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Infectious disease transmission and behavioural allometry in wild mammals

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Summary

1. Animals' social and movement behaviours can impact the transmission dynamics of infectious diseases, especially for pathogens transmitted through close contact between hosts or through contact with infectious stages in the environment.

2. Estimating pathogen transmission rates and R_0 from natural systems can be challenging. Because host behavioural traits that underlie the transmission process vary predictably with body size, one of the best-studied traits among animals, body size might therefore also predict variation in parasite transmission dynamics.

3. Here, we examine how two host behaviours, social group living and the intensity of habitat use, scale allometrically using comparative data from wild primate, carnivore and ungulate species. We use these empirical relationships to parameterize classical compartment models for infectious micro- and macroparasitic diseases, and examine how the risk of pathogen invasion changes as a function of host behaviour and body size. We then test model predictions using comparative data on parasite prevalence and richness from wild mammals.

4. We report a general pattern suggesting that smaller-bodied mammal species utilizing home ranges more intensively experience greater risk for invasion by environmentally transmitted macroparasites. Conversely, larger-bodied hosts exhibiting a high degree of social group living could be more readily invaded by directly transmitted microparasites. These trends were supported through comparison of micro- and macroparasite species richness across a large number of carnivore, primate and ungulate species, but empirical data on carnivore macroparasite prevalence showed mixed results.

5. Collectively, our study demonstrates that combining host behavioural traits with dynamical models of infectious disease scaled against host body size can generate testable predictions for variation in parasite risk across species; a similar approach might be useful in future work focused on predicting parasite distributions in local host communities.

Key-words: allometric scaling, body mass, host–pathogen dynamics, macroecology, parasite species richness, ranging behaviour, social contact

Introduction

The transmission dynamics of many parasites and pathogens depend on host behaviour. In particular, group living and social interactions can provide contacts between hosts that facilitate disease transmission, especially for pathogens transmitted via close contact (e.g. touching, grooming, food sharing; reviewed in Altizer *et al.* 2003). Animal movements and territoriality can further impact pathogen transmission by influencing host contact with infectious stages in the environment (e.g. macroparasites with free-living infectious stages; Ezenwa 2004a,b; Nunn & Dokey 2006). Thus, in conjunction with pathogen traits such as transmission mode and virulence, host behaviours and resulting contact patterns can fundamentally influence infection patterns in nature (Altizer *et al.* 2003; Gudelj & White 2004; Mossong *et al.* 2008; Hamede *et al.* 2009; Hawley *et al.* 2011).

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Estimating pathogen transmission rates in wildlife hosts can be difficult (Grenfell *et al*. 2001; McCallum, Barlow & Hone 2001) and often requires fine-scale information on contacts between individuals coupled with diagnostic measures of infection through time. The difficulty in obtaining such data makes estimating transmission rates empirically intractable for many wildlife populations (McCallum, Barlow & Hone 2001; Fenton *et al*. 2002). However, a number of host behavioural traits underlying the transmission process vary predictably with body size, one of the most widely available and best-studied species-level traits (Haldane 1956; Peters 1986). For example, comparative studies in mammals have shown that body size correlates negatively with activity rate (Werner 1992) and positively with home range size (McNab 1963) and the propensity for social aggregation (Clutton-Brock, Albon & Harvey 1980). Adult body mass also correlates positively with longevity and some reproductive measures (e.g. time to first reproduction, interbirth interval), across species; at the population level, these traits can predict variation in demographic rates and carrying capacity that determine population dynamics (Lindstedt & Calder 1981).

Here, we incorporate allometric scaling of behavioural traits directly into the transmission parameter of classical infectious disease models to examine their effects on epidemiological outcomes. Previous work has examined the effects of allometric scaling in host demographic parameters on directly transmitted infectious diseases (De Leo & Dobson 1996; Bolzoni *et al*. 2008b). An important prediction was that transmission coefficients required for pathogens to invade should increase with host body size for pathogens with frequency-dependent transmission (De Leo & Dobson 1996). Later work based on an SEI framework and applied to the case of rabies in carnivores showed that the frequency of pathogen outbreaks and the probability of limit cycles within populations could scale allometrically (Bolzoni *et al*. 2008b) and that for multi-host pathogens, the cycle period in a host community is driven by the dynamics within the smallest species. Although this prior work showed that the minimum transmission parameter depended on allometric scaling of host vital rates, a need remains to explore how host behaviours that feed more directly into transmission will scale with body size, and the epidemiological outcomes of this variation.

Our first goal was to empirically derive scaling coefficients for estimates of social group size and ranging behaviour for three groups of mammals (primates, carnivores and ungulates) through an existing global trait data base, and apply these relationships to scale the transmission parameter relative to host body size. These behavioural traits are important because many mammal species exhibit group living where close-contact interactions among group members could increase transmission, particularly of microparasites (reviewed in Nunn & Altizer 2006). For macroparasites transmitted environmentally, infection risk might be expected to increase with ranging behaviours that increase the diversity of habitats sampled by hosts, thus supporting a greater diversity of macroparasites through greater encounter rates. In contrast, recent empirical studies in mammals showed that the intensity of habitat use (e.g. through territorial defence) increases the diversity, prevalence and intensity of macroparasite infections, perhaps due to the accumulation of infectious parasite stages within the hosts' environment and repeated resampling of these habitats by hosts (Ezenwa 2004b; Nunn & Dokey 2006; Lindenfors *et al*. 2007; Bordes *et al*. 2009). Our second goal was to examine the consequences of sociality and home range usage for the invasibility of host populations by microparasites transmitted directly through close contact and by macroparasites transmitted environmentally. Because our models predicted opposing effects of body size on invasion by the two major parasite types examined here, our final goal was to use prevalence and parasite richness data from an existing data base of wild mammal parasites to test whether model predictions were generally supported within particular host taxonomic groups.

