Behavioral Correlates of Parasite Risk Among Humans, Primates, and Other Mammals: Social Contact, Exploratory Tendency, and the Foundations of Culture

Behavioral correlates of parasite risk among humans, primates, and other mammals: Social contact, exploratory tendency, and the foundations of culture

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Abstract

Social learning and innovation are the behavioral processes that together create the emergent phenomenon of culture, which allows organisms to behaviorally adapt to and thrive in new environments. However, these processes can be difficult to quantify in practice. Instead, social contact and environmental exploration are two measurable behavior patterns that underlie the processes of social learning and innovation, respectively. The benefits afforded by these behavior patterns are many, but they also have their costs. Specifically, I test the hypothesis that the social and exploratory behaviors of animals influence their infection by parasites. To better understand the association between cultural behavior and parasitism, I utilize three complementary analytical approaches in this dissertation, each with different study systems, and each focusing on distinct facets of this association.

In the first study, I utilized phylogenetic comparative methods among 127 primate species to investigate two competing hypotheses about the broad associations between cultural behavior and parasitism: that cultural behaviors increase exposure to parasites, or that parasite infection drives the emergence of cultural behaviors as compensatory mechanisms. I investigated

these hypotheses by assembling datasets on parasite richness and recorded instances of social learning, innovation, and exploration. Bayesian Markov Chain Monte Carlo Phylogenetic Generalized Least Squares (PGLS) analyses indicated that the variety of social learning behaviors covaried positively with richness of socially transmitted parasites, but not with richness of environmentally transmitted parasites. Conversely, the variety of innovative and exploratory behaviors for a primate species covaried positively with environmentally transmitted parasite richness but not with socially transmitted parasite richness. This provided support for the hypothesis that cultural behaviors increase exposure to parasites.

Delving further into the social facet of cultural behavior and parasite exposure, my second study employed stochastic simulations of parasite transmission across theoretical populations to investigate how patterns of social contact within groups can impact parasitism. Simple group size indices have proven to be poor predictors of disease risk within a group, and so more complex metrics of social contact merit investigating. By simulating disease transmission through social networks, I developed a novel method for simultaneously accounting for the effects of the structure and size of groups, with respect to disease outbreaks. This structure-standardized group size, which I called "effective network size" was then used in PGLS analyses of 22 primate species for which social network structures had been published to determine whether effective network size was a better predictor than group size of parasite risk, measured by parasite species richness. I found that effective network size performed no better than raw group size, but that the approach has promise for further applications.

In my third study, which focused on the exploratory facet of behavior and parasite risk, I designed a longitudinal field study to investigate how exploratory tendencies in a model taxa, rodents, affected their parasite infections as well as their likelihood of associating with humans,

which may ultimately affect human exposure to rodent-borne diseases. I predicted that more exploratory rodents, which are expected to interact with their environments more intensely and to venture more often into new environments, would have a greater richness and intensity of parasites and would be found more often in homes (commensal) than less exploratory rodents. I captured rodents in homes and wildlife conservancies in central Kenya and assessed the exploratory tendencies of each captured rodent. I also collected gastrointestinal and ectoparasites from them. As expected, commensal rodents were more exploratory than wild rodents, and these more exploratory individuals had greater intensities of ectoparasites and gastrointestinal parasites. However, contrary to my prediction, commensal rodents had a lower average richness of gastrointestinal parasites. Thus, exploratory behaviors were predictive of parasite infection intensity but not richness. These more heavily burdened animals in human homes may also play an important role in transmitting their parasites to humans.

As my three complementary studies show, the social and exploratory behaviors that form the foundations of human and animal culture can have significant impacts on parasitism. These increases in parasite diversity and infection intensity may be most apparent in humans, a species which is undoubtedly dependent on culture for their success and livelihood, from hunter-gatherers to farmers.

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Dedication

For dad

Chapter 1

Introduction: How do host behavior and parasite distribution interact to produce infection risk?

Behavior plays a central role in many aspects of an organism's life. Depending on the outcome of a behavior, an animal may either successfully attract a mate or may fail in this endeavor; it may either escape from a predator or it may be killed. Thus, the outcome of behavior has direct consequences on the fitness of the behaver, and behaviors that increase fitness will become more prevalent in populations through the process of natural selection (Tinbergen, 1963). However, a behavior that is advantageous in one context may have negative consequences in another, presenting fitness tradeoffs for the behaver. For instance, by digging around in new parts of its environment, a chipmunk may be better able to find food and sustain itself, but it may also be exposing itself more often to questing ticks on trees or larval stages of gastrointestinal parasites in the soil (Boyer et al., 2010). Similarly, by living in a large, gregarious herd, a zebra may be protected against predators, but it will also be exposed to greater numbers of parasitic worms from its groupmates (Ezenwa, 2004). In this dissertation, I consider the fitness tradeoffs of such social and exploratory behaviors in the context of exposure to and infection with parasites.

Parasitism can have diverse negative effects on host fitness. For the sake of this dissertation, the term "parasite" refers to any disease-causing agent ranging from pathogens, sometimes referred to as micro-parasites, to macro-parasites, such as helminths, ticks, and fleas. Of course there are the obvious negative impacts of parasites on fitness via morbidity (Bush et al., 2001) and mortality (Milton, 1996; Formenty et al., 1999; Walsh et al., 2003). However, parasites can also have negative impacts on cognitive development and function, making it more difficult to compete in cognitively demanding environments (Kavaliers and Colwell, 1995).

Parasites can also hinder growth, making it more difficult to physically compete with conspecifics for mates and with other species for resources (Checkley et al., 2008), and they can require individuals to allocate more time and energy to resting and immune function (Hart, 1990). These fitness consequences of parasitism are often amplified when a greater diversity of parasites are found in a host or species, leading to multiplicative fitness effects from co-infection; for example, co-infection leads to higher host mortality in a wide variety of host and parasite taxa (Jolles et al., 2008; Ezenwa et al., 2010; Bordes and Morand, 2011). The resting and reduction of activity in sick individuals can itself increase the likelihood of coinfection by making these less mobile hosts easier targets for disease vectors such as mosquitoes, ticks, and fleas (Moore, 2002).

Parasitic infection risk can be measured in number of ways, and for the purposes of this dissertation, I will focus on the most widely used of these: intensity, prevalence, and richness (Nunn and Altizer, 2006). Infection intensity refers to the quantity of a specific parasite or group of parasites within an infected host. Prevalence is defined as the proportion of potential hosts in a population that are infected with a particular parasite species. Parasite species richness is the number of the different species of parasites infecting a given individual, population, or host species.

Parasites can also be split into broad categories based on their modes of transmission (Nunn and Altizer, 2006). Close-contact transmission can occur through coughing, sneezing, touching, sexual contact, and other behaviors that involve close contact among different individuals. Non-close-contact transmission entails transmission through the environment through media like soil and water; these modes often require ingestion of or contact with infectious substrates. Similar to non-close-contact transmission, vector-borne parasites and those

requiring intermediate hosts are also transmitted through the ingestion of infectious substrates (in the case of infected intermediate hosts), or contact with vector habitats, like brush in which questing ticks may live. Additionally, these transmission modes are not necessarily mutually exclusive; the same parasite that is transmitted by close contact from coughing or sneezing can also persist in the environment on fomites. The type of contact required for each transmission mode is also salient to the behaviors which may affect transmission; social interactions are likely to increase close-contact transmission (Thrall and Antonovics, 1997), and exploratory behaviors should increase the transmission of non-close-contact transmitted parasites.

Given the fitness effects that parasitism can have on hosts, natural selection is expected to shape traits and behaviors that alter the risks of parasitism. On the individual level, behavior's association with parasitism is typically split into two broad categories: behaviors that affect an individual's exposure to parasites and behaviors of a host in response to, or manipulated by parasites. However, parasitism can also impact the behavior of populations through selective pressures on specific types of behaviors (Barber and Dingemanse, 2010). Being in an environment with more socially transmitted parasites could in theory select over time for less sociable animals, while being in a habitat with more environmentally transmitted parasites could select for less exploratory animals. This represents a shift of the balance between costs and benefits of such behaviors; for instance, exploratory tendencies can benefit animals by allowing them to find new or better food sources, but this benefit must outweigh the many costs of this tendency, including parasitism (Reader and Laland, 2001). Beyond this fact, the impacts of host behavior on parasite exposure and of parasitism on host behavior are often reciprocal, and we see this on the species level with behaviors that maximize benefits relative to costs of infection (Ezenwa et al., 2016).

Animals can have a variety of behavioral responses to infection. These include behavioral manipulation by parasites, which themselves have selective pressures to reproduce and be transmitted to other organisms; thus, these manipulations often vary by the transmission mode of the parasite. In close-contact transmitted infections like rabies, this manipulation takes the form of increasing the ranging behaviors of infected hosts so that they may interact with new potential hosts (Baer, 1991). For non-close-contact and intermediate transmitted parasites like *Toxoplasma gondii*, increasing the boldness of infected intermediate hosts can make them more likely to be consumed by definitive hosts, which in the case of *T. gondii* are cats (Berdoy et al., 2000). Sickness responses to parasite infection include not only fever, but also behavioral responses that either allow the body to shunt resources to immune functions, like increases in resting and sleep, or that reduce exposure to additional parasites, including self-quarantining and reducing intake of foods, or even self-medication to reduce or clear parasite infections (Hart, 2010). However, the line can be blurry between behavioral manipulation by parasites and sickness responses to them; for instance, a reduction of activity and movement in baboons infected with *Echinoccus* tapeworm cysts can also make them easier targets for predators, the definitive hosts of this parasite (Moore, 2002).

Behaviors that affect exposure to parasites fall into two categories: “avoidance” behaviors that reduce an individual’s encounters with parasites, and “risky” behaviors that increase these encounters. Avoidance measures include disgust reactions to specific stimuli that indicate parasite presence and grooming to remove ectoparasites (Schaller, 2006; Hart, 2011). The notion of risky behaviors has been applied to phenomena of disease exposure by human epidemiologists since the 1970s, often in the context of sexually transmitted diseases (STD), where behaviors like engaging in unprotected sexual intercourse increase exposure to STDs

(Sallis et al., 2000; Taylor-Seehafer and Rew, 2000). Risky behaviors have also been used to understand and predict infection patterns among animals (Kiesecker et al., 1999; Rich et al., 2013), and this research forms the intellectual foundation of the following chapters of this dissertation.

Exploratory behavior is defined here as venturing into novel areas, either spatially or behaviorally. Novel, in this respect, refers to the fact that such a location, object, or individual has either been previously unencountered in its entirety, or is an unencountered combination of familiar experiences. As such, this may be one of the riskier behaviors that animals engage in with regard to parasite exposure, as well as exposure to many other risks, like predators. In previous research, rather than reporting exploration as an anecdotal observation of a new behavior, exploratory tendencies are often considered to be stable, individual-level personality traits that predict the amount of exploration an organism will engage in over its lifetime (Dingemanse and Réale, 2005). The benefits of exploratory behaviors include new or more efficient solutions to problems, such as acquiring food (Reader and Laland, 2003), or the discovery of novel mating or dominance displays (Kummer and Goodall, 1985). Innovation, a behavioural process which can be measured by proxy through exploratory tendencies, has also been proposed to be a precondition for the development of culture in primates and humans (van Schaik et al., 1999; Day et al., 2003; Reader et al., 2011). But exploratory behavior is not without its costs: trying new solutions to old problems carries obvious risks, such as poisoning from eating toxic novel foods, and new opportunities for predation and injury from interacting with new objects, environments, or individuals (Reader and Laland, 2001).

Exploratory behaviors can also increase the likelihood that individuals encounter parasites in the environment and are infected with these parasites (Barber and Dingemanse,

2010). Previous studies have found that increased exploratory tendency is associated with higher levels of parasite exposure and infection in a variety of taxa, and at different scales, from individual to species-level effects. The first study to suggest this relationship anecdotally indicated that pumpkinseed fish captured in fish traps (i.e. that were more exploratory) tended to have higher infection intensity of blackspot trematodes, but lower intensities of whitegrub trematodes than the overall pumpkinseed population (Wilson et al., 1993). Among mammals, a single study of chipmunk personality has found that individuals with greater exploratory tendencies, as measured by novelty-seeking (neophilic) responses in hole-board tests, had greater infestations intensities of ticks (Boyer et al., 2010).

The vast majority of research on exploratory behvaior and parasitism, however, has been conducted in birds, as personality and behavioural tendencies have been studied for decades within this clade (Spence, 1960). In great tits, behavioural assays have indicated that more exploratory females, but not males, were more likely to be infected with avian malaria (Dunn et al., 2011a). In comparative studies of 108 avian taxonomic families, the number of recorded innovations for a species, as a proxy for exploratory tendency on a species level, was positively associated with louse infestation intensity (Garamszegi et al., 2007; Soler et al., 2011; Vas et al., 2011), and vector-borne haematozoan prevalence was positively associated with the number of feeding innovations recorded for a species (Garamszegi et al., 2007). In addition to direct measures of parasite risk, the previous study on feeding innovations in birds found that these behaviors were also positively associated with relatively larger immune organs like the spleen and bursa of Fabricius, which were considered indirect markers of disease risk in these species.

Increased exploratory tendencies in house finches are also correlated with greater innate immune defense investment, potentially due to the greater likelihood of encountering parasites

for exploratory individuals (Zylberberg et al., 2014). Indirect evidence for this relationship also comes from bullfinches in Barbados, among which boldness, but not exploratory tendency, as measured by behavioral responses to novelty, was positively associated with proximity to human dwellings, as was immunocompetence, a proxy for parasite risk here measured by phytohemagglutinin antigens (Audet et al., 2015); this increased immunocompetence may have resulted from higher quality and abundance of foods near human dwellings. More recent research in the same study system has since found that birds in closer proximity to human dwellings do indeed also show more exploratory tendencies (Ducatez et al., 2016).

However, it can be difficult to determine the causality of correlations between exploratory tendencies and parasitism. As one example of this, a study of personality in uninfected tadpoles found that more exploratory individuals were actually less likely to become infected with water-borne trematode parasites (Koprivnikar et al., 2012). The authors of this paper argue that this phenomenon may be due to the fact that more exploratory individuals must have better behavioural avoidance mechanisms, given their greater chances of encountering parasites. Additionally, infection can reduce activity, and thus assayed exploratory tendencies (Barber and Dingemanse, 2010), and parasite infections early in life can have lasting effects on personality (Galic et al., 2009; Rico et al., 2010; Butler et al., 2011). Thus, the relationship between exploratory behavior and parasite risk is complicated. No studies have considered the effects of exploratory tendencies broadly on all ectoparasites and endoparasites, at least not in a single study. The conflicting results to date may indicate context-dependent outcomes of exploratory tendency on parasite infection. Studies designed specifically to make sense of such context-dependent outcomes, as are presented later in this dissertation, have the potential to greatly further this field of research.

On the other hand, greater social contact, defined here as the tendency of an animal to associate with, tolerate, and socially interact with other individuals, as well as the structuring and intensity of those interactions, can also be a risky behavior from the perspective of parasite exposure. The benefits of increased sociality and social contact include greater access to social information about the presence of food, protection from predators, and greater selection of mates (Rieucau and Giraldeau, 2011; Thornton and Clutton-Brock, 2011). Social contact, and specifically the opportunities for social learning that often accompany it, is another important requirement for the development of culture in primates and humans, along with innovation (Heyes and Galef, 1996; Whiten, 2000; Byrne et al., 2004). But there are costs associated with social contact, as well; these include increased competition for food and mates, as well as increases in intragroup and intergroup aggression (Wrangham et al., 1996).

Again, as with exploratory behaviors, increased levels of social contact can have significant impacts on parasite exposure (Barber and Dingemanse, 2010). Social contact can be measured in a variety of ways, and the most basic of these is to consider a social group's size, where larger groups provide more opportunities to come into contact with social partners. One of the earliest studies into the effects of group size on parasitism posited that social groups act like islands for parasites, in the sense of island biogeography theory; the larger a group, the greater the diversity of parasites that the group can support (Freeland, 1979). An early meta-analysis of group size and parasitism found that larger social groups tended to have higher prevalences and intensities of close-contact transmitted parasites (Côte and Poulin, 1995). Two separate updates to these meta-analyses have also found that intensity and prevalence of parasites are positively associated with social group size, but richness is not; additionally, effect sizes of these associations have been relatively weak (Rifkin et al., 2012; Patterson and Ruckstuhl, 2013).

When investigating these patterns in single host species, similar patterns arise as is to be expected; for instance, larger groups of feral island horses were found to have greater gastrointestinal parasite infection intensities (Rubenstein and Hohmann, 1989), but the richness of streblid fly parasites infecting neotropical bats had no relationship with social group sizes in these hosts (Bordes et al., 2008).

Another useful, although rough measure of social contact is population density. Here, we assume that animals in closer proximity to one another have closer contact, and thus greater chances of transmitting parasites. In phylogenetic comparative models, greater host density has been associated with higher prevalence and intensity of parasite infections (Arneberg et al., 1998), and richness of non-close-contact transmitted parasites (Lindenfors et al., 2007). Among primate species, population density was positively associated with parasite species richness for helminths, protozoa, and viruses separately in phylogenetic comparative analyses (Nunn et al., 2003), and similar results were found for richness in independent contrasts tests among terrestrial mammals (Morand and Poulin, 1998). Meta-analyses specifically investigating the relationship between host population density and parasite species richness in animals, plants, and fungi also found a positive association (Kamiya et al., 2013). Even among herd-living species without intense social interactions, like African bovids, the tendency to gather into dense groups has been positively associated with prevalence and intensity of strongyle parasites (Ezenwa, 2004; Vitone et al., 2004).

However, neither social group size nor population density truly capture the complexity of social interactions like affiliation, cooperation, or aggression that are required for most forms of social learning to occur (Reader and Biro, 2010). Instead, the most appropriate method for understanding effects of social contact on the spread of socially learned behaviors across a

population is through social network analysis (Franz and Nunn, 2009; Jacobs and Petit, 2011). Previous studies have shown the importance of network approaches for understanding behavior spread through populations in primates (Franz and Nunn, 2009), and specifically in humans (Centola, 2010). Studies focusing on the social and temporal structuring of human populations have also found that group sizes that take such structuring into account can explain the number and complexity of cultural behaviors sustained in these groups (Henrich, 2004; Powell et al., 2009). The structure of social networks are also important for understanding the spread of disease through populations (Kasper and Voelkl, 2009; Craft et al., 2010; Sueur et al., 2011; White et al., 2015). Field studies of the effect of social network structuring on disease spread have found that more clustered networks tend to have reduced spread of close-contact transmitted parasites (Loehle, 1995; VanderWaal et al., 2013; Balasubramaniam et al., 2016). Theoretical and comparative studies have also found support for sub-structuring, clustering, and modularity slowing the spread of disease (Griffin and Nunn, 2012; Nunn, 2012; Nunn et al., 2015). These types of analyses are most suited to understanding the interactions between social interactions and close-contact parasite transmission, but there remains no standardized or universally agreed-upon measure to account for the effects of social network structure and group size on information and disease flow through groups.

The three chapters that follow this introduction investigate the links between parasite risk and the behavioral patterns that accompany the emergence of animal and human culture, exploratory tendency and social contact. Chapter 2 of this dissertation investigates associations between social learning, exploration, and parasite risk across primate species, where the behavioral measures are based on literature surveys and parasite risk is measured as parasite species richness. Using phylogenetic comparative methods, I found that social learning rates in

different primate species positively associated with close-contact-transmitted parasite richness, and exploration rates covaried with non-close-contact-transmitted parasite richness. Building on these findings, I further investigate the link between social contact and parasite risk at the population level in Chapter 3. Specifically, I attempt to resolve the issue of group size not being a particularly strong predictor of parasite risk, despite the logical predictions for an effect. My accounting for structure in group sizes preformed no better than raw group size at predicting parasite richness among primate species, but the approach holds promise for future studies. Based on the exploratory behavior findings in Chapter 2, Chapter 4 investigates how exploratory behavior in rodents influences parasite richness and infection intensity, as well as their risk of transmitting these parasites to humans through cohabitation. I found that more exploratory rodents had greater intensities of endo- and ectoparasite infections but no effect on the richness of parasites. Additionally, these more exploratory, more heavily infected rodents were more likely to be found in close proximity to humans, perhaps also increasing human exposure to such parasites. Given the importance of exploratory tendecies and social contact to the development of culture, particularly in primates and humans, understanding their relationships with parasitism should be of high importance to the study of human evolution, which is why they will be the overarching focus of the following chapters.

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Chapter 2

Behavioral flexibility and learning as drivers of disease risk among primates

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Abstract

Culturally transmitted traits are observed in a wide array of animal species, yet we understand little about the costs of the behavioral patterns that underlie culture, such as innovation and social learning. We propose that infectious diseases are a significant cost associated with cultural transmission. We investigated two hypotheses that may explain such a connection: that social learning and exploratory behaviors (specifically, innovation and extractive foraging) either compensate for existing infection or increase exposure to infectious agents. We used Bayesian comparative methods, controlling for sampling effort, body mass, group size, geographic range size, terrestriality, latitude, and phylogenetic uncertainty. Across 127 primate species, we found a positive association between pathogen richness and rates of innovation, extractive foraging, and social learning. This relationship was driven by two independent phenomena: socially contagious diseases were positively associated with rates of social learning, and environmentally transmitted diseases were positively associated with rates of exploration. Because higher pathogen burdens can contribute to morbidity and mortality, we propose that parasitism is a significant cost associated with the behavioral patterns that underpin culture, and that increased pathogen exposure is likely to have played an important role in the evolution of culture in both non-human primates and humans.

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2.1 Introduction

Cultural transmission has allowed humans and nonhuman animals to flexibly adapt to and shape their environments. The capacity to learn new behaviors – both individually through innovation and from others through social learning – allows flexibility in the face of changing environments (Reader and Laland, 2003; Sol et al., 2005; Whiten et al., 2012; Sih, 2013). Individual learning of a novel behavior, or innovation, occurs through exploration and experimentation (Reader and Laland, 2001; 2003), while social learning occurs when one individual learns from the behavior of another individual (Tomasello and Call, 1997; Dunbar and Shultz, 2007; Whiten et al., 2012). Much of the research on innovation and social learning has focused on the ecological and social benefits of behaviors acquired by these processes. For example, learned foraging behaviors can enable individuals to more effectively acquire energy, either by accessing new resources or by more efficiently exploiting existing food sources (Reader and Laland, 2003). Similarly, extractive foraging (feeding on embedded or encased foods such as nutmeat, shellfish, bone marrow, and buried tubers) can provide access to novel or nutritionally rich food resources. Extractive foraging has been linked to exploratory behavior and innovation, and also has been proposed to be a condition favouring the development of complex culture (van Schaik et al., 1999; Day et al., 2003; Reader et al., 2011).

Although many studies have focused on the benefits of innovation, extractive foraging, and social learning, relatively few studies have considered the costs of these behaviors. Costs may include the immediate costs of performing a behavior and constitutive costs associated with developing the ability to perform a behavior (Mery and Kawecki, 2003). In terms of immediate costs, innovation and extractive foraging may carry risks such as poisoning from eating toxic food sources, predation, or injury from interacting with new objects or individuals (Reader and

Laland, 2001). Additionally, social learning may favour increased proximity among individuals, which could increase competition for resources. In terms of constitutive costs, brain size may be a cost of innovation and social learning, with rates of both innovation and social learning positively correlated with brain volume across primates (Reader and Laland, 2002), (Chittka and Niven, 2009). Production and maintenance of energetically-expensive brain tissue can be accommodated by decreasing energetic investment in functions such as digestion, locomotion, or reproduction (Navarrete et al., 2011). Larger brains may also generate life history costs involving greater allocation of resources to large-brained offspring (Barrickman et al., 2008; Bordes et al., 2011).

We tested the hypothesis that innovation, extractive foraging, and social learning are associated with increased disease risk (Garamszegi et al., 2007; Boyer et al., 2010; Vas et al., 2011). Higher rates of innovation and more varied extractive foraging, which are indicators of greater environmental exploration, may result in greater exposure to infectious agents in the environment. For example, primates foraging on insects can be exposed to acanthocephalans, and primates digging in soil may be exposed to helminths (Nunn and Altizer, 2006; Ghai et al., 2014). If social learning is associated with increased social contact either directly or via increased joint use of resources (Reader and Biro, 2010), more frequent social learners may be more exposed to socially-contagious parasites. Because we were interested in how increases in species-level behavioral variation affected parasite variation at the species level, we used measures of richness, the number of observed unique behaviors or parasites for a species, as our main indicator variables. This decision was further motivated by our goal of capturing a wide diversity of parasites and behaviors, a goal which would be hindered if we were to test the hypotheses with single-parasite measures of prevalence (the proportion of individuals infected

with a specified parasite in a single population) or infection intensity (the number of reproductive parasites present within a single infected individual at a point in time).

Given our hypotheses, we propose that social learning and exploration may have parasite-related fitness costs that, if great enough, would offset their benefits. These parasite-related costs could thus partially account for the observed interspecific variation in social learning and exploration across primates (Reader et al., 2011). Beyond the obvious negative impacts of parasites on fitness via morbidity (Bush et al., 2001) and mortality (Milton, 1996; Formenty et al., 1999; Walsh et al., 2003), infectious diseases also have negative impacts on cognitive development and function (Kavaliers and Colwell, 1995), hinder growth (Checkley et al., 2008), and require individuals to allocate more time and energy to resting and immune function (Hart, 1990). These fitness consequences of parasitism are often amplified when a greater diversity of parasites are found in a host or species; for example coinfection leads to higher host mortality in a wide variety of host and parasite taxa (Jolles et al., 2008; Ezenwa et al., 2010; Bordes and Morand, 2011).

Previous research has linked higher rates of exploratory or innovative behaviors with higher levels of parasitism in rodents (Boyer et al., 2010) and birds (Garamszegi et al., 2007; Soler et al., 2011; Vas et al., 2011). Additionally, indicators of social contact patterns such as group size, population density, and social network properties are positively correlated with parasitism (Côte and Poulin, 1995; Altizer et al., 2003; Griffin and Nunn, 2012; Rifkin et al., 2012), although none of these patterns have been linked directly to social learning. Unfortunately, the causes behind these correlations are poorly understood, with two major competing hypotheses. The first, the "exposure hypothesis," posits that increased exploration or social learning leads to increased exposure to parasites. The second, the "compensation

hypothesis," posits that increased exploration and social learning are compensatory responses to higher parasite levels. These two hypotheses have been proposed in the discussions of previous work finding links between exploration, social learning, and parasite risk (Reader and Laland, 2001; Garamszegi et al., 2007; Boyer et al., 2010; Vas et al., 2011), but never explicitly tested. Additionally, no study on the correlation of parasitism and behaviors underpinning culture has yet included primates, which is remarkable given the large number of studies on social learning and innovation in primates and their importance for understanding the evolution of human culture (Tomasello and Call, 1997; Whiten et al., 2012).

Based on the general hypothesis that exploratory behavior and social learning have parasite-related costs, we contrast these two specific, but not necessarily exclusive hypotheses [Table 2.1]. Under the "compensation hypothesis," if parasite-related costs were driving a general need for further exploration and social learning in primates, we predict positive correlations between these behaviors and all measures of parasite richness, regardless of transmission mode. However, under the "exposure hypothesis," we predict that richness of parasites transmitted through social contact will covary positively with rates of social learning, but not with our measures of environmental exploration (innovation and extractive foraging), while richness of parasites transmitted through contact with the environment are predicted to covary most strongly with rates of innovation and extractive foraging, but not with social learning.

Table 2.1. Predicted associations between parasite richness and behavioral richness under the competing hypotheses: (a) the "exposure hypothesis", and (b) the "compensation hypothesis."

(a) – "Exposure Hypothesis"	**Socially Transmitted Parasites**	**Environmentally Transmitted Parasites**
Social Learning	Positive Association	No Association
Exploration	No Association	Positive Association

(b) – "Compensation Hypothesis"	**Socially Transmitted Parasites**	**Environmentally Transmitted Parasites**
Social Learning	Positive Association	Positive Association
Exploration	Positive Association	Positive Association

2.2 Methods

2.2.1 Overview

To test our competing hypotheses, we examined three behavioral measures of social learning and environmental exploration, namely the number of reports of social learning, innovation and extractive foraging per species, with innovation and extractive foraging together indexing "exploratory behavior," as they both relate to the exploration and exploitation of an animal's environment. We use "parasite" to refer to any infectious disease-causing agent, ranging from macro-parasites like helminths and arthropods to micro-parasites, or pathogens, such as viruses, bacteria, protozoa, and fungi. In addition to a general analysis of all parasites, we investigated social learning and exploratory behavior in relation to parasites that are either socially transmitted or environmentally transmitted, and thus relevant to our two specific hypotheses. Socially transmitted parasites rely on direct host-to-host contact for their transmission, such as viruses that cause respiratory infections and are spread through sneezing, coughing, and physical contact. Environmentally transmitted parasites spread through environmental substrates such as soil and water, in which parasite infectious stages are found e.g. *Giardia spp.*; (Nunn and Altizer, 2006). Thus, we ran five analyses: one global analysis testing for an association between all behaviors and all parasites, and four sub-analyses addressing each of the predictions of our specific hypotheses.

2.2.2 Behavioral and parasite datasets

Behavioral data were obtained from Reader et al. (2011), a survey of over 4000 articles published between 1925-2000, principally coming from four primate behavior journals (*Primates*, *American Journal of Primatology*, *Folia Primatologica*, and *International Journal of*

Primatology), but also from searches of other relevant literature and studies cited by publications that were located in the first round of search. Keywords were used to classify behavior patterns. Innovation was defined as the discovery of novel solutions to environmental or social problems (example keywords: "innovation", "invention", "opportunistic", "departure from normal behavioral repertoire", "not previously observed", "unusual", "no published accounts", "first observation", "unique", "exceptional", "previously unreported", "not documented before", "never seen before", "novel", "new"). Extractive foraging was defined as feeding on foods that must first be extracted from matrices in which they are embedded or encased, including nutmeat, shellfish, snails, eggs, brains, bone marrow, roots, tubers, and ant and termite mounds. Social learning was defined as learning skills and acquiring information from others (example keywords: "social learning", "social transmission", "cultural transmission", "traditional", "teaching", "imitation", "protoculture", "[goal] emulation", "observational learning", "learning from each other", "culturally acquired", "local enhancement", "stimulus enhancement", "socially mediated learning"). Examples came from varied behavioral contexts, including foraging behavior, locomotion, anti-predator behavior and social displays. Further details of how data were collated, examples of behavioral reports, and discussion of the validation and utility of the dataset are given in (Reader and Laland, 2001; 2002; Reader and MacDonald, 2003; Lefebvre et al., 2004; Reader et al., 2011).

For reports to be included in the behavioral database as distinct examples, they needed to be reported as separate behavior patterns by the original authors. The database was then screened for possible repeated examples, with reports in the same species, context, and involving the same food and substrate only counted once. For example, two reports in one species of fracturing dead branches to consume larvae would be counted only once, whereas two reports, one of fracturing

branches to consume larvae and another of fracturing branches to access fungi would be counted as two reports. Similarly, digging soil to access larvae and fracturing branches to access larvae would count as two reports. Thus, a specialist extractive forager that uses a single extractive foraging technique to access a foodstuff would only be counted for one report in our database. However, instances of social learning and innovation of the same behavior were counted as distinct events, since the social learner and individual learned from are different individuals. Additionally, examples of innovative tool use and of extractive foraging with tools were also excluded because we lacked a clear hypothesis linking tool use to parasitism.

