Journal of Mammalogy, 100(6):1831–1836, 2019
DOI:10.1093/jmammal/gyz136
Published online September 11, 2019

Stomach nematodes of cotton rats: parasites, commensals, or mutualists?

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We related presence and burden of stomach nematodes to body mass and reproductive allocation in hispid cotton rats (*Sigmodon hispidus*) from two long-running field studies in Virginia (1983–1984, $n = 286$; and 1988–1990, $n = 425$) and one from Georgia 1987–1989 ($n = 459$). Eighty percent of rats from the earlier Virginia sample were infected, with mean nematode mass of 1,311 mg. In the later samples, 23% (Virginia) and 33% (Georgia) were infected with mean nematode mass of 493 and 769 mg, respectively. Presence of nematodes was positively correlated with host body length for each sex in each sample. We used analysis of covariance to examine length-adjusted residuals for presence of nematodes and mass of nematodes for association with somatic and reproductive response variables. Both body and reproductive masses were either positively associated or not related to nematode presence in the two low-prevalence samples, and either negatively associated or not related to nematode presence in the high-prevalence sample. No relationships were detected between host mass and nematode mass per host in either sex in any sample. There was no effect of nematode presence on litter size of pregnant females, but there was a positive effect of nematode mass on litter size in Georgia. Recent theory provides several possible explanations for such neutral-to-positive effects of stomach nematodes on host fitness, including the evolution of host tolerance to the parasites, fecundity compensation by the hosts, and positive effects on host health via immune modulation.

Key words: body mass, fecundity compensation, host tolerance, immune modulation, *Mastophorus*, *Physaloptera*, reproductive allocation

Certain helminth parasites of mammals cause severe individual- and population-level effects in their hosts; examples include the lungworm *Protostrongylus* spp. in bighorn sheep (*Ovis canadensis*—Uhazy et al. 1973) and the brainworm *Parelaphostrongylus tenuis* in many New World cervids (though not *Odocoileus virginianus*—Anderson 1972). However, the term "parasite" sensu stricto may be improperly applied to certain alimentary canal-dwelling helminths in wild mammals, even though these endosymbionts are seemingly quite invasive, causing tissue damage by burrowing into the mucosa to extract nutrients from their host (Schell 1952). Regardless, net effects of these gut endosymbionts can be neutral or beneficial, involving such mechanisms as altering host responses to microbial pathogens, regulating the gut microbiome, and decreasing inflammatory disease through downregulation of the immune system (Maizels and Yahzdanbahksh 2003; Elliott and Weinstock 2012; Aivelo and Norberg 2018). Increasing specialization and adaptive radiation of parasites in a long-parasitized host may lead to co-adapted gene-complex pairs that reduce negative effects on primary hosts (Price 1980). The evolution of mammalian host tolerance to high parasite burdens and chronic infection with gut helminths was recently reviewed by Kutzer and Armitage (2016) and King and Li (2018).

Even if a parasite increases mortality of adult females, litter size at weaning may be greater in heavily parasitized mammalian hosts (Kristan 2004). This may be a manifestation of fecundity compensation, in which the reduction of residual reproductive value drives females toward greater reproductive allocation, i.e., greater emphasis on current fecundity (Schwanz 2008). Many female cotton rats (*Sigmodon hispidus*), especially at the northern edge of their distribution

where winters are more severe, are de facto semelparous because of their short life span and consequently have larger litters than females in lower-latitude populations (Bergstrom and Rose 2004). Any other causes of increased mortality, such as parasitism, could increasingly elicit fecundity compensation, varying with conditions over time and space, in a mammal that is capable of producing litters as large as 13 (Cameron and McClure 1988; Bergstrom and Rose 2004). Thus, there is a range of both strategies and tactics that a host with short generations and high fecundity could employ to either tolerate or mask the tendency for a tissue-invasive parasite to cause negative fitness effects, and it is possible that a parasite burden of moderate intensity could even increase host fitness through immune system benefits (Maizels and Yahzdanbahksh 2003).