Materials and methods

DATA: ALLOMETRIC SCALING OF BEHAVIOURAL TRAITS

We collected data on body size, social group size and the intensity of home range usage to calculate allometric scaling coefficients for wild mammal species of four orders (Primates, Carnivora, Perissodactyla and Artiodactyla). Data on body size and social group size were obtained from PanTHERIA, a freely accessible global data base comprising over 25 biological and ecological traits for more than 5000 mammal species (Jones *et al*. 2009). Body size (mean adult body mass in g) is defined in PanTHERIA as the mass of male or female specimens (excluding pregnant females) from captive, wild or provisioned populations (Jones *et al*. 2009). Social group size is defined as the number of individuals that spend a majority of a 24-h period together in a group where there is evidence of social cohesiveness (Jones *et al*. 2009). We used social group size as an indicator of interindividual contacts that can facilitate the transmission of directly transmitted pathogens. Data for which a high proportion of species had mean group size >1 were available for primates (n = 166 species) and ungulates (n = 92 species, both Artiodactyla and Perissodactyla); data on carnivore social group size estimates were not analysed because of the relatively small number of species with reported social group size >1. Group size estimates were regressed against mean adult body mass (g) for each species on a log–log scale (Fig. 1a).

In addition, we measured the intensity of home range use by calculating the defensibility index (D-index), a metric based on the ratio of day range length (DRL) (the absolute distance traversed by an animal within a 24-h period; DRL) to home range size (the area used by an animal on a daily or seasonal time-scale; Mitani & Rodman 1979; Nunn & Dokey 2006). Intuitively, hosts that traverse large home ranges (those with high DRL) come into contact with a greater diversity of habitats and may therefore have a higher likelihood of contacting other hosts and/or

