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Epizootiology: [Chapter 9 in *Biology of the Acanthocephala*]

Brent B. Nickol
University of Nebraska - Lincoln, bnickol1@unl.edu

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9.1 Introduction

In practice, epizootiology deals with how parasites spread through host populations, how rapidly the spread occurs and whether or not epizootics result. Prevalence, incidence, factors that permit establishment of infection, host response to infection, parasite fecundity and methods of transfer are, therefore, aspects of epizootiology. Indeed, most aspects of a parasite could be related in some way to epizootiology, but many of these topics are best considered in other contexts. General patterns of transmission, adaptations that facilitate transmission, establishment of infection and occurrence of epizootics are discussed in this chapter.

When life cycles are unknown, little progress can be made in understanding the epizootiological aspects of any group of parasites. At the time Meyer's monograph was completed (1933), intermediate hosts were known for only 17 species of Acanthocephala, and existing descriptions are not sufficient to permit identification of two of those. Laboratory infections of intermediate hosts had apparently been produced for only two species. Study at that time was primarily devoted to species descriptions, host and geographical distribution, structure and ontogeny. Little or nothing was known about adaptations that promote transmission and the concept of paratenic hosts was unclear.

In spite of the paucity of information, Meyer (1932) summarized pathways of transmission among principal groups of hosts, visualized the relationships among life cycle patterns for the major groups of Acanthocephala, and devised models for the hypothetical origin of terrestrial life cycles from aquatic ones. Nevertheless, most of our knowledge regarding epizootiology has been recently acquired.

9.2 Transmission

9.2.1 *General patterns*

Hosts and developmental stages. All organisms must disperse from sites of propagation to other microhabitats if overcrowding is to be avoided and ranges extended. Parasites must disperse not only in space but also from one host to another. Among the acanthocephalans, only eggs exist outside of a host, free in the environment, and transmission from one definitive host to another requires that eggs be ingested by invertebrate animals appropriate to serve as intermediate hosts and that the final ontogenetic stages achieved there find their way to vertebrate animals where maturation can occur.

Acanthocephalan species for which life cycles have been confirmed by laboratory infections require vertebrates for definitive hosts and arthropods as intermediate hosts. Occurrences of adults in carnivorous invertebrates, such as *Neorhadinorhynchus atlanticus* in squid (Gaevskaya, 1977; Naidenova & Zuev, 1978; Gaevskaya & Nigmatullin, 1981), probably represent transitory infections acquired by transfer of adults from prey. There is no laboratory evidence that adulthood is reached in any invertebrate.

Acanthocephalans parasitic in terrestrial definitive hosts usually have insects, frequently species of Coleoptera or Orthoptera, for intermediate hosts. Microcrustaceans, usually species of Amphipoda, Copepoda, Isopoda or Ostracoda, are generally intermediate hosts for those parasitic in aquatic definitive hosts. In the life cycle of some species, another host occurs between the arthropod intermediate and vertebrate definitive hosts. In such hosts, forms parasitic in intermediate hosts penetrate the intestinal wall and localize in mesenteries or visceral organs where maturity does not occur. Although such intercalated hosts may be required to complete transfer of acanthocephalans from intermediate hosts to the trophic level at which potential definitive hosts feed, there is no evidence that they are essential for achievement of infectivity to the final host. The term 'paratenic host', proposed by Baer (1951) and elaborated by Beaver (1969), has attained wide usage for such animals in which ontogeny does not proceed.

No species of acanthocephalan has been demonstrated to require more than the arthropod intermediate host in order to develop infectivity to vertebrates in which maturity can occur. *Neoechinorhynchus emydis*, which matures in turtles, might be an exception. Whitlock (1939) discovered an unidentified species of *Neoechinorhynchus* in snails from Michigan and Lincicome & Whitt (1947) found *N. emydis* in snails from Kentucky. Hopp (1954) demonstrated that *N. emydis* from *Campeloma rufum* was infective to turtles, but he was unable to infect these snails with eggs of *N. emydis*.

Ostracods, however, were readily infected. In preliminary trials, two turtles fed *N. emydis* from laboratory-infected ostracods did not harbor acanthocephalans at necropsy (Hopp, 1954). Ward (1940*b*) concluded that bluegill sunfish, *Lepomis pallidus* (=*L. macrochirus*), were true second intermediate hosts for *N. cylindratus*, but forms from ostracods are now known to reach maturity directly in several species of the Centrarchidae.

Acanthocephalan ovaries fragment early in life and ova are produced in the resulting masses of cells (§7.5.3). Fertilization is internal and within the female a series of membranes develops around the zygote. Upon discharge from the female, cleavage has produced a larva surrounded by a series of membranes and a shell. Strictly, this stage should, perhaps, be called a shelled embryo. However, for convenience, the term 'egg' is usually applied. The term acanthor designates the larval stage that emerges from the egg upon its ingestion by an arthropod (Van Cleave, 1937). After penetration of the wall of the alimentary canal, the acanthor undergoes a series of ontogenetic stages in the body cavity of its intermediate host. Van Cleave (1937) proposed the term acanthella for each in this series of stages. Misuse of the term prompted him later to reaffirm and clarify this term (Van Cleave, 1947). The final ontogenetic stage in intermediate hosts, which is infective to potential definitive hosts, was termed the juvenile (Van Cleave, 1937, 1947) until Chandler (1949) introduced the term cystacanth. As originally proposed, cystacanth designated the final, infective stage found in invertebrates and juvenile was retained to designate re-encysted forms in paratenic hosts. Cystacanth has achieved general usage as a name for the stage infective to a final host regardless of whether it is found in the arthropod intermediate host (Van Cleave, 1953) or in a vertebrate paratenic host (Van Cleave, 1953). Some objection can be made to use of the term for juveniles in paratenic hosts because not all such stages actually 'encyst'. However, argument can be made for the consistent use of cystacanth to designate a stage of ontogeny, i.e. infective to a potential definitive host, regardless of whether the form is actually encysted.

Transmission from intermediate host directly to definitive host. Many, if not most, acanthocephalans utilize only an intermediate host and a definitive host in their life cycles. Eggs of some species, *Acanthocephalus dirus*, *Polymorphus marilis* and *P. minutus*, might not always be shed individually from females and may leave the definitive host only in the body of passed females, to be released upon deterioration of the adult (Muzzall & Rabalais, 1975*a*; Denny, 1968; Nicholas & Hynes, 1958, respectively). However, eggs of most species are passed free in the feces of the definitive host and are ingested by arthropods in which larval development occurs.

Cystacanths developed in these intermediate hosts attain adulthood in definitive hosts upon ingestion of the intermediate host. In these cases, intermediate hosts are fed upon directly by vertebrates that will become definitive hosts and the path of transmission is clear. Many laboratory studies have confirmed such life cycles for a variety of species.

Paratenic hosts. Cystacanths have long been known to occur in mesenteries and visceral organs of vertebrates, usually poikilotherms, but an understanding of the relationships of these hosts to intermediate and definitive hosts has been slow in developing. Meyer (1932) listed vertebrates harboring cystacanths together with invertebrates as '*Zwischenwirte*' or intermediate hosts.

The suggestion by Van Cleave (1920*a*) that larvae of some acanthocephalans locate extraintestinally in vertebrates when ingested before being fully developed is generally accepted to explain the occurrence of adults of some species in the intestine and cystacanths of the same species in the viscera of conspecific hosts. Late acanthellae of *Leptorhynchoides thecatus* cannot establish in fishes. Those that have developed in amphipods for less than 26 days are unable to infect rock bass, *Ambloplites rupestris*. If development has been from 26 to 29 days in amphipods, the cystacanths localize extraintestinally when fed to rock bass. When at least 30 days of development has occurred before ingestion by rock bass, the worms attach to the intestine and mature (DeGiusti, 1949*a*). This suggests that there is a period in development during which some acanthocephalans are able to survive in vertebrates but are unable to maintain themselves in the intestine and mature. Such adaptations would clearly increase survivorship for those species that mature in predators by giving larvae that were ingested before being completely developed a second chance to reach a definitive host.

Other species of Acanthocephala occur viscerally in certain species of vertebrates in which they are not known to occur intestinally, regardless of the age at which ingestion occurs. Perhaps Mingazzini (1896) was the first to illustrate the epizootiological role of this kind of paratenic host when he produced laboratory infections in falcons, *Falco tinnunculus*, by feeding them cystacanths of *Centrorhynchus aluconis* and *C. buteonis* taken extraintestinally from *Zamenis gemonensis* (Reptilia: Colubridae). Use of paratenic hosts to bridge trophic levels between predatory vertebrates and arthropods is now considered to be an important adaptation that enables acanthocephalans to utilize as definitive hosts groups of animals to which transmission would otherwise be unlikely.

Predatory fishes frequently acquire acanthocephalans from smaller

paratenic hosts, usually other fishes, that constitute prey. Hamann (1891*b*) found cystacanths of *Pomphorhynchus laevis* in the viscera of six small fishes and noted that these were frequently prey for larger ones. Riquier (1909) demonstrated that cystacanths of *P. laevis* are indeed, infective to carnivorous fish (*Esox lucius*), but paratenic hosts are not required for development. Cystacanths from intermediate hosts, *Gammarus pulex*, attain adulthood when fed directly to flounder (Engelbrecht, 1957) or goldfish (Kennedy, 1972). Other species of *Pomphorhynchus* also use paratenic hosts. *Pomphorhynchus rocci* occurs in the intestine of several piscine species, including *Morone saxatilus*, striped bass, and encysted in the viscera of striped bass (Paperna & Zwerner, 1976). *Pomphorhynchus bulbocolli* occurs in the mesenteries of several species of fish (Ward, 1940*a*). *Leptorhynchoides thecatus* and *Neoechinorhynchus cylindratus* are other species that occur as adults in carnivorous fishes and commonly use smaller fishes as paratenic hosts. At least two species of *Pallisentis*, *P. basiri* and *P. nagpurensis*, occur viscerally in coarse fishes that are often eaten by larger fishes (Hasan & Qasim, 1960; George & Nadakal, 1973, respectively). Species of the genus *Serrasentis* occur as adults in marine fishes and use various species of other marine fishes as paratenic hosts (Van Cleave, 1924).

Piscivorous birds frequently acquire acanthocephalans of the genera *Arhythmorhynchus*, *Corynosoma*, *Hexaglandula* and *Southwellina* from poikilothermic vertebrate hosts. *Arhythmorhynchus frassoni* occurs in the viscera of Brazilian fishes (Travassos, 1926; Golvan, 1956*a*) and *A. uncinatus* in mesenteries of sheepshead, *Archosargus probatocephalus*, from the coast of Florida (Bullock, 1960). *Corynosoma clavatum* occurs as adults in several species of cormorants, *Phalacrocorax*, in the Southern Hemisphere (Edmonds, 1957*a*) and as cystacanths in mesenteries of *Platycephalus fuscus*, flathead fish (Johnston & Edmonds, 1952). Cormorants, herons and kingfishers in Brazil are definitive hosts for *Hexaglandula mutabilis*, cystacanths of which occur in marine fishes of several genera (Travassos, 1926). *Southwellina hispida* has a broad geographical distribution in *Nycticorax nycticorax*, black-crowned night heron (Schmidt, 1973*a*), and occurs in mesenteries of fishes, frogs and snakes in Japan (Van Cleave, 1925; Yamaguti, 1935, 1939), and in the mesenteries of fishes in Texas (Chandler, 1935; Bullock, 1957*a*). Species of the genus *Andracantha* parasitize a variety of piscivorous birds (Schmidt, 1975), including the American bald eagle (Nickol & Kocan, 1982) and, although no life cycle is known for any species of this genus, it is likely that some, if not all, use fishes as paratenic hosts.

Amphibians and reptiles also serve as paratenic hosts for some acantho-

cephalan species that mature in flesh-eating birds. Species of *Centrorhynchus* and the related *Sphaerirostris* from around the world, are well known as cystacanths in frogs, lizards and snakes. Adults occur in raptors and other kinds of carnivorous birds. Golvan (1956*b*) and Schmidt & Kuntz (1969) listed many of the definitive and paratenic hosts for species of these genera. Many species of *Oligacanthorhynchus* occur as adults in birds of prey, and the literature abounds with worldwide reports of cystacanths in the viscera and mesenteries of reptiles, usually snakes.

