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Sex differences in parasitic infections among arthropod hosts: is there a male bias?

Letitia A. D. Sheridan, Robert Poulin, Darren F. Ward and Marlene Zuk

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A higher susceptibility to diseases or parasites in males than females may be an ultimate consequence of the different reproductive strategies favored by selection in the two sexes. At the proximate level, the immunosuppressant effects of testosterone in vertebrates provide a mechanism that can cause male biases in parasite infections. Invertebrates, however, lack testosterone and other steroid hormones. We used a meta-analysis of published results to investigate whether sex biases in parasite infections were generally observed among arthropod hosts despite the absence of the immune-endocrine coupling provided by testosterone. Overall, male and female arthropods did not differ in prevalence or intensity of parasite infections. This is based on an analysis of sex differences corrected for sample size and, when possible, variability in the original data. Sex biases in parasite infection were not more likely to be observed in certain host or parasite taxa, and were not more pronounced in experimental studies than in surveys of naturally infected hosts. Our results suggest that because of the absence of endocrine-immune interactions in arthropods, males are not generally more prone to parasite infections than females despite the greater intensity of sexual selection acting on males.

L. A. D. Sheridan and R. Poulin (correspondence), Dept of Zoology, Univ. of Otago, P.O. Box 56, Dunedin, New Zealand (robert.poulin@stonebow.otago.ac.nz). – D. F. Ward, School of Zoology, La Trobe Univ., Bundoora, Victoria 3083, Australia. – M. Zuk, Dept of Biology, Univ. of California, Riverside, CA 92521, USA.

Ultimately, most if not all differences between the sexes are the product of sexual selection (Clutton-Brock and Parker 1992, Owens and Thompson 1994). In most species, males invest in costly secondary sexual features or courtship displays to attract females, while at the same time competing intensely with other males for access to females. This results in greater inter- and intrasexual selection pressures on males than females, with the life of a male being more socially and energetically stressful than that of a female. One consequence of this might be the higher mortality incurred by males in many taxa (e.g. Promislow 1992, Promislow et al. 1992).

Another consequence might be the higher parasite infection levels commonly observed in the males of many vertebrate species relative to females. Recent literature surveys have found small but consistent male biases in infections by helminth and arthropod parasites in birds and mammals (Poulin 1996a, Schalk and Forbes 1997). The proximate mechanism most often associated with male-biased parasitic infection is the immune suppression associated with androgens, primarily testosterone, the hormones necessary for the development of male sexual traits and behavior (Grossman 1985, Alexander and Stimson 1988, Schuurs and Verheul 1990, Zuk 1990, 1996, Folstad and Karter 1992). In contrast, female oestrogens may actually boost humoral immunity (Grossman 1985). These hormone-mediated differences between the sexes can produce males that are relatively more susceptible to parasite infections than females. In nature, sexual differences in susceptibility to parasites may be masked to

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some degree by differences in exposure resulting from sex-specific behaviors (e.g. see McCurdy et al. 1998). Thus the proximate effect of sex hormones on susceptibility to parasites is more easily detectable in experimental studies, where exposure is controlled, than among naturally infected males and females (see Schalk and Forbes 1997).

The bulk of the research carried out thus far on sex biases in parasitism has been performed on vertebrates, and almost nothing is known of the general patterns and processes in invertebrates (Zuk and McKean 1996). On the one hand, the operation of sexual selection should be the same in invertebrates as in vertebrates (Clutton-Brock and Parker 1992), ultimately producing different reproductive strategies in males and females and causing sex biases in parasite infections. On the other hand, the proximate mechanism operating in vertebrates, i.e. the immunosuppressive effects of testosterone, is absent in invertebrates. The relationship between sex and parasite infections may therefore be less likely to develop in invertebrates (Zuk and McKean 1996). However, other mechanisms could produce sex biases in parasite infections among invertebrates. For instance, males may have less energy to invest in immune responses than females because males engage in intrasexual competition and courtship of females. It would be important to quantify the general pattern of sex biases in parasitism among invertebrate species to determine whether the expected ultimate effects of sexual selection on parasite infections also occur in taxa that lack testosterone and other steroid hormones (Zuk and McKean 1996).