Our behavioral data provide a measure of the variety of reports within each behavioral category for each species, rather than data on the frequency of use, time spent, or reliance on social learning, innovation, and extractive foraging. Inter-observer reliabilities are high, and the measures have been validated against other compilations as well as against experimental cognitive tests (Reader and Laland, 2002; Reader et al., 2011; Maclean et al., 2014). However, the majority of these reports were observational, and thus reports of social learning (which is difficult to characterize without controlled experiments) should be interpreted with caution, while innovation and extractive foraging are more easily characterized by observational studies (Reader and Laland, 2002; Reader and Biro, 2010; Reader et al., 2011). In this respect experimental investigation of species differences would be valuable, but such data are challenging to objectively gather for the large number of species investigated here (Reader et al., 2011).

Behavioral data were summarized as the total number of distinct behaviors (a measure of behavioral richness) that could be categorized as either social learning, innovation, or extractive foraging for each primate species in the dataset. To facilitate simpler and more intuitive

interpretations of the hypotheses given in the introduction, we combined innovation and extractive foraging counts for each species. Because each of these measures separately quantified an aspect of exploration, we investigated whether it would be justified to combine these two variables into a single variable describing exploratory behavior as a whole. Results of separate MCMC Bayesian PGLS models for innovation and extractive foraging regressed against parasite species richness, controlling for common covariates, showed converging results [Table 2.2], and were thus combined into a single variable for analyses presented in the main text.

We extracted parasite species richness from the Global Mammal Parasite Database (GMPD) (Nunn and Altizer, 2005). The GMPD was collated by searching published literature for reports of parasites from wild primate populations, using online reference databases such as Biological Abstracts, AGRICOLA, Medline, PrimateLit, and Web of Science. Edited volumes, reviews, and studies that were cited by publications that were located in the first round of searches were also examined. Latin binomials of primate species were used as search keywords, as well as primate genus name following Corbet and Hill (1991) and common taxonomic variants (Rowe, 1996; Groves, 2001). Parasites were recorded in the database following positive identification of a genus or species of parasite within a mammal host from one of these published articles; only peer-reviewed identifications were noted in the database. The database is continuously updated with new records; the dataset used for this study was extracted from the database in September 2010, and thus includes records up to this date.

2.2.3 Control variables

We controlled for four variables that may influence parasite richness (Poulin, 1995; Morand 2000; Nunn et al., 2003): (i) average body size of a species, because larger-bodied individuals consume more resources and provide more niches for parasites (Morand 2000);

Table 2.2. Results of a minimal Bayesian PGLS models including all predictor variables and flat priors (model further explained in section 2.2.5), run independently for both innovation and extractive foraging. Parasite species richness was the response variable and all others (behavior richness, body mass, group size, and geographic range) were predictors; terrestriality and absolute latitude were excluded from these initial tests. Reported outputs for each predictor are the mean slopes (β) and proportion of models with positive slopes (support) sampled from 3,000,000 iterations. Model mean R^2 and mean λ were estimated as the means of all iterations and 95% highest posterior density credibility intervals (95% HPD CI) values for λ were calculated from all results. Effect sizes for multiple regression models are presented as Cohen's f^2. Innovation and extractive foraging models converge to nearly identical results for each variable studied, justifying the combination of the two into a single variable.

Parasite Transmission Mode	Behavioral Measure	Behavior Richness		Body Mass		Group Size		Geographic Range		Lambda		Mean R^2	Cohen's f^2
		Mean β	Support	Mean β	Support	Mean β	Support	Mean β	Support	Mean λ	95% HPD CI		
All	*Innovation*	0.21	98.9%	0.22	99.9%	0.05	74.3%	0.05	92.2%	0.20	<0.01 - 0.47	0.17	0.20
All	*Extractive Foraging*	0.20	97.6%	0.22	99.8%	0.04	68.2%	0.06	92.9%	0.22	<0.01 - 0.51	0.16	0.19

(ii) average group size for a species, because larger groups are more likely to maintain a parasite than are smaller ones (Côte and Poulin, 1995; Altizer et al., 2003; Rifkin et al., 2012); (iii) geographic range of the species, because species that cover more area are more likely to encompass the ranges of multiple parasites, likely have larger populations to sustain more parasites, and are more likely to encounter greater variation in habitat types which could support different parasites (Gregory, 1990; Nunn and Altizer, 2006), and may show greater behavioral diversity (Kamilar and Marshack, 2012); and (iv) the absolute value of the latitudinal mid-point of each species' range (henceforth "absolute latitude"), because previous studies have shown that parasite richness decreases as host species move away from the equator (Nunn et al., 2005). An additional analysis that also included a binary variable for substrate use (arboreal versus terrestrial, because terrestrial species would be expected to encounter a greater variety of environmentally-transmitted parasites) revealed an identical, albeit weaker, pattern of results, perhaps due to the decrease in power or the lack of resolution that a binary variable can provide. Owing to these various issues with this variable, we left substrate use out of our main multivariate analyses, but we present and discuss additional results involving associations between learning categories, substrate use and parasite transmission in section 2.3.3. Data on mean adult body mass, mean group size, total geographic range, and absolute latitude were collected from the PanTHERIA database (Jones et al., 2009), and, when data were unavailable, from the All the World's Primates database (Rowe and Myers, 2011).

We also investigated the effects of terrestriality on parasite richness measures. More terrestrial species may encounter a greater variety of environmentally transmitted parasites than do more arboreal primates (Nunn et al., 2003; Nunn and Altizer, 2006). However, the variable was not included as a predictor in the combined models presented in the main portion of the

results for several reasons. First, previous comparative studies have failed to find a consistent association between general measures of parasitism and terrestriality (Nunn et al., 2003). In addition, including this variable reduced the power of the models below an acceptable level. Finally, the measure of terrestriality available for our large sample of primates (2002) is a binary categorisation of what is, in reality, a continuum of tree and ground use. But because the links between terrestriality and socially-transmitted or environmentally transmitted infectious agents have not previously been investigated, we ran our multivariate analyses with a binary categorization of terrestriality, obtained from Nunn and van Schaik (2002).

More intensive sampling could lead to higher counts of both parasites and behaviors (Cooper and Nunn, 2013). We controlled for differences in research effort by regressing parasite and behavior counts on citation counts, and using the residuals from these models in our analyses. Although the relationship between sampling effort and richness is, in theory, an asymptotic one of diminishing returns at higher sampling efforts, we saw no such levelling-off of this relationship for any of our study species, and thus chose to model this relationship with a linear fit. When testing for a correlation between two sets of residuals obtained with identical x-variables, spurious positive results can arise due to measurement error in x (Nunn, 2002). To control for this, the "Economos problem" of correlated residuals, we used separate, independent sources to estimate sampling effort for the parasite and behavioral data (Deaner et al., 2003). We collated our parasite richness data with data on the number of references for each host species using the Primate Information Network's "PrimateLit" bibliographic database (http://primatelit.library.wisc.edu/), accessed in May 2010. Similarly, we collated our behavioral richness data with data on the number of references for each species using the Zoological Record citation index for 1993-2001. Both bibliographic databases cover a range of subject areas and

both field and captive studies, and they were chosen to ascertain general research effort in the study of a given species. Log_{10}-transformed parasite richness and behavioral richness data were then regressed against the independently obtained measures of sampling effort while controlling for phylogeny, with residuals from these regressions used in the analyses. Mean R^2 for behavioral richness ~ citation count models was 0.387, and mean R^2 for parasite species richness ~ citation count was 0.184.

2.2.4 Phylogeny and phylogenetic uncertainty

After compiling data from both databases, 127 primate species were found to co-occur between datasets and were thus included in this study. Following the classification system of Corbett and Hill (C&H) (1991), the 127 species consisted of 26 strepsirrhines, 1 tarsier, 38 New World monkeys, 53 Old World monkeys, 5 gibbons, and 4 great apes (excluding humans) [Table 2.3]. Seventy-four percent of all primate species were sampled from the C&H taxonomy. Most species included in the parasite and behavior databases match the Corbet & Hill (C&H) taxonomy (1991), with the following exceptions: five lemur species in the genus *Eulemur* were indexed under the alternate C&H genus of *Petterus*; the howler monkey, *Alouatta pigra* was indexed under the alternate C&H species designation of *Alouatta villosa*; and two species, *Alouatta guariba* and *Callicebus personatus*, were not identified in the C&H system. Species are listed by alphabetical order in Table 2.3.

In all analyses reported – including those controlling for sampling effort – we incorporated uncertainty in primate phylogeny and the underlying evolutionary model by using Bayesian phylogenetic comparative methods (Markov chain Monte Carlo Phylogenetic Generalized Least Squares models, or MCMC PGLS), as implemented in

Table 2.3. List of primate species included in the parasite and behavior database, according to the Corbet & Hill taxonomy (1991). Species marked with asterisks indicate minor changes from the original taxonomies used in data collection (as noted in Section 2.2.4).

Allenopithecus nigroviridis	*Cercopithecus ascanius*	*Hylobates concolor*	*Pan paniscus*
Alouatta belzebul	*Cercopithecus campbelli*	*Hylobates hoolock*	*Pan troglodytes*
Alouatta caraya	*Cercopithecus cephus*	*Hylobates lar*	*Papio anubis*
Alouatta guariba *	*Cercopithecus diana*	*Hylobates moloch*	*Papio cynocephalus*
Alouatta palliata	*Cercopithecus lhoesti*	*Hylobates syndactylus*	*Papio hamadryas*
Alouatta villosa *	*Cercopithecus mitis*	*Indri indri*	*Papio papio*
Alouatta seniculus	*Cercopithecus mona*	*Lagothrix lagotricha*	*Papio ursinus*
Aotus azarae	*Cercopithecus neglectus*	*Lemur catta*	*Perodicticus potto*
Aotus trivirgatus	*Cercopithecus nictitans*	*Leontopithecus chrysomelas*	*Pithecia irrorata*
Arctocebus calabarensis	*Cercopithecus petaurista*	*Leontopithecus chrysopygus*	*Pithecia pithecia*
Ateles belzebuth	*Cercopithecus pogonias*	*Leontopithecus rosalia*	*Pongo pygmaeus*
Ateles fusciceps	*Cercopithecus preussi*	*Lepilemur mustelinus*	*Presbytis cristata*
Ateles geoffroyi	*Cheirogaleus major*	*Macaca arctoides*	*Presbytis entellus*
Ateles paniscus	*Cheirogaleus medius*	*Macaca assamensis*	*Presbytis melalophos*
Avahi laniger	*Colobus angolensis*	*Macaca cyclopis*	*Presbytis obscura*
Brachyteles arachnoides	*Colobus badius*	*Macaca fascicularis*	*Presbytis phayrei*
Cacajao calvus	*Colobus guereza*	*Macaca fuscata*	*Presbytis vetulus*
Callicebus moloch	*Colobus polykomos*	*Macaca maurus*	*Propithecus diadema*
Callicebus personatus *	*Daubentonia madagascariensis*	*Macaca mulatta*	*Propithecus tattersalli*
Callimico goeldii	*Erythrocebus patas*	*Macaca nemestrina*	*Propithecus verreauxi*
Callithrix argentata	*Petterus coronatus* *	*Macaca nigra*	*Saguinus fuscicollis*
Callithrix jacchus	*Petterus fulvus* *	*Macaca ochreata*	*Saguinus leucopus*
Cebuella pygmaea	*Petterus macaco* *	*Macaca radiata*	*Saguinus midas*
Cebus albifrons	*Petterus mongoz* *	*Macaca sinica*	*Saguinus mystax*
Cebus apella	*Petterus rubriventer* *	*Macaca sylvanus*	*Saguinus oedipus*
Cebus capucinus	*Euoticus elegantulus*	*Macaca tonkeana*	*Saimiri boliviensis*
Cebus olivaceus	*Galago moholi*	*Mandrillus leucophaeus*	*Saimiri oerstedii*
Cercocebus albigena	*Galago senegalensis*	*Mandrillus sphinx*	*Saimiri sciureus*
Cercocebus aterrimus	*Galagoides demidoff*	*Microcebus murinus*	*Tarsius bancanus*
Cercocebus galeritus	*Gorilla gorilla*	*Miopithecus talapoin*	*Theropithecus gelada*
Cercocebus torquatus	*Hapalemur griseus*	*Nycticebus coucang*	*Varecia variegata*
Cercopithecus aethiops	*Hapalemur simus*	*Otolemur crassicaudatus*	

BayesTraits (Pagel, 1999) and assuming flat priors. Because we have imperfect knowledge of the exact evolutionary history of living primates, our analyses controlled for phylogenetic uncertainty by using a set of 100 dated, bifurcating phylogenies, downloaded from *10kTrees* Version 3 for the 125 species identified in the C&H taxonomy, plus two additional species not identified in the C&H taxonomy (Arnold et al., 2010). Regression models were run for 3,300,000 iterations, with a 300,000 iteration burn-in, and sampled every 100 iterations. Rate deviation parameters were set to maintain acceptance rates between 25% and 35%, and we estimated λ, which scales the internal branch lengths of a phylogeny and is generally used to quantify phylogenetic signal (Pagel, 1999; Freckleton et al., 2002). A value of λ equal to one indicates that evolution of a given trait has occurred according to a Brownian motion model of evolution on the phylogeny and thus shows phylogenetic signal; a value of λ equal to zero indicates that trait variation is independent of phylogeny; and values of λ between zero and one indicate an intermediate phylogenetic signal. We included an estimate of λ to control for the effect of phylogeny in the statistical models; this is preferable to using phylogenetic independent contrasts (PIC), because PIC assumes a λ of 1, rather than allowing λ to take intermediate values. Three runs of each model were tested to ensure convergence to common values and plateaued likelihood, and consistent findings were confirmed before reporting results. All models reported in this study resulted in convergence to common values for all variables tested. Regression coefficients used for controlling sampling effort were obtained as the mean of the posterior distribution.

2.2.5 Bayesian Statistical Models and Evaluation Criteria

First, we investigated the effect of all different behaviors on all parasites, which we will refer to as the "total" model, using the following linear model: $Residual[PSR] \sim intercept + \beta_{Residual[BR]}*Residual[BR] + \beta_{BM}*Body\ Mass + \beta_{GS}*Group\ Size + \beta_{GR}*Geographic\ Range + \beta_{AL}*Absolute\ Latitude + error$ (where PSR is parasite species richness, BR is behavioral richness, BM is body mass, GS is group size, GR is geographical range and AL is absolute latitude). The "total" model included many parasites that were documented as being transmitted by both social and environmental contact.

Second, we extracted richness of exclusively socially transmitted and exclusively environmentally transmitted parasites from the GMPD. Fifty-four host species (11 strepsirhines, 13 Old World monkeys, 26 New World monkeys, and 4 great apes) were found to have sufficient data for testing our hypotheses (i.e. for each species, at least one parasite species was present for each of the two categories of mutually exclusive transmission modes). We controlled for sampling effort, and re-estimated parameters for the statistical model as used in the "total" model. We also investigated the association between social learning and exploratory behaviors using our methods.

Levels of support for an association between two variables were based on the proportion of regression coefficients with slopes in the predicted direction, assigned as follows: >95% of slopes in the predicted direction were interpreted as "strong support" and 90% to 95% as "likely support." Variance inflation factors (VIF) of all models were tested to detect multicollinearity in the statistical models, with critical values set at ≥10 (Petraitis et al., 1996). All VIFs for predictors across all models were well below the critical value, with a maximum value of 1.51.

2.3 Results

2.3.1 Total Richness Results

Across primate species, the number of reports of social learning, innovation, and extractive foraging ("total behavior richness") covaried positively with total parasite richness [Figure 2.1], with both variables controlled for sampling effort. We found "strong support" (see section 2.2.5 for definitions of support) for this association in our MCMC Bayesian PGLS model, with over 99% of sampled iterations exhibiting positive slopes. Our models also included additional controls for body mass, geographic range, absolute latitude, and group size, which are commonly investigated as predictors of parasite richness (Morand 2000; Nunn and Altizer, 2006). We found "likely support" for a positive association between total parasite richness and body mass, "strong support" for a positive association between total parasite richness and geographic range, and "strong support" for a negative association between total parasite richness and absolute latitude. The model revealed intermediate phylogenetic signal (mean λ=0.29; see section 2.2.4 for explanation of lambda) and despite strong support for several key variables, fit the data modestly, with mean R^2=0.15 [Table 2.4].

2.3.2 Transmission Mode Results

To focus specifically on whether particular types of exploration and learning influence exposure to particular parasites, we isolated exclusively socially transmitted and exclusively environmentally transmitted parasites in tests for associations with socially learned behaviors and exploratory behaviors. This isolation was important, because many parasites exhibited multiple transmission modes, which could lead to spurious correlations in the tests of our specific predictions (Reader and Laland, 2002; Pedersen et al., 2005). If we had included

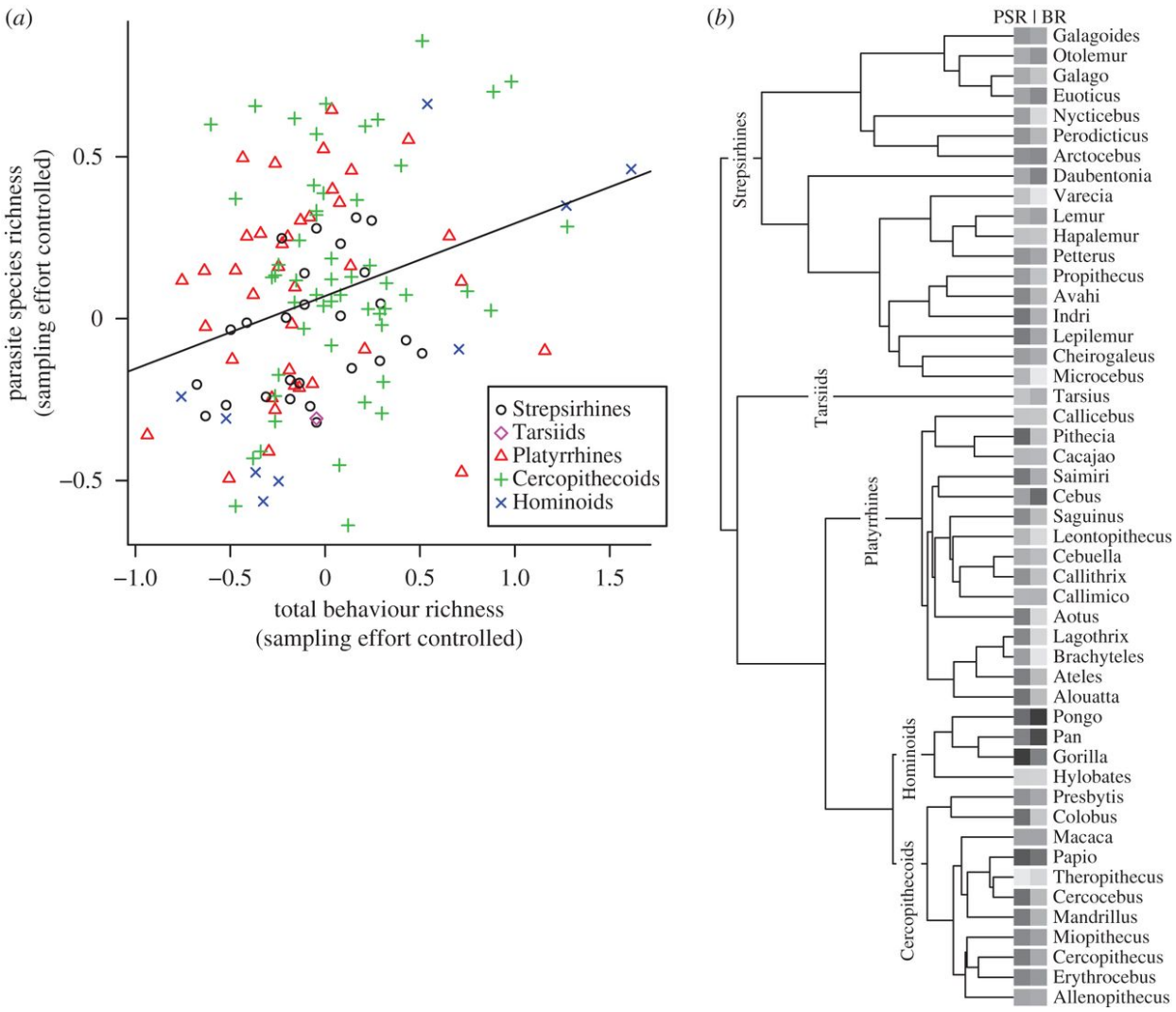

Figure 2.1. The "total" Bayesian PGLS model reveals an association between behavior and parasite richness. a.) Parasite species richness covaries positively with behavior richness per primate species. Species are coloured and grouped by monophyletic taxa, with a line-of-best-fit indicating the regression after controlling for body mass, group size, geographic range, absolute latitude, phylogeny, and sampling effort. b.) 10kTrees (Arnold et al., 2010) consensus phylogeny of genera included in the analysis, with relative measures of parasite species richness (PSR, first column) and behavior richness (BR, second column) indicated at the tips, along with genus name. Shades of blocks indicate magnitude of a genus' PSR or BR, with darker shades representing greater relative values.

parasites that were transmitted by both social and environmental contact, then we would be much more likely observe results in support of the "compensation hypothesis," because such inclusions would lead to a convergence in our results for the four specific models. Tests of socially transmitted parasites supported the "exposure hypothesis." Specifically, the number of reports of social learning was positively associated with measures of exclusively socially transmitted parasite richness, again in models that controlled for sampling effort and the other previously mentioned controls. This association between social learning and socially transmitted parasite richness received "strong support" with 98% of iterations sampled exhibiting positive slopes [Figure 2.2a]. However, we found no support for an association between the richness of exploratory behavior and exclusively socially transmitted parasite richness, with only 36.5% of iterations sampled exhibiting positive slopes for counts of exploratory behaviors [Figure 2.2b]. In both social transmission parasite models, body mass and geographic range were also "strong" predictors of positive associations with socially transmitted parasite richness. Group size and absolute latitude were not clearly associated with socially transmitted parasite richness [Table 2.4].

Analyses of environmentally transmitted parasites provided additional support for the "exposure hypothesis." The richness of exploratory behavior was positively associated with measures of exclusively environmentally transmitted parasite richness, showing "likely support" with nearly 93% of MCMC samples exhibiting positive slopes [Figure 2.2d]. Conversely, we found no evidence for an association between social learning and measures of exclusively environmentally transmitted parasite richness, with just under 66% of iterations sampled exhibiting positive slopes for relative counts of social learning [Figure 2.2c]. Body mass was a supported predictor of environmentally transmitted parasite richness (94-97% support), while

Table 2.4. Results of the five Bayesian PGLS models tested in this study. Parasite species richness was the response variable and all others (behavior richness, body mass, group size, geographic range, and absolute latitude) were predictors.. Reported outputs for each predictor are the mean slopes (β) and proportion of models with predicted slopes ('support') sampled from 3,000,000 iterations. Model mean R^2 and mean λ were estimated as the means of all iterations and 95% highest posterior density credibility intervals (95% HPD CI) values for λ were calculated from all results.

Parasite Transmission Mode	Behavioral Measure	Behavior Richness		Body Mass		Group Size		Geographic Range		Absolute Latitude		Lambda		Mean R^2
		Mean β	Support	Mean β	Support	Mean β	Support	Mean β	Support	Mean β	Support	Mean λ	95% HPD CI	
Social	*Exploration*	-0.02	36.5%	0.18	99.8%	-0.08	10.4%	0.09	98.8%	-0.01	64.2%	0.26	<0.01-0.54	0.20
Social	*Social Learning*	0.16	98.0%	0.14	99.3%	-0.06	17.2%	0.08	98.3%	-0.01	73.7%	0.20	<0.01-0.47	0.27
Environmental	*Exploration*	0.13	92.9%	0.12	94.3%	0.05	71.2%	-0.01	42.3 %	-0.03	84.9%	0.19	<0.01-0.50	0.11
Environmental	*Social Learning*	-0.04	65.5%	0.15	96.8%	0.04	66.2%	<0.01	50.0%	-0.03	80.4%	0.21	<0.01-0.53	0.08
Total	*Total*	0.19	99.7%	0.09	91.1%	0.05	74.3%	0.07	95.7%	-0.04	98.3%	0.29	<0.01-0.56	0.15

group size, absolute latitude, and geographic range were not clearly associated with environmentally transmitted parasite richness [Table 2.4].

Previous studies have demonstrated a positive relationship between the frequency of social learning and innovation (Reader et al., 2011). The results presented here are not in conflict with these findings, as we also found such a relationship in our own analyses [Figure 2.3], again with a modest correlation coefficient ($R^2 = 0.23$). Such a result indicates that a large proportion of the unexplained variance must be attributable to other variables, two of which we have presented evidence for: environmentally and socially transmitted parasite species richness.

2.3.3 Terrestriality Results

We found that substrate use predicted parasite richness, with terrestriality covarying positively with socially transmitted parasites, but not with environmentally transmitted parasites [Table 2.5]. This result was contrary to our predictions. One reason for this may be that terrestrial primate species are in closer social and spatial contact than are arboreal species (Dunbar, 1991), an idea supported by our finding that terrestrial species demonstrate increased rates of social learning.

The results of strong positive associations between terrestriality and different measures of parasite transmission may have been partially due to strong associations between terrestrial habits and increased social learning. More terrestrial primate species consistently showed higher levels of both social learning [Figure 2.4a] and exploratory behavior [Figure 2.4b], findings that may be attributable to the increased social cohesion of terrestrial species and their increased time in interaction with terrestrial substrates, respectively. Thus, terrestriality is associated with both an increased richness of socially transmitted parasites and increased rates of social learning, and

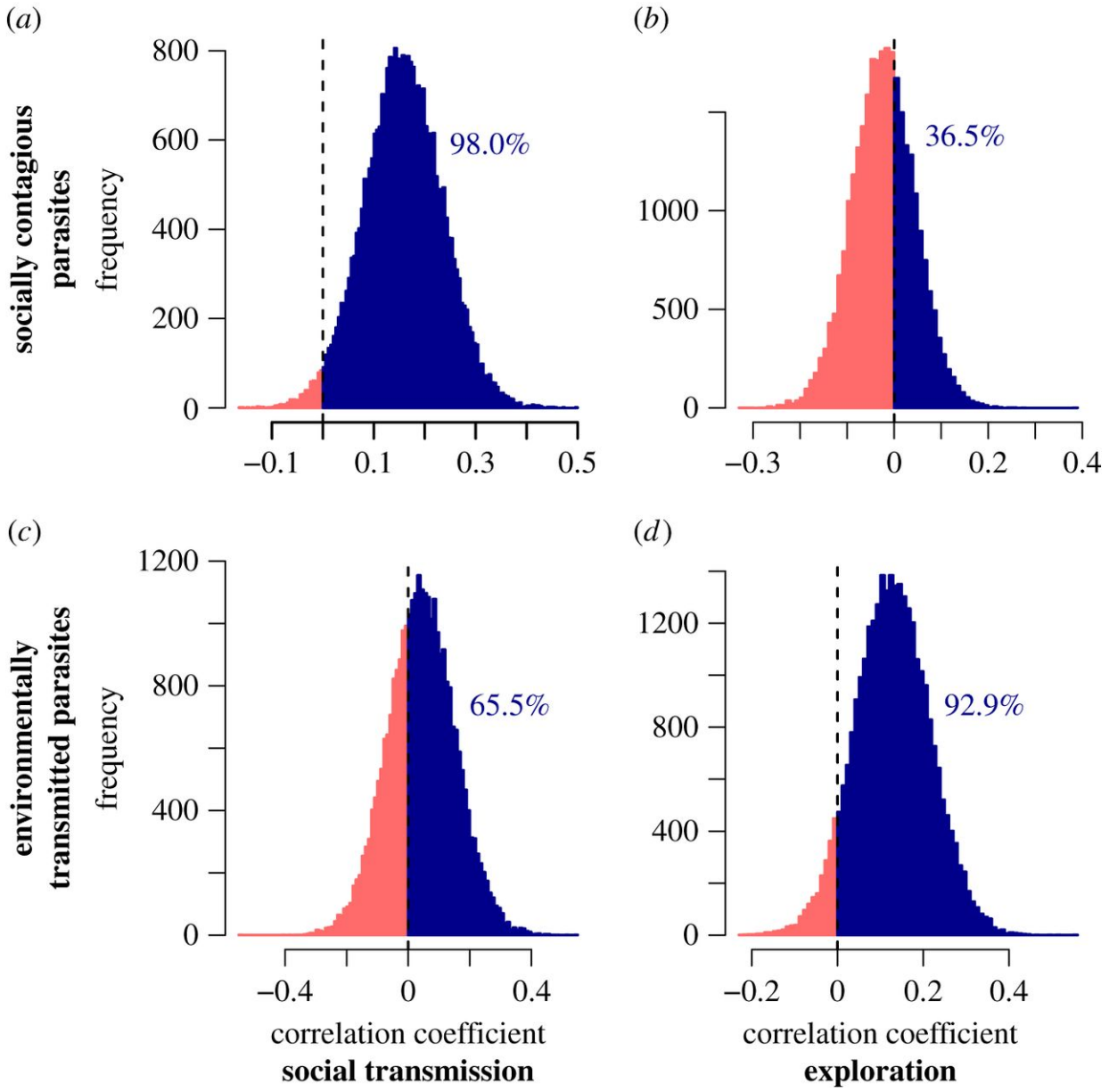

Figure 2.2. Matrix of histograms displaying posterior distributions of β_{BR} (model coefficient for behavior richness) for all transmission mode-specific models. These models each also account for body mass, group size, geographic range, absolute latitude, sampling effort, and phylogenetic uncertainty. Values marked in dark blue represent estimates for β_{BR} that indicate positive associations, light red represents negative associations, and percentages above histograms indicate percentage of positive values estimated in each model (measure of support). Y-axes indicate the parasite transmission mode for which parasite richness was collected, and X-axes the type of behavior examined for a given model. Predicted associations of the "exposure hypothesis" (socially contagious parasite richness and social learning; environmental parasite richness and exploration) show support for positive associations, while non-predicted associations under this hypothesis show no support.

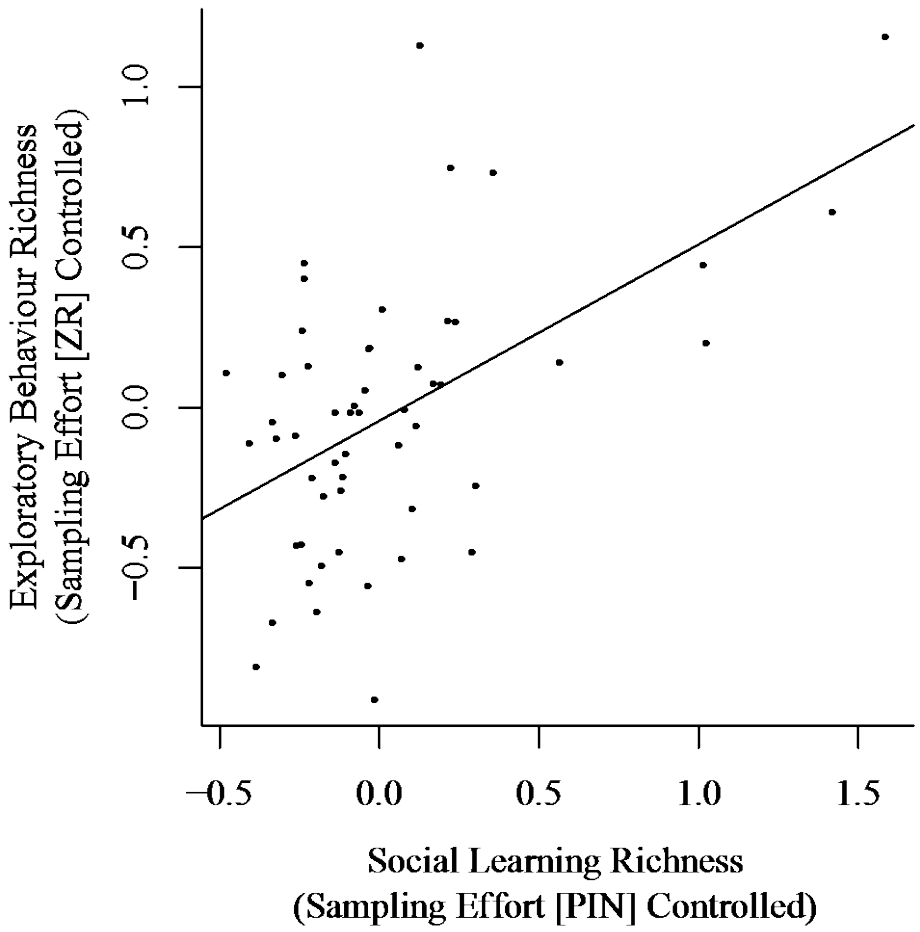

Figure 2.3. Association between social learning richness and exploratory behavior richness. This figure is based on the data included in the exclusive parasite transmission mode models. The line, with a slope of 0.51 and R^2=0.23, indicates average results from 3,000,000 iterations of MCMC Bayesian PGLS with 99.99% of models supporting a positive association after controlling for all socio-ecological variables and research effort in the multiple regression model. Parenthetical descriptors of sampling efforts are as follows: ZR - sampling effort estimated from the Zoological Record; PIN - sampling effort estimated from Primate Information Network's "PrimateLit" database.

an independent association exists between social learning and socially transmitted parasites even while taking terrestriality into account [Table 2.5].

