Parasite infection and host dynamics in a naturally fluctuating rodent population

J.C. Winternitz, M.J. Yabsley, and S.M. Altizer

Abstract: Parasites can both influence and be affected by host population dynamics, and a growing number of case studies support a role for parasites in causing or amplifying host population cycles. In this study, we examined individual and population predictors of gastrointestinal parasitism on wild cyclic montane voles (*Microtus montanus* (Peale, 1848)) to determine if evidence was consistent with theory implicating parasites in population cycles. We sampled three sites in central Colorado for the duration of a multiannual cycle and recorded the prevalence and intensity of directly transmitted *Eimeria* Schneider, 1875 and indirectly transmitted cestodes from a total of 267 voles. We found significant associations between host infection status, individual traits (sex, age, and reproductive status) and population variables (site, trapping period, and population density), including a positive association between host density and cestode prevalence, and a negative association between host density and *Eimeria* prevalence. Both cestode and *Eimeria* intensity correlated positively with host age, reproductive status, and population density, but neither parasite was associated with poorer host condition. Our findings suggest that parasites are common in this natural host, but determining their potential to influence montane vole cycles requires future experimental studies and long-term monitoring to determine the fitness consequences of infection and the impact of parasite removal on host dynamics.

Key words: Microtus montanus, montane vole, *Eimeria*, helminths, population cycles, parasite regulation.

Résumé : Les parasites peuvent influencer la dynamique des populations de leurs hôtes, mais également être influencés par celles-ci. De fait, un nombre croissant d'études de cas appuie la thèse voulant que les parasites contribuent à causer ou amplifier les cycles des populations de leurs hôtes. Nous avons examiné des prédicteurs aux échelles tant individuelle que de la population de parasitisme gastrointestinal chez des campagnols montagnards (*Microtus montanus* (Peale, 1848)) cycliques à l'état sauvage afin d'établir si les observations concordaient avec la thèse d'un rôle des parasites dans les cycles de populations d'hôtes. Nous avons échantillonné trois localités du centre du Colorado pendant la durée d'un cycle pluriannuel et noté la prévalence et l'intensité d'*Eimeria* Schneider, 1875, un parasite à transmission directe, et de cestodes à transmission indirecte chez 267 campagnols au total. Nous avons noté des associations significatives avec l'état d'infection et des caractères individuels (sexe, âge et état reproducteur) des hôtes et des variables relatives à la population (localité, période de capture et densité de population), dont une association positive entre la densité des hôtes et la prévalence de cestodes et une association négative entre la densité des hôtes et la prévalence d'*Eimeria*. Les intensités des cestodes et d'*Eimeria* étaient toutes deux corrélées positivement à l'âge et à l'état reproducteur des hôtes et à la densité de leur population, mais ni l'un ni l'autre des parasites n'était associé à un moins bon état des hôtes. Si nos résultats suggèrent que les parasites sont répandus chez cet hôte naturel, d'autres études expérimentales et une surveillance à long terme visant à déterminer les conséquences des infections sur la valeur sélective et l'impact du retrait des parasites sur la dynamique des hôtes sont nécessaires pour établir si ces parasites peuvent influencer les cycles des campagnols montagnards.

Mots-clés : Microtus montanus, campagnol montagnard, *Eimeria*, helminthes, cycles de population, régulation des parasites.

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Introduction

Understanding parasite impacts on host populations can inform a basic understanding of population regulation (Elton 1924) and can aid efforts in wildlife conservation and management (O'Brien and Evermann 1988; Scott 1988; Smith et al. 2009). For example, population declines can be caused or exacerbated by infectious diseases, as evidenced by endangered Tasmanian devils (*Sarcophilus harrisii* (Boitard, 1841)) and transmissible facial cancer (McCallum 2008), amphibian population declines due to chytridiomycosis (Skerratt et al. 2007), and for great ape populations suffering from Ebola and other emerging diseases (reviewed in Leendertz et al. 2006). More generally, parasites can have both regulatory and destabilizing effects on the dynamics of vertebrate host populations (reviewed in Hudson et al. 2002). However, only a handful of studies have directly implicated parasites as causing or amplifying host population cycles. These include the classic examples of Red Grouse (*Lagopus lagopus scotica* (Latham, 1787)) and their ceacal nematodes on the Scottish

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moors (Dobson and Hudson 1992; Hudson et al. 1998), feral Soay sheep (*Ovis aries* L., 1758) and gastrointestinal nematodes on the island chain of St. Kilda (Gulland 1992), and deer mice (*Peromyscus maniculatus* (Wagner, 1845)) and their gastrointestinal helminths in eastern North America (Pedersen and Greives 2008).

The relationship between host abundance and parasite transmission is a core concept in epidemiology (Anderson and May 1978; Lloyd-Smith et al. 2005). For parasites to regulate their hosts, their transmission must increase with host density (Anderson and May 1978). However, in natural systems, parasite life history, transmission mode, host recruitment, behavior, and climate can obscure the relationship between host density and parasite prevalence or even cause a negative relationship to appear (Montgomery and Montgomery 1988; Haukisalmi and Henttonen 1990; Lloyd-Smith et al. 2005). For example, increased range size (Ostfeld et al. 1985) or territorial exclusion (Boonstra et al. 1998) at low densities could lead to individuals foraging farther distances, increasing their exposure to parasites in the environment. Beyond reductions in abundance, mathematical models suggest three conditions under which parasites can induce host population cycles: (1) delayed density-dependence in parasite recruitment, (2) moderate to low degree of parasite aggregation among hosts, and (3) negative effects of parasitism on host fecundity or the recruitment of juveniles (Anderson and May 1978; Dobson and Hudson 1992; Hudson et al. 1998).

