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NEMACYCLE: A CODING SYSTEM FOR REPRESENTATION OF NEMATODE LIFE-CYCLES

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ABSTRACT: Parasitism, which probably originated several times within nematodes, has produced complicated life-cycles, a knowledge of whose detailed mechanisms is very important for understanding evolution of life traits. In the scientific literature descriptions of life-cycles are generally presented following different organizations; also, graphic representations of life-cycles are frequently more artistic than meaningful; thus, because different authors make use of different designs, it is difficult to analyse and compare life-cycles. In the present work we attempt to propose an alternative based: first, on a standardization of the presentation of the information given in the text, and second, on a graphic representation of the cycles using a new coding system. This system, called «Nemacycle», is based on the linear representation of the cycles designed using the same basic symbols. This makes it easier to recognize which cycles include identical or similar sequences of events and can be helpful for conceptualization and comparison of component traits. Several tentative examples of life-cycles are given and discussed by way of illustration.

KEY WORDS: Nematodes, parasites, life-cycle, Nemacycle, *Trichuris trichiurus, Calodium hepaticum, Trichinella spiralis, Pearsonema plica, Dioctophyme renale, Pelodera orbitalis, Strongyloides* spp., *Rhabdias* spp.

INTRODUCTION

Parasitism, which probably originated several times within nematodes, has produced complicated life-cycles. Various adaptations, associated with different transmission strategies, arose during evolutionary times, sometimes producing convergent life traits. This results in many different patterns which can be observed in the life-cycles of the various orders and families. Descriptions and graphic representations of life-cycles are frequently presented by different authors making use of different designs. This makes it difficult to analyse and compare life-cycles. The aim of our work is to propose a general pattern for the representation of parasitic nematode life-cycles. Our proposal is based on: first, a standardization of the presentation of the information given in the text, and second, a graphic representation of the cycles using a new coding system. This system, called «Nemacycle», is based on the linear representation of the cycles designed using the same basic symbols. This make it easier to recognize which cycles include identical or similar sequences and should be helpful for conceptualization and comparison of component traits. Tentative examples of parasite cycles representation, using the system, are given.

METHOD

Organisation of text: Each description of a life-cycle is presented following the same successive sections: «*Hosts*» gives an enumeration of the definitive host(s) and when necessary, intermediate and (or) paratenic, and (or) sporadic host(s); «*Range*» gives the range of the parasite; «*Description of life-cycle*» gives a synopsis of the consecutive stages of the life-cycle; the «*Timing*» section collects all available information on the duration of different stages and life spans of the parasite. Three facultative last sections may be added, entitled «*Particularity of life-cycle*», «*Advanced seclusion*» and «*Dubious or unknown sequences*», where the outstanding sequence(s) in a life-cycle are sketched in figures using a white arrow, an exclamation or a question mark, respectively. Most of the information given in this part was found in ANDERSON (1993).

«Nemacycle», a tentative coding assemblage: Fig. 1 displays the basic symbols. The different stages for a parasite such as adult, larvae, embryo and eggs are figured using A, L, E or ω, respectively. Each letter may be accompanied by a symbol, a number or a small cap letter to indicate which variety is heeded. A solid circle figures egg shell. Larvae may remain enclosed in moult(s) of previous stages(s): this is figured using elliptical outline(s). Hosts are designated using different kinds of bold outlined boxes. Paratenic hosts could be drawn with full lines, when they are necessarily present in the cycle, or using dashed lines when they are considered optional.

A solid square around an adult parasite or larval stage means that this stage is «*endoparasitic*». By endoparasitism we consider all those cases in which the adult parasites or their larval stages are not living in the cavity of the digestive tract of their hosts: for instance, if they are living in the gut mucosa, in other tissues, in a blood ves-