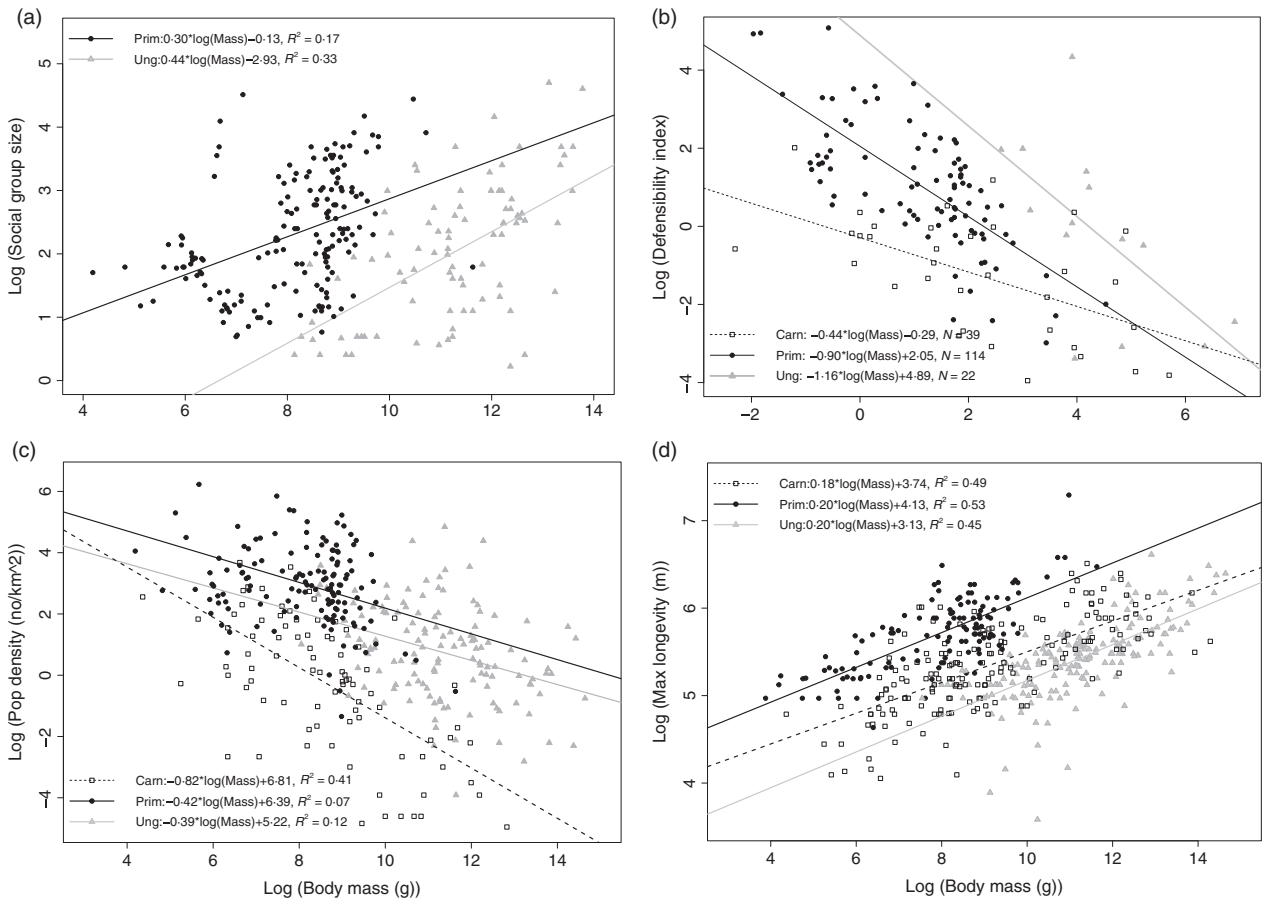


Fig. 1. Allometric scaling of social group size, defensibility index, population density and maximum longevity for primates, ungulates and carnivores on a log–log scale. Relationships are shown for host groups for which relevant data were available. Scaling coefficients for each host clade are found within regression equations (e.g. primate social group size scales with body mass with slope (b) = 0·30 and y-intercept (a) = −0·13). All relationships were statistically significant at the $\alpha = 0{\cdot}05$ level based on regression analysis. Global scaling coefficients and R^2 values are social group size: slope (b) = 0·10, y-intercept (a) = 1·23, R^2 = 0·04; defensibility index: slope (b) = −0·67, y-intercept (a) = 1·50, R^2 = 0·36; population density: slope (b) = −0·74, y-intercept (a) = 8·94, R^2 = 0·57; maximum longevity: slope (b) = 0·09, y-intercept (a) = 4·57, R^2 = 0·17. All global relationships were statistically significant at the $\alpha = 0{\cdot}001$ level.

environmentally transmitted macroparasites. However, previous work showed that the intensity of range use, rather than the absolute distance traversed per day, is a better predictor of macroparasite infection. For example, the species richness of macroparasites transmitted through 'non-close' contact was greater in mammal hosts repeatedly utilizing a defended home range, and large home ranges decreased macroparasite transmission (Bordes *et al.* 2009). Thus, the probability of host infection might increase as a host uses a given habitat more intensely and re-acquires infectious stages shed into the environment. In support of this prediction, bovids that showed greater territoriality (i.e. more intense use of a finite habitat) had more intense infections by parasitic helminths and protozoa (Ezenwa 2004b). Our model reflects these empirical patterns, and we assume that the intensity of home range usage scales positively with non-close-contact transmission of macroparasites. To calculate D-index, we obtained data on DRL (the distance traversed by an animal within a 24-h period) for primates (n = 114), carnivores (n = 39) and ungulates (n = 26) from (Carbone *et al.* 2005), and home range sizes for each species from PanTHERIA (Jones *et al.* 2009).

Because many macroparasites and some microparasites persist in host populations over long time-scales, in addition to D-index we examined whether the effects of allometric scaling in parasite transmission depended on the relative effects of host vital rates. Using data available from PanTHERIA, we quantified allometric scaling of host longevity (in years, taking the inverse to represent host mortality rate), and population density (animals km^{-2}, as a correlate of carrying capacity) for ungulates (n = 174), primates (n = 125 species) and carnivores (n = 169 species; Fig. 1c,d). Host longevity is defined in PanTHERIA as the maximum adult age of males or females from captive, wild or provisioned populations, measured either through direct observation, capture–recapture estimates or projected from physical wear (Jones *et al.* 2009). Population density was estimated through either direct or indirect counts of males and females, measured in any sized area within a defined boundary over any duration of time using non-captive, non-provisioned populations (Jones *et al.* 2009). Though not yet well characterized, allometric scaling patterns could also affect within-host dynamics of infectious diseases and might be important for parameters such as host recovery and disease-induced mortality (Cable, Enquist & Moses 2007), in addition to predicting the fitness of adult parasites within a host. Although these other complexities of scaling are interesting, data needed to test the relationships between host body size and relevant parameters were not available for the present study, and therefore, they were not explored in the analyses described here.

It is well-established that closely related species show similar values for many traits (Felsenstein 1985), and this pattern has been shown for many traits of carnivores (e.g. Gittleman 1985), primates (e.g. Nunn & Barton 2000) and ungulates (e.g. Mysterud, Pérez-Barbería & Gordon 2001). Rather than to confirm that relatedness is an underlying driver of allometric scaling, our goal was to model the consequences of traits for infection dynamics. Thus, we followed previous authors (De Leo & Dobson 1996; Cable, Enquist & Moses 2007; Bolzoni *et al.* 2008a,b; Brose 2010) by retaining phylogenetic signal in scaling coefficients to parameterize models based on relationships as they are observed in nature.

MODEL: ALLOMETRICALLY SCALED MICROPARASITE TRANSMISSION

We extended a basic differential equation model that describes changes in the number of susceptible (S), infected (I) and recovered individuals (R) in a population (Fig. 2a) following (Anderson & May 1991). To isolate the effects of host behaviour on pathogen transmission (and because we assumed a rapid acute infection process), we initially excluded host birth and death processes in the absence of infection, but assumed that infected individuals can die from disease (at per capita rate, α) or recover from infection (at per capita rate, γ). Following previous work (e.g. Getz & Pickering 1983; Heesterbeek & Metz 1993; Thrall, Antonovics & Wilson 1998; McCallum, Barlow & Hone 2001), we assumed that the microparasite transmission rate (β) is a product of the contact rate (f) among hosts in a population and the per capita probability of parasite transmission (i.e. the probability that infection will be transmitted for any given contact between a pair of S and I individuals; τ). For closed social groups, parameter f is the contact rate in the group and for open social groups, parameter f reflects the proportion of the overall population that typical individuals have contact with (in which case f can increase by group size or membership turnover). In all cases, the transmission rate is $\beta = f\tau$. We adopted the standard expression for allometric scaling, aM^b, where M refers to body mass, and a and b represent the intercept and slope of a scaling relationship between body mass and group size (Fig. 1). In our model, contact rate scales with group size in accordance with the classic model of density-dependent transmission, $f = aM^b$. Consequently, $\beta = \tau aM^b$. Compartmental models and corresponding equations incorporating allometric scaling of contact rates are provided in Fig. 2.