Microtine rodent population cycles have fascinated population ecologists since Elton (1924) first observed them almost 100 years ago, and yet the mechanisms for these cycles are still unknown (Boonstra et al. 1998). Some likely drivers are variation in climate, resource abundance, predators, parasites, and the interactive effects of multiple drivers (Turchin 1993). Montane voles (*Microtus montanus* (Peale, 1848)) exhibit frequent population cycles and can fluctuate from 300 to <25 animals/ha (Smith and Merrick 2001). Montane voles have short life spans (6–12 months) and can produce up to four litters per year (Jannett 1977), giving them potential for explosive growth. Montane voles are also infected with a range of parasites that can reach high prevalence in the populations, including cestodes, nematodes, and protozoans (Kinsella 1967; Timm 1985). Although the fitness consequences of parasite infection have never been assessed in montane voles, these parasite taxa are known to reduce host fitness in numerous vertebrate species through reducing body condition, survival, and reproductive success (Scott and Lewis 1987; Scott 1988; Hakkarainen et al. 2006; Turner et al. 2011), thus creating the potential for effects on host population cycles.

The goals of this study were to examine the potential for individual and population impacts of parasitism on montane voles. We first characterized the community of gastrointestinal parasites at three sites inhabited by voles near the Rocky Mountain Biological Laboratory in Colorado, USA. We next asked whether individual host traits, including age, sex, body condition, and reproductive status were associated with measures of parasitism. We further explored population changes in parasitism, including within- and between-year changes in prevalence and host abundance, and tested for host density effects on the spatial distribution of infection. We predicted that for parasites to influence host dynamics, either parasite prevalence or infection intensity should increase with vole population density. We also examined whether parasites were weakly aggregated among hosts, as has been shown for other regulating parasites. Finally, we tested for negative effects of parasites on host body condition or reproductive status, as would be expected if parasites negatively affect host fitness.

Materials and methods

Sites and field sampling

Voles were trapped for 3 consecutive years (2008–2010) at three replicate sites within 5 km of the Rocky Mountain Biological Laboratory, located in the Upper East River Valley (39°N, 107°W), Colorado, USA. Trapping sites (A, Kettle Ponds 1; B, Kettle Ponds 2; C, Research Meadow) were separated by a minimum of 0.5 km at approximately 2900 m elevation. Vegetation at each site was dominated by the grasses *Festuca thurberi* Vasey (Thurber fescue), *Elymus trachycaulum* (Link) Gould ex Shinners (slender wheatgrass), *Bromus polyanthus* Scribn. ex Shear (Great Basin brome), and *Bromus inermis* Leyss. (smooth brome), and the forb *Lupinus argenteus* Pursh (silvery lupine). Voles were captured using Longworth live traps (23 cm × 8x8 cm) spaced 10 m apart, with the size of each sampling grid based on available open space. Site A encompassed a 60 m × 80 m trapping grid (7 trap stations × 9 trap stations: 63 total traps), site B encompassed a 60 m × 40 m trapping grid (7 trap stations × 5 trap stations: 35 total traps), and site C contained 60 m × 60 m trapping grids (7 trap stations × 7 trap stations: 49 total traps). Trapping grids were baited with a mixture of peanut butter, rolled oats, and millet and a piece of polyfill bedding material was added to each trap for warmth. Traps were opened between 1800 and 2000 and checked the following day between 0600and 0900. Animals were captured 4–5 consecutive days per site every 2 weeks throughout the breeding season (15 June – 15 August).

Animals were uniquely identified with numbered ear tags (National Brand Tag Company). We recorded individual sex, reproductive status, mass (to the nearest 0.5 g) using a spring balance, and body length (to the nearest millimetre) using digital calipers. Males were considered to be reproductive if a scrotal sac was visible and females if they had a perforate vagina, visible nipples, or were pregnant by palpation. Body mass combined with evidence of sexual maturity was used as an index of age class (juvenile: <15 g and not reproductively active; subadult: 16–30 g; adult: >30 g) following Negus and Pinter (1966) and Sullivan et al. (2003). A 2 mm tail tip was collected from nonjuveniles and stored in 95% ethanol after anesthetizing the animals with isoflurane gas for 1 min. Fecal samples were collected from the used traps and traps were scrubbed with 20% bleach solution to reduce parasite transmission. Animals were processed with permission of the Colorado Department of Natural Resources Division of Wildlife (Scientific Collections Licenses No. 08-10TR2006) and handled in accordance with the animal care guidelines of the American Society of Mammalogists (Sikes and Gannon 2011), the Rocky Mountain Biological Laboratory Animal Care and Use Committee, and the University of Georgia Institutional Animal Care and Use Committee (AUP No. A2010 5-092).

Parasite identification and quantification

Fecal samples were stored in 10% formalin with half the sample stored in 2.5% potassium dichromate for coccidia sporulation and identification (Duszynski and Wilber 1997). Salt flotation using sodium nitrate solution (specific gravity 1.2–1.5) was used to isolate intestinal parasites eggs (Dryden et al. 2005). Cover slips were scanned in five replicate zigzag transects at both 100× and 400×; parasite oocytes and eggs were quantified per gram of feces. Noninvasive fecal egg counts are commonly used in longitudinal studies as an approximation of worm burden and have been shown to correlate with the ability of the host's immune system to regulate worm burden and fecundity (Stear et al. 1995; Nielsen et al. 2010). Although the relationship between fecal egg counts and the actual number of adult worms is system specific, this measure has been shown to be useful in other wild rodent systems and can provide a valuable noninvasive approximation of worm burden (Brenner 1970; Keymer and Hiorns 1986; Scott 1988; Ferrari et al. 2004; Froeschke and Sommer 2005; Harf and Sommer 2005).

