Parasitol Res (2017) 116:73–80
DOI 10.1007/s00436-016-5263-3

ORIGINAL PAPER

Prevalence of avian haemosporidian parasites is positively related to the abundance of host species at multiple sites within a region

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Received: 21 May 2016 / Accepted: 14 September 2016 / Published online: 5 October 2016

Abstract Parasite prevalence is thought to be positively related to host population density owing to enhanced contagion. However, the relationship between prevalence and local abundance of multiple host species is underexplored. We surveyed birds and their haemosporidian parasites (genera *Plasmodium* and *Haemoproteus*) at multiple sites across eastern North America to test whether the prevalence of these parasites in a host species at a particular site is related to that host's local abundance. Prevalence was positively related to host abundance within most sites, although the effect was stronger and more consistent for *Plasmodium* than for *Haemoproteus*. In contrast, prevalence was not related to variation in the abundance of most individual host species among sites across the region. These results suggest that parasite prevalence partly reflects the relative abundances of host species in local assemblages. However, three nonnative host species had low prevalence despite being relatively abundant at one site, as predicted by the enemy release hypothesis.

Keywords Avian malaria · Enemy release hypothesis · *Haemoproteus* · Host abundance · *Plasmodium*

Introduction

Local species assemblages are typically composed of many rare and few common species (McGill et al. 2007), while locally rare species may be abundant in other parts of their

Electronic supplementary material The online version of this article (doi:10.1007/s00436-016-5263-3) contains supplementary material, which is available to authorized users.

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ranges (Murray and Lepschi 2004). Furthermore, phylogenetically informed analyses show that abundance (i.e., the number of individuals of a particular species) is an evolutionarily labile trait, meaning that closely related species often differ greatly in abundance (Webb and Gaston 2003; McGill 2008; Ricklefs 2011; Ricklefs 2012) despite their presumably similar ecological requirements. These observations suggest that localized, species-specific factors, rather than evolutionarily conserved species traits, may act independently across regions to influence population abundance. One hypothesis proposes that coevolution between specialized parasites and their hosts might generate these abundance patterns (Ricklefs 2011; Ricklefs 2012). Indeed, specialized soil pathogens limit the local abundance of temperate (Packer and Clay 2000; Packer and Clay 2003) and tropical tree species (Mangan et al. 2010), and parasites, including viruses, can depress population densities of their vertebrate hosts (Hudson et al. 1998; LaDeau et al. 2007), although few studies have investigated geographic variation in these effects (but see Ricklefs et al. 2016). Additional support for the influence of pathogens on populations comes from nonnative host species, which are often more common in their introduced ranges than in their native ranges, possibly because they have left their parasites behind (the "enemy release hypothesis," Torchin et al. 2003; Marzal et al. 2011).

Individuals of rare forest tree species may survive less well in the presence of conspecifics than do individuals of abundant species (Comita et al. 2010). This appears to result from rarer species being more susceptible to host-specialized soil pathogens (Mangan et al. 2010) and, potentially, to other natural enemies (Janzen 1970). Conversely, relatively common species might support higher pathogen prevalence than relatively rare species because of higher contagion in denser host populations, as demonstrated for plants in a coastal grassland by Parker et al. (2015). Several studies have shown that both vectored and nonvectored parasite prevalence (i.e., the proportion of individuals of a host species infected by a parasite) increases with host population density (Arneberg et al. 1998; Brown et al. 2001; Krasnov et al. 2002; O'Brien and Brown 2011) and that such relationships can be both parasite and host species specific (Stanko et al. 2006; Isaksson et al. 2013). However, few studies have examined the relationship between parasite prevalence and the abundance of host species at a particular site, representing a host assemblage. Ricklefs et al. (2005) found a U-shaped relationship between the prevalence of dipteran-vectored haemosporidian parasites and the abundances of hosts in a sample of birds at one site in the Ozark Mountains of southern Missouri, where the rarest and commonest bird species had the highest prevalence. However, the general relationship between parasite prevalence and host abundance remains to be determined, both among species within local sites and within species among multiple sites.

Here, the relationship between avian host abundance and the prevalence of haemosporidian blood parasites (genera *Plasmodium* and *Haemoproteus*) at multiple sites across eastern North America is explored. On one hand, haemosporidian parasite control of host abundance might result in negative relationships between parasite prevalence and the abundances of host species within sites. Such patterns would suggest that abundant host species are better able to resist or avoid infection than are less common host species. On the other hand, prevalence might be positively related to host abundance within sites if vectors are more likely to encounter individuals of more abundant host species (Medeiros et al. 2015).