Cystacanths of some species of *Porrorchis* that occur as adults in coucals (*Centropus*), owls (*Bulbo* and *Tyto*), and kites (*Milvus*) in Australia, India, the Philippines and Taiwan are found in the mesenteries of amphibians and reptiles. *Porrorchis hylae* has long been known from the viscera of Australian frogs (Johnston, 1914). Southwell & MacFie (1925) described the adult from *Centropus phasianus*, collected in northern Australia, as *Echinorhynchus bulbocaudatus*, and specimens from *C. viridis*, red-winged coucal, collected in the Philippines were later named *E. centropusi* by Tubangui (1933). *Echinorhynchus bulbocaudatus* and *E. centropusi* are regarded (Edmonds, 1957*b*; Schmidt & Kuntz, 1967*b*) as synonyms of *P. hylae*. Coucals are known to feed upon the species of frogs in which cystacanths of *P. hylae* occur (M'ackness, 1977). *Porrorchis hylae* occurs also in India, but snakes, rather than frogs, are paratenic hosts (Gupta & Jain, 1975). Schmidt & Kuntz (1967*b*) found many species of flesh-eating birds to be definitive hosts in Taiwan where snakes, lizards and frogs are paratenic hosts. *Porrorchis indicus* from India, *P. leibyi* from Taiwan and Palawan, and *P. oti* from Japan are other species of the genus that occur as adults in flesh-eating birds and are known to occur in the viscera of snakes, lizards and frogs (Das, 1957*b*; Schmidt & Kuntz, 1967*b*; Yamaguti, 1939, respectively). The presence of cystacanths of another member of the Porrorchinae, *Lueheia lueheia*, in mesenteries of amphibians, reptiles and even birds of Brazil (Travassos, 1926) is difficult to assess. Adults are only known to occur in insectivorous birds of the families Formicariidae and Furnariidae. Similarly, cystacanths of *L. inscripta* occur in the body cavity of lizards, *Anolis cristatellus*, in Puerto Rico (Acholonu, 1976), but adults occur in passerine birds. Perhaps a yet to be discovered predator is the usual definitive host for these species or perhaps the cystacanths occur in these paratenic hosts but have no epizootiological significance.

Aquatic mammals, especially seals and whales, are frequently definitive hosts for acanthocephalans acquired from fishes. Species of *Bolbosoma* and *Corynosoma*, cosmopolitan parasites of cetaceans and pinnipeds, have been reported many times from a seemingly endless number of piscine paratenic hosts. In spite of the fact that host specificity for fishes is

apparently low, the prevalence and intensity of the acanthocephalans seem to vary according to the species of fish. Some acanthocephalan species are more abundant in certain fishes and others more abundant in different species of fish. This is demonstrated by the fact that seasonal variation in the structure of *Corynosoma semerme* and *C. strumosum* populations in ringed seals, *Pusa hispida*, from the Bothnian Bay of the Baltic Sea reflects the migratory habits of herrings. Among fishes that are paratenic hosts in the Bothnian Bay, *C. strumosum* is more predominant than *C. semerme* only in *Clupea harengus*, Baltic herring, and *Lota lota*, burbot (Helle & Valtonen, 1980), but Helle & Valtonen (1981) cited references to indicate that the seals do not feed on burbot. In all seasons *C. semerme* is more predominant in seals than is *C. strumosum*, but the difference, which is great in the spring, is much less in the autumn when herrings are available to the seals (Helle & Valtonen, 1981). Similar relations between paratenic hosts and acanthocephalan population structures in aquatic mammals are likely to be evident when parasite distribution within piscine paratenic hosts is analyzed with the same care at other localities.

Acanthocephalans that occur as adults in carnivorous mammals and are known to use paratenic hosts belong primarily to the genera *Macracanthorhynchus* and *Oncicola*. Adults of *M. catulinus* occur in many species of carnivores throughout the eastern European and Asian portion of the USSR, and cystacanths are found in smaller mammals. Petrochenko (1958) listed definitive and paratenic hosts to which Barus, Kullmann & Tenora (1970) added rodents as paratenic hosts in Afghanistan. *Macracanthorhynchus catulinus* also uses poikilothermic vertebrates as paratenic hosts: *Varanus benghalensis* and *Uromastix hardwicki* (Sauria) in Afghanistan (Barus & Tenora, 1976); frogs, snakes and lizards in Azerbaidzhan (Farzaliev & Petrochenko, 1980); snakes, *Vipera lebetina*, in central Asia (Markov, Zinyakova & Lutta, 1967); and *Naja oxiana* in Turkmenistan and Tadzhikistan (Markov, Bogdanov, Makeev & Khutoryanski, 1968). Tenebrionid beetles serve as intermediate hosts (Rizhikov & Dizer, 1954) for *M. catulinus*.

In North America, *Macracanthorhynchus ingens* parasitizes carnivores, primarily *Procyon lotor*, raccoon (Chandler & Melvin, 1951), and cystacanths occur in a variety of frogs and snakes (Moore, 1946*b*; Elkins & Nickol, 1983). Moore (1946*b*) demonstrated that eggs fed to beetles of the genera *Phyllophaga* and *Ligyrus* developed into cystacanths; however, only *Narceus americana*, a species of millipede (Crites, 1964), and *Parcoblatta pensylvanica*, a species of woodroach (Elkins & Nickol, 1983), are known to serve as intermediate hosts in nature.

Several pathways are apparently feasible for transmission of *Macra-*

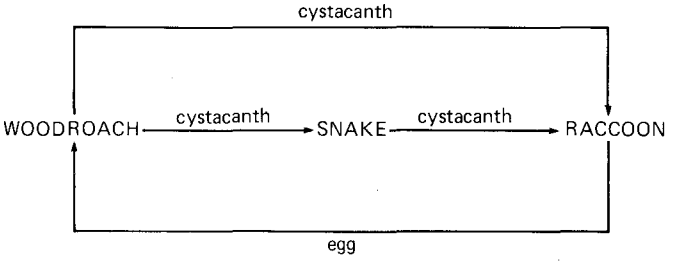
canthorhynchus ingens to raccoons (Fig. 9.1). Cystacanths from woodroaches develop into adulthood when fed to raccoons or penetrate the intestine and re-encyst in the viscera when fed to snakes. Cystacanths from snakes develop into adults when fed to raccoons (Elkins & Nickol, 1983). Additionally, it is probable that frogs may be intercalated as paratenic hosts between intermediate hosts and reptilian paratenic hosts or between intermediate hosts and raccoon definitive hosts.

Adults of species of *Oncicola* also parasitize carnivorous mammals, primarily the Canidae and Felidae. The role of paratenic hosts in the epizootiology of these species is a little less clear because the infectivity of encysted forms has not been verified by laboratory infections. Except for *O. spirula*, a parasite of primates, no intermediate host is known for species of this genus, but cystacanths occur in several avian and mammalian species.

In southwestern North America, *Oncicola canis* occurs in the viscera of *Dasypus novemcinctus*, armadillo (Van Cleave, 1921; Chandler, 1946*a*), beneath the epithelial lining of the esophagus of turkey poults (Price, in Christie, 1929), and in the connective tissue and on the outer surface of the esophagus and crop of *Colinus virginianus*, bob-white quail (Cram, 1931). Cystacanths of *O. canis* from the armadillo are frequently calcified (Chandler, 1946*a*), suggesting that armadillos may not be significant paratenic hosts. *Oncicola oncicola* occurs in the connective tissue and musculature of armadillos (*Tatus* sp.) in Brazil (Travassos, 1917) and subcutaneously and in the musculature of domestic chickens in Costa Rica (Zeledón & Arroyo, 1960).

Australian and Asian species of *Oncicola* also use birds for paratenic hosts. *Oncicola pomatostomi*, which occurs intestinally in canine and feline definitive hosts in Australia, Borneo, Malaysia and the Philippines, has been reported from under the skin of 19 species of birds (Schmidt, 1983).

Fig. 9.1. Schematic diagram of pathways of transmission for *Macracanthorhynchus ingens* demonstrated feasible by laboratory infections and field studies. (From data in Elkins & Nickol, 1983.)



An unidentified species of *Oncicola* has also been reported (Padmavathi, 1967) from the musculature of gallinaceous birds in Madras.

It is clear that paratenic hosts play an important role in transmission of many acanthocephalans, but frequently they lead to termination of the life cycle before reproduction can occur. More or less frequently, encysted forms are found in viscera of animals from which transmission is unlikely or impossible. This seems to be the case for *Oncicola schacheri*. Schmidt (1972*c*) reported that adults of this species occur in Lebanese fox and cystacanths in the mesenteries of badgers, *Meles meles*, but completion of the life cycle by use of badgers as paratenic hosts seems improbable.

At two locations in the USA, Erie County, New York (Nickol & Oetinger, 1968) and near Lincoln, Nebraska, cystacanths of *Plagiorhynchus cylindraceus* occur commonly in mesenteries of shrews. Adults parasitize passerine birds, usually robins or starlings, and although robins are known to kill shrews (Penny & Knapton, 1977) and feed them to their nestlings (Weeden & Weeden, 1973), the epizootiological significance is probably negligible. *Mediorhynchus grandis* has also been reported from the mesenteries of a shrew (Collins, 1971), but because adults occur in non-predatory birds, usually icterids, transmission from the shrew and subsequent attainment of maturity seem unlikely. Cystacanths in hosts from which transmission leading to maturation probably cannot occur, have been reported for many other acanthocephalan species. Such instances could provide a means by which distribution to new kinds of hosts may occur through continued exposure and adaptation, or they may simply represent retention of a general adaptation that is useful only to certain species. In either event, they reflect the frequency with which acanthocephalans, as a group, successfully incorporate paratenic hosts into their pathways of transmission and thereby achieve distribution to groups of animals that would be inaccessible otherwise.

Postcyclic parasitism. When ingested as adults within their definitive hosts, some acanthocephalans survive and parasitize the predator. This phenomenon is termed postcyclic parasitism and hosts that are parasitized as a result are either eupostcyclic or parapostcyclic hosts, depending on whether the predator is conspecific with the prey (Bozkov, 1976). Little is known regarding postcyclic parasitism by Acanthocephala, but it is possible for individuals of at least five species, *Acanthocephalus ranae*, *Echinorhynchus salmonis*, *Moniliformis moniliformis*, *Neoechinorhynchus cristatus* and *Octospiniferoides chandleri*, to be transferred from definitive host to definitive host in this manner (Bozkov, 1980; Hnath, 1969; Moore, 1946*a*; Uglem & Beck, 1972; DeMont & Corkum, 1982, respectively).

DeMont & Corkum (1982) theorized that postcyclic transmission could explain some of the 'mysteries of acanthocephalan transmission', for example, the occurrence of acanthocephalans without known paratenic hosts in definitive hosts that do not feed on the appropriate intermediate hosts.

If postcyclic transmission of some species occurs more frequently in nature than is generally assumed, hosts in which maturation and reproduction of parasites are not at a rate sufficient to maintain the population or in which enteric survival occurs without maturation may not always exert inhibitory effects on acanthocephalan populations.

9.2.2 *Adaptations that increase probability of transmission*

Most species of parasite have high levels of fecundity and are towards the *r* end of the *r*–*K* continuum (Esch, 1977), and it is often argued that *r*-selection results from the uncertainties of transmission. In spite of this, Croll (1966) suggested that because of the great demands of reproduction the number of eggs produced should be the bare minimum required to overcome the natural toll of transmission and successfully propagate the next generation.

Croll's view was challenged by Jennings & Calow (1975) who contended that high fecundity is a natural consequence of the stable, nutrient-rich environment of adult parasites. They believed that in such an environment energy supplies are not limiting so that accumulation of energy reserves to buffer against competition and possible reductions in food supply or allocation of energy to other *K*-strategies would not be at the expense of egg production. Selection pressure to reduce fecundity to the minimum suggested by Croll would not be present. In such circumstances, parasites could readily produce eggs in excess of the minimum number required to insure successful transmission. Whether or not parasites are under selective pressure to hold egg production to a minimum, it is clear that characteristics of potential hosts are commonly exploited in a manner to facilitate transmission, and elaborate adaptations that help insure larval success have evolved.