Morphological measurements were used to identify three major taxonomic groups of intestinal parasites (Table 1): coccidia, cestodes, and nematodes. *Eimeria* Schneider, 1875 is the only intestinal coccidian genus reported from voles (Levine and Ivens 1965). Oocysts of this protozoan parasite are shed in the host feces and transmitted via a fecal–oral route. *Eimeria* can reduce overwinter survival of rodents (Fuller and Blaustein 1996), increase susceptibility to predation (Voříšek et al. 1998), and reduce breeding success (Hakkarainen et al. 2006). We identified five *Eimeria* morphospecies in montane voles following sporulation and these were designated as *Eimeria* A through *Eimeria* E. The most common *Eimeria* sp. found in our study (*Eimeria* A) was not similar to any of the previously described *Eimeria* spp. of *Microtus* based on measurements and general morphology of 325 oocycts (Levine and Ivens 1965). All *Eimeria* spp. have direct life cycles. We identified two species of cestodes in this study that were identified as *Andrya* Railliet, 1883 and *Paranocephala* Lühe, 1910 based on the size and morphology of eggs. In particular, comparison of morphometric evidence with reports in the primary literature indicated that these species were *Andrya macrocephala* Douthitt, 1915 or *Andrya primordialis* Douthitt, 1915 (Rausch and Schiller 1949; Rausch and Tiner 1949) and *Paranoplocephala infrequens* (Douthitt, 1815) Baer, 1927 (Rausch and Kuns 1950). Because both of these species have similar transmission (ingestion of infected intermediate hosts (orabatid mites) from the environment), data on all cestodes were combined for most analyses. We also observed eggs of one nematode species (2% prevalence) morphologically similar to *Syphacia obvelata* (Rudolphi, 1802) (Kinsella 1967).

Multiple measures of infection status were examined for individual voles: parasite species richness, and for each parasite, the presence or absence and intensity of infection. Because multiple parasite species could infect any single host, we summed the number of intestinal parasite species (counting separately each morphospecies of *Eimeria*, cestodes, and nematodes for a maximum of eight species) as one measure of parasitism. Analyses of parasite prevalence and intensity were conducted for each parasite type (combining data for all morphospecies) separately. Intensity was recorded for the two most prevalent parasite groups: cestode intensity was estimated as the number of oocytes or eggs per gram of feces, and *Eimeria* intensity was scored categorically on a scale of 0–3 per scan of the entire cover slip (1 = 1–10 oocysts; 2 = 11–100 oocysts; 3 = >100 oocysts).

Statistical analysis

Estimation of host density using capture–mark–recapture

Estimates of population size were calculated using the Schnabel method, based on recapture data and assuming closed populations (Sutherland 1996). Individual-level methods to estimate population size were not employed because of the low recapture rate per individual (captures per animal = 2.92 ± 0.1 (mean ± SE), range = 2–9). We divided the breeding season into five 2-week long trapping intervals and estimated population size separately for each trapping period and site. Population size was divided by site grid size, giving vole density per hectare.

Correlates of parasitism: effects of individual host traits, density, and time

To test the level of aggregation among hosts for each type of parasite, we examined the k parameter of the negative binomial distribution (Wilson et al. 2002), and the index of discrepancy (D) of Poulin (1993) using the program Quantitative Parasitology by (Rózsa et al. 2000). Measures of aggregation were examined for the most common parasite morphospecies (*Eimeria A* and cestode A) owing to sample size limitations for the less common parasites. To test for potential association between the presence of cestodes and *Eimeria*, we used χ^2 tests, and we also used Pearson's correlations to test for associations between cestode and *Eimeria* intensity focusing on the subset of animals positive for both parasites.

To examine how individual vole traits and population variables influenced the risk of infection, we ran generalized linear models (GLMs) with the presence of *Eimeria* and the presence of cestodes as separate dependent variables (assuming a binary logistic link function). The independent predictor variables were age, sex, site, trapping period, and year with vole population density as a continuous covariate, and we included all biologically relevant two-way interactions. To examine factors associated with parasite intensity, we ran a series of GLMs with the same independent predictors (listed above) and the following dependent variables, in separate analyses: endoparasite species richness (assuming a Poisson distribution and log-linear-link function), cestode intensity (assuming a negative binomial distribution and log-link function), and *Eimeria* intensity (using a linear-link function). We ran a separate analysis using similar predictor variables with a reduced sample set of only adults with reproductive scores ($N = 70$) to consider the relationship between adult reproductive status and parasite prevalence and intensity. Host breeding status was scored as follows: 0 (nonreproductive; $N = 7$), 1 (perforate vaginal opening or visible nipples; $N = 25$), 2 (pregnant or lactating; $N = 20$), and 3 (scrotal; $N = 16$). We log-transformed host density prior to all analyses and we simplified the model using AIC_c (Akaike's information criteria corrected for smaller sample sizes) following Crawley (2002). Unless otherwise stated, all

Table 1. Mean parasite prevalence, species richness (by taxonomic group), and parasite intensity for all montane voles (*Microtus montanus*) captured in the 3-year time series ($N = 238$).

	Prevalence (%)	No. of species (mean ± SE)	Intensity (mean ± SE)
All intestinal parasites	62.2	1.51±0.07	NA
Coccidia	52.9	1.39±0.06	1.7±0.80
Eimeria A	45.2		1.7±0.08
Eimeria B	8.7		2.3±0.19
Eimeria C	8.3		2.4±0.16
Eimeria D	8.3		1.9±0.17
Eimeria E	2.2		1.75±0.25
Cestodes	24.4	0.25±0.03	263.9±59.7
Cestode A	23.7		298.2±66.5
Cestode B	2.8		54.3±36.8
Nematode	1.2	NA	NA

Note: Mean species number and mean intensity were calculated using positive animals only. *Eimeria* intensity was based on categorical scoring as described in the Materials and methods. NA, not applicable.

analyses were performed using the statistical software SPSS version 19.0 (SPSS Inc., Chicago, Illinois, USA), and results are reported as likelihood ratio χ^2 for analyses of continuous dependent variables (e.g., body condition, parasite intensity) and as Wald χ^2 for analyses of binomial dependent variables (e.g., presence of infection).