Nematodes in the genera *Physaloptera* (Order Spirurida: Family Physalopteridae) and *Mastophorus* (Order Spirurida: Family Spirocercidae) are acquired by their definitive rodent hosts by the consumption of insects (ground beetles, earwigs, crickets, among others), which are their intermediate hosts (Schell 1952). Hispid cotton rats (*S. hispidus*, hereafter "cotton rats") are susceptible to these stomach nematodes (*Physaloptera hispida* and *Mastophorus muris*) because they sometimes ingest insects, probably incidental to their grazing (Kincaid and Cameron 1982). Once inside their definitive hosts, both species of these dioecious nematodes feed by rasping holes in the stomach wall, often aggregating in tight clusters that ulcerate the gastric mucosa (Schell 1952; Wertheim 1962). After several weeks to attain maturity and mate, females lay embryonated eggs that exit the host in its feces. These eggs, or their hatched larvae, are eaten by insects, molt to the infective stage, and are consumed by a compatible mammal, thus completing the life cycle (Schell 1952).

Although these two species of stomach nematodes of cotton rats (Harkema and Kartman 1948; Hugghins 1951; Schell 1952; Coggins 1972; Kinsella 1974; Briese and Smith 1980) also parasitize some other rodents in eastern North America (Kinsella 1988, 1991), little is known about their effects on the ecology and reproduction of their hosts. Here, we examine the effects of stomach nematode infection and burden on the somatic growth and reproductive allocation of their hosts from three long-running studies in which monthly field samples of cotton rats were obtained from the southeastern United States, one with very high prevalence (percentage of host population infected) and burden (mass per host) of gut nematodes from eastern Virginia in 1983–1984, and one each from southern Georgia and eastern Virginia in the late 1980s, in which parasite prevalences and burdens were substantially lower. We present results of general linear models for each sex and sample separately, while examining residual effects of nematode presence and nematode mass on body mass and reproductive mass of cotton rats, and on litter size of pregnant females (for two of the three samples), after removing the effect of body length as a covariate (the latter is a surrogate for age in cotton rats—Cameron and Spencer 1981).

Materials and Methods

Cotton rats ≥ 50 g (minimum mass for sexual maturity in our populations—Rose and Mitchell 1990; Bergstrom and Rose 2004) were collected each month from a range of sites in southeastern Virginia and southern Georgia. During the first Virginia sample (October 1983 to November 1984), cotton rats were trapped at a 440-ha old field undergoing secondary succession after removal of trees in 1980. The vegetation, dominated by bluestem (*Schizachyrium* spp.) and panic (*Panicum* spp.) grasses, ideal habitat for cotton rats, was neither mowed nor burned during this period of collection. All cotton rats from the later two samples (September 1987 to December 1989 in Georgia and January 1988 to January 1990 in Virginia) were trapped from several highway, railroad, and power-line rights of way, strips near an airport runway, and fallow farm fields in southeastern Virginia and south-central Georgia, habitats that were mowed at least annually.

Using Fitch live traps (33 × 6 × 6 cm with 355-ml can—Rose 1994) and Sherman traps (23 × 9 × 8 cm; H. B. Sherman Trap Co., Tallahassee, Florida), our goal was to collect a monthly sample of 10–15 cotton rats of each sex from each region, but we suspended trapping after 8 nights if we did not collect at least 10 rats in any month. However, month of collection was not a variable examined in this study, and all animals collected from each of the three samples described above were treated as a single sample. After collection, cotton rats were immediately taken to a university laboratory and euthanized with chloroform and either necropsied immediately or frozen for later necropsy and examination (when freshly thawed). Immediately after euthanasia, we weighed (g) and measured (mm) each cotton rat to determine body mass and total and tail lengths (mm). Subtracting tail length from total length gave us body length, which was our primary variable for size and a proxy for age. During necropsy, we removed and weighed (mg) testes and seminal vesicles for males, and the total of these two weights was recorded as reproductive mass for males. We removed and weighed the entire uterus (mg) for females, and this was recorded as reproductive mass for females. After weighing, we carefully dissected each uterus and counted the number of implanted embryos in both horns: this number constituted the litter size for each female found to be pregnant. After opening the stomach, we determined the aggregate mass of nematodes (mg) as a single measurement of fresh, wet mass for each rat.

Identification of *Physaloptera* spp. and *Mastophorus* spp. requires clearing in phenol or glycerin before microscopic examination of mouthparts and other features. Samples of nematodes from our studies were identified by specialists at Indiana University and Virginia Polytechnic Institute and State University as *P. hispida*, but nematodes from four animals sent to Louisiana State University were identified as *M. muris.* Both species are of similar size, life cycle, and mode of feeding. It is also possible that some of our rats may have harbored *Strongyloides* spp., a similar stomach nematode known to infect cotton rats elsewhere (Chandikumar et al. 1990; S. L. Gardner, University of Nebraska, pers. comm.). Hereafter, we use the term "stomach nematode" for these parasites.