The objective of this study was to investigate the occurrence and general direction of sex biases in parasite infections among arthropod species. We performed a meta-analysis of published data on male and female infection levels in which we controlled for sample sizes as well as assessing the influence of other variables. These other variables were host and parasite taxonomy and whether the hosts had been naturally or experimentally infected by the parasites. We examined the effect of these variables because sex biases in parasitism might be more likely to develop in certain host taxa infected by certain parasite taxa, or more easily detected when exposure is experimentally controlled.

Methods

Data collection

We searched the literature for data on fungal, protozoan or metazoan infections in females and males from the same natural population or from the same experimental study. Specifically, we searched all issues of *Parasitology* and the *Journal of Parasitology* available at the University of Otago, as well as RP's reprint collection and our own data sets. Most of the data (88% of the male-female comparisons used) came from studies that did not focus on sex biases in parasitism but reported these data nonetheless for descriptive purposes. It is therefore unlikely that our data set suffered from a problem of under-representation of non-significant differences.

Two measures of parasitism were considered and recorded separately for each sex: prevalence (percentage of hosts that are infected) and intensity (mean number of parasites per infected hosts). To be included, a study had to report prevalence and/or intensity of infection by a parasite species and sample sizes for both sexes of a host species. If available, we also recorded the standard deviation in intensity for both males and females. Finally, we only kept studies in which the type of infection (experimental or natural) was clearly stated. Some studies provided more than one comparison to the data set. A total of 33 studies contributed to the data set (see Appendix 1).

Statistical analysis

We treated each host-parasite species combination as an independent observation. While phylogenetic effects may influence sex biases in infection (Harvey and Pagel 1991), it is difficult to control simultaneously for both the host and parasite phylogenies. Previous meta-analyses of sex biases in parasite infections have similarly treated each host-parasite combination as statistically independent (Poulin 1996a, Schalk and Forbes 1997, McCurdy et al. 1998).

Comparisons of prevalence and intensity of infection between the sexes were computed for each host-parasite system to produce standard measures that are independent of sample size (Hedges and Olkin 1985). Differences in prevalence were calculated as

$$(p_f - p_m)(J), \text{ where } J = 1 - [3/(4(N_f + N_m - 2) - 1)]$$

The difference between the prevalence in females, p_f, and that in males, p_m, is weighed by J, which is a correction for small sample sizes, N_f and N_m. As the total sample size increases, J approaches one so that more weight is given to comparisons based on many host individuals (Hedges and Olkin 1985). This correction is important because estimates of prevalence are often influenced by host sample size (Gregory and Blackburn 1991). Using the above formula, we get positive comparisons when prevalence is greater in females, and negative comparisons when it is greater in males. Similarly, differences in intensity were computed as

$$(I_f - I_m)J/I_f$$

Again, the difference between the intensity in females (I_f) and that in males (I_m) is corrected for sample size. Here, female and male sample sizes used in the computation of J are the numbers of infected host individuals, whereas in comparisons of prevalence we used the numbers of individuals examined. Also, differences in intensity are expressed as a proportion of the intensity in females. This procedure was necessary because the actual intensities of infection recorded in the studies we used vary greatly among systems (see Poulin 1996a). In a true meta-analysis, differences between mean values for males and females should be adjusted for the variability among individuals, i.e. each difference should be divided by the pooled standard deviation of the two groups (Hedges and Olkin 1985). This procedure was used for the small subset of comparisons in intensity between males and females for which standard deviations were available.