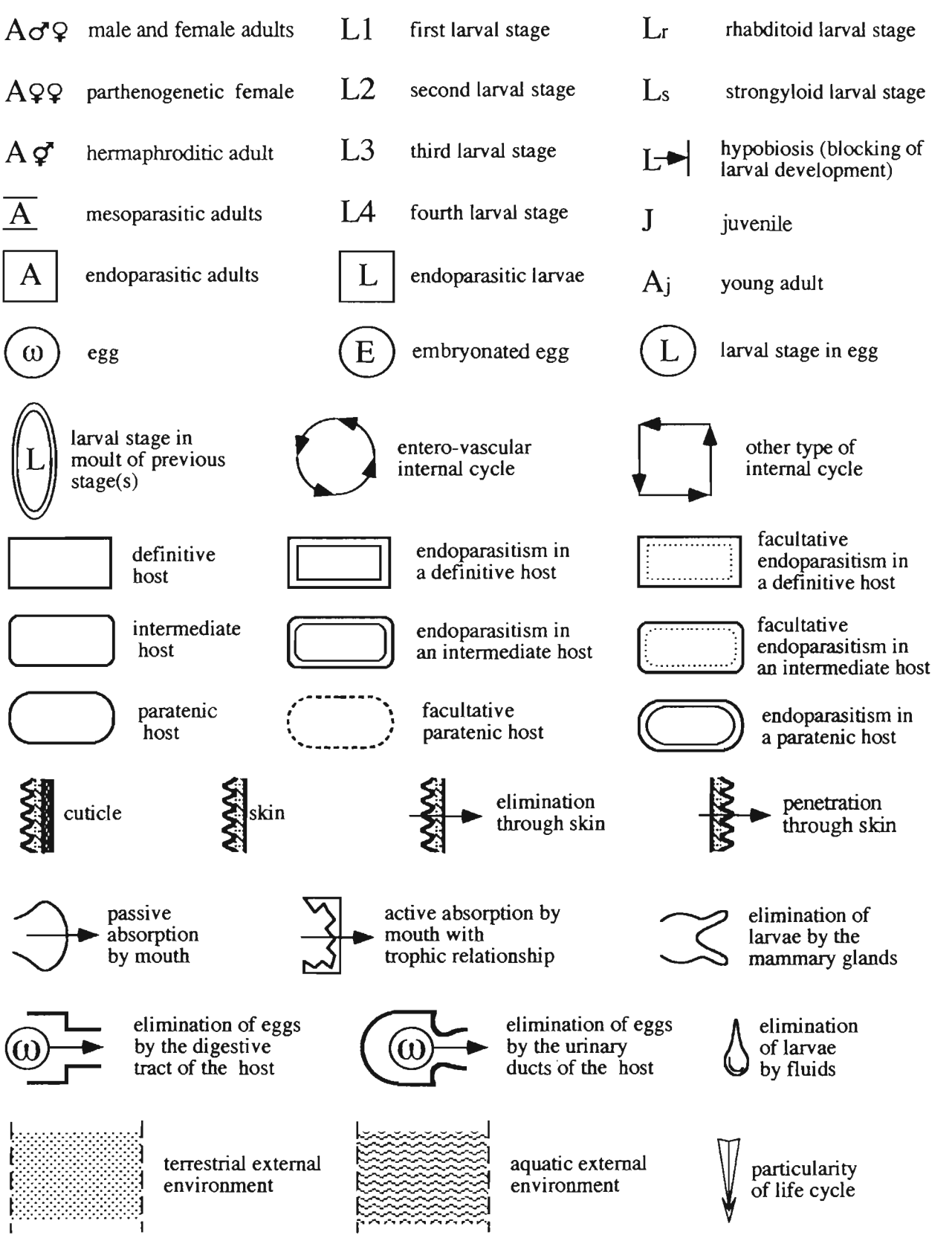

Fig. 1.– «Nemacycle»: a set of symbols for coding parasitic nematode life-cycles. Explanations in text.

sel, in the urinary bladder, etc. Endoparasitism may be optional; for instance, a parasite may, or may not, enter the gut mucosa: in this case the corresponding square box is dashlined. Endoparasites establish themselves in different ways. The most frequent is when parasites cross the gut mucosa barrier, enter the blood or lymphatic vessels and, carried by flow and (or) stopped by capillary networks, get established at the level of tissue. We call this phenomenon «*entero-vascular internal cycle*» which is represented by four circular black arrows. Any «*other type of internal cycle*» (for instance when parasites penetrate through the skin and then migrate until they find their specific location), is represented by four squared black arrows.

Several other symbols are utilized to represent how the parasites invade a host: through skin (or cuticle), by passive absorption (the host swallows eggs or infesting larvae when eating infected food), or by active absorption related with trophic relationships (the definitive host is a predator, scavenger or cannibal of the intermediate host, or the blood-sucking intermediate host ingests parasite larvae when feeding on the definitive host). The same symbols, with a reversed black arrow are used to sketch how the parasites may leave (or get eliminated by) a host: through skin (or cuticle), discharged with faeces (elimination by the digestive tract), or with urine (elimination by the urinary ducts), eliminated by fluids (tears, sperm, etc.), or with milk (elimination by the mammary gland). Additional symbols, a vertical white arrow or an exclamation mark, are used for making apparent outstanding sequence(s) in a life-cycle. The exclamation mark being restricted to «*advanced seclusion*», which means particularity of life-cycle allowing a parasite to improve «*seclusion*» (see discussion section for explanation). A question mark announces a dubious or unknown sequence.

Using the code: Each parasite life-cycle is linearly represented, beginning on the left side with elimination of eggs or larvae by the definitive host, and ending when adult parasites have reached the location where they are able to produce offspring.

RESULTS

Trichuris trichiurus (Linnaeus, 1788) (Fig. 2A)

Host(s): *Definitive*: primates including humans. *Intermediate*: none. *Paratenic*: none. *Sporadic*: experimentally, guinea pigs and rabbits (FÜLLEBORN, 1923).

Range: Cosmopolitan. Regarded as the second most common parasitic infection in humans in the tropics (BUNDY & COOPER, 1989).

Description of life-cycle: Eggs (50 × 20 µm) dispersed with faeces of definitive host, develop to the infective stage (L1) in the external environment and are passively ingested by a susceptible definitive host. In the digestive tract eggs are hatched and the larvae optionally penetrate the epithelium. After four moults L1 larvae give rise to adult males and females living in the caecum and colon. Adults (♂ 30-45 mm; ♀ 35-50 mm) with cephalic end buried in the mucosal epithelium of the gut, inducing formation of a syncitium on which they are presumed to be feeding (LEE & WRIGHT, 1978).

