JYVÄSKYLÄ STUDIES IN BIOLOGICAL AND ENVIRONMENTAL SCIENCE
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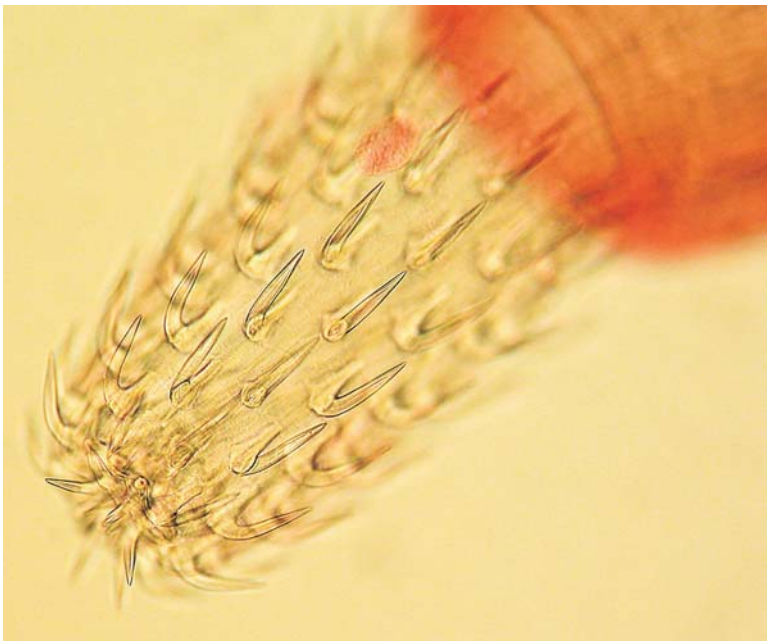
Daniel P. Benesh

Larval Life History, Transmission Strategies, and the Evolution of Intermediate Host Exploitation by Complex Life-Cycle Parasites



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Cover picture: Proboscis of Acanthocephalus lucii
by Daniel P. Benesh

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ABSTRACT

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Complex life-cycle parasites use their intermediate hosts both as an energy source and as a vessel for transmission to the next host in the life cycle. Parasites that grow rapidly to a large size may have high fitness (e.g. time spent in uninfective stages is limited), yet those that grow too aggressively may reduce host viability and their own probability of successful transmission. I examined aspects of both the growth and transmission strategy of an acanthocephalan (*Acanthocephalus lucii*) in its isopod intermediate host. In an experimental infection, the relative rate of larval parasite growth slowed over time, and eventually parasites seemed to reach a threshold biomass sustainable by their hosts. Consequently, late during this infection parasite growth depended on the level of resources a given host could provide. The rapid, unconstrained growth of young *A. lucii* appeared worse for isopod viability than the slow, constrained growth of larger parasites. Female *A. lucii* grew larger than males and their size was more strongly related to host size, suggesting they invest more in growth and are consequently more limited by resources. Patterns of sexual dimorphism across acanthocephalan species suggest that sexual selection driving adult dimorphism may promote sexual divergence in larval growth strategies. Isopods infected with *A. lucii* cystacanths spent less time hiding and had darker abdominal coloration than uninfected isopods. The magnitudes of these two altered traits were not correlated, even when both traits were measured somewhat repeatably from individual hosts. Parasite-induced host alteration seemed to increase over time as parasites grew. Refuge use by infected isopods decreased over eight weeks, and, in general, the altered coloration of infected isopods seemed to increase with parasite growth. An increasing probability of host mortality, as well as a decreasing potential for additional parasite growth, could favor increased parasitic manipulation of host phenotype.

Keywords: *Acanthocephalus lucii*, complex life cycles, host manipulation, isopod, life history, sexual selection, virulence

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LIST OF ORIGINAL PUBLICATIONS

This thesis is based on five original papers, which will be referred to in the text by their Roman numerals (I-V). I am the lead author in all papers, and I was responsible for the majority of the planning and execution of each study.

I Benesh, D. P. & Valtonen, E. T. 2007. Effects of *Acanthocephalus lucii* (Acanthocephala) on intermediate host survival and growth: implications for exploitation strategies. Journal of Parasitology 93: 735-741.

II Benesh, D. P. & Valtonen, E. T. 2007. Proximate factors affecting the larval life history of *Acanthocephalus lucii* (Acanthocephala). Journal of Parasitology 93: 742-749.

III Benesh, D. P. & Valtonen, E. T. 2007. Sexual differences in larval life history traits of acanthocephalan cystacanths. International Journal for Parasitology 37: 191-198.

IV Benesh, D. P., Valtonen, E. T. & Seppälä, O. Multidimentionality and intra-individual variation in host manipulation by an acanthocephalan. Submitted manuscript.

V Benesh, D. P., Seppälä, O. & Valtonen, E. T. The relationship between larval size and the host manipulation strategy of *Acanthocephalus lucii* (Acanthocephala). Manuscript.

1 INTRODUCTION

1.1 Parasitism and complex life cycles

Parasitism is a symbiotic relationship in which one organism (the parasite) lives on or in another organism (the host), benefiting at its expense (Zelmer 1998). The effects parasites have on their hosts range from relatively benign nutrient stealing all the way to killing the host. Parasitism has independently evolved numerous times in the history of life (de Meeûs & Renaud 2002), and it is generally assumed that the diversity of parasitic organisms easily surpasses that of their free-living counterparts (Windsor 1998). The vast numbers of niches offered by free-living organisms and the advantages associated with parasitism have presumably favored adoption of such a lifestyle. The environments inhabited by parasites, i.e. hosts, are relatively predictable with a steady supply of food, unlike the often stochastic fluctuations in the external environment. Moreover, some selective forces which act heavily on free-living animals, such as predation, are often not applicable to parasites. On the other hand, hosts have sophisticated mechanisms for killing invaders, i.e. immune responses, so the habitat of parasites can also be hostile.

Given the stability of a parasite's environment, it is perhaps surprising that many parasite life cycles involve the mandatory use of multiple hosts. These complex life-cycle (CLC) parasites complete different phases of their ontogeny in different hosts. For example, development may begin in one host (a first intermediate host), continue in another (a second intermediate host), before sexual reproduction occurs in a final host (the definitive host). Transmission between hosts generally, though not exclusively, occurs through trophic interactions, i.e. parasites move from hosts low on a food web to those on higher trophic levels via predation. CLCs are characteristic of many parasite taxa, both in protozoan (e.g. apicomplexans, kinetoplastids) and metazoan groups (e.g. cestodes, trematodes, nematodes, and acanthocephalans). Given some of the obvious disadvantages associated with a CLC, such as getting from host to host and dealing with multiple, perhaps very different host responses to

infection, a parasite's potential gains must be substantial to favor the evolution of such a lifestyle. Life history theory, how organisms allocate limited resources to growth and reproduction over time (Stearns 1992), has been used to generate hypotheses about what these advantages may be. Invasion of hosts higher on a food web could allow for the evolution of larger body size and greater fecundity, whereas incorporation of hosts at lower trophic levels could reduce mortality and increase the likelihood of reaching a definitive host (Choisy et al. 2003, Parker et al. 2003a). CLCs may also increase the number of opportunities for outcrossing (Brown et al. 2001b, Rauch et al. 2005).

Compartmentalization of the life cycle into distinct niches is not a phenomenon unique to parasites. Many marine invertebrates, insects, fish, and amphibians, for example, exploit different resources during their life cycles (Ebenman 1992). However, a CLC likely imposes constraints on the evolution of parasite life history which are not applicable to free-living animals. For instance, a high mortality rate generally selects for more rapid development (Williams 1966, Stearns 1992). Faster parasite development, however, requires additional consumption of host resources, increasing the damage inflicted on the host. At some point, the level of parasitic exploitation may be great enough to kill the host, and, as a consequence, overly aggressive parasites die before being transmitted. Thus, unlike free-living organisms, parasites are under some pressure to exploit their environment, the host, prudently. This tradeoff between the benefits of exploiting the host (faster growth) and the potential costs of over-exploitation (decreased transmission) theoretically determines how virulent a parasite should evolve to be (Anderson & May 1982, Frank 1996, Ebert & Herre 1996).

1.2 Exploitation of intermediate hosts by helminths

The tradeoff between the costs and benefits of host exploitation presumably shapes the larval life history of helminth parasites in their intermediate hosts. Before transmission between hosts can occur, generally trophically, these parasites must reach an infective developmental stage. Parasites use host resources to grow, but the intermediate host must remain somewhat viable for transmission events to occur (Fig. 1). The rate and duration of parasite growth in the intermediate host, therefore, likely reflects a balance between the benefits of consumption (quick development and a large larval size) and decreased host viability/parasite transmission (Parker et al. 2003b).