The null hypothesis (i.e. no sex bias in levels of infection) is that differences in prevalence and intensity are symmetrically distributed around a mean of zero. We used one-group, two-tailed t-tests to compare the standardized comparisons to the expected mean of zero. The use of directional tests instead of two-tailed tests could be justified given our specific hypothesis (Rice and Gaines 1994). Using one-tailed tests would not affect our conclusions, and thus we report the results of two-tailed tests. Analyses were performed following the log transformation of prevalence and intensity values in the computations of differences between sexes; however, untransformed values are reported in all figures and tables. We present results of analyses across the entire data set, as well as separate analyses for subsets of the data in order to highlight the influence, if any, of the type of infection, host taxonomy and parasite taxonomy.

Results

We obtained 61 comparisons of prevalence and 31 comparisons of intensity of infection between males and females (see Appendix 1). The majority were from natural infections. In general, sample sizes were large. For instance, total sample size (males plus females) for prevalence comparisons averaged 1351 (range 44–18540) for natural infections and 337 (range 60–1160) for experimental infections. Sample sizes were smaller for intensity comparisons, because only infected hosts were used, but they were still generally good (overall average = 203). No host taxon was involved in a disproportionate number of comparisons. With respect to parasite taxonomy, however, protozoans and nematodes were better represented in the data set than other taxa (Appendix 1).

Among comparisons of prevalence, sex differences were symmetrically distributed around zero, with almost as many male-biased differences as there were female-biased ones (Fig. 1). The overall average difference in prevalence between the sexes was only about 1% and it did not differ from zero (Table 1). In addition, no sex bias was observed in any of the subsets of the larger data set considered here, i.e. sex differences were not influenced by either host or parasite taxonomy, or by whether the hosts had been naturally or experimentally infected (Table 1).

Sex differences in intensity of infection showed a slightly skewed frequency distribution; however, this results from two strongly male-biased comparisons, with all others having values close to zero (Fig. 2). The overall average difference in intensity between the sexes was relatively very small, less than 1% of the intensity in females (Table 2). We obtained similar results in a separate analysis using only the 12 comparisons that could be corrected for the pooled standard deviation of the original data (mean difference = -1.78, $t = 1.061$, $P = 0.311$). As with comparisons of prevalence, there was no effect of host or parasite taxonomy or mode of infection on sex differences in intensity of infection (Table 2).

Discussion

In birds and mammals, males are typically more susceptible to parasite infections than females (Poulin 1996a, Schalk and Forbes 1997), an observation usually attributed to the immunosuppression associated with testosterone (Schuurs and Verheul 1990, Zuk 1990,

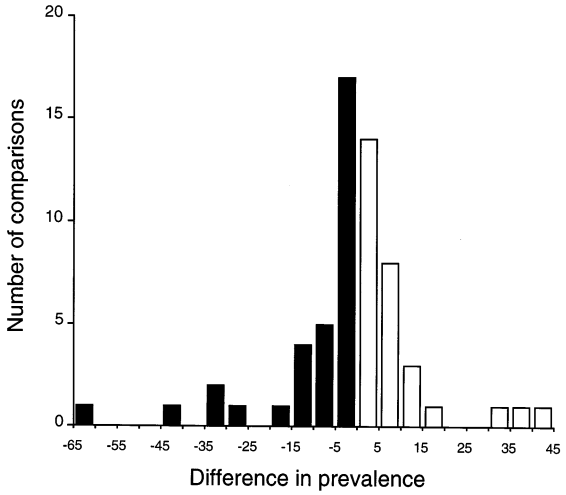

Fig. 1. Frequency distribution of sex differences in parasite prevalence, corrected for sample size, in 61 host-parasite systems involving arthropod hosts. Black columns indicate male-biased differences, and open columns indicate female-biased differences.

Table 1. Differences in prevalence of parasite infection between male and female hosts. Results are presented for the entire data set as well as for various subsets of the data.