Association between parasitism and host condition

To test whether infection measures were associated with poorer host body condition, we estimated body condition in four ways: as a relative index (Ri) using the residuals of an ordinary least squares (OLS) regression of ln(mass) against ln(length) (Schulte-Hostedde et al. 2005), as Fulton's index (untransformed mass/length^3) (Fulton 1904), as the body-mass index (BMI = untransformed mass/length^2) (Garrow and Webster 1985), and as a scaled index (SMI) using the scaled mass index (Peig and Green 2009). The SMI uses the population mean, a scaling exponent estimated from an OLS regression, and the length and body mass of each individual to produce a predicted body mass (Peig and Green 2009). We ran multiple GLMs using linear-link functions on each condition index, with the following infection measures as independent variables—model 1: endoparasite species richness; model 2: *Eimeria* presence + cestode presence; model 3: *Eimeria* intensity (using data from positive animals only); and model 4: log cestode intensity (again, using data for positive animals only). Other independent variables included in all GLMs were age, sex, site, trapping period, log density, and year. We excluded pregnant females ($N = 16$) from these analyses as their mass was grossly affected by the size of their gestational mass, and their condition would thus not provide a biologically meaningful comparison to other groups. Pregnant females were included in a separate analysis to explore the effects of reproductive status on the association between infection and body condition in adults only ($N = 70$). As a final test of effects of parasitism on host condition, we ran a repeated-measures ANOVA using data from recaptured individuals and using all indices of condition. Specifically, we tested whether a change in body condition between the first and the second capture event depended on the change in infection status. Detailed methods and results for this recapture analysis are provided in the supplementary material.[1]

Association between parasites and host density

To further examine the association between host population density and parasitism, we averaged parasite and host data at the level of site and trapping interval for a total of 25 observations over 3 years. We used a mixed model with a built-in temporal autocorrelation function to examine how changes in parasite prevalence and intensity co-varied with host density. Here, we assumed that both parasite prevalence and host density followed a time-decaying covariance process, so that correlations within each variable decreased linearly over time (trapping period) for each site and each year (SUBJECTS) by using the REPEATED option with a first-order autoregressive process. Density was included as a fixed effect, and site and year were random effects. Because the shedding of parasite transmission stages may occur several weeks after infection, we examined whether host density was associated more strongly with prevalence or intensity from the same sampling interval (T) versus the next 2-week interval ($T + 1$). Moreover, because parasitism could itself cause changes in host density, we also compared host density with parasite measures from the previous 2-week time interval ($T - 1$). Thus, we ran three separate temporal combination analyses for each of three separate parasite measures: cestode prevalence, *Eimeria* prevalence, and cestode intensity (sample sizes for *Eimeria* intensity data were not sufficient to include this variable in analyses). To meet assumptions of normality in this analysis, we used log-transformed host density, log-transformed cestode intensity, and logit-transformed parasite prevalence (Warton and Hui 2011).

To examine spatial effects in the potential relationship between parasite prevalence and host density, we tested whether transmission is localized within a grid as opposed to ran-

[1]Supplementary materials are available with the article through the journal Web site (http://nrcresearchpress.com/doi/suppl/10.1139/z2012-083).

domly, as might be the case with indirectly transmitted parasites, using Ripley's *K* function in the spatstat package version 1.14-8 in R (Baddeley and Turner 2005). Detailed methods for this spatial analysis are provided in the supplementary material.[1]

Results

A total of 472 capture events occurred during the breeding season (June–August) across all 3 years (2008–2010). Of these, we used only first capture data to determine correlates of infection status ($N = 267$ animals). No marked animals were captured in multiple sites, indicating parasite transmission between sites was probably rare. Vole density changed by a factor of 30 between trapping intervals within each year (2008, 2009, 2010) and a factor of 20 between years (peak at 2009, low at 2010; Fig. 1). Mean vole density ranged from 105 to 161 animals/ha between sites: site A = 105 voles/ha (range across years 18–174 voles/ha); site B = 161 voles/ha (range across years 12–410 voles/ha); site C = 160 voles/ha (range across years 16–381 voles/ha) (Fig. 1). These density estimates based on field data are consistent with longer term patterns of vole abundance over 14 years (Fig. 1 inset; R.J. Smith, unpublished data).

Overall prevalence for *Eimeria* spp. was 53%, with an intensity score of 1.7 ± 0.80 (mean ± SE) among the subset of infected animals (Table 1). *Eimeria* were moderately aggregated in the population during 2009 (Table 2), with a relatively small proportion of voles harboring a large number of oocytes. *Eimeria* prevalence also appeared to differ among sampling locations and fluctuated weakly across trapping intervals and years (Table 2, Fig. 2). Overall prevalence for cestodes was 24%, with an intensity of 263.9 ± 59.7 eggs/g feces (mean ± SE) among infected animals (Table 1). Cestodes were highly aggregated at the population level, although aggregation was lower in 2009, the year with the highest vole density (Table 2). Cestode prevalence and intensity did not differ among sampling locations but changed between years (Table 2). We found no evidence for a statistical association between cestode and *Eimeria* presence as binary variables ($\chi^2_{[1]} = 2.13$, $P = 0.14$, $N = 267$). When we focused on data for animals infected with both parasites simultaneously, the intensity of cestodes and *Eimeria* was positively correlated ($r = 0.51$, $P = 0.005$, $N = 29$). Nematode prevalence was 1.2% among voles for 2008–2010. Because of the low prevalence of nematodes, we excluded this group from further analysis.

Individual and population predictors of infection status

Older animals had a significantly higher probability of cestode and *Eimeria* infection, greater cestode intensity, and higher endoparasite species richness (Table 3). Males had significantly higher cestode prevalence than females (Table 3). *Eimeria* intensity was not predicted by attributes of individual hosts, but site was a significant predictor of both *Eimeria* prevalence and intensity (Table 3). Parasite species richness also varied among sites (Table 3). Seasonal and yearly changes in cestode prevalence were observed with higher prevalence towards the middle of each breeding season (Table 3, Fig. 2) and in 2009 (Table 3). Cestode intensity was also greater in 2009 and was positively correlated with host density (Table 3). There was an interaction between host age and density such that the positive relationship between cestode intensity and host density was stronger among subadults than adults (Table 3). *Eimeria* intensity also increased with host density (Table 3). Analyses restricted to the subset of adults with reproductive data showed that cestode intensity was greatest in pregnant and lactating females and in scrotal males, and lowest for nonreproductive adults (Table 4). Site, density, and year were again significant predictors of cestode intensity in this reduced data set. *Eimeria* prevalence and intensity, as well as endoparasite species richness, varied by site and trapping interval (Table 4).