2.4 Discussion

2.4.1 Support for the "Exposure Hypothesis"

Much previous research on innovation, extractive foraging, and social learning has focused on the benefits of these behaviors, yet they may also be associated with considerable costs. Here, the associations we report provide evidence for disease-related costs of behavioral flexibility in primates. Specifically, the total number of parasites covaried positively with richness of reports of innovation, extractive foraging, and social learning. Moreover, parasite transmission mode was linked to the behavioral subcategories we addressed in this study: greater richness of socially learned behaviors was associated with a higher number of socially transmitted parasites, and greater richness of exploratory behaviors was associated with a higher number of environmentally transmitted parasites. Our analyses thus revealed support for the "exposure hypothesis" in primates, and are consistent with the idea that some aspects of social learning and exploration lead to greater exposure to different types of parasites.

Because the richness of socially learned behaviors was positively associated with socially transmitted parasites but not with environmentally transmitted parasites, we propose that social learning either requires, causes, or motivates increased social contact and proximity, leading to the increased spread of socially transmitted parasite species within primate host populations. Alternatively, it could be that some other factor influences both social learning and the transmission of socially transmitted infections. For example, certain parasites may lead to higher rates of social contact (Bouwman and Hawley, 2010), or contagious disease and social learning

Table 2.5. Results of the four mutually exclusive transmission mode Bayesian PGLS models, including terrestriality. Parasite species richness was the response variable and all others (behavior richness, terrestriality, body mass, group size, geographic range, and absolute latitude) were predictors. Reported outputs for each predictor are the mean slopes (β) and proportion of models with predicted slopes (support) from 3,000,000 iterations. Model mean R^2 and mean λ were estimated as the means of all iterations and 95% highest posterior density credibility intervals (95% HPD CI) values for λ were calculated from all results.

Parasite Transmission Mode	Behavioral Measure	Behavior Richness		Terrestriality		Body Mass		Group Size		Geographic Range		Absolute Latitude		Lambda		Mean R^2
		Mean β	Support	Mean β	Support	Mean β	Support	Mean β	Support	Mean β	Support	Mean β	Support	Mean λ	95% HPD CI	
Social	*Exploration*	-0.04	26.50%	0.14	97.50%	0.14	99.00%	-0.1	5.20%	0.09	99.10%	-0.01	75.60%	0.21	<0.01 - 0.48	0.26
Social	*Social Learning*	0.13	94.40%	0.1	91.80%	0.11	97.10%	-0.08	11.70%	0.08	98.20%	-0.02	80.70%	0.19	<0.01 - 0.46	0.29
Environmental	*Exploration*	0.11	90.00%	0.09	82.80%	0.1	86.90%	0.03	65.20%	-0.01	43.60%	-0.03	87.60%	0.2	<0.01 - 0.50	0.12
Environmental	*Social Learning*	<0.01	51.20%	0.11	86.20%	0.12	92.50%	0.02	57.50%	<0.01	50.90%	-0.03	84.80%	0.22	<0.01 - 0.53	0.09
Total	*Total*	0.2	99.80%	-0.08	14.80%	0.11	94.20%	0.06	79.70%	0.07	96.00%	-0.04	97.80%	0.29	<0.01 - 0.55	0.16

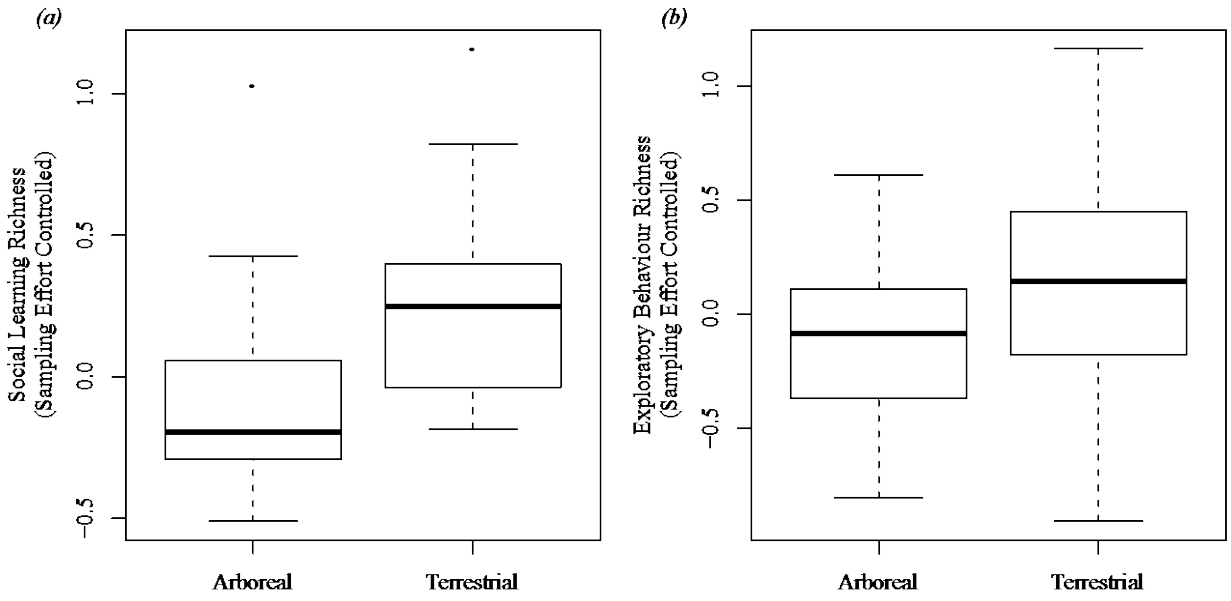

Figure 2.4. Effects of terrestriality on behavioral richness among primates. We investigated the effect of terrestriality on behavior richness in the exclusive parasite transmission mode models. Primate species were categorized as either arboreal or terrestrial. There were significantly more reports per citation of both social learning (a; Support=99.3%) and exploratory behavior (b; Support=94.8%) in terrestrial compared to arboreal primates. Box plots indicate median line, 25th and 75th percentile, with whiskers denoting the 1.5 interquartile range and points indicating outliers.

may covary with other factors that influence social contact, such as grouping and mating patterns (Terborgh and Janson, 1986), fission-fusion dynamics (Aureli et al., 2008), or social network structure (Griffin and Nunn, 2012). Furthermore, since the majority of behavioral reports included in this study were observational rather than experimental, the reports of social learning should be interpreted with caution, as wild studies of this phenomenon are often conducted without sufficient controls to unambiguously identify this learning process (Reader and Biro, 2010). Clearly, experimental data, particularly from wild populations, would be valuable in further tests of our findings.

Because exploratory behavior richness was positively associated with environmentally transmitted parasite richness but not with socially transmitted parasite richness, we propose that increased exploration exposes the host to new infectious diseases. We used innovation and extractive foraging to index exploratory behavior, and thus either these behaviors themselves or their correlates could increase parasite exposure. Exploration was not associated with socially transmitted parasite richness, thus providing no support for the "compensation hypothesis." Similarly, our findings are not consistent with the idea that environmentally and socially transmitted parasites provoke compensatory responses to a different degree, that exploratory behavior and social learning differ in their efficacy as compensatory responses, or these two possibilities in combination. Thus, overall, our analyses support the "exposure hypothesis" and not the "compensation hypothesis."

That social learning and exploration are independently associated with parasites that show distinctly different transmission modes might seem counterintuitive given that social learning, innovation, and extractive foraging are positively correlated in primates (Reader and Laland, 2002; Reader et al., 2011), a result that we replicated using our methods [Figure 2.3],

again finding a modest correlation coefficient (R^2=0.23). Thus, social learning and exploratory behavior are correlated but not collinear, leaving ample independent variation that can be accounted for by factors such as parasitism. Moreover, the disassociation between the results presented in our study concerning exploratory behavior and social learning provide reassurance that the associations with parasite richness are not the result of an unmeasured variable that correlates equally with both behavioral and parasite richness. Furthermore, the finding that social learning and exploratory behavior differentially predict parasite richness provides evidence for divergent validity of these two measures. If measures of social learning and exploratory behavior were confounded, for example through shared sampling biases, we would not expect support for the "exposure hypothesis." Instead, our data suggest that species characterized by high levels of both exploration and social learning, e.g. Hominoidea, *Macaca*, *Cebus,* and *Papio*; (Reader et al., 2011) may pay a 'double cost' of both socially and environmentally transmitted parasites.

2.4.2 Socio-ecological Predictors of Parasite Richness

We also found support for some of the additional ecological, demographic, and geographic hypotheses that we investigated. First, all of our analyses provided support for a positive association between mean body mass and parasite richness. This may reflect that larger bodied organisms have more "niches" available for colonization or that larger-bodied organisms are exposed to more parasites through greater food intake. Secondly, we found positive associations of geographic range size with total and socially transmitted parasite richness, suggesting that socially contagious parasites have the strongest association with expanding range, perhaps driven by increased contact with other closely related species as ranges expand or larger population sizes being able to support more parasites.

No supported positive associations between mean group size and measures of parasite richness were detected in any model tested in this study, consistent with previous comparative work on primates with an earlier version of this database (Nunn et al., 2003). Our results involving social learning and socially transmitted parasites suggest that more refined measures of sociality and social contact within groups may prove more useful for investigating socially transmitted infectious agents (Reader and Biro, 2010; Griffin and Nunn, 2012; Nunn, 2012).

2.4.3 Ameliorating the Costs of Parasitism

Increased parasitism may have profound impacts on host fitness; hence, species expressing greater behavioral flexibility may also possess mechanisms for ameliorating these costs, including through behaviorally flexible traits (Bush et al., 2001). These coping mechanisms fall into two broad categories: physiological/immunological adaptations and avoidance/elimination/self-medication behaviors, such as grooming and the ingestion of medicinal plants or their addition to shelters (Nunn and Altizer, 2006), (Huffman, 1997). Some of these anti-parasite strategies may themselves be facilitated by social processes (such as ectoparasite removal during allogrooming) or be socially learned (Nunn and Altizer, 2006). Comparisons have previously been made between animal self-medication behaviors and human medicine (Hart, 2011), and further study of animal behavioral responses to disease may shed light on the evolution of human medical practices. Additionally, we only investigated one aspect of parasitism; other measures such as prevalence, intensity, or virulence of parasites could provide further insights to the hypotheses that we tested (Nunn and Altizer, 2006).

Based on our findings, we propose that parasites and the infectious diseases that they cause pose substantial costs to behavioral patterns that underlie both human culture and animal

traditions. Enhanced behavioral flexibility may have involved the evolution of counterstrategies to overcome these costs, such as medicative behaviors, or ways to increase the benefits of behavioral flexibility, such as increased cognitive sophistication in social learning (Rendell et al., 2011). As humans, we have experienced a marked increase over recent evolutionary history in our learned behavioral repertoires and in the diversity of parasites that infect us (Barrett et al., 1998). Further comparative and experimental investigation into infectious diseases as a constraint on the evolution of culture may therefore broaden our understanding of cognitive and cultural evolution both in humans and in other animals.

Chapter 3

Effective network size: A novel measure of socially-structured group size

* in review as: McCabe CM, Nunn CL. 2017. Effective network size predicted from simulations of pathogen outbreaks through social networks provides a novel measure of structure-standardized group size. *Frontiers in Veterinary Science.*

Abstract

The transmission of infectious disease through a population is often modeled assuming that interactions occur randomly in groups, with all individuals potentially interacting with all other individuals at an equal rate. However, it is well known that interactions are heterogenous. Here, we propose a measure of effective group size, which refers to a network of random interactions that corresponds to outbreak characteristics of the structured network. We simulated SI and SIR models on maximally-complete networks (i.e. with random interactions among all individuals in the group) to produce idealized outbreak duration distributions for a disease on a network of a given size. We simulated these same diseases on randomly structured networks and then used the resulting outbreak duration distributions to predict an equivalently sized maximally-complete network. The size of this equivalent, idealized network is what we then used as our "effective network size" for the population. Outbreak durations of simulations on randomly structured networks were more variable than those on complete networks, but tended to have similar mean durations of disease spread. We applied this method to investigate whether effective group size improves on group size for predicting parasite richness across primate species. In phylogenetic generalized least squares (PGLS) analyses, our novel measurement of effective network size performed no better than raw network size at predicting parasite species

richness, perhaps because our measures of parasite richness and social network structure were taken from different groups. Overall, our study provides a proof of concept for simulation-based approaches toward constructing metrics of effective network size, while also revealing the conditions under which this approach is most promising. Future work could develop this relationship mathematically or be applied to larger samples of networks that have corresponding data on parasitism, including in humans.

3.1. Introduction

Theoretical models allow us to make sense of complex phenomena by applying a set of simplifying assumptions. In many cases, however, empirical observations of the phenomena do not conform to these assumptions. Understanding how observations compare to their theoretical ideals is thus critical to the interpretation of any such model. Within biology, one of the earliest attempts to compare observations to their theoretical ideals was the work of Sewall Wright on effective population size (Wright, 1931). Effective population size models take an observed population with a certain amount of genetic diversity and predict the size of an idealized population under the assumptions of Fisher-Wright populations that groups are of finite and fixed sizes, individuals mate randomly, and generations do not overlap (Felsenstein, 1971; Crow, 2010; Weissman and Barton, 2012). The generalizability of effective population size allows biologists to compare populations, which is useful in many contexts, including wildlife management and conservation policies (Gomez-Uchida et al., 2013).

Infectious disease represents another phenomenon in which the concept of an idealized population is useful. As with effective population size, a set of simplifying assumptions exist which can be repurposed to formulate theoretically idealized populations, given an observed population. Compartmental disease models aim to predict disease transmission assumptions similar to those in Fisher-Wright populations. For example, they assume that: a) individuals transmit pathogens freely throughout the population, similar to the Fisher-Wright assumption of random mating (the free association assumption); b) individuals do not immigrate or emigrate, maintaining a Fisher-Wright constant population size; and c) there is no age structure within the population, with non-overlapping generations (Anderson and May, 1979). However, these assumptions are rarely upheld in natural populations. As shown through early critiques of

compartmental disease models like Anderson and May (1979), and more recently through the resurgence of social network studies, interactions are not random, but instead structured along social ties between specific individuals based on affiliative interactions, mating, and other social behaviors (White et al., 2015).

Here, we investigated how changes specifically to the free association assumption, through structuring in social networks, affect the time it takes for a disease to fully transmit through a population. To assess the deviation of an observed population from a theoretical ideal in disease transmission through structured groups, we must define what represents an idealized population and disease outbreak. In a review of network modeling of epidemics, Keeling and Eames (2005) suggest that a variety of idealized networks exist, depending on the end goal of the model. The purpose of our model is to allow free-association between individuals in a social network. The earliest modeling of disease transmission through networks was conducted on lattices (Cardy and Grassberger, 1985), with regularly-structured connections between individuals [Figure 3.1a]. However, lattices show too much deviation from the Fisher-Wright assumption of completely free and random association to be used as an idealized population. Instead, given the assumptions of basic compartmental models, the most fitting network arrangement to be used as an ideal is a maximally-complete network, in which each individual has uniform ties to each other individual in the network, allowing effectively free association among nodes [Figure 3.1b].

As for the transmission mode, either deterministic or stochastic models are used to model the transmission of disease. As we are aiming to simplify assumptions about the transmission of disease, deterministic models would provide more straightforward, less complicated views of disease transmission. However, deterministic models require an intimate knowledge of the

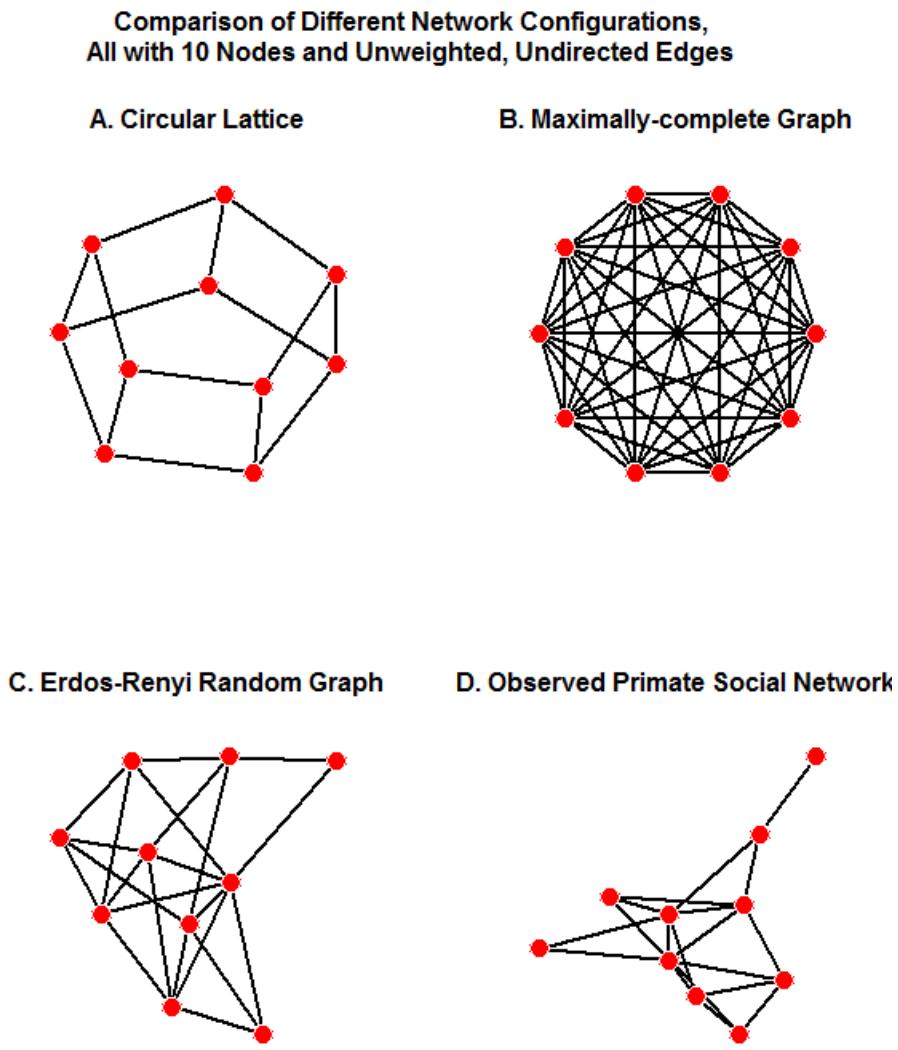

Figure 3.1. Examples of a population of 10 individuals showing various representative network structures, as discussed in the text. These different structures and their applications are: (A) lattice structure as has been used in other network models for disease transmission, where ties are regular, but not exhaustively complete; (B) maximally-complete structure as was used for our idealized networks with free association, each individual is connect to all other individuals in the population; (C) Erdõs-Rényi generation structure with every possible tie existing with a probability of 0.25, thus the number of ties in this graph are a quarter of those present in panel B; (D) an example of an empirically observed network of social interactions among primates (*Pan troglodytes*).

dynamics of disease transmission within a population; unknown variables, such as the effect of social structure on outbreaks, make this sort of modeling impossible. Stochastic models, which are often more representative of real-world variations that underlie disease transmission, allow for uncertainty in variables or dynamics by simulating many different, randomly selected values for important variables (Kimura et al., 2007). For this reason, we employed stochastic models for our study.

Infectious diseases may be transmitted and maintained in populations by a variety of transmission modes. For instance, Susceptible-Infected (SI) models are useful for investigating the transmission of diseases caused by chronic infections, where no recovery is possible; these models include specialized types of SI diseases, like sexually transmitted diseases, where transmission rates vary depending on which sex of individual is interacting. For following disease outbreaks through a population where recovery is possible, the simplest sufficient compartmental model would be a Susceptible-Infected-Recovered (SIR) model, where susceptible individuals may become infected by other infected individuals, but they will eventually recover with immunity to further infection (if they do not die from the disease). To capture the large amount of variation among diverse types of diseases, and to be as relevant as possible to researchers studying a potentially wide variety of pathogens, we investigated SI and SIR models in this study.

Previous work on determining effective network size has focused on very specific aspects of network structure, and has thus maintained restricted conceptions of what constitutes an idealized network. In the only comparable epidemiological research on this topic, Caillaud et al. proposed a measure of "epidemiological effective group size." This metric considered the variation in sub-group size within a meta-population and the impact of this variation on the

outbreak of a disease within the meta-population. By using maximally-complete networks of sub-groups connected to other maximally-complete sub-groups, the researchers calculated the likelihood of an epidemic outbreak throughout the meta-population based on the size of the index sub-group. Thus, Caillaud et al.'s (2013)metric is essentially a novel measure of the critical community size needed to maintain an outbreak (Bartlett, 1960), which in addition to the previous measure, also takes variation in meta-population structure and sub-group size into account.

Additionally, another notion of effective group size has been utilized in estimating the number of distinct cultural behaviors, or cultural richness, that is maintained within a human population. This approach was first theoretically developed by Henrich (2004) using assumptions of even mixing for cultural transmission of multiple behaviors through a population; results of this analysis demonstrated that a decrease in the size of a population through geographic isolation could explain the loss of complex cultural behaviors among Tasmanian islanders. This method was further developed by Powell et al. (2009) to incorporate spatial and temporal variability through estimates of population density and migration rates, respectively. Using this method, the researchers showed that the variability in human population density and migratory activity, resulting in "effective population sizes" for human groups, explained much of the geospatial distribution in cultural behaviors during the Late Pleistocene Epoch. These methods are closely related to those described in our study, in that each is using population structure to explain observed richness, either cultural or parasitic. However, the models for explaining observed richness of human behavior did not explicitly incorporate social network structure; this is the main contribution of our own method.

The first aim of our study is to quantify the relationship between networks of various sizes and outbreak durations for diseases of various transmission modes and epidemiological parameters (focusing on variation in per contact probability of transmission). Here, we expect that infectious diseases transmitted through larger networks will show longer outbreak durations than disease transmitted through smaller networks (Caillaud et al., 2013). We are particularly interested in the mathematical relationship between group size and outbreak duration, as predicted from regression models. The second aim is to provide a proof of concept for our method by generating randomly structured networks and simulating disease transmission through those structured networks to predict equivalent maximally complete networks; we call these the "effective network size" of a social group. Just as we use outbreak durations to establish a baseline mathematical relationship with maximally complete network size, we use this same relationship between network size and outbreak duration to predict the effective network size of randomly structured groups from the outbreak durations of their SI and SIR simulations. Among these simulations, we compare the accuracy and precision of using regression models to predict effective network size from distributions of outbreak durations on the randomly structured networks.

Finally, we apply our new metric for representing disease transmission through networks to investigate the links between group size and parasitism in primates. We predict the effective network size of primate social groups based on their social networks, and then test whether these estimates of effective network size explain variation in parasite richness across species. Previous tests of the hypothesis that animal species with larger group sizes exhibit higher richness of parasites have often produced weak or non-significant results (Nunn et al., 2003; Rifkin et al.,

2012; Nunn et al., 2015). We predicted that effective network size would be a better predictor of parasite richness than raw network size.

3.2 Methods

3.2.1 Simulation and regression of disease transmission on maximally-complete networks

To address the first aim of correlating idealized networks with disease transmission times, we generated maximally-complete, unweighted, undirected networks for groups of size 3 to 200 in R, version 3.3.2 (R Core Team, 2016) with packages igraph (Csardi and Nepusz, 2006) and statnet (Handcock et al., 2008). We then simulated SI models (with a per-contact transmission rate, β, of 0.10, and per capita interactions per day set at 3 times the group size) and SIR models (with an additional parameter, γ, or the daily recovery rate set at 0.10) to saturation or extinction (the points at which pathogens could not be transmitted further) on each of these networks 1000 times. β and γ were both parameterized at 0.1, following previous disease simulations as described in Griffin and Nunn (2012). Per capita social interaction rates per day can be difficult to parameterize, given the wide range of such values in the literature; we chose an arbitrary rate of 3 interactions per individual per day in the analyses presented here.

Our algorithms for disease transmission on networks took place in multiple stages. The first stage involved generating and recording social networks as edgelists, where each social tie between two individuals is recoded as its own row of data. We also tracked the infection status of each node, or individual in the network, as either susceptible, infected, or recovered. From among these nodes, one was selected as an index case, and was infected at the outset of the simulation.

We then selected consecutive random edges, or social ties between individuals, to determine whether the disease would be transmitted from one node to another; the number of edges that were selected depended on the per capita interactions per day, or 3N, and the number of individuals in the network (ranging from 3 to 200). So, for a network of 10 individuals, we chose 30 random edges each day, allowing for the possibility of repeated sampling of social ties. For each of these edges, we checked whether transmission was possible; in our models, the only opportunity for disease transmission was the case where an edge connected an infected individual with a susceptible one, ignoring any directionality in the interaction. Each edge over which transmission was possible resulted in an actual transmission event (where the susceptible individual becomes infected) with probability $\beta = 0.10$, as described above; this would result in 10% of interactions between susceptibles and infecteds resulting in transmission. After all random edges had been considered for a day, each infected individual in SIR models randomly recovered with a probability γ. The simulation then moved to the next day, and only stopped when the criteria for simulation completion were met. No maximum duration was set for either SI or SIR models (because these models would eventually reach either saturation or extinction).

We also considered transmission models where each tie in a graph was sampled once per day, rather than randomly in proportion to the number of nodes. We call this the "alternative model," and give results alongside the other models in section 3.3. By simulating disease spread on maximally complete networks, we inevitably encountered issues of the number of ties in a network not growing proportionally to the number of nodes. This relationship is due to the exponential relationship between network size and the maximum number of ties in that network, as shown in Equation 1:

$$\boldsymbol{Ties\ in\ Network\ of\ size\ N} = \frac{\boldsymbol{N(N-1)}}{\boldsymbol{2}} = \frac{\boldsymbol{N^2 - N}}{\boldsymbol{2}}$$

By simulating the spread of disease on a network of given size N, if we wish to represent each tie once in a day of simulations, the number of ties sampled will be exponentially greater than the number of nodes (for any network larger than 2, which all of our networks were). This leads to unreasonably rapid disease outbreaks at large network sizes. For this reason, a per capita interaction rate, i.e. 3 interactions per node, was chosen, because scaling interactions per day by the size of a network resulted in a total number of ties selected per day that scaled linearly with network size, as shown in Equation 2:

$$\frac{\boldsymbol{3N}}{\boldsymbol{N(N-1)/2}} = \frac{\boldsymbol{6}}{\boldsymbol{N-1}}$$

Additionally, we also considered transmission models where ties were weighted. In such models, ties with greater weights, or intensity of interaction between two individuals, were sampled more often than lesser weighted ties. In these models, ties were still sampled randomly at the per capita interaction rate per day, but the likelihood of sampling a given tie was proportional to its weight. This model is called the "weighted model" in analyses that follow.

We recorded the number of days until the simulation ended as "outbreak duration." For SI models, simulations ended at saturation, defined as the point at which all individuals had transitioned from susceptible to infected. For SIR models, simulations ended at extinction, defined as the point at which no infected individuals were present in the population, either because all susceptible individuals had been infected and subsequently recovered, or because all infected individuals recovered without being able to sustain further transmission to remaining susceptible nodes. We then found a line of best fit through the results for each transmission mode, using the regression model:

$$\boldsymbol{Network\ Size \sim Outbreak\ Duration}$$

For SIR models, we only considered simulations where all individuals had been infected and subsequently recovered were considered sufficient. This resulted in exclusion of 26.9% of simulations in which the disease failed to infect every individual.

To determine under which conditions our method would be most useful, regression models were calculated with raw network size as the response and outbreak duration as the predictor. The association between raw network size and outbreak duration was exponential rather than linear, as would be expected from an exponential growth system like disease transmission. To determine the area of the graphs where we could reliably predict network size from outbreak duration, we used piecewise OLS regressions to predict two separate relationships between outbreak. We did not transform these data at this point, because by splitting the relationship into two separate regressions with piecewise regression, this approach allowed us to identify portions of the graph where prediction could be made appropriately. In the first portion, duration outbreak would show a relatively shallow relationship with network size, making prediction reasonable. But in the second, much steeper portion, relatively small increases in outbreak duration would show much larger increases in predicted network size, making prediction tenuous. We estimated piecewise regression models in R with package segmented (Muggeo, 2003) to determine where the breakpoint between the two portions of the graph would be; this method optimizes the linear fit of each portion by randomly varying the breakpoint until the best split is achieved. We also simulated the simpler SI models with varying values of β to determine if raising or lowering this parameter had any effect on the breakpoint in these piecewise regressions. Such a result would indicate that altering β would allow for better or worse predictions of large network sizes from longer outbreak durations.

In addition to considering piecewise regression models, we separately ran regression models with log-transformed network sizes to achieve a linear fit. For each set of 1000 iterations of disease simulation on a given network, outbreak durations were quite variable. Thus, we used reduced major axis (RMA), estimates of model II regressions to control for the uncertainty in outbreak duration in addition to that in network size, calculated in R with package lmodel2 (Legendre, 2014). RMA estimates consider the variation in both the independent and dependent variable when fitting regression models rather than, as in OLS models, only considering variation in the dependent variable. RMA provided the most suitable control for estimating how variation in outbreak duration would affect our predictions of fixed network sizes.

We then exponential-transformed the output of these equations to counteract the prior log-transformation; these exponential-transformed equations formed the basis for calculating "effective" network sizes from outbreak durations of diseases simulated on observed networks. Back-transformations from log transformed data introduce bias into predicted values because of the difference between errors in log-transformed variables and their untransformed counterparts (Smith, 1993; Hayes and Shonkwiler, 2006). We considered accounting for this bias by using the "consistent I estimator" from Hayes and Shonkwiler (2006), and compared this approach to our own method of calculating network size from the uncorrected RMA models; the equation for the consistent I estimator is:

$$\boldsymbol{y = e^{ln(a)+b\ ln(x)+(\frac{s^2}{2})}}$$

Where a is the intercept, b is the slope, x is the independent variable, and s^2 is the mean squared error for the model. Because mean squared error is constant within each model, such a correction would create a consistent upward shift in all estimates of network size by a value of $s^2/2$; this would not have any impact on further linear models' slope coefficients, and so uncorrected RMA

model back-transformations were chosen for simplicity of interpretation throughout the main text. However, for the sake of comparison, back-transformed results are presented along the untransformed results in section 3.3.

