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The effects of parasitism by the barnacle *Loxothylacus panopaei* (Gissler) (Cirripedia: Rhizocephala) on growth and survival of the host crab *Rhithropanopeus harrisii* (Gould) (Brachyura: Xanthidae)

Fernando Alvarez[a,*], Anson H. Hines[b], Marjorie L. Reaka-Kudla[a]

[a]*Department of Zoology, University of Maryland, College Park, MD 20742, USA*
[b]*Smithsonian Environmental Research Center, P.O. Box 28, Edgewater, MD 21307, USA*

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Abstract

The crab *Rhithropanopeus harrisii* (Gould) (Brachyura: Xanthidae) was infected in the laboratory with the parasitic barnacle *Loxothylacus panopaei* (Gissler) (Cirripedia: Rhizocephala). Crabs and barnacles were collected in the Rhode River in the Chesapeake Bay, MD. Forty-three out of 153 *R. harrisii*, including stages from megalopa (<1 mm of carapace width) to crab 8 stage (±8 mm of carapace width), developed a mature parasite (externa) after exposure to rhizocephalan cypris larvae. The duration of the internal phase of the parasite (from infection to the emergence of the externa) averaged 33 days and was independent of host size. Recently emerged externae (virgin externae) exposed to male cypris larvae, matured after fertilization in an average of 15 days. Host molting frequency and molt increments did not differ significantly between parasitized and control crabs. Survival to the crab 9 stage (±9.5 mm of carapace width) was 6% for hosts parasitized during the megalopal stage (<1 mm of carapace width), while it was 50% for the controls. The results of this study are discussed relative to the prevalences of *L. panopaei* found in the Chesapeake Bay.

Keywords: Chesapeake Bay; Crab; Host-parasite interaction; Parasitic castration; Rhizocephalan barnacle

1. Introduction

Rhizocephalan barnacles are all parasites, mainly of decapod crustaceans

* Corresponding author. Present address. Colección de Crustáceos, Instituto de Biología, Universidad Nacional Autónoma de México, A.P. 70–153, C.P. 04510, D.F. México.

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(Bocquet-Vedrine, 1972; Overstreet, 1983), which have a highly modified life cycle compared to free living barnacles. Rhizocephalans pass through two larval stages, nauplius and cypris, as the rest of barnacles, and present a gonochoristic condition (Hoeg 1991). The female cypris larva infects a susceptible host (in crabs, usually a recently molted organism) and develops as an endoparasite (internal phase) followed by the emergence of the reproductive body (externa) through the abdomen of the host, usually after host molting. Rhizocephalans can be described as macroparasites (sensu Anderson & May, 1981). From one to a few may be found on each host. After emergence, the externa has to be fertilized by male cypris larvae before maturation and reproduction ensue. From the moment of initial infection by the female cypris to the time where the externa emerges, the host undergoes a series of physiological and morphological changes, which include complete castration and cessation of growth (O'Brien & Van Wyk, 1984; Hoeg & Lützen 1985).

In most host species, rhizocephalans are found on hosts of intermediate size (Daugherty, 1969; O'Brien & Van Wyk, 1984, Hoggarth, 1990; Wardle & Tirpak, 1991), indicating that infection may occur exclusively in juvenile individuals. The pattern of reduced size in hosts has attracted considerable interest because of its effect in commercial fisheries species (Bishop & Cannon, 1979; Sloan, 1984; Weng, 1987) and because other allometric changes (often considered to be hormonally based) may accompany stunted growth (Reinhard, 1950; Nielsen, 1970; Hochberg et al., 1992). These instances of altered growth and allometry provide opportunities for examining the physiological mechanisms that control molting frequency (including premature anecdysis), relative growth, and molt increments (Andrieux et al., 1981; Herberts, 1982; O'Brien & Skinner, 1990).

In this study, experimental infections of the xanthid crab *Rhithropanopeus harrisii* (Gould) by the rhizocephalan barnacle *Loxothylacus panopaei* (Gissler) were performed in the laboratory. Duration of development (internal phase) and time to maturation (from emergence to first reproduction) of parasites infecting a range of host sizes were established. Molting frequency and molt increments for parasitized crabs were compared to those of controls. Host survival after infection was determined. Finally, the size distribution of parasitized *R. harrisii* in the field is discussed relative to the results presented here.