We used ANOVA: general linear models to conduct analysis of covariance (ANCOVA), and also Pearson correlations and 2-sample *t*-tests, in the Minitab 17.3.1 statistical package (Minitab 2016). Before these analyses, Anderson–Darling statistics were generated in Minitab 17.3.1 for each variable and sample, which were then ln-transformed in cases where the sample distribution departed significantly from normality. This research was conducted under the guidelines for acceptable field research of mammals by the American Society of Mammalogists (Sikes et al. 2016) and before the Old Dominion University and Valdosta State University Institutional Animal Care and Use Committees evaluated proposals for research on wild mammals.

Results

Prevalence and burden of stomach nematodes in cotton rats.—Prevalence of nematodes in 286 cotton rats from Virginia in 1983–1984 was 79.8%, and infected animals had a mean nematode mass of 1.31 g ± 0.08 (*SE*). Mean nematode mass did not differ significantly between males and females (*t*-test, $P > 0.1$). Prevalence was much lower, 23.1%, for 425 cotton rats from Virginia in 1988–1990, when infected cotton rats had a mean nematode mass of 0.49 g ± 0.07 *SE*, again with no significant difference between sexes. Prevalence of nematodes in a sample of 459 cotton rats from Georgia in 1987–1989 was 33.0%, and infected rats had a mean nematode mass intermediate between the two Virginia samples—0.77 g ± 0.1 *SE*; again, nematode mass did not differ between sexes.

Effects of nematode presence and burden on body mass and reproductive mass of cotton rats.—Initial data exploration revealed that nematode mass was significantly positively correlated with cotton rat body mass for each sex in all three samples. But given that adult rats continue to grow in body length as they age (Cameron and Spencer 1981; Bergstrom and Rose 2004) and that mass is positively correlated with body length, further analyses sought to factor out body length as a covariate, thereby examining the residual differences in body mass and reproductive mass for any effects of parasitism. As an internal confirmation of the age–length relationship, we examined the best independent indicator of age, which was parity of adult females (i.e., a multiparous female [≥ 2 litters] is necessarily older, on average, than a primiparous female [1 litter], which is in turn older than a nulliparous adult female [0 litter]). In female cotton rats from each of the three samples, parity, as defined above (and coded 2, 1, 0, respectively, for analysis), was highly positively correlated with body length (Pearson *r*, all $P << 0.001$).

For each sex of each sample, body length, as a covariate in ANCOVA with presence of stomach nematodes as the main effect, was highly positively correlated with both body mass and reproductive mass (Table 1). After removing this age–length effect, the presence of stomach nematodes had mixed effects on body and reproductive masses, with either positive or null effects from the two later Virginia and Georgia samples (in which prevalence and parasite burdens were lower). For the earlier Virginia sample (with higher prevalence and parasite burdens), presence of stomach nematodes had negative effects on reproductive mass in both sexes and null effects on body mass (Table 1).

Restricting the analysis to only the rats that harbored stomach nematodes, with mass of nematodes as the main effect and body mass and reproductive mass the response variables, body length was a weaker covariate, though positive when significant, which was in one-half of the cases. After removing the effects of body length, there was no relationship between mass of nematodes and either body mass or reproductive mass in either sex in any of the three samples (Table 2).

Effects of nematode presence and burden on litter size of cotton rats.—Again, removing any effect of body length as a covariate, for the two samples for which we had both nematode mass and associated litter-size data for females (Virginia 1983–1984 and Georgia 1987–1989), we found no effect on litter sizes of being parasitized in either sample. Further, we found a positive effect of greater mass of nematodes on litter size in the Georgia sample, and no effect of mass of nematodes on litter size in the Virginia sample (which had a higher prevalence and parasite burden; Table 3).

Table 1.—Results of analysis of covariance (ANCOVA) in all cotton rats sampled in Virginia and Georgia, with presence of stomach nematodes as main effect, body length as covariate, and body mass and reproductive mass (uterine mass for females, testes plus seminal vesicles mass for males) as response variables. After removing body-length effect on reproductive and body masses, the remaining effect of being parasitized by nematodes was either not significant (n.s.), marginally significant (m.s. +/–), significantly positive (+), or significantly negative (–) on mass. Reproductive masses were ln-transformed for normality.