Parasitism and host body condition

All host condition indices were significantly positively correlated (adjusted R^2 based on linear regressions ranged from 0.57 to 0.97). For analyses that included data on animals from all age classes in the same model, three out of four indices of condition (Ri, BMI, and SMI) varied significantly between age classes for each of the three GLMs run with (*i*) endoparasite species richness, (*ii*) parasite presence or absence, and (*iii*) parasite intensity. For example, analysis of Ri in relation to endoparasite richness showed that condition measures were greater for adults than subadults or juveniles (age effect: $\chi^2_{[2]} = 73.98$, $P = 0.001$) and the same result was seen when comparing Ri to cestode and *Eimeria* presence (age effect: $\chi^2_{[2]} = 78.29$, $P = 0.001$) and intensity (age effect: $\chi^2_{[2]} = 11.49$, $P = 0.001$). For each of these analyses, condition measures scaled separately for adults, subadults, and juveniles. Fitted models tested using Fulton's index performed no better than the intercept-only model. No measure of parasitism was significantly associated with any of the four condition indices for this full data set.

For analyses of body condition restricted to adults ($N = 70$), we report results from residual condition measure Ri only because analysis of other condition measures produced similar results. Body condition was significantly associated with both site ($\chi^2_{[2]} = 10.17$, $P = 0.01$) and host density ($\chi^2_{[1]} = 4.5$, $P = 0.03$), but not with cestode or *Eimeria* presence or absence. For a separate analysis restricted to infected animals only that included cestode and *Eimeria* intensity as predictor variables ($N = 14$), log cestode intensity was positively associated with adult condition ($\chi^2_{[1]} = 17.33$, $P = 0.0001$). Adult condition also depended on reproductive status ($\chi^2_{[2]} = 19.73$, $P = 0.001$), site ($\chi^2_{[2]} = 34.41$, $P = 0.001$), and trapping interval ($\chi^2_{[4]} = 14.14$, $P = 0.001$). For the GLM including endoparasite species richness, both site ($\chi^2_{[2]} = 10.17$, $P = 0.01$) and increasing density ($\chi^2_{[1]} = 4.55$, $P = 0.04$) were significant variables.

Host density and population parasite measures

We found that both cestode prevalence and intensity were positively associated with vole population density, and *Eimeria* prevalence was negatively associated with vole density, using generalized linear models to examine mean values across site-sampling interval combinations. Our models assumed that correlations in parasite and density measures decayed linearly with increasing time between sampling intervals (treating sampling interval within year as a repeated measure, with the same time-decaying covariance structure

Fig. 1. Intra-annual and multiannual population dynamics of montane voles (*Microtus montanus*) near the Rocky Mountain Biological Laboratory in central Colorado. The bottom panel displays population density by site (A, B, C) for 3 years (2008–2010) during the breeding season from early June to early August. The inset displays yearly density of montane voles recorded at site A from 1997 to 2010 (R.J. Smith, unpublished data).

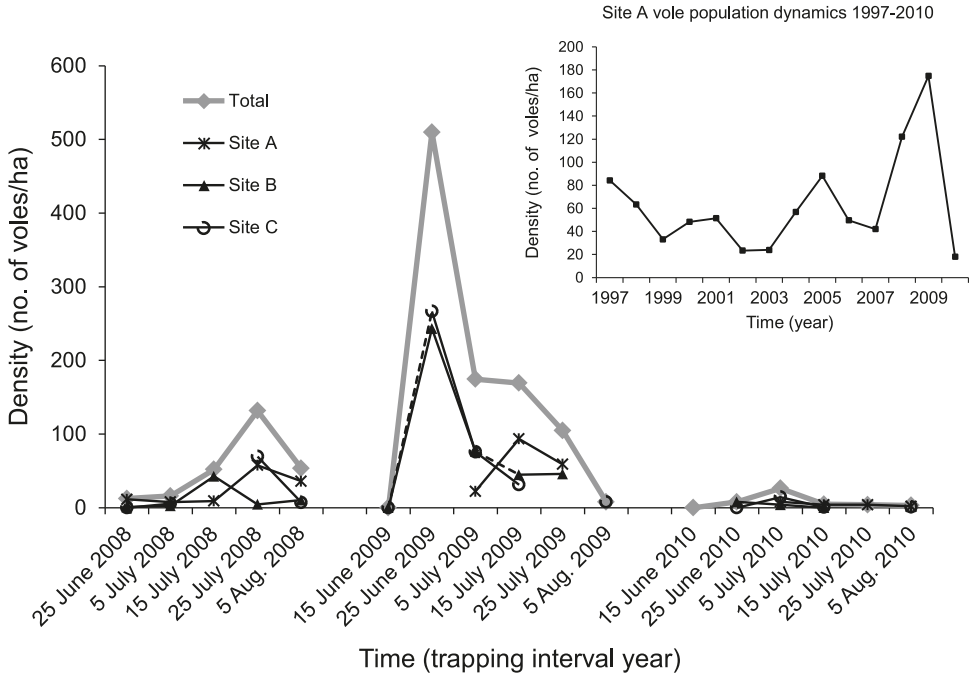


Table 2. Parasite parameters measured separately for each sampling location and year.

Species	Site and year	*N*	Prevalence (%)	Intensity (mean ± SE)	Abundance (mean ± SE)	K^a	D^b
Cestode species	A	64	21.9	206.9±76.1	45.3±19.4	0.048	0.901
	B	90	21.1	326.5±154.9	68.9±34.9	0.030	0.947
	C	113	25.7	229.6±55.2	58.9±16.9	0.047	0.900
	2008	47	12.8	153.2±67.5	19.6±16.9	0.038	0.947
	2009	214	26.2	265.2±61.6	69.4±17.9	0.047	0.907
	2010	6	0.0	NA	0	NA	NA
Eimeria species	A	64	53.1	90.8±8.8	86.90±9.4	0.482	0.640
	B	90	68.9	94.6±5.0	91.85±5.5	0.030	0.947
	C	113	40.7	88.6±3.9	36.30±5.0	0.088	0.858
	2008	47	46.8	NA	NA	NA	NA
	2009	214	55.6	92.4±3.9	63.80±63.8	0.215	0.746
	2010	6	16.7	10.0±0.0	9.35±9.4	NA	0.714

Note: NA, not applicable.