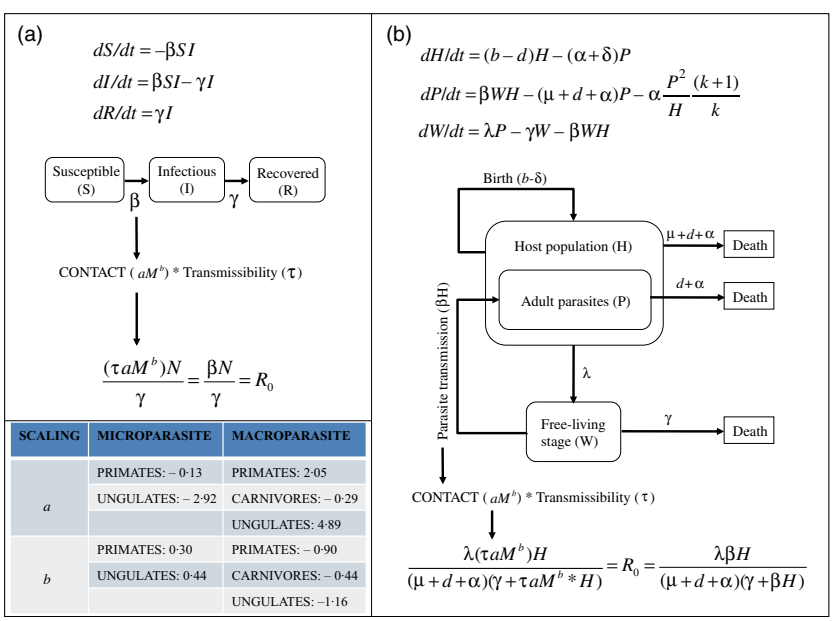
Using these equations, we examined how the basic reproductive number of a directly transmitted pathogen (R_0) changes with allometric scaling of transmission through social contact. R_0 is the number of secondary infections produced by one infectious individual introduced into a wholly susceptible population (Anderson & May 1991; Dobson & Hudson 1992) and must exceed 1 for a pathogen to invade the host population. R_0 expressions for the allometrically scaled and unscaled models are given in Fig. 2a. In our exploration of R_0, τ was set at the value forcing R_0 through 1 at the average body mass for all species within a clade, and, thus the allometrically scaled expression is

$$R_0 = \frac{(a'aM^b)N}{\gamma}$$

All parameters except for body size were fixed; thus, predicted values of R_0 reflect the dynamics of a particular pathogen infecting multiple host species spanning a broad range of body sizes. Due to the simple nature of this model, the loss term, γ, can take on an inclusive definition extending to, for example, loss of infected individuals through disease-induced mortality and host recovery from infection.

MODEL: ALLOMETRICALLY SCALED MACROPARASITE TRANSMISSION

We extended a system of three ordinary differential equations (e.g. Dobson & Hudson 1992) describing the dynamical interactions between adult macroparasites (P), a host population (H) and free-living parasite larvae (W) (Fig. 2b). Hosts have per capita birth and death rates (b) and (d) independent from effects of the macroparasite. The macroparasite transmission parameter (β) comprises both the encounter rate between hosts and parasite



SCALING	MICROPARASITE	MACROPARASITE
a	PRIMATES: − 0·13	PRIMATES: 2·05
	UNGULATES: − 2·92	CARNIVORES: − 0·29
		UNGULATES: 4·89
b	PRIMATES: 0·30	PRIMATES: − 0·90
	UNGULATES: 0·44	CARNIVORES: − 0·44
		UNGULATES: −1·16

Fig. 2. Compartment models illustrating population dynamics of infectious microparasites (a) and macroparasites (b) within a host population, and their respective systems of ordinary differential equations with corresponding expressions for the parasite's basic reproductive number, R_0, and illustrating how allometric scaling coefficients were incorporated into the transmission term, β. The inset table shows scaling coefficients [y-intercepts (a) and slopes (b)] derived by regressing data on host behavioural traits against body mass for primate, ungulate and carnivore species.

larvae, and the probability of successful parasite transmission given an encounter (τ). Adult parasites have background death rates within the host (μ), and also die if their host dies of either natural causes (*d*) or due to infection (α). The larval population depends on the fecundity of adult parasites (λ), larval mortality rate in the environment (γ) and the rate at which larvae are transmitted successfully to new susceptible hosts (β). We modelled macroparasite aggregation in host populations by assuming a negative binomial distribution with dispersion parameter, *k*. Infected hosts can suffer a decrease in fecundity due to macroparasite burden. For a host infected with *x* macroparasites, fecundity is adjusted to $b - x\delta$, and host mortality is given by $d + x\alpha$, where δ and α are the per capita effects of each adult parasite on host fecundity and mortality, respectively.