Seasonal distribution. Van Cleave (1916) was among the earliest to report that acanthocephalans are not distributed similarly among hosts throughout the year (§7.9.5). He found that *Gracilisentis gracilisentis* occurs in gizzard shad, *Dorosoma cepedianum*, of the Illinois River only from October through April and that *Tanaorhamphus longirostris* is present in the same piscine species only from June through December. Seasonal distribution of acanthocephalans was still largely unstudied by 1932, and Meyer's

monograph (1932, 1933) contains little information regarding seasonal occurrence. The rapidity with which studies on the subject have accumulated is evidenced from a recent review (Chubb, 1982) of seasonal occurrence in freshwater fishes. The seasonal distribution is summarized for 34 species and is discussed in relation to the world's climatic zones.

Obviously periods of seasonal activity, including feeding by potential intermediate and definitive hosts, must coincide long enough for transmission to definitive host, maturation and reinfection of intermediate hosts to occur. Eggs could lie dormant at the end of the cycle with reinfection of the intermediate host population being postponed, but there is little indication that they do so. Petrov (1973), however, reported that eggs of a species of *Polymorphus* retained their infectivity to invertebrates for 90 days in the salt lake Kuspek, Kazakhstan, USSR. Kennedy (1972) found that feeding response was an important factor in controlling the level of *Pomphorhynchus laevis* parasitism in dace, *Leuciscus leuciscus*, and that water temperature influenced this response. Unless negated by another factor, such as temperature-dependent rejection (Kennedy, 1972), prevalence and intensity should clearly be greatest during times when intermediate hosts with infective larvae constitute an appropriate portion of the diet of potential definitive hosts. When this occurs throughout the year, there may be no seasonal periodicity in the occurrence of acanthocephalans.

Echinorhynchus salmonis shows no seasonal periodicity in prevalence, intensity or development and maturation in rainbow smelt, *Osmerus mordax*, from Lake Michigan where the amphipod intermediate host, *Pontoporeia affinis*, is available to fish throughout the year (Amin, 1978*a*, 1981). Likewise, availability of intermediate hosts in all seasons has been postulated to explain the uniform seasonal prevalence of *Acanthocephalus clavula* in the fish of Llyn Tegid (Chubb, 1964), *Echinorhynchus truttae* in brown trout, *Salmo trutta*, from North Wales (Awachie, 1965) and the lack of seasonal periodicity in both prevalence and intensity of *Neoechinorhynchus saginatus* in fallfish, *Semotilus corporalis*, of New Hampshire (Muzzall & Bullock, 1978).

It may not follow, however, that absence of seasonal periodicity implies that rates of recruitment and mortality are constant throughout the year. Hine & Kennedy (1974*a*, *b*) found infected *Gammarus pulex* in the River Avon during each month of the year. In that river *Pomphorhynchus laevis* shows no seasonal cycle in incidence or intensity in fishes (Kennedy, 1974; Rumpus, 1975; Kennedy & Rumpus, 1977). Because high water temperatures in the laboratory reduce the success with which *P. laevis* establishes in *Carassius auratus*, goldfish, Kennedy (1972) believed that

increased feeding by fish in summer balanced a lower rate of parasite establishment. Thus, seasonal differences in rates of recruitment and mortality (from failure to establish in fish) could occur even if parasite occurrence shows no periodicity.

There are instances in which species of invertebrates appropriate as intermediate hosts are consumed by potential definitive hosts throughout the year, but definite seasonal cycles in acanthocephalan distribution occur. Awachie (1965) demonstrated that even though infective larvae of *Echinorhynchus truttae* occurred in *Gammarus pulex* throughout the year and that the amphipods formed an important part of the food of brown trout all year, seasonal differences in densities of infective larvae led to cycles of intensity in fish. Cases such as this suggest that fish of a different species, one for which seasonal cycles are significant, might be more important hosts.

Seasonal cycles in occurrence of acanthocephalans are often linked directly with seasonal changes in the external environment, especially changes in water temperature. Variations in habits of hosts during the year, for instance changes in diet, also cause seasonal differences in prevalence and intensity of infections (Halvorsen, 1972). While obviously true (intermediate hosts must be available and eaten), this view more or less implies that parasite activity remains constant and alterations in host habits result in distribution of parasites. Parasites are frequently not so passive. Acanthocephalans often show adaptations that are directly related to seasonal events in the life histories of definitive and intermediate hosts in a way that helps insure transmission.

Reproductive effort by parasites may be limited to coincide with breeding cycles in host populations. Muzzall (1978) described seasonal cycles of *Fessisentis friedi* in isopods, *Caecidotea communis*, from New Hampshire that suggest peak acanthocephalan egg production occurs as pickerel move into shallow water to spawn. Maximum intensity and egg production of *Pomphorhynchus bulbocolli* in white suckers, *Catostomus commersoni*, have also been related (Muzzall, 1980) to migration and spawning. In Lake Michigan, *Echinorhynchus salmonis* reaches peak maturity in chinook salmon, *Oncorhynchus tshwytscha*, during spawning (Amin, 1978*a*). These seasonal patterns result in peak egg production by the acanthocephalans when fishes are in shallow waters where appropriate invertebrates are most abundant.

A similar potential endocrine relation may exist between the reproductive cycles of acanthocephalans and the maturation of amphibian hosts. In Georgia, aquatic isopods, *Asellus scrupulosus*, are intermediate hosts for *Fessisentis necturorum*. Prevalence and intensity in salamanders,

Ambystoma opacum, increase from January through March while the salamanders are larvae. Parasitism declines in April and May during metamorphosis and the worms are absent from adults (Nickol & Heard, 1973). Adult *A. opacum* are found in water much less frequently than are adults of other species of salamander and do not even enter standing water to breed. Instead, eggs are laid in moist litter that is later inundated. In Louisiana and Illinois, *F. necturorum* and *F. fessus* occur in *Necturus beyeri* and *Siren intermedia*, respectively (Nickol, 1967, 1972). These amphibians, which retain gills and remain aquatic throughout their lives, are parasitized into summer, long after acanthocephalans are gone from *A. opacum*. Avery (1971) found no seasonal difference in levels of *Acanthocephalus anthuris* infection in newts, *Triturus helveticus*, which remain in the water for the entire year in England. Gravid females were present in adults and larvae throughout the year. No seasonal difference was detected in larval or adult *T. vulgaris*, but adults were surveyed only during months in which they were aquatic. If acanthocephalans were lost from these species at metamorphosis, the aquatic existence of adults could have resulted in their reinfection.

Kennedy (1970) concluded that when maturation of helminths from freshwater fishes showed seasonal cycles, peak egg production would almost always occur late in spring or early in summer. Many species of Acanthocephala might conform to this generalization. Prevalence, density and maturity of *Acanthocephalus dirus* in Wisconsin fishes are greatest in the spring. Eighty per cent of the female acanthocephalans contain fully formed eggs in May (Amin, 1975*c*). Walkey (1967) showed this cycle for *Neoechinorhynchus rutili* in England. There was no seasonal difference in prevalence or intensity in three-spined sticklebacks, *Gasterosteus aculeatus*, but the proportion of immature worms plunged by May so that gravid worms were most common during the late spring and early summer and rarest in winter. This finding is consistent with a later report by Tesarcik (1972) who noted that *N. rutili* was recruited into carp populations from late February to early March in Czechoslovakia. Egg production then occurred from March through July (Tesarcik, 1970). Although he did not note periods of maturity, Bibby (1972) reported a seasonal distribution in Wales of *N. rutili* in minnows, *Phoxinus phoxinus*, that fits with this cycle.

There are many other reports, including those by von Möller (1974, 1975) for *Echinorhynchus gadi*, by Muzzall & Rabalais (1975*a*) for *Acanthocephalus dirus*, by Paperna & Zwerner (1976) for *Pomphorhynchus rocci*, and by Jilek (1978) for *Tanaorhamphus longirostris*, of acanthocephalan infections of vertebrates increasing in prevalence and intensity in the spring. Egg production then occurs late in spring and perhaps

throughout the summer. Numbers decline in the fall and reach a minimum during winter. Komarova (1950) found that the prevalence and intensity of *A. lucii* in perch, *Perca fluviatilis*, of the Dneiper River rose sharply in March and April, but that the worms were immature until summer. Towards the end of summer, acanthocephalans decreased in perch until they were absent in the fall. Anderson (1978) described a similar seasonal cycle for *A. lucii* in Norway, although peaks were a little later. In England, the prevalence of *A. lucii* in perch also rises sharply in March. At the same time the percentage of *Asellus aquaticus* and *Gammarus pulex*, intermediate hosts, is at its yearly highest in perch stomachs (Mishra, 1978). *Dentitruncus truttae* has nearly the same seasonal dynamics in Italy. Prevalence of detectable cystacanths in gammarid intermediate hosts is highest from February through March. It falls during the remainder of the year. Prevalence in trout, *Salmo trutta*, begins to rise after April and continues to rise through October, after which it falls (Orecchia, Paggi, Manilla & Rossi, 1978).

Although most studies of seasonal distribution have been concerned with parasites of poikilothermic hosts, there is indication that some of those from homeotherms conform to the general pattern. Hair (1969) found *Plagiorhynchus cylindraceus* more numerous in starlings, *Sturnus vulgaris*, of South Carolina during spring and summer. They were absent from birds during five winter months. *Moniliformis clarki* is most prevalent and produces more eggs in voles, *Microtus ochrogaster* and *M. pennsylvanicus*, in Indiana during the summer than in spring and winter (Fish, 1972). Sadaterashvili (1977) reported egg production and development of *Macracanthorhynchus hirudinaceus* in swine to be slower in winter than in summer.

These studies of seasonal distribution show that annual cycles in vertebrates frequently begin in spring and that most transmission between vertebrates and invertebrates occurs during summer and early fall. Development in intermediate hosts infected late in the season is slow or non-existent until temperatures rise again in the spring. Petrochenko (1958) reported that at 9–12 °C *Polymorphus magnus* developed in *Gammarus lacustris* at only one third the rate exhibited at 18–25 °C. Others (including DeGiusti, 1949*a*; Awachie, 1966; Lackie, 1972; Nickol, 1977; Tokeson & Holmes, 1982) have also observed halted or retarded larval development in invertebrates at low temperatures. During late winter at Cooking Lake, Alberta, less than 3% of the *G. lacustris* contained cystacanths of *P. marilis*. After these amphipods were held in aquaria at temperatures greater than 15 °C, 20–30% of them developed cystacanths (Tokeson & Holmes, 1982).

Ability to resume development is not always related to temperature alone. Tokeson & Holmes (1982) found that the developmental rate of *Polymorphus marilis* in amphipods placed in temperatures favoring development, after being held at temperatures below the developmental threshold, depended upon the time spent at low temperatures. Because development was faster after longer periods below the threshold, they postulated a diapause condition that might prevent development to the cystacanth stage during sporadic intervals of warm weather in early fall.

A common type of cycle, then, consists of spring recruitment by vertebrates after the environment has warmed and potential vertebrate and invertebrate hosts have become active; summer transmission between vertebrates and invertebrates; slow development in invertebrates during the winter; and completion of larval development with rising temperatures in the spring.

Development of parasites geared to the same stimuli that promote increased activity in hosts insures that larvae reach infectivity during times when transmission is most likely. Fully developed, infective larvae probably increase the mortality of hosts even in the absence of predation. Larger forms might make greater metabolic demands of their hosts and increase stress in an already stressful season. Further, alterations of host behavior that may accompany parasite infectivity (Bethel & Holmes, 1974) could result in the intermediate host responding in an unadvantageous manner that could increase stress at a time when transmission is unlikely.