Timing: Full development of eggs in 21-25 days at 26° C (FÜLLEBORN, 1923); 28 days at 28-30° C (HASEGAWA, 1924); 21 days at 30° C (SPINDLER, 1929); 14 days at 37° C (ARTEMENKO, 1937). After DINNIK & DINNIK (1937, 1939) full development at: 120 days at 20° C; 57 days at 25° C; 17,5 days at 30° C; 11 days at 35° C. Estimated prepatent period in humans 2-3 months (FAUST *et al.*, 1975; BUNDY & COOPER, 1989). Estimated number of eggs/female/day: 3000-20000 (FAUST *et al.*, 1975). Estimated parasitic life span of worms in humans 1-2 years, occasionally 4 years (BUNDY & COOPER, 1989).

Calodium hepaticum (Bancroft, 1893) (Fig. 2B)

Host(s): *Definitive*: rodents. *Intermediate*: none. *Paratenic*: carnivorous, necrophagous or cannibal mammals. *Sporadic*: dogs, cats, hyraxes, peccaries, primates and humans.

Range: Cosmopolitan.

Description of life-cycle: Small groups of uncleaved eggs (55 x 32 µm) encapsulated in liver tissue of definitive host. Eggs, when eaten by a carnivorous, necrophagous or cannibal paratenic host, pass through the gut, are dispersed with faeces, develop to the infective stage (L1) in the external environment and are passively ingested by a putative definitive host (FREEMAN & WRIGHT, 1960). L1 larvae (140-190 µm) cross the gut mucosa, travel to the liver by the hepatic portal system (entero-vascular internal cycle) and after four moults give rise to adult males and females (♂, ♀ 20 mm). After mating female worms move into the liver and deposit small groups of uncleaved eggs which become encapsulated by host tissue (LUTTERMOSER, 1938; PAVLOV, 1955). Adults enclosed by multinucleate cytoplasmic masses originating from liver cells which WRIGHT (1974) attested to prove that they are feeding on liver parenchyma.

Timing: Full development of eggs in moist environment at room temperature in 35-40 days (LUTTERMOSER, 1938; PAVLOV, 1955). Experimental infestation of mice with eggs (FREEMAN & WRIGHT, 1960; WRIGHT, 1961) gave: L1 larvae in the liver 2 days postinfection; moulting L1, 3-4 days postinfection; L3 appeared in 5 days; L4 appeared in 9 days; full development of males 18 days; of females 20 days (23 days after SHIMATANI, 1961); appearance of first eggs in liver parenchyma at 21 days; death of males achieved after 40 days; of females after 59 days.

Particularity of life-cycle: ↑1: Eggs unable to be released from the living host (BANCROFT, 1893; FÜLLEBORN, 1924) and also unable to develop after liver decaying (SHORB, 1931; LUTTERMOSER, 1938; ORLOV, 1948; PAVLOV, 1955). ↑2: Carnivorous, necrophagous or cannibal mammalian paratenic host necessarily required for eggs releasing, dissemination and further development.

Advanced seclusion: !: Entero-vascular internal cycle leading L1 larvae to the liver through the hepatic portal system.

Trichinella spiralis (Owen, 1835) (Fig. 2C)

Host(s): *Definitive*: carnivorous, necrophagous or cannibal mammals. *Intermediate*: carnivorous, necrophagous or cannibal mammals. *Paratenic*: none. *Sporadic*: likely any mammal when feeding on infested raw meat.

Range: Cosmopolitan.

Description of life-cycle: L1 (745-975 μm long) infective larvae encapsulated into «nurse» striated muscles cells, having a developed reproductive primordia (KOZEC, 1971a, b). When eaten by a carnivorous, necrophagous or cannibal mammal, larvae are digested out of the muscle capsule at level of stomach and gut, invade the small intestine mucosa where they undergo 4 moults and give rise to adult males (1,4-1,6 mm long) and females (3-3.5 mm long), whose entire body is embedded in mucosal epithelium. Mating occurs in the epithelium of the hosts where females deposit L1 larvae (69-99 μm long). Larvae migrate through the intestine mucosa to capillary venules and lymphatic, then to general circulation (entero-vascular internal cycle) from where they emerge to striated muscles, especially of diaphragm, larynx, tongue, intercostal, biceps, abdomen, psoas, pectoral, gastrocnemius and deltoid.

Timing: L1 larvae invade gut mucosa 10-60 mn postinfection (GOULD, 1945; DESPOMMIER *et al.*, 1978), and reach adulthood within 36 hours. Moults take place at 10, 17, 24 and 29 hours in males and 12, 19, 26 and 36 hours in females (ALI KHAN, 1966). Mating takes place from 32 to 144 hours postinfection (GOULD *et al.*, 1957). Each female produces approximately 500 larvae (KHAMBOONRUANG, 1971) which after invading their «*nurse*» cell develop within 19 days (DESPOMMIER *et al.*, 1975). Life span of encapsulated larvae may embrace the entire life of the host. Life span of adult worms in the gut of host not reputed to exceed 1 month (RAPPAPORT, 1943; COKER, 1955; LARSH, 1963; KHAMBOONRUANG, 1971).