Population/sample	Sex	Response (*n*)	Main effect (presence of nematodes)	Covariate (body length)
Virginia 1983–1984	F	Reproductive mass (137)	$- F_{1,134} = 5.3, P = 0.023$	$+ F_{1,134} = 59.0, P < 0.001$
		Body mass	n.s. $F_{1,134} = 0.5, P = 0.5$	$+ F_{1,134} = 95.1, P < 0.001$
	M	Reproductive mass (143)	$- F_{1,141} = 7.8, P = 0.006$	$+ F_{1,141} = 74.0, P < 0.001$
		Body mass	n.s. $F_{1,141} = 0.74, P = 0.39$	$+ F_{1,141} = 162.3, P < 0.001$
Virginia 1988–1990	F	Reproductive mass (203)	$+ F_{1,201} = 5.7, P = 0.018$	$+ F_{1,201} = 63.9, P < 0.001$
		Body mass	$+ F_{1,201} = 9.25, P = 0.003$	$+ F_{1,201} = 245.4, P < 0.001$
	M	Reproductive mass (219)	n.s. $F_{1,216} = 1.8, P = 0.18$	$+ F_{1,216} = 71.9, P < 0.001$
		Body mass	$+ F_{1,216} = 9.25, P = 0.003$	$+ F_{1,216} = 182.5, P < 0.001$
Georgia 1987–1989	F	Reproductive mass (214)	n.s. $F_{1,211} = 0.04, P = 0.84$	$+ F_{1,211} = 12.5, P = 0.001$
		Body mass	m.s.+ $F_{1,211} = 3.1, P = 0.078$	$+ F_{1,211} = 168.4, P < 0.001$
	M	Reproductive mass (242)	n.s. $F_{1,239} = 2.5, P = 0.12$	$+ F_{1,239} = 24.8, P < 0.001$
		Body mass	$+ F_{1,239} = 8.7, P = 0.003$	$+ F_{1,239} = 419.9, P < 0.001$

Table 2.—Analysis of covariance (ANCOVA), as in Table 1, but including only cotton rats that had stomach nematodes, with the continuous variable mass of stomach nematodes as main effect; nematode and reproductive masses were ln-transformed for normality. n.s. = not significant; + = significantly positive.

Population/sample	Sex	Response (*n*)	Main effect (mass of nematodes)	Covariate (body length)
Virginia 1983–1984	F	Reproductive mass (112)	n.s. $F_{1,109} = 0.8$, $P = 0.72$	n.s. $F_{1,109} = 1.9$, $P = 0.2$
		Body mass	n.s. $F_{1,109} = 0.5$, $P = 0.5$	+ $F_{1,109} = 5.5$, $P = 0.047$
	M	Reproductive mass (117)	n.s. $F_{1,114} = 0.9$, $P = 0.65$	+ $F_{1,114} = 13.6$, $P = 0.001$
		Body mass	n.s. $F_{1,115} = 1.1$, $P = 0.4$	+ $F_{1,115} = 27.4$, $P < 0.001$
Virginia 1988–1990	F	Reproductive mass (57)	n.s. $F_{1,54} = 1.2$, $P = 0.4$	+ $F_{1,54} = 5.7$, $P = 0.04$
		Body mass	n.s. $F_{1,54} = 1.2$, $P = 0.4$	+ $F_{1,54} = 17.4$, $P = 0.002$
	M	Reproductive mass (45)	n.s. $F_{1,42} = 0.5$, $P = 0.9$	n.s. $F_{1,42} = 0.04$, $P = 0.86$
		Body mass	n.s. $F_{1,42} = 1.2$, $P = 0.5$	n.s. $F_{1,42} = 0.02$, $P = 0.9$
Georgia 1987–1989	F	Reproductive mass (79)	n.s. $F_{1,76} = 0.48$, $P = 0.94$	n.s. $F_{1,76} = 0.1$, $P = 0.76$
		Body mass	n.s. $F_{1,78} = 2.0$, $P = 0.19$	n.s. $F_{1,78} = 2.3$, $P = 0.18$
	M	Reproductive mass (67)	n.s. $F_{1,64} = 0.64$, $P = 0.83$	n.s. $F_{1,64} = 0.73$, $P = 0.42$
		Body mass	n.s. $F_{1,63} = 1.5$, $P = 0.32$	+ $F_{1,63} = 8.5$, $P = 0.03$

Table 3.—Analysis of covariance (ANCOVA) results, showing a) the effects of presence of stomach nematodes on litter size in pregnant cotton rats after removing the effect of body size for two samples, and b) same as a), only the main effect is mass of stomach nematodes. n.s. = not significant; + = significantly positive.

