Article bibliographique

NON-INSECT HOSTS FOR THE ENTOMOGENOUS RHABDITOID NEMATODES *NEOAPLECTANA* (STEINERNEMATIDAE) AND *HETERORHABDITIS* (HETERORHABDITIDAE)

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The rhabditoid genera *Neoaplectana* (Steinernematidae) and *Heterorhabditis* (Heterorhabditidae) are considered as entomogenous nematodes. This is because all natural hosts of these nematodes were insects and because under laboratory conditions, these nematodes develop most suitably in insects. In contrast to the typical obligate parasites of invertebrates, members of the above two families are unique in producing third-stage juveniles which carry and release symbiotic bacteria (*Xenorhabdus* sp.) into the body cavities of their hosts, thus establishing an environment conducive for nematode development and multiplication. The major characteristic which makes these nematodes " parasitic " is the ability of the third-stage dauer (= infectives) to penetrate into the body cavity of living insects and initiate development. Since the host normally dies within 24 hours after the nematode has entered, development in *Heterorhabditis* and *Neoaplectana* is similar to that of microbotrophic rhabditoids which feed on bacteria in decaying habitats. The major difference is that these two genera have become dependent on the presence of *Xenorhabdus* for optimum development. The major functions of the bacteria are a source of nourishment and the production of antibiotic compounds to maintain a stable habitat. The effect of these compounds on microorganisms in the environment have been cited by Gregson and McInerney (1986).

The basic prerequisites for successful development of these nematodes require completion of the following steps : 1) penetration into the body cavity of the potential host, 2) release of the symbiotic bacteria, 3) development to the adult stage, and 4) multiplication and production of infective stage juveniles.

Whereas these conditions are normally met in holometabolous insects which represent the majority of natural hosts for these nematodes, they are rarely met in other animals. The present paper discusses examples in non-insects where penetration, mortality, development or occasionally infective juvenile formation occur.

Table 1

Mortality of *Oncomelania hupensis* snails by *Neoaplectana* and *Heterorhabditis* nematodes (modified from Li *et al.* (1986)).

Nematode species	*Number of* O. hupensis *tested*	*Number of dead* O. hupensis	*Number of snails with developing nematodes*
N. glaseri	10	9	9
N. carpocapsae	10	5	5
N. bibionis	10	6	3
H. heliothidis (NC strain)	10	5	0
H. heliothidis (T 327 strain)	10	9	1
Control	10	0	0

Table 2

Increase in numbers of infective stage *N. glaseri* after infecting the snail, *Oncomelania hupensis* with three dosages (modified from Li *et al.* (1986)).

Dosage (nemas/pot)	*Number of* O. hupensis *examined*	*Average number of living and dead nematodes from infected* O. hupensis		*% increase of nematodes over initial dosage*
		living	dead	
106,814 (340/cm²)	15	1 773 (1 200-2 300)	774 (30-1 800)	15
84,248 (300/cm²)	5	3 080 (1 900-4 500)	460 (300- 600)	69
62,832 (200/cm²)	5	2 040 (1 600-2 500)	370 (100- 600)	65

Phylum Mollusca

Class Gastropoda

A study by Li *et al.* (1986) was the first to show that *Neoaplectana* and *Heterorhabditis* nematodes could infect a snail. They reported mortality in the semi-aquatic *Oncomelania hupensis* (Hydrobiidae) from infections with *H. heliothidis, N. bibionis, N. carpocapsae* and *N. glaseri.* All four species showed some development in the snail, although *N. glaseri* appeared to be the best adapted to this host (Tab. 1). In further experiments, living snails were added to 4.5 cm plastic dishes containing moistened soil together with various concentrations of *N. glaseri.* After being held at 17-23° for one week, the snails were crushed and the number of nematodes counted. Nematode penetration, bacterial release, nematode development and production of infective juveniles occurred with *N. glaseri* (Li *et al.*, 1986) (Tab. 2).