Particularity of life-cycle: ↑1: Viviparous female nematodes directly producing L1 infective stages. ↑2: Fully-developed muscle larvae having a reproductive primordia. Precocious development of genitalia and viviparity are assumed to be advantageous by ANDERSON (1993), because they are «related to extreme rapidity of maturation in the final host which allows the parasite to reproduce before the host's immune system has been effectively mobilized».

Advanced seclusion: !:Viviparity allows *Trichinella* spp. to suppress egg development in the external environment, which improves seclusion because the exposure of the parasite to outside contingencies is so reduced in time and space that it can be considered fortuitous. In turn, storage and accumulation of infective stages in muscles of definitive hosts make a predator/prey relationship necessary for natural transmission, also making definitive hosts behave as intermediate hosts for the continuation of the cycle.

Pearsonema plica (Rudolphi, 1819) (Fig. 2D)

Host(s): *Definitive*: canids and mustelids. *Intermediate*: none. *Paratenic*: terrestrial oligochaetes (*Lumbricus rubellus* and *L. terrestris*). *Sporadic*: none recorded.

Range: Cosmopolitan.

Description of life-cycle: Eggs eliminated in the external environment through the urinary ducts of the definitive host, develop to the infective stage (L1) and are passively ingested by a paratenic host where they hatch. L1 larvae get encapsulated in muscles. When eaten by a putative definitive host, L1 larvae penetrate the gut mucosa and achieve the first moult. L2 larvae shift in the portal blood vessel (entero-vascular internal cycle) and migrate to the urinary bladder, where they have three additional moults, giving birth to male and female adults.

Timing: Full development of eggs in external environment in 20-21 days at 26-28° C (PETROV & BOROVKOVA, 1942). L1 infective within 1 day after ingestion by earthworms; L1 develop to L2 in 8-10 days; L2 develop to L3 in about 30 days and to adult stage in 53-63 days (ENIGK, 1950).

Particularity of life-cycle: ↑: Parasite of the urinary bladder.

Advanced seclusion: !: A regular paratenic host allows infective larvae (L1) to be protected, which improves seclusion and also facilitate parasite transmission, (i) because infective stages are accumulated, (ii) because active search for prey by predators replaces fortuitous passive absorption.

Dioctophyme renale (Goeze, 1782) (Fig. 2E)

Host(s): *Definitive*: mustelids. *Intermediate*: aquatic oligochaete (*Lumbricus variegatus*). *Paratenic*: fishes; also recorded in frogs (KARMANOVA, 1968; MACE & ANDERSON, 1975). *Sporadic*: canids especially *Canis lupus*; bears: *Ursus ursus*; racoons: *Procyon lotor, Nasua nasua* (KARMANOVA, 1968; MACE, 1976); occasional reports in humans and domesticated animals, rarely in rats and seals.

Range: Cosmopolitan in temperate regions of the world.