a) Population/sample	Sex	Response (*n*)	Main effect (presence of nematodes)	Covariate (body length)
Virginia 1983–1984	F	No. of embryos (63)	n.s. $F_{1,60} = 0.0$, $P = 0.99$	n.s. $F_{1,60} = 1.4$, $P = 0.2$
Georgia 1987–1989	F	No. of embryos (108)	n.s. $F_{1,105} = 0.1$, $P = 0.8$	n.s. $F_{1,105} = 1.25$, $P = 0.3$
b) Population/sample	**Sex**	**Response (*n*)**	**Main effect (mass of nematodes)**	**Covariate (body length)**
Virginia 1983–1984	F	No. of embryos (63)	n.s. $F_{1,60} = 0.7$, $P = 0.4$	+ $F_{1,60} = 3.0$, $P = 0.01$
Georgia 1987–1989	F	No. of embryos (103)	+ $F_{1,100} = 4.2$, $P = 0.04$	n.s. $F_{1,100} = 1.1$, $P = 0.35$

Discussion

Patterns of infection.—Greater prevalence in the Virginia old field (80%) than in the Virginia (23%) or Georgia (33%) mowed sites may have been due to the old field having greater abundances of insects. This conjecture is further supported by the greater parasite burdens, or mean mass of stomach nematodes, in infected cotton rats from the old field than those from mowed sites (1.31 g, versus 0.49 g and 0.77 g). Nematode prevalence reported in other cotton rat populations include 43% of adult cotton rats infected with *P. hispida* in South Carolina (Briese and Smith 1980), 50% for both species of stomach nematodes in Florida uplands (Kinsella 1974), and for *M. muris*, 76% of cotton rats sample in Georgia and 50% of cotton rats sampled in North Carolina (Harkema and Karman 1948).

Effects of parasites on host vigor and fitness.—The expectation is that most parasites will have negative effects on host vigor, host fitness, or both (Rousset et al. 1996), but more field studies are needed to determine if this is true in wild mammalian hosts (Thomas et al. 2000). We found no negative effect of the presence or increased parasite burden (stomach nematode mass) on length-adjusted body mass (i.e., somatic growth and maintenance) of cotton rats of either sex in any of our three samples, and we actually found positive effects of nematode presence on body mass in our two samples with lower prevalence and burden (Table 1). As for reproductive allocation, only in the high-prevalence, high-burden sample did we find negative effects of nematode presence on length-adjusted reproductive masses (in the latter case, for both sexes; Table 1). As for fecundity–fertility effects, length-adjusted litter size was not affected by nematode presence in our two samples of pregnant females, from different regions and with different prevalences and burdens. Greater mass of stomach nematodes in those pregnant females that were infected was associated with larger litters in the Georgia sample, whereas there was no effect of parasite burden on litter size in the high-prevalence, high-burden Virginia sample (Table 3). On balance in this study, presence of stomach nematodes had positive effects on somatic growth and maintenance in cotton rats in environments where parasite prevalence and burden were comparatively low, whereas they had neutral effects in an environment where parasite prevalence and burden were high. Presence of stomach nematodes had either neutral or positive effects on reproductive allocation in cotton rats in an environment where parasite prevalence and burden were low, whereas presence of stomach nematodes had negative effects on reproductive allocation in cotton rats in environments where parasite prevalence and burden were high (Table 1). Presence of nematodes had no effect on litter size in either sample. For infected cotton rats, increased nematode burden had no effect on somatic maintenance nor on reproductive allocation in any of the three samples, and greater parasite burden was associated with larger litters in the lower-prevalence, lower-burden sample, and had no effect on litter size in the higher-prevalence, higher-burden sample.

There is a growing body of literature on the evolution in hosts of tolerance to the effects of gut helminth parasites, mediated by the immune system and complex interactions with