3.2.3 Accuracy, precision of predicting effective network size from randomly structured graphs

To investigate the second aim, we generated large sets of Erdõs-Rényi (E-R) graphs [Figure 3.1c] for predetermined group sizes and predetermined proportions of ties present; although, to reduce variability, these were used as set numbers of ties, rather than probability that ties would be present between two given nodes) in R with package igraph (Csardi and Nepusz, 2006). Random graphs were used as the baseline in this case because they represented the only source from which we could obtain a large enough sample size to validate our methods. Group sizes for these were kept smaller than the maximally-complete networks to allow for direct comparison of outbreak duration distributions, and are in good agreement with the observed network sizes of primates ranging from 4 to 35 typically (Kasper and Voelkl, 2009). Tie proportions were kept relatively low to increase differentiation from maximally-complete networks. We sampled blocks of 111 networks for each combination of group size (10, 30, 50) and tie proportion (15%, 25%, and 35% of possible ties), generating 999 total random networks. To ensure that disease simulations could reach full saturation and (for SIR) subsequent extinction, we screened each randomly generated network to ensure that all nodes were part of a single, connected network.

We then simulated the same SI and SIR models (as discussed in section 3.2.1) over 1000 iterations on each of our 999 randomly generated models, recording outbreak durations of the models. Because all outbreak durations for random networks of size N are expected to be greater

than those of the idealized network of size N, these simulations were conducted to determine the scale of increase in outbreak durations and consequently in effective network size. The mean of outbreak durations for a given random network with a given transmission model were used as the predictor variable in the RMA regression equations described in section 3.2.1. Only simulations which reached saturation were analyzed here, and so some runs of the SIR simulations were removed due to stochastic extinction events. This reduced the sample size of analyzed simulation runs, and may have biased our results for SIR comparisons. These values were then exponential-transformed and rounded to the nearest integer to arrive at a directly comparable effective network size for each random network. Thus, effective network sizes were calculated twice for each random network; once for SI models and once for SIR models.

To gauge the accuracy and precision of our methods, we compared each distribution of outbreak durations on a given E-R network (hereafter, observed) to that of the original outbreak durations on the maximally-complete network of the same size as the predicted effective network size of the observed network (hereafter, effective). We compared these distributions graphically and statistically. For accuracy, we compared the observed and effective network distributions in means of outbreak durations, with more similar means indicating that simulating disease spread on effective networks is more accurately capturing expected spread on the observed network. For precision, we compared the observed and effective network distributions in standard deviations of outbreak durations, with more similar standard deviations indicating that the precision of simulating disease spread on effective networks is similar to what would be obtained on the actual networks. We statistically compared the distributions of outbreak durations between observed and effective network simulations with Kolmogorov-Smirnov tests in R with package

dgof (Arnold and Emerson, 2011). Significance on these tests indicates that the two distributions likely did not come from the same original distribution.

3.2.4 Application to tests of association between group size and parasitism

We used our predictive models to estimate effective network sizes of primate social networks that had been recorded in the literature [e.g. Figure 3.1d]. These networks mainly consisted of the dataset collected by Griffin and Nunn (2012), although they were supplemented with more recent publications as well. A full listing of the sources for each of these networks, as well as the species and interaction type to which each corresponds, is provided in Table 3.1. Effective network sizes were again calculated by simulating SI and SIR transmission and then inputting the resulting outbreak duration means into the equations described in section 3.2.1. The primate phylogeny used in our analysis [Figure 3.2] was downloaded from 10kTrees, version 3 (Arnold et al., 2010), following the Corbet and Hill taxonomy (1991), and imported into R with package ape (Paradis et al., 2004).

Parasite richness estimates considered were total parasite richness recorded in the literature, and subsets including only socially-transmitted parasites and subsetted to helminths, protozoa, and viruses. Richness measurements were obtained from the Global Mammal Parasite Database (Nunn and Altizer, 2005). We tested the fit of the following linear model with PGLS analyses in R with package caper (Orme et al., 2013):

$$\boldsymbol{Parasite\ Richness} \sim (\boldsymbol{Group\ size} \,||\, \boldsymbol{Mass} \,||\, \boldsymbol{Geographic\ range}) + \boldsymbol{Citation\ count}$$

Citation counts were based on the number of citations found in Web of Science for each primate species and included as a measure of sampling effort as a covariate in each model. We included geographic range size estimates and mean body mass estimates for each primate species from

Table 3.1. Raw and effective network sizes of primate species included in the PGLS models, as well as source information for each of the networks. "Network Size" is the count of nodes in the observed primate network. "ENS" indicates effective network size, with "SI" or "SIR" indicating the type of transmission model used for estimating effective network size, and "Weighted" indicating that tie weights were also included in simulations for estimating effective network size.

Species	Group Size	ENS SI	ENS SIR	Weighted ENS SI	Weighted ENS SIR	Group Status	Interaction Class	Source
Alouatta caraya	5	7	8	9	7	Captive	Grooming	(Jones, 1983)
Ateles geoffroyi	15	36	22	75	23	Free-ranging	Grooming	(Ahumada, 1992)
Cebus apella	12	20	18	43	18	Wild	Grooming	(Izawa, 1980)
Cebus capucinus	6	9	10	9	9	Wild	Grooming	(Perry, 1996)
Cercopithecus aethiops	8	11	13	15	12	Wild	Grooming	(Seyfarth, 1980)
Cercopithecus mitis	16	43	21	57	26	Wild	Grooming	(Cords, 2000)
Colobus guereza	8	13	13	43	13	Wild	Grooming	(Dunbar and Dunbar, 1976)
Eulemur fulvus	11	16	16	20	15	Free-ranging	Proximity	(Jacobs et al., 2011)
Lemur catta	12	16	17	20	16	Wild	Proximity	(Kendal et al., 2010)
Macaca arctoides	19	31	26	53	26	Captive	Grooming	(Butovskaya et al., 1994)
Macaca assamensis	19	36	26	79	28	Wild	Grooming	(Cooper et al., 2005)
Macaca fascicularis	10	20	15	70	17	Captive	Grooming	(Butovskaya et al., 1996)
Macaca mulatta	28	34	35	37	30	Captive	Proximity	(Massen and Sterck, 2013)
Macaca radiata	16	25	22	32	22	Wild	Grooming	(Sugiyama, 1971)
Miopithecus talapoin	8	11	13	16	12	Captive	Grooming	(Wolfheim, 1977)
Pan troglodytes	7	10	11	12	9	Wild	Grooming	(Sugiyama, 1988)
Papio ursinus	14	24	21	27	18	Wild	Grooming	(King et al., 2011)
Saguinus fuscicollis	7	10	12	16	11	Captive	Grooming	(Vogt, 1978)
Saguinus mystax	6	9	10	10	9	Wild	Grooming	(Löttker et al., 2007)
Theropithecus gelada	7	15	12	16	10	Captive	Socio-positive	(Dunbar, 1982)

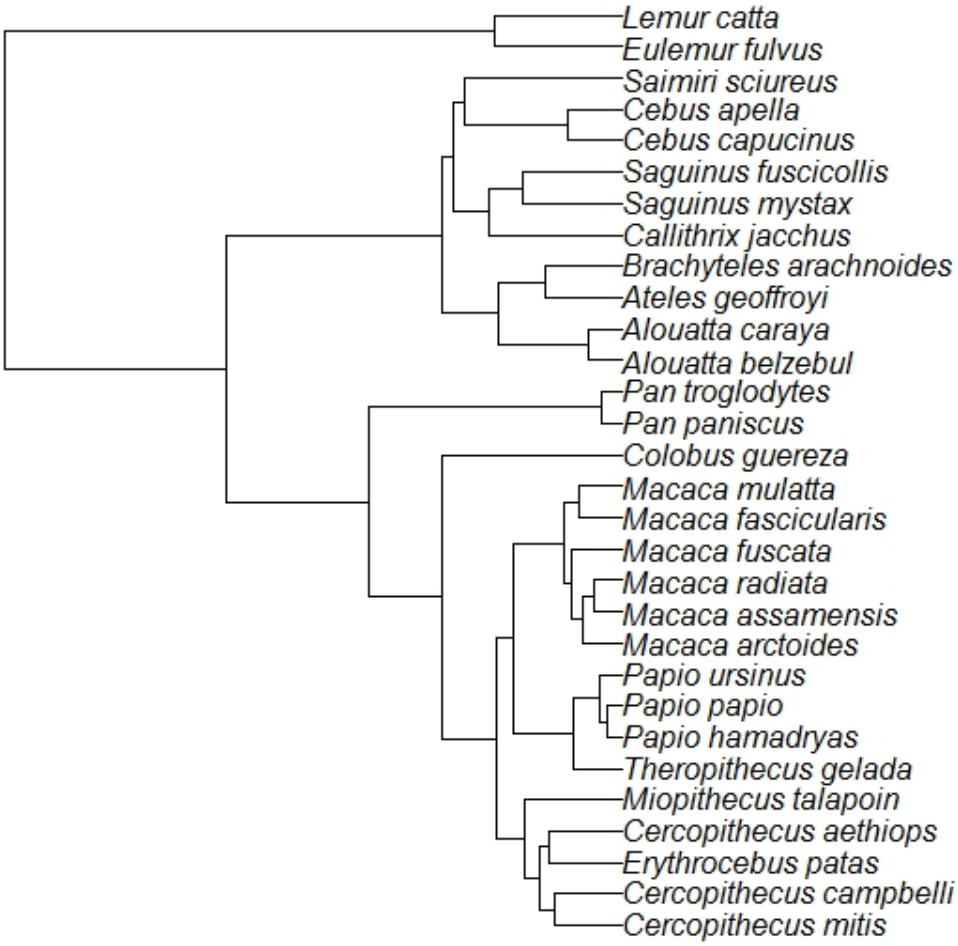

Figure 3.2. Phylogenetic tree of primate species for which we tested hypotheses about associations between group size and parasite richness. Tree shows poor coverage of certain taxa of primates, such as strepsirrhines, for which only 2 species, *Lemur catta* and *Eulemur fulvus*, had published social network data.

McCabe et al. (2014), as these have been previously supported in the literature as reliable predictors of parasite richness in primates (Nunn et al., 2003; McCabe et al., 2014). We conducted these analyses as a rough indicator of whether our small sample of primate species might impact the outcome of our tests; if we detected positive associations among these variables, but not among our measures of group size, then we could conclude that we had a representative sample of primate species.

Each measure of richness listed above was predicted to positively covary with group size. The measurements of group size tested were: observed (raw) sizes of primate networks, and effective network sizes from SI and SIR simulations, repeated for weighted and unweighted networks. All variables were log-transformed to meet assumptions of the PGLS analyses and z-transformed to facilitate comparison between different predictor variables (Mundry, 2014). Although some primates were represented multiple times in our dataset of published social networks, only one network per species ever met our condition of having an effective network size of less than cutoffs that were determined in the piecewise regression; thus, we did not need to account for any intraspecific variation. In total, 20 primate species were included in our analyses. Effective network sizes were predicted to be better estimators of parasite richness than the observed raw network sizes. We tested this prediction in a model comparison framework with AICc as the model selection criterion, using a cutoff of 2 AICc units for preferring a model over other models. AICc values were calculated in R with package MuMIn (Barton, 2016).

3.3 Results

Optimization of piecewise regression models estimated a break at a network size of 80 nodes, indicating that predictions of effective network sizes above 80 individuals would be

considerably less reliable than those of 80 or below. Furthermore, altering the values for β had no effect on the breakpoints, although as would be expected, the ranges of outbreak durations were inversely related to the value for β [Figure 3.3]. All piecewise regressions revealed breaks at between 79.45 and 81.75 nodes. RMA model II regressions of log-transformed maximally-complete network size versus outbreak duration for SI and SIR models fit relatively well, with R^2 of 0.532 and 0.438 respectively [Figure 3.4]. The regression equations, listed in Figure panels 3.4a and 3.4b, were then used to calculate effective network size. Alternative model results, with ties sampled regularly rather than randomly, showed similar results for SIR models, but tended to oversample ties in large networks for SI models, leading to unreasonably short outbreak durations in these networks [Figure 3.5].

We then compared the distributions of E-R graph (observed) outbreak durations to those of their equivalent maximally complete (effective) network's outbreak durations to assess accuracy and precision. This was done to determine whether disease outbreaks on observed networks were accurate, or similar to those on maximally complete networks, in terms of the distributions of the outbreak durations from simulations on effective and observed networks. Figure 3.6 shows the results of the SI model comparisons, and back-transformed results show similar patterns [Figure 3.7]. Accuracy of our RMA predictive model was high, with means similar between observed and effective network outbreak durations [Figure 3.6b], but outbreak durations from observed network simulations showed higher standard deviations than those from effective networks [Figure 3.6c]. Kolmogorov-Smirnov tests show that these two sets of distributions were often significantly different, with a critical value for the D-statistic at 0.60 [Figure 3.6d]. However, this method is extremely sensitive to small changes in distributions and

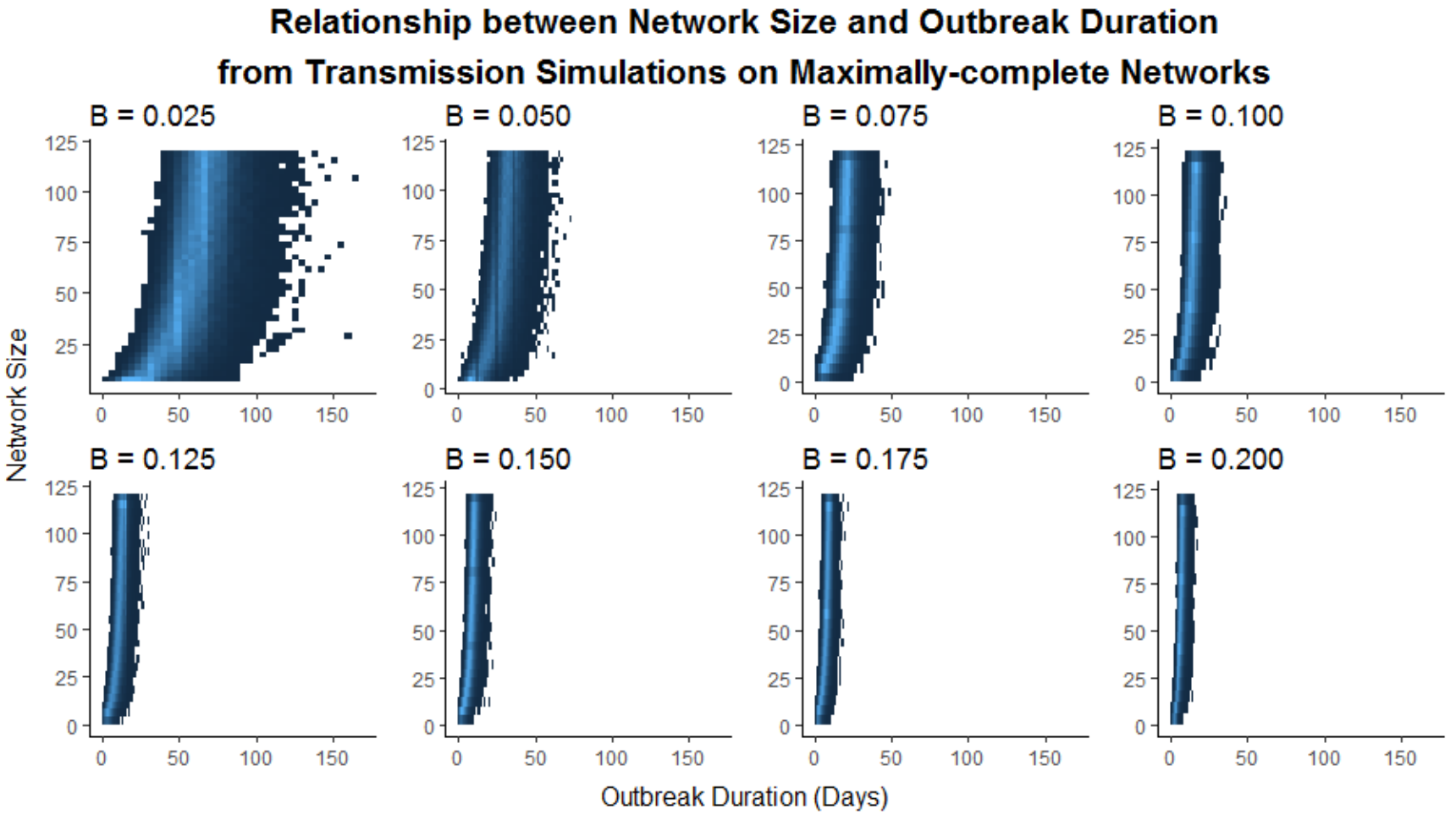

Figure 3.3. Comparison between distributions of outbreak durations for SI simulations with varying values for β. Lower values for β have larger ranges of outbreak durations, but the shapes of curves are qualitatively similar when scaled to the maximum outbreak duration for a given value of β.

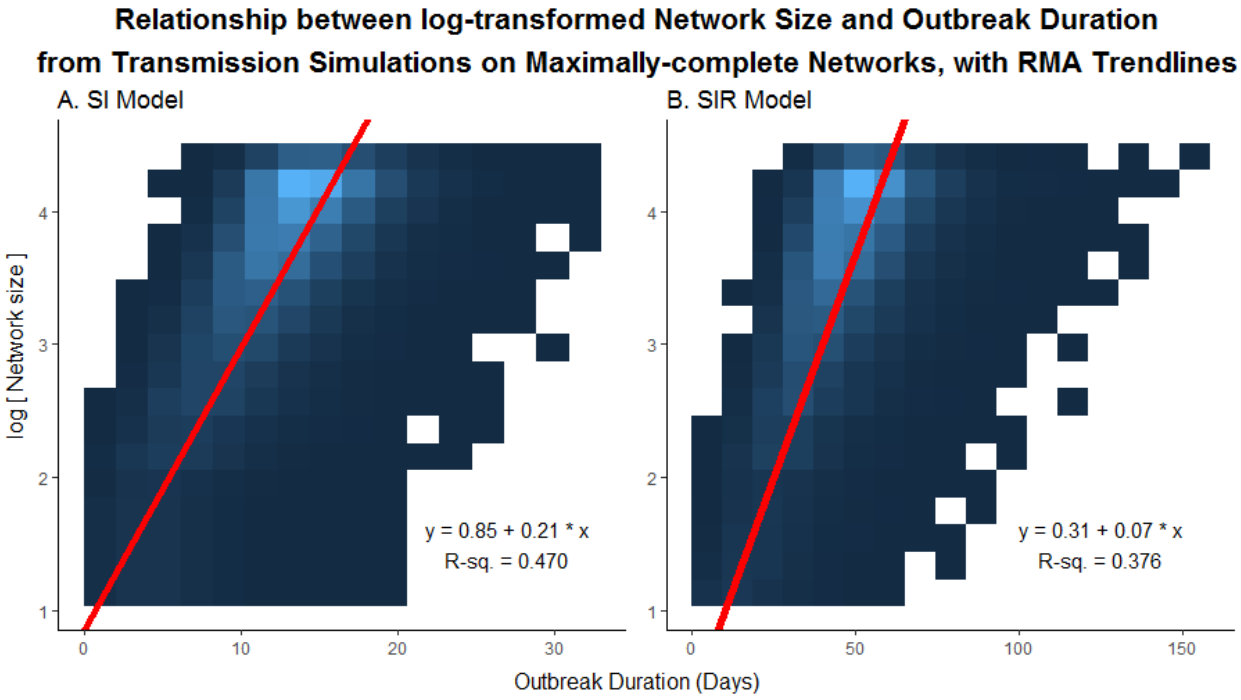

Figure 3.4. Associations between log-transformed network size and outbreak duration for different disease models. Data points for each graph, limited to networks of 80 nodes or less (n=78,000), were too dense to make scatterplot representations intelligible, thus heatmaps were used to illustrate the results, with lighter colors of blue representing a higher density of data points. Log-transforming network size makes for a linear relationship, and reduced major axis (RMA) model 2 regression lines, represented in red, account best for the joint variation in the x and y axes.

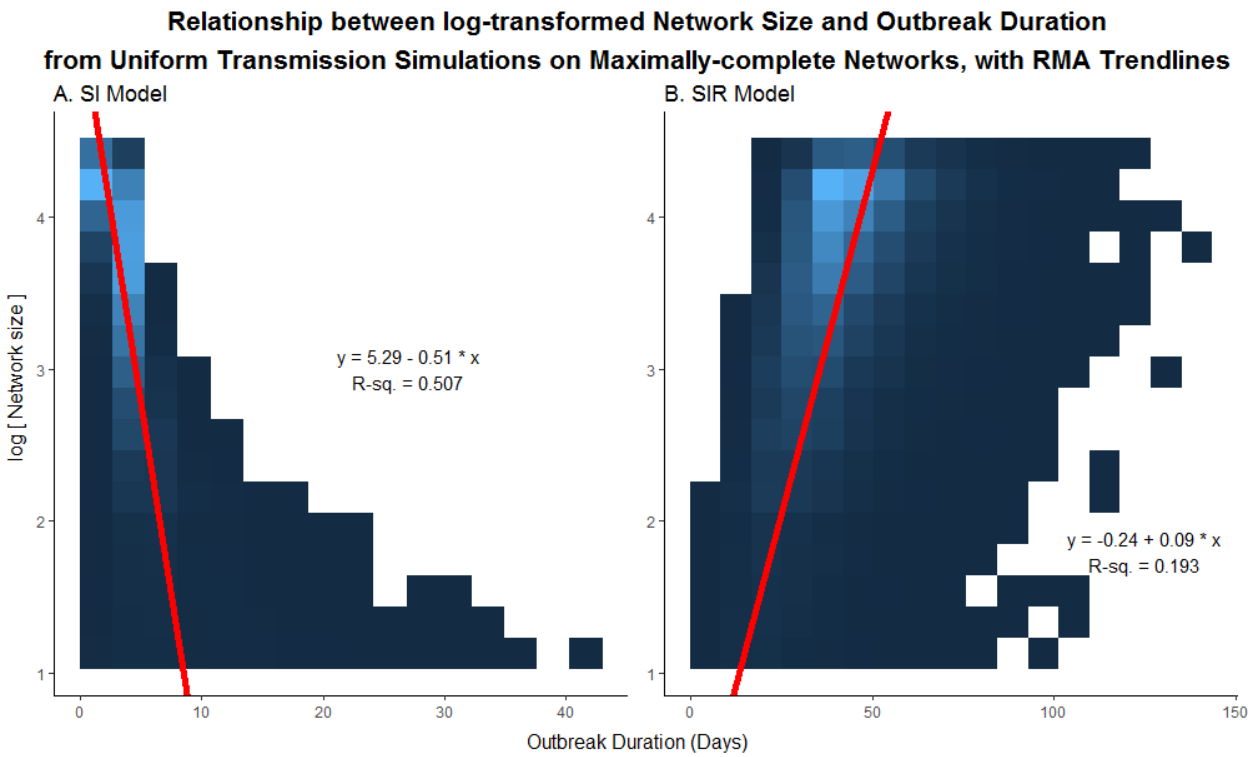

Figure 3.5. Associations between log-transformed network size and outbreak duration for different disease models using alternative models with uniform tie selection per day rather than random tie selection using per capita interaction rates per day. Data points for each graph, limited to networks of 80 nodes or less (n=77,000), were too dense to make scatterplot representations intelligible, thus heatmaps were used to illustrate the results, with lighter colors of blue representing a higher density of data points. Log-transforming network size makes for a linear relationship, and reduced major axis (RMA) model 2 regression lines, represented in red, account best for the joint variation in the x and y axes. Both SI and SIR models show a much steeper increase in outbreak duration as network size increases than do the per capita interaction rate models, but the relationship is most apparent in SI models, where recovery does not counteract the spread of disease. Given the exponential relationship between network size and the maximum number of possible ties present in that network [Supplementary Equation 1], there are more and more possible routes for disease transmission through a maximally connected network as the number of nodes increases, causing a seemingly negative relationship between network size and outbreak duration. For this reason, per capita interaction rates for networks were chosen as a more representative transmission model for our methods.

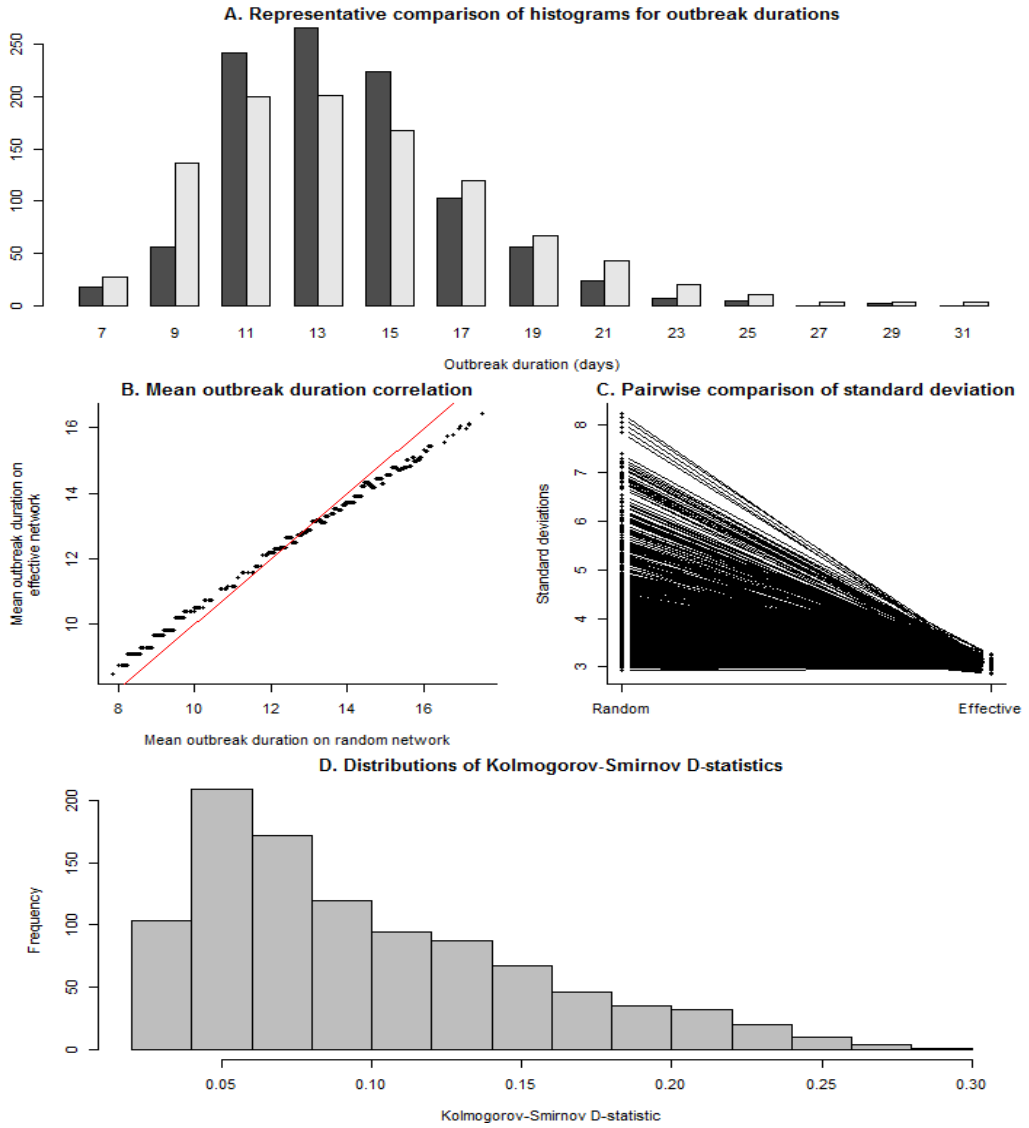

Figure 3.6. Comparison between distributions of outbreak durations for SI simulations on observed and effective network. Throughout the figure, the term "observed" refers to results from simulations on E-R graphs, and "effective" refers to results from simulations on RMA-predicted equivalent maximally-complete networks. Network sizes are limited to a maximum of 80 individuals, as this was the condition under which we were reasonably confident in our results. Panel A, a histogram with a representative pair of observed (dark gray) and effective (light gray) distributions of outbreak durations plotted together for viewing overlaps, shows that the distributions, compared on a pairwise scale had a considerable amount of overlap. Panel B shows means of outbreak durations from observed networks plotted against those from their predicted effective networks; red line indicates 1:1 equivalence, at which effective means match observed means. Panel C shows a paired line plot of standard deviations in outbreak durations for simulations on observed and effective networks; observed networks showed higher standard deviations than their paired effective networks. Panel D shows a histogram of Kolmogorov-Smirnov D-statistics for pairwise statistical comparisons between observed and effective network outbreak durations, with values above 0.60 indicating significantly different distributions.

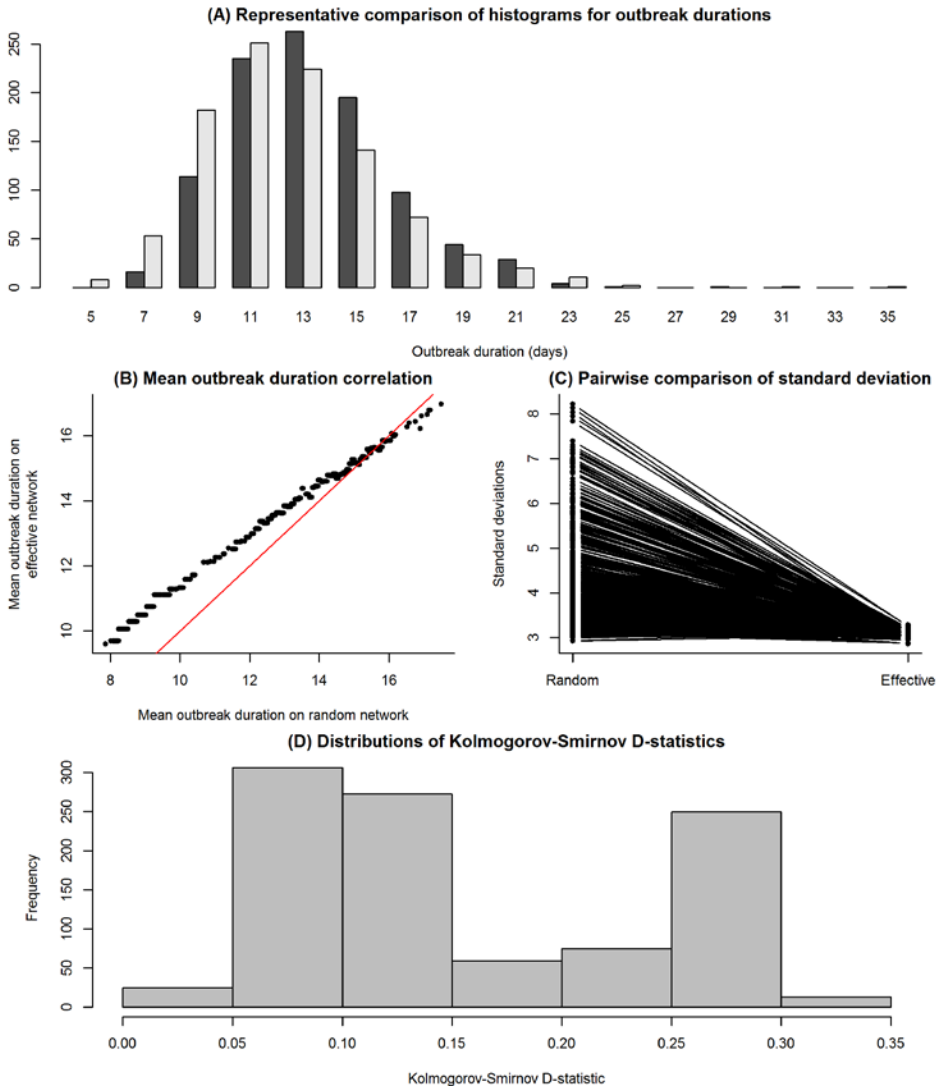

Figure 3.7. Comparison between distributions of outbreak durations for SI simulations on observed and effective network, where effective network size estimates have been back-transformed by the consistent I estimator (24). Throughout the figure, the term "observed" refers to results from simulations on E-R graphs, and "effective" refers to results from simulations on RMA-predicted equivalent maximally-complete networks. Network sizes are limited to a maximum of 80 individuals, as this was the condition under which we were reasonably confident in our results. Panel A, a histogram with a representative pair of observed (dark gray) and effective (light gray) distributions of outbreak durations plotted together for viewing overlaps, shows that the distributions, compared on a pairwise scale had a considerable amount of overlap. Panel B shows means of outbreak durations from observed networks plotted against those from their predicted effective networks; red line indicates 1:1 equivalence, at which effective means match observed means. Panel C shows a paired line plot of standard deviations in outbreak durations for simulations on observed and effective networks; observed networks showed higher standard deviations than their paired effective networks. Panel D shows a histogram of Kolmogorov-Smirnov D-statistics for pairwise statistical comparisons between observed and effective network outbreak durations, with D>0.60 indicating significantly different distributions.

may not be best suited for determining similarity between the observed and effective network outbreak duration distributions.