Finally, the enemy release hypothesis predicts that nonnative hosts reach high abundances as a result of escaping their parasites and that generalized parasites (i.e., those with broad host breadth) should attain higher prevalence on native host populations than on nonnative host populations (Keane and Crawley 2002), presumably because they are better adapted to native hosts. This prediction was evaluated by comparing parasite prevalence in populations of native and nonnative hosts at one sampling site (Chicago, IL) where three nonnative host species were particularly well sampled (Medeiros et al. 2013; Ellis et al. 2015).

Materials and methods

Sampling design

Small blood samples (<1 % of an individual's body weight) were collected from 5867 individuals of 99 species of bird (mostly in the order Passeriformes) across 13 sites in eastern North America (Fig. 1) between 1999 and 2014 (full dataset available in Ellis et al. 2015). Individuals of all species were sampled at most of the sites; however, efforts at four sites (CHAMP, CHI2, MS, and the 2012 sample from PA) targeted one or a few species only. Most of the samples were obtained between late May and August, but sampling extended into April and September at some sites. All birds were released after capture, and all sampling took place under appropriate federal and state permits (Master Banding Permits by state for samples collected by the authors: IN [22423], MO [21688], TN [23734], IL [06507; 23469], PA [23343], LA [23299]) and IACUC protocols (University of Missouri-St. Louis [309824-1], University of Illinois-Urbana-Champaign [03034; 14072], Rhodes College [111 and 114], Wilkes College [128], Tulane University [0449]). Blood samples were stored in Puregene® (Germantown, MD) or Longmire's (Longmire et al. 1997) lysis buffer.

Molecular analyses

DNA was extracted from samples stored in lysis buffer using an ammonium acetate-isopropanol precipitation protocol (Svensson and Ricklefs 2009), and DNA samples were

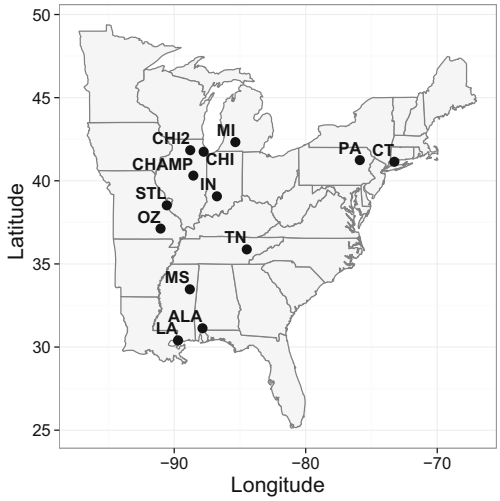

Fig. 1 Sampling sites: *ALA* Alabama, *CHAMP* Champaign (IL), *CHI* Chicago (IL), *CHI2* western Chicago (IL), *CT* Connecticut, *IN* Indiana, *LA* Louisiana, *MI* Michigan, *MS* Mississippi, *OZ* Ozarks (Missouri), *PA* Pennsylvania, *STL* St. Louis (Missouri), *TN* Tennessee

screened using a PCR protocol designed to amplify a small fragment of haemosporidian mitochondrial rDNA (Fallon et al. 2003). For samples that were positive for haemosporidian parasites (based on the PCR screen), a portion of the parasite cytochrome *b* gene was subsequently amplified and sequenced using one or more protocols (Waldenström et al. 2004; Fallon et al. 2005; Ricklefs et al. 2005; Fecchio et al. 2013). Parasite lineages were defined based on cytochrome *b* sequence divergence and host and geographic distributions (Svensson-Coelho et al. 2013; Ricklefs et al. 2014). The average genetic distance among *Haemoproteus* lineages in this study was 0.049 ± 0.018 SD (range 0.004–0.096), and the average among *Plasmodium* lineages was 0.054 ± 0.019 (0.002–0.092). Mixed infections were separated by phasing when possible (Browning and Browning 2011; Matthews et al. 2016). Lineage names, GenBank Accession numbers, and genus assignments are provided in Table S1.