Assuming that parasite transmission occurs through encountering infectious stages in the environment (e.g. if parasites are shed through faeces or urine), transmission is expected to increase with the intensity of home range use. We found that *D*-index (intensity of home range use) decreases with host body mass as aM^b in carnivores and primates (e.g. Fig. 1b). As in the microparasite model, *M* is mean adult body mass, and *a* and *b* represent the intercept and slope of a linear scaling relationship between body mass and *D*-index on a log–log scale. For the macroparasite model, R_0 refers to the number of parasites produced by an adult parasite over its life span. Allometrically scaled and unscaled expressions for R_0 are given in Fig. 2b.

Because host birth and death rates are known to scale with body size for many animals, and because many macroparasite infections occur on longer time-scales over which host birth and death are likely to occur, we examined a second iteration of R_0 of macroparasites that included scaling of host demography using empirical data from primates and carnivores (Fig. 1c,d) to scale population density and mortality rate (the inverse of host longevity). Host population density is described as gM^h (for primates $g = 5{\cdot}21$, $h = -0{\cdot}28$; for carnivores $g = 6{\cdot}81$, $h = -0{\cdot}82$). Host mortality rate is described as cM^d (for primates, $c = 4{\cdot}13$, $d = 0{\cdot}20$ and for carnivores $c = 3{\cdot}74$, $d = 0{\cdot}18$). The new expression for R_0 is given by:

$$R_0 = \left(\frac{\lambda(a' a M^b)(gM^h)}{(\mu + \alpha + \frac{1}{cM^d})(\gamma + (a^1 a M^b)(gM^h))} \right)$$

As with the microparasite model, τ was set at the value forcing R_0 through 1 at the average body mass for all species within a clade, and all parameters except for body size were fixed. Other parameters specific to macroparasites (e.g. longevity and within-host development and fecundity) are also likely to scale with host body size. We did not allometrically scale these parameters because comprehensive data on parasite longevity and fecundity in relation to host body size were not available for this analysis.

EMPIRICAL TESTS OF MODEL PREDICTIONS

Hosts exhibiting behaviours that promote parasite fitness should accumulate more parasite species for which $R_0 > 1$ (Morand 2000; Nunn *et al.* 2003). Moreover, because simple epidemiological models predict a direct relationship between R_0 and equilibrium prevalence, pathogen prevalence might represent a proxy for pathogen invasion (Keeling & Rohani 2008). To test model predictions for micro- and macroparasites, we examined relationships between host body size and parasite prevalence and species richness. We obtained data from the Global Mammal Parasite Database (Nunn & Altizer 2005) on parasites recorded in the primary literature to infect free-living populations of primate, ungulate and carnivore species. Microparasite data were restricted to include viruses and bacteria transmitted directly through close non-sexual contact (defined by close proximity or direct contact such as through biting, scratching or other touching; Pedersen *et al.* 2005). For macroparasites of primates, we selected helminth species transmitted environmentally, defined as transmission via fomites, contact with contaminated soil or water, or through the consumption of intermediate hosts (Pedersen *et al.* 2005). Data on transmission mode for macroparasites of carnivores and ungulates were not available; thus, analyses for these groups were restricted to helminths under the assumption that the vast majority of helminths infecting mammals have external transmission stages (Poulin & Morand 2000; Roberts & Janovy 2009).

Parasite data collected from wild host species can reflect different kinds of sampling bias, with more parasite species being reported from better studied hosts (Walther *et al.* 1995). For our analysis, it was important to determine whether sampling bias varied predictably with host body size, and to control for sampling effort in estimating parasite richness. We followed previous studies in using citation counts from Web of Science for the Latin binomial of each host species as an index of sampling effort; this measure indicates how well a host species has been broadly studied and is known to correlate positively with measures of parasite richness (Nunn *et al.* 2003; Lindenfors *et al.* 2007). Additionally, larger host species might simply be better studied overall (due to being more apparent on the landscape, or of greater conservation concern, for example Cardillo *et al.* 2005) and thus might be better sampled for parasites. We tested for relationships between body size and sampling effort within each mammal order to identify where such sampling biases may exist, and we also included sampling effort as a covariate in parasite richness tests described below.

Prevalence

We used Spearman's rank correlation to examine relationships between parasite prevalence and adult body mass, to test whether microparasite prevalence increased with body mass (as might be mediated by social groups size) and whether macroparasite prevalence declined with body mass (as mediated by home range use). We first identified parasite species that had information on prevalence for host species differing in body mass (Appendix S1, Supporting information), and tests were conducted for microparasites and macroparasites separately for each host order.

Parasite species richness

Next, we tested whether parasite species richness varied with adult body size, following previously published protocols for compiling parasite richness from records of infections in wild mammal populations (Nunn *et al.* 2003). We predicted that microparasite richness would increase with host body size, and that macroparasite richness would decrease with body size, based on modelling results and the rationale described above. We tested for significant associations between body mass and parasite richness using multivariate regression (carnivores and ungulates) and Spearman's rank correlation (for primates, due to non-normally distributed errors). We accounted for uneven sampling effort in

these analyses by including the number of Web of Science citations per host species as a covariate and by using the residuals of parasite species richness regressed against citation counts as the dependent variable in rank correlation tests. All variables (parasite richness, citation counts and body mass) were $\log_{10}$-transformed prior to analysis.