2. Materials and methods

Rhithropanopeus harrisii was collected in the Chesapeake Bay from the Rhode River at the Smithsonian Environmental Research Center. Plastic trays, containing oyster shell, were suspended from a dock and left to be colonized by *R. harrisii*. Routinely, ovigerous females and parasitized crabs were collected from these trays and transferred to the laboratory. Once in the laboratory, crabs were kept individually in plastic containers at a constant temperature of 24°C and under a 12 h day/12 h night photoperiod. Unfiltered water from the Chesapeake Bay was used throughout the experiments and its salinity was maintained at 15‰. To insure that no rhizocephalan larvae were present in the water used in the

laboratory, in order to prevent uncontrolled infections, the water was aereated for 7 days prior to being used. This period of time would be enough since the infective cypris larva develops in 48 h and remains infective for 4 days (Alvarez, 1993).

Ovigerous females of *R. harrisii* were monitored daily until the larvae became free swimming. Crab zoeae were cultured in groups of 100 in 300 ml plastic containers. Water was changed daily or every other day as needed. The zoeae were fed daily newly hatched *Artemia* nauplii. At the third crab stage, the *Artemia* nauplii diet was gradually replaced by adult *Artemia* and pellet food made of dried shrimp. Organisms on the fifth crab stage and older were maintained on pellet food only. Fifty four crabs obtained from these cultures were never exposed to rhizocephalan larvae and served as controls, while 156 others were used in the experiments. All crabs were measured with a vernier caliper every time they molted and the date was recorded. Parasitized crabs were maintained in a similar way and monitored daily for newly released rhizocephalan larvae. Each parasite brood was collected and placed into 300 ml plastic containers. Due to their lecithotrophic development, the rhizocephalan larvae were left undisturbed until they were presented with potential crab hosts.

Infection experiments were performed using crabs that had molted within 24 h prior to exposure, since intermolt crabs are not susceptible to infection (Alvarez, 1993). Exposures lasted for 12 h and utilized cypris larvae which were 2 days old, following the protocol of Walker et al. (1992). A minimum of 100 cypris larvae were used in every exposure. After the exposure period, the crabs were placed in clean water and routine care was resumed. The remaining cypris larvae were discarded. Both control and experimental crabs were monitored daily, although external signs of a successful infection usually are not visible until the crab molts again. Crabs ranging in size from <1 mm in carapace width (CW) (megalopae) to adults >10 mm in CW were used in these experiments.

Crabs bearing virgin parasite externae, which had been infected in the laboratory or collected in the field, were exposed to cypris larvae for 12 h for fertilization. Because virgin externae grow rapidly after fertilization, crabs collected in the field were observed for a minimum of 5 days to ensure that they had not been previously fertilized. The crabs were subjected to the same post-exposure care as above and were monitored daily for visible changes in growth of the externae of the parasites (signifying successful fertilization), until the first batch of larvae was released.

Data analysis comprised linear regression and comparison of regression slopes and elevations with analysis of covariance (ANCOVA). All crab measurements were taken in mm and mean values are followed by ± 1 SE.

3. Results

Forty-three out of 156 crabs exposed to cypris larvae were successfully infected and developed an externa (overall parasitization rate = 27.5%). Cypris larvae attached to the crabs in a number of sites: on the dorsal surface of the carapace,

inside the orbits and in the ocular peduncle, on all the segments of legs, along the suture lines of the thoracic segments, and on the segments of the second and third maxillipeds. Many cypris larvae entered the branchial chambers of the potential hosts, but it is uncertain whether or not these larvae attach and penetrate into the host. They can enter in sufficiently large numbers to suffocate and eventually kill the crab. Cypris larvae, once attached to the crab, remained in place and molted to the kentrogon stage (the last external stage before penetrating into the host) in a process that took about 3 days until injection of the parasite primordium occurred.

Crabs measuring 0.8 mm (megalopa stage) to 8.5 mm (crab 8 stage) were successfully infected. Development time of the parasite from infection to the emergence of the externa varied between 18 and 57 days ($\bar{X} = 33.5 \pm 2.1$ days) and was independent of host size ($r = -0.27$, $n = 29$, $p > 0.05$; Fig. 1).