Figure 3.8 shows the results of the SIR model comparisons between effective and observed network simulations, and back-transformed results again showed similar results [Figure 3.9]. Accuracy, or similarity between mean values of outbreak durations for simulations on effective and observed networks was again high [Figure 3.8b], but outbreak durations from observed network simulations actually showed lower standard deviations than those from effective networks [Figure 3.8c]. Kolmogorov-Smirnov tests show that these two sets of distributions were often significantly different, again with a critical value for the D-statistic at 0.60 [Figure 3.8d].

Networks included in the PGLS models are presented in Table 3.1 with information about their sources in the literature and calculated effective network sizes. Results for PGLS analyses indicated, in general, no better performance of effective network size over raw network size in predicting parasite richness [Table 3.2]. Similar results were found for weighted models, although AICc values for weighted models were lower across the board than were their equivalent unweighted ones for the same sample of species and richness [Table 3.3]. Body size and geographic range were also not predictors of parasite richness across the board for the species included [Table 3.2]. However, within these results, body mass was positively associated with close-contact transmitted parasite richness, and it had a lower AICc value than all other close-contact richness models.

3.4 Discussion

These results demonstrate the potential for using effective network size to compare infectious disease risk across groups of different sizes, including potentially for understanding

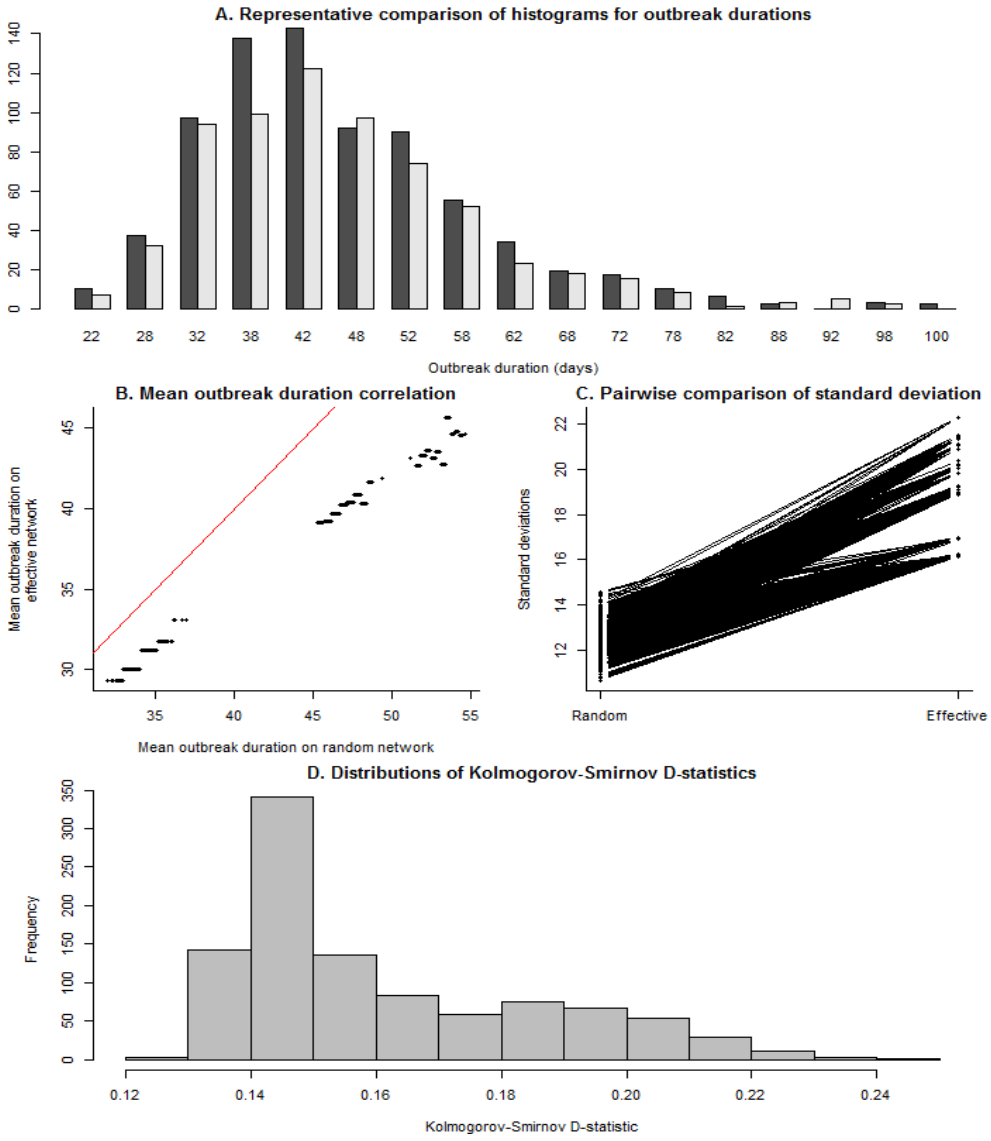

Figure 3.8. Comparison between distributions of outbreak durations for SIR simulations on observed and effective network. Again, the term "observed" refers to results from simulations on E-R graphs, and "effective" refers to results from simulations on RMA-predicted equivalent maximally-complete networks. Network sizes are also limited to a maximum of 80 individuals, as this was the condition under which we were reasonably confident in our results. Panel A, a histogram with a representative pair of observed (dark gray) and effective (light gray) distributions of outbreak durations plotted together for viewing overlaps, shows that the distributions, compared on a pairwise scale had a considerable amount of overlap. Panel B shows means of outbreak durations from observed networks plotted against those from their predicted effective networks; red line indicates 1:1 equivalence, at which effective means match observed means. Panel C shows a paired line plot of standard deviations in outbreak durations for simulations on observed and effective networks; observed networks showed higher standard deviations than their paired effective networks. Panel D shows a histogram of Kolmogorov-Smirnov D-statistics for pairwise statistical comparisons between observed and effective network outbreak durations, with values above 0.60 indicating significantly different distributions.

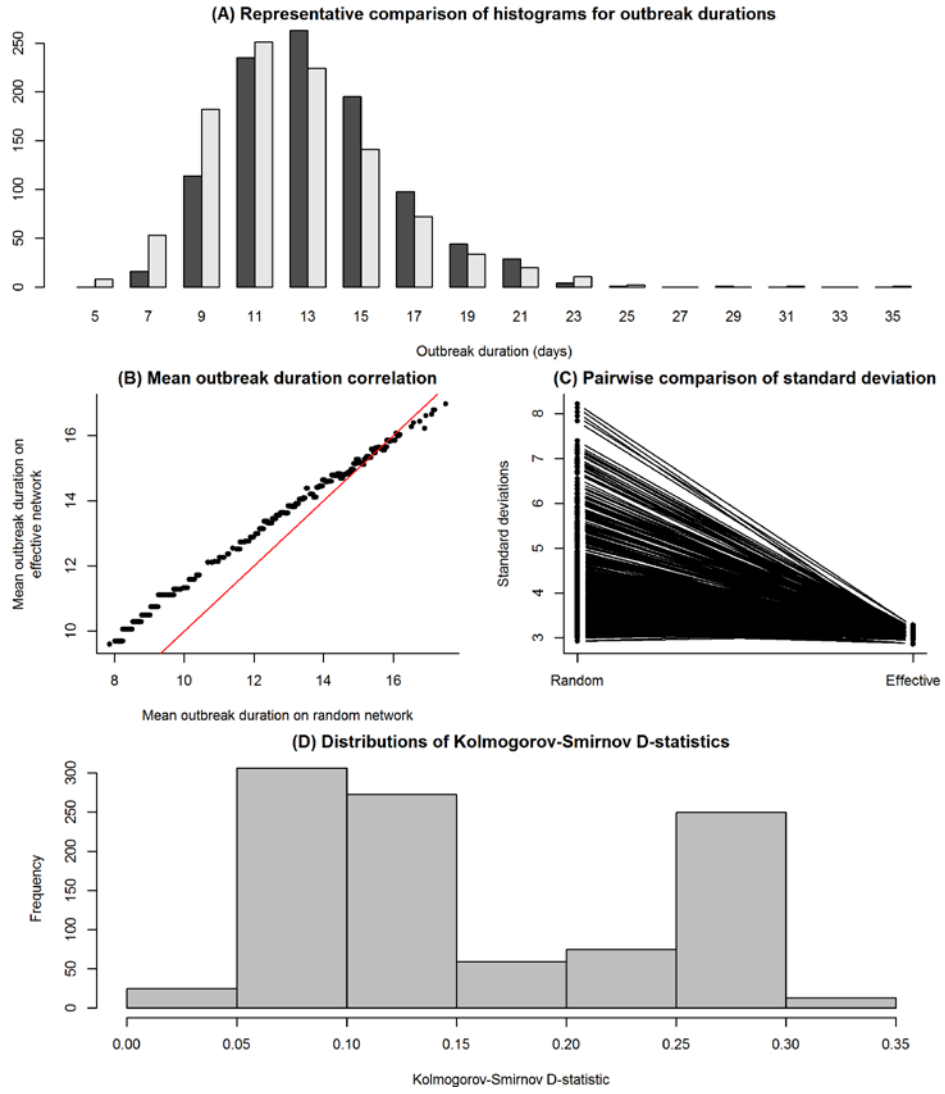

Figure 3.9. Comparison between distributions of outbreak durations for SIR simulations on observed and effective networks, where effective network size estimates have been back-transformed by the consistent I estimator (24). Again, the term "observed" refers to results from simulations on E-R graphs, and "effective" refers to results from simulations on RMA-predicted equivalent maximally-complete networks. Network sizes are also limited to a maximum of 80 individuals, as this was the condition under which we were reasonably confident in our results. Panel A, a histogram with a representative pair of observed (dark gray) and effective (light gray) distributions of outbreak durations plotted together for viewing overlaps, shows that the distributions, compared on a pairwise scale had a considerable amount of overlap. Panel B shows means of outbreak durations from observed networks plotted against those from their predicted effective networks; red line indicates 1:1 equivalence, at which effective means match observed means. Panel C shows a paired line plot of standard deviations in outbreak durations for simulations on observed and effective networks; observed networks showed higher standard deviations than their paired effective networks. Panel D shows a histogram of Kolmogorov-Smirnov D-statistics for pairwise statistical comparisons between observed and effective network outbreak durations, with values above 0.60 indicating significantly different distributions.

Table 3.2. Richness model comparison between effective network sizes, observed raw network size, geographic range size, and body mass for 22 primate species. Reported are slope estimates, p-values, and AICc values for each model. Models are indicated by the independent variable in rows ($N_{en,SI}$ = effective network size from SI equation; $N_{en,SIR}$ = effective network size from SIR equation; N_{obs} = observed raw network size; GR = geographic range; BM = body mass) and the dependent variable, which specific measure of parasite richness was used, in columns.

	Parasite Species Richness				
	Total	*Close-transmitted*	*Helminth*	*Protozoa*	*Virus*
$N_{en,SI}$	β = -0.238 p = 0.253 AICc = 55.6	β = -0.105 p = 0.653 AICc = 51.2	β = -0.241 p = 0.288 AICc = 49.4	β = -0.174 p = 0.459 AICc = 47.6	β = -0.198 p = 0.422 AICc = 33.8
$N_{en,SIR}$	β = -0.270 p = 0.201 AICc = 55.2	β = -0.151 p = 0.544 AICc = 51.0	β = -0.292 p = 0.215 AICc = 48.9	β = -0.356 p = 0.133 AICc = 45.4	β = 0.071 p = 0.784 AICc = 34.5
N_{obs}	β = -0.256 p = 0.227 AICc = 55.4	β = -0.132 p = 0.603 AICc = 51.2	β = -0.285 p = 0.232 AICc = 49.0	β = -0.340 p = 0.157 AICc = 45.7	β = 0.069 p = 0.785 AICc = 34.5
GR	β = 0.188 p = 0.409 AICc = 56.4	β = 0.440 p = 0.171 AICc = 49.3	β = 0.047 p = 0.868 AICc = 50.8	β = 0.247 p = 0.389 AICc = 47.3	β = -0.040 p = 0.918 AICc = 34.6
BM	β = -0.124 p = 0.567 AICc = 56.8	β = 0.562 p = 0.020 AICc = 45.9	β = -0.196 p = 0.389 AICc = 49.9	β = 0.248 p = 0.353 AICc = 47.1	β = 0.287 p = 0.353 AICc = 33.5

Table 3.3. Richness model comparison between effective network sizes, observed raw network size, geographic range size, and body mass for 20 primate species, using weighted models which sample ties in proportion to their weights. Reported are slope estimates, p-values, and AICc values for each model. Models are indicated by the independent variable in rows ($N_{en,SI}$ = effective network size from SI equation; $N_{en,SIR}$ = effective network size from SIR equation; N_{obs} = observed raw network size; GR = geographic range; BM = body mass) and the dependent variable, which specific measure of parasite richness was used, in columns.

	Parasite Species Richness				
	Total	*Close-transmitted*	*Helminth*	*Protozoa*	*Virus*
$N_{en,SI,w}$	β = -0.204 p = 0.328 AICc = 53.4	β = -0.257 p = 0.280 AICc = 47.7	β = -0.206 p = 0.361 AICc = 47.2	β = -0.255 p = 0.293 AICc = 44.6	β = -0.475 p = 0.062 AICc = 29.8
$N_{en,SIR,w}$	β = -0.227 p = 0.277 AICc = 53.1	β = -0.201 p = 0.423 AICc = 48.3	β = -0.235 p = 0.317 AICc = 47.0	β = -0.311 p = 0.201 AICc = 43.9	β = 0.239 p = 0.385 AICc = 33.7
N_{obs}	β = -0.256 p = 0.227 AICc = 52.7	β = -0.134 p = 0.599 AICc = 48.7	β = -0.287 p = 0.230 AICc = 46.4	β = -0.336 p = 0.168 AICc = 43.6	β = -0.069 p = 0.785 AICc = 34.5
GR	β = 0.376 p = 0.169 AICc = 52.2	β = 0.535 p = 0.117 AICc = 46.1	β = 0.253 p = 0.523 AICc = 47.7	β = 0.070 p = 0.879 AICc = 46.0	β = -0.040 p = 0.918 AICc = 34.6
BM	β = -0.110 p = 0.615 AICc = 54.2	β = 0.546 p = 0.025 AICc = 44.0	β = -0.182 p = 0.429 AICc = 47.4	β = 0.216 p = 0.432 AICc = 45.3	β = 0.287 p = 0.354 AICc = 33.5

disease transmission across a mosaic of many loosely connected groups within a larger meta-population structure. Unfortunately, as our PGLS results indicated, these novel measures of effective network size proved to be, in most cases, no better than raw group size at predicting parasite richness of a species. This could have been for a variety of reasons. Primary among these, the estimates for parasite richness and primate social networks that we used in this study came from separate sources, which complicates any potential relationship between network structure and richness. Relationships between social networks and parasitism are more than likely dependent on the specific group being observed, and so ideally, measures of parasite richness and network structure should come from the same group. Empirical studies considering disease transmission on observed social networks have shown that relationships between network structure and parasite risk exist, but often when the parasite measures and network structures are taken from the same population (Loehle, 1995; VanderWaal et al., 2013; Romano et al., 2016), with a notable exception of network modularity showing negative associations with parasite richness in a comparative study of primates (Griffin and Nunn, 2012).

Previous studies have applied similar network-level metrics, like centrality and modularity, to the study of disease transmission through populations (Potterat et al., 1999; Borgatti, 2005; Kasper and Voelkl, 2009; Rushmore et al., 2013; Romano et al., 2016). Nearly all of these measures capture only one aspect of networks, and require this aspect to be considered in isolation from other important information about the network, specifically, its size. This issue is problematic for some metrics like modularity, whose value is mathematically positively associated with network size (Griffin and Nunn, 2012; Nunn et al., 2015). Our measure of effective network size provides a metric for disease transmissibility among individuals in a group that also accounts for the size of the population from which it was

estimated. This differs from the previously mentioned approach by Caillaud et al. (2013), which focused on understanding sub-group heterogeneity of meta-populations in light of epidemic thresholds. Specifically, our approach uses network structure and group size to predict how quickly a disease can be transmitted and maintained by individuals in a population.

Of course, social networks can be represented in many ways, and our approach still simplifies networks considerably from their real-world manifestations. First, nearly all social ties in the real world vary in intensity (weight). However, we conducted most of our methods discussed above on unweighted networks. The unweighted networks were used as a less "noisy" test of our methods. We did, however, also test for associations between effective network sizes and parasite richness using weighted primate networks, which generally did not show improved performance over using raw network size. However, results from these analyses do indicate that effective network sizes estimated from weighted networks were better predictors of parasite richness than unweighted networks. Additional factors complicating this relationship include, first, that social network structure more than likely has much greater intraspecific variation than other traits like body size, and the network structures presented in the literature may not be representative of the primate species which they represent. Additionally, the approach we used for our study, stochastic disease simulation, produces variability of its own, which may further obfuscate a relationship between structure and parasite risk.

Additional sources of variability are also worth considering. For example, individuals vary in traits that make them more or less susceptible to a disease or to transmitting it, including through age-related effects on immune function (Cohen et al., 1997). Networks may also vary in their structure across time, adding yet another variable that complicates analyses (Read et al., 2008; Hamede et al., 2009; Rushmore et al., 2013; Springer et al., 2016). However, the majority

of research focuses on the importance of structural aspects of static networks for predicting and mitigating disease transmission, as this allows for more straightforward interpretation and comparison among different populations (Glass et al., 2006; Andre et al., 2007; Craft, 2015). Our proposed effective network size presents one potential application through the use of our metric in predicting parasite richness in primates.

Although this study has only focused on simulation-based solutions for determining effective network size, mathematical solutions for determining effective network size likely also exist. One such approach for these mathematical solutions was shown by Caillaud et al. (2013), but mathematicians and theoreticians interested in the effects of group size on disease transmission could still significantly further such research. In addition to this, the number of studies that have published primate social network structures is still small. For this reason, we encourage scientists researching social interaction in primates to publish network information on species for which they already have data, and to begin more studies of social network analysis in primate groups.

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Chapter 4

Behavioral and infectious disease comparisons of commensal and non-commensal rodents, with implications for human health

* in prep as: McCabe CM, Agwanda B, Weinstein SB, Young HS, Nunn CL. Human-rodent commensalism and implications for parasite transmission in the modern agricultural transitions of central Kenya.

Abstract

Commensal rodents have probably inhabited human dwellings since at least the advent of agriculture, and since then, they have served as a source of novel parasites and pathogens for humans. Here, we investigate hypotheses for why some individuals and species of rodents are found in human dwellings, whether as novel environments to explore, as sources of abundant stores of grains, or as thermoregulatory buffers. We also test the hypothesis that commensal individuals and species of rodents are more heavily parasitized than non-commensal rodents, posing increased disease risks to humans. To test these hypotheses, we live-trapped rodents throughout Laikipia, Kenya, both inside human dwellings and in adjacent wildlife conservancies. We exhaustively sampled each captured rodent for gastrointestinal parasites and ectoparasites. We also assessed their neophilia and neophobia (measures of exploratory tendencies), diet breadth, and body size. Supporting our predictions, we found that exploratory individuals were more likely to be captured in homes than in wildlife conservancies, although diet breadth and body size did not have any effect on commensality. Commensal rodent individuals had a lower average richness of gastrointestinal parasites, but greater gastrointestinal and ectoparasite infection intensity than those captured in wildlife conservancies. Additionally, more exploratory

individuals had higher infection intensities of gastrointestinal parasites and ectoparasites, and larger-bodied rodents had higher parasite species richness and infection intensities. We suggest that the greater exploratory tendencies of commensal rodents may be driving these animals into the novel habitats of human dwellings, where they are infected by more parasites of fewer species, perhaps owing to the decreased richness but increased density of mammalian hosts present in and around homes. The patterns of ectoparasite infection intensity among commensal rodents may be of particular concern for the health of cohabiting humans, as these ectoparasites may carry blood-borne zoonotic pathogens.

4.1 Introduction

Throughout recent human history, transitions from nomadic hunting and gathering to sedentary agricultural lifestyles have introduced novel costs and benefits. By providing consolidated and storable food resources (Bar-Yosef and Meadow, 1995; Kuijt and Finlayson, 2009), agriculture has resulted in shorter inter-birth intervals and higher carrying capacities per hectare of land (Groube, 1996). However, this lifestyle has also been accompanied by many costs: higher population densities led to increased transmission and maintenance of many new "crowd" diseases (Dunn, 1968; Groube, 1996), and large food stores became monopolized by relatively few individuals, creating social inequalities (Bar-Yosef and Meadow, 1995; Kuijt and Finlayson, 2009). Additionally, permanent homes and granaries attracted other animals as cohabitants, including rodents, insects, and birds (Barrett et al., 1998; Kuijt and Finlayson, 2009).

Animals that live in or around human settlements have been traditionally referred to as commensals. However, the designation of commensality as an ecological class of interspecies relationship, where one species benefits while the other is not affected, may not be particularly fitting in the case of cohabitating small mammals. Most researchers consider these relationships to be more parasitic in nature, i.e. with a benefit to the small mammal and cost to humans (Hulme-Beaman et al., 2016). Because the term "commensal" is the convention within the field, however, we use it to describe animals cohabiting with humans.

Animals may become commensal for many reasons, including to seek shelter from the elements, protection from predators, or increased access to resources (Tchernov, 1984). Rodents are a particularly common commensal taxon today, and evidence suggests that they have been associated with human settlements for more than 10,000 years (Wyncoll and Tangri, 1989).

Rodents, especially house mice (*Mus musculus*) and brown, black, and Polynesian rats (*Rattus norvegicus, R. rattus*, and *R. exulans*, respectively), are considered pest species among modern agriculturalists because they forage on crops in fields or on stored grains and other resources in granaries and homes (Gratz, 1999a). Evidence from the archaeological record suggests that even in the early stages of transitions to sedentary lifestyles, humans identified rodents as pests. This has been inferred from evidence of measures to deter rodents' predation on stored foods, e.g. by installing risers in granaries to keep grains out of reach from rodents (Kuijt and Finlayson, 2009).

In addition to depleting food stores, commensal rodents are major sources of parasitic and infectious diseases for humans. Throughout the world, commensal rodents have been found to host many species of helminths that are zoonotic, meaning that they are carried by other animals, but can also infect humans (Froeschke and Matthee, 2014; Chaisiri et al., 2015; Han et al., 2015). Commensal rodents harbor some infectious diseases with few or no obvious symptoms, such as with typhus, while others manifest with highly lethal symptoms, as is often the case with plague (Cavanaugh et al., 1969; Azad, 1990; Gratz, 1997). These rodent-borne diseases can then be transferred to humans by vectors that include lice, fleas and ticks. Rodents can also spread infectious diseases without the help of vectors, for example, through the aerosolization of their urine or feces, as is the case for Hantavirus and Lassa virus transmission (McCormack et al., 1987; Wong et al., 1989; Fichet-Calvet et al., 2005). Outbreaks of rodent borne diseases have decimated human populations in the past, most notably plague in Europe (Haensch et al., 2010). Understanding disease risks associated with commensal rodents may help to better understand the causes and effects of major disease-related bottlenecks throughout human history (Speicher et al., 2010).

Rodents are remarkably diverse and abundant, with 1591 species identified in the taxonomy of Corbet and Hill (1991), yet few rodent species have become commensal with humans, with *M. musculus*, *R. norvegicus*, *R. rattus*, and *R. exulans* being the only globally distributed commensal species (Gratz, 1999b). Few papers have investigated why some rodent species, or any other animals for that matter, may make the shift to commensality with agriculturalists while others remain in largely undisturbed habitats. The reasons given for the emergence and maintence of commensality typically fall into one of two categories: behavioral or physiological. In addition to predicting commensality, both of these types of traits may have significant consequences for disease risk and parasitism in rodents, and thus also spillover to humans.

Behaviorally, research on the drivers of commensality has focused on the ability of rodents to cope with the stress of living in novel, confined areas with high densities of other rodents. An important mediating phenomenon in this context is exploratory tendency, commonly defined as the balancing effect of neophobia, or fear of novelty, and neophilia, or novelty-attraction (Wright et al., 2010; Liebl and Martin, 2012; Audet et al., 2015; Ducatez et al., 2016). Exploratory tendency refers to the willingness of an animal to approach new areas, objects, or organisms. This tendency varies among and within species, but is generally stable within a given adult individual (Reader, 2003). It can be indexed by assessing frequencies of neophilic versus neophobic reactions to novel stimuli or situations (Mettke-Hofmann et al., 2002; Greenberg, 2003). Increased exploratory tendency is also linked with less risk-aversion (Laviola et al., 2003), which implies that more exploratory rodents will more often live with or around potential threats, such as humans.

The combination of low fear (neophobia) of human cohabitants along with a high willingness to explore new areas or food sources (neophilia) is thus expected to make human homes attractive novel habitats for exploratory individuals and species, as has been found empirically for birds (Audet et al., 2015). In a comparative study, more exploratory species of primates also had a greater diversity of parasite species (2014). Other studies have found that more exploratory species of birds had higher intensities of haematozoa blood parasites (Garamszegi et al., 2007), while more exploratory individuals of a chipmunk species had greater infestations of ticks (Boyer et al., 2010).

We expand on these studies by investigating the exploratory tendencies of multiple rodent species, but by also studying a wide variety of ectoparasites and gastrointestinal parasites, considering both their richness within hosts as well as the intensity of infection among hosts. Moreover, we place this research in a human historical and evolutionary context by linking metrics of exploration to commensality. As such, our study offers the first substantial test for associations between exploratory tendencies and multiple measures of parasite infection across a wide taxonomic range of parasites and hosts.

We also consider other factors that influence commensality. Physiologically, research on understanding the selective advantages of commensality has focused on feeding adaptations and thermoregulation (Wyncoll and Tangri, 1989). Dietary specializations and preferences are important dimensions of niche separation (Heroldová et al., 2008). Rodents exhibit a wide range in extent and nature of dietary specializations: some are generalists (Taylor and Green, 1976; Keesing, 1998; Morand 2000), while others are specialists on cereals (Taylor and Green, 1976), leaves (Downs et al., 2003), or dicot seeds (Shaw et al., 2002). As humans are likely to eat a broad range of foods, rodents showing naturally large diet breadths may be those most

immediately adapted to commensal lifestyles. The breadth of an animal's diet is positively associated with parasitism, with those eating broader diets generally hosting greater diversities of parasites (Dunn, 1968). However, since many agriculturalists store their food in large stockpiles, this could lead to large, dense populations of commensal rodents heavily utilizing such patches. Both of these factors, host density and patch-use intensity, covary positively with parasite risk in primates (Nunn et al., 2003; Nunn and Dokey, 2006) and may also increase parasite risk in commensal rodents in regard to both richness and intensity. The diversity of foods found in human homes might also lead to a greater number of rodent species living together in these homes. Local host richness has been shown to be positively associated with parasite richness in such ecological communities (Poulin, 1997).

Body size is an important determinant of thermoregulation and metabolic rate in mammals that may influence commensality: the smaller-bodied an animal is, the more heat it loses and the higher its metabolic rate is per unit mass (Nagy, 1987). More temperate and constant internal temperatures, relative to those of the external environment, of human settlements may draw commensal animals that are under thermoregulatory stress (Roberts et al., 1974; Yom-Tov, 2003). Smaller rodents are also more difficult to detect and are preyed upon less than larger-bodied rodents, as shown in a study of owl predation on desert rodent species (Kotler et al., 1988); thus, smaller-bodied rodents may be better suited for escaping detection in human homes. A comparative meta-analysis of 62 studies of parasite risk across animal, plant, and fungus hosts confirms that smaller-bodied species also have lower parasite richness than larger-bodied ones (Kamiya et al., 2013).

To shed light onto questions about origins of commensality among rodent species and individuals, we captured rodents in homes and in wildlife conservancies, measured them, and

measured physiological (body size, diet specialization) and behavioral traits (exploratory tendency) that we hypothesized should affect their propensity for commensality. Then, to test whether commensality or any of the traits we proposed above to be related with it impact parasite richness and intensity within species or individuals within species, we surveyed each individual captured for a variety of internal (gastrointestinal) and external parasites. We chose a study site in central Kenya to investigate these questions for three reasons. First, Maasai and Turkana groups of traditionally nomadic, pastoral societies in and around Laikipia have been transitioning to sedentary, agricultural lifestyles for two decades. Second, there is a high diversity of native rodent species, with previous research by Young et al. (2015b) finding 19 unique species, only one of which was an identified invasive species, *R. rattus*. Third, emerging rodent-borne diseases have been identified in this area, including plague, babesiosis, bartenellosis, Lyme disease, and their ectoparasite vectors, to name a few (Jones et al., 2008; Young et al., 2014; 2015a; Campana et al., 2016; Guerra et al., 2016).

We hypothesized that disease risk, behavior, and physiology will each show associations with commensality, and we tested three specific predictions stemming from this hypothesis. First, behaviorally, we predict that less neophobic and more neophilic rodents will be drawn to the novel environments of human homes and will be captured there more often than less exploratory ones. Second, with regard to diet, we predict that rodents with greater diet breadth will be found in commensal habitats more often than will dietary specialists. Third, we predict that smaller-bodied rodents will be found in homes more often than larger-bodied ones, due to their increased heat loss (Bergmann, 1847; Bejan, 2001), resultant need for thermoregulation, and increased ability to evade detection.

As exploratory tendency, diet breadth, and body size have also been associated with infectious disease risk in various taxa, we hypothesized that these traits may facilitate an ecological connection between commensality and parasitism. First, we predict that more exploratory rodent individuals and species will have greater richness and intensities of parasites than less exploratory ones. Second, we also predict that rodent individuals and species with broader diets will have greater richness and intensity of parasites than dietary specialists. Third, we predict that smaller-bodied rodent individuals and species will have lower richness and intensities of parasites than larger-bodied ones. Additionally, we predict that rodents living in habitats with greater numbers of unique host species will have a greater richness of parasites, and those living at higher host densities, such as may be found in homes, will have higher parasite infection intensities. Taking these potential effects together, we predict that commensals will carry a greater number of parasites than non-commensals, both in terms of infection intensity and richness. We test these predictions in separate within and between species analyses.

4.2 Materials and Methods

4.2.1 Field Collection

We established eight independent trapping blocks throughout Laikipia, Kenya, predominantly settled by Maasai and Turkana peoples. At each of the eight blocks, we sampled triads of a recently settled agriculturalist village, an existing pastoral village, and an undisturbed wildlife conservancy habitat. The two villages and conservancy within each triad were always within 10 km of each other, with a mean distance between villages and conservancy within a triad of 3.98 km ± 3.57 (SD) [Figure 4.1].