Deviations from the common 'spring recruitment cycle' are often related to deviations in vertebrate–invertebrate relationships and environmental disturbances. Prevalence of *Echinorhynchus salmonis* in yellow perch, *Perca flavescens*, from Ontario rises in the fall after which it declines until the parasite is absent from fish during the summer (Tedla & Fernando, 1970). Watson & Dick (1979) also found that *E. salmonis* was most common in whitefish, *Coregonus clupeaformis*, in late fall in Manitoba when the amphipod intermediate host, *Pontoporeia affinis*, was a prominent whitefish food item.

In instances where invertebrates are rare or absent during winter months, the cycle may be altered also, resulting in recruitment to the definitive host population in the fall. Maturation is then slow with egg production occurring in the spring. Eure (1976) found this to be true for *Neoechinorhynchus cylindratus* in a heated reservoir in South Carolina. Recruitment into largemouth bass, *Micropterus salmoides*, occurred in the fall while ostracod numbers decreased. By November ostracods were scarce and most acanthocephalans were in bass. Egg production was delayed until spring when ostracods were again abundant. Ice cover might

affect seasonal distribution of acanthocephalans of waterfowl similarly. Spencer (1974) found no *Gammarus lacustris* infected with *Polymorphus minutus* in a Colorado lake from December through March. Prevalence in amphipods peaked in July and started down in August. Similarly, Hynes (1955) reported that *G. lacustris* infected with *P. minutus* began die-offs during fall. Apparently, during the winter *P. minutus* is found in mallards or occurs in amphipods as small, undetected acanthors. A similar species, *P. magnus*, is rare or absent in waterfowl during the spring and summer, but becomes common in the fall (Okorokov, 1953; Moskalev, 1976).

Environmental disturbances may not affect host relationships of all parasites in the same manner. Contrary to Eure's (1976) findings, Boxrucker (1979) detected no seasonal difference in prevalence or intensity of *Pomphorhynchus bulbocolli* in a thermally heated sample area in Wisconsin, but found that in a non-heated reference area this species displayed the familiar pattern of increasing prevalence and intensity during the spring followed by declining numbers in the fall. Elevated winter temperatures may have resulted in more rapid parasite development in amphipods and in fish feeding more extensively on them than would occur during winter in non-heated environments.

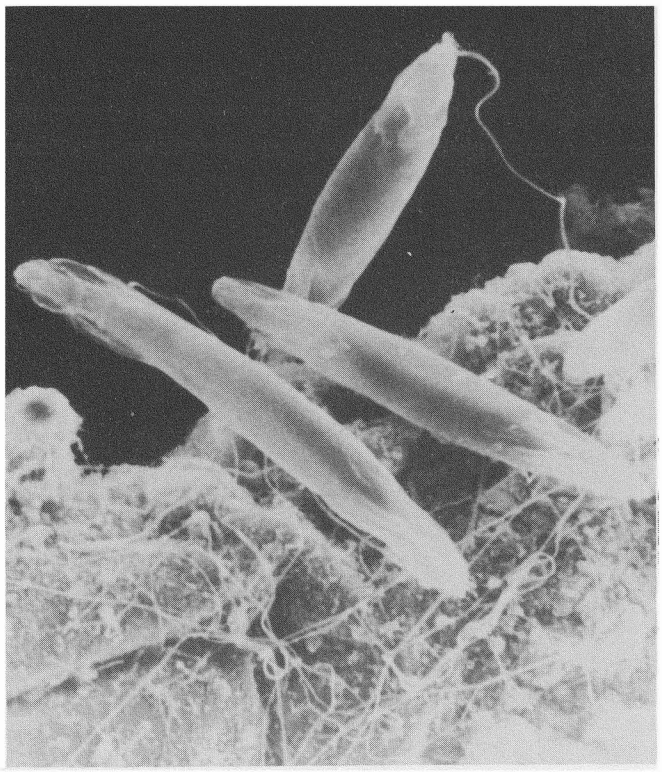
Egg fibrils. There has been considerable attention devoted to the number (see Table 7.6), composition and structure (von Brand, 1940; Monné, 1964; Wright, 1971; Stranack, 1972; Whitfield, 1973), and homologies (West, 1964) of egg envelopes of acanthocephalans. Even early observers frequently illustrated eggs with fibrils contained in the envelopes surrounding acanthors. The illustrations were reproduced without comment by Meyer (1932). Yamaguti (1935) may have been the first to mention fibrils specifically, but he associated them with the outer envelope as did Petrochenko (1953) who described enveloping threads on the surface of *Pseudoacanthocephalus caucasicus* eggs. Monné & Hönig (1954) recognized the fibrils as a separate, distinct envelope, under the outermost covering and first applied the term fibrillar coat. West (1964) incorporated this term in his scheme for naming acanthor envelopes and associated the filamentous nature with aquatic forms.

Monné & Hönig (1954) noted that crushing or treatment with sulphuric acid caused unraveling of the fibrillar coat due to destruction of the outer envelope, but Whitfield (1973) was the first to postulate a function for egg fibrils. He recognized three envelopes in eggs of *Polymorphus minutus*. His envelope II comprises a refractile inner zone, IIb (fertilization membrane in West's scheme), and an outer layer, IIa (fibrillar coat of West), of fibrillar threads. Whitfield then suggested that keratin-destabilizing condi-

tions in the alimentary canal of intermediate hosts could cause lengthening and softening of envelope IIb. Such destabilization could burst envelope I causing release of the fibrillar envelope IIb. The looping threads could serve to slow down passage through the gut giving acanthors better opportunity to complete hatching and to initiate penetration of host tissue. A similar view was adopted by Oetinger & Nickol (1974) in explaining the fibrils of *Acanthocephalus dirus* eggs (Fig. 9.2). They observed, however, that fibrils could be released by the action of bacteria and protozoans on the outer envelope before eggs were ingested.

Uznanski & Nickol (1976) observed that eggs of *Leptorhynchoides thecatus* in fish feces lack outer membranes and possess free fibrils. Scanning electron microscopy revealed the fibrils to be edges of a membranous band wrapped like a bandage around the egg (Fig. 9.3). The band is often torn to produce ribbons of various lengths and numbers. These fibrils entangle in algae and suspend eggs among algal filaments

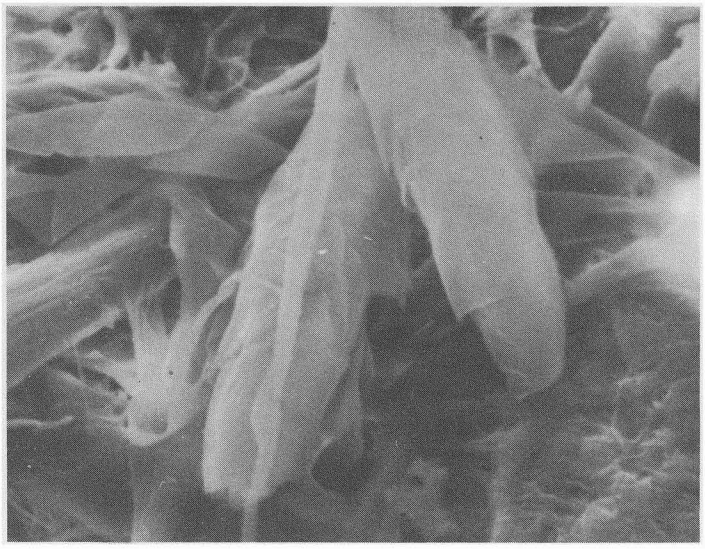
Fig. 9.2. Eggs of *Acanthocephalus dirus* with freed filaments viewed at 500X by scanning electron microscopy. (Micrograph by David F. Oetinger.)



where *Hyalella azteca*, potential intermediate hosts, feed. Feeding experiments resulted in significantly greater prevalences and intensities of infection in amphipods held in containers to which eggs were added after algae (permitting entanglement) than in those held in containers to which eggs were added before the algae (prohibiting entanglement).

Adaptations resulting in egg production at the proper place and time to promote likelihood of successful transmission have been considered. Because eggs are the only free-existing stage in acanthocephalan life cycles, they are of considerable importance in dispersal and transmission. In spite of the fact that eggs are frequently viewed as passive stages disseminated randomly from definitive hosts, it appears that adaptations occur that help to facilitate transmission. Fibrils on egg envelopes represent one such adaptation and there may be others. For example, the outer membrane of *Pallisentis nagpurensis* eggs swells upon contact with water allowing the eggs to float (George & Nadakal, 1973). Planktonic copepods, *Cyclops strennus*, are the intermediate hosts and floating eggs might be more accessible to them. It is clear that fibrils, and perhaps devices for floating, can promote retention of eggs in microhabitats of potential intermediate hosts. Fibrils may also retard passage through alimentary canals of intermediate hosts.

Fig. 9.3. Eggs of *Leptorhynchoides thecatus* with freed fibrillar bands viewed at 610X by scanning electron microscopy. (Reproduced from Uznanski & Nickol, 1976, with permission of the editor of the *Journal of Parasitology*.)



Pigmentation. There are many reports showing that animals that contrast with their backgrounds are more frequently taken by predators. Animals that differ from conspecifics of their population may also be more vulnerable (Holmes & Bethel, 1972). Infective stages of acanthocephalans are known to modify intermediate hosts in ways that may result in increased probability of consumption by potential definitive hosts. These modifications are frequently morphological, involving parasite and host pigmentation, or behavioral.

Although cystacanths of most species are colorless, some of those occurring in aquatic crustaceans are brightly colored. Among the best known and most widely distributed of these colored cystacanths is *Corynosoma constrictum* in central North America. Cystacanths of *C. constrictum* appear through the body wall of *Hyalella azteca* as conspicuous, bright orange spheres. Cystacanths of *Polymorphus contortus*, *P. marilis*, and *P. paradoxus* in *Gammarus lacustris* are also orange (Denny, 1969). Among European species, *Echinorhynchus truttae* and *P. minutus* cystacanths are bright orange (Barrett & Butterworth, 1968) as are those of *Pomphorhynchus laevis* in amphipod intermediate hosts (Kennedy, Broughton & Hine, 1978; Van Maren, 1979*a*, *b*).

Analysis of the carotenoids of *Polymorphus minutus* cystacanths by Barrett & Butterworth (1968) revealed that in spite of the fact that a variety of carotenoids was present in *Gammarus pulex*, only one, esterified astaxanthin, occurred in the cystacanths. They concluded that developing *P. minutus* either selectively absorb this pigment or convert all the others into astaxanthin.

Pigmentation of cystacanths may be independent of that in adults. While all known pigmented cystacanths are shades of orange, adults of several species are red, orange, yellow or brown, and pigmented individuals may occur alongside unpigmented worms of the same species. Ravindranathan & Nadakal (1971) found adults of *Pallisentis nagpurensis* to be orange, red, brown, yellow or colorless. There is apparently no correlation between the type of intermediate host and the carotenoid found in adult parasites (Barrett & Butterworth, 1973).

Although no metabolic role has been demonstrated for pigmentation in acanthocephalans, pigmented cystacanths certainly render infected crustaceans more conspicuous. Such animals readily stand out from uninfected conspecifics and thus may be more likely selected by sight-feeding vertebrates. Likelihood of transmission could be enhanced by making infected invertebrates more obvious or by allowing a predator to single out an individual from the group. Holmes & Bethel (1972) developed a scenario comparing unidirected efforts at prey capture to 'flockshooting' by

hunters. Theoretical approaches to predator–prey relationships suggest advantages in transmission for pigmented cystacanths, but little experimental verification has been attempted.

Alterations of intermediate host pigmentation may also promote acanthocephalan transmission. By interfering with normal pigmentation of intermediate hosts, cystacanths of some species cause an abnormal contrast with backgrounds. Munro (1953) was the first to note a relation beween isopod pigmentation and infection with acanthocephalans. In Scotland, he found more than 90% of the *Asellus aquaticus* to be darker than normal when infected with larval polymorphids. Individuals of *A. aquaticus* infected with *Acanthocephalus anguillae* are also more darkly colored (Balesdent, 1965). In Italy, *A. coxalis* infected with *A. clavula* have been described as more darkly pigmented than uninfected individuals (Fresi, 1967*a*, *b*).