Twenty-four crabs were followed from infection to the emergence of the parasite. The relationships between intermolt period (in days) versus premolt size were significant for both parasitized ($y = -1.571 + 4.432X$, $n = 48$, $r = 0.7987$, $p < 0.001$) and control crabs ($y = -3.651 + 5.145X$, $n = 54$, $r = 0.8479$, $p < 0.001$); the two regressions did not differ significantly (ANCOVA with premolt size as covariate; slopes, $F_{(1,100)} = 0.961$, $p > 0.05$; elevations, $F_{(1,101)} = 0.653$, $p > 0.05$; Fig. 2). Nine of the experimental crabs that were infected as megalopae (<1 mm) underwent 3 to 7 molts (average 5) until the parasite emerged. The remaining 15 crabs, representing crab instars 3 to 8 (where the normal intermolt period is longer, ≈1 month), molted one time, with the exception of a 3rd crab instar that molted 4 times before the externa of the parasite emerged.

The regressions of molt increments as a function of premolt size were significant for control ($n = 54$, $r = 0.589$, $p < 0.01$) and parasitized crabs ($n = 48$, $r = 0.349$, $p < 0.05$). Molt increments did not differ significantly between control and parasitized crabs (ANCOVA with premolt size as covariate; slopes $F_{(1,99)} = 0.032$, $p > 0.05$; elevations, $F_{(1,101)} = 1.16$, $p > 0.05$; Fig. 3). The size that was reached at

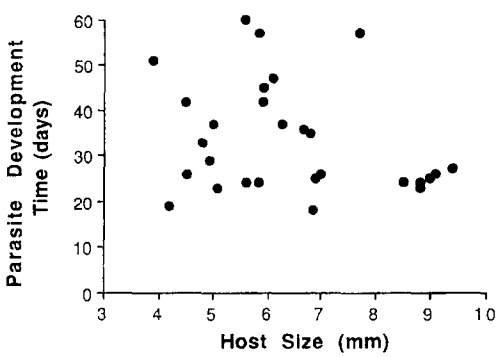

Fig. 1. Scattergram of development time of the internal phase (from infection to emergence of the externa) of *Loxothylacus panopaei*, in hosts of various sizes.

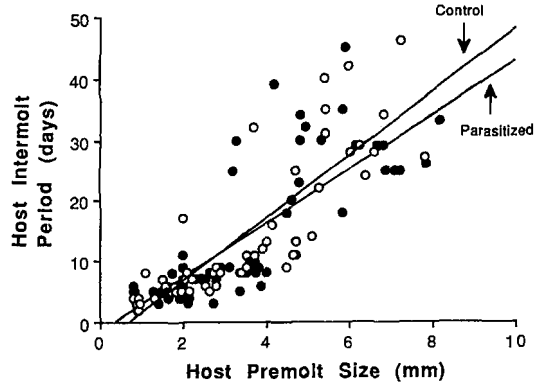

Fig. 2. Plot of intermolt period versus premolt size of *Rhithropanopeus harrisii*. ○, data from the control crabs; ●, parasitized crabs.

each instar, after the megalopal stage, was virtually the same for control and parasitized crabs (Fig. 4).

In order to fertilize the newly emerged parasites, 28 crabs bearing virgin externae were exposed to rhizocephalan cypris larvae. Six crabs died within 5 days from the time of exposure, and the parasites in the remaining 22 crabs matured and started to reproduce. The average maturation time (from fertilization until the first larvae were released) was 15.5 ± 0.8 days (range 12 to 22 days).

A set of 125 megalopae, 54 control and 71 experimental, was followed from infection until the crab 9 stage (145 days). First, survival was followed with respect to the crab stage that was reached. Twenty-eight (39.5%) of the 71 megalopae

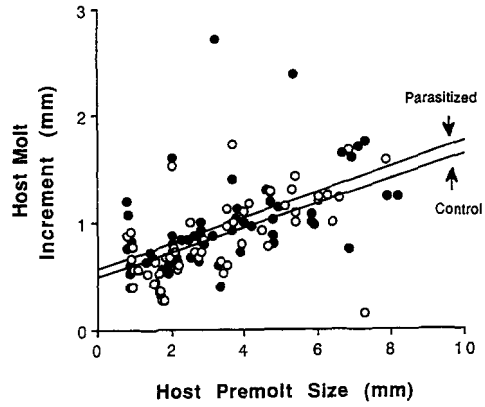

Fig. 3. Plot of molt increment versus premolt size of *Rhithropanopeus harrisii*. ○, data from the control crabs; ● parasitized crabs.