Phylum Arthropoda

Class Crustacea

Under laboratory conditions, Poinar and Paff (1985) demonstrated that both *N. carpocapse* and *H. heliothidis* were able to infect and develop in the hemocoel of the terrestrial isopods, *Armadillidium vulgare* and *Porcellio scaber.* Although the body wall broke down shortly after the host's deaths and the body cavity was invaded by other microorganisms, some reproduction occurred and a few hundred infective stages of both nematode species were produced (Tabs 3 & 4). It was interesting that no infection of either isopod species resulted when *N. glaseri* was used. In laboratory experiments, Kermarrec and Mauléon (1985) placed the fresh water prawns, *Atyia innocous* and *Macrobrachium acanthurus* in a liter of water containing 2×10^5 infective stages of *N. carpocapsae.* No effect of the nematodes on the shrimp was noted.

Table 3

Effect of *Neoaplectana* species on non-insect invertebrate hosts (NS = not stated)

Phylum (Class)	*Host*	*Nematode*	*Penetration*	*Bacterial release*	*Development*	*Multiplication*	*Reference*
Mollusca							
(Gastropoda)	*Oncomelania hupensis*	*N. glaseri*	+	N.S.	+	+	Li *et al.*, 1986
	Oncomelania hupensis	*N. carpocapsae*	+	N.S.	+	–	Li *et al.*, 1986
	Oncomelania hupensis	*N. bibionis*	+	N.S.	+	–	Li *et al.*, 1986
Arthropoda							
(Symphyla)	*Scutigerella immaculata*	*N. carpocapsae*	+	N.S.	+	+	Swenson, 1966
(Collembola)	*Onychiurus armatus*	*N. carpocapsae*	+	N.S.	N.S.	N.S.	Rahayu, 1983
(Arachnida)	*Pholcus phalangiodes*	*N. carpocapsae*	+	+	+	–	Poinar & Thomas, 1985 A
	Latrodectus mactans	*N. carpocapsae*	+	+	+	–	Poinar & Thomas, 1985 A
	Pirata sp.	*N. carpocapsae*	+	+	+	–	Poinar & Thomas, 1985 A
	Phlangium sp.	*N. carpocapsae*	+	+	+	+	Poinar & Thomas, 1985 A
	Garypus californicus	*N. carpocapsae*	+	+	+	+	Poinar *et al.*, 1985
(Crustacea)	*Armadillidium vulgare*	*N. carpocapsae*	+	+	+	+	Poinar & Paff, 1985
	Armadillidium vulgare	*N. glaseri*	–	–	–	–	Poinar & Paff, 1985
	Porcellio scaber	*N. carpocapsae*	+	+	+	+	Poinar & Paff, 1985
	Porcellio scaber	*N. glaseri*	–	–	–	–	Poinar & Paff, 1985
(Diplopoda)	*Oxidus gracilis*	*N. carpocapsae*	+	+	–	–	Poinar & Thomas, 1985 B

Class Symphyla

Swenson (1966) showed that the DD-136 strain of *N. carpocapsae* was able to infect, develop and reproduce in the garden symphylan, *Scutigerella immaculata.* Infective-stage nematodes emerged from parasitized hosts five to seven days after infection (Tab. 3). Since garden symphylans are sometimes considered as plant pests, it may be feasible to use nematodes for their control.

Class Collembola

Springtails were originally considered to be insects, however they are now placed in a separate class. Using the DD-136 strain of *N. carpocapsae,* Rahayu (1983) was able to obtain some infection of *Onychiurus armatus* under laboratory conditions (Tab. 3). Rahayu (1983) also reported that the application of this nematode to sugar beet fields at seedling and two-leaf stages consider-

ably reduced the numbers of Collembola. Similar effects of the DD-136 strain of *N. carpocapsae* on field populations of *Onychiurus* collembola had been reported by Edwards and Oswald (1981).

Class Arachnida

In laboratory experiments, Poinar and Thomas (1985) showed that both *N. carpocapsae* and *H. heliothidis* could enter and kill the aerial spiders, *Pholcus phalangiodes* and *Latrodectus mactans*, a ground spider, *Pirata* sp. and a harvestman, *Phalangium* sp., respectively. Although the nematodes developed to the adult stage in all four hosts, they reproduced and formed infective juveniles only in *Phalangium* sp. An average of 7 200 infectives (N = 6) of *N. carpocapsae* and 26 000 infectives of *H. heliothidis* were produced from each parasitized *Phalangium* sp. (Tabs 3 & 4). The absence of reproducing forms in the other arachnids was attributed to the appearance of foreign bacteria which made the cadavers unhospitable for the developing nematodes.