Other species of developing acanthocephalans have the opposite effect on crustacean pigmentation. Hindsbo (1972) found that 94.9% of the adult *Gammarus lacustris* from two ponds in Denmark were of the normal gray-to-brown color, but that the remainder were blue. Dissection showed that all blue amphipods, but less than 4% of the brown ones, were infected with a species of *Polymorphus*, probably *P. minutus*. Hindsbo attributed the abnormal color of infected amphipods to the blue hemolymph showing through unpigmented cuticle. Seidenberg (1973) reported 'dichromatism' of the isopod *Asellus intermedius* in Illinois in which all light-colored isopods were infected with *Acanthocephalus dirus* and most dark individuals were not. He related infection and pigmentation in a manner to suggest that developing acanthocephalans promoted depigmentation. *Lirceus lineatus*, when infected with *A. dirus*, are also non-pigmented (Muzzall & Rabalais, 1975*b*, *c*).

Integumental pigmentation of *Asellus intermedius*, *Lirceus garmani* and *L. lineatus* differs with species, sex, reproductive state and size of the isopod. Comparison of pigmentation between uninfected and infected isopods requires consideration of these factors. Normal pigmentation is altered in each of these species when isopods harbor *Acanthocephalus dirus* cystacanths (Oetinger & Nickol, 1981). Acanthocephalan-infected isopods fail to develop pigmentation comparable to uninfected forms, rather than becoming depigmented (Oetinger & Nickol, 1981). Spectrophotometric study of integumental pigment extracts from *A. dirus*-infected and uninfected *A. intermedius* and methanolic hydrochloric acid extracts from *A. dirus* suggests that competition between developing acanthocephalans and developing isopods for amino acids may cause the pigmentation dystrophy (Oetinger & Nickol, 1982*a*). Only small isopods can be infected

routinely in the laboratory (Oetinger & Nickol, 1982*b*). Occasional acanthocephalan infections in normally pigmented isopods apparently are the result of infrequent infection of older individuals in which pigmentation has already developed (Oetinger & Nickol, 1981, 1982*a*, *b*). Some epigean isopods possess reduced pigmentation when associated with hypogean habitats (Lisowski, 1979), and thus not all isopods with reduced pigmentation are infected. However, it is clear that developing acanthocephalans of some species cause pigmentation dystrophy that renders infected isopods more conspicuous against the dark background of their habitat (Fig. 9.4).

Experimental demonstration that acanthocephalan-induced conspicuousness actually leads to increased vulnerability to predation has been attempted. Feeding experiments in which unpigmented, blue amphipods (*Gammarus lacustris*) infected with *Polymorphus minutus* were offered to ducks along with the normal brown, uninfected forms revealed that the chance of unpigmented individuals being eaten was 2.5 times greater than that for the pigmented forms (Hindsbo, 1972). Similarly, significantly more light-colored isopods (*Asellus intermedius*) infected with *Acanthocephalus dirus* were eaten than uninfected, dark forms when offered to creek chubs, *Semotilus atromaculatus*, in aquaria (Camp & Huizinga, 1979). However,

Fig. 9.4. Uninfected (above) and *Acanthocephalus dirus*-infected (below) *Lirceus lineatus*. (Photograph by David F. Oetinger.)

P. minutus and *A. dirus* evoke behavioral changes (Hindsbo, 1972; Camp & Huizinga, 1979) in their intermediate hosts. The importance of altered pigmentation as opposed to modified behavior in promoting differences in predation is unclear. Bethel & Holmes (1977) attempted to distinguish between the effects in promoting increased vulnerability of amphipods to predation by ducks. They painted oval marks, about the size and color of cystacanths, on carapaces of uninfected amphipods. Upon exposure to mallards, the proportions of marked and unmarked amphipods that were eaten were not significantly different. However, mallards are surface feeders and although cystacanths of *P. paradoxus*, for which mallards are a principle definitive host, are brightly colored, other species with orange cystacanths (*Corynosoma constrictum* and *P. marilis*) occur much more frequently in diving ducks, such as scaup, for which brightly colored prey might be more significant.

Alteration of host behavior. Holmes & Bethel (1972) pointed out that the degree of overlap between the habitats or feeding niches of definitive and intermediate hosts could influence the evolution of transmission mechanisms. They suggested that when the habitats only overlap partially, alterations might occur in responses of intermediate hosts so as to move them into the area of overlap with potential definitive hosts. Modifications in definitive host behavior that would result in release of parasite eggs in the appropriate invertebrate habitat might also be expected.

Little is known of the effects of acanthocephalans on behavior of definitive hosts. In man, however, *Moniliformis moniliformis* may cause nearly unbearable tinnitus (Grassi & Calandruccio, 1888) and violent turning of the head (Al-Rawas, Mirza, Shafig & Al-Kindy, 1977). Rabies-like symptoms associated with *Oncicola canis* in dogs and coyotes (Parker, 1909) led Van Cleave (1921) to speculate that acanthocephalan-induced pain could drive a host 'mad'. Intestinal parasitism is the principle cause of pinniped strandings on Australian beaches where cestodes and acanthocephalans are the main parasites seen (Bergin, 1976). Although acanthocephalan-induced behavioral responses have not been studied critically in definitive hosts, it is conceivable that transmission could be influenced by alterations of host habitat selection and distribution.

On the other hand, modifications of behavior that promote transmission have been demonstrated clearly in intermediate hosts of Acanthocephala. Differences in response to light between infected and uninfected amphipods were studied thoroughly by Holmes & Bethel (1972) and by Bethel & Holmes (1973). Their results showed that uninfected *Gammarus lacustris*

and *Hyalella azteca* are strongly photophobic and negatively phototactic. *G. lacustris* becomes photophilic, without showing a differential response to different light intensities, but negatively phototactic when infected with cystacanths of *Polymorphus marilis* and photophilic and positively phototactic when infected with *P. paradoxus*. *H. azteca* are strongly photophilic and select regions of highest illumination when infected with *Corynosoma constrictum*. Holmes & Bethel demonstrated that differences in responses to light by these amphipods placed cystacanths of *C. constrictum*, *P. marilis* and *P. paradoxus* in different microhabitats from each other and from uninfected amphipods. This microhabitat difference was magnified by differences in evasive response displayed by amphipods. For example, uninfected amphipods found at the surface among leaves and emergent vegetation dove and vanished immediately when surrounding water or vegetation was disturbed. However, upon disturbance, amphipods infected with *P. paradoxus* cystacanths never dove but instead clung persistently to surrounding floating material, even when it was shaken or removed from the water. If material on which to cling was not present, infected amphipods swam to the top of the water and began 'skimming' along the surface rapidly digging or grasping at the air–water interface with their gnathopods. On the other hand, *G. lacustris* harboring cystacanths of *P. marilis* were photophilic, but displayed normal evasive responses (Bethel & Holmes, 1973).

Other species of amphipod infected with different species of Acanthocephala display similar behavioral modifications. *Gammarus lacustris* and *G. fossarum* infected with cystacanths of a species of *Polymorphus* (probably *P. minutus*) and *Pomphorhynchus laevis*, respectively, display a much higher degree of positive phototropism than do uninfected individuals (Hindsbo, 1972; Van Maren, 1979*a*), and *P. laevis*-infected *G. pulex* spend more time in open water and less on the substrate, move more often towards the surface and rest more in surface vegetation than do uninfected *G. pulex* (Kennedy, Broughton & Hine, 1978).

Certain species of isopods and ostracods also display modifications of behavior when infected by acanthocephalans. Non-parasitized *Lirceus lineatus* spend significantly more time under leaves and less time 'wandering' than do those harboring *Acanthocephalus dirus* cystacanths (Muzzall & Rabalais, 1975*c*), and similar behavioral differences occur between *Asellus intermedius* infected with cystacanths of *Acanthocephalus dirus* and uninfected conspecifics (Camp & Huizinga, 1979). *Cypridopsis vidua* and *Physocypria pustulosa* form aggregations milling about at the water surface when infected with cystacanths of *Octospiniferoides chandleri*, while

uninfected ostracods concentrate at the bottom (DeMont & Corkum, 1982).

Evidence that behavioral differences are acanthocephalan-induced comes not only from comparisons of behavior of infected and uninfected invertebrates but also from study of the relation between modified behavior and acanthocephalan development. Not all acanthocephalan species are infective to vertebrates immediately upon reaching the cystacanth stage. Some, such as *Plagiorhynchus cylindraceus* in pillbugs (Schmidt & Olsen, 1964), *Polymorphus trochus* in amphipods (Podesta & Holmes, 1970) and *Mediorhynchus centurorum* in woodroaches (Nickol, 1977), require a period of development as a cystacanth before attaining infectivity. *Polymorphus paradoxus* is not infective to vertebrates during the first week after becoming a cystacanth. The behavioral modifications of infected amphipods are not manifested until 15–20 days after the cystacanth stage is reached. By this time, cystacanths are infective (Bethel & Holmes, 1974).

Experiments to test whether acanthocephalan-induced modifications of intermediate host behavior actually increase vulnerability to predation have been conducted. Bethel & Holmes (1977) showed that amphipods infected with cystacanths of *Polymorphus paradoxus* were significantly more vulnerable to predation by mallards and to accidental ingestion by muskrats, and demonstrated how altered amphipod behavior relates to feeding techniques of these two types of definitive host. Likewise, *Gammarus pulex* harboring *Pomphorhynchus laevis* are eaten by predatory fishes in significantly greater proportions than are uninfected individuals, even when infected amphipods constitute a much smaller proportion of the amphipod population (Kennedy, Broughton & Hine, 1978).

9.3 Establishing infection

9.3.1 *Host recognition and activation*

Acanthocephalans, like most parasites, reach an ontogenetic stage at which no further development occurs until an external stimulus is received. Such stimuli are provided by potential hosts and result in activation and resumption of development. Without these stimuli there is no spread of the parasite.

Hatching of eggs. The egg and cystacanth represent acanthocephalan stages at which development is suspended until external stimuli are provided after ingestion by suitable invertebrates and vertebrates, respectively. However, cystacanths of some species are activated after ingestion by certain vertebrates (which become paratenic hosts), but become parenteric and do not resume development until ingested by a different vertebrate (which

becomes a definitive host). Eggs, free in the environment after being passed from the definitive host, and cystacanths, in the body cavity of intermediate or paratenic hosts, represent the stages of epizootiological significance. These stages must achieve transmission while viable, respond to host stimuli and establish infections.

There are numerous records concerning the duration of infectivity of some eggs for various species, but little information on the effect of aging on infectivity of a population of eggs. Retention of viability of at least some eggs ranges from less than 3 weeks (Hynes & Nicholas, 1957) for *Polymorphus minutus* to more than 3 years (Kates, 1942) for *Macracanthorhynchus hirudinaceus*. Environmental factors are a major influence on retention of viability (Kates, 1942), but freezing does not seem to be harmful and may, in fact, extend viability. *M. hirudinaceus* eggs are viable 140 days after freezing (Kates, 1942), *P. minutus* eggs are infective after 6 weeks at −22 °C (Hynes & Nicholas, 1963) and *Plagiorhynchus cylindraceus* eggs are viable for at least an additional 9 months after 30 h at −80 °C (Nickol & Dappen, 1982). As time passes, however, there appears to be a decrease in the percentage of eggs that is viable. The greatest percentage of *Moniliformis moniliformis* eggs hatches after storage for between 3 and 5 days. After 5 days of storage, the percentage that hatches gradually decreases (Edmonds, 1966).