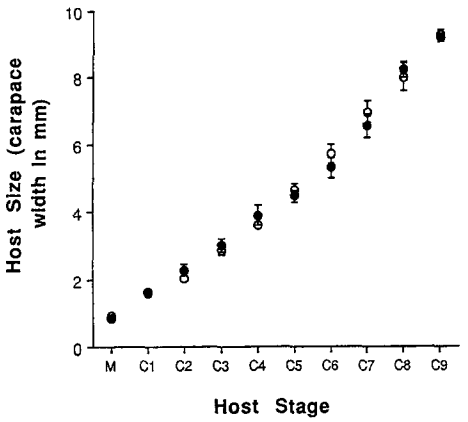

Fig. 4. Plot of average size that is reached at each developmental stage in *Rhithropanopeus harris* from megalopa (M) to crab 9 (C9) stage. ○, data from the control crabs; ●, parasitized crabs. T error bars correspond to plus or minus 1 SE.

exposed to rhizocephalan cypris died within 3 days of exposure. At the crab stage, the externa of the parasite emerged in 5 of the remaining 28 live cral (18%), in 4 of the remaining 21 live crabs (18%) at the crab 5 stage, and in 2 the remaining 13 crabs (15%) at the crab 7 stage (Fig. 5). Twenty-seven (50%) the 54 control crabs survived to the crab 9 stage, whereas only 4 (6%) of the exposed crabs reached this stage.

The high megalopal mortality of exposed crabs may be an artifact of the hig concentrations of rhizocephalan larvae to which they were exposed in order insure that infection would take place. To verify that megalopal mortality alor

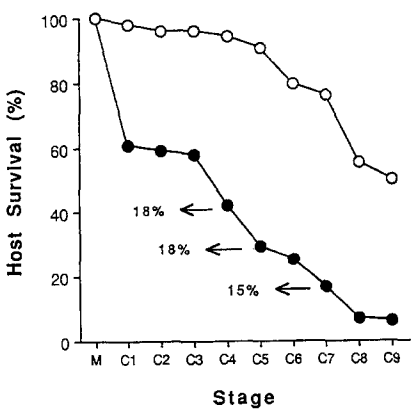

Fig. 5. Plot of survival of *Rhithropanopeus harrisii*, expressed as percentage of the total initial samp versus developmental stage. ○, data from the control crabs; ●, crabs that were exposed rhizocephalan larvae at the megalopal stage (M = megalopa, C1–C9 = crab 1 through crab 9). T arrows indicate the percentage of individuals which developed an externa at that stage.

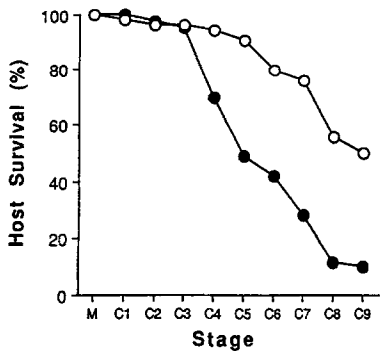

Fig. 6. Plot of survival of *Rhithropanopeus harrisii*, expressed as percentage of the total initial sample, versus developmental stage. ○, data from the control crabs; ●, crabs that were exposed to rhizocephalan larvae at the megalopal stage (M = megalopa, C1–C9 = crab 1 through crab 9). In this graph the igh megalopal mortality shown in Fig. 5 has been omitted.

was not causing the observed differences in ultimate survival, a second comparison was made using the number of exposed crabs surviving to the crab 1 stage as the initial number (Fig. 6). It can be seen graphically that a marked difference persists with respect to survival of the control and experimental crabs, and that the pronounced decrease in survival of exposed crabs begins in the crab 4 stage, when the externae of the parasite began to emerge (Fig. 6).

Second, survival is analyzed as a function of time. Megalopal mortality after exposure to cypris larvae again is pronounced. Sixty-one percent of the control crabs survived 145 days, while only 15.5% of the exposed crabs did. Separate linear regressions of percent survival for the control and experimental data sets were significant (control: $y = 102.2–0.308X$, $n = 30$, $r = -0.9683$, $p < 0.01$; experimental: $y = 69.19–0.41X$, $n = 30$, $r = -0.9394$, $p < 0.01$; Fig. 7). When compared, the slopes were not homogeneous (ANCOVA with time in days as covariate, $F_{(1,28)} = 5.988$, $p < 0.021$). When survival of parasitized crabs was corrected for megalopal mortality, the significant regression ($y = 88.99 - 0.509X$, $n = 29$, $r = -0.9851$, $p < 0.01$) also differed significantly from that of the control crabs (ANCOVA with time in days as covariate, $F_{(1,28)} = 41.209$, $p < 0.0001$).