Description of life-cycle: Eggs (65 x 45 μm) eliminated

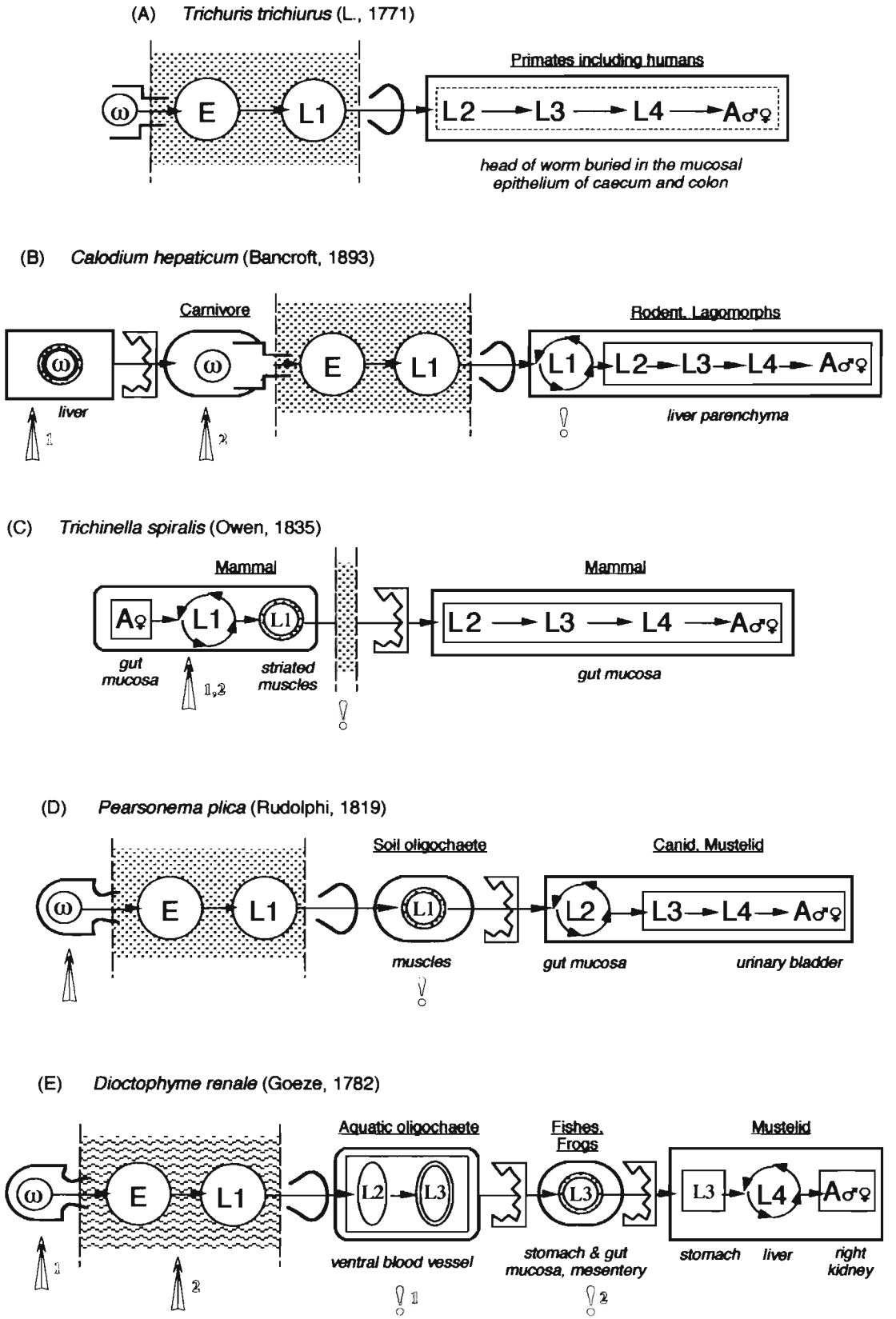

Fig. 2.– Example life-cycles in the Enoplia.

in the external environment through the urinary ducts of the definitive host. When incubated in water, they develop to the infective stage (L1) and are passively ingested by an intermediate host where they hatch. L1 invade the coelom of the intermediate host, then stay in the ventral blood vessel and have two moults. L2 (about 2 mm long) retain the first-stage cuticle. L3 (6-12 mm long) retain cuticle of first and second moults. The intermediate host is eaten by a fish (paratenic host) where the L3 larvae are found encapsulated on the stomach and gut mucosa and on the mesentery. Several paratenic hosts having predator/prey relationships may improve infesting larvae accumulation before being eaten by a carnivorous mammal (the definitive host) in which L3 larvae achieve their development, having their third moult in the stomach mucosa from which L4 larvae shift into the portal blood vessel (entero-vascular internal cycle) and then into the liver. MACE & ANDERSON (1975) observe the proximity of the right lobe of the liver to both the stomach and the right kidney of minks and strongly suggest that larvae actively invade the kidney through the body cavity. In the kidney L4 give birth to male and female adults (200-800 mm long, 8-12 mm wide).

Timing: Full development of L1 in eggs, in water between 14-30° C, about 35 days (MACE & ANDERSON, 1975), 6 months according to GOLVAN (1990). L1 remain active about 1 week after development (MACE & ANDERSON, 1975). L1 develop to L2 in about 50 days; L2 develop to L3 in about 100 days; usually only 1-2 fully-developed L3 found per host. L3 develop to L4 5 days postinfection in the definitive host; L4 observed in the liver 50 days postinfection (MACE & ANDERSON, 1975). Development to adult stage in definitive host 108-134 days postinfection (MEASURES & ANDERSON, 1985).

Particularity of life-cycle: ↑1: Parasite of the right kidney. ↑2: Eggs develop necessarily in water.

Advanced seclusion: !1: A regular intermediate host protects development of infective larvae (L1) to L3 stage, which improves seclusion. !2: Although experimental infection of susceptible definitive hosts with larvae directly from oligochaete is possible (KARMANOVA, 1968; MACE & ANDERSON, 1975), definitive hosts generally get infected when eating paratenic hosts, which facilitates parasite transmission: (i) because paratenic hosts accumulate infective stages, and (ii) because active search for prey by predators replaces fortuitous passive absorption.

Pelodera orbitalis Sudhaus et Schulte, 1986 (Fig. 3A)

Host(s): *Definitive*: none. *Intermediate*: none. *Paratenic*: none. *Sporadic*: rodents.

Range: Holarctic.

Description of life-cycle: Eggs (25 × 10 μm) laid in nests of rodents by adult free-living rhabditoid females. They develop to the first larval stage (Lr1; 200-300 μm) in the external environment and, after one moult, give rise to Lr2 (300-350 μm). Lr2 stages transform into free-living fourth-stage (Lr4; M 600 μm; V 800 μm) and adults (♂, 1100 μm; ♀ 1200 μm) in three different ways (larval triphenism), either: (i) free-living third stage larvae (Lr3; 600 μm) rapidly transformed into fourth stage, (ii) dauer third stage larvae (Lr3; 600 μm) which retain the cuticle of the second stage and arrest their development until suitable conditions are attained, or (iii) infective third stage larvae (Lr3; 730 μm) which can be found in the orbit and conjunctival sacks of rodents with which they are associated. Parasitic larvae feed on lachrymal secretions, increase in size, and then leave the orbit and enter the nest or runways of the host (SCHULTE, 1989).