Data set	No. comparisons (no. host species)	Mean female minus male prevalence (SD)	t*	P
All data	61 (46)	−1.29 (15.58)	0.647	0.520
Type of infection				
Experimental	11 (8)	0.19 (17.93)	0.035	0.973
Natural	50 (39)	−1.62 (15.20)	0.752	0.456
Host taxon				
Crustaceans				
Decapods	9 (9)	2.28 (7.65)	0.892	0.398
Others	7 (7)	−5.74 (11.76)	1.291	0.244
Ticks	5 (2)	2.57 (8.99)	0.639	0.558
Insects				
Orthopterans	11 (8)	−12.09 (24.67)	1.626	0.135
Blattarians	8 (4)	−1.79 (13.51)	0.376	0.718
Coleopterans	6 (2)	1.87 (4.56)	1.003	0.362
Dipterans	14 (13)	4.58 (15.97)	1.074	0.302
Odonates	1 (1)	0.10 (–)	–	–
Parasite taxon				
Protozoans	26 (19)	−0.81 (12.23)	0.337	0.739
Fungi	2 (2)	19.12 (29.85)	0.906	0.531
Helminths				
Nematodes	16 (11)	−6.73 (20.06)	1.342	0.200
Others	7 (7)	−7.81 (11.09)	1.863	0.112
Arthropods				
Isopods	7 (7)	4.56 (6.52)	1.850	0.114
Mites	3 (3)	11.46 (16.86)	1.178	0.360

* From one-group, two-tailed tests.

1996, Folstad and Karter 1992, Zuk and McKean 1996). To date, no one had investigated the possible existence of a similar general pattern among arthropods or other invertebrates, which lack testosterone and other steroid hormones. The results of the present meta-analysis indicate that there is no general sex bias in parasite infection among arthropods. This absence of bias is independent of host or parasite taxonomy. In addition, no consistent bias emerged from the data of experimental infections, in which sexual differences in susceptibility to parasites should be easier to detect.

In meta-analyses of this nature, it is often tempting to blame the lack of significant effects on various sources of error. For instance, the data on natural infections came from hosts that had been sampled at different times of the year, and parasite infections can show seasonal fluctuations. Also, most studies contributed comparisons in both prevalence and intensity of infection, such that our two data sets were not truly independent (Appendix 1). These and other factors can generate noise in the data set or bias the analysis one way or the other. However, the approach used here is the same as that used in surveys of sex-biased infections in vertebrates, where clear patterns emerged from similar types of data (Poulin 1996a, McCurdy et al. 1998). The average sex differences in infection levels between male and female mammals or birds reported in the literature (Poulin 1996a) can be 3–4 times greater than the ones presented here for male and female arthropods. Our analysis had the power to detect a significant sexual bias had an effect size similar to that in vertebrates been found in arthropod hosts. The results presented here are therefore a good indication that there is no consistent sex bias in parasite infections among arthropod hosts comparable to what is observed in mammals or birds.

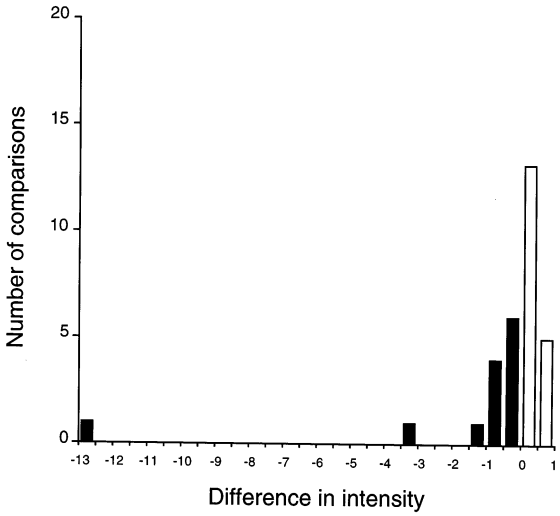

Fig. 2. Frequency distribution of sex differences in parasite intensity, corrected for sample size, in 31 host-parasite systems involving arthropod hosts. The differences are expressed as a proportion of the intensity in females. Black columns indicate male-biased differences, and open columns indicate female-biased differences.