Hosts of well-studied parasites

We conducted a final test to find whether the body size distribution of hosts infected by a subset of well-sampled parasites differed from that of randomly selected hosts within the same taxonomic order. This approach was taken to account for the likelihood that specialist parasites from smaller-bodied hosts might be less well known, and thus not represented in tests of parasite richness. By focusing on the host distribution of commonly reported parasites, our goal was to examine the body sizes of affected hosts relative to all others in our data set. We first identified parasites infecting five or more host species per order. We compared the body sizes of the hosts infected by this subset of parasites to the distribution of body sizes for randomly selected hosts from across the same clade. For each clade, we applied one-tailed Wilcoxon rank sum tests to examine whether body sizes were larger for hosts infected by microparasites relative to randomly chosen hosts, and smaller for hosts infected by well-studied macroparasites relative to randomly chosen hosts. We used the R statistical environment for all analyses, modelling and figures (R Core Team 2014). All parasite species richness and prevalence data for microparasites and macroparasites; defensibility index data for primates, carnivores and ungulates; and code for R_0 simulations for both micro- and macroparasites are available in the Dryad Digital Repository, doi: 10.5061/dryad.kr202.

Results

EMPIRICALLY DERIVED SCALING COEFFICIENTS

Social group size (an indicator of contact rates) increased significantly with host body size (Fig. 1a), with this relationship scaling more steeply for ungulates than for primates. *D*-index (indicating the intensity of habitat use) scaled negatively with host body mass (Fig. 1b); here, the scaling exponent declined more steeply for carnivores than for primates. As *D*-index is comprised of both DRL and home range size, we also examined whether one of these metrics might be driving the overall negative allometric scaling of *D*-index. We found that DRL scaled positively with body size for carnivores and ungulates, but there was no allometric scaling in primates. Home range size also scaled positively for carnivores, ungulates and primates. Thus, the negative relationship between body size and *D*-index was driven by a steeper positive slope for home range size and body size, relative to the more shallow slope for DRL and body size within these clades (Fig. S1, Appendix S2, Supporting information). Population density decreased significantly with body size for both primates and carnivores (Fig. 1b,c), and as expected, host longevity scaled positively with body size for both primates and carnivores (Fig. 1d). The scaling coefficients we observed fell within previously published ranges (Peters 1986 and references therein; Gittleman 1985; Wrangham, Gittleman & Chapman 1993; West, Brown & Enquist 1997; Carbone & Gittleman 2002; Carbone *et al.* 2005) . The scaling coefficients (slopes and intercepts) provided in Fig. 1 were incorporated into micro- and macroparasite models as indicated in the Materials and methods text, with results described below.

MODEL PREDICTIONS FOR PARASITE INVASION

For microparasites, scaling transmission according to empirically derived coefficients for social group size (Fig. 2) showed that R_0 increased with body size for both primates and ungulates (Fig. 3a). When host body size was small, the probability of invasion was greater for primates than for ungulates, and the probability of invasion as a function of body size increased more steeply for ungulate species. Conversely, scaling the transmission of macroparasites based on coefficients for *D*-index showed that R_0 decreased with body size for both primates and carnivores (Fig. 3b). In this case, the probability of pathogen invasion for the smallest species was lower for carnivores than for primates, but R_0 for moderate and large species was similar for both groups. The negative relationship between macroparasite R_0 and host body size was unaffected by the addition of allometric scaling in host population density and host mortality. This is due to the relatively small contribution of host longevity to R_0 and the saturating effect of host population density on R_0. Consequently, our analysis predicted no qualitative change in the relationship between body mass and R_0 after incorporating host demographic scaling. Although allometric scaling of demographic parameters was not explicitly included in the microparasite model (envisioning instead acute infections that spread on a fast time-scale relative to population turnover), earlier results on allometric scaling and demography (De Leo & Dobson 1996) indicated that behavioural and demographic components of transmission would reinforce each other in density-dependent transmission scenarios, with both factors leading to an increase in transmission as body mass increases, although through different processes.

EMPIRICAL SIGNATURES OF MODEL PREDICTIONS

Empirical tests of relationships between host body size, parasite prevalence and parasite species richness are summarized in Table 1, with statistical details provided in Appendix S1 (Supporting information). In general, we found support for the predictions of allometrically scaled models of micro- and macroparasites. In ungulates, microparasite prevalence increased with host body size (Spearman's rank correlation = 0·078, $n = 49$, $P = 0{\cdot}045$), and hosts infected by well-studied microparasites were larger in size compared to randomly sampled hosts from