There is empirical support in a variety of helminth-intermediate host systems for the basic premises presumed to govern the evolution of virulence. Accelerated growth decreases the amount of time parasites spend in uninfective ontogenetic stages that cannot survive transmission to the next host in the life cycle. Moreover, the potential fitness benefits linked to a larger larval size may include better establishment success in the definitive host (Rosen & Dick 1983, Steinauer & Nickol 2003), higher adult fecundity (Fredensborg & Poulin 2005),

and less developmental time to maturity (Poulin 2007). Conversely, many parasites exploit their intermediate hosts at levels which reduce host viability (e.g. Hynes & Nicholas 1958, Sorenson & Minchella 2001, Duclos et al. 2006), and the common phenomenon of intensity-dependent mortality (e.g. Nie & Kennedy 1993, Ashworth et al. 1996, Fredensborg et al. 2004) suggests that high parasite burdens can decrease host viability. On the other hand, in a variety of systems neither parasitism nor infection intensity affects host viability (e.g. Uznanski & Nickol 1980, Wedekind 1997, Hurd et al. 2001). Thus, there seems to be considerable variation in the effects parasite species have on intermediate hosts, perhaps suggesting that a variety of solutions to the virulence tradeoff have evolved.

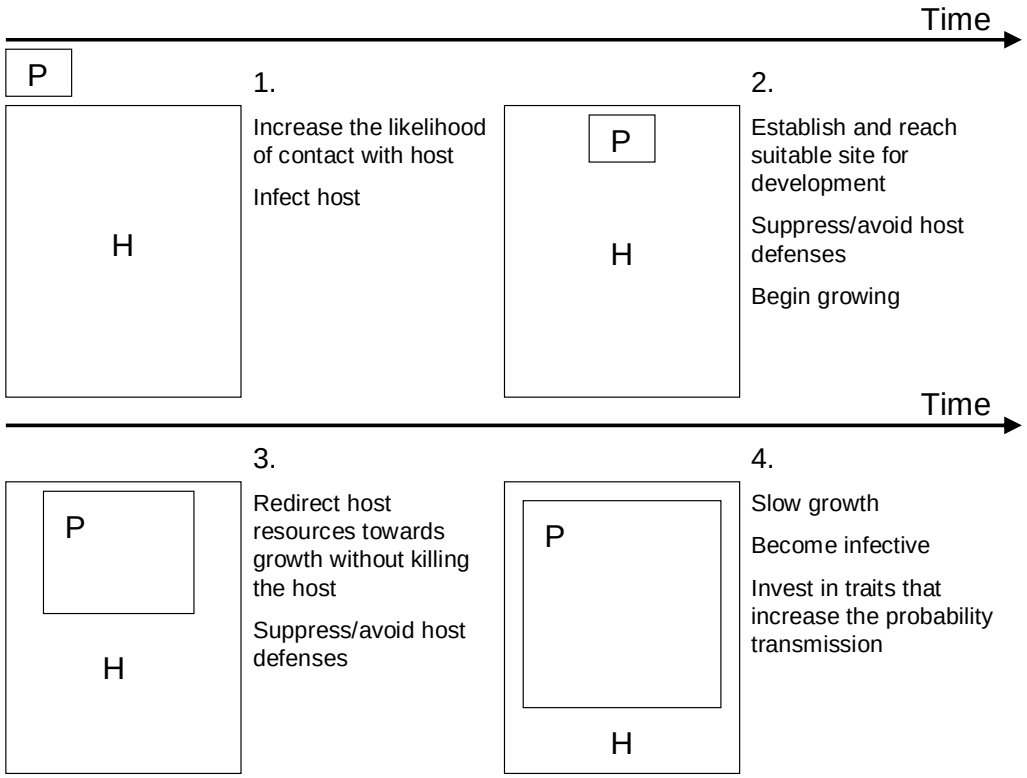

FIGURE 1 A schematic diagram of the pressures on trophically-transmitted parasites (P) in their intermediate hosts (H) over time. Time increases from boxes 1 to 4.

For CLC parasites, many factors may modify the cost/benefit ratio associated with exploiting the intermediate host. Parasites that castrate their host, for example, may divert host resources away reproduction, and thereby liberate more resources for parasite growth and/or host maintenance (Hurd et al. 2001, Sorenson & Minchella 2001). The life history characteristics of the intermediate host, such as its size and life span, may determine the amount of time and resources available for parasite development (Poulin 1994a). The likelihood of sharing an intermediate host with conspecifics could also shape parasite growth strategies (Parker et al. 2003b). If parasite abundance is high and within-host competition is likely, reduced parasite growth could ensure that the host

remains viable in the case of a multiple infection. Finally, factors affecting the life history evolution of adult parasites (e.g. age-dependent mortality (Gemmill et al. 1999), sexual selection (Poulin & Morand 2000), phylogenetic constraints (Morand & Poulin 2003)) probably affect larval growth patterns. For example, sexual selection driving male/female size dimorphism in adult parasites (Shine 1989, Andersson 1994) could indirectly favor sexually divergent larval growth patterns.

The conditions experienced by parasites developing in an intermediate host are unlikely to be entirely predictable. Environmental heterogeneity could be represented by variation in host condition or the presence, size, and number of co-occurring parasites. Parasites that can alter their growth in response to this unpredictability are presumably at an advantage, and, consequently, a certain level of developmental plasticity may be expected within parasite species (Davies & McKerrow 2003, Poulin 2003, Poulin 2007). Indeed, the growth of larval helminths often varies with proximate conditions. For example, in many species, individual parasites grow larger in bigger or faster-growing hosts (Wedekind 1997, Dezfuli et al. 2001, Steinauer & Nickol 2003, Barber 2005), suggesting parasites can adjust their growth in accordance with resource availability. Parasites also commonly develop to a smaller size in the presence of competitors (e.g. crowding effects; Shostak et al. 1985, Wedekind 1997), which may be a response to resource limitations or a strategy to maintain host viability (Parker et al. 2003b, Michaud et al. 2006).

Parasite growth in the intermediate host eventually slows or stops and parasites become capable of infecting the next host in the life cycle. At this ontogenetic stage, the host primarily serves as a vessel for bringing the parasite to the next host in the life cycle; it is presumably no longer a major source of energy (Fig. 1). For trophically-transmitted parasites, the likelihood of any particular intermediate host being eaten by an appropriate target host is assumed to be low (Dobson 1988). This generates a selective burden on parasites to increase intermediate host susceptibility to predation by target hosts. Consequently, many trophically-transmitted parasites seem to alter their intermediate host's phenotype in ways that increase transmission to the next host in the life cycle (Moore 2002, Thomas et al. 2005). This phenomenon has been collectively referred to as host manipulation.

Though the adaptiveness of host alteration has been vigorously debated (Holmes & Zohar 1990, Poulin 1995, Thomas et al. 2005), the increase in parasite transmission associated with altered host phenotypes in some systems appears relatively clear. Both field observations (e.g. Brown et al. 2001a, Perrot-Minnot et al. 2007) and laboratory experiments (e.g. Bethel & Holmes 1977, Moore 1983, Bakker et al. 1997) indicate that some parasites render their intermediate hosts more susceptible to predation. Moreover, in many cases, though not all, manipulated host traits only arise as parasites become infective to the next host in the life cycle (e.g. Bethel & Holmes 1974, Pulkkinen et al. 2000, Seppälä et al. 2005), strongly suggesting the host modifications represent parasite adaptations to increase transmission (Poulin 1995). Given the obvious potential benefits associated with host manipulation, i.e. increased transmission, one could expect

this to be a ubiquitous parasite strategy. In some respects, host manipulation is a very common phenomenon; a variety of different parasite species are known to alter a variety of different host characteristics (reviewed by Moore 2002). However, the number, type, and magnitude of host trait alterations vary considerably between parasite species (Poulin 1994b, Thomas et al. 2005). The absence of severe host manipulation in all trophically-transmitted parasites suggests that there may be costs or constraints associated with the evolution of host manipulation (Poulin 1994a). These costs could be ecological in that host manipulation increases the probability of predation by unsuitable hosts (e.g. Mouritsen & Poulin 2003), or they could be physiological in that parasites must expend energy to change host phenotype (Thompson & Kavaliers 1994).

On an evolutionary time scale, selection presumably minimizes the costs of host manipulation relative to the benefits. On a contemporary scale, however, the cost/benefit ratio associated with host manipulation may change with environmental conditions or infection age, perhaps favoring plastic manipulation strategies (Thomas et al. 2002). Within a parasite species, considerable variation in manipulated traits is often observed between individual hosts, and explaining this variation is essential to understanding the evolution of host manipulation (Perrot-Minnot 2004, Thomas et al. 2005). To explore the sources of this variation, however, altered traits must be measured representatively for *individual* hosts. For instance, if an altered trait stochastically varies in magnitude over time, short experiments may only capture a portion of an individual host's trait variability. Consequently, much of the variation observed in an altered trait may reflect random noise in the data rather than genuine differences between hosts.