[a]*K* is the corrected aggregation index.

[b]*D* is Poulin's (1993) index of discrepancy, which measures the disparity between observed and uniform distributions.

for all sites and years). For cestodes, the effect of host density on parasite infection was significant for the $T + 1$ scenario (with host density at time T associated with parasite prevalence at time $T + 1$; $F_{[1,6]} = 52.78$, $b = 0.384$, $P = 0.0001$; Fig. 3) but not for the T or $T - 1$ scenarios. A separate model showed that mean cestode intensity at time $T + 1$ was also significantly positively associated with vole population density at time T ($F_{[1,13]} = 11.54$, $b = 0.90$, $P = 0.021$; both T and $T - 1$ scenarios showed no significant association between cestode intensity and host density). In contrast to the cestode results, *Eimeria* prevalence at time $T + 1$ was significantly negatively associated with vole population density ($F_{[1,11]} = 21.76$, $b = -0.63$, $P = 0.001$; both T and $T - 1$ scenarios were not significant). For each of these analyses, there were no significant associations between measures of infection and site or year. Finally, using Ripley's K summary statistic, we did not find evidence of focal transmission of cestodes or *Eimeria* for any site or year.

Discussion

Overall, our results showed that montane vole abundance changed dramatically within and between years, but provided only limited support for an association between host population dynamics and parasitism. Evidence in support of an association between cestodes and montane vole population

Fig. 2. The multiyear dynamics of cestode and *Eimeria* prevalence and cestode intensity plotted alongside log-transformed density of montane voles (*Microtus montanus*). Note log scale on *y* axis.

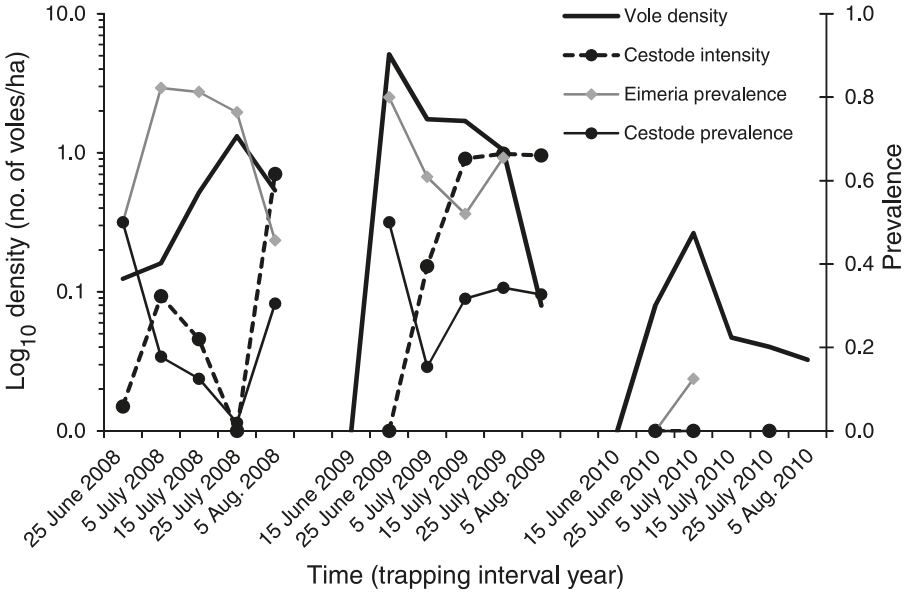


Table 3. Results of generalized linear models ($N = 230$) using data for all montane voles (*Microtus montanus*) (juveniles, subadults, and adults) showing the effect of host variables (age and sex), population variables (site and density), design variables (trapping interval and year), and their interaction on (*i*) the probability of cestode infection, (*ii*) cestode morphotype A intensity, (*iii*) the probability of *Eimeria* infection, (*iv*) *Eimeria* morphotype A intensity, and (*v*) endoparasite species richness.

	Cestode infection (0 or 1)		Cestode A intensity ($N = 52$)		*Eimeria* infection (0 or 1)		*Eimeria* A intensity ($N = 145$)		Endoparasite species richness	
	df	χ^2	df	χ^2	df	χ^2	df	χ^2	df	χ^2
Host variables										
Age	2	11.5**	1	13.4***	2	8.9**	—	—	2	9.3**
Sex	1	4.2*	—	—	—	—	—	—	—	—
Population variables										
Site	—	—	—	—	2	27.1***	2	67.3***	2	13.9***
Density	—	—	1	6.6**	—	—	1	8.2**	—	—
Design variables										
Trapping interval	1	7.7**	—	—	—	—	—	—	—	—
Year	2	11.4**	1	8.1**	—	—	—	—	2	4.62
Interaction										
Age × density	—	—	2	11.8***	—	—	—	—	—	—

Note: Only results for the final simplified models are given (full model: dependent variable = age + sex + site + density + trap interval + year + age × density + age × sex + age × trap interval + sex × density + sex × trap interval). Test statistics are Wald χ^2 for binary (0 or 1) response variables (probability of cestode and *Eimeria* infection) and likelihood ratio χ^2 tests for other dependent variables. *, $P < 0.05$; **, $P < 0.01$; ***, $P < 0.001$.

cycles (for a general review see Tompkins et al. 2002) includes delayed density-dependence in cestode recruitment, with parasites appearing to track host density with a 2-week time lag. Our results also showed that cestode aggregation was weaker during the year (2009) with the highest host density and parasite prevalence, and that reproductively active adults had greater cestode intensity, opening the door for cestodes to negatively affect host reproduction. *Eimeria* intensity increased with host density in one analysis, but a separate analysis showed that *Eimeria* prevalence at time $T + 1$ decreased with host density, suggesting that either mortality of infected animals is higher, or that *Eimeria* transmission is lower, when host density is high. Importantly, we did not find evidence for negative effects of parasitism by either cestodes or *Eimeria* on host body condition as a proxy for host fitness.