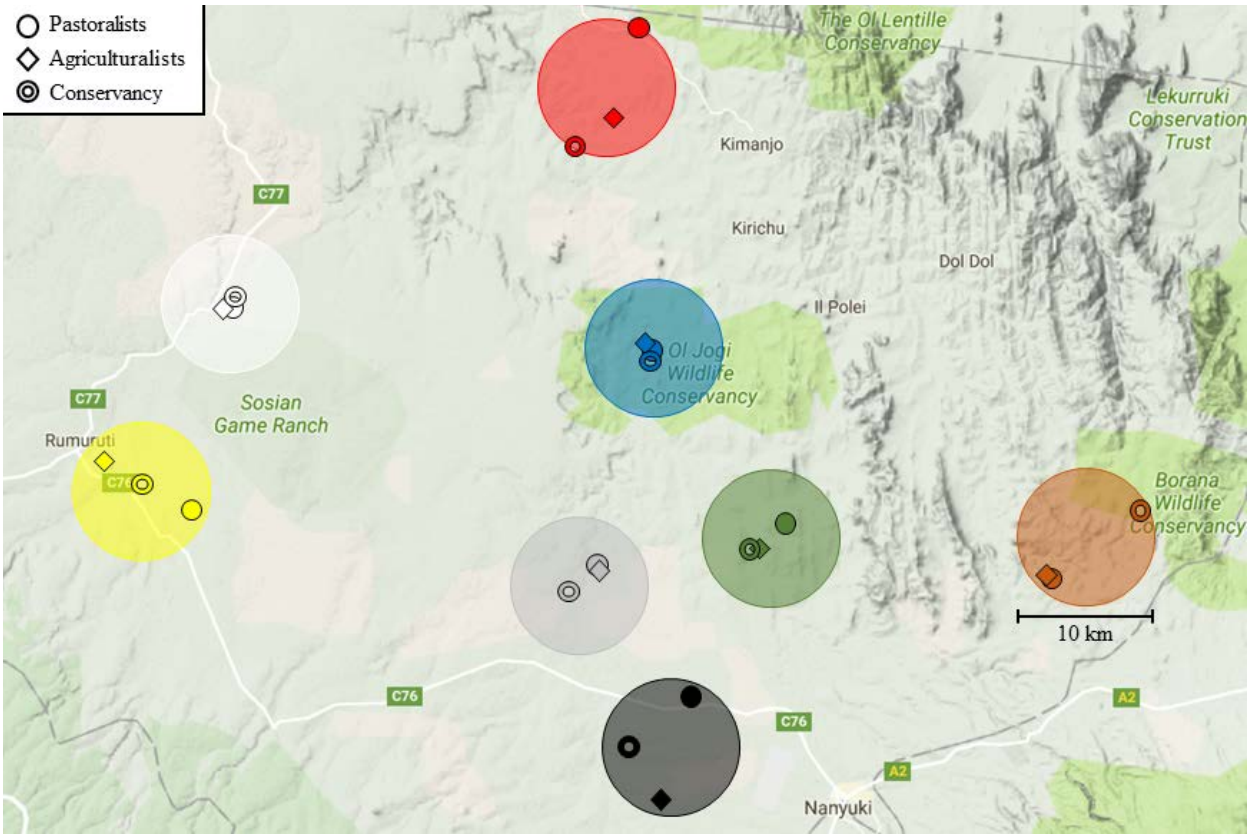

Figure 4.1. Map of the eight trapping locations throughout Laikipia, Kenya, overlaid onto a topographical map from Google Earth (Map data ©2017 Google; 0.562449º N, 36.456455º W to 0.011060º S, 37.332947º W). Circles represent pastoral villages, diamonds agricultural villages, and open circles wildlife conservancies; each village or conservancy within a triad (demarcated by the larger, translucent circles) fell within 10km of the others, with a mean distance between villages and/or conservancies of 3.98km. As shown in the map, triads varied in their proximity to each other, as well as in their proximity to major roads, marked in white with green labels.

In wildlife conservancies, we trapped on 1 ha plots set with Large Folding Aluminum (LFA) Sherman traps every 10 m x 10 m, for a total of 100 traps per square grid. Trapping within each human village occurred only in enclosed, manufactured structures, including homes, granaries, and kitchens. In each village, 20 structures were chosen for sampling. Inside each of these structures, 5 LFA Sherman traps were set haphazardly (i.e., not as part of a 10 x 10 one hectare grid) for a total of 100 traps set in each of the villages. To maintain as much continuity as possible in trapping protocols, traps were set on the ground in homes and in conservancies, preferentially near some sort of cover, like beds and cupboards in homes, and trees and rocks in conservancies. Traps were baited with a mixture of peanut butter and oats. In villages, lengths and widths of homes were also measured and recorded to obtain a total area within human homes that was sampled for each village, for comparison to the total area sampled in each conservancy (1 ha, or 10,000 m^2).

We simultaneously trapped in each of the three sites within a triad for three consecutive days, to allow for acclimation of the rodents to the presence of traps in their environment. Additionally, trapping at each of the eight blocks was replicated every three months to capture seasonal variation in behavior, ecology, and parasite exposure. The number of unique rodent species captured at each location over the course of the study was recorded for each village or conservancy as the local rodent host richness. The total trapping yield (number of animals caught over the course of the study) for each site was calculated and divided by both the total trapping area sampled (within homes for villages, and 1 ha in conservancies) and the number of nights that traps were set at the site to obtain an estimate of host density as the number of rodents captured per m^2 per night.

Every captured rodent was identified to species, sexed, aged (as juvenile or adult), weighed, and measured. Each individual was also combed for ectoparasites, which were counted after exhaustive combing. All captured individuals were then taken into the field lab to quantify exploratory tendency, diet breadth, and parasitic infections. Individuals were placed in short-term housing boxes with sterile cotton nesting materials, water, and food (oats and peanut butter) ad libitum. After 36 hours of housing, rodents were sacrificed through isoflurane inhalation overdose and blood samples were taken by cardiac puncture exsanguination. Animal capture, handling, housing, testing, and euthanasia were approved under Harvard University IACUC protocol 14-02-188.

4.2.2 Behavioral Assessment of Neophilia and Neophobia

Two well-established behavioral tests were used to quantify exploratory tendencies in rodent subjects (Kliethermes and Crabbe, 2006). The first was the open-field test [Figure 4.2a], in which the subject was placed in a relatively large, walled enclosure and allowed to explore this space for 5 minutes. This test is meant to give some sense of how fearful (neophobic) the rodent is, when placed in a novel, open environment (Archer, 1973; Walsh and Cummins, 1976). The open-field arena was illuminated with red-filtered light to mimic low light levels while still allowing observation (McLennan and Taylor-Jeffs, 2004). Activity was videotaped for later analysis in Noldus EthoVision XT 11.5. Table 4.1 summarizes the behaviors recorded in this test and their relevance to quantifying neophobia (no behaviors measured in this test directly assessed neophilia).

The second behavioral test was the modified hole-board test [Figure 4.2b], in which the subject was released into a walled arena with four shallow holes positioned equidistantly

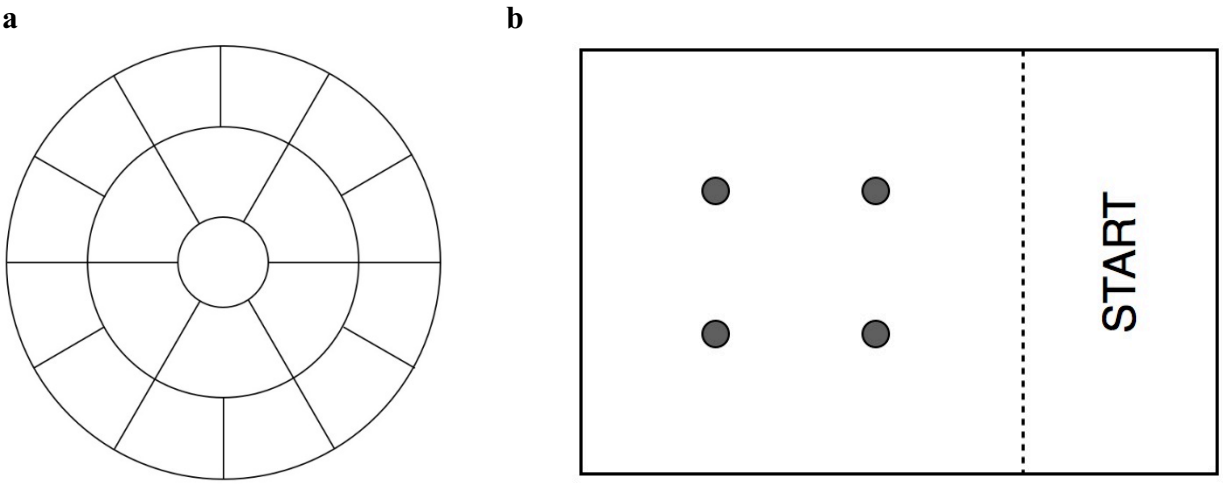

Figure 4.2. Diagrams of the open-field and hole-board arenas. (a) Open-field: subjects were placed in a random outer segment facing the outer wall of the arena; dimensions: diameter of full arena = 75 cm, area of arena = 1.77 m2, diameter of interior (intermediate of three concentric circles) = 50 cm, diameter of center (smallest of three concentric circles) = 25 cm. (b) Hole-board: START box represents the holding cell for rodent before exposure to experiment; dashed line represents plexiglass barrier between holding cell and experimental arena; dimensions: holes were 2.5 cm in diameter, 2 cm deep, and spaced 10 cm from other holes and 15 cm from the border walls.

throughout on the floor of the arena. The total number of head dips into holes and total duration of head dips are indicative of an animal's tendency to explore the novel environment, thus capturing neophilia (File and Wardill, 1975; Martin and Réale, 2008). Lighting and duration of this test were as in the hole-board test, and the trials were also videotaped for analysis in Noldus EthoVision XT 11.5. After each test, the testing arenas were thoroughly washed with 1:20 ethanol to water solution to ensure removal of scents from previous subjects (Deacon, 2006). Again, table 4.1 summarizes the behaviors recorded in this test and relevance to neophilia (marked with asterisks in the table) or neophobia.

After full analysis of the videos, two separate principal components analyses (PCA) of the two neophilic and eight neophobic behaviors, respectively, were used to obtain measures (principal components) of these behavioral tendencies for each individual sampled. Because neophilia and neophobia are two distinct, but closely related behavioral phenomena (Greenberg and Mettke-Hofmann, 2001), we chose to analyze them in separate PCAs so that the signal of one would not be lost among the variation of the other. The number of principal components that capture the majority of variation in each PCA (cumulatively, greater than 50%) will be included in the linear models that follow. Previous studies on exploratory tendencies in small mammals have favored hole-board head dips (both frequency and cumulative duration) as the most direct measure of exploration of the environment through neophilia (Boyer et al., 2010); thus, head dipping frequency and cumulative duration were included in the first PCA, measuring neophilia. Less direct proxies of exploratory tendencies include those that measure the presence or absence of fear in response to novel stimuli (Kliethermes and Crabbe, 2006); such behaviors indicate the degree of neophobia in an individual. These behaviors included immobility (freezing), thigmotaxis (moving close to walls), activity level (velocity), and movement (in the open field

Table 4.1. List of types of neophobic and neophilic behaviors recorded in the two types of behavioral tests (open-field and hole-board) using head-body-tail tracking in Noldus EthoVision XT 11.5 software. Velocity was calculated between each frame as the two-dimensional movement of the animal's body-point. Freezing was considered to be moving at less than 20% of an animal's recorded maximum velocity. Thigmotaxis, or movement in close proximity to the walls of the arena, was recorded when an animal's body point was detected in the outer 25% of the arena. The interior of the open-field test was considered to be the center 50% of the arena. Dips for hole-board tests were recorded when an animal's head-point was detected within 1cm of one of the four holes. The two behaviors marked with an asterisk are those that were considered in the neophilia PCA, and the remaining eight were included instead in the neophobia PCA.

	Open-field	**Hole-board**
Neophobic Behavior	Freezing	Freezing
	Thigmotaxis	Thigmotaxis
Neophilic Behavior	Duration in Interior	* Number of Dips *
	# Lines Crossed	* Duration of Dips *
	Activity (Velocity)	Activity (Velocity)

test, these were indicated by time spent in the center of the arena and number of boundaries crossed). These behaviors were included in a separate PCA measuring the degree of neophobia showed by each individual.

4.2.3 Assessment of Diet Breadth

To assess the extent of dietary specialization and breadth at the species level we used diet composition data compiled from literature. From the literature, we find that central Kenya is home to various dietary specializations among rodents, including species focusing on cereals, leaves, and seed, respectively (Taylor and Green, 1976; Shaw et al., 2002; Downs et al., 2003), and generalists that consume all of these food types, along with invertebrates and fruit (Taylor and Green, 1976; Keesing, 1998). However, classifications of diet specialization are not available for all rodent species included in our study (Happold, 2013). For this reason, we used the MammalDIET database for diet classifications (Kissling et al., 2014; WD et al., 2014). This database included both literature classifications of diet for mammal species and phylogenetically controlled extrapolations for species on which no data was present. The database provided presence-absence data for the following types of food in diets: grains/seeds, leaves, fruit, insects, and meat (but no rodents in our dataset were characterized as having meat present in their diets). From these presence-absence categories, we calculated diet breadth measurement for each species in our study by summing the food types present in each species' diet [Table 4.2].

4.2.4 Sample Collection and Laboratory Analyses of Parasites

To permit parasitological examination, captured rodents were euthanized with halothane and frozen. Each sampled animal was measured, sexed, and dissected. During dissection, we

Table 4.2. Distribution of rodents captured during the study, grouped by species in alphabetical order of scientific name. Common names, as well as taxonomic family and subfamily are listed as provided in Young et al. (2015b). Diet breadth and trophic classification were taken from the MammalDIET database (Kissling et al., 2014; WD et al., 2014). Sample size refers to the number of individuals captured of each species across all habitats.

Species	Common Name	Family	Subfamily	Diet Breadth	Trophic Class	Sample Size
Acomys kempi	Kemp's spiny mouse	Muridae	Deomyinae	1	Carnivore	19
Acomys percivali	Percival's spiny mouse	Muridae	Deomyinae	1	Carnivore	4
Aethomys hindei	Hinde's rock rat	Muridae	Murinae	1	Omnivore	22
Arvicanthis nairobae	Nairobi grass rat	Muridae	Murinae	2	Herbivore	120
Dendromus melanotis	Gray climbing mouse	Nesomyidae	Dendromurinae	3	Omnivore	6
Gerbilliscus robustus	Fringe-tailed gerbil	Muridae	Gerbillinae	3	Omnivore	34
Grammomys dolichurus	Woodland thicket rat	Muridae	Murinae	1	Omnivore	5
Mastomys natalensis	Natal multi-mammate mouse	Muridae	Murinae	3	Omnivore	1
*Mus sp.**	* cryptic mouse species	Muridae	Murinae	2	Omnivore	19
Rattus rattus	Black rat	Muridae	Murinae	2	Omnivore	16
Saccostomus mearnsi	East African pouched mouse	Nesomyidae	Cricetomyinae	1	Omnivore	18
Tatterillus harringtoni	Harrington's gerbil	Muridae	Gerbillinae	2	Omnivore	4
Zelotomys hildegardae	Hildegarde's broad-headed mouse	Muridae	Murinae	1	Carnivore	1

removed the gastrointestinal tract and separated the stomach, small intestine, cecum and large intestine into separate dishes. To obtain exhaustive parasite samples, gut sections were then opened lengthwise and the contents were scraped and preserved in ethanol (final concentration ~80%). Worms were removed from gut contents under a dissecting scope and identified to morphospecies and broad taxonomic categories (nematode, cestode, acanthocephalan, trematode, pentastome). Adult and juvenile parasites were visually identified to species by taxonomic specialists, and will later be genetically identified to species through DNA barcoding. Ectoparasites were exhaustively combed off of each rodent and into a shallow collecting basin of ethanol immediately after the rodent's capture. These ectoparasites were removed from the collecting basin, counted in aggregate, and preserved in vials for later identification.

4.2.5 Statistical Analyses

For between-species comparisons of parasite risk, we employed phylogenetic generalized least squares (PGLS) analyses in R 3.3.3 (R Core Team, 2016) with packages ape (Paradis et al., 2004) and caper (Orme et al., 2013), estimating λ using maximum likelihood. The phylogenetic tree used for our analyses [Figure 4.3] was pruned from the mammalian supertree of Bininda Emonds et al. (2008), and polytomies were randomly resolved for each PGLS test. Because species are the unit of replication for analyses such as PGLS, our sample size here was only 13 species. Due to this low sample size of our phylogenetic dataset, we were constrained by power issues to only test one parameter per model (Mundry, 2014). Thus, for each measure of parasite risk we show results of four tests, one each for commensality, exploratory tendency (neophilia PC1, as the most direct measure of novelty-attraction), diet breadth, and body size. Parasite richness, intensity, and body size measures were log10-transformed and z-scaled to meet the

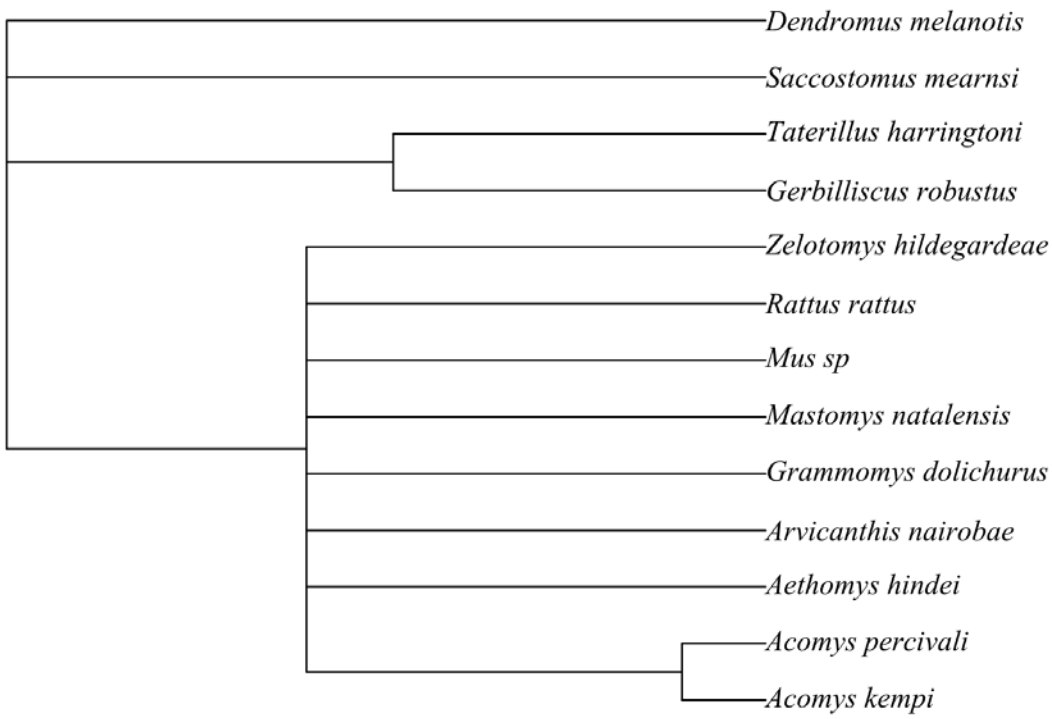

Figure 4.3. Phylogeny of rodent species captured in our study, pruned from the corrected mammal supertree of Bininda-Emonds et al. (2008). Low resolution of tree was corrected for in PGLS analyses by randomly resolving polytomies in R.

assumptions of PGLS. Also, because each of the species included in our analysis was represented by multiple individuals, but PGLS in caper cannot incorporate variation in predictor variables, we were forced to only choose one individual per species for each PGLS model. To incorporate the variation in predictor variables, we randomly sampled a representative individual for each species 1000 times, with each individual within a species equally likely to be picked, and present the distribution of slope estimates from this resampling (Sandel et al., 2016). Assessment criteria were set as in McCabe et al. (2014), with cut-offs at 85% slope estimates in the predicted direction indicating "weak support," 90% for "support," and 95% for "strong support."

For tests of within-species variation, we employed generalized linear models to account for the non-Gaussian distributions in errors of our response variables represented by count or presence-absence data. Habitat type (a three factor measure of commensality, further splitting commensal habitats into agricultural homes and pastoral homes), neophilia, neophobia, diet breadth, and body mass in grams were included in all models as fixed effects. In addition to this, we used mixed models with sampling period nested within settlement ID, and species ID as random effects.

All generalized linear mixed-effects models (GLMM) were analyzed with the lme4 package (Bates et al., 2015) in R, with a standard $\alpha=0.05$ indicating significance. Among the GLMMs, parasite counts (richness and intensity) were assumed to have Poisson distributions, and commensality was coded as a binary variable (1 for commensal including both agricultural and pastoral, 0 for non-commensal), and thus was assumed to have a binomial distribution. All

estimates of goodness-of-fit for GLMMs are reported as conditional R^2 values, taking into account both the fixed and random effects.

Additionally, in an attempt to assess causality, four models were compared by their AICc values, computed in R package MuMIn (Barton, 2016): one where commensality was predicted by exploratory tendency, diet breadth, and body size; one where exploratory tendency was predicted by commensality, diet breadth, and body size; one where diet breadth was predicted by commensality, exploratory tendency, and body size; and one where body size was predicted by commensality, exploratory tendency, and diet breadth. The interpretations of the models are as follows: if a predictor variable is significantly associated with commensality, then it is influencing commensality; if a predictor variable, including commensality, is significantly associated with exploratory tendency, then it is influencing exploratory tendency; if a predictor variable, including commensality, is significantly associated with diet breadth, then it is influencing diet breadth; and if a predictor variable, including commensality, is significantly associated with body size, then it is influencing body size. Only the model with the lowest AICc value will be interpreted in the conclusions, provided that its AICc score is at least 2 less than the next nearest model (a standard cut-off for this method of model selection).

4.3 Results

4.3.1 Behavioral and Physiological Predictors of Commensality

Among the 247 individuals considered in our analyses, we identified 13 rodent species distributed across agricultural homes, pastoral homes, and wildlife conservancies [Figure 4.4]. *Mus* species were cryptic and difficult to identify without genetic barcoding methods, similar to findings of Young et al. (2015b), and so they were lumped into a single cryptic species

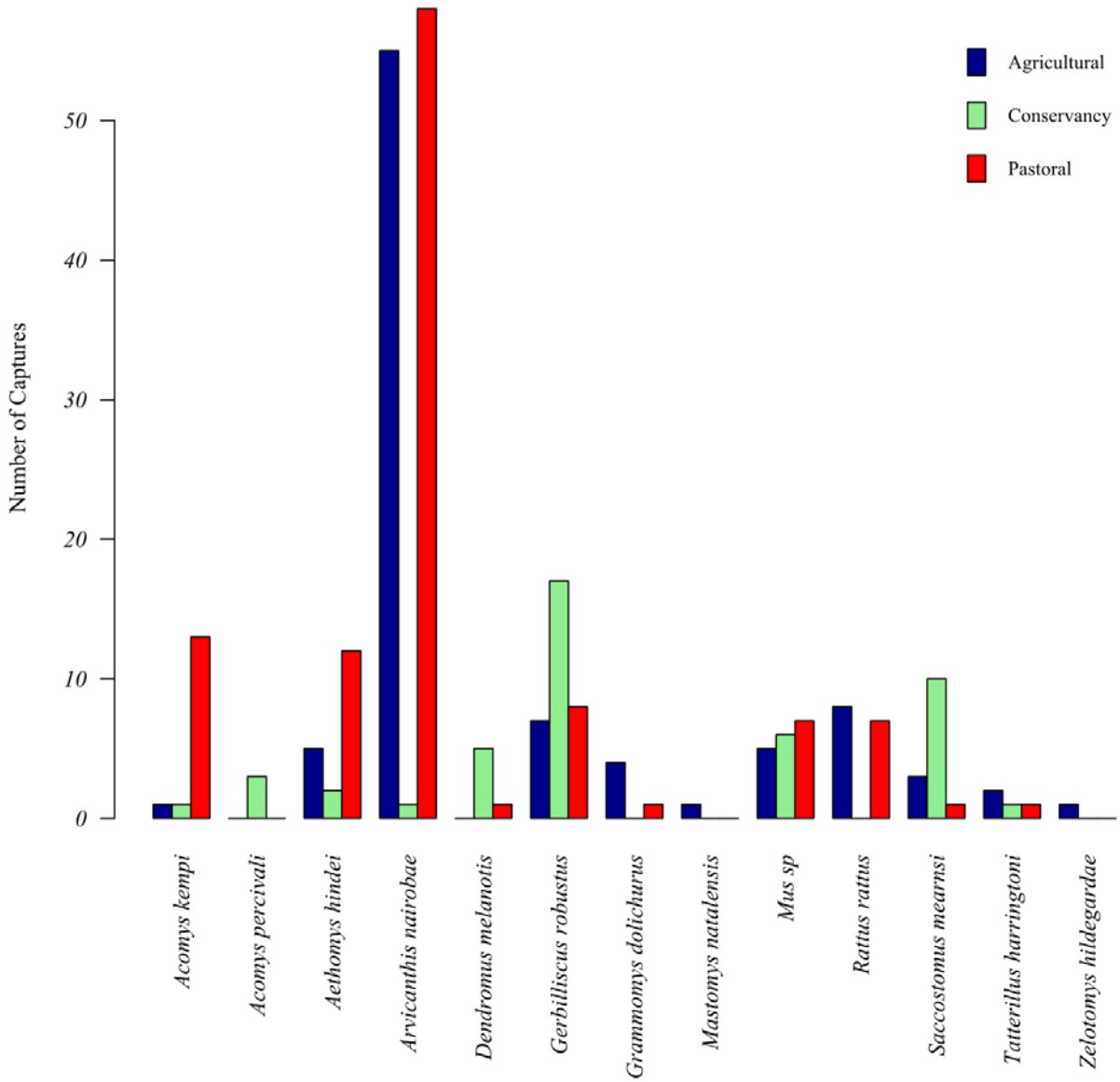

Figure 4.4. Distributions of rodent captures by species across different habitats. Blue bars represent the numbers of each species captured in agricultural homes, green bars in wildlife conservancies, and red bars in pastoral homes.

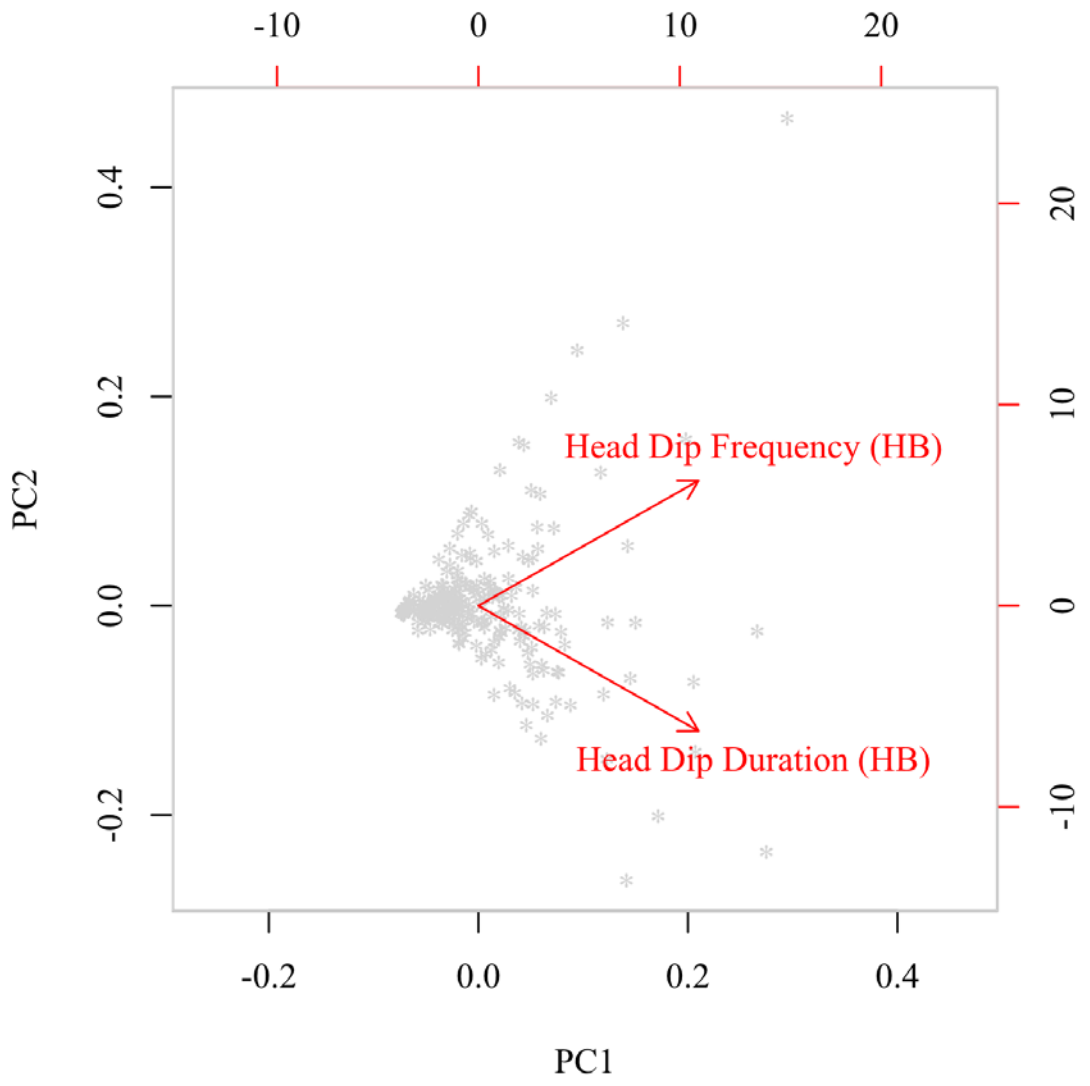

Figure 4.5. Biplot of the first two principal components from analysis of neophilia. Points in grey represent individual rodents, and red arrows indicate loadings of specific behaviors on the two principal components. Parenthetical "HB" indicates that both behaviors were recorded during hole-board tests.