Table 4

Effect of *Heterorhabditis* species on non-insect invertebrate hosts

Phylum (Class)	*Host*	*Nematode*	*Penetration*	*Bacterial release*	*Development*	*Multiplication*	*Reference*
Mollusca							
(Gastropoda)	*Oncomelania hupensis*	*H. heliothidis*	+	+	+	–	Li *et al.*, 1986
Arthropoda							
(Arachnida)	*Pholcus phalangiodes*	*H. heliothidis*	+	+	+	–	Poinar & Thomas, 1985 A
	Latrodectus mactans	*H. heliothidis*	+	+	+	–	Poinar & Thomas, 1985 A
	Pirata sp.	*H. heliothidis*	+	+	+	–	Poinar & Thomas, 1985 A
	Phlangium sp.	*H. heliothidis*	+	+	+	+	Poinar & Thomas, 1985 A
	Garypus californicus	*H. heliothidis*	+	+	+	+	Poinar *et al.*, 1985
(Crustacea)	*Armadillidium vulgare*	*H. heliothidis*	+	+	+	+	Poinar & Paff, 1985
	Porcellio scaber	*H. heliothidis*	+	+	+	+	Poinar & Paff, 1985
(Diplopoda)	*Oxidus gracilis*	*H. heliothidis*	+	+	–	–	Poinar & Thomas, 1985 B

The pseudoscorpion, *Garypus californicus*, also proved susceptible to infection by *N. carpocapsae* and *H. heliothidis* (Poinar, Thomas & Lee, 1985). The nematodes released their symbiotic bacteria, developed to the adult stage and multiplied in the host cadaver (Tabs 3 & 4). However, as a result of foreign bacteria in the environment, only a few infectives were formed.

Class Diplopoda

Under laboratory conditions, Poinar and Thomas (1985 *b*) showed that both *N. carpocapsae* and *H. heliothidis* were able to infect and kill the garden millipede, *Oxidus gracilis.*

Although the penetrating infectives did liberate their *Xenorhabdus* bacteria, a quick encapsulation reaction and enmeshement of the nematodes in the host's tracheoles and connective tissue restricted further development.

Phylum Vertebrata

Class Pices

Kermarrec and Mauleon (1985) challenged 30 specimens of the fish, *Lebister reticulatus* with 2 10^5 infective-stage *N. carpocapsae* in a liter of water. No nematode development or any effect on the fish was noted (Tab. 5).

Class Reptilis

Kermarrec and Mauléon (1985) challenged ten individuals of the lizard *Anolis marmoratus* with 5 000 infective stages of *N. carpocapsae per os* every third day for 30 days. No mortality of the lizards were observed (Tab. 5). In a single case, some necrosis of the intestinal epithelium adjacent to a mass of dead infective-stage nematodes was noted. Poinar and Miller (unpubl. rept., 1988) challenged snapping turtle hatchlings *(per os)* with 2 000 infectives of *N. carpocapsae* and *H. heliothidis*, respectively (N = 20 for each nematode species). No mortality or signs of disease were noted in the turtles after two weeks.

Class Amphibia

Under laboratory conditions, Kermarrec and Mauléon (1985) demonstrated that *N. carpocapsae* could quickly kill tadpoles of *Bufo marinus* (Tab. 5). Chal-