Timing: No data found.

Particularity of life-cycle: ↑: Triphenism. Infective larvae longer and slender than other larvae, sensitive to heat and vibration in the environment, highly active and not requiring films of moisture in which to move, able to somersault and propel themselves from the substrata, which allows them to search actively for a suitable host.

Advanced seclusion: !: In superficially invading the host, infective third-stage larvae find a protected environment which also provides a richer food. This allows them to store lipid droplets in their intestinal cells and to grow somewhat (SCHULTE, 1989), to survive conditions of food deprivation and use for subsequent development. In addition, infected rodents act as an agent of dispersal in situations where places for free-living development may be sparse.

Strongyloides spp. (Fig. 3B)

Host(s): *Definitive*: tetrapods. *Intermediate*: none. *Paratenic*: none. *Sporadic*: none.

Description of life-cycle: Parthenogenetic parasitic females produce only genotypically female eggs by mitotic (apomictic) parthenogenesis (TRIANTAPHYLLOU & MONCOL, 1977; MONCOL & TRIANTAPHYLLOU, 1978; GEORGI, 1982). Eggs embryonate in the intestine, hatch and may evolve in three different ways: (i) *autoinfection*, i.e. development of Lr1 in the gut to the infective stages (Ls3) (FAUST & DE GROAT, 1940; FAUST, 1949), which invade the gut mucosa or perianal skin, then migrate (entero-vascular internal cycle) to the lung; in lungs larvae leave blood vessels, enter air spaces, move up to the trachea where they are swallowed (LOOSS, 1905; FÜLLEBORN & SCHILLING-TORGAU, 1911; FÜLLEBORN, 1914), and come back to the intestine where they grow into parthenogenetic females; (ii) *homogonic pathway*, Lr1 are eliminated with faeces into the external environment and develop into infective female larvae (Ls3); (iii) *heterogonic pathway*, Lr1 are eli-