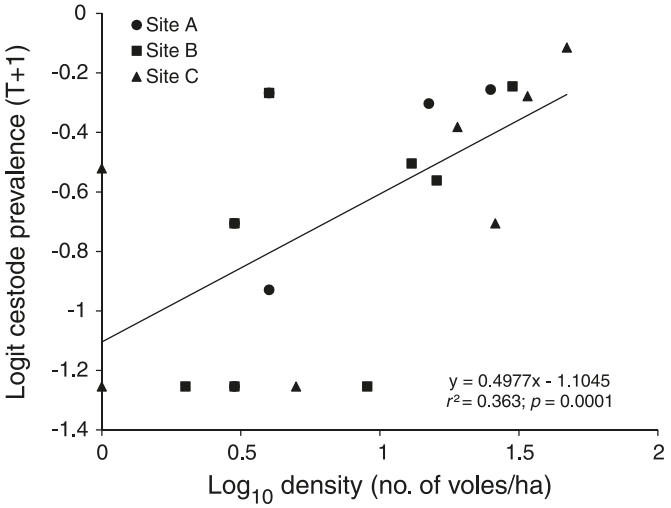
Support for regulatory and destabilizing effects of parasites on host populations has been limited to a relatively small number of intensive field studies, including work on Soay sheep, Red Grouse, and deer mice (Gulland 1992; Hudson et al. 1998; Pedersen and Greives 2008). One factor these systems share in common is evidence that the individual and population impacts of parasites increase directly with host population size. Despite these often-cited examples, many field studies have shown no association between parasite prevalence and host density. For example, no or negative relationships between host density and prevalence of intestinal parasites were found in fossorial water voles (*Arvicola terrestris*

Table 4. Results of generalized linear models on a subset of adult montane voles (*Microtus montanus*) with reproductive status recorded ($N = 70$) showing the effect of host variables (sex and interval reproductive number), population variables (site and density), design variables (trapping interval and year), and their interaction on (*i*) the probability of cestode infection, (*ii*) cestode morphotype A intensity, (*iii*) the probability of *Eimeria* infection, (*iv*) *Eimeria* morphotype A intensity, and (*v*) endoparasite species richness.

	Cestode prevalence		Cestode A intensity ($N = 19$)		*Eimeria* prevalence		*Eimeria* A intensity ($N = 38$)		Endoparasite species richness	
	df	χ^2	df	χ^2	df	χ^2	df	χ^2	df	χ^2
Host variables										
Sex	1	4.4*	1	6.4**	—	—	—	—	—	—
Reproductive number	—	—	3	27.7***	—	—	—	—	—	—
Population variables										
Site	—	—	2	23.8***	2	15.3***	2	22.4***	2	12.6**
Density	—	—	1	19.9***	—	—	—	—	—	—
Design variables										
Trapping interval	—	—	—	—	1	4.4*	—	—	1	3.80
Year	—	—	1	32.8***	—	—	—	—	—	—

Note: Only results for the final simplified models are given (full model: dependent variable = sex + reproducive number + site + density + trap interval + year + sex × density + sex × trap interval + reproductive number × density + reproductive number × sex + reproductive number × trap period). Test statistics are Wald χ^2 for binary (0 or 1) response variables (probability of cestode and *Eimeria* infection) and likelihood ratio χ^2 tests for other dependent variables. *, $P < 0.05$; **, $P < 0.01$; ***, $P < 0.001$.

Fig. 3. The correlation between logit-transformed cestode prevalence 2 weeks later ($T + 1$) and density of montane voles (*Microtus montanus*) at time T averaged for 3 sites for 5 trapping intervals per each of 3 years (2008–2010; $N = 18$). Prevalence was not possible to record for all trapping intervals because of missing fecal samples.



(L., 1758) (Deter et al. 2006), bank voles (*Myodes glareolus* (Schreber, 1780)) (Haukisalmi and Henttonen 1990), wood mice (*Apodemus sylvaticus* (L., 1758)) (Montgomery and Montgomery 1988), and deer mice (Theis and Schwab 1992). Moreover, Fenton et al. (2002) showed in a meta-analysis across experimental studies that the relationship between parasite transmission rate and host density could be either positive or negative, with negative relationships between host density possibly due to the spatial structuring of hosts.

It is important to note that there are multiple factors that could complicate the relationship between host abundance and parasite transmission and impacts. In particular, many cestodes have indirect life cycles that rely on the presence of one or more intermediate hosts. In a comparative study looking across 44 mammalian species and three orders of nematodes, Arneberg (2001) found that host population density was positively correlated with parasite prevalence only for parasites with direct life cycles. The cestodes observed in our study are known to use free-living oribatid mites as their intermediate hosts (Rausch and Tiner 1949). These mites are likely to be abundant in vole habitats (St. John et al. 2006), and because no clustering of prevalence was observed in the spatial analysis, they might not be a limiting step in parasite transmission. In fact, cestode intensity in our study increased as vole density increased, and parasitism peaked just as vole abundance started to decline, consistent with the idea that cestode prevalence and intensity can track host density.

We were surprised to find no evidence for a positive relationship between *Eimeria* prevalence and host density, given that most coccidia are directly transmitted (without intermediate hosts) and have short generation times. It is possible that *Eimeria* transmission depends more strongly on local host density at very small spatial scales as opposed to total host density (Deter et al. 2006). Consistent with this idea, site influenced the probability of *Eimeria* infection at the individual level, with specific sampling locations having consistently higher prevalence, indicating that landscape heterogeneities could influence parasite abundance (reviewed in Hess et al. 2002; Salvador et al. 2011). Moreover, the relationship between *Eimeria* and host density may be confounded by host age; specifically, prevalence could be diluted by uninfected newborns entering the population of continuously susceptible subadults and adults, explaining why *Eimeria* prevalence appears to decrease following increases in host density (Fig. 2). *Eimeria* intensity was positively correlated with host density in 2009, which may suggest lowered resistance to parasite reproduction at high host densities. Indeed, if the mortality of infected hosts increased with greater intensity of infection, this might cause the negative relationship between *Eimeria* prevalence and host density observed here.