Table 2. Differences in intensity of parasite infection between male and female hosts, expressed as a proportion of the intensity in females. Results are presented for the entire data set as well as for various subsets of the data.

Data set	No. comparisons (no. host species)	Mean female minus male intensity (SD)	t*	P
All data	31 (21)	−0.50 (2.38)	1.176	0.249
Type of infection				
Experimental	8 (5)	−1.82 (4.59)	1.118	0.300
Natural	23 (17)	−0.05 (0.48)	0.488	0.631
Host taxon				
Crustaceans	5 (5)	−0.63 (1.54)	0.913	0.413
Ticks	5 (2)	−2.34 (5.81)	0.900	0.419
Insects				
Orthopterans	7 (5)	−0.33 (0.58)	1.483	0.189
Blattarians	4 (3)	−0.04 (0.57)	0.138	0.899
Coleopterans	7 (3)	0.21 (0.39)	1.434	0.202
Others	3 (3)	0.05 (0.26)	0.335	0.770
Parasite taxon				
Protozoans	10 (7)	−1.27 (4.05)	0.988	0.349
Helminths				
Nematodes	13 (8)	−0.04 (0.53)	0.257	0.801
Others	5 (5)	−0.55 (1.59)	0.771	0.484
Arthropods	3 (3)	0.09 (0.04)	3.483	0.073

* From one-group, two-tailed tests.

The absence of a universal and consistent sex bias does not mean that there is no bias in any specific host-parasite system. Significant differences between males and females in prevalence and/or intensity of infection were reported for some (9 out of 24 comparisons for which a statistical test was reported) of the comparisons included in our data set. In some cases (six), males were more parasitized than females, whereas the opposite was true in other comparisons (three). The authors of only one (Wedekind and Jakobsen 1998) of these studies attributed the observed difference to sexual selection; other authors either did not discuss the difference or attributed it to ecological causes. It may be that there exists no general pattern among arthropods, but that the specific biology of hosts and parasites may produce biases one way or the other in some host-parasite species combinations. This interpretation is not consistent with the generally more intense sexual selection pressure acting on males than on females and producing more stressful reproductive strategies in males (Clutton-Brock and Parker 1992, Owens and Thompson 1994). One factor that may obscure sex differences in susceptibility to parasites could be size dimorphism: in most invertebrates, females are often larger than males because of fecundity-driven selection (see Poulin 1996b for review). The larger size of females could result in greater exposure to parasites and provide more resources to incoming parasites, thus negating a higher susceptibility of males to infection. This may be a common phenomenon, since positive correlations between host size and intensity of infection, regardless of host sex, were reported in many studies that we surveyed.

Experimental studies should be more sensitive than field surveys to intrinsic differences in susceptibility to parasites between males and females, if these exist, because they control for differences in exposure. Combined in the meta-analysis, the results of experimental studies did not suggest any clear sex bias. Taken individually, however, they can provide clear patterns within host species. For example, Wedekind and Jakobsen (1998) found that male copepods had significantly higher prevalence and intensity of infection than females in experimental infections with the larval cestode *Schistocephalus solidus*. Their study suggests that the immune response of males is somehow weaker than that of females. These results and interpretations mirror the findings of other studies on bacterial infections in arthropods (e.g. Gray 1998). Since the majority of the studies included in our data set involved naturally infected hosts, it will be important to use an experimental approach in more invertebrate-parasite systems to elucidate the influence of sex on infection levels.

In addition, the relatively simple immune system of arthropods and other invertebrates (Loker 1994) should be amenable to studies of sex differences in its functioning. All our conclusions are strictly about sex differences in actual parasitic infections, and not about sex differences in immune response. It may be that male and female invertebrates invest differentially in defense against pathogens because of differences in sexual selection, but that this differential investment is not reflected in terms of parasite infection to the same degree as in vertebrates because of the greater simplicity of the invertebrate immune system.