Host abundance

Mist net capture effort was not standardized across sites or across years within sites, and therefore, captures could not be used to estimate host abundance. Alternatively, host abundance within each capture site was estimated from the North American Breeding Bird Survey (https://www.pwrc.usgs.gov/bbs/). Routes deemed acceptable by survey organizers (i.e., routes that met all survey requirements in a particular year) located within 80 km of the sampling sites were selected, and route data corresponding to the year that each site was sampled, plus 1 year before and 1 year after the sample was taken, were used. For example, Chicago, IL, was sampled in 2006 and 2007, so route data from 2005 to 2008 within the 80 km buffer were used (for the sites sampled in 2014, we used route data from years 2013 and 2014). These spatial and temporal windows were used to account for potential bias due to observer error (Sauer et al. 1994) and were chosen a priori. Avian species abundances were averaged across routes and across years for each sampling site to obtain a measure of abundance for each host at each site (Fig. S1).

Statistical analyses

To test for a general relationship between parasite prevalence and host abundance within sampling sites (Fig. S2), a generalized linear binomial mixed-effect model was created in the lme4 package in R (Bates et al. 2014). The proportion of infected individuals of each species was included as the response variable (coded as two vectors—the number of infected individuals and the number of uninfected individuals per species—as is typical for binomial models in R, Crawley 2007) and site-specific host abundance as the explanatory variable. A random effect of site (random intercept) and an individual-level random effect were included to account for overdispersion in the model (Bolker et al. 2009; Harrison 2015). This analysis was restricted to the seven best sampled sites (Table 1; Fig. S1; Ellis et al. 2015); samples from the other six sites included too few host species or did not sample most host species well enough to test this relationship. Because accurate estimates of parasite prevalence depend on adequate sampling, the model was run on three subsets of the data: one in which the data for each site included only host species sampled five or more times and two more subsets where the data for each site included only host species sampled at least 10 and 15 times, respectively. Prevalence in these models was based on all infections (hereafter, "total prevalence") and on infections of *Plasmodium* and *Haemoproteus* parasites separately. Nine models were run in all, the results of which are reported in Table S2. Because results were consistent across models, only the three models using a sample size cutoff of five host individuals per species per site are reported in the "Results" section. Parameter estimates are reported with 95 % confidence intervals, estimated using the "Wald" method in the "confint.merMod" function in the lme4 R package.

A categorical explanatory variable coding whether a host species migrates, or not, was not significant in any model and was dropped from the analysis (results not presented). Three nonnative host species were sampled in the region (*Passer domesticus*, *Sturnus vulgaris*, *Haemorhous mexicanus*), but a categorical variable coding species as native or nonnative also was not significant in any model and was dropped. *H. mexicanus* is arguably less of a nonnative than the other two species since its native distribution extends across western North America. However, because some haemosporidian parasites of *H. mexicanus* show geographic structuring between their western (native) and eastern (nonnative) ranges (Kimura et al. 2006), they were considered nonnatives for this

Table 1 Results of generalized linear models of the effect of log-transformed host abundance on parasite prevalence (total prevalence, *Plasmodium* prevalence only, and *Haemoproteus* prevalence only) within the best sampled sites in the region

Site	Prevalence	β	Confidence interval		P
			2.5 %	97.5 %	
Chicago	Total	0.35 (−1.34)	0.14 (−1.88)	0.56 (−0.83)	0.003 (<0.001)
	Plasmodium	0.61 (−0.87)	0.33 (−1.47)	0.91 (−0.30)	<0.001 (0.007)
	Haemoproteus	−0.37	−0.77	0.07	0.093
Connecticut	Total	0.35	0.02	0.71	0.061
	Plasmodium	0.63	−0.04	1.61	0.131
	Haemoproteus	0.24	−0.14	0.66	0.243
Indiana	Total	0.90	0.35	1.51	0.005
	Plasmodium	0.64	0.10	1.20	0.032
	Haemoproteus	0.85	0.09	1.63	0.040
Michigan	Total	0.58	0.07	1.17	0.051
	Plasmodium	0.42	−0.20	1.15	0.232
	Haemoproteus	0.76	−0.07	1.73	0.115
Ozarks	Total	0.73	0.37	1.14	<0.001
	Plasmodium	0.36	0.01	0.74	0.059
	Haemoproteus	0.90	0.28	1.65	0.014
St. Louis	Total	0.56	0.13	1.02	0.022
	Plasmodium	0.51	0.11	0.94	0.026
	Haemoproteus	0.39	−0.18	1.02	0.205
Tennessee	Total	0.77	0.11	1.48	0.043
	Plasmodium	0.36	−0.28	1.04	0.295
	Haemoproteus	0.97	−0.12	2.39	0.132

The estimate of the coefficient of natural log-transformed (abundance plus 1) (β), its 95 % confidence interval, and P value are reported. The Chicago models included a categorical variable coding for native and nonnative host species, and the coefficients describing nonnative species relative to native ones and their confidence intervals and P values are reported in parentheses next to the estimates for log-transformed abundance. The *Haemoproteus* model in Chicago was run without including a variable coding for nonnative species because nonnatives were not infected with *Haemoproteus* in Chicago

study. The absence of an effect of nonnative species in the models might have been related to small samples from most of the sites (Table S3). However, all three of these species were well sampled at the Chicago site, allowing for a test of their effect at that site alone (see following paragraph).