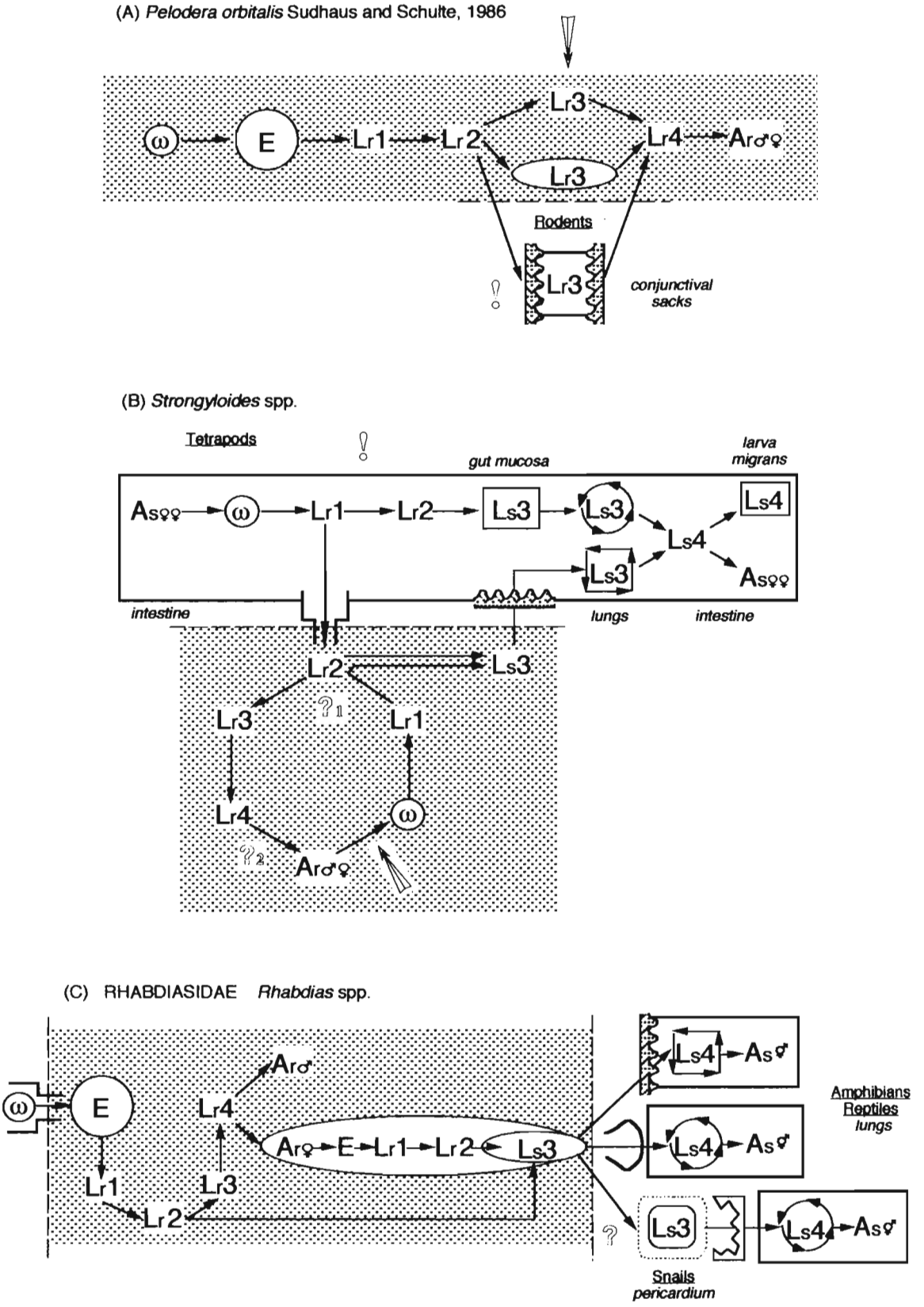

Fig. 3.– Example life-cycles in the Rhabditia.

minated with faeces into the external environment and grow and moult four times to produce a single free-living generation giving birth to both males and females; larvae arising from eggs of heterogonic females develop into infective female larvae only (Ls3). Ls3 invade the definitive host by penetration through the skin and then to blood or lymphatic vessels from which they are carried to lungs and complete their cycle in the same way as autoinfective larvae. In an immunocompromised host, infection can lead to fatal disseminating strongyloidiasis (larva migrans) (PURTILO *et al.*, 1974; SCOWDEN *et al.*, 1978; SCHAD *et al.*, 1984; GENTA, 1986). Transplacental transmission has been proposed to account for early infection in young newborn animals (ENIGK, 1952; STONE, 1964). Infective larvae of some species can remain in the superficial tissues, pass into the milk and infest the suckling young (MONCOL & BATTE, 1966; SUPPLERER & PFEIFFER, 1967; KATZ, 1969; STEWART *et al.*, 1969; LYONS *et al.*, 1969, 1973; MONCOL & GRICE, 1974; ZAMIRDEN & WILSON, 1974; MONCOL, 1975; WILSON *et al.*, 1976; NOLAN & KATZ, 1981).

Timing: Prepatent period variable among species and hosts: 3 days in *S. ransomi* according to FRICKERS (1953); 8 to 17 days in *S. stercoralis*; 9 days in *S. papillosus*, according to TURNER (1955); 7 to 10 days in *S. agoutii* according to GRIFFITH (1940). Peak egg production generally occurs around the first 3-8 weeks of the patent period and may persist for months.

Particularity of life-cycle: ↑: Heterogonic females produce eggs by meiotic parthenogenesis after male sperm has penetrated the egg. Penetration of sperm is necessary for initiation of egg development, although sperm and egg pronuclei generally do not fuse; diploidy recovered through recombination of nuclei of the second division of egg.

Advanced seclusion: !: Autoinfection allows the parasite to totally accomplish its cycle in a single host individual, which reduces exposure of eggs and larval stages to outside contingencies.

Dubious or unknown sequences: ?1: Factors which determine whether larvae will develop into free-living rhabditiform females or into strongyliform infective larvae are unknown. ?2: Stimuli acting on whether larvae will develop into males or females during heterogonic development are unknown as well.

Rhabdias spp. (Fig. 3C)

Host(s): *Definitive*: amphibians, reptiles. *Intermediate*: none. *Paratenic*: snails possibly. *Sporadic*: none.

Range: Cosmopolitan.

Description of life-cycle: Eggs deposited in lungs of definitive hosts pass up the respiratory system, are swallowed and pass into faeces of the host. Lr1 hatch from eggs in the external environment and may evolve in two different ways: (i) in *homogonic development* Lr1 develop directly into Lr2 and then Ls3 infective larvae; (ii) in *heterogonic development* free-living larval stages result in reproducing rhabditoid adults. Females produce a few large eggs which develop to ensheathed infective Ls3. The female then becomes senescent and the larvae consume her internal organs (matricidal endotoky) and break free of maternal cuticle. Infective larvae may infect the host by skin penetration, or passive oral absorption (CHU, 1936; CHABAUD *et al.*, 1961; BAKER, 1979), or may be ingested when definitive host eat snail paratenic hosts (trophic relationship). When penetrating skin, larvae lose their sheaths, then migrate to the body cavity (BAKER, 1979) where they grow to L4 and to the subadult stage of the parasitic hermaphrodite, which invade the lung.

Timing: In *R. americanus* of toads and *R. ranae* of frogs, mature adults appeared in culture within 27 hours at room temperature (22° C) (BAKER, 1979). When hosts were infested experimentally, subadults first appeared in lungs after 18 days and gravid females after 30 days (*R. americanus*), 9 days (*R. ranae*) (BAKER, 1979). Field studies of *R. ranae* in *Rana sylvatica* showed that one, and possibly two genrations of the parasite occurred annually, that transmission occurred mainly during spring and autumn, that frogs acquired infection the same summer as they transformed and that the parasite survived winter as adults in the frogs (BAKER, 1979).