Quantifying host manipulation is further complicated by the fact that many, if not most, parasites affect several aspects of their host's phenotype, including behavior, appearance, and physiology (e.g. Hindsbo 1972, Moore 1983, Bakker et al. 1997). Thus, documenting the extent that an individual host is manipulated by its parasites requires recording multiple traits. The various, parasite-induced modifications in host phenotype could arise via either linked or independent means (Cézilly & Perrot-Minnot 2005), and these mechanistic relationships may constrain how host manipulation evolves. For example, if two host alterations share the same underlying mechanism, then selection presumably acts on the "complex" of altered traits rather than each trait separately. Correlating the magnitudes of altered traits has been proposed as an initial step in evaluating their mechanistic similarity, i.e. positive correlations between traits being suggestive of related mechanisms (Cézilly & Perrot-Minnot 2005). However, to confidently correlate the magnitudes of different modified characteristics, representative trait values should be obtained for individual hosts. Thus, experimental setups that representatively quantify the extent individual hosts are manipulated are necessary to explore both the interrelationships between manipulated traits as well as the causes of between-host variation in altered characteristics.

1.3 Aims of the study

The generalized goal of this study was to investigate several aspects of intermediate host exploitation by an acanthocephalan parasite (*Acanthocephalus lucii* in its isopod intermediate host). I examined both larval parasite growth and host manipulation. By examining these two aspects of intermediate host exploitation in a single system, I hoped to gain a more comprehensive understanding of how parasites use their intermediate hosts. First, I investigated the effects of infection and parasite development on host survival and growth (I). Concomitantly, I assessed how several proximate factors, e.g. host size, host molting, and infection intensity, influenced parasite growth (II). I also explored how larval growth strategies may diverge between male and female parasites, and I considered whether sexual selection acting on adult body size may be responsible for these patterns (III). With regard to the parasite's transmission strategy, I assessed whether several traits differed between infected and uninfected hosts, and I searched for experimental setups capable of measuring apparently manipulated host traits with high repeatability (IV). Repeatable experimental designs presumably yield individually representative trait measurements, thereby permitting robust correlations between different altered traits to be conducted (IV). Finally, I investigated whether parasite growth explained any of the between-host variation in a manipulated trait (isopod coloration) that was measured with high repeatability (V).

2 STUDY SYSTEM

Acanthocephalus lucii exhibits a typical acanthocephalan life cycle (Schmidt 1985, Fig. 2); the definitive host is a vertebrate and the intermediate host is an arthropod. A variety of freshwater fish can serve as the definitive host including pike (*Esox lucius*), ruffe (*Gymnocephalus cernuus*), and burbot (*Lota lota*) (Chubb 1982 and references therein). European perch (*Perca fluviatalis*), however, is the most commonly reported definitive host species, and it is probably responsible for maintaining the parasite population in most localities (Brattey 1988). The dioecious adults mate in the intestine of fish, and females release shelled acanthors (eggs) into the environment with the host's feces. Freshwater isopods of the species *Asellus aquaticus* serve as intermediate host, and they become infected by ingesting these eggs. Parasites develop in the isopod's body cavity from an acanthor to the infective cystacanth stage, and this normally takes several weeks, depending on the temperature (Andryuk 1979, Bratty 1986). The life cycle is completed when an isopod harboring an infective cystacanth is eaten by an appropriate definitive host.

Studies examining the survival of isopods infected with *A. lucii* have given mixed results (Brattey 1986, Hasu et al. 2006, Hasu et al. 2007). None have examined parasite growth and host mortality concurrently. Typically, there is one worm per infected isopod (Brattey 1986), and it grows to a fairly large size relative to that of the host (Andryuk 1979, Fig. 2). Perhaps as a consequence of this aggressive growth, *A. lucii* development is retarded in high intensity infections (Pilecka-Rapacz 1986). Larval *A. lucii* also exhibit clear sexual size dimorphism (females are larger than males; Andryuk 1979). As parasites reach the infective cystacanth stage, the respiratory opercula of their hosts (appendages used to circulate water for respiration) become conspicuously darker (Brattey 1983), but isopod response to light or a disturbance is unaffected by infection (Lyndon 1996). Infected isopods are more susceptible to predation by perch, suggesting some aspect of the infection increases the probability of parasite transmission (Brattey 1983).

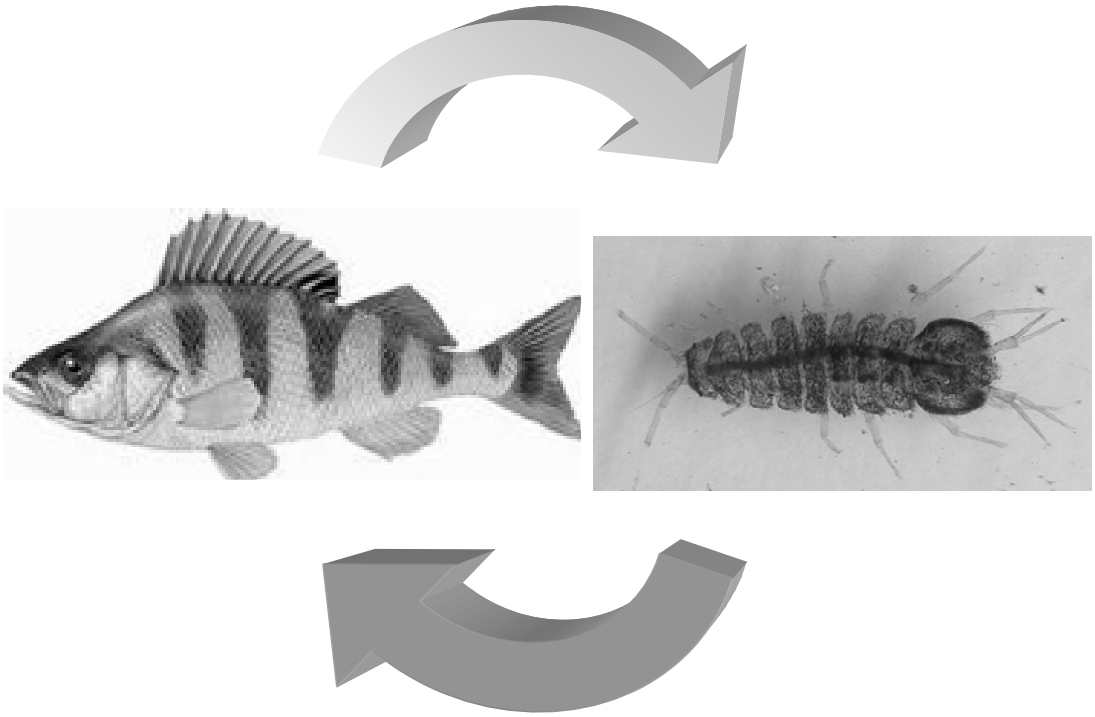

FIGURE 2 The life cycle of *Acanthocephalus lucii*. Isopods (*Asellus aquaticus*) serve as intermediate hosts and freshwater fishes, typically European perch (*Perca fluviatilis*), are the definitive hosts. Parasites grow rather large in the isopod intermediate host. In the picture above, the isopod's intestine, which normally runs parallel to the body axis, is laterally displaced as a consequence of the large worm in the animal's body cavity. (perch picture from www.fishing.pl, isopod picture taken by author)

This system has several advantages with regard to studying intermediate host exploitation. First, the larval parasite size to host size ratio is relatively large (Lafferty and Kuris 2002), suggesting parasites use intermediate host resources rather extensively, but it is still unclear how this affects the host. Second, although *A. lucii* seems to heavily exploit its isopod host, there is variation within the species in growth patterns, i.e. males and females grow to clearly different sizes. The causes and consequences of this larval dimorphism, however, are unexplored. Third, female isopods are castrated by infection (Brattey 1983), and, in similar systems, infected male hosts suffer decreased competitive ability and/or motivation to mate (Zohar & Holmes 1998, Sparkes et al. 2006). Thus, because host fitness is presumably relegated to nearly zero (Kuris 1997), alterations in host behavior or appearance are more likely to represent parasite, not host, adaptations, i.e. the host becomes an extension of the parasite phenotype (Dawkins 1982). Finally, the synchronization of host pigment modification with the onset of parasite infectivity strongly suggests this alteration serves to increase the probability of *A. lucii* transmission (Brattey 1983).