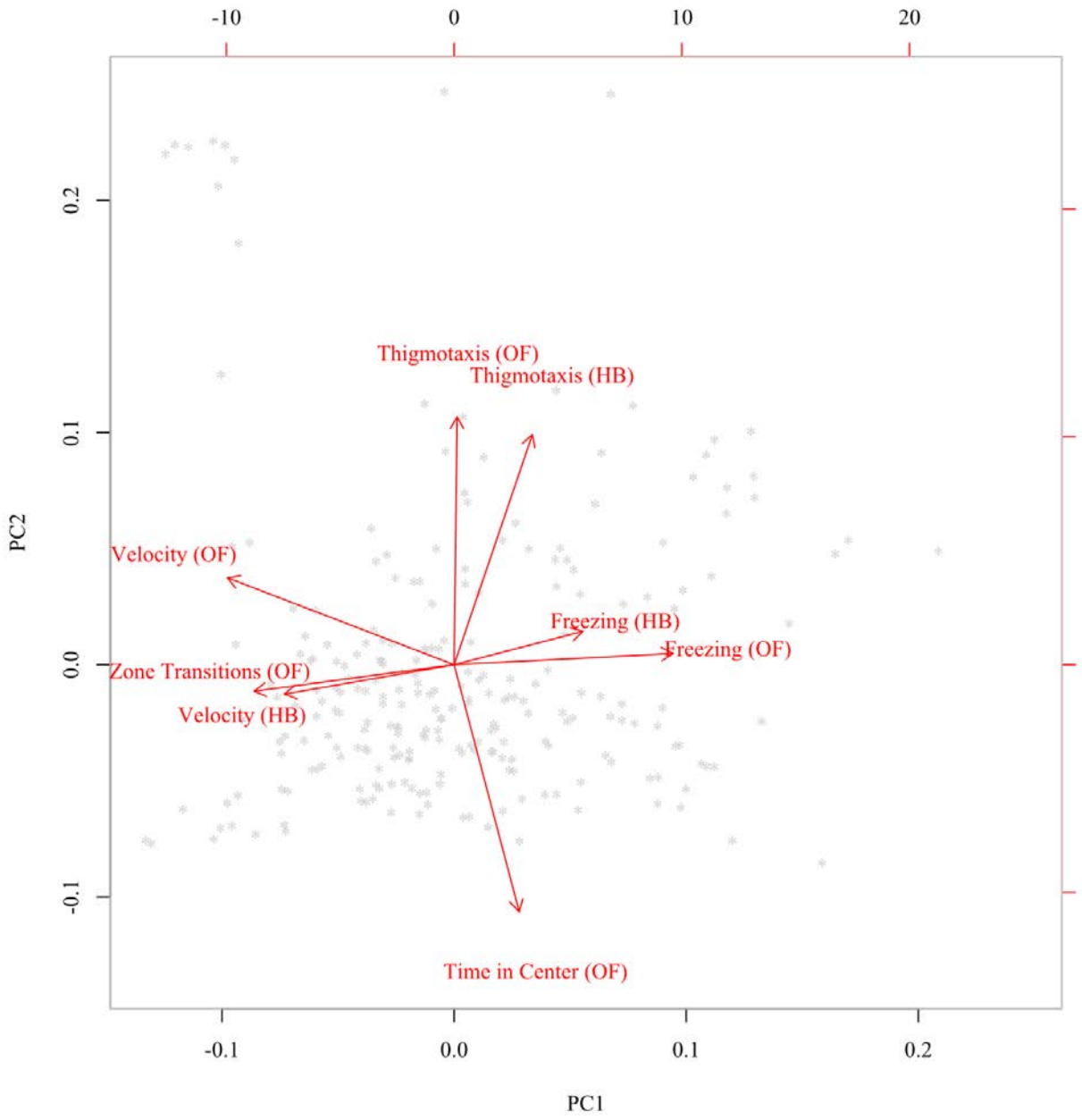

Figure 4.6. Biplot of the first two principal components from analysis of neophobia. Points in grey represent individual rodents, and red arrows indicate loadings of specific behaviors on the two principal components. Parenthetical "HB" indicates behaviors that were recorded during hole-board tests, and "OF" indicates behaviors that were recorded during open field tests.

aggregate. With our binomial-distributed response variable of commensality, we tested for relationships with exploratory tendency (as measured here by neophilia, as the most direct estimate of novelty-attraction), diet, and body size.

PCA results yielded the predicted associations of the behavioral proxies with their related behavioral tendencies. For the PCA representing neophilia, both the frequency and duration of head dips on the hole-board experiment were positively loaded on PC1 [Figure 4.5]. Thus, higher scores on PC1 of this analysis were indicative of more neopilic tendencies. In this analysis, PC1 accounted for 75.51% of the variance in neophilic behaviors observed, and PC1 and PC2 cumulatively accounted for 100% of the variance (as there were only 2 behaviors considered); thus, only PC1 was included in the models. For the PCA representing neophobia, zone transitions (number of lines crossed in the open field test) and velocity (in both tests) were loaded negatively on PC1, while freezing behaviors in both tests were loaded positively on PC1 [Figure 4.6]. Thus, higher scores on PC1 indicate less mobile, more neophobic individuals. On PC2, thigmotaxis (movement in close proximity to walls of the arenas) was loaded positively, and time in the center of the open field arena was loaded negatively, capturing another axis of neophobia that may be particularly important to behaviour inside human homes, which all had walls. Thus, higher scores on PC2 are also indicative of more neophobic tendencies. In this analysis, PC1 accounted for 29.76% of the variance in neophobic behaviors observed, and PC1 and PC2 cumulatively accounted for 58.05% of the variance; thus, both PC1 and PC2 were included in the models.

In intraspecific (individual-level) models predicting the relationships between commensality and the three variables that were predicted to be associated with it, the only marginally significant relationship was between exploratory tendency and commensality.

Table 4.3. Results of a mixed-effects model investigating associations between an individual's commensality (whether or not it was captured within a home) and predicted correlates of commensality, namely exploratory tendency (neophilia), diet breadth, and body size from 247 individuals. Models also included additional random effects (sex, age, season, species ID, and site ID). Numbers in parentheses indicate standard errors, and those above are coefficients, with asterisks denoting significance (* $p < 0.1$; ** $p < 0.05$; *** $p < 0.01$). For model selection purposes, AICc values are also included at the bottom of the table for each model.

Predictors of Commensality

	Dependent variable:			
	Commensality (A)	Neophilia PC1 (B)	Diet breadth (C)	Body mass (g) (D)
Intercept	2.198	0.178	0.526***	-0.316
	(1.990)	(0.525)	(0.166)	(0.643)
Neophilia PC1	0.467*		-0.009	-0.057
	(0.263)		(0.041)	(0.035)
Commensality		0.471**	-0.024	0.121
		(0.236)	(0.144)	(0.141)
Diet breadth	-0.299	-0.231		-0.037
	(1.004)	(0.223)		(0.327)
Body mass (g)	0.359	-0.147	-0.007	
	(0.365)	(0.101)	(0.065)	
Akaike Inf. Crit.	165.570	808.717	666.995	533.168
Marginal R-squared	0.044	0.043	0.001	0.006
Conditional R-squared	0.676	0.331	0.210	0.667

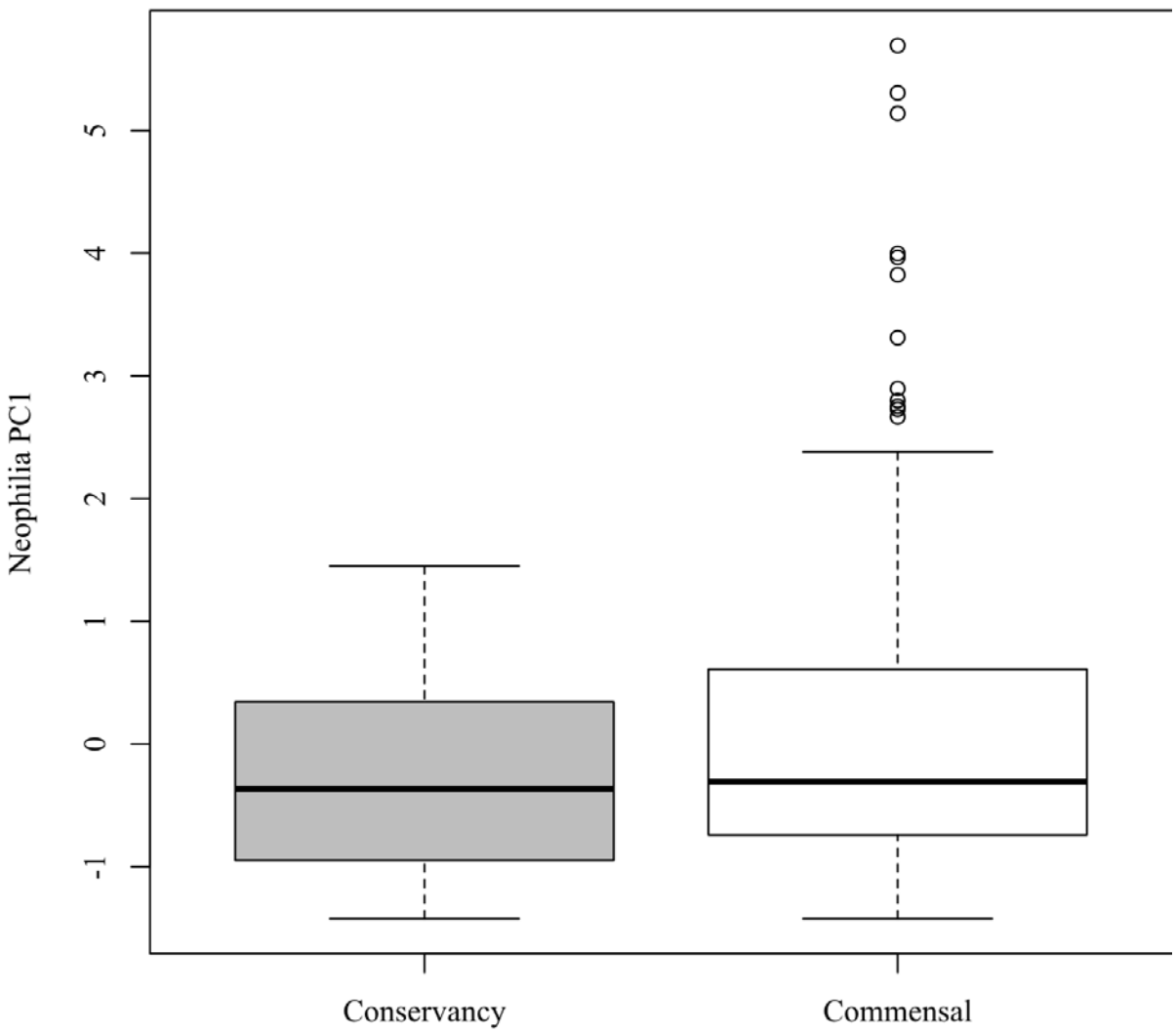

Figure 4.7. Marginally significant effect of neophilia PC1 on the commensality of captured rodents. Effect reported in text also accounts for the body size, diet breadth, village and season captured, and species, and assumes a Binomial distribution of the dependent variable (commensality). Commensal rodents had higher neophilia PC1 scores than did conservancy-captured rodents.

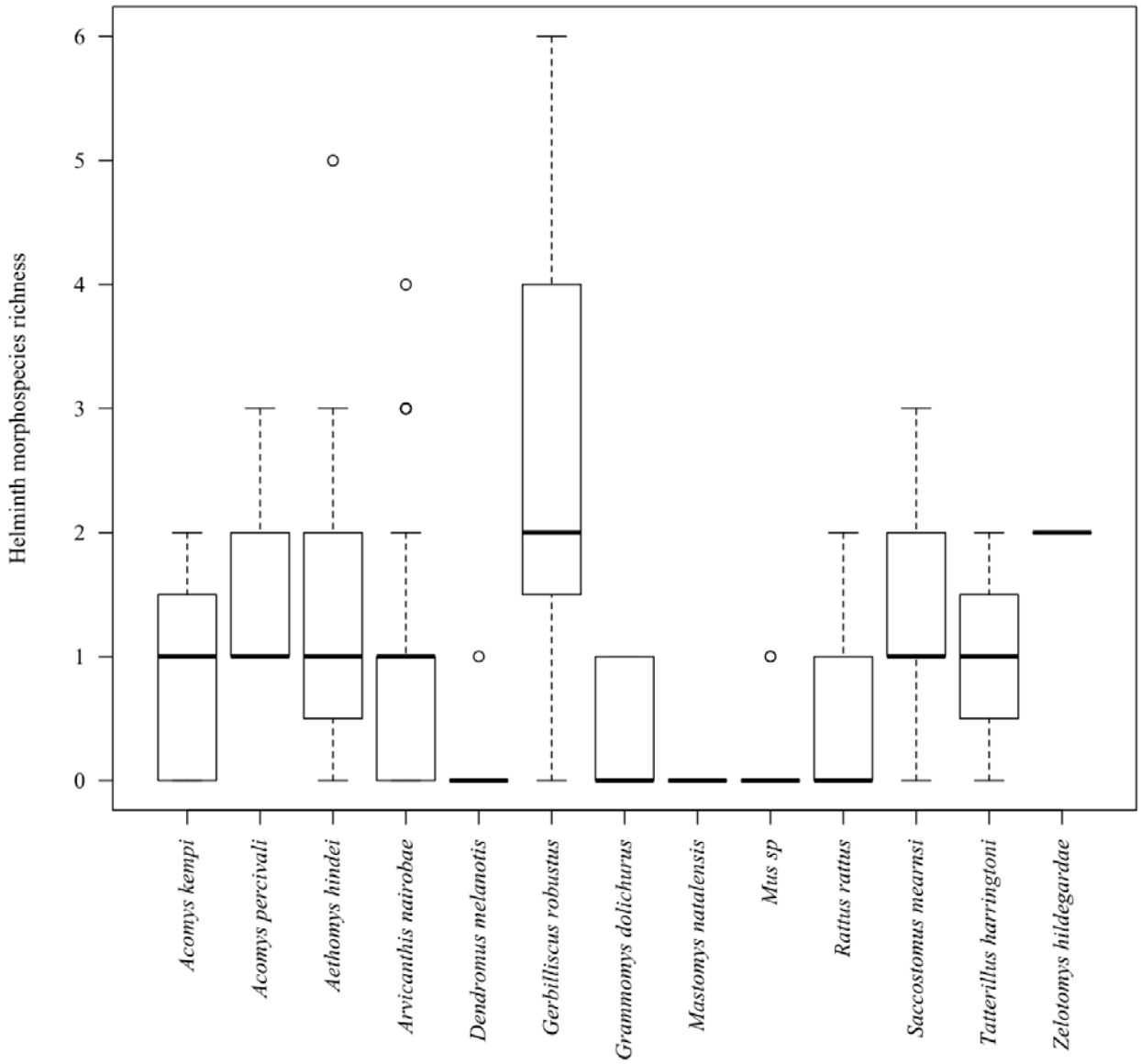

Figure 4.8. Distributions of the numbers of helminth morphospecies identified from each individual of given host species. Across the range of variation between species. *Gerbilliscus robustus* had the highest average richness of gastrointestinal parasites, while a cryptic species aggregate of genus *Mus* showed a median richness of zero parasite morphospecies.

Neophilia PC1 was positively associated with the likelihood of being captured in a home versus being captured in a wildlife conservancy (β=0.467, SE=0.263, p=0.076; Table 4.3a; Figure 4.7), and commensality was positively associated with neophilia PC1 (β=0.471, SE=0.236, p=0.046; Table 4.3b). In these two models, no other variables were significantly associated with the response variables. AICc comparisons between models showed that by far the most informative model for associations among the four variables of interest was the one where neophilia PC1 predicted commensality, suggesting that exploratory tendencies are influencing commensality among individuals. Of particular importance in Table 4.3 are the very low marginal R^2 values relative to the conditional R^2 values; this indicates that the random effects, most likely species ID, are important to obtaining good fit in the models.

4.3.2 Commensality and Parasite Richness

We identified 28 distinct morphospecies of gastrointestinal parasites from our samples: 1 acanthocephalan, 11 cestodes, 14 nematodes, 1 pentastome, and 1 trematode. The distribution of gastrointestinal parasite species richness across species is shown in Figure 4.8. The average parasite richness among these individuals was 1.12 species per host ± 1.26 (SD), average infection intensity was 21.79 adult worms per host ± 73.68 (SD), and average exhaustive ectoparasite count was 8.65 ± 19.87 (SD).

Between-species models for gastrointestinal parasite richness show body mass to be the only supported predictor, with “strong support” at 95.5% of models showing slopes in the predicted direction [Figure 4.9b]. This result indicated that larger-bodied rodent species had greater parasite richness than smaller-bodied species. All other predictors were not supported.

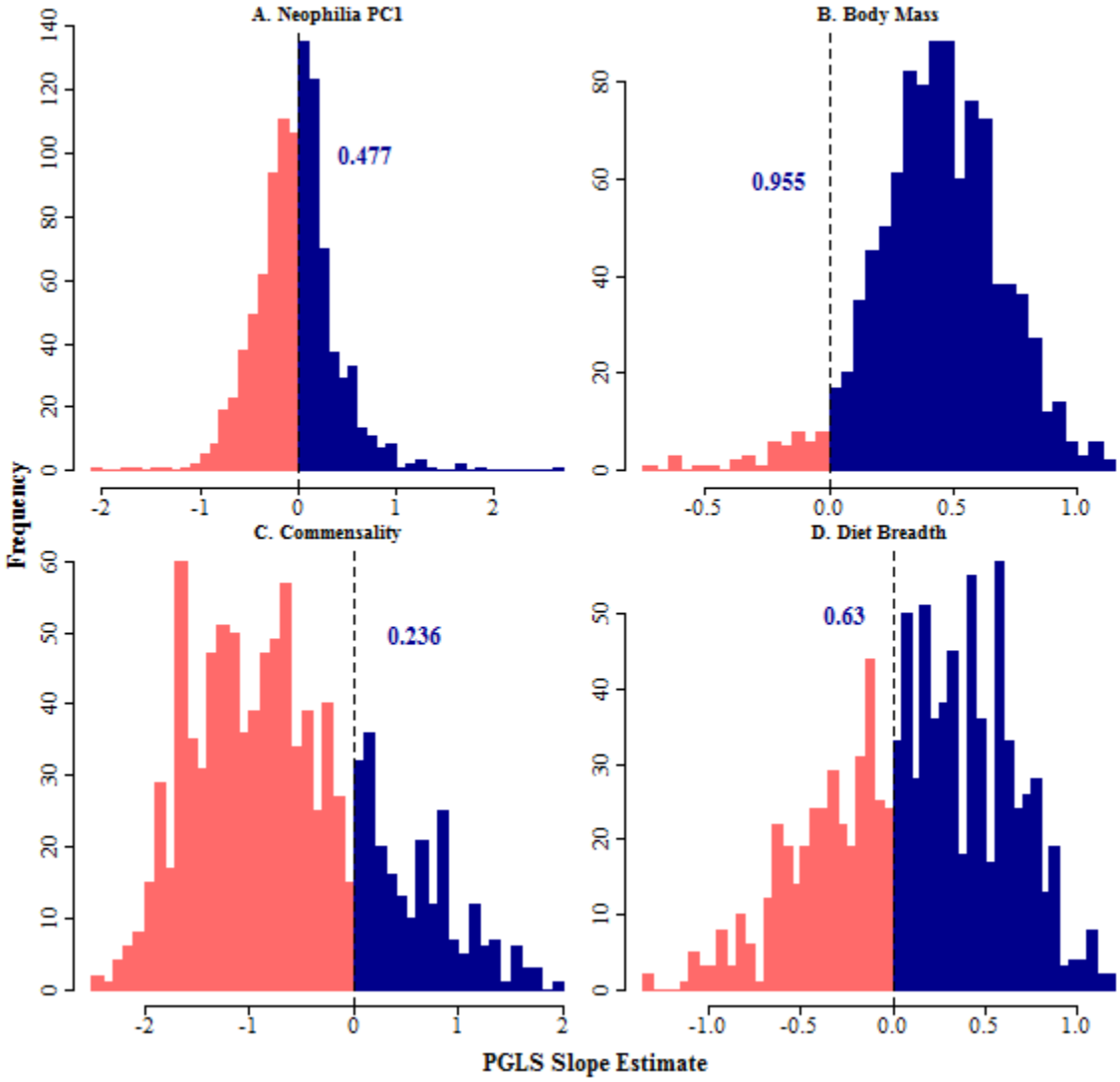

Figure 4.9. Distributions of slope coefficients from 1,000 resamplings of PGLS models for various predictor variables associated with total gastrointestinal morphospecies richness. Panels indicate which predictor variable was tested: A. neophilia PC1, B. body mass (in grams), C. commensality, and D. diet breadth. Portions of each distribution in blue indicate positive associations, and red portions indicate negative associations. Numbers in blue indicate the support level for each association.

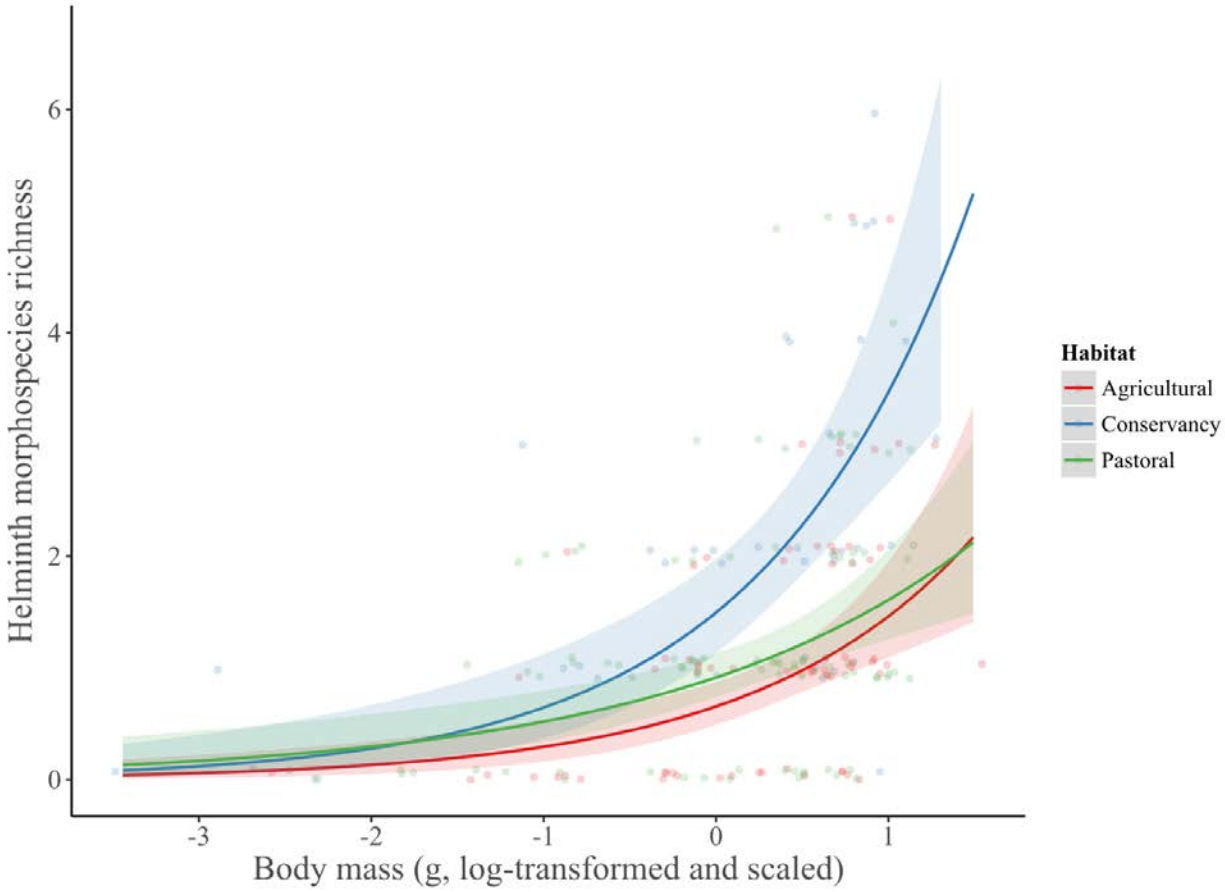

Figure 4.10. Marginal effects of habitat type (agricultural, pastoral, or wildlife conservancy) and body mass (in grams, log-transformed and scaled) on total gastrointestinal parasite morphospecies richness among 247 captured rodent individuals. Effects reported also account for diet breadth, village and season of capture, species, and assume a Poisson distribution of richness. Richness increases with body size and is greater in wildlife conservancy-captured animals than those captured in agricultural or pastoral homes.

Table 4.4. Results of a mixed-effects models investigating associations between an individual's gastrointestinal parasite infection richness and habitat type, diet breadth, neophilia, neophobia, body size, and local host richness from 247 individuals. Models also included additional random effects (trapping season nested within site ID, and species ID). Numbers in parentheses indicate standard errors, and those above are coefficients, with asterisks denoting significance (* $p < 0.1$; ** $p < 0.05$; *** $p < 0.01$).

Parasite Species Richness

	Dependent variable:		
	Total Helminth Richness	Cestode Richness	Nematode Richness
	(A)	(B)	(C)
Intercept	-0.067	-3.376***	-0.167
	(0.513)	(0.813)	(0.558)
Conservancy habitat	0.389*	-0.461	0.551**
	(0.207)	(0.492)	(0.236)
Pastoral habitat	0.164	0.123	0.173
	(0.154)	(0.347)	(0.173)
Diet breadth	-0.201	0.535*	-0.292
	(0.251)	(0.298)	(0.281)
Neophilia PC1	0.018	-0.129	0.057
	(0.062)	(0.163)	(0.067)
Neophobia PC1	-0.028	-0.091	-0.028
	(0.050)	(0.120)	(0.056)
Neophobia PC2	0.035	0.101	0.040
	(0.060)	(0.124)	(0.071)
Body mass (g)	0.728***	0.977***	0.709***
	(0.124)	(0.306)	(0.139)
Local host richness	0.029	0.061	0.029
	(0.042)	(0.103)	(0.043)
Marginal R-squared	0.335	0.314	0.297
Conditional R-squared	0.479	0.446	0.433

Concerning gastrointestinal parasite richness patterns within species, habitat and body size were the only significant predictors of morphospecies richness [Figure 4.10], with one exception of diet breadth associated with cestode richness. Rodent individuals from wildlife conservancies had marginally greater total gastrointestinal parasite richness than commensals (β=0.389, SE=0.207, p=0.061; Figure 4.10; Table 4.4a). Individual body size also correlated positively with total gastrointestinal parasite richness (β =0.728, SE=0.124, p<0.001; Figure 4.10; Table 4.4a). All other relationships were not statistically significant. The model fit the data with R^2=0.479. This observed pattern appears to be driven by the effects of body size and commensality on nematode richness specifically. Here, rodent individuals from wildlife conservancies had significantly greater nematode richness than commensals (β=0.551, SE=0.236, p=0.019; Table 4.4c). Again, individual body size also correlated positively with total gastrointestinal parasite richness (β =0.709, SE=0.139, p<0.001; Table 4.4c). The model for nematode richness fit the data with R^2=0.433. Cestodes showed a differing pattern, with individuals with broader diets, perhaps encompassing cestodes' intermediate hosts, having marginally higher richness (β =0.535, SE=0.298, p=0.073; Table 4.4b). The model for cestode richness fit the data with R^2=0.446.

4.3.3 Commensality and Parasite Intensity

Between-species models for gastrointestinal parasite infection intensity again showed body mass to be the only supported predictor, with "strong support" at 96.5% of models showing slopes in the predicted direction [Figure 4.11b]. This result indicated that larger-bodied rodent species had greater gastrointestinal parasite infection intensity than smaller-bodied species. All other predictors were not supported. PGLS models for ectoparasite infestation intensity also

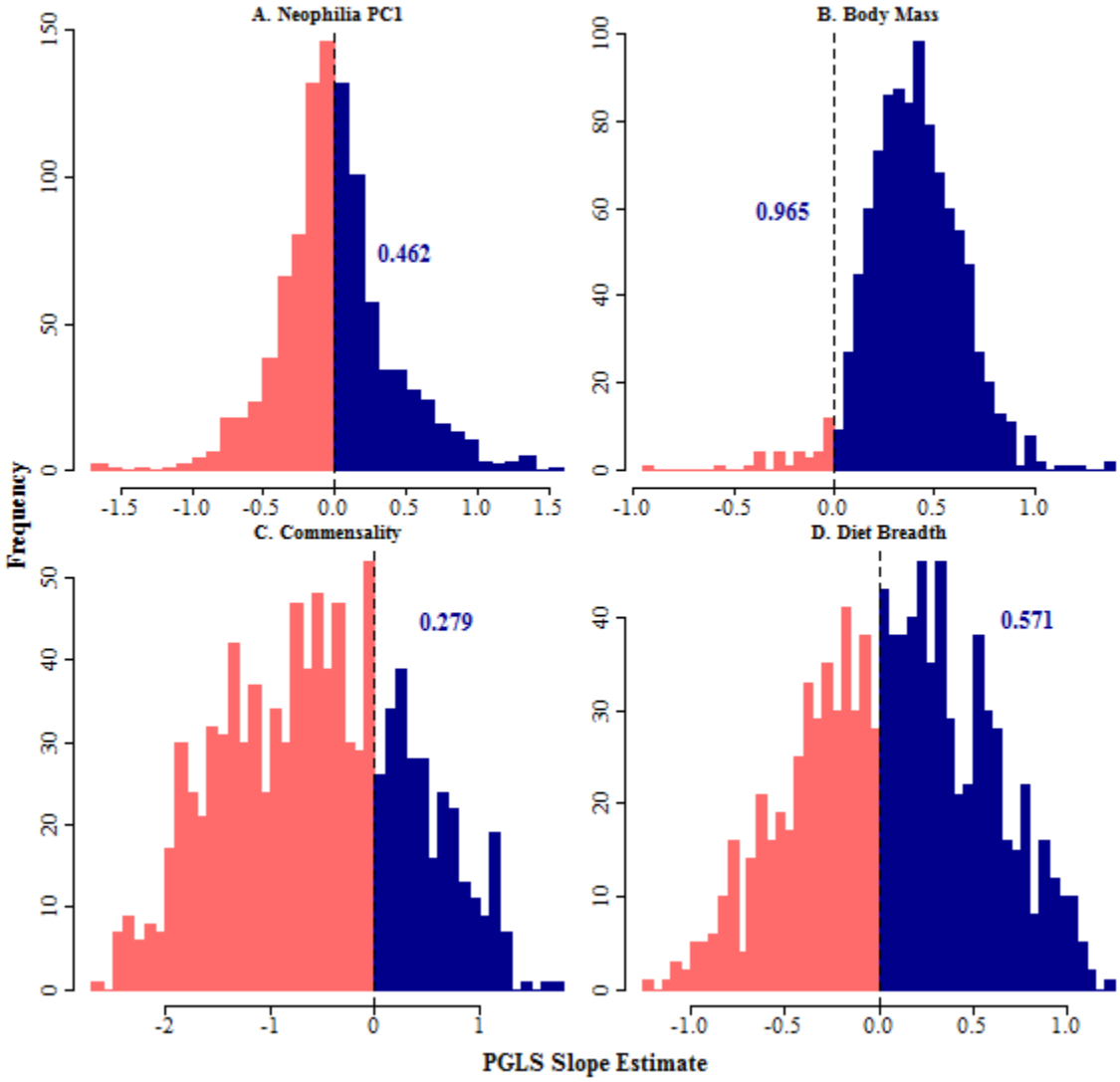

Figure 4.11. Distributions of slope coefficients from 1,000 resamplings of PGLS models for various predictor variables associated with gastrointestinal infection intensity. Panels indicate which predictor variable was tested: A. neophilia PC1, B. body mass (in grams), C. commensality, and D. diet breadth. Portions of each distribution in blue indicate positive associations, and red portions indicate negative associations. Numbers in blue indicate the support level for each association.