Survival increased for crabs that were infected in more advanced stages of development. Thirty-four crabs, representing 5 stages (crab 4 to crab 8), were exposed to rhizocephalan cypris. Of these, 9 crabs (29.5%) died within 5 days of exposure, 20 crabs (26.5%) were infected and developed an externa at the next molt, and 15 crabs (44%) survived uninfected.

4. Discussion

Reduced sizes of crabs parasitized by rhizocephalan barnacles have been reported in a number of studies (i.e. Reinhard, 1956; Bocquet-Vedrine, 1972;

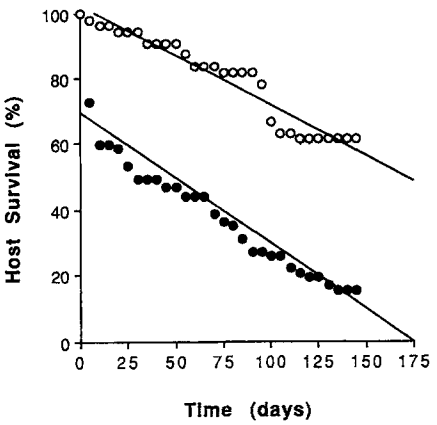

Fig. 7. Plot of survival of *Rhithropanopeus harrisii*, expressed as percentage of the total initial sample versus time in days. ○, data from the control crabs; ●, crabs that were exposed to rhizocephalan larvae at the megalopal stage (M = megalopa, C1–C9 = crab 1 through crab 9).

O'Brien & Van Wyk, 1984), but few studies have followed parasitized individuals through successive molts to observe the steps by which these changes occur. O'Brien (1984) demonstrated that the number of molts is reduced in parasitized versus control crabs (*Heterosaccus californicus* on the crab *Pugettia producta*) and Veillet (1945), Vernet-Cornubert (1958) (*Sacculina carcini* on *Carcinus maenas*), and Hawkes et al. (1987) (*Briarosaccus callosus* on *Paralithodes camtschatica*) showed that molt increments are smaller in parasitized than nonparasitized crabs.

In this study, the duration of the intermolt period and the increase in size in molt increments in control versus parasitized crabs did not differ significantly. As expected from these results, the size that is reached at each stage from megalopa to the crab 9 instar was virtually the same for control and parasitized crabs, corroborating the absence of an effect of the parasite on the growth pattern of the host. Thus, *L. panopaei* affects the size of *R. harrisii* primarily by producing anecdysis ("parasitic anecdysis", O'Brien & Van Wyk, 1984), whereas the crab normally would show indeterminate growth (Turoboyski, 1973).

"Precocious maturity" (described for the majid crab *Pugettia producta* parasitized by *Heterosaccus californicus*, O'Brien, 1984) does not occur in *R. harrisii* because the great majority of crabs bearing an externa already have reached the size at which sexual maturity is acquired. The length of the gonopods suggests that male *R. harrisii* become sexually mature between 4.0 and 4.5 mm. Setation of the pleopods and the minimum size at which they become ovigerous suggest that females become sexually mature between 4.5 and 5.0 mm (Ryan, 1956; Turoboyski, 1973). In both cases, these sizes coincide with the minimum size of parasitized crabs.

Development time of the parasite (from infection to emergence of the externa) was independent of host size and variable; the average of 33.5 ± 2 days is longer than the 26 days reported for *R. harrisii* megalopae only by Walker et al. (1992).

Considering that cypris larvae 2–6 days old are capable of infecting a crab, that the development time of the parasite averages 33 days, and that the externa mature in an average of 15 days after fertilization (Alvarez, 1993), a conservative estimate of *L. panopaei* generation time is 54 days. Since the internal development time of the parasite is roughly equal to the intermolt period (30 days) in crabs 4 mm and bigger, the average molt increment is about 1 mm, and the most heavily parasitized sizes of crabs in the field are 6 to 9 mm (Fig. 8), it can be estimated that the stages of *R. harrisii* most susceptible to infection are crab stages 5 to 8 (≈5–8 mm). Further, because *R. harrisii* of a wide range of sizes can be infected and because development time of *L. panopaei* is independent of host size, infection of juvenile crabs in this species does not shorten the generation time of the parasite, as is the case in other crab-rhizocephalan associations (O'Brien, 1984; O'Brien & Van Wyk, 1984).