Eggs are not known to hatch under natural circumstances until ingested by an appropriate invertebrate. Alternate drying and wetting results in 'artificial' hatching among eggs removed from body cavities of some species; however, hatching does not occur when eggs from host feces are subjected to such treatment (Manter, 1928; Moore, 1942). The mechanism of such artificial hatching is apparently different from that of 'natural' hatching and fundamental differences occur between acanthors freed artificially and naturally (Uglem, 1972*a*,*b*; Nickol, 1977). After artificial hatching, no species has been demonstrated to be infective to invertebrates and acanthors of *Neoechinorhynchus cristatus* are known to be non-infective (Uglem, 1972*b*). Further, the process of hatching induced artificially is passive (Manter, 1928) and Uglem (1972*b*) interpreted it as osmotic release of dead material. Acanthors stimulated to hatch by more natural means play an active role in their release. Mechanics of hatching have been detailed by Crook & Grundmann (1964) for *Moniliformis clarki* and by Uglem (1972*b*) for *N. cristatus*. In each of these cases, acanthors used hooks of their aclid organs (Schmidt & Olsen, 1964) to assist in penetration of embryonic membranes. Others (DeGiusti, 1949*a*; Merritt & Pratt, 1964) have also noted active participation by the acanthor during hatching. Hatching induced by alternate drying and rewetting, which could be

prompted by periods of sunshine followed by rain (Manter, 1928), is of no apparent epizootiological significance.

Upon ingestion by appropriate invertebrates, hatching begins within 15 min (Schmidt & Olsen, 1964) for *Plagiorhynchus cylindraceus*, 45 min (DeGiusti, 1949*a*) for *Leptorhynchoides thecatus*, 60 min (Awachie, 1966; Uglem, 1972*b*; Hynes & Nicholas, 1957, respectively) for *Echinorhynchus truttae*, *Neoechinorhynchus cristatus*, and *Polymorphus minutus*, 90 min (Nickol, 1977) for *Mediorhynchus centurorum*, 2 h (Edmonds, 1966) for *Moniliformis moniliformis*, 4 h (Harms, 1965*a*) for *Octospinifer macilentus*, 6 h (Merritt & Pratt, 1964) for *N. rutili*, and 24 h (Moore, 1942) for *Mediorhynchus grandis*. Hatching of *M. grandis* eggs continues for at least 48 h (Moore, 1962).

Attempts to discover stimuli for 'natural hatching' were begun when Hynes & Nicholas (1957) were unable to induce hatching of *Polymorphus minutus* eggs by placing them in macerated portions of suitable *Gammarus* gut. Schmidt & Olsen (1964) achieved hatching of *Plagiorhynchus cylindraceus* eggs by mixing them with crushed digestive glands of an appropriate invertebrate. *In vitro* hatching of *Moniliformis moniliformis* eggs is prompted by solutions of a variety of electrolytes provided that the molarity is between 0.2 and 0.4 and that the pH is greater than 7.5. In 0.25M sodium bicarbonate, hatching occurs over the temperature range of 10 °C to 37 °C. In the presence of carbon dioxide, hatching occurs at pH as low as 6 and the percentage of those hatching is greater over the entire range of suitable pHs (Edmonds, 1966). Lackie (1972*b*) found that when eggs removed from body cavities and stored more than 48 h in 60% sucrose were placed in a solution of 0.3M sodium bicarbonate at room temperature, more than 10% hatched within 24 h. Because these aliquots included a proportion of eggs not fully developed, hatching success under natural conditions should be considerably greater. During hatching, acanthors release chitinase (Edmonds, 1966). Because chitin reportedly occurs in the embryonic membranes of some acanthocephalans (von Brand, 1940; Monné & Hönig, 1954) and because external chitinase has no effect on eggs (Edmonds, 1966), it appears that hatching requires contributions from both the host and parasite. Crompton (1970) compiled information to demonstrate that the physicochemical conditions under which Edmonds (1966) achieved hatching occur in invertebrate alimentary canals, suggesting that the stimulus is physiological. There may be a tendency to view feeding habits and presence or absence of a hatching stimulus in digestive tracts of various invertebrates as the basis for intermediate host specificity. This is not, however, the entire explanation. Eggs of *Neoechinorhynchus cristatus* are ingested and hatch in four species of Ostracoda, but acanthors penetrate

the intestine in only two of them and develop to cystacanths in only one species (Uglem, 1972*a*,*b*).

Initiation of infection in intermediate hosts. Factors other than ingestion and hatching must be considered in the epizootiology of acanthors. Success in initiating infection, the effect of previous infection and the ultimate ability to develop cystacanths are important considerations in the spread of parasites through host populations. It is difficult, if not impossible, to know what percentage of eggs is viable in nature. In cockroaches, *Periplaneta americana*, approximately 25% of the fully developed eggs of *Moniliformis moniliformis* given orally in small numbers succeed in reaching the cystacanth stage. Although the percentage of success decreases as the number of eggs administered to each cockroach increases, there is no evidence of a saturation level and previous infection does not seem to affect the success of superimposed infections (Hynes & Nicholas, 1957). Various life cycle studies (Ward, 1940*b*; Hynes & Nicholas, 1957; Harms, 1965*a*; Nickol & Dappen, 1982) reveal that under laboratory conditions, where exposure and numbers of eggs are great, it is usually possible to infect between 70% and 80% of the appropriate invertebrates. Of course, wild-caught invertebrates are infected at much lower levels, probably due to lower exposure rates.

Cystacanth activation. After reaching the cystacanth stage, acanthocephalan development is again suspended until an external stimulus is supplied by an appropriate vertebrate. Little information is available regarding the length of time cystacanths remain infective in intermediate or paratenic hosts, but those of *Moniliformis moniliformis* remain infective to rats after at least one year in *Periplaneta americana* (Buckner & Nickol, 1975). It is generally assumed that cystacanths remain infective throughout the life of the intermediate host.

Although cystacanths of *Moniliformis moniliformis*, *Echinorhynchus truttae* and *Polymorphus minutus* are known to be freed from their intermediate hosts in the vertebrate stomach, activation does not appear to occur until they reach the small intestine (Graff & Kitzman, 1965; Awachie, 1966; Lingard & Crompton, 1972, respectively). *Polymorphus minutus* cystacanths introduced directly into the small intestine of ducks established infections (Lingard & Crompton, 1972), demonstrating that all necessary conditions for activation are present there.

Bile salts are important in cystacanth activation. Graff & Kitzman (1965) achieved *in vitro* activation of at least 80% of the *Moniliformis moniliformis* cystacanths treated with sodium taurocholate, sodium glycocholate and

sodium cholate in a pH range of 7.4 to 8.5. Sodium taurocholate was the most effective bile salt, giving activation more rapidly and at lower concentrations. Activation was stimulated by carbon dioxide and inhibited by oxygen. Cystacanths activated by this treatment were infective to rats. Pretreatment of cystacanths with proteolytic enzymes was unnecessary for activation and any effect questionable. Rats in which the bile duct openings were surgically transferred to the cecum were refractory to infection.

Cystacanths of *Polymorphus minutus* can be activated *in vitro* in a basic salt solution, but addition of duck bile or any of several bile salts markedly enhances activation. Temperatures between 42 °C and 44 °C and a pH of 7.0 are optimal for activation (Lackie, 1974). Kennedy *et al.* (1978) studied effects of a variety of stimuli on activation of *Pomphorhynchus laevis* cystacanths. They found that natural bile (either piscine or mammalian) was the most effective of the media tested.

Conditions under which cystacanth activation has been achieved *in vitro* are consistent with those expected to exist *in vivo*. The temperature, pH, osmotic pressure and bile salt concentrations in the environment of *Polymorphus minutus* in ducks (Crompton, 1969; Crompton & Edmonds, 1969) are similar to those found by Lackie (1974) to be favorable for activation of *P. minutus* cystacanths. The speed with which activation occurs *in vitro* is also consistent with *in vivo* observations (Lackie, 1974).

The role in host specificity played by stimuli for cystacanth activation apparently varies. Although Kennedy *et al.* (1978) concluded that stimuli for some species are more specific than for others, the requirements for activation are comparatively general. As Crompton (1970) pointed out, the fact that cystacanths of many species are activated by conditions in vertebrates destined to become paratenic hosts suggests that activation stimuli may not be a principal factor in determining the host specificity of many species.

Initiation of infection in definitive hosts. After activation, cystacanths encounter obstacles in addition to defensive responses by hosts. Several laboratory studies (Kennedy, 1974; Buckner & Nickol, 1975) report great intraspecific differences in cystacanth success. The success of *Pomphorhynchus laevis* cystacanths depends upon water temperatures and the nutritional state of the host but not on the number of cystacanths administered (Kennedy, 1972). On the other hand, survival of *Leptorhynchoides thecatus* cystacanths is density-dependent at high levels of infection. Green sunfish, *Lepomis cyanellus*, fed 10, 20, or 40 *L. thecatus* cystacanths retain only 10 to 15 of them. When fed 40 cystacanths, parasite survivorship declines until, after 2 weeks, the worm level is similar to that

in fish fed 20 cystacanths (see Fig. 11.6). Resources in green sunfish may limit *L. thecatus* to about 15, or approximately two per pyloric cecum (Uznanski & Nickol, 1982).

In addition to environmental temperature, nutritional state of the host and the number of cystacanths ingested, the sex of the host may also affect the success rate of cystacanths. For example, male rats retain a greater number of *Moniliformis moniliformis* cystacanths than do females (Crompton & Walters, 1972). The monograph by Crompton (1970) should be consulted for a more complete review of these and other aspects of the infection process.

9.3.2 *Host response*

Site location. After activation, acanthocephalans must locate in the proper site for continued development. Frequently this site is not that at which activation occurs. In intermediate hosts, acanthors are freed from embryonic membranes in the alimentary canal and must penetrate surrounding tissue and continue development in a parenteric site. Even in definitive hosts, the initial site of attachment is not always that required for continued growth (Holmes, 1962*a*; Uglem & Beck, 1972). Site location does not bear directly on epizootiology, but defensive response by the host during migration and development does, when spread of the parasite is prevented by stopping egg production or by killing infective stages.

Morphological considerations. Structural features do not seem to be important defensive devices against acanthocephalans. There is no evidence that anatomical factors such as villus length or the size and shape of the crypts of Lieberkühn, suspected (Smyth & Smyth, 1968) of rendering some carnivores refractory to *Echinococcus granulosus*, serve as deterrents to acanthocephalan attachment.

Various grinding organs found in alimentary canals of many potential hosts pose possible risks to invading acanthocephalans, but direct evidence of damage to eggs, acanthors or cystacanths is lacking. Indeed, mechanical damage by amphipod mouth parts is suspected to assist hatching of *Polymorphus minutus* eggs (Hynes & Nicholas, 1957) and grinding by the gastric mill promotes liberation of *Echinorhynchus truttae* acanthors (Awachie, 1966). Chitinous peritrophic membranes of many insects and some crustaceans represent possible barriers to infection. The toll on invading acanthors is unmeasured, but the presence of viable cystacanths in these organisms attests to the fact that at least some are successful in penetration.

Absence of evidence that morphological features inhibit successful

transmission may be due to lack of study. Certainly many acanthors succumb to cellular responses in the epithelium (Moore, 1962; Robinson & Strickland, 1969; Nickol & Dappen, 1982) or in the serosa (DeGiusti, 1949*a*; Hynes & Nicholas, 1958). The extent to which prior damage contributes to these fatalities is unknown. In *Gammarus pulex* dead or damaged acanthellae of *Polymorphus minutus* are readily encapsulated and melanized (Crompton, 1967). Future studies could reveal that structural features are important, just as are feeding preferences and physiological traits, in determining host specificity and intensity of parasitism.

Cellular response. There are many reports describing pathology of acanthocephalans in definitive hosts, but there is little evidence that host responses retard or prevent egg production. It is well known, though, that individuals of some species parasitize the alimentary canal of vertebrates in which they cannot attain maturity (Holmes, 1979). The exact mechanisms, or missing requirements, that prevent egg production are unknown. Nutritional studies such as those of Nesheim, Crompton, Arnold & Barnard (1977), Parshad, Crompton & Nesheim (1980) and Crompton, Singhvi & Keymer (1982) illustrate that this failure to attain maturity, at least in some cases, is not a result of a defensive response by the host.