Taken as a whole, our results suggest that there may be a difference between vertebrates and arthropods

with respect to some of the consequences of sexual selection. We should expect basic differences between the sexes in terms of basic parameters such as mortality rates, in both vertebrates and invertebrates. However, the potential for endocrine-immune interactions provides a means for selection to act in vertebrates in a way that it might not be able to achieve in invertebrates. Invertebrates lack the negative feedback system between the immune system and the expression of sexual features and behavior provided by testosterone in vertebrates. Thus, higher levels of parasitic infections may be a general cost for male vertebrates but not so general for male invertebrates.

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Appendix 1. Data on female and male infection levels used in the analyses.

Host taxon	Parasite taxon	Type of study*	Female infection	Male infection	Sample size†	Source**
Prevalence of infection						
decapod	nematomorph	N	1.12	1.84	2500	1
decapod	trematode	N	57.14	67.85	647	2
decapod	isopod	N	62.85	46.15	471	3
decapod	isopod	N	35.20	33.90	4327	4
decapod	isopod	N	0.28	0.30	18540	5
decapod	isopod	N	69.90	61.15	2868	6
decapod	isopod	N	33.33	26.67	51	7
decapod	isopod	N	0.92	1.43	397	7
decapod	isopod	N	1.47	2.33	333	7
amphipod	acanthocephalan	N	2.70	4.16	4451	8
amphipod	cestode	N	3.50	3.90	870	9
amphipod	trematode	N	46.45	58.85	940	10
amphipod	mite	N	62.32	58.86	1183	11
copepod	protozoan	N	30.14	30.56	765	12
copepod	cestode	E	40.00	70.00	182	13
isopod	acanthocephalan	N	27.00	26.10	5340	14
tick	protozoan	E	38.00	26.80	500	15
tick	protozoan	E	11.63	1.51	944	16
tick	protozoan	E	88.20	91.40	138	17
tick	protozoan	E	10.00	20.00	60	18
tick	protozoan	E	31.29	26.70	1160	19
orthopteran	protozoan	N	56.25	66.67	58	20
orthopteran	protozoan	N	17.07	48.88	264	21
orthopteran	protozoan	N	46.62	51.39	349	21
orthopteran	protozoan	N	13.64	23.08	83	20
orthopteran	protozoan	N	38.71	44.00	87	22
orthopteran	protozoan	N	53.33	57.90	64	22
orthopteran	protozoan	N	36.28	30.00	132	22
orthopteran	protozoan	N	51.72	53.57	57	22
orthopteran	nematode	N	15.39	77.42	44	20
orthopteran	nematode	N	15.39	57.58	46	20
orthopteran	mite	N	83.33	52.00	49	22
blattarian	protozoan	N	27.27	21.88	76	23
blattarian	protozoan	N	90.91	96.88	76	23
blattarian	protozoan	N	45.46	46.88	76	23
blattarian	protozoan	N	31.82	25.00	76	23
blattarian	nematode	N	54.17	87.10	79	22
blattarian	nematode	N	68.97	61.11	65	22
blattarian	nematode	N	87.50	79.50	178	24
blattarian	nematode	N	97.73	100.00	76	23
coleopteran	nematode	N	19.20	16.60	156	25

Appendix 1. (Continued)