The isopods employed in this study came from two different locations in central Finland. Isopods used in experimental infections (I, II, V) were collected

from Niemijärvi (62°12'N 25°45'E), a small pond in which the only fish species present is *Carassius carassius*, the crucian carp. Because a suitable definitive host for the parasite is not present in the pond, this population served as a source of unexposed, uninfected isopods. Isopods naturally infected with *A. lucii* were collected from Lake Jyväsjärvi (62°14'N 25°44'E). Natural infections were used to assess parasite growth (III), host survival (I), and host phenotype manipulation (IV, V). In these experiments, infection was not a randomly assigned treatment, so there may be pre-existing differences between uninfected and infected isopods. I acknowledge the possibility that such differences might impact the measured characteristics. However, natural infections are preferable for some studies, because experimental exposures often produce unnaturally high infection intensities (I, II, Brattey 1986, Hasu et al. 2007).

3 RESULTS AND DISCUSSION

3.1 Larval parasite growth

In an experimental infection, the relative growth rate of larval *A. lucii* in isopods was most rapid at the beginning of the infection, but it then slowed over time (II). This pattern has been observed in other systems (e.g. Michaud et al. 2006), and is expected to be a generalized phenomenon in helminths (Parker et al. 2003b). It is probably favorable because it reduces the amount of time parasites spend in ontogenetic stages that are unable to infect the next host in the life cycle. Additionally, in the case of *A. lucii*, rapid growth may be beneficial in avoiding host defenses. A small proportion of developing *A. lucii* died in their intermediate host, and most of these dead parasites were relatively small (II). This concurs with other reports; dead acanthocephalans in their intermediate hosts are usually young acanthors or acanthellae (e.g. Nickol & Dappen 1982, Gleason 1989, Volkmann 1991). Thus, a rapid, initial growth rate may minimize the amount of time parasites spend in 'risky' ontogenetic stages.

Though rapid growth may have long-term metabolic costs (Metcalfe & Monaghan 2001), the most important cost presumably constraining parasite growth is the risk of killing the host via over-exploitation (Parker et al. 2003b). Studies on the relationship between *A. lucii* parasitism and isopod viability have returned mixed results. Brattey (1986) found exposure to *A. lucii* to consistently reduce isopod survival over 60 days, Hasu et al. (2006) observed exposed, infected isopods to actually survive better than controls, though only gravid female isopods were observed, and Hasu et al. (2007) noted that adult isopods exposed to *A. lucii* had higher mortality than juveniles, though they also harbored larger parasite burdens. My study used adult male and non-ovigerous female isopods (I), the stages most likely to be infected in nature (Brattey 1986), and the animals were observed for a longer period of time than in previous studies. Isopods that were exposed to and infected with *A. lucii* had reduced survival compared to unexposed controls, but this reduction was not consistent over time (I). Reduced host survival was primarily seen early during

the infection; after approximately 40 days post-exposure, parasitism did not increase host mortality (I). The rapid growth exhibited by young larval *A. lucii* may, thus, be worse for host viability than the slower growth of larger parasites observed later in the infection. Periods of rapid parasite growth seem associated with elevated host mortality in other helminths as well (e.g. Shostak et al. 1985, Duclos et al. 2006). This undermines some theoretical expectations in that high parasite mass, not growth rate, is assumed to be responsible for decreased host viability (Parker et al. 2003b).

Though there seemed to be a relationship between parasite growth and host viability, infection intensity did not affect host survival (I). This result was unexpected, although similar observations of intensity-independent mortality have been made on other acanthocephalans (Lackie 1972, Uznanski & Nickol 1980). The energetic stress experienced by the host is assumed to increase with infection intensity, but this need not be the case. For instance, each host may have a ceiling level of parasite biomass that can be maintained, regardless of how many individual parasites are present. Late during the infection, total worm volume was unrelated to intensity, which implies parasites reached this host-defined threshold biomass (II). By contrast, early on, total worm volume increased with the number of parasites, suggesting the consumption of host resources was greater in high-intensity infections (II). Nonetheless, even during the early part of the infection, host mortality did not appear to increase with intensity; if anything the opposite trend was observed (I). This may suggest that the expected connection between parasite load and host survival is modified by other factors. Indeed, the virulence of *A. lucii* in isopods seems to vary, for example, with host development (Hasu et al. 2006, Hasu et al. 2007).

Predictable variation in the probability of host death may favor plasticity in parasite growth strategies, e.g. parasites could reduce growth rates when host mortality is likely. Early during development, though, the average size of larval parasites was unrelated to either intensity, host molting rate, or host size (II). Thus, early *A. lucii* ontogeny seems fairly inflexible. As the experiment progressed, however, parasite growth seemed to vary with resource availability. For instance, late in the infection, average parasite size was higher in larger hosts that molted more frequently (II). Larger isopods presumably provide larval parasites more resources and/or space to grow. In other words, the host-defined "ceiling" for parasite growth is at a higher level in bigger hosts. Moreover, *A. lucii* infection seems to affect isopod growth either positively or not at all (I, Hasu et al. 2007), so this "ceiling" may be continuously moving up. The negative relationship between average worm size and intensity late in the infection, i.e. a crowding effect, was also presumably a consequence of resource constraints on parasite growth (II). Crowding effects have been observed in late stages infections of other acanthocephalans in their intermediate hosts (Awachie 1966, Dezfuli et al. 2001, Poulin et al. 2003a, Steinauer & Nickol 2003), and limited resources are generally assumed to be responsible for such observations.

In the high-intensity experimental infection, parasites appeared to reach an upper size limit defined by each host, and, as a consequence, parasite growth

was largely determined by resource availability (II). At more natural, lower intensities, however, *A. lucii* does not seem to exploit the intermediate host at a maximum level. For instance, A. *lucii* cystacanths sharing a host with one conspecific were on average smaller than those that did not share a host, i.e. single-cystacanth infections (V). However, the average size of parasites from double infections was reduced by less than half compared to parasites from single infections. This indicates that worms, in the absence of any competitors, do not reach the host's "resource ceiling", perhaps as an adaptive life history strategy (Parker et al. 2003b, Michaud et al. 2006). Such submaximal growth is particularly evident for male parasites; they grow to a much smaller size than females (Andryuk 1979). Even though larval *A. lucii* often exploit host resources below threshold levels, resource availability still seems to be an important determinant of parasite growth. In natural, single-worm infections, cystacanth volume was correlated with host size (III). The slope of this correlation, however, differed between male and female parasites. Female cystacanth size increased tightly with host size whereas this relationship had a much gentler slope for male cystacanths (III). This pattern suggests that females invest more into growth than males, and, consequently, their size is more resource-dependent than male size. Similar, sex-specific larval life history strategies have been observed in other acanthocephalans (Amin et al. 1980, Oetinger & Nickol 1982, Steinauer & Nickol 2003). This larval size dimorphism may also reflect differences in energy reserves, e.g. female *A. lucii* survived longer in vitro than males (III).

Sexual selection acting on adult parasites could be responsible for the divergence between male and female larval growth strategies. For female parasites, a large adult size may entail a fecundity advantage, but male size, on the other hand, may vary less predictably with reproductive success (Stearns 1992). This asymmetry in selective pressures presumably drives the evolution of adult size dimorphism (Shine 1989, Andersson 1994), and perhaps by association larval size dimorphism. Across acanthocephalan species, the sexual size dimorphism of adults and cystacanths was correlated (III). However, size dimorphism tends to be less pronounced in cystacanths than in adults, which may suggest that the level of sexual dimorphism attainable in intermediate hosts is constrained by resources (III). Indeed, in the case of *A. lucii* the difference between the size of male and female cystacanths tended to increase as resource availability, i.e. host size, increased (III). The correlation between cystacanth and adult sexual size dimorphism was weaker when using phylogenetically independent contrasts (III). This suggests that related species exhibit similar dimorphism levels and phylogeny may constrain, to some degree, the evolution of acanthocephalan life history (Poulin et al. 2003b).

3.2 Alteration of host phenotype

Five traits were compared between uninfected isopods and isopods harboring *A. lucii* cystacanths: hiding, activity, substrate color preference, body (pereon) coloration, and abdominal (pleon) coloration. Infected isopods tended to spend less time hiding under a leaf shelter and they had darker abdominal pigmentation than uninfected isopods (IV). The other three traits were apparently unaffected by *A. lucii* infection (IV). The darkened opercula of infected isopods (Brattey 1983) are presumably responsible for their overall darker abdominal coloration. Other *Acanthocephalus* species also alter their intermediate host's pigmentation, either increasing (Lyndon 1996) or decreasing it (Oetinger & Nickol 1981). Unlike other *Acanthocephalus* species (Muzzall & Rabalais 1975, Camp & Huizinga 1979, Hetchtel et al. 1993, Lyndon 1996), however, *A. lucii* was not known to alter the behavior of its intermediate host. Both the behavioral and visual dimension of isopod alteration could increase the probability of *A. lucii* transmission (Brattey 1983, Bakker et al. 1997).