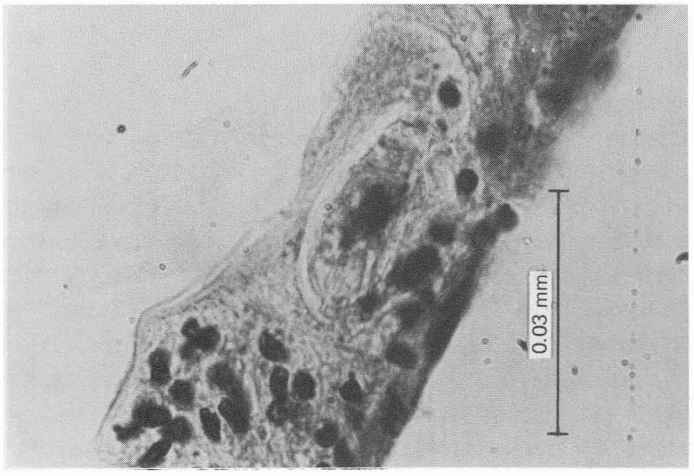
Invertebrates, on the other hand, frequently display cellular responses that result in destruction of invading acanthors. In some cases these responses serve to limit intensity of infection in normal intermediate hosts. A portion of the invading *Moniliformis moniliformis* acanthors is encapsulated, melanized and killed in *Periplaneta americana* (Robinson & Strickland, 1969; Schaefer, 1970) and the intensity of the hemocyte response may be greater when large numbers of acanthors are present (Robinson & Strickland, 1969). Some *Leptorhynchoides thecatus* are destroyed by cellular responses in *Hyalella azteca*, the usual intermediate host, especially at low temperatures when development is slowed (DeGiusti, 1949*a*).

In other instances, defensive responses may account for the host distribution of acanthocephalan species. Larvae of *Macracanthorhynchus hirudinaceus* develop successfully in many species of scarabaeid beetles (Kates, 1943), but not all scarabaeids are equally satisfactory hosts. Defensive reactions by some result in encapsulation and death of many invading acanthors (Miller, 1943). In Great Britain all three native species of *Gammarus* can be found infected by *Polymorphus minutus*. Hynes & Nicholas (1958), however, described strains of the parasite adapted to each of the amphipod species. In the 'wrong' amphipod, development is slowed and many parasites are encapsulated and melanized (Hynes & Nicholas,

1958). Similarly, *Mediorhynchus grandis* is capable of development in dock beetles, but development is delayed and many parasites are partially or wholly chitinized; development is much more satisfactory in grasshoppers (Moore, 1962). At sites where *Plagiorhynchus cylindraceus* acanthors penetrate the gut walls of isopods (*Armadillidium vulgare*), there is an immediate accumulation of hemocytes that may completely engulf the parasites leading to their degeneration (Schmidt & Olsen, 1964). This response (Fig. 9.5) is made by adult isopods and contributes to their great resistance to infection; juvenile isopods display no such response and are highly susceptible (Nickol & Dappen, 1982).

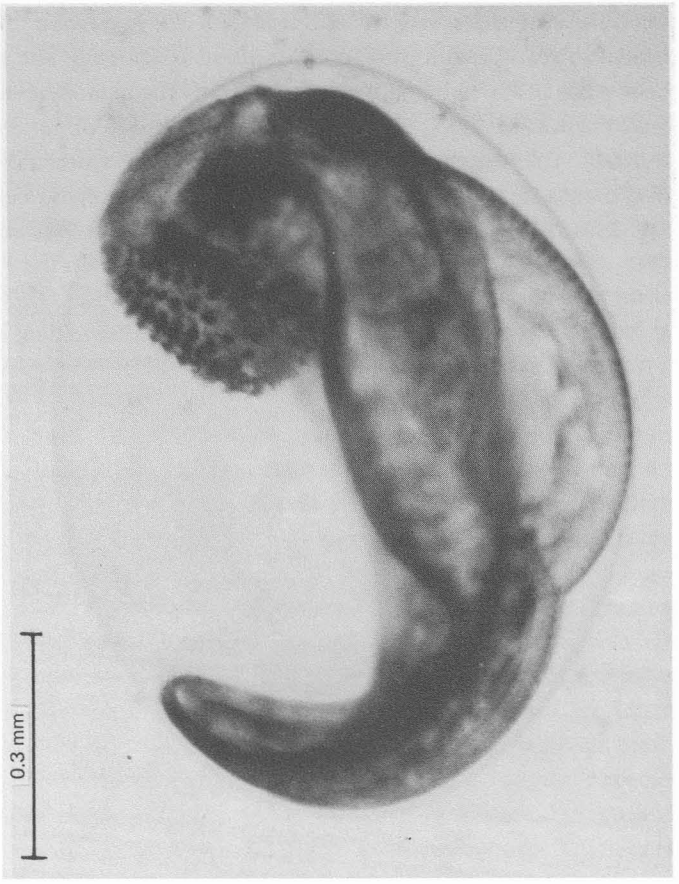
Acanthocephalans of many species are invested in a thin, transparent envelope as they develop in the hemocoel of arthropod intermediate hosts (Fig. 9.6). Perhaps influenced by the knowledge that arthropods make defensive hemocyte responses to acanthors and encouraged by Salt's (1963) review of defensive reactions by insects to metazoan parasites that frequently result in parasite encapsulation and death, the envelope frequently has been viewed as originating from host hemocytes as a defensive reaction (Bowen, 1967; Mercer & Nicholas, 1967; Poinar, 1969; Ravindranath & Anantaraman, 1977). After penetrating epithelial tissue of the host, acanthors undergo development beneath the serosa before entering the hemocoel (DeGiusti, 1949*a*; Crompton, 1964) leading Crompton

Fig. 9.5. Photomicrograph of a cross-section through the mid-hindgut of an adult *Armadillidium vulgare* 47 hours after feeding of *Plagiorhynchus cylindraceus* eggs, showing an acanthor in the epithelium and the hemocyte response. (Reproduced from Nickol & Dappen, 1982, with permission of the editor of the *Journal of Parasitology*.)



(1964) to the initial conclusion that the envelope originates as a result of a wound-healing response by the host to its stretched or damaged serosa. Subsequent descriptions (Bowen, 1967; Denny, 1969) were consistent with that view. Robinson & Strickland (1969), however, injected *Moniliformis moniliformis* acanthors directly into the hemocoel of cockroaches and demonstrated that envelope formation does not require parasite development in the serosa. Earlier workers (Meyer, 1938*a*; Pflugfelder, 1956; Moore, 1962) attributed the envelope's origin to the parasite. Rotheram & Crompton (1972) and Lackie & Rotheram (1972) concluded that the

Fig. 9.6. Early cystacanth of *Moniliformis moniliformis* removed from the hemocoel of *Periplaneta americana*, showing surrounding envelope.



envelope comprises a membranous coat formed from microvillate projections of the early acanthella and later separated from the parasite's surface. Based on observations of *M. moniliformis*, *Plagiorhynchus cylindraceus* and *Prosthenorchis elegans* every second or third day until cystacanths were fully formed, Wanson & Nickol (1973) noted that the membranous coat around the early acanthella bears armature of the acanthor and concluded that as the acanthella develops from the central nuclear mass of the acanthor, remnants of the acanthor's tegument remain as the envelope. Lackie & Lackie (1979) provided additional evidence of parasite origin for the envelope by demonstrating that most cystacanths of *M. moniliformis* grown in locusts and transplanted within their envelopes to cockroaches did not evoke hemocyte response, while locust tissue was readily encapsulated by cockroaches.

Hemocytes collect on the surface of the envelope during early stages of acanthella development but are soon dispersed or destroyed (Robinson & Strickland, 1969; Lackie & Rotheram, 1972; Wanson & Nickol, 1973) and the envelope apparently provides protection from further hemocyte response. Lackie (1975) examined the envelope's role in determining intermediate host specificity for *Moniliformis moniliformis* and observed that the degree of protection afforded by the envelope varies depending upon the species of cockroach. He suggested that avoidance of hemocytic encapsulation resulted from quantitative variation of some unknown parameter of surface properties rather than from the roach's all-or-none discrimination of a qualitative difference. Brennan & Cheng (1975) provided evidence that mucins in the envelope ultimately prevent melanization of *M. moniliformis* by blocking required enzymatic activity in the cockroach hemolymph. It is possible that the degree to which mucins of an acanthocephalan species retard enzymatic activity of various invertebrates represents the quantitative variation hypothesized by Lackie (1975).

It is apparent that the envelope surrounding developing acanthocephalans of some species is a product of the parasite rather than a result of a defensive response by the host hemolymph. Although some acanthors may be under hemocyte assault in the gut wall while others in the same host are forming envelopes under the serosa (Rotheram & Crompton, 1972), hemocytic encapsulation is independent of envelope formation. Both of these phenomena, in turn, are distinct from cystacanth encapsulation that sometimes occurs in visceral organs of vertebrates. Upon ingestion by vertebrates in which they cannot grow to adulthood, cystacanths of some species localize in mesenteries or visceral organs where they frequently are encapsulated. *Neoechinorhynchus cylindratus* is the only species for which structure of the cyst has been studied carefully. The walls of these cysts

are of host origin and comprise an inner layer of collagenous connective tissue and a thinner outer layer of fibroblastic cells (Bogitsh, 1961). While the cystacanths remain viable, the vertebrates serve as paratenic hosts but, frequently, encapsulated cystacanths are necrotic and appear to have been destroyed. The duration of viability is unknown but Crompton (1970) suggested that encapsulation follows the pattern of chronic inflammatory reactions; thus, it is likely that degeneration is an extended process.

Humoral response. There is evidence that humoral responses by invertebrate and vertebrate hosts play a role in limiting acanthocephalan infections. Juvenile isopods, *Armadillidium vulgare*, are highly susceptible to infection by *Plagiorhynchus cylindraceus*, but adults are nearly refractory. In addition to an effective cellular response to invading acanthors, electrophoresis reveals that adult isopods produce an increase in one of the protein components of their hemolymph when subjected to penetrating acanthors. Hemolymph of juvenile isopods shows no such change. This finding is interpreted as indirect evidence for humoral resistance (Nickol & Dappen, 1982).

Evidence that poikilothermic vertebrates are capable of responding immunologically to acanthocephalans is clearer, but whether the response results in resistance to parasitism is unresolved. Numbers of a cestode, *Proteocephalus exiqus*, and of an unidentified species of *Neoechinorhynchus* show an inverse relation in ciscoes, although the two species occupy different intestinal sites. A nonspecific immunity may limit either tapeworms or acanthocephalans when one of them is present in large numbers (Cross, 1934). *Pomphorhynchus laevis* matures in chub, *Leuciscus cephalus*, and evokes production of a precipitating antibody (Harris, 1970). Antibodies are not produced in four other piscine species in which *P. laevis* occurs but does not mature (Harris, 1972). These findings led Harris (1972) to speculate that the antibodies are produced only in response to excretory or secretory products of mature worms. The role of antibody response in limiting reinfection was not considered. In goldfish, *Carassius auratus*, the rate of *P. laevis* establishment is not affected by the presence of an existing infection (Kennedy, 1974), but *P. laevis* is not known to mature in goldfish and may not evoke an antibody response in them. Thus, the role of antibody response in protection against infection is still undetermined.

The possibility that acquired immunity limits acanthocephalan infections in homeotherms is better documented. Little information regarding immunity in birds is available although protein changes occur in the serum of *Polymorphus*-infected ducks (Petrov & Nikitin, 1975). Burlingame &

Chandler (1941) showed that rats infected with *Moniliformis moniliformis* possess decided resistance to superinfection. They interpreted their results as evidence of crowding or environmental effects rather than immunological reactions. This conclusion was based on the facts that survival of worms in the initial infection was not dose-dependent and that survival of superimposed worms that succeeded in establishing within the 'zone of viability' was equal to survival in primary infections. However, localization of the secondary worms was different from those of similar age in primary infections because 15–40% of them were found posterior to the zone of viability.

Others have attributed the resistance described by Burlingame & Chandler (1941) to immunological causes. Density-dependent expulsion in primary infections occurs at doses of 100 cystacanths, far more than the 20–25 used by Burlingame and Chandler. Recovery in primary infections of this magnitude has been shown to drop from 66% to 26% after 2 weeks (Miremad-Gassmann, 1981*a*) and from about 85% to about 15% between the fourth and eighth weeks (Andreassen, 1975*a*). At lower initial doses, expulsion is delayed (Andreassen, 1975*a*; Miremad-Gassmann, 1981*a*). Worms from secondary and tertiary infections are recovered in smaller percentages, are smaller, and are located more posteriorly in the intestine than are those from primary infections (Andreassen, 1975*a*; Miremad-Gassmann, 1981*a*).