the microbiota (King and Li 2018), effectively turning these once-parasites into commensals. There are also examples of parasites apparently having mutualistic (positive) effects on mammalian hosts, at least during portions of the host life cycle. These include Mueller (1963), who reported that mice given subcutaneous injections of larvae of the cestode *Spirometra mansonoides* grew faster than controls, being 31% heavier at 20 weeks and 49% heavier at 57 weeks. Controlled infections of laboratory mice with various intestinal nematodes can induce the immune system to produce anti-inflammatory cytokines, which improve host health (Maizels and Yahzdanbahksh 2003). Certain gut helminths of mouse lemurs (*Microcebus rufus*) are associated with maintenance of healthy microbiomes (Aivelo and Norberg 2018). In some cases, while physiological health and even survival rates can be diminished by gut helminths, nevertheless litter sizes can increase due to fecundity compensation (increased current reproductive output offsetting increased mortality in *Peromyscus maniculatus* infected with a trematode—Schwanz 2008). An apparently similar increase in current reproductive output was caused by laboratory infection of wild-derived house mice (*Mus musculus*) with the intestinal nematode *Mastophora muris* (Kristan 2004). Both *P. hispida* and *M. muris* are restricted to the stomach and possess a thick noncellular cuticle that protects them from the acidic environment. After embryonated eggs or larvae (L_3 stage) are ingested by insects and after two larval molts during development in the colon wall of the intermediate host, the life cycle is completed when a rodent eats the insect. Once inside the stomach of the definitive host, the larvae of *P. hispida* grow rapidly and metamorphose into adults (to 6–8 mm by day 18 and 14–17 mm by day 25); females mature and lay eggs by 73–90 days (Schell 1952). Details of growth and development are better known for *P. hispida* (Schell 1952) than for *M. muris* (Wertheim 1962).

Time to maturity in female *P. hispida* (10–12 weeks) is not much shorter than the mean life span of cotton rats and many other small-mammal hosts. The average life span of cotton rats in the Piedmont of Georgia was estimated to be only 6 months (Odum 1955); however, the fact that multiparous females were captured in every calendar month in the lower Coastal Plain of Georgia, including > 40% of those captured in February, implies they may live significantly longer than 6 months (Bergstrom and Rose 2004). By contrast, the complete lack of multiparous females January through April in southeastern Virginia (Bergstrom and Rose 2004) implies a life span more comparable to that found by Odum (1955). The important point is that cotton rats are not long-lived animals that can acquire parasites over more than a year. When availability of nematodes is low in the environment, prevalence in cotton rats is low (their intake of insects is generally low because it may be largely incidental to their grass folivory—Kincaid and Cameron 1982), but the probability of acquiring these parasites increases steadily with age, hence the positive association of nematode presence with body length. Yet, once nematodes are acquired, a cotton rat is likely to retain its parasites for the rest of its life, though our results do not support that parasite burden increases with body length (age). Prevalences ranging from 23% to 80% among sample populations and nematode residency inside a host lasting most of the adult life of the host constitute chronic parasitism, and as a result, various strategies of accommodation and even adaptation are to be expected within the host population.

The reasons for positive effects of nematodes on somatic growth and maintenance in cotton rats remain unclear. However, several studies and reviews report links between helminth infection and protective immune mechanisms, including suggestions that helminths govern immune-mediated inflammation and healthy microbiomes (Weinstock et al. 2002; Maizels and Yahzdanbahksh 2003; McKay 2006; Anthony et al. 2007; Helmby 2009; Elliott and Weinstock 2012; Weinstock and Elliott 2014; King and Li 2018). If body mass truly is an adaptive feature, the results of our study suggest that the hispid cotton rat–nematode symbiosis is a potential candidate model to use in testing the association between immune-mediated inflammation and autoimmune responses in hosts.

Because only potentially adult cotton rats, i.e., those ≥ 50 g, were studied, we were able to examine effects of stomach nematodes on reproductive allocation and litter sizes. The very presence of the nematode seems to trigger a positive response in reproductive allocation in environments that yield moderate prevalence of infection (perhaps with lower insect abundance than in our earlier Virginia sample), and in these environments, litter size increases with increasing parasite burden. This may be a further indication of immune-mediated benefits of the nematode to host vigor, or it may be fecundity compensation, where the female hosts respond by shifting their life history toward the semelparity end of the spectrum, increasing their current reproductive output to offset increased mortality from higher parasite burdens.

Acknowledgments

We thank owners who granted permission to trap cotton rats on their properties, S. Brant and other parasitologists in three labs for identifications, and our universities for equipment and other support. The work was partly funded by the Langdale Foundation of Valdosta, Georgia and the Valdosta State University Foundation. We benefitted from assistance by V. Whitley, T. Stahl, K. Brosell, T. Eastman, E. Evans, and A. White, and comments by two reviewers, M. Steele and S. Gardner.

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Submitted 21 June 2019. Accepted 17 August 2019.

Associate Editor was Luis Ruedas.