Although the mixed-effect models provide an estimate of the relationship between parasite prevalence and host abundance generalized across all sites, it was instructive to examine the results of separate models for each of those sites. To that end, generalized linear models with a quasi-binomial error structure (to account for overdispersion) were run for each of the seven best sampled sites individually (using the five individuals per species cutoff) and those results are reported in Table 1. In the Chicago site, where the three nonnative species were well sampled, a categorical variable coding host species as native or nonnative was included in the model.

Generalized linear binomial mixed-effect models were also used to test whether parasite prevalence was related to the abundances of individual host species among the sites where they were sampled. For this analysis, the data were restricted to host species sampled in at least four sites, with at least five ($n = 19$ species), ten ($n = 8$), or 15 ($n = 2$) individuals sampled per site. Since this analysis did not involve comparing multiple host species within the same site, all sites were included, even those with few species sampled. For example, the site MS only included samples of northern cardinals (*Cardinalis cardinalis*), and so, it was used as one of the sites in the northern cardinal model. Parasite prevalence was again calculated in three ways (total, *Plasmodium*, *Haemoproteus*) producing nine models in all. The results of those models are summarized in Table S4. Results were again similar across all models, so the results of the three models using host species sampled at least five times in each of at least four sites are presented. Results of separate generalized linear models for each of the best sampled host species did not provide additional information and are not presented.

The natural logarithm of host abundance plus one (to account for zeros in host abundance) was used in all analyses,

and all graphics were created with the ggplot2 package in R (Wickham 2009). R v.3.2.1 (R Core Team 2015) was used for all analyses.

Results

Parasite prevalence and host abundance of multiple host species within sites

Binomial mixed-effect models revealed a positive effect of host abundance on total prevalence of haemosporidian infection ($\beta = 0.47$, 95 % confidence interval (CI) = 0.27, 0.67; Fig. 2a, Table S2) and on the prevalence of *Plasmodium* ($\beta = 0.47$, 95 % CI = 0.26, 0.68). That is, the most common host species in an assemblage were the most likely to be infected by haemosporidian parasites. The effect of host abundance on *Haemoproteus* prevalence was smaller, and the 95 % CI of the coefficient approached zero ($\beta = 0.36$, 95 % CI = 0.03, 0.70). Separate models for each site of the effect of host abundance on parasite prevalence showed some variation among sites (Table 1), but *Plasmodium* prevalence was still more often positively related to host abundance than was *Haemoproteus* prevalence. In Chicago, the three nonnative species were less infected than native species (total prevalence, $\beta = -1.34$, 95 % CI = −1.88, −0.83, $P < 0.001$; *Plasmodium* prevalence, $\beta = -0.87$, 95 % CI = −1.47, −0.30, $P = 0.007$). Interestingly, none of the nonnative species in Chicago were infected with *Haemoproteus* parasites. However, *P. domesticus* was infected with *Haemoproteus* parasites in St. Louis and in Tennessee (Ellis et al. 2015).