Isopod hiding behavior was initially recorded on a scale of 1 hour. A subsample of isopods was observed a second time under the same conditions to evaluate whether this experimental setup was repeatable. Successive observations on the hiding behavior of individual isopods were not similar, i.e. the experiment had low measurement repeatability (IV). Thus, the 1-hr experiment did not seem to yield individually representative values of isopod hiding behavior. Two additional experiments were conducted to assess whether hiding behavior could be measured with some level of repeatability. Isopods were observed for longer periods of time (8 hrs and 8 wks), so as to capture a larger portion of each individual's behavioral variability. Extending the period of observation from 1 to 8 hrs did not produce repeatable measurements, but isopod hiding behavior was relatively consistent within individuals over 8 weeks of observation (IV). Thus, the hiding experiment conducted on the scale of weeks presumably yielded more individually representative measurements of isopod behavior than the shorter-scale experiments.

The two traits that were found to differ between infected and uninfected isopods appear to be unrelated. There was no correlation between isopod hiding and abdominal pigmentation, regardless of the experimental setup used to measure hiding behavior (IV). This may suggest these two traits are manipulated via independent mechanisms that are unconstrained by potential trade-offs (Cézilly & Perrot-Minnot 2005). For the 1- and 8-hr experiments, however, confidence in this null relationship is undermined by the low repeatability with which hiding behavior was measured. Recorded trait values may or may not be representative of individual averages, i.e. there is probably considerable noise in the data. For the isopods observed 8 weeks, though, there was also no correlation between the two manipulated traits (IV). Because this longer experiment seemed to measure individual hiding behavior relatively representatively, this null correlation indicates, less equivocally, that hiding and coloration are unrelated. Thus, these traits could have originated via separate,

positive effects on transmission (Bakker et al. 1997). Moreover, if these traits are unrelated, selection could act on the magnitude of each trait independently.

Intra-individual variation in isopod behavior was observed in all three hiding experiments. The apparently stochastic, short-term variation in isopod behavior probably contributed to the negligible measurement repeatability of the 1- and 8-hr experiments (IV). The hiding behavior of individual isopods also varied in the 8-week experiment, but, unlike the apparently random fluctuations observed on a short scale, changes in isopod behavior were directional on the scale of weeks. In general, the time isopods spent exposed tended to increase over time (IV). While this trend leveled off for the uninfected isopods, the proportion of time infected isopods spent exposed continued to increase throughout the experiment (IV). As a consequence of these patterns, the difference between infected and uninfected isopod hiding behavior was largest at the end of the 8-week experiment. Though acclimation to laboratory conditions may explain some of this variation, parasites may also change their host manipulation strategy over time.

3.3 Parasite life history and host manipulation strategies

It remains to be established whether the temporal decrease in the hiding behavior of infected isopods actually increases the likelihood of parasite transmission. Nonetheless, there are two factors which probably increase the favorability associated with parasite transmission over time. First, the likelihood of host death and failed parasite transmission presumably increases over time (Fig. 3A). Second, the possibility for continued parasite growth in the intermediate host diminishes over time, i.e. parasite growth slows as resources/space become limited (II, Fig. 3B). Thus, the likely benefits associated with remaining in the intermediate host (continued growth) decrease with time, while, concomitantly, the potential costs (likelihood of host death) increase. This increasingly unfavorable cost/benefit ratio probably encourages increased expression of parasite traits related to transmission, i.e. host manipulation.

Thus, the temporal changes in the hiding behavior of infected isopods could reflect adaptive plasticity in host manipulation by *A. lucii*. If this is the case, other manipulated traits would presumably vary with time in a similar manner. Time, however, is probably only an indirect determinant of the cost/benefit ratio associated with transmission. For example, parasites in large hosts, which are perhaps older and more likely to die, may have more incentive to be transmitted than those in small hosts, even if the infections are the same age. Thus, the notion that *A. lucii* flexibly manipulates host phenotype to optimize fitness, would be more strongly supported if manipulative effort varies with factors representative of transmission profitability (Thomas et al. 2002). Infected isopods exhibited not only altered hiding behavior, but also darker abdominal coloration (IV). The photographic method for measuring isopod coloration was highly repeatable (IV, V). Consequently, the acquired

coloration values were presumably representative for individual hosts, thereby permitting the sources of between-host variation in this trait to be explored.

Given that continued growth is presumably the major benefit for parasites staying in the intermediate host, the relationship between isopod coloration and several factors affecting parasite growth, i.e. host size (II, III), parasite sex (III), and competition (II), were assessed. In naturally-infected isopods, abdominal coloration tended to become darker in larger hosts (V). Larger isopods usually harbor larger parasites (II, III), which suggests host pigment alteration increases with parasite growth. Moreover, small isopods infected with a male parasite tended to have darker abdominal pigmentation than those infected with a female parasite, but this difference was absent in larger hosts (V). Sexual divergence in host alteration may be a consequence of the unique growth strategies adopted by males and females (III). Females, given their larger size, may have more to gain than males by remaining in and growing mutually with small hosts. In the high-intensity experimental infection (I, II), isopods harboring a larger worm biomass tended to have darker operculae, particularly if this worm biomass was distributed among fewer individuals (V). This observation also suggests the alteration of host pigmentation increases with parasite growth. The apparent connection between parasite growth and host manipulation, however, may be modified in multiple infections. In naturally-collected, 2-cystacanth infections, parasites were smaller than expected, yet this did not necessarily entail reduced modification of host coloration relative to single-cystacanth infections (V). This may indicate the alteration of host coloration by individual parasites is additive.

As larval *A. lucii* grew over time, the alteration of both host hiding behavior and host coloration seemed to increase in magnitude (IV, V). Assuming that it becomes less and less worthwhile to remain in the intermediate host over time (Fig. 3), these alterations could reflect adaptive plasticity in the transmission strategy of *A. lucii*. An ecological consequence of such a strategy may be that large, heavily manipulated hosts are taken more easily by predators, resulting in higher parasite abundance in hosts of medium size. Indeed, in at least one *A. lucii* population, parasite abundance peaks in intermediate-sized isopods and is reduced in large isopods (Brattey 1986). The circumstances presumably favoring the manipulation strategy of *A. lucii* exist in other trophically-transmitted parasites: parasite growth must eventually slow or stop and the probability of host mortality must ultimately increase. Thus, increasing host manipulation over time might be a common parasite strategy. Though other helminth species exhibit distributions in their intermediate host population that are similar to *A. lucii*, i.e. abundance peaks in mid-sized hosts (Thomas et al. 1995, Rousset et al. 1996), it is not known whether these parasites modify intermediate host phenotype in a flexible manner.