Table 5

Results of exposing rhabditoid entomogenous nematodes to vertebrates

Class	*Host*	*Nematode*	*Method Application*	*Dose # infectives*	*Effect*	*Reference*
Pices	*Lebistes reticulatus*	*N. carpocapsae*	in water	2×10^5	none	Kermarrec & Mauléon, 1985
Reptilia	*Anolis marmoratus*	*N. carpocapsae*	per os	50,000	none	Kermarrec & Mauléon, 1985
Amphibia	*Bufo marinus*	*N. carpocapsae*	in water	100/ml	mortality of young tadpoles	Kermarrec & Mauléon, 1985
	Hyla regilla	*N. carpocapsae*	in water	100, 200 and 400/ml	mortality of young tadpoles	Poinar & Thomas, 1988
	Hyla regilla	*H. heliothidis*	in water	100, 200 and 400/ml	mortality of young tadpoles	Poinar & Thomas, 1988
	Xenopus laevis	*N. carpocapsae*	in water	200, 400 and 800/ml	mortality of young tadpoles	Poinar & Thomas, 1988
	Xenopus laevis	*H. heliothidis*	in water	200, 400 and 800/ml	mortality of young tadpoles	Poinar & Thomas, 1988
Aves	*Gallus gallus* (chicken)	*N. carpocapsae*	per os	60,000	none	Kermarrec & Mauléon, 1985
Mammalia	*Microtus pennsylvanicus*	*N. carpocapsae*	per os	200,000	none	Schmiege, 1963
	Rattus rattus	*N. carpocapsae*	per os	200,000	none	Nutrilite Products, Inc. (in Poinar, 1979)
	Rattus rattus	*N. glaseri*	interperitoneally	5-6,000	some nematode viability after 5 days	Jackson & Bradbury, 1970
	Rattus rattus	*N. carpocapsae*	interperitoneally	50,000	some nematode viability after 2 days	Gaugler & Boush, 1979
	Rattus rattus	*N. carpocapsae*	per os	50,000	none	Gaugler & Boush, 1979
	mice	*N. carpocapsae*	subcutaneously	1,000	no development	Poinar *et al.*, 1982
	mice	*H. heliothidis*	subcutaneously	1,000	no development	Poinar *et al.*, 1982
	mice	*N. carpocapsae*	per os	62,500 (over 25 days)	no development	Kermarrec & Mauléon, 1985
	mice	*N. carpocapsae*	per os	1 000; 10,000	no development	Kobayashi *et al.*, 1987
	mice	*N. bibionis*	per os	1 000; 10,000	no development	Kobayashi *et al.*, 1987
	mice	*N. glaseri*	per os	1 000; 10,000	no development	Kobayashi *et al.*, 1987
	mice	*H. heliothidis*	per os	1 000; 10,000	no development	Kobayashi *et al.*, 1987
	mice	*N. carpocapsae*	subcutaneously	2×10^4	ulcers in skin	Kobayashi *et al.*, 1987
				10×10^4	no nema develop.	Kobayashi *et al.*, 1987
	mice	*N. bibionis*	subcutaneously	2×10^4	ulcers in skin	Kobayashi *et al.*, 1987
				10×10^4	no nema develop.	
	mice	*N. carpocapsae*	interperitoneal	2×10^4	no development	Kobayashi *et al.*, 1987
				10×10^4		
	mice	*N. bibionis*	interperitoneal	2×10^4	no development	Kobayashi *et al.*, 1987
				10×10^4		

lenging 60 tadpoles with a dose of 100 nematodes/ml (1 000 ml container) resulted in 100 % mortality after five days. The authors noted that dead clustered bundles of infective stages were recovered from the bucal cavity and intestine of the tadpoles, however no explanation of the cause of death was given. After hundreds of dissections, they recovered an adult female nematode from the body cavity of one tadpole.

Poinar and Thomas (1988) performed similar tests with *N. carpocapsae* and *H. heliothidis* against the tadpoles of the western tree frog, *Hyla regilla* and the clawed frog, *Xenopus laevis*. Both nematode species could kill young tadpoles of both host species, respectively (Tab. 5). The infectives were ingested by the tadpoles, penetrated through the gut wall, entered the body cavity and in a few cases with *N. carpocapsae* released their symbiotic bacteria. In two instances, the infectives of *N. carpocapsae* developed into mature females in the body cavity of their tadpole hosts.

Both Kermarrec and Mauléon (1985) and Poinar and Thomas (1988) noted that the susceptibility of tadpoles decreased markedly with age, which was probably correlated with a thickening of the epithelial gut wall, thus making it more difficult for the nematodes to enter. The latter authors also noted a rapid defense reaction involving encapsulation of the nematodes by blood cells in *H. regilla*.

CLASS AVES

Tests with rhabditoid entomogenous nematodes against birds were conducted only by Kermarrec and Mauléon (1985). No effect was noted when 60 000 infectives of *N. carpocapsae* were given *per os* to 30 domestic chickens *(Gallus gallus).*

CLASS MAMMALIA

The first mammalian test with entomogenous rhabditid nematodes was reported by Schmiege (1963) who fed four field mice *(Microtus pennsylvanicus)* each a dose of 200 000 infective stage *N. carpocapsae* over a five-day period. Dead nematodes were recovered from the feces and no damage to the mice was reported (Tab. 5).