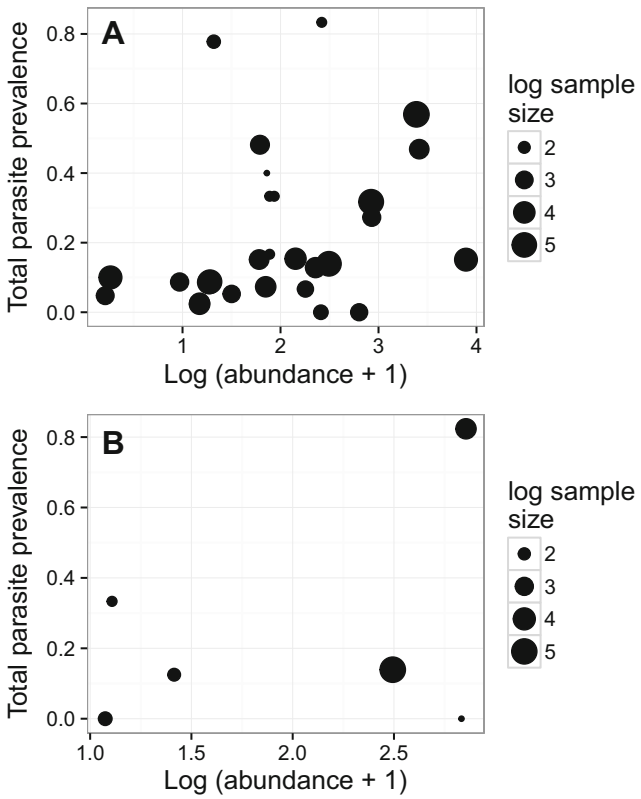

Fig. 2 **a** An example of a positive relationship between total parasite prevalence and natural log of (host abundance plus 1) within a single site, in this case, the Ozarks of southern Missouri. Each *point* represents a host species sampled at least five times in the Ozarks, and *points* are scaled to the natural log of sample size. **b** An example of a nonsignificant relationship between total parasite prevalence and the natural log of (abundance plus 1) of a single host species across the region, in this case, the host *Vireo griseus*. Each *point* represents a site at which *V. griseus* was sampled at least five times, and *points* are scaled to the natural log of sample size

Parasite prevalence and host abundance of individual host species among sites

The within-host species analysis (i.e., the binomial mixed-effect model with host species as a random effect) revealed no significant relationship between haemosporidian prevalence and the abundances of individual host species among sites at which they were sampled ($\beta = 0.29$, 95 % CI = −0.05, 0.64, Fig. 2b, Table S4). When modeled separately, *Plasmodium* ($\beta = 0.07$, 95 % CI = −0.29, 0.43) and *Haemoproteus* ($\beta = 0.26$, 95 % CI = −0.16, 0.69) prevalence also was not related to the abundances of individual host species among the sites where those host species were sampled.

Discussion

Within the sites surveyed here, parasite prevalence was generally positively related to host abundance (Fig. 2a, Table 1, Fig. S2), a phenomenon that is likely mediated by higher vector-host encounter rates in denser host populations. This relationship was stronger for *Plasmodium* parasites than for *Haemoproteus* parasites. Parasites of these two genera have different dipteran vectors: culicid mosquitos [Culicidae] vector *Plasmodium*, while biting midges [Ceratopogonidae] vector *Haemoproteus* (Valkiūnas 2005). These vectors may respond differently to variation in host abundance. In an analysis of avian host utilization by mosquitos at one site investigated here (Chicago), Medeiros et al. (2015) found that abundant hosts were more often bitten by mosquito vectors and were also more likely to be infected by *Plasmodium* parasites compared with less abundant hosts. Their results are consistent with the idea that vector-host encounter rates contribute to the positive relationships between host abundance and parasite prevalence documented here at multiple sites.

Vector exposure might, however, be related to factors in addition to host abundance. This is consistent with the relationships between prevalence and host abundance within sites being statistically noisy (Fig. S2). Many avian hosts are bitten by mosquitos at different rates than expected based on their relative abundances in an assemblage (Kilpatrick et al. 2006; Hamer et al. 2009). Over- and under-utilization of hosts by vectors may, at least in part, be related to host traits (e.g., nesting height, nest type, height of primary foraging stratum, or roosting habitat) that may mediate vector exposure (Fecchio et al. 2013; Svensson-Coelho et al. 2013; González et al. 2014; Lutz et al. 2015; Matthews et al. 2016). For example, in the Tennessee site described here, Matthews et al. (2016) found that host species nesting in open-cup nests had higher prevalence of *Plasmodium* than hosts with closed nests. While this may have been a result of increased exposure to mosquito vectors in open-cup nesting birds, the same effect was not found for *Haemoproteus* infection, so nest type may not influence exposure to biting midges. Parasite transmission may also be limited by parasite-host (Medeiros et al. 2013) and parasite-vector (Carlson et al. 2015) compatibility. Finally, immunological competence of hosts associated with their embryonic development periods might also influence parasite prevalence (Ricklefs 1992). The noise in the prevalence and host abundance relationships within sites might also derive in part from the methods used to estimate abundance, which we cannot evaluate.