The pattern of host size selectivity may be the result of a minimum viable host size for the parasite, below which the crab may be too small to sustain the energy demands of the parasite. In *R. harrisii*, the effects of rhizocephalan infection on growth of the crab host are not as clear as they are in other species where parasitized crabs are of intermediate size (O'Brien & Van Wyk, 1984). The first susceptible stage for infection in *R. harrisii* is the megalopa; during the development time (culminating in the emergence of the externa) of the parasite (≈1 month), the crab will reach 4 mm CW. Thus, 4 mm is the minimum crab size at which the parasite can be detected by the presence of an externa. Beyond the crab 4 stage (≈4 mm CW), the intermolt period of the crab increases to about 1 month (at 24°C); crabs at these stages will produce a rhizocephalan externa at the next molt. Crabs remain susceptible to infection to at least the crab 9 stage, and, thus, some of the largest crabs in the population may bear parasites (Fig. 8). Consequently, due to the wide range of susceptible stages and the relatively small size of adults of this host species, we do not observe in *R. harrisii* the typical pattern of parasitized crabs of intermediate size. The wide range of susceptible sizes in the populations of *R. harrisii* may account for the rapid spread of the

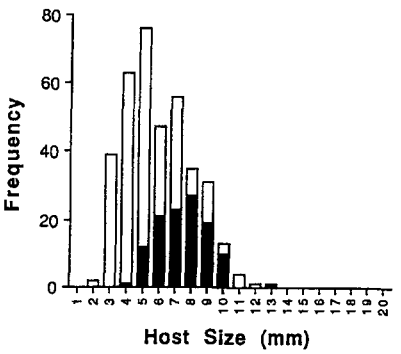

Fig. 8. Bar graph of the size frequency of *Rhithropanopeus harrisii* from the Rhode River, Maryland. Open bars represent unparasitized crabs and solid bars parasitized crabs.

parasite. For example, in the Rhode River, Chesapeake Bay, *L. panopaei* appeared on *R. harrisii* for the first time in 1989 but, by 1991, 72% of the host population was infected (Hines et al., 1995).

Parasitism of *L. panopaei* on *R. harrisii* had a significant effect on survival. Under laboratory conditions, a high initial mortality is observed after exposure of crabs to infective cypris larvae. This could be an artifact of the high concentrations of larvae to which the crabs were exposed in order to assure infection. As the cypris are attracted to the potential host, they enter the branchial chambers in great numbers, probably obstructing water flow and ultimately suffocating and killing the crab. Cypris larvae have been observed before in the branchial chamber of *R. harrisii* (Walker et al., 1992), but there is no evidence that cypris settlement occurs in the gills. As far as is known, settlement occurs exclusively on external areas at the base of setae and other soft areas of the carapace. After the initial exposure period, the next stage at which mortality increases is at the molt from crab 3 to crab 4, corresponding to the point where the first externae emerge. A distinction has to be made between mortality that occurs before the parasite becomes external and survival of crabs with externae. As shown here, a large proportion of crabs that survive the initial exposure to the larvae of the parasite, and that do not develop an externae still die. It is possible that they were infected, their physiological condition was poor, and they could not withstand the energy drain inflicted by the developing parasite. Due to this confounding effect, mortality due to parasitism during the internal phase of the parasite is probably underestimated. However, the crabs that do develop an externa generally survive for many months in the laboratory (Alvarez, 1993). Although it has not been formally quantified, similar observations of high survival of parasitized crabs have been made for other species parasitized by rhizocephalans (Foxon, 1940; O'Brien, 1984; Lutzen, 1987; Hoggarth, 1990). This may occur because only the most fit hosts survived the initial stages of infection, particularly in species where infection can occur in very small hosts.