Particularity of life-cycle: ↑: Both homogonic and heterogonic development may occur in a single species, although one may predominate (CHU, 1936; WILLIAMS, 1960).

Advanced seclusion: !: Development inside the cuticle of heterogonic females and matricidal endotoky suppress exposure of eggs and larval stages to the outside contingencies.

Dubious or unknown sequences: ?: The possible role of snails as paratenic hosts is not fully elucidated. However infective larvae of both *Rhabdias bufonis* and *R. americanus* have been said to invade and survive in tissues of snails (FÜLLEBORN, 1928; BAKER, 1979). CHU (1936) found that infective larvae of *R. fuscovenosa*, a parasite of snakes, failed to penetrate snails, tadpoles, frogs and toads.

DISCUSSION

JEANNEL (1942) and later CHABAUD (1965) attested that from the ancestral pattern of free-living nematodes, in which the entire development occurs in the external environment with a saprophytic free life, to the most sophisticated cycles observed in certain parasites, a general tendency can be recognized. After RACOVITZA (1929), they named it as the concept of «*seclusion*» which involves any adaptation used by the free-living stages of a parasite to elude the roughness, irregularities

and hazards of the external environment. This suggests that improving seclusion can be used as an Ariadne's clew for understanding evolution of parasite life-cycles.

For instance, Fig. 2A sketches a simple and monoxeneous life-cycle in which all parasite stages are living in the intestines without true endoparasitism; however, L2, L3 or L4 larvae may, optionally and momentary, live in the mucosa. Cycle 2B is very similar but endoparasitism of L2 and L3 is necessary, which can be interpreted an advance (!): L4 have an entero-vascular internal cycle and become endoparasitic in the liver, where adult nematodes also are living and producing eggs. Eggs being encysted in the liver cannot be eliminated outside and the cycle runs the risk of being broken off. This necessitates the appearance of a particularity of the life-cycle, a paratenic carnivorous host which, after eating the definitive host, releases the eggs through its own digestive tract. However, occasional cannibalism (a mouse eating a dead congener) sometimes makes the presence of a paratenic host unnecessary. The cycle of *Trichinella spiralis* looks similar to cycle 2B, but the female nematode directly produces L1 larvae (! innovation) and active absorption (with trophic relationships) becomes the rule. Cycles 2A, 2B and 2C show how seclusion may evolve: (i) endoparasitism probably improves seclusion for larvae and adults (READ & SKORPING, 1995), but also results in egg encystation; (ii) utilisation of trophic relationships obviates this difficulty, either by the introduction of a paratenic host, or by occasional use of cannibalism. The cycle of *T. spiralis*: (iii) generalizes cannibalism, making trophic relationships the rule for absorption of infesting stages, which can be considered advantageous because this substitutes the active search for prey by predators to fortuitous passive absorption; (iv) directly produces infesting stages (L1) which allows suppression of egg development in the external environment. This results in a very short and quite non-existing free living stage and an almost perfect seclusion of both larval and adult parasite stages.

Cycles 2D and 2E illustrate another pattern of evolution in which early larval stages improve seclusion by annexation of a paratenic host with the triple benefit of: (i) better protection, (ii) an accumulation of infective stages, (iii) paratenic hosts being devoured by definitive hosts, substitution of the active search for prey by predators to fortuitous passive absorption. In *D. renale* (Fig. 2E) points (ii) and (iii) are reinforced by the presence of a third host. *D. renale* shares with *C. hepaticum* an entero-vascular internal cycle leading parasite subadult stages to accumulate in the liver; but here, as suggested by MACE (1976), the proximity of the right lobe of the liver to the stomach and the right kidney of minks allows the parasite to ultimately settle in an organ connected to the outside by the urinary ducts, through which eggs can be eliminated.

Free living Rhabditida are considered as the possible ancestors of most of the Secernentean parasites. In this group free cycles can be observed with an optional parasitism of larval stage three in the conjunctival sacks or hair follicles of a mammal (Fig. 3A). This could explain why in a large part of the parasitic cycles, the third stages remain able to infest their host through skin. In some other Rhabditida a free-living cycle with outside sexual reproduction alternates with a parasitic one (Fig. 3B and C). All together these cycles illustrate how nematodes can evolve from a free-living life-cycle to a more or less obligatory parasitic life-cycle.

CONCLUSION

The different ways used by the parasites in their pursuit of new hosts and investigation of new areas and improvement of seclusion gave birth to different sketches which, generally, display a high similarity when considering closely related parasite taxa. In other words, any parasitic cycle partly includes attributes shared with several other more or less closely related taxa, and partly includes specific components which, occasionally, are shared with less numerous taxa. When such a component can be interpreted as an innovation, a great similarity becomes visible with the so-called «*share derived characters*» applied as evidences of close phylogenetic relationship for cladistic analysis. This presumably signifies that a systematic, and perhaps a phylogeny of life-cycle patterns in nematodes could be considered. The present work is a first approach and we now intend to improve our coding system so as to be able to represent every case of parasitic nematode cycles. This will allow us to publish a general atlas of these cycles and to go further in our investigations by using a comparison of the biological features of parasites.

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