Serum from rats fed 100 *Moniliformis moniliformis* cystacanths contains antibody capable of sensitizing homologous skin for at least 16 weeks after infection (Andreassen, 1975*b*) and Miremad-Gassmann (1981*b*) found specific antibodies in *M. moniliformis*-infected rats. The lemnisci and tegument are suspected either to form or to store metabolic antigens, because indirect immunofluorescence revealed that specific circulating antibodies bound predominantly to them (Miremad-Gassmann, 1981*b*).

There seems little question that acanthocephalans of at least some mammals, and perhaps of birds, poikilothermic vertebrates and invertebrates, induce resistance potentially capable of limiting the number of parasites and reducing the likelihood of epizootics. The extent to which immunity actually influences populations in nature is largely unknown.

9.4 Epizootics

9.4.1 *Epizootics in wild animals*

Definitive hosts. Helminth prevalence is seldom monitored regularly in wild populations, making detection of sudden increases unlikely. However, the particular number of eggs produced by most acanthoceph-

alans (even if the number is minimal for survival of the species) and the variety of adaptations that promote transmission present an ever-present risk of hyperinfestation for some animals.

Isolated instances of morbidity and death are frequently attributed to acanthocephalans. A dog with rabies-like symptoms that died in San Antonio, Texas, harbored about 300 specimens of *Oncicola canis*, some of which had perforated the intestine (Parker, 1909). *Plagiorhynchus cylindraceus* has occasionally been linked with paralysis of robins and starlings (Jones, 1928; Webster, 1943; Holloway, 1966) and death of bluebirds (Thompson-Cowley, Helfer, Schmidt & Eltzroth, 1979). The pathological significance of acanthocephalans in dying or dead animals is difficult to interpret because the link is frequently circumstantial. Further, densities are often no greater in dead animals supposedly killed by the acanthocephalans than in conspecific individuals that showed no adverse effects. Whether or not acanthocephalans cause or contribute to isolated morbidity and death, these cases probably do not indicate epizootics. More likely they represent instances of acanthocephalans supplementing effects of other stresses or of hyperinfestation in single animals rather than being a reflection of sudden increases of prevalence in the population.

On the other hand, sudden local increases in morbidity and mortality, including epizootics, do occasionally occur. From 1966 through 1970, mottled sculpin, *Cottus bairdi*, found dead in a creek in La Salle County, Illinois, suffered unusual pathology and dense infections from *Acanthocephalus dirus*. The intestine of one of the fish was detached from the stomach and in another instance a portion of sculpin intestine with 22 worms attached was found in the bottom of the stream. Many other sculpin harbored large numbers of *A. dirus*. After this apparent epizootic, no sculpin could be found in that region of the creek possibly due to extermination by acanthocephalans (Schmidt, Walley & Wijek, 1974).

Polymorphus magnus and *P. mathevossianae* were among the helminths deemed responsible for the 1968 death of 40% of the cygnets inhabiting a lake in the Kurgal'dzhin (USSR) nature reserve (Maksimova, 1972). Sanford (1978) described gross and histopathological findings in cygnets from two Canadian flocks of mute swans, *Cygnus olor*, whose deaths were attributed to heavy infections of *P. minutus*. In one case cygnets from one of seven ponds were noticeably smaller than those on the other ponds. One of the small cygnets died and was found to harbor more than 300 acanthocephalans. Seven of eight of the 1976 and at least six of seven 1977 cygnets from the other flock also died, apparently from large numbers of *P. minutus*. The birds had gradually lost weight and body condition over the summer and died in close succession from August through mid-October.

Eiders, *Somateria mollissima*, seem especially vulnerable to sudden outbreaks of acanthocephaliasis. *Polymorphus minutus* was linked to a 1947 epizootic with high mortality in eiders in Denmark (Christiansen, 1948), and *P. botulus* was regarded as the major contributing factor to 1956 and 1957 epizootics during which numerous eider ducks were found dead and dying in Maine and Massachusetts (Clark, O'Meara & Van Weelden, 1958). During the summers of 1956 and 1957, eiders from breeding colonies in the Netherlands remained in large numbers near a coastal island. Many were apparently ill and some were found dead. This epizootic was also attributed (Swennen & Van Den Broek, 1960) to *P. botulus*. Infections with as many as 3500 individuals of *P. minutus* may have been partially responsible for unusually high mortality of juvenile eiders in an archipelago of southwestern Finland during the summers of 1976 through 1979 (Itämies, Valtonen & Fagerholm, 1980).

Several factors, including the physical characteristics of the landscape, appear to initiate acanthocephalan-induced epizootics. Van Maren (1979*b*) described a locality on the river Merloux, in France, at which 48% of a dense population of *Gammarus fossarum* harbored *Polymorphus minutus*. At this site, in a meadow with ducks and chickens, the stream is narrow and shallow. It is an excellent habitat for amphipods and provides an unusually good opportunity for them to become infected. Away from this site, at a point not far downstream, only 2% of the amphipods were infected and farther downstream infected amphipods were not found.

There is evidence that constant exposure to acanthocephalans leads to a relatively small, stabilized density of worms among a population of vertebrates, but that sudden exposure to large numbers of intermediate hosts results in severe infections (Hynes & Nicholas, 1963). When a new or escaped flock of ducks encounters an environment such as the one described by Van Maren (1979*b*), an epizootic may ensue.

Environmental disturbances due to human activities or climatic conditions may also induce epizootics. *Polymorphus minutus* was first present in wildfowl on a reserve in Kent, England, after activities of a sand and ballast company altered the course of a traversing river. Although infected birds appeared healthy, in the succeeding 3 years the prevalence of *P. minutus* in mallards rose steadily to 39%, then 45%, and finally 71% (Crompton & Harrison, 1965). Often, environmental changes, which are frequently only temporary, or human activity produce a rapid increase of acanthocephalans routinely present in small or moderate numbers. Increasingly frequent outbreaks of disease have accompanied growing numbers of birds along the Swedish coast since spring hunting of seafowl was outlawed in the mid-1950s (Peresson, Borg & Fält, 1974). These

epizootics, in which approximately 90% of the ducklings died during the summer of 1970, are attributed to serious invasion by endoparasites, including *P. botulus* and *P. minutus*, brought about by overpopulation (Peresson *et al.*, 1974). Ice-covered waters during late winter often limit the places where eiders search for food. Congregation of large numbers in small areas leads to heavy infections. Grenquist (1970) believed these conditions to have caused a high level of mortality from *P. minutus* or *P. botulus* in eiders in Finland. Similarly, concentration of swans in summer on a reduced level of water is blamed for the Kurgal'dzhin reserve epizootic (Maksimova, 1972).

Intermediate hosts. Attempts to produce laboratory infections frequently result in the death of invertebrates, probably as a result of simultaneous penetration of the intestinal wall by large numbers of acanthors. Some of the species known to cause death under these conditions are *Macracanthorhynchus hirudinaceus* in June beetle larvae (Miller, 1943), *Neoechinorhynchus cylindratus* in ostracods (Ward, 1940*b*) and *N. rutili* in ostracods (Merritt & Pratt, 1964). Other species interfere with reproduction by intermediate hosts by retarding development of ovaries (Schmidt & Olsen, 1964), preventing appearance of secondary sex characteristics (Munro, 1953) or by suppressing both oogenesis and secondary sex characteristics (LeRoux, 1931). Hynes (1955) suggested that such interference with reproduction could account for the sudden, unaccountable disappearances of amphipods observed in certain lakes.

Studies of the seasonal occurrence of acanthocephalans in invertebrates frequently reveal different prevalences throughout the year, usually in relation to reproduction by adults in definitive hosts. While it is clear that pathological effects could result in unusually high mortality, acanthocephalan-induced 'die-offs' of invertebrates are unknown outside the laboratory.

9.4.2 *Epizootics in captivity*

Animals in zoological gardens, fish hatcheries and other confinements repeatedly suffer pathogenic effects of acanthocephaliasis and frequently die as a result. Most of the reported cases of acanthocephalan-induced epizootics among captive animals are of primates harboring *Prosthenorchis elegans* and, occasionally, *Oncicola spirula*. It is not unusual for recently captured, imported animals to die from acanthocephalan infections that were probably acquired before confinement. Four of 10 newly acquired squirrel monkeys, *Saimiri oerstedii*, died of *P. elegans* at the Gorgas Hospital in the Panama Canal Zone; the other six were

uninfected (Takos & Thomas, 1958). Nine of 30 marmosets acquired by the Medical College of Virginia and Dartmouth Medical School died of massive infections of *P. elegans* or from a combination of the parasite and other infections from 4 to 122 days after acquisition (Richart & Benirschke, 1963). Deaths of a marmoset, *Hapale jacchus*, and a mongoose, *Atilax paludinosus*, within 8 months of arrival at Regent's Park, London, were attributed to acanthocephalans (T-W-Fiennes, 1966). Heavy infections with a species of *Prosthenorchis* were deemed responsible for the death of 12 of 13 squirrel monkeys that died within 18 days of arrival at a primate colony in the USA (Morin, Renquist, Johnson & Strumpf, 1980).

Acanthocephalans do not always cause death directly, but they may cause lesions that enable other pathogens to become established (Schmidt, 1972*a*). A baboon, *Papio papio*, that harbored many *Oncicola spirula* died of generalized tuberculosis in a Paris zoo (Brumpt & Urbain, 1938), suggesting that acanthocephalans may act synergistically with other pathogens.

Circumstances of confinement usually result in the concentration of definitive hosts in an environment abounding with invertebrates suitable as intermediate hosts. This is especially true of primate colonies, where roaches suitable for development of *Prosthenorchis elegans* and *Oncicola spirula* are plentiful (Brumpt & Desportes, 1938). Under such conditions parasites of introduced animals may spread rapidly among others of the colony. *Prosthenorchis elegans* and *O. spirula* occur naturally only in the New World. Importation, however, has permitted spread to both Old and New World primates in zoos and primate colonies around the world, where it commonly results in loss of entire stocks (Schmidt, 1972*a*). *Prosthenorchis elegans* or *O. spirula* epizootics occurred among Old World primates in Paris (Brumpt & Urbain, 1938) and Rotterdam (Van Thiel & Wiegand-Bruss, 1945). Moore (1970) described an epizootic among the great apes at the Hogle Zoo in Salt Lake City, Utah, that involved five gibbons, three orang-utans and one gorilla. Three of the gibbons died. It was concluded that the epizootic began with the introduction of a gibbon that had been acquired from a source in Florida known to handle large numbers of South American monkeys. The gibbon had diarrhea upon arrival and grew progressively weaker during the following months. The other animals subsequently became ill. Importation of *O. spirula*-infected gibbons purchased from a New York dealer had previously (Chandler, 1953) been blamed for an epizootic during which many animals became ill and three gibbons, two squirrel monkeys and a pig-tailed macaque died at the Houston Zoological Gardens.

Among confined animals, epizootics in mammals are most conspicuous

and receive the most attention. However, an epizootic caused by *Acanthocephalus dirus* among trout at the New Hampshire State Fish Hatchery at New Hampton (Bullock, 1963) attests to the fact that under the right circumstances poikilotherms are also subject to severe outbreaks of acanthocephaliasis.

9.5 Regulation of numbers

Fecundity of acanthocephalans, like most groups of parasites, is great relative to that of their hosts. Even if egg production is the minimum required for survival of the species, as Croll (1966) suggested, the number of eggs produced by each female is considerable (Kates, 1944; Crompton & Whitfield, 1968*a*; Crompton *et al.*, 1972). The large number of infectious agents and various adaptations to enhance the probability of successful transmission provide potential for massive infections in individual hosts. The relative infrequency of epizootics is due, in part, to mechanisms that regulate numbers (see Chapters 10 and 11).