In our study, host body condition did not appear to decline during periods of high host density, which has been shown in other species where parasites exacerbate host declines (Gulland 1992; Pedersen and Greives 2008). In particular, a negative

relationship between population density and host condition is expected when increased crowding leads to resource shortages (Begon et al. 2006), which can cause chronic stress (Christian 1950), increased susceptibility to parasites (Beldomenico et al. 2008), and population crashes after periods of high density (Charbonnel et al. 2008). The lack of evidence for declines in host condition observed here could indicate that voles experienced no shortage of resources or social stress during their high density phase, as might occur if other factors (such as predation) kept density well below the carrying capacity of the environment. We also found no changes in age structure or sex ratio in relation to host population density, and mean condition remained relatively constant during all years and across all trapping intervals.

Importantly, we found little support for negative effects of parasites on individual host condition. In general, the negative effects of gastrointestinal helminths are thought to be mediated through the host's energy balance, where there is increased metabolic demand from competition for host resources and from the host mounting defensive responses (Holmes 1995). Lack of negative effects of intestinal parasites on condition or breeding status have also been observed in water voles (Deter et al. 2006), common voles (*Microtus arvalis* (Pallas, 1778)) (Laakkonen et al. 1998), bank voles (Tenora et al. 1979), snowshoe hares (*Lepus americanus* Erxleben, 1777) (Keith et al. 1985), and other studies (reviewed in Irvine 2006). Surprisingly, we found a positive association between cestode intensity and adult vole condition in this study. Similar associations have been observed in other host–parasite systems and could be caused by differences in parasite exposure in relation to foraging or other behaviors. For example, Halvorsen (1986) found that heavy, dominant reindeer were more heavily infected with gastrointestinal nematodes, likely due to greater access to contaminated pasture and a higher rate of nematode ingestion with increased food intake, and a review of laboratory studies investigating effects of small-mammal host diet on parasite biology by Crompton (1987) found that cestode establishment, growth, and reproduction can be affected by host nutritional status. In this study, montane voles in better condition could have foraged more effectively, leading to greater exposure to cestode infection and more nutritional resources to support parasite establishment. Additionally, heavier animals with greater condition measures were more likely to be in breeding condition, and thus could have traded off reproduction against immune defenses, making them vulnerable to chronic infections (Perrin et al. 1996; Schwanz 2008). It is also important to note that the condition indices measured here might not be a reliable proxy of individual fitness. As such, other methods such as parentage analysis (Gooderham and Schulte-Hostedde 2011), recapture survival estimates, and experimental manipulation of parasite loads in the field may be more effective in identifying links between host fitness and parasitism.

Both the probability of transmission and immunological susceptibility to parasites are important determinants of infection and each of these factors can depend crucially on individual host traits such as age and sex. Our study showed that cestode prevalence was greater in males than in females, likely due to androgens (testosterone) suppressing immune response (Folstad and Karter 1992) or to behavioral differences between the sexes that could cause differential exposure (Moore and Wilson 2002). Moreover, cestode prevalence was greater in adults, probably due to increased foraging outside the nest in adults and constant exposure to parasites throughout life. On the other hand, *Eimeria* prevalence was high for all age classes and for both sexes, suggesting high transmission efficiency in *Eimeria* (both within and outside of the nest; Jannett 1978). High prevalence of *Eimeria* in adults further suggests that animals are susceptible to infection following repeated exposures. Moreover, our finding of relatively high prevalence of both cestode and *Eimeria* parasites in adult voles indicates that parasites are probably not eliminated by the host's immune response, as might be inferred from an age-infection relationship where prevalence or intensity first increased and then decreased with host age (Cattadori et al. 2005).

A recent review by Tompkins et al. (2011) highlighted that most of the available evidence of parasite impacts on regularly fluctuating host populations depends heavily on other factors also being in play, especially changes in resource abundance and external environmental factors. A common theme appears to be that parasites can affect the rate and magnitude of host population crashes, but that the ultimate cause of host population fluctuations depends on other variables. For example, in the study of Pedersen and Greives (2008), acorn masting was proposed as the main driver of wild mouse population cycles, but parasites appeared to accelerate the population crashes. Holmes (1995) argued that the real significance of disease in altering wildlife population dynamics is likely to be through its interactions with nutrition, predation, and their synergy. For montane voles, although parasites were not documented to directly lower host condition indices, they may still contribute to population dynamics indirectly, if, for example, they cause increased susceptibility to predation, as has been suggested in other host–parasite interactions (e.g., Murray et al. 1998; Møller and Nielsen 2007).

Montane vole populations are a useful natural system to investigate the potential impacts of parasitism because their rapid fluctuations in host density can provide a backdrop to examine population host–parasite interactions. As such, this study adds to the growing body of work that considers the conditions under which parasites affect host dynamics, and the types of parasites most likely to do so. Our study showed that greater parasite loads (cestode intensity and intestinal parasite species richness) were harbored by larger, older, and higher condition animals, and that one parasite type (cestodes) appeared to positively track changes in host density, whereas *Eimeria* prevalence was negatively associated with host density. We found no evidence that parasites negatively affect host body condition or reproductive status. Although a large number of studies on host–pathogen systems found little evidence that parasites impact host condition or population dynamics (Tenora et al. 1979; Keith et al. 1985; Laakkonen et al. 1998; Deter et al. 2006; Irvine 2006), observational association studies cannot clearly demonstrate that parasites have no effects on their hosts, but rather can suggest fruitful hypotheses to test with experimental studies. A key challenge for ecologists studying the population biology of animal–parasite interactions is to undertake careful analysis of the sublethal impacts of infection, such as through experimental manipulation of parasite loads, before concluding that

parasites have minimal influence on host population cycles (Irvine 2006). As such, a growing number of treatment studies are revealing the detrimental impacts of parasites previously thought to be benign. Another outstanding challenge is for ecologists to identify generalities that might reduce the importance of parasites as regulating forces on natural populations.

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