Despite the positive relationship between abundance and prevalence across multiple host species within sites (Fig. 2a), parasite prevalence in individual host species was independent of abundance among multiple sites (Fig. 2b). This result contrasts with the positive relationships between parasite prevalence and within-host species abundance reported in the literature on other host-parasite systems. For example, prevalence of directly transmitted ectoparasites of rodents (Krasnov et al. 2002) and of a vector-transmitted arbovirus of birds (O'Brien and Brown 2011) increased with host population density (i.e., within-host species abundance), although the swallow bugs (Hemiptera: Cimicidae: *Oeciacus vicarius*) that vector the arbovirus likely are less mobile than avian haemosporidian vectors. At a site in England, the prevalence of one *Plasmodium* parasite, but not another, increased in response to experimental increases in the population density of the avian host *Parus major*, suggesting that relationships between prevalence and host abundance may be parasite lineage specific (Isaksson et al. 2013). It is unclear why the analysis presented here did not reveal a relationship between prevalence and the abundance of individual host species across sites. Future investigations of this relationship should sample more sites within the distributions of individual hosts.

While no general relationship between parasite prevalence and the abundance of individual host species at multiple sites was found, the prevalence of *Plasmodium* parasite OZ08 was negatively related to the abundance of the host *Icteria virens* at four sites where *I. virens* was sampled at least five times per site (Fig. S3). The majority of infections of *I. virens* in the dataset are from lineage OZ08, which mostly infects *I. virens* (Ellis et al. 2015). The ability of specialized antagonists (e.g., host-specific pathogens) to reduce host population sizes has been hypothesized to generate the variation in abundance often observed among closely related species (Ricklefs 2015; Ricklefs et al. 2016). The negative relationship between *I. virens* and its parasite OZ08 may represent the influence of a parasite on the population of its host.

One of the predictions of the enemy release hypothesis is that nonnative hosts are less impacted by generalized parasites (e.g., resulting in lower prevalence) than are native hosts (Keane and Crawley 2002). However, tests of the hypothesis have tended to focus on the prediction that overall parasite prevalence is lower in the nonnative range of the host than in its native range, rather than comparing prevalence between nonnatives and natives in the same assemblage (Torchin et al. 2003; Marzal et al. 2011). The analysis presented here demonstrated that three nonnative host species had lower haemosporidian prevalence than did native species in the site at which they were best sampled (Chicago; Table 1, Fig. S2). While this result was not replicated in the present study, it is consistent with the results of Lima et al. (2010) who demonstrated that introduced house sparrows (*P. domesticus*) in Brazil had lower prevalence of haemosporidian parasites than native birds in the same location. This suggests that nonnative host species may be more resistant to the parasites that they encounter in their new ranges than are the native hosts with which they coexist or that endemic parasites have lower infectivity in nonnative hosts. In either case, the difference in prevalence between native and nonnative hosts is consistent with evolution of pathogens to infect local hosts.

Overall, the results presented here demonstrate that parasite prevalence varies in direct relation to host abundance within local host assemblages, although other factors (e.g., host life history traits) also account for some of the variance in prevalence and may explain differences between the parasite genera. Future studies should focus on how general these patterns are through time or among other parasites, other geographic regions, and different host species. Future work also should investigate the potential of vector feeding rates to drive parasite prevalence within sites (Medeiros et al. 2015) and to produce the differences between the parasite genera reported here.

Acknowledgments Many people helped with collecting samples and permitting, for which we are grateful: B. Ryder, P. Marra, K. Tranquillo, P. Pyle, the Indiana MAPS teams, A. Burke, field biologist B. Keith and the Kalamazoo Valley Bird Observatory/Kalamazoo Nature Center, the Connecticut Audubon Society, S. Latta, W. Walstrom, D. Outlaw, E. Levenson, and K. Riley. P. Parker, I. Jiménez, S. Mangan, and an anonymous reviewer provided useful feedback on earlier versions of the manuscript. Our work would not have been possible without the help

of numerous government and conservation agencies. We also thank the thousands of volunteers and organizers of the Breeding Bird Survey. Field and laboratory work was supported by the Harris World Ecology Center, the St. Louis Audubon Society, the Missouri Audubon Society, the Curators of the University of Missouri, the NSF Malaria Research Coordination Network, and a University of Missouri-St. Louis Dissertation Fellowship awarded to V.A.E.

Compliance with ethical standards

Ethical standards All applicable federal, state, and institutional guidelines for the care and use of animals were followed in this study.

Conflict of interest The authors declare that they have no conflict of interest.

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