Specimens of *Anguillicola novaezelandiae* have an aspinose cuticle, a relatively small buccal capsule with minute circumoral teeth, and an esophagus expanded at its posterior half; they are small, plump worms compared with the filiform shape of specimens of *A. australiensis*. Moravec and Taraschewski (1988) suggest that *Anguillicola novaezelandiae* is indigenous to New Zealand and was introduced to Italy.

The morphology of *A. papernai* is unusual in that the buccal capsule is recessed inside the head end, and the anterior and posterior parts of the body are adorned with fibrous, papilla-like protuberances. This species is known only from Africa.

**Effects of *A. crassus* on anguillid hosts**

The ability of *A. crassus* to cause disease in the eel host depends on which of the three anguillid species it is infecting. In Japanese aquaculture facilities the prevalence and intensity of *A. crassus* infections are much higher for the European eel than for the native Japanese eel (Nagasawa et al. 1994). Prevalence of *A. crassus* among wild eel populations in Europe has approached 100% in some localities (Kennedy and Fitch 1990). Infections with *A. crassus* caused mass mortality of eels in Lake Balaton, Hungary, only one year after the first record of its occurrence in that country (Molnár et al. 1993).

Among European eels, severe infections have caused enlarged abdomens, swim bladder rupture, and sometimes host death (Nagasawa et al. 1994). Other symptoms have included dilation of blood vessels in the swim bladder wall; thickened swim bladder wall; skin ulcers in the posterior part of the abdomen; a red, swollen anus; and secondary bacterial infections (van Banning and Haenen 1990). Behavioral changes of heavily infected European eels include decreased appetite, more frequent occurrence near the surface (van Banning and Haenen 1990), and decreased swimming speeds (Sprengel and Lüchtenberg 1991). Neither inflammation of the swim bladder nor pathological changes of infected swim bladders have ever been reported for Japanese eels farmed in Japan and Korea (Nagasawa et al. 1994). Clearly, this exotic parasite has an increased virulence in European eel hosts.

A study conducted by Haenen et al. (1994) from 1986 to 1992 on *A. crassus* infections of European eels in the Netherlands showed that after 1988, prevalence and intensity of infections decreased, and swim bladder lesions became less severe. Thus, Haenen et al. (1996) hypothesized that *A. crassus* had evolved to become less pathogenic and/or that eels had developed resistance to infection. To test this hypothesis, Haenen et al. (1996) infected parasite-free eels with various doses of *A. crassus* third-stage juveniles. Some eels were reinfected 56 days p.i. to study the effect of a primary infection on the development of resistance and to look for antibodies in eel blood against the parasite. Results showed (1) no effects of primary and/or secondary infection dose on *A. crassus* infection levels; (2) increased severity of swim bladder lesions among reinfected eels; and (3) no evidence of antibody production against *A. crassus*. Although Haenen et al. (1996) stated that experimental eels were under much stress, they concluded that European eels had not developed resistance against infection with *A. crassus*.

Ooi et al. (1996) reported high mortality of American eels from eel farms in Taiwan shortly after their introduction into the country for aquaculture. In at least one case, American eel mortality was due to infection with *A. crassus*. We observed effects among infected American eels similar to those noted for European eels, including enlarged abdomens and hematoma at the perianal region. Further studies are needed to determine the relative pathogenicity of *A. crassus* on American eels.

**Outlook for population-level effects of *A. crassus* on *A. rostrata***

Apart from the acute response of Lake Balaton European eels to *A. crassus*, direct effects to eel populations have not been demonstrated. Möller et al. (1991) found no differences in condition factor or liver-somatic index between infected and uninfected European eels.

Based on a 19% decrease in swimming performance in experimental tanks of infected European eels, Sprengel and Lüchtenberg (1991) suggested that infected eels might be more vulnerable to commercial trawl fisheries. Also, because eels depend on selective tidal stream transport for estuarine migrations and retention (McClose and Kleckner 1982), compromised swim bladder performance would be expected to affect feeding, growth, and the ability to escape predation.

The relationship between spawning stock and recruitment in American eels is obscured by panmictic spawning but could be affected by species-wide changes in quantity or attributes of spawning stock. Castonguay et al. (1994) recently suggested that high eel productions coupled with a high female:male ratio in St. Lawrence estuary could lead increased mortality of female yellow and silver eels in that
system with species-wide declines in recruitment. Spawning can only occur after a successful spawning migration by silver eels to the Sargasso Sea. Therefore, it is conceivable that reduced swim bladder function caused by infections of Anguillicola crassus in several important systems such as the Chesapeake Bay and Hudson River could reduce spawning success and cause depression of recruitment throughout North American waters. We strongly urge colleagues to examine American eels for the presence of Anguillicola crassus so we can quickly ascertain its prevalence across North American systems.

Acknowledgments

This study was supported by grants from the Hudson River Foundation and Maryland Sea Grant. We thank Horn Point Laboratory in Cambridge, Maryland, for supplying laboratory space and microscopes.

References