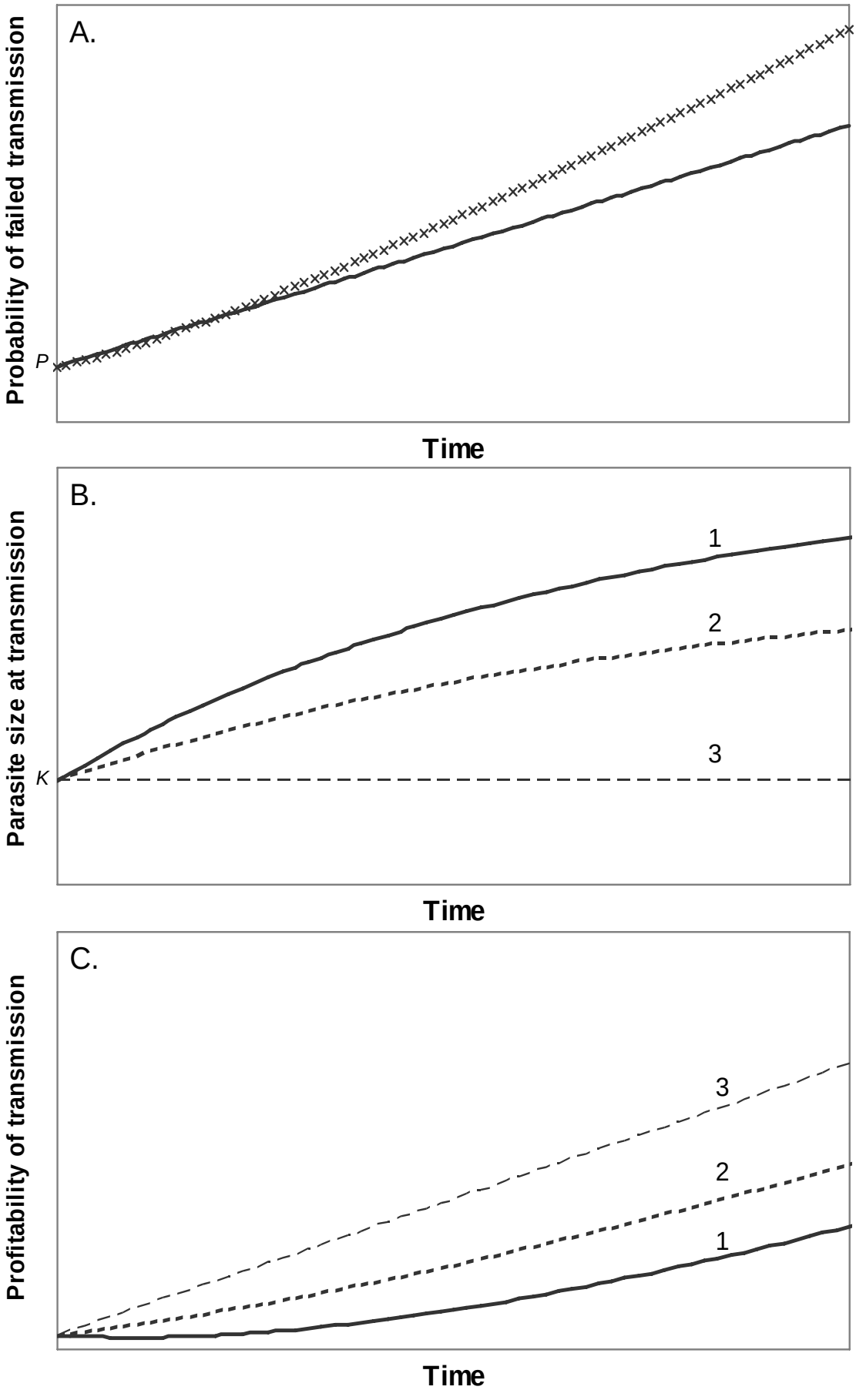
A.
Probability of failed transmission
P
Time
B.
Parasite size at transmission
1
2
3
K
Time
C.
Profitability of transmission
3
2
1
Time

FIGURE 3 Theoretical considerations on how different larval growth strategies may affect the incentive for parasites to be transmitted to the next how in the life cycle. (A) When t=0, the probability of parasites failing to be transmitted, e.g. through natural host mortality, is *p*. The value of *p* may increase in a linear fashion if the host mortality rate is constant (solid line) or it may vary non-linearly if host mortality is age-dependent (e.g. if older hosts have a particularly high likelihood of mortality, x-ed line). (B) When t=0, three different parasites become infective at a size *k*, but their growth patterns diverge thereafter. Parasite 1 grows toward a high asymptotic size, parasite 2 approaches a lower asymptotic size, and parasite 3 stops growing completely. (C) Assuming that parasite fitness increases with size at transmission (*k*) and that the probability of failed transmission (*p*) determines how potentially costly it is to stay in the intermediate host, the ratio of *p* to *k* can indicate how favorable parasite transmission is at a given time. With the assumption that *p* increases linearly over time, the ratio of *p* to *k* (profitability of transmission) was graphed for the three different parasite growth patterns in B. At t=0, the fitness costs associated with *p* were assumed to be higher than the fitness benefits associated with *k*, thereby resulting in positive transmission favorability (which could also be a trigger to become infective). For parasite 1, the incentive to be transmitted increases slowly initially because considerable parasite growth is still possible. Parasite 2, by contrast, reaches a smaller size and has less incentive to remain in the intermediate host. Finally, for parasite 3, it is only costly to remain in the intermediate host after reaching infectivity, so the profitability of transmission increases at a constant rate. These theoretical profitability functions could dictate how parasites express traits related to transmission, such as the manipulation of host phenotype.

The details of a parasite's growth strategy may dictate how profitable transmission to the next host is (Fig. 3). Unlike *A. lucii*, for example, many parasites exhibit relatively fixed growth strategies. After developing to an infective stage, growth stops. For these species, there may be no additional benefits, only costs, associated with remaining in the intermediate host after infectivity is reached, and this could favor discrete changes in the level of host manipulation (e.g. Bethel & Holmes 1974, Pulkkinen et al. 2000, Seppälä et al. 2005). Thus, different parasite growth patterns can seemingly lead to different host manipulation strategies (Fig. 3). However, it is important to note that while the profitability of parasite transmission presumably increases with time, the level of host manipulation need not respond accordingly. The optimal magnitude of host manipulation depends on the relationship between the level of host alteration and the probability of parasite transmission as well as the costs this manipulative effort entails (Poulin 1994a). For example, if a large increase in host manipulation yields no increase in transmission probability, there may be no selection for elevated manipulative effort, regardless of how the profitability of transmission is changing over time.

4 CONCLUSIONS

For complex-life cycle parasites, intermediate hosts serve as both an energy source and as transportation to the next host in the life cycle. Connections between these two functions have long been recognized. The evolution of parasite virulence, for instance, is assumed to reflect a balance between the benefits of parasite growth and the cost of reducing host viability (Anderson & May 1982). In the case of *A. lucii*, rapid larval growth may decrease intermediate host survival (I, II). However, in later infections the size of *A. lucii* seems to be primarily determined by resource availability (II, III), and the slow, apparently constrained growth of parasites may have few consequences for host survival (I). Therefore, parasite growth at this stage, rather than directly reducing host viability, may be important in dictating how favorable it is to move to the next host. As parasites grow larger and hosts become older, it becomes less and less worthwhile to stay in the intermediate host, presumably favoring increased investment in traits related to transmission such as host manipulation. Perhaps accordingly, the overall level of host manipulation by *A. lucii* (both altered host hiding and abdominal coloration) seemed to increase over time as parasites grew larger (IV, V). The apparent interconnection between parasite size and host manipulation suggests that the factors affecting parasite life history also influence transmission strategies. For example, sexual selection acting on adult life history may promote sexually divergent growth patterns and, by association, manipulation strategies (III, V). Therefore, untangling the multitude of factors shaping parasite life history (e.g. host size, host life-span, host defenses, intraspecific parasite competition, parasite transmission rates, sexual selection, phylogenetic constraints) is necessary to understand the diversity of intermediate host exploitation strategies exhibited by helminth parasites.

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YHTEENVETO (RÉSUMÉ IN FINNISH)

Väkäkärsämatotoukkien elinkierto- ja transmissiostrategiat sekä väli-isännän hyväksikäytön evoluutio

Loiset, joilla on usean isännän elinkierto, käyttävät väli-isäntiään sekä energialähteenä että keinona siirtyä seuraavaan isäntään. Ne loiset, jotka kasvavat nopeasti isokokoiseksi, voivat saavuttaa korkean kelpoisuuden (esim. aika, jolloin loinen ei vielä kykene infektoimaan isäntää, jää mahdollisimman lyhyeksi). Toisaalta loiset, jotka kasvavat liian aggressiivisesti, saattavat alentaa isännän elinkykyä ja samalla loisen omaa trasmissiotodennäköisyyttä (selviämistä seuraavaan isäntään). Olen tutkinut yhden väkäkärsämatolajin (*Acanthocephalus lucii*) kasvuun ja transmissiostrategioihin liittyviä tekijöitä vesisiirassa, joka on loisen 1. väli-isäntä. Kokeellisessa infektiossa loisen toukkien suhteellinen kasvu hidastui aikaa myöten ja loiset näyttivät saavuttavan kynnysbiomassan, jonka siiraisäntä pystyy kestämään. Tästä johtuen infektion myöhemmässä vaiheessa loisen kasvu näyttäisi olevan riippuvainen kulloisenkin isännän resurssien määrästä. Nuorten *A. lucii* -loisten nopea ja rajoittamaton kasvu näyttäisi olevan haitallisempaa isännän elinkyvylle kuin isompien loisten hidas ja isännän resursseista riippuvainen kasvu. *A. lucii* -loisnaaraat kasvoivat suuremmiksi kuin koiraat, ja niiden koko riippui voimakkaammin isännän koosta. Tämä voisi tarkoittaa sitä, että naaraat investoivat enemmän kasvuun, jolloin isännän resurssien määrä rajoittaa niitä enemmän kuin koiraita. Seksuaalinen dimorfismi (naaraat kookkaampia kuin koiraat) on yleinen piirre väkäkärsämatolajeilla. Siten seksuaalivalinta, joka ylläpitää aikuisten loisten dimorfismia, saattaa suosia sukupuolten erilaistumista myös väkäkärsämatojen toukkavaiheissa. *A. lucii* -loisen infektoimat siirat, joissa loinen on kehittynyt infektiiviseksi, viettävät vähemmän aikaa piiloutuneena ja ovat tummempia vatsapuoleltaan kuin loisettomat siirat. Näiden kahden mitatun ominaisuuden voimakkuus ei kuitenkaan korreloinut, ei edes silloin kun molempia ominaisuuksia mitattiin toistuvasti samoista yksilöistä. Loisten aikaansaama isännän piirteiden muuttuminen lisääntyi ajan myötä loisten kasvaessa. Kahdeksan viikon havainnoinnin aikana infektoitujen siirojen suojapaikkojen käyttö väheni ja loisittujen siirojen väritys tummui loisten kasvun myötä. Isännän kuoleman kasvava todennäköisyys ja vähenevä potentiaali loisen lisäkasvulle saattavat suosia voimakkaampaa loisen manipulaatiota isännän ilmiasua kohtaan.