This pattern of prevalence in smaller than average hosts also has important evolutionary implications. The first consequence for the parasites is that they do not reach a larger size. Since there is a positive correlation between host size and parasite size and between parasite size and number of larvae produced in the decapod-rhizocephalan system (Alvarez, 1993), it would appear that parasites are exploiting hosts in a suboptimal way if they occur on relatively small hosts. Published explanations for this pattern consider that rhizocephalan parasites may effectively shorten their generation time by decreasing the host's number of molts or molt increments, reducing the host's maximum size (O'Brien & Van Wyk, 1984), or that a spatial constraint may place younger crabs at a higher risk of infection than older crabs, since in estuarine species larval crabs may occupy the same habitat as the parasite's larvae (Walker et al., 1992). Regardless of the processes that originate this pattern, a size refuge is created where individuals that attain a certain size would thereafter be relatively free from parasitism. If so, in host species where rhizocephalan prevalence remains at high levels for prolonged periods of time, parasitism may represent a selective force for hosts to reach

larger sizes in a shorter period of time. Further, in sympatric, closely related host species that share the same parasite species (e.g. Alaskan king crabs, Hawkes et al., 1987), differences in maximum adult size may give those larger species an advantage, since in that case a larger proportion of the population can reach the size refuge.

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References

Alvarez, F., 1993. The interaction between a parasitic barnacle, *Loxothylacus panopaei* (Cirripedia: Rhizocephala), and three of its crab host species (Brachyura: Xanthidae) along the east coast of North America. Ph.D. Dissertation, University of Maryland, College Park, Maryland, pp. 1–188.

Anderson, R.M. & R.M. May, 1981. The population dynamics of microparasites and their invertebrate hosts. *Proc. Philos. Trans. R. Soc.*, Vol. 291, pp. 451–524.

Andrieux, N., C. Herberts & J. De Frescheville, 1981. Relations hote-parasite entre les crustacés *Carcinus* et *Sacculina carcini*: effet d'extraits de parasite et de l'hemolymphe de crabe infesté sur le protéinogramme de crabes sains. *Ann. Parasitol.*, Vol. 56, pp. 441–448.

Bishop, R.K. & L.R.G. Cannon, 1979. Morbid behaviour of the commercial sand crab, *Carcinus pelagicus* (L.), parasitized by *Sacculina granifera* Boschma, 1973 (Cirripedia: Rhizocephala). *J. Fish Dis.*, Vol. 2, pp. 131–144.

Bocquet-Vedrine, J., 1972. Les rhizocephales. *Cah. Biol. Mar.*, Vol. 13, pp. 615–626.

Daugherty, S.J., 1969. Aspects of the ecology, life history, and host-parasite relationship of *Loxothylacus panopaei* (Sacculinidae) in Chesapeake Bay. M.A. Thesis, College of William and Mary, Williamsburg, Virginia, 68 pp.

Foxon, G.E.H., 1940. Notes on the life history of *Sacculina carcini* Thompson. *J. Mar. Biol. Assoc. U.K.*, Vol. 24, pp. 253–264.

Hawkes C.R., T.R. Meyers & T.C. Shirley, 1987. Growth of Alaskan king crabs, *Paralithodes platypus* (Brandt), parasitized by the rhizocephalan *Briarosaccus callosus* Boschma. *Crustaceana*, Vol. 52, pp. 78–84.

Herberts, C., 1982. Host-parasite relation between the shore crab *Carcinus maenas* and *Sacculina carcini* (Rhizocephala): identification and characterization of a specific fraction correlated with parasitism. *J. Invertebr. Pathol.*, Vol. 39, pp. 60–65.

Hines, A.H., F. Alvarez & S.A. Reed, 1995. Geographic and temporal variation in parasitic castratin of xanthid crabs by the rhizocephalan cirripede *Loxothylacus panopaei*. Bull. Mar. Sci. (in press).

Hochberg, R.T., T.M. Bert, P. Steele & S.D. Brown, 1992. Parasitization of *Loxothylacus texanus* on *Callinectes sapidus*: aspects of population biology and effects on host morphology. *Bull. Mar. Sci.*, Vol. 50, pp.117–132.

Hoeg, J.T., 1991. Functional and evolutionary aspects of the sexual system in the Rhizocephala (Crustacea: Cirripedia: Rhizocephala). In, *Crustacean sexual biology*, edited by R. Bauer & J. Martin, Columbia University Press, New York, pp. 208–227.

Hoeg, J.T. & J. Lutzen, 1985. *Crustacea Rhizocephala.* Marine Invertebrates of Scandinavia, No. 6, Norweigan University Press.