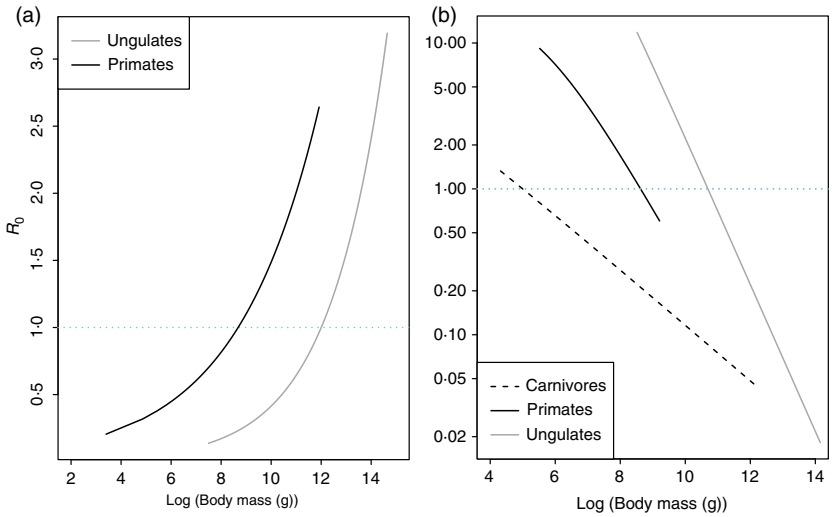


Fig. 3. Predicted values of R_0 based on mathematical models applied across mean body masses of primates and ungulates for close-contact transmitted microparasites (a), and primates, carnivores and ungulates for macroparasites transmitted via non-close contact (b). For panel a, mean adult body mass (M) ranged from 30 to 150 000 g for primates and 1800–2 285 900 g for ungulates based on the smallest and largest species; values for model parameters a and b were obtained through regression described in the Materials and methods; other parameters were set at: τ (primates) = $8{\cdot}42 \times 10^{-4}$, τ (ungulates) = $9{\cdot}06 \times 10^{-4}$, $\gamma = 0{\cdot}01$. For panel b, M ranged from 250 to 10 000 g for primates, and 75–200 000 g for carnivores and 5000–1400 000 g for ungulates; parameters a and b were obtained through regression described in the Materials and methods and depicted in Fig. 1; other parameters were set at: τ (primates) = 100·6, τ (carnivores) = 0·387, τ (ungulates) = 10007·8, H = 10, $\lambda = 10$, $\mu = 0{\cdot}5$, $\alpha = 0{\cdot}005$, $\gamma = 8{\cdot}0$, d (primates) = 326·7, d (carnivores) = 237·98, d (ungulates)=244·8, $\beta = 0{\cdot}5$.

Table 1. Empirical tests of predictions from allometrically scaled models of microparasites of ungulates and primates, and macroparasites of carnivores, primates and ungulates

	Microparasites		Macroparasites			
Question	Ungulates	Primates	Primates	Carnivores	Ungulates	Test
1. Are larger host species better studied?	No	Yes	Yes	No	No	Linear regression
2. Is there a correlation between prevalence and body mass?	S*	S*	X*	X*	ns	Spearman's rank correlation
3a. Is PSR influenced by host body mass and/or citation count?	S*	//	//	S*	S*	Multivariate regression
3b. Is there a correlation between residual PSR and body mass?	//	ns	ns	//	//	Spearman's rank correlation
4. Does body size differ between hosts with high vs. low parasite species richness?	ns	ns	X*	S*	S*	Wilcoxon rank sum test
5. Are body sizes of hosts infected by well-studied parasites different from hosts selected randomly across the clade?	S*	S*	S*	ns	ns	Wilcoxon rank sum test

PSR, parasite species richness; S*: statistically significant results in the predicted direction (at α=0·05); X*: statistically significant results in the opposite direction from model prediction; ns, not statistically significant; NA designates analyses that were not relevant (all clades were analysed with multivariate regressions, except for the primates which violated normality assumptions and were therefore analysed using Spearman's rank correlations, 3b).
Detailed statistical results of all tests are given in Appendix S1, Supporting information.

across the clade (Wilcoxon rank sum test = 1658·8, nA = nB = 52; $P = 0{\cdot}02$, one-tailed test). For primates, we found that larger primate species were indeed better studied ($F_{1,34} = 12{\cdot}19$, P-value = 0·001), and that hosts infected by the best-studied microparasite species were larger relative to randomly sampled hosts from across the clade (Wilcoxon rank sum test = 302·5, nA = nB = 20; P-value < 0·003, one-tailed test). In carnivores, macroparasite analyses showed a negative relationship between parasite richness and host body size. However, macroparasite prevalence increased with host body size in this group (Spearman's rank correlation = 0·050, $n = 9$, $P = 0{\cdot}003$). Counter to model predictions, we also found that macroparasite species richness increased with body size in primates. However, primates infected by well-studied macroparasites were smaller compared to primates

sampled randomly across the clade (Wilcoxon rank sum test = 657, nA = nB = 30; $P < 0{\cdot}002$, one-tailed test). In ungulates, we found that host species with higher macroparasite species richness were smaller than those with low parasite richness (Wilcoxon rank sum test = 347, nA = 25, nB = 40; $P = 0{\cdot}04$, two-tailed test), consistent with model predictions.

Discussion

We combined empirical data on mammal body size and behaviours with population dynamic models to predict the invasion probability for micro- and macroparasites across multiple host groups. Scaling pathogen transmission through behavioural allometry led to opposite predictions about the types of hosts most easily invaded by micro- vs. macroparasites. In particular, large-bodied primates and ungulates living in large social groups were predicted to be more susceptible to invasion by directly transmitted microparasites compared to small-bodied species living in smaller social groups. In contrast, for macroparasites transmitted through external infectious stages, our model predicted that smaller primates, carnivores and ungulates, which typically utilize their home ranges more intensively, would be at greater risk for macroparasite invasion than larger-bodied species. These findings support a growing number of studies suggesting that home range size is inversely linked with infection risk, and that territoriality increases infection risk in numerous mammal species (Ezenwa 2004b; Nunn & Dokey 2006; Lindenfors *et al.* 2007; and reviewed in Bordes *et al.* 2009). Empirical analyses using prevalence and parasite species richness generally supported our model predictions with the exception of results for macroparasite prevalence in carnivores.