Host taxon	Parasite taxon	Type of study*	Female infection	Male infection	Sample size†	Source**
coleopteran	nematode	N	2.50	7.60	156	25
coleopteran	nematode	N	0.50	0.40	1214	25
coleopteran	nematode	N	3.80	2.20	1214	25
coleopteran	nematode	N	3.80	0.80	1214	25
coleopteran	nematode	N	39.20	30.20	1214	25
dipteran	protozoan	E	52.10	40.95	149	26
dipteran	protozoan	E	49.30	61.80	141	26
dipteran	protozoan	E	72.05	34.50	148	26
dipteran	protozoan	N	9.10	9.50	623	27
dipteran	protozoan	N	5.97	4.23	394	28
dipteran	protozoan	N	16.90	14.10	6344	27
dipteran	protozoan	E	47.85	65.05	142	26
dipteran	protozoan	E	4.20	4.00	146	26
dipteran	fungus	N	73.00	32.60	176	29
dipteran	fungus	N	80.00	82.00	100	29
dipteran	nematode	N	2.33	3.36	799	30
dipteran	nematode	N	21.68	19.86	1347	30
dipteran	nematode	N	1.85	2.08	570	30
dipteran	nematode	N	6.23	4.09	4569	30
odonate	mite	N	98.20	98.10	1847	31
Intensity of infection						
decapod	trematode	N	2.90	3.10	412	2
decapod	isopod	N	1.55	1.38	250	3
decapod	isopod	N	1.08	1.04	54	5
copepod	cestode	E	0.50	2.20	81	13
isopod	acanthocephalan	N	3.39	2.85	1417	14
tick	protozoan	E	1.12	15.51	63	16
tick	protozoan	E	21.45	9.20	338	19
tick	protozoan	E	3.98	2.08	162	15
tick	protozoan	E	17.10	14.43	124	17
tick	protozoan	E	1.67	2.00	9	18
orthopteran	protozoan	N	2.32	3.92	35	22
orthopteran	protozoan	N	10.68	3.07	46	22
orthopteran	protozoan	N	8.98	15.63	35	22
orthopteran	protozoan	N	2.48	3.43	30	22
orthopteran	protozoan	N	19.67	17.82	37	20
orthopteran	nematode	N	7.00	14.63	26	20
orthopteran	nematode	N	12.00	15.26	21	20
blattarian	nematode	N	1.71	2.32	53	22
blattarian	nematode	N	5.10	3.80	148	24
blattarian	nematode	N	1.97	1.94	42	22
blattarian	acanthocephalan	E	12.36	7.10	141	32
coleopteran	nematode	N	17.50	11.40	420	25
coleopteran	nematode	N	1.00	1.00	28	25
coleopteran	nematode	N	3.00	1.00	5	25
coleopteran	nematode	N	6.90	3.00	37	25
coleopteran	nematode	N	5.20	8.10	28	25
coleopteran	nematode	N	6.50	2.70	8	25
coleopteran	cestode	E	2.09	1.84	120	33
dipteran	nematode	N	1.91	2.38	22	30
dipteran	nematode	N	2.21	1.62	279	30
odonate	mite	N	35.60	31.40	1813	31

* E, experimental infection; N, natural infection.

† For prevalence, total number of hosts examined; for intensity, total number of infected hosts in natural infections, or total number of hosts exposed to infection in experimental studies.

** Sources: 1, Born 1967; 2, Stromberg et al. 1978; 3, Kuris et al. 1980; 4, van Wyk 1982; 5, Field 1969; 6, Beck 1979; 7, Wenner 1978; 8, Ward 1986; 9, Stark 1965; 10, Thomas et al. 1995; 11, Kitron 1980; 12, Wickstead 1963; 13, Wedekind and Jakobsen 1998; 14, Seidenberg 1973; 15, Purnell et al. 1973; 16, Young et al. 1975; 17, Young et al. 1980; 18, Purnell et al. 1975; 19, Irvin et al. 1981; 20, Luong and Zuk unpubl.; 21, Zuk 1987; 22, Ward et al. 1998; 23, Tsai and Cahill 1970; 24, Dobrovolny and Ackert 1934; 25, Fincher et al. 1969; 26, Moloo et al. 1993; 27, Moloo et al. 1973; 28, Rogers et al. 1972; 29, Schlein et al. 1985; 30, Welch 1959; 31, Andres and Cordero 1998; 32, Lackie 1972; 33, Keymer 1982.