REFERENCES

Anderson, R. M. & May, R. M. 1982. Coevolution of hosts and parasites. Parasitology 85: 373-398.

Andersson, M. 1994. Sexual selection. 624 p., Princeton University Press, Princeton.

Andryuk, L. V. 1979. Developmental cycle of the thorny-headed worm, Acanthocephalus lucii (Echinorhynchidae). Parazitologiia 13: 530-539. (in Russian).

Amin, O. M., Burns, L. A. & Redlin, M. J. 1980. The ecology of Acanthocephalus parksidei (Acanthocephala: Echinorhynchidae) in its isopod intermediate host. Proc. Helminthol. Soc. Wash. 47: 37-46.

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

Awachie, J. B. E. 1966. The development and life-history of Echinorhynchus truttae Schrank 1788 (Acanthocephala). J. Helminthol. 40: 11-32.

Bakker, T. C. M., Mazzi, D. & Zala, S. 1997. Parasite-induced changes in behavior and color make Gammarus pulex more prone to fish predation. Ecology 78: 1098-1104.

Barber, I. 2005. Parasites grow faster in faster growing fish hosts. Int. J. Parasitol. 35: 137-143.

Bethel, W. M. & Holmes, J. C. 1974. Correlation of altered evasive behavior in Gammarus lacustris (Amphipoda) harboring cystacanths of Polymorphus paradoxus (Acanthocephala) with the infectivity to the definitive host. J. Parasitol. 60: 272-274.

Bethel, W. M. & Holmes, J. C. 1977. Increased vulnerability of amphipods to predation owing to altered behavior induced by larval acanthocephalans. Can. J. Zool. 55: 110-115.

Brattey, J. 1983. The effects of larval Acanthocephalus lucii on the pigmentation, reproduction and susceptibility to predation of the isopod Asellus aquaticus. J. Parasitol. 69: 1172-1173.

Brattey, J. 1986. Life history and population biology of larval Acanthocephalus lucii (Acanthocephala: Echinorhynchidae) in the isopod Asellus aquaticus. J. Parasitol. 72: 633-645.

Brattey, J. 1988. Life history and population biology of adult Acanthocephalus lucii (Acanthocephala: Echinorhynchidae). J. Parasitol. 74: 72-80.

Brown, S. P., Loot, G., Grenfell, B. T. & Guégan, J.-F. 2001a. Host manipulation by Ligula intestinalis: accident or adaptation? Parasitology 123: 519-529.

Brown, S. P., Renaud, F., Guégan, J.-F. & Thomas, F. 2001b. Evolution of trophic transmission in parasites: the need to reach a mating place? J. Evol. Biol. 14: 815-820.

Camp, J. W. & Huizinga, H. W. 1979. Altered color, behavior, and predation susceptibility of the isopod Asellus intermedius infected with Acanthocephalus dirus. J. Parasitol. 65: 667-669.

Cézilly, F. & Perrot-Minnot, M.-J. 2005. Studying adaptive changes in the behaviour of infected hosts: a long and winding road. Behav. Processes 68: 223-228.

Choisy, M., Brown, S. P., Lafferty, K. D. & Thomas, F. 2003. Evolution of trophic transmission in parasites: why add intermediate hosts? Am. Nat. 162: 172-181.

Chubb, J. C. 1982. Seasonal occurrence of helminths in freshwater fishes. Part IV. Adult Cestoda, Nematoda, and Acanthocephala. Adv. Parasitol. 20: 1-292.

Davies, S. J. & McKerrow, J. H. 2003. Developmental plasticity in schistosomes and other helminths. Int. J. Parasitol. 33: 1277-1284.

Dawkins, R. 1982. The extended phenotype. 336 p., W. H. Freeman, Oxford.

de Meeûs, T. & Renaud, F. 2002. Parasites within the new phylogeny of eukaryotes. Trends Parasitol. 18: 247-251.

Dezfuli, B. S., Giari, L. & Poulin, R. 2001. Costs of intraspecific and interspecific host sharing in acanthocephalan cystacanths. Parasitology 122: 483-489.

Dobson, A. P. 1988. The population biology of parasite-induced changes in host behavior. Q. Rev. Biol. 63: 139-165.

Duclos, L. M., Danner, B. J. & Nickol, B. B. 2006. Virulence of Corynosoma constrictum (Acanthocephala: Polymorphidae) in Hyalella azteca (Amphipoda) throughout parasite ontogeny. J. Parasitol. 92: 749-755.

Ebenman, B. 1992. Evolution in organisms that change their niches during the life cycle. Am. Nat. 139: 990-1021.

Ebert, D. & Herre, E. A. 1996. The evolution of parasitic diseases. Parasitol. Today. 12: 96-101.

Frank, S. A. 1996. Models of parasite virulence. Q. Rev. Biol. 71: 37-78.

Fredensborg, B. L. & Poulin, R. 2005. Larval helminths in intermediate hosts: does competition early in life determine the fitness of adult parasites? Int. J. Parasitol. 35: 1061-1070.

Fredensborg, B. L., Mouritsen, K. M. & Poulin, R. 2004. Intensity-dependent mortality of Paracalliope novizealandiae (Amphipoda: Crustacea) infected by a trematode: experimental infections and field observations. J. Exp. Mar. Biol. Ecol. 311: 253-265.

Gemmill, A. W., Skorping, A. & Read, A. F. 1999. Optimal timing of first reproduction in parasitic nematodes. J. Evol. Biol. 12: 1148-1156.

Gleason, L. N. 1989. Movement of Pomphorhynchus bulbocolli larvae from the hemocoel to the peripheral circulation of Gammarus pseudolimnaeus. J. Parasitol. 75: 982-985.

Hasu, T., Valtonen, E. T. & Jokela, J. 2006. Costs of parasite resistance for female survival and parental care in a freshwater isopod. Oikos 114: 322-328.

Hasu, T., Holmes, J. C. & Valtonen, E. T. 2007. Isopod size (Asellus aquaticus) and acanthocephalan (Acanthocephalus lucii) infections. J. Parasitol. 93: 450-457.

Hetchtel, L. J., Johnson, C. L. & Juliano, S. A. 1993. Modification of antipredator behavior of Caecidotea intermedius by its parasite Acanthocephalus dirus. Ecology 74: 710-713.

Hindsbo, O. 1972. Effects of Polymorphus (Acanthocephala) on colour and behaviour of Gammarus lacustris. Nature 238: 333.

Holmes, J. C. & Zohar, S. 1990. Pathology and host behaviour. In: Barnard, C. J. & Behnke, J. M. (eds.), Parasitism and host behaviour: 34-63. Taylor and Francis, London.

Hurd, H., Warr, E. & Polwart, A. 2001. A parasite that increases host lifespan. Proc. R. Soc. Lond., B, Biol. Sci. 268: 1749-1753.

Hynes, H. B. N. & Nicholas, W. L. 1958. The resistance of Gammarus spp. to infection by Polymorphus minutus (Goeze, 1782) (Acanthocephala). Ann. Trop. Med. Parasitol. 52: 376-383.

Kuris, A. 1997. Host behavior modification: an evolutionary perspective. In: Beckage, N. E. (ed.), Parasites and pathogens, effects on host hormones and behaviour: 231-245. Chapman and Hall, New York.

Lackie, J. M. 1972. The course of infection and growth of Moniliformis dubius (Acanthocephala) in the intermediate host Periplaneta americana. Parasitology 64: 95-106.

Lafferty, K. D. & Kuris, A. M. 2002. Trophic strategies, animal diversity, and body size. Trends Ecol. Evol. 17: 507-513.

Lyndon, A. R. 1996. The role of acanthocephalan parasites in the predation of freshwater isopods by fish. In: Greenstreet, S. P. R. & Tasker, M. L. (eds.), Aquatic predators and their prey: 26-32. Blackwell Scientific, Oxford.