Allometric scaling relationships are best known for their generality, describing relationships between body size and a diversity of life history and physiology variables for organisms spanning multiple kingdoms across many orders of magnitude in size (Peters 1986; West & Brown 2005; Sibly, Brown & Kodric-Brown 2012). Scaling factors derived from large-scale data sets on animal life-history traits such as mortality and population density were previously incorporated into population dynamical models of infectious diseases (De Leo & Dobson 1996; Bolzoni *et al.* 2008a,b). Here, we report scaling relationships for behavioural traits that generally show more noise in relation to body size than more commonly analysed life-history traits (Dial, Greene & Irschick 2008). For primates in particular, body size explained only 17% of the total variation in social group size in our analysis. By comparison, body size explained *c.* 86% of the variation in population density from an analysis of data aggregated across multiple taxonomic groups of animals (Peters 1986). Our work showed that body size explained a greater degree of variation in defensibility index across primates and carnivores (38–53%), suggesting that some behavioural measures scale more closely with body size. It is more important to note that here we derived order-specific scaling coefficients for different mammal groups, whereas past studies generally incorporated scaling factors derived from data aggregated across multiple animal groups. Applying coefficients derived at larger taxonomic scales into models applied at smaller taxonomic scales (e.g. within a host class or order) could subsume a potentially large degree of variation and reduce confidence in model outputs.

Our empirical analyses of infection data from wild mammal–parasite associations supported theoretical predictions of R_0 in many cases, although we note that prevalence and richness might serve only as weak proxies for R_0 (Nunn *et al.* 2003; Keeling & Rohani 2008). Consistent with model predictions, the prevalence of well-studied microparasites increased with body mass for ungulates, and ungulates infected by well-studied microparasites were larger than a random sample of ungulates. For primates, hosts infected by well-studied microparasites were generally larger relative to randomly sampled primates. For primates and ungulates of similar body size, primates were expected to show a greater microparasite R_0 owing to the steeper allometric scaling of their social group sizes. For macroparasites, parasite richness declined with body size in both carnivores and ungulates. Likewise, primate hosts infected by well-studied macroparasites were smaller than randomly sampled hosts. We note that, counter to model predictions, macroparasite prevalence increased with body size in carnivores. This pattern that might arise from several underlying causes, including the fact that macroparasites tend to be highly aggregated in host populations, with a high fraction of the parasite population harboured by a small number of hosts (Shaw & Dobson 1995; Wilson *et al.* 2002). Thus, for macroparasite species that show high aggregation, prevalence might not represent the best index of parasite transmission. It is also important to note that additional traits, such as investment in immune defence or host behavioural defences could confound field observations of parasite species richness, particularly if such traits also scale allometrically. For example, pathogenesis has been shown to scale positively with host metabolism for some infectious agents with environmental stages (e.g. anthrax; Cable, Enquist & Moses 2007), which runs counter to the negative scaling of home range usage that we observed across ungulates and primates.

Findings here indicate that host body mass could predict patterns of parasitism observed at the level of host communities For example, for directly transmitted acute microparasitic infections, host communities comprised of many large-bodied species might harbour more parasites compared to communities dominated by smaller-bodied species. In the case of macroparasites with free-living infectious stages, host communities dominated by small-bodied species should harbour more parasites. Host–parasite associations will further depend on geographical and ecological constraints (e.g. spatial overlap among host

species, parasite tolerance of environmental conditions) and on biotic interactions governing coinfection within individual hosts (e.g. Graham 2008).

Collectively, our results illustrate how parasite invasibility can be modulated by host behaviour and predicted by host body mass, based on assumptions linking R_0 to processes that govern exposure to infectious stages. One exciting area for future research will be develop a unified modelling framework that combines the scaling effects of transmission, host demography and within-host dynamics. For example, the rate at which a host develops disease following initial infection by microparasites was shown to scale negatively with host metabolic rate (Cable, Enquist & Moses 2007). In our framework, the joint effects of contact rate and disease-induced mortality would reinforce the positive relationship between host body size and microparasite fitness, but the next outcome for R_0 would depend on the relative strengths of other scaling relationships as well. A synthesis of more data is required to facilitate a comprehensive view of allometric scaling of pathogen transmission dynamics. Finally, given that most hosts are infected by multiple parasites, we note that macro- and micro-parasite transmission is more than likely to occur simultaneously in any host group in nature. Thus, there is great potential to incorporate the joint effects of scaling on interactions between micro- and macroparasites, which could act synergistically or antagonistically to amplify or mute the effects of allometry on wildlife disease.

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Data accessibility

Data on parasite prevalence and species richness as well as code for R_0 simulations for both micro- and macroparasites have been published in the Dryad Digital Repository, http://dx.doi.org/10.5061/dryad.kr202 (Han *et al*. 2015).

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Supporting Information

Additional Supporting Information may be found in the online version of this article.

Appendix S1. Statistical analyses summarized in Table 1.

Appendix S2. Statistical analyses of allometric scaling in day range length and home range size for primates, ungulates, and carnivores.

Fig. S1. Allometric scaling of day range length (km) (mean distance traversed in 24 h) and home range size (km^2) for primates, ungulates and carnivores on a log–log scale.