Hoggarth, D.D., 1990. The effects of parasitism by the rhizocephalan, *Briarosaccus callosus* Boschma on the lithodid crab, *Paralomis granulosa* (Jacquinot) in the Falkland Islands. *Crustaceana*, Vol. 59, pp. 156–170.

Lutzen, J., 1987. Life history parameters calculated from growth rings in parasitic barnacles of the family Peltogasteridae (Crustacea: Cirripedia: Rhizocephala). *J. Crust. Biol.*, Vol. 7, pp. 493–506.

Nielsen, S.O., 1970. The effects of the rhizocephalan parasites *Peltogaster paguri* Rathke and *Gemmosaccus sulcatus* (Lilljeborg) on five species of paguridan hosts (Crustacea: Decapoda). *Sarsia*, Vol. 42, pp. 17–32.

O'Brien, J., 1984. Precocious maturity of the majid crab, *Pugettia producta*, parasitized by the rhizocephalan barnacle, *Heterosaccus californicus*. *Mar. Biol.*, Vol. 166, pp. 384–395.

O'Brien, J. & P. Van Wyk, 1984. Effects of crustacean parasitic castrators (epicaridean isopods and rhizocephalan barnacles) on growth of crustacean hosts. In, *Crustacean, Issues 3*, edited by A.M. Wenner, A.A. Balkema, Rotterdam, The Netherlands, pp. 191–218.

O'Brien, J. & D.E. Skinner, 1990. Overriding of molt-inducing stimulus of multiple limb autotomy in the mud crab *Rhithropanopeus harrisii* by parasitization with a rhizocephalan. *J. Crust. Biol.*, Vol. 10, pp. 440–445.

Overstreet, R.M., 1983. Metazoan symbionts of crustaceans. In, *The biology of the Crustacea, Vol. 6*, edited by A.J. Provenzano, Academic Press Inc., pp. 156–251.

Reinhard, E.G., 1950. An analysis of the effects of a sacculinid parasite on the external morphology of *Callinectes sapidus*. *Biol. Bull.*, Vol. 98, pp. 277–288.

Reinhard, E.G., 1956. Parasitic castration of Crustacea. *Exp. Parasitol.*, Vol. 5, pp. 79–107.

Ryan, E.P., 1956. Observations on the life histories and the distribution of the Xanthidae (mud crabs) of Chesapeake Bay. *Am. Midl. Nat.*, Vol. 56, pp. 138–162.

Sloan, N.A., 1984. Incidence and effects of parasitism by the rhizocephalan barnacle, *Briarosaccus callosus* Boschma, in the golden king crab, *Lithodes aequispina* Benedict, from deep fjords in northern British Columbia, Canada. *J. Exp. Mar. Biol. Ecol.*, Vol. 84, pp. 111–131.

Turoboyski, K., 1973. Biology and ecology of the crab *Rhithropanopeus harrisii* ssp. *tridentatus*. *Mar. Biol.*, Vol. 23, pp. 303–313.

Veillet, A., 1945. Recherches sur le parasitisme des crabes et des galathées par les rhizocéphales et les épicarides. *Ann. Inst. Océanogr.*, Vol. 22, pp. 193–341.

Vernet-Cornubert, G., 1958. Recherches sur la sexualité du crabe *Pachygrapsus marmoratus* (Fabricius). *Arch. Zool. Exp. Gén.*, Vol. 96, pp. 101–276.

Walker, G., A.S. Clare, D. Rittschof & D. Mensching, 1992. Aspects of the life-cycle of *Loxothylacus panopaei* (Gissler), a sacculinid parasite of the mud crab, *Rhithropanopeus harrisii* (Gould): a laboratory study. *J. Exp. Mar. Biol. Ecol.*, Vol. 157, pp. 181–193.

Wardle, W.J. & A.J. Tirpak, 1991. Occurrence and distribution of an outbreak of infection of *Loxothylacus texanus* (Rhizocephala) in blue crabs in Galveston Bay, Texas, with special reference to size and coloration of the parasite's external reproductive structures. *J. Crust. Biol.*, Vol. 11, pp. 553–560.

Weng, H.T., 1987. The parasitic barnacle, *Sacculina granifera* Boschma, affecting the commercial sand crab, *Portunus pelagicus* (L.), in populations from two different environments in Queensland. *J. Fish Dis.*, Vol. 10, pp. 221–227.