Jackson and Bradbury (1970) injected from 5 000 to 6 000 infectives of *N. glaseri* into the peritoneal cavity of female rats. Both living and dead nematodes were recovered from the peritoneal cavity of treated rats five days later (Tab. 5). The majority of the recovered nematodes were still infectives. None had developed to the adult stage and rarely had a recovered nematode developed beyond the third stage. When the living nematodes recovered from the injected rats were placed in culture, some developed to adults but there was no reproduction. At the time of removal from the rat peritoneal cavity, many nematodes were covered with layers of rat peritoneal exudate cells. Those removed after three days showed no cuticular alterations, but nematodes recovered four and five days following injection showed two cuticular changes, a swollen surface sheet and a disrupted striation pattern. The authors concluded that the latter condition was not a result of high temperature but was caused by host cells, possibly as a result of enzymatic action.

Mammalian tests with entomogenous rhabditoid nematodes also were conducted by Nutrilite Products, Inc. in 1970 (in Poinar, 1979). All stages of *N. carpocapsae* were fed to laboratory rats. All nematodes recovered from the feces were dead and there was no inflammation of the alimentary tract or nematode developpment inside the rat (Tab. 5).

Since these early attempts, mice and rats have been challenged with various species of *Neoaplectana* and *Heterorhabditis heliothidis* (Tab. 5). Gaugler and Boush (1979) introduced 50 000 *N. carpocapsae per os* and intraperitoneally, respectively, into five-week-old albino rats. All treated rats showed normal weight gains over the 36-day test period and gross tissue examination of the treated rats sacrificed on day 36 revealed neither nematodes nor changes attributable to nematodes. Dead nematodes were recovered from the feces of rats treated *per os.* Some nematodes did remain alive after two days when introduced intraperitoneally. However no development was noted and many were encapsulated by peritoneal macrophages or occasionally enmeshed in gelatinous sheets of host material. However, the nematodes were unable to cause injury, become established or survive for over two days in the rat body.

Poinar *et al.* (1982) inoculated 1 000 infective stages of *H. heliothidis* and *N. carpocapsae,* respectively, subcutaneously into adult Swiss albino mice. No disease symptoms or nematode development were noted in the adult mice during the 14-day postinoculation period.

Kobayashi, Okano and Kirihara (1987) placed doses of 1 000 and 10 000 infective stages of *N. glaseri, H. heliothidis, N. bibionis* and *N. carpocapsae,* respectively, into five-week-old male mice with a stomach probe. The same doses of *N. bibionis* and *N. carpocapsae* were injected subcutaneously or interperitoneally into similar mice. Mice with oral doses were dissected after seven days. No nematodes were recovered and no disease symptoms were noted in these mice.

Although no hosts died, the subcutaneous injection of nematodes into the hypodermal tissue of mice resulted in ulcers in the injured portion of the skin. These ulcers became dislodged on the fourth day after injection and contained dead nematodes. Dead nematodes were also recovered from the inoculated area on the 14th day after injection. The authors concluded that the ulcers were not specifically formed by the penetrating nematodes, but from an allergic reaction resulting from the injection of foreign material into the hypodermis. An allergic reaction was also considered responsible for mouse deaths after the interperitoneal injection of 100 000 infective stage *N. carpocapsae.*

Discussion

Representatives of the classes Gastropoda, Symphyla, Collembola, Arachnida, Crustacea and Diplopoda are able to be infected by neoaplectanid and heterorhabditid nematodes. However infective juvenile production is not completed in every case. Thus, aside from the four basic prerequisites for successful development mentioned earlier (penetration, bacterial release, development and infective juvenile production), there are additional conditions. Many invertebrates are not attacked because they are too small, they are not attractive to the infectives, there are no natural openings (or thin enough cuticles in the case of *Heterorhabditis* spp.) or the habitat prohibits nematode activity (too dry or too wet). An aspect of arthropod structure important in nematode infection is tegument integrity. With isopod crustaceans and spiders, the body wall breaks down rapidly after death, resulting in the exposure of the nematodes to the external environment. Ideal development for the nematodes requires a closed environment containing *Xenorhabdus* bacteria and hemolymph and the absence of host defense reactions. A breakdown of the intersegmental membranes results in the loss of this closed environment and interference by contaminants. It would appear that in the case of invertebrates, one reason why these

nematodes are such successful parasites of insects is the integral structure of the intersegmental host membranes after death.

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