Metcalfe, N. B. M. & Monaghan, P. 2001. Compensation for a bad start: grow now, pay later? Trends Evol. Ecol. 16: 254-260.

Michaud, M., Milinski, M., Parker, G. A. & Chubb, J.C. 2006. Competitive growth strategies in intermediate hosts: experimental tests of a parasite life-history model using the cestode, Schistocephalus solidus. Evol. Ecol. 20: 39-57.

Moore, J. 1983. Responses of an avian predator and its isopod prey to an acanthocephalan parasite. Ecology 64: 1000-1015.

Moore, J. 2002. Parasites and the behavior of animals. 315 p., Oxford University Press, Oxford.

Morand, S. & Poulin, R. 2003. Phylogenies, the comparative method, and parasite evolutionary ecology. Adv. Parasitol. 54: 281-302.

Mouritsen, K. M. & Poulin, R. 2003. Parasite-induced trophic facilitation exploited by a non-host predator: a manipulator's nightmare. Int. J. Parasitol. 33: 1043-1050.

Muzzall, P. M. & Rabalais, F. C. 1975. Studies on Acanthocephalus jacksoni Bullock, 1962 (Acanthocephala: Echinorhynchidae). III. The altered behavior of Lirceus lineatus (Say) infected with cystacanths of Acanthocephalus jacksoni. Proc. Helminthol. Soc. Wash. 42: 116-118.

Nickol, B. B. & Dappen, G. E. 1982. Armadillidium vulgare (Isopoda) as an intermediate host of Plagiorhynchus cylindraceus (Acanthocephala) and isopod response to infection. J. Parasitol. 68: 570-575.

Nie, P. & Kennedy, C. R. 1993. Infection dynamics of larval Bothriocephalus claviceps in Cyclops vicinus. Parasitology 106: 503-509.

Oetinger, D. F. & Nickol, B. B. 1981. Effects of acanthocephalans on pigmentation of freshwater isopods. J. Parasitol. 67: 672-684.

Oetinger, D. F. & Nickol, B. B. 1982. Developmental relationships between acanthocephalans and altered pigmentation in freshwater isopods. J. Parasitol. 68: 463-469.

Parker, G. A., Chubb, J. C., Ball, M. A. & Roberts, G. N. 2003a. Evolution of complex life cycles in helminth parasites. Nature 425: 480-484.

Parker, G. A., Chubb, J. C., Roberts, G. N., Michaud, M. & Milinski, M. 2003b. Optimal growth strategies of larval helminths in their intermediate hosts. J. Evol. Biol. 16: 47-54.

Perrot-Minnot, M.-J. 2004. Larval morphology, genetic divergence, and contrasting levels of host manipulation between two forms of Pomphorhynchus laevis (Acanthocephala). Int. J. Parasitol. 34: 45-54.

Perrot-Minnot, M.-J., Kaldonski, N. & Cézilly, F. 2007. Increased susceptibility to predation and altered anti-predator behaviour in an acanthocephalan-infected amphipod. Int. J. Parasitol. 37: 645-651.

Pilecka-Rapacz, M. 1986. On the development of acanthocephalans of the genus Acanthocephalus Koelreuther, 1771, with special attention to their influence on intermediate host, Asellus aquaticus L. Acta Parasitol. Pol. 30: 233-250.

Poulin, R. 1994a. The evolution of parasite manipulation of host behaviour: a theoretical analysis. Parasitology 109 (Supl.): 109-118.

Poulin, R. 1994b. Meta-analysis of parasite-induced behavioural changes. Anim. Behav. 48: 137-146.

Poulin, R. 1995. "Adaptive" changes in the behaviour of parasitized animals: a critical review. Int. J. Parasitol. 25: 1371-1383.

Poulin, R. 2003. Information about transmission opportunities triggers a life-history switch in a parasite. Evolution 57: 2899-2903.

Poulin, R. 2007. Evolutionary ecology of parasites, 2nd edition. 332 p., Princeton University Press, Princeton, NJ, USA.

Poulin, R. & Morand, S. 2000. Testes size, body size and male-male competition in acanthocephalan parasites. J. Zool. 250: 551-558.

Poulin, R., Nichol, K. & Latham, A. D. M. 2003a. Host sharing and host manipulation by larval helminthes in shore crabs: cooperation or conflict? Int. J. Parasitol. 33: 425-433.

Poulin, R., Wise, M. & Moore, J. 2003b. A comparative analysis of adult body size and its correlates in acanthocephalan parasites. Int. J. Parasitol. 33: 799-805.

Pulkkinen, K., Pasternak, A. F., Hasu, T. & Valtonen, E. T. 2000. Effect of Triaenophorus crassus (Cestoda) infection on behavior and susceptibility to predation of the first intermediate host Cyclops strenuus (Copepoda). J. Parasitol. 86: 664-670.

Rauch, G., Kalbe, M. & Reusch, T. B. H. 2005. How a complex life cycle can improve a parasite's sex life. J. Evol. Biol. 18: 1069-1075.

Rosen, R. & Dick, T. A. 1983. Development and infectivity of the procercoid of Triaenophorus crassus Forel and mortality of the first intermediate host. Can. J. Zool. 61: 2120-2128.

Rousset, F., Thomas, F., de Meeûs, T. & Renaud, F. 1996. Inference of parasite-induced host mortality from distributions of parasite loads. Ecology 77: 2203-2211.

Schmidt, G. D. 1985. Development and life cycles. In: Nickol, B. B. & Crompton, D. W. T. (eds.), Biology of the Acanthocephala: 273-306. Cambridge University Press, Cambridge.

Seppälä, O., Karvonen, A. & Valtonen, E. T. 2005. Manipulation of fish host by eye flukes in relation to cataract formation and parasite infectivity. Anim. Behav. 70: 889-894.

Shine, R. 1989. Ecological causes for the evolution of sexual dimorphism: a review of the evidence. Q. Rev. Biol. 64, 419-461.

Shostak, A. W., Rosen, R. B. & Dick, T. A. 1985. The use of growth curves to assess the crowding effect on procercoids of the tapeworm Trianophorus crassus in the copepod host Cyclops bicuspidatus thomasi. Can. J. Zool. 63: 2343-2351.

Sorenson, R. E. & Minchella, D. J. 2001. Snail-trematode life history interactions: past trends and future directions. Parasitology 123: S3-S18.

Sparkes, T. C., Weil, K. A., Renwick, D. T. & Talkington, J. A. 2006. Development-related effects of an acanthocephalan parasite on pairing success of its intermediate host. Anim. Behav. 71: 439-448.

Stearns, S. C. 1992. The evolution of life histories. 262 p., Oxford university press, Oxford.

Steinauer, M. L. & Nickol, B. B. 2003. Effect of cystacanth body size on adult success. J. Parasitol. 89: 251-254.

Thomas, F., Renaud, F., Rousset, F., Cézilly, F. & de Meeûs, T. 1995. Differential mortality of two closely related host species induced by one parasite. Proc. R. Soc. Lond., B, Biol. Sci. 260: 349-352.

Thomas, F., Brown, S. P., Sukhdeo, M. & Renaud, F. 2002. Understanding parasite strategies: a state-dependent approach? Trends Parasitol. 18: 387-390.

Thomas, F., Adamo, S. & Moore, J. 2005. Parasitic manipulation: where are we and where should we go? Behav. Processes 68: 185-199.

Thompson, S. N. & Kavaliers, M. 1994. Physiological bases for parasite-induced alterations of host behaviour. Parasitology 109 (Supl.): 119-138.

Uznanski, R. L. & Nickol, B. B. 1980. Parasite population regulation: lethal and sublethal effects of Leptorhynchoides thecatus (Acanthocephala: Rhadinorhynchidae) on Hyalella azteca (Amphipoda). J. Parasitol. 66: 121-126.

Volkmann, A. 1991. Localization of phenoloxidase in the midgut of Periplaneta americana parasitized by larvae of Moniliformis moniliformis (Acanthocephala). Parasitol. Res. 77: 616-621.

Wedekind, C. 1997. The infectivity, growth and virulence of the cestode Schistocephalus solidus in its first intermediate host, the copepod Macrocyclops albidus. Parasitology 115: 317-324.

Williams, G. C. 1966. Adaptation and natural selection. 320 p., Princeton University Press, Princeton.

Windsor, D. A. 1998. Most of the species on earth are parasites. Int. J. Parasitol. 28: 1939-1941.

Zelmer, D. A. 1998. An evolutionary definition of parasitism. Int. J. Parasitol. 28: 531-533.

Zohar, S. & Holmes, J. C. 1998. Pairing success of male Gammarus lacustris infected by two acanthocephalans: a comparative study. Behavioral Ecology 9: 206-211.