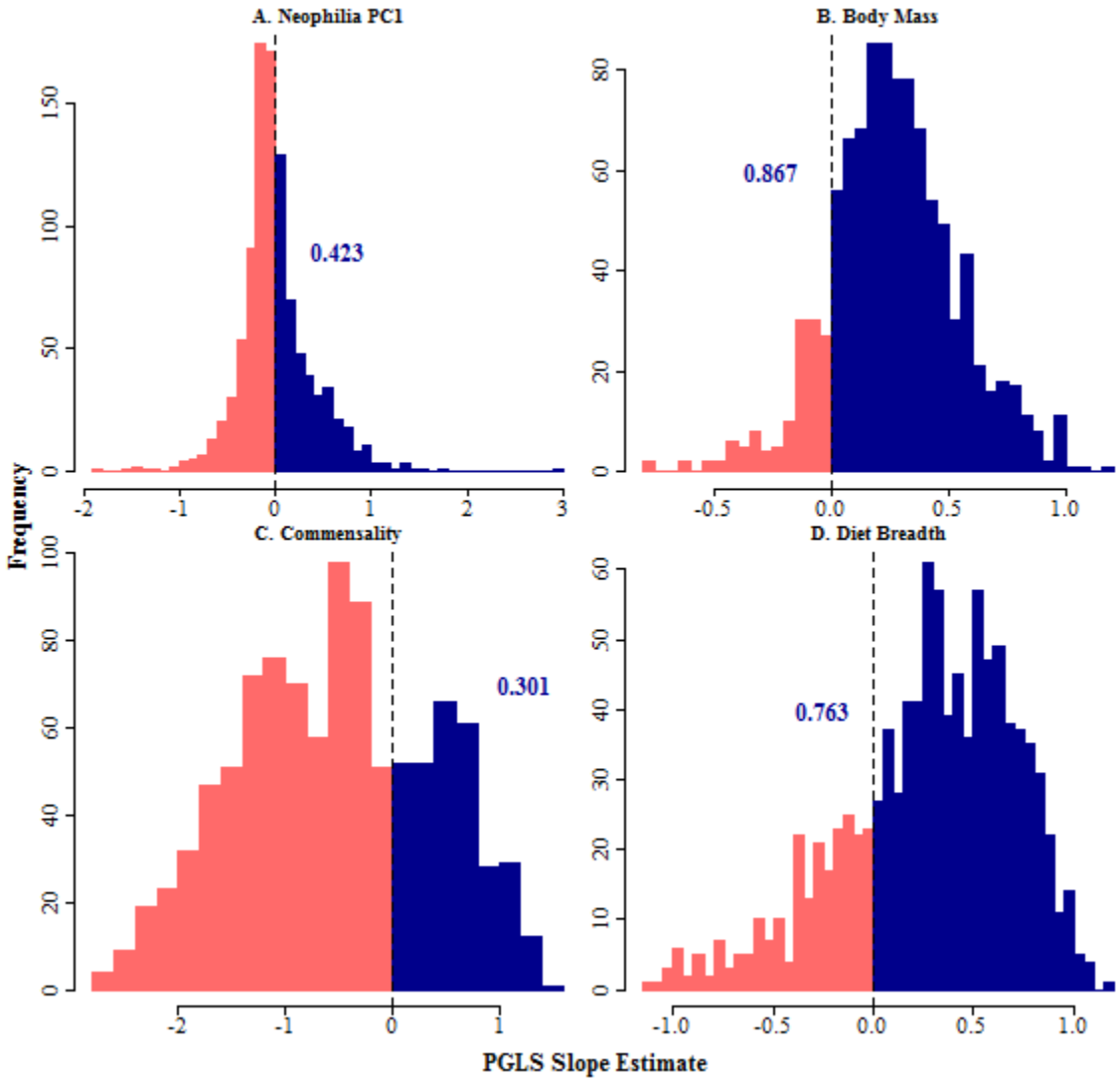

Figure 4.12. Distributions of slope coefficients from 1,000 resamplings of PGLS models for various predictor variables associated with ectoparasite infestation intensity. Panels indicate which predictor variable was tested: A. neophilia PC1, B. body mass (in grams), C. commensality, and D. diet breadth. Portions of each distribution in blue indicate positive associations, and red portions indicate negative associations. Numbers in blue indicate the support level for each association.

Table 4.5. Results of a mixed-effects models investigating associations between an individual's parasite infection intensity and habitat type, diet breadth, neophilia, neophobia, body size, and local rodent trapping densities (n/(nights*m^2)) from 247 individuals. Models also included additional random effects (trapping season nested within site ID, and species ID). Numbers in parentheses indicate standard errors, and those above are coefficients, with asterisks denoting significance (* $p < 0.1$; ** $p < 0.05$; *** $p < 0.01$).

Parasite Infection Intensity

	Dependent variable:			
	Total Helminth Intensity (A)	Cestode Intensity (B)	Nematode Intensity (C)	Ectoparasite Intensity (D)
Intercept	3.083**	-1.375	2.598*	0.941
	(1.313)	(1.751)	(1.527)	(0.981)
Conservancy habitat	-0.218***	-1.996***	-0.194**	-0.385***
	(0.074)	(0.359)	(0.081)	(0.115)
Pastoral habitat	0.500***	2.007***	0.385***	0.526***
	(0.053)	(0.257)	(0.056)	(0.101)
Diet breadth	-1.153*	0.639	-1.330*	0.248
	(0.669)	(0.745)	(0.777)	(0.481)
Neophilia PC1	0.178***	0.061	0.219***	0.156***
	(0.017)	(0.049)	(0.019)	(0.026)
Neophobia PC1	-0.092***	0.042	-0.105***	0.064***
	(0.012)	(0.041)	(0.012)	(0.021)
Neophobia PC2	-0.076***	0.065	-0.136***	0.084***
	(0.017)	(0.042)	(0.019)	(0.027)
Body mass (g)	1.873***	1.132***	1.976***	0.536***
	(0.044)	(0.135)	(0.048)	(0.057)
Local trapping density (n/(nights*m^2))	4.470***	-58.041***	8.859***	-14.021***
	(1.130)	(8.779)	(1.246)	(2.597)
Marginal R-squared	0.390	0.321	0.376	0.061
Conditional R-squared	0.427	0.321	0.517	0.576

showed support for predictions by body mass, with 86.7% of models lending "weak support" to a positive relationship between body mass and ectoparasite intensity [Figure 4.12b]. These results indicated that larger-bodied rodent species had higher average ectoparasite infestation intensities. When looking at patterns of total gastrointestinal parasite infection intensity within host species, habitat, exploratory tendency, body size, and local host density all were significant predictors of gastrointestinal parasite intensity [Table 4.5a]. Individuals captured in wildlife conservancies had lower gastrointestinal parasite intensity than those captured in homes (β=-0.218, SE=0.074, $p<0.004$). More neophilic individuals had higher intensities of gastrointestinal parasite infection intensity (β=0.178, SE=0.017, $p<0.001$; Figure 4.13), and more neophobic individuals had lower intensities, both in regard to their mobility as scored on PC1 (β=-0.092, SE=0.012, $p<0.001$) and their thigmotaxis as scored on PC2 (β=-0.076, SE=0.017, $p<0.001$). Rodent individuals living in areas with higher host densities had greater gastrointestinal parasite infection intensity than those living in less dense communities (β =4.470, SE=1.130, $p<0.001$). Larger-bodied individuals also had higher infection intensities (β =1.873, SE=0.044, $p<0.001$). Additionally, greater diet breadth was marginally associated with lower gastrointestinal parasite intensity (β =-1.153, SE=0.669, $p=0.085$). The model fit the data with R^2=0.427 and was driven by results for cestode and nematode intensities, identical in respect to significance and directionality [Table 4.5b and 4.5c].

For within species measurements of ectoparasite infestation intensity from exhaustive combings, habitat, exploratory tendency, body size, and local host densities all were significant predictors [Table 4.5d]. Rodent individuals trapped inside wildlife conservancies had lower ectoparasite infestation intensity than those caught within homes (β=-0.385, SE=0.115, $p<0.001$). More neophilic individuals again had greater intensities than less neophilic ones (β=0.156, SE=0.026, $p<0.001$), although neophobia was also positively associated with ectoparasite

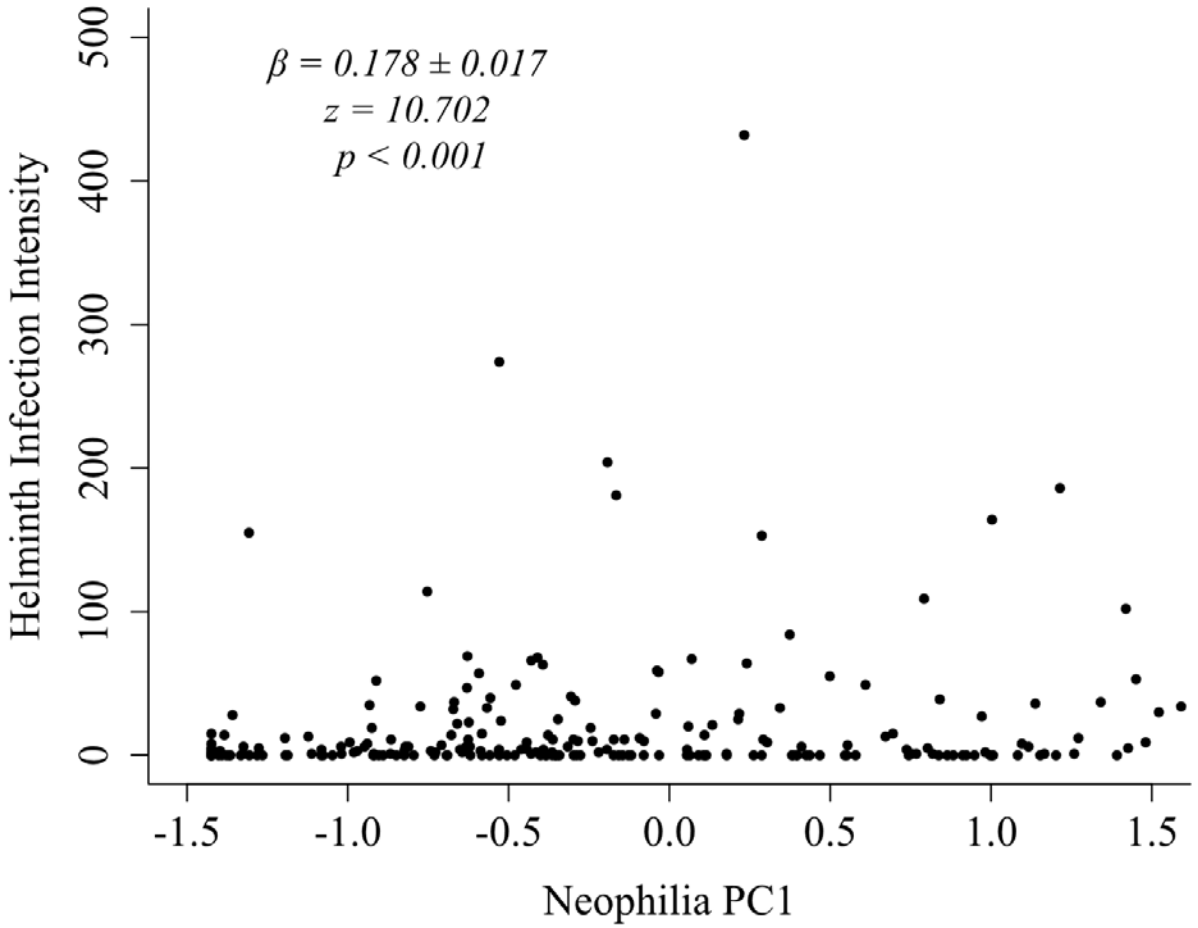

Figure 4.13. Scatterplot of exploratory tendencies (measured by neophilia PC1) of rodent individuals and their gastrointestinal parasite infection intensities. GLMMs indicate a significant positive association between these two variables, as is also shown below, although this specific graph does not control for the other fixed and random effects in the GLMM.

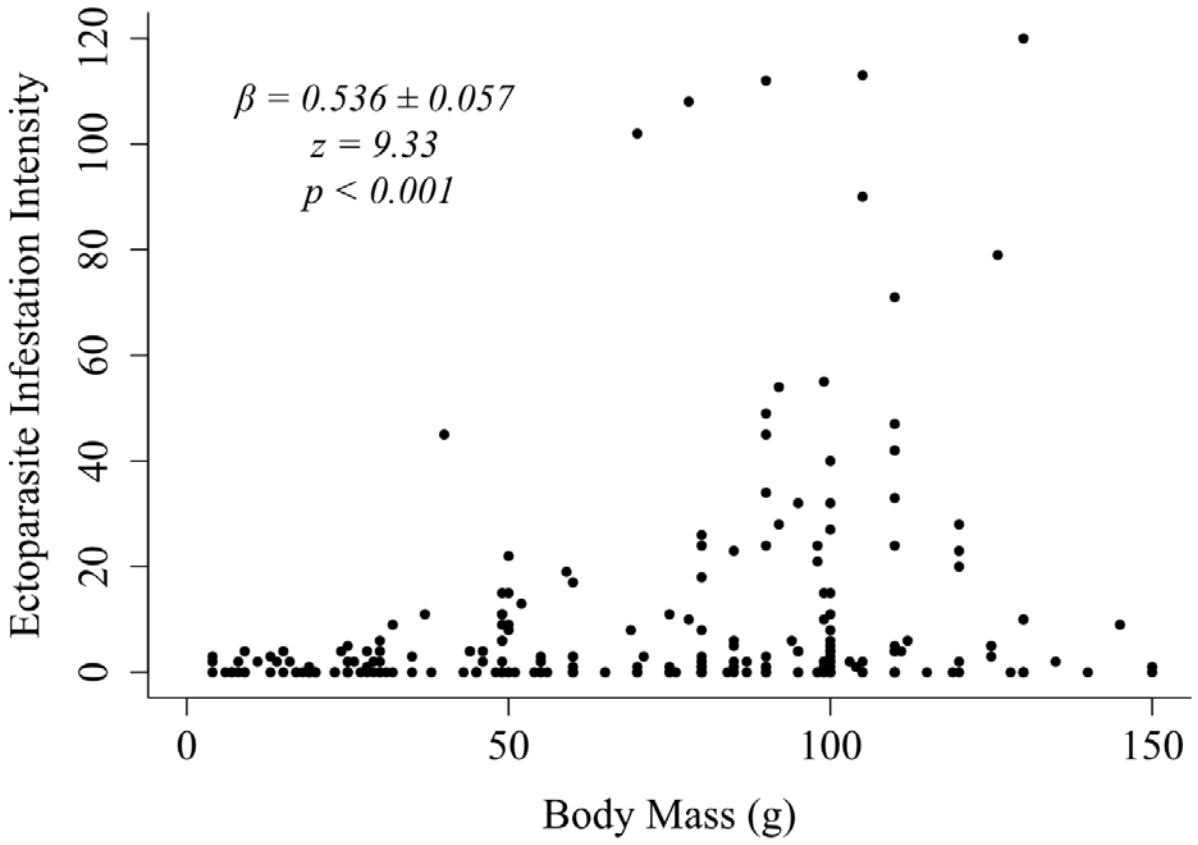

Figure 4.14. Scatterplot of body size (measured by mass, in grams) of rodent individuals and their ectoparasite infestation intensities. GLMMs indicate a significant positive association between these two variables, as is also shown below, although this specific graph does not control for the other fixed and random effects in the GLMM.

intensity (PC1: β=0.064, SE=0.021, p=0.002; PC2: β=0.084, SE=0.027, p=0.002). In contrast to the PGLS results, diet breadth had little effect on counts of ectoparasites within species, but body size was again positively correlated with intraspecific variation in ectoparasite infestation intensity (β =0.536, SE=0.057, p<0.001; Figure 4.14). Rodent individuals captured from locations with higher local host densities, contrary to predictions, had lower ectoparasite infestation intensities (β =-14.021, SE=2.597, p<0.001). The model fit the data with R^2=0.576.

4.4 Discussion

4.4.1 Predictors of commensality among rodent species and individuals

The only trait that was predictive of commensality within species in our study was exploratory tendency. Our model comparison framework in section 4.3.1 of the results indicated that the best supported model was the one in which exploratory tendency, as measured by neophiliac PC1, was predictive of commensality. We did not test for species level patterns in commensality due to methodological limitations of our analyses, but the low marginal R^2 values relative to the conditional ones indicated that the random effects, most likely species ID, were driving the fit of the models. Among individuals, though, this effect of exploratory behavior predicting commensality may be a by-product of their recent establishment in the relatively new agricultural villages in Laikipia. Theoretical research has indicated that exploratory tendencies may not be advantageous for sustained commensality, although they do play a role in establishing in new commensal environments (Wright et al., 2010). Recent research into exploratory tendencies of commensal bullfinches has noted such a phenomenon of decreased

exploratory tendency in established commensal species, although invading generations of the birds were more neophilic (Audet et al., 2015).

The lack of support for generalization for human foods among commensal rodents may indicate that the broad range of foods present in human homes might not actually be an important draw for commensal rodents; these animals may instead be drawn to homes for shelter from the elements and protection from natural predators (Tchernov, 1984). However, the fact that commensal individuals were found to be no larger or smaller-bodied than non-commensals suggests that thermoregulation may not be a particularly salient draw for these animals either. Second, diet as recorded in the MammalDIET database might not be a reasonable assessment of actual dietary adaptation and breadth; this may be exacerbated by the use of extrapolation to fill in missing species in this dataset. Body size was not an important factor in the commensality of rodent individuals within species either, suggesting that the commensality of rodents is not determined by thermoregulatory stress.

4.4.1 Parasite risk of commensal rodent species and individuals

As for parasitism, we found that habitat type (agricultural, pastoral, or wildlife conservancy) had a considerable effect on parasitism. With respect to gastrointestinal parasites, wildlife conservancy-captured rodents had a greater richness of parasites than commensals did, but lower infection intensities than commensals. The increase in gastrointestinal parasite richness among wildlife conservancy rodents relative to commensals may be due to an increased diversity of rodent and more broadly, mammalian hosts in wild habitats, which would potentially increase the number of parasites encountered (Wood et al., 2014). Although not driven by biotic diversity (local host richness), this effect could have been driven by an increase in habitat diversity present

in wildlife conservancies relative to human homes, with grasslands, scrub forests, and water holes, contributing to an increase in the diversity of parasites encountered for conservancy animals.

The more neophilic tendencies of commensal rodents did not have any effect on gastrointestinal parasite richness, but they were associated with higher intensities of both gastrointestinal parasite infections and ectoparasite infestations within species. This finding may be attributable to exploratory individuals interacting with the enclosed environments of human dwellings more intensely, but experiencing repeated exposures to the same parasites rather than novel ones. However, as shown by the positive effect of local host density on gastrointestinal parasite intensities, the denser communities of rodent hosts within homes may also be driving gastrointestinal parasite intensity. Further supporting this trend, more neophobic individuals also had lower gastrointestinal parasite intensities. However, more neophobic individuals, as well as those living at lower densities were shown to have higher intensities of ectoparasite infestations; such a result may indicate that when rodents are more densely packed and less fearful, ectoparasites can spread evenly among them, but when the opposite is true, ectoparasites may become clustered on the relatively fewer hosts.

With respect to ectoparasite infestation intensity, commensal rodents hosted far more ectoparasites than those captured in wildlife conservancies. This increase in ectoparasites among commensal rodents may signal some effect of livestock (cattle, goats, sheep) living in close proximity to the homes, as additional reservoirs for ectoparasites (Fuehrer et al., 2012). Further research into the species identifications of our collected ectoparasites may yield essential

information about ectoparasites found on our captured rodents that have come from, or are shared with livestock species.

Additionally, body size exhibited a consistent and strong positive association with gastrointestinal morphospecies richness and intensity, as well as ectoparasite infestation intensity. This pattern was observed both within species and between species in our PGLS models. Such a consistent association is to be expected for body size and parasite risk measures, as this pattern has been repeatedly observed in comparative studies and meta-analyses of mammal and other animals (Dunn et al., 2011b; Kamiya et al., 2013).

4.4.3 Implications

Our findings that commensal rodents are infected with a lower diversity, but greater intensity of parasites than their non-commensal counterparts suggest that commensal rodents may not pose as great a threat to human health, at least in terms of novel pathogens, as has been proposed in other research, although the greater intensity of parasites may help to maintain endemic infections shared between rodents and humans. However, such a claim requires data on specifically zoonotic pathogens to be truly relatable to human health. If these rodents were shown to carry zoonotic or endemic human pathogens, then they could potentially act as amplifying hosts for the relatively fewer parasites already present in and around the home in humans or other domesticated animals. Additionally, the increased infestation intensity of ectoparasites among commensal rodents may be of particular concern, as these ectoparasites can and regularly do carry blood-borne pathogens that may infect humans or livestock. Thus, future research is imperative for determining the health impacts of commensal rodents and their ectoparasites on cohabiting humans and their economically important herds.

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Chapter 5

Conclusion: Synthesis of results and implications for human evolution

5.1 Synthesis of results

The results presented in this dissertation indicate that there are consistent, but context dependent parasite risks associated with the behavioral patterns that favor the emergence of culture in humans and other animals. These parasite risks depend on the type of behavior, where behaviors that increase social contact and social learning are associated with greater socially transmitted parasite risk, and behaviors that increase exploration and innovation are associated with greater environmentally transmitted parasite risk. These risks also depend on the measurement of parasite risk that is considered. In this dissertation, I considered the measurements of parasite species richness, infection intensity, and indirectly, prevalence, as an indicator of disease saturation in social network models. The results showed that measurements which may be good predictors at one scale, for instance, a species level for richness, may not be good predictors at other scales, like at an individual level.

The results found for parasite richness associating with exploration and innovation at the species-level scale in Chapter 2 were not supported when considering individual-level interactions between exploratory tendencies and parasite richness in Chapter 4, and this could have been for a variety of reasons. First, although it would be tempting to treat the measurements of exploration and innovation rates in Chapter 2 as roughly equivalent with the exploratory tendencies measured in Chapter 4, this comparison may be flawed. The innovation and exploration rates measured in Chapter 2 were counted on a species-by species basis, and they were also counted in aggregate as anecdotal proxies for the exploration rate of an individual. In

comparison, the exploratory tendencies measured in Chapter 4 were quantified on an individual basis, and they were measured as personality traits that would favor exploration and innovation. Additionally, the two studies considered two different taxonomic groups of hosts, primates in Chapter 2 and rodents in Chapter 4. The types of exploratory behaviors exhibited by these two taxa are likely very different, and this may have effects on the diversity and intensity of parasites encountered. And finally, I cannot entirely rule out the fact that perhaps exploratory tendencies as personality traits may not be as correlated with innovation and exploration rates as has been previously assumed, as the observation of innovation and exploration requires broad, longitudinal datasets to allow for such categorization.

Regardless, the measurements of innovation and exploration rates in Chapter 2 and of exploratory tendencies in Chapter 4 did both show positive associations with parasite risk, albeit in different measurements: richness and intensity, respectively. The patterns of increased parasite intensity among exploratory individuals could conceivably lead to greater richness of parasites in aggregate for species, if more exploratory individuals are more likely to encounter and become heavily infected with rare parasites in the environment. Further theoretical and empirical research into such an association could provide the missing link between greater intensities of infection at the individual level and greater richness of parasites at the species level.

As for the effects of social contact on the transmission of close-contact parasites, the results were less convincing. On the species level among primates, a strong relationship was indeed noted between the social learning rates of individuals and the richness of socially transmitted parasites in Chapter 2. However, when delving further into this relationship in Chapter 3, there was no support for a link between social contact (as measured by effective network size) and the richness of parasites in primates. This result is made even more confusing

by the findings discussed in the introduction of Chapter 3 that indicated that social contact structure among human groups was positively associated with richnesses of cultural behavior variants, a very closely related metric to that in Chapter 2. But of course, as can be the case with these complicated relationships, an association between A and B and an association between B and C do not necessitate a relationship between A and C. This may have been the case with my attempts to find a relationship between social contact structure and the richness of socially transmitted parasites among primates. However, other such explanations for this finding may be more telling. First, the measurements of parasite richness and social network structure came from two different sources, which may have obfuscated an otherwise observable relationship. Secondly, the variation in social contact structure within a species may be more important than that between species. Studies discussed in Chapter 3 show that the overwhelming majority of previous associations found between social contact structure and richness of either parasites or cultural behaviors have been observed within species rather than between them. And finally, the issue of a small and unrepresentative sample of species and social networks could not be ruled out. As is the case, efforts to find such a relationship between social network structure and parasite risk would be greatly improved upon if more social networks of primates were published in the literature, as is likely to happen, given the current trajectory of social network research among primatologists.

5.2 Implications for human evolution

Taken together, these three chapters represent a significant advancement in the understanding of parasite risks associated with cultural behaviors. Although the research presented herein considers these associations largely among non-humans, the findings are almost

wholly transferable to humans, as a species which relies so heavily on social learning and exploration for its success and livelihood. The expansion of humans into novel environments and subsistence patterns over the course of our evolutionary history has undeniably been accompanied by an increase in parasite risk. Our reliance on social learning and socially-structured interactions have also led to larger and denser social groups which have supported greater numbers of socially transmitted diseases.

It is also serendipitous, and perhaps ironic that the same behavioral patterns that may have increased our exposure to parasites may also have helped us to overcome these infections. Perhaps because of their tradeoffs with exposure to new parasites, the same exploration and innovation that have driven the evolution of self-medicative behaviors in non-humans has also almost certainly formed the foundation of traditional ethnobotanical medicine as well as modern science-based medical approaches in humans. Additionally, our complex social structures and patterns of social learning have their foundations deep within the animal phylogeny, and these same behaviors that may have increased our exposure to socially transmitted diseases have also facilitated our transmission of knowledge about the treatment and prevention of such diseases.

References

Ahumada J. 1992. Grooming behavior of spider monkeys (Ateles geoffroyi) on Barro Colorado Island, Panama. Int J Primatol 13:33.

Altizer SM, Nunn CL, Thrall PH, Gittleman JL, Antonovics J, Cunningham AA, Dobson AP, Ezenwa V, Jones KE, Pedersen AB, Poss M, Pulliam JRC. 2003. Social organization and parasite risk in mammals: Integrating theory and empirical studies. Annu Rev Ecol Evol Syst 34:517–547.

Anderson RM, May RM. 1979. Population biology of infectious diseases: Part I. Nature 280:361–367.

Andre M, Ijaz K, Tillinghast JD, Krebs VE, Diem LA, Metchock B, Crisp T, McElroy PD. 2007. Transmission Network Analysis to Complement Routine Tuberculosis Contact Investigations. Am J Public Health 97:470–477.

Archer J. 1973. Tests for emotionality in rats and mice: a review. Anim Behav 21:205–235.

Arneberg P, Skorping A, Grenfell B, Read AF. 1998. Host densities as determinants of abundance in parasite communities. Proc R Soc Lond B Biol Sci 265:1283–1289.

Arnold C, Matthews LJ, Nunn CL. 2010. The 10kTrees website: A new online resource for primate phylogeny. Evol Anthropol 19:114–118.

Arnold TB, Emerson JW. 2011. Nonparametric Goodness-of-Fit Tests for Discrete Null Distributions. The R Journal 3:34–9.

Audet J-N, Ducatez S, Lefebvre L. 2015. The town bird and the country bird: problem solving and immunocompetence vary with urbanization. Behavioral Ecology 27:637.

Aureli F, Schaffner CM, Boesch C, Bearder SK, Call J, Chapman CA, Connor R, Fiore AD, Dunbar RIM, Henzi SP, Holekamp K, Korstjens AH, Layton R, Lee P, Lehmann J, Manson JH, Ramos-Fernández G, Strier KB, Schaik CPV. 2008. Fission-Fusion Dynamics: New Research Frameworks. Curr Anthropol 49:627–654.

Azad AF. 1990. Epidemiology of murine typhus. Annu Rev Entomol 35:553–569.

Baer GM. 1991. The Natural History of Rabies. Boca Raton: CRC Publishers.

Balasubramaniam K, Beisner B, Vandeleest J, Atwill E, McCowan B. 2016. Social buffering and contact transmission: network connections have beneficial and detrimental effects on Shigella infection risk among captive rhesus macaques. PeerJ 4:e2630.

Bar-Yosef O, Meadow RH. 1995. The origins of agriculture in the Near East. In: Price TD, Gebauer AB, editors. Last hunters- first farmers. Santa Fe, NM: School of American Research. p 39–94.

Barber I, Dingemanse NJ. 2010. Parasitism and the evolutionary ecology of animal personality. Philos Trans R Soc Lond B Biol Sci 365:4077–4088.

Barrett R, Kuzawa CW, McDade T, Armelagos GJ. 1998. Emerging and re-emerging infectious diseases: The third epidemiologic transition. Annu Rev Anthropol 27:247–271.

Barrickman NL, Bastian ML, Isler K, van Schaik CP. 2008. Life history costs and benefits of encephalization: A comparative test using data from long-term studies of primates in the wild. J Hum Evol 54:568–590.

Bartlett MS. 1960. The Critical Community Size for Measles in the United States. J R Stat Soc A 123:37–44.

Barton K. 2016. MuMIn: Multi-Model Inference. https://CRANR-projectorg/package=MuMIn.

Bates D, Mächler M, Bolker B, Walker S. 2015. Fitting Linear Mixed-Effects Models Using lme4. Journal of Statistical Software 67:1.

Bejan A. 2001. The tree of convective heat streams: its thermal insulation function and the predicted 3/4-power relation between body heat loss and body size. International Journal of Heat and Mass Transfer 44:699.

Berdoy M, Webster JP, Macdonald DW. 2000. Fatal attraction in rats infected with *Toxoplasma gondii*. Proc R Soc Lond B Biol Sci 267:1591–1594.

Bergmann C. 1847. About the relationships between heat conservation and body size of animals. Goett Stud 1:595–708.

Bininda-Emonds ORP, Cardillo M, Jones KE, MacPhee RDE, Beck RMD, Grenyer R, Price SA, Vos RA, Gittleman JL, Purvis A. 2008. The delayed rise of present-day mammals (corrigendum). Nature 456:274–274.

Bordes F, Morand S, Krasnov BR. 2011. Does investment into “expensive” tissue compromise anti-parasitic defence? Testes size, brain size and parasite diversity in rodent hosts. Oecologia 165:7–16.

Bordes F, Morand S, Ricardo G. 2008. Bat fly species richness in Neotropical bats: Correlations with host ecology and host brain. Oecologia 158:109–116.

Bordes F, Morand S. 2011. The impact of multiple infections on wild animal hosts: A review. Infect Ecol Epidemiol 1:7346.

Borgatti SP. 2005. Centrality and network flow. Soc Networks 27:55–71.

Bouwman KM, Hawley DM. 2010. Sickness behaviour acting as an evolutionary trap? Male house finches preferentially feed near diseased conspecifics. Biol Lett 6:462–465.

Boyer N, Réale D, Marmet J, Pisanu B, Chapuis J-L. 2010. Personality, space use and tick load in an introduced population of Siberian chipmunks *Tamias sibiricus*. J Anim Ecol 79:538–547.

Bush AO, Fernandez JA, Esch GW, Seed JR. 2001. Parasitism: The Diversity and Ecology of Animal Parasites. Cambridge: Cambridge University Press.

Butler MW, Toomey MB, McGraw KJ, Rowe M. 2011. Ontogenetic immune challenges shape adult personality in mallard ducks. Proc R Soc Lond B Biol Sci 279:326–333.

Butovskaya M, Kozintsev A, Welker C. 1996. Conflict and reconciliation in two groups of crab-eating monkeys differing in social status by birth. Primates 37:261.

Butovskaya ML, Kozintsev AG, Kozintsev BA. 1994. The structure of affiliative relations in a primate community: Allogrooming in stumptailed macaques (Macaca arctoides). Hum Evol 9:11.

Byrne RW, Barnard PJ, Davidson I, Janik VM, McGrew WC, Miklósi Á, Wiessner P. 2004. Understanding culture across species. Trends Cogn Sci 8:341–346.

Caillaud D, Craft ME, Meyers LA. 2013. Epidemiological effects of group size variation in social species. J R Soc Interface 10:20130206.

Campana MG, Hawkins MTR, Henson LH, Stewardson K, Young HS, Card LR, Lock J, Agwanda B, Brinkerhoff J, Gaff HD, Helgen KM, Maldonado JE, McShea WJ, Fleischer RC. 2016. Simultaneous identification of host, ectoparasite and pathogen DNA via in-solution capture. Mol Ecol Resour 16:1224–1239.

Cardy JL, Grassberger P. 1985. Epidemic models and percolation. J Phys A Mat Gen 18:L267–L271.

Cavanaugh DC, Ryan PF, Marshall JD Jr. 1969. The role of commensal rodents and their ectoparasites in the ecology and transmission of plague in southeast Asia. Bull Wildlife Dis Assoc 5:187–194.

Centola D. 2010. The spread of behavior in an online social network experiment. Science 329:1194–1197.

Chaisiri K, Siribat P, Ribas A, Morand S. 2015. Potentially Zoonotic Helminthiases of Murid Rodents from the Indo-Chinese Peninsula: Impact of Habitat and the Risk of Human Infection. Vector-Borne Zoonot Dis 15:73–85.

Checkley W, Buckley G, Gilman RH, Assis AM, Guerrant RL, Morris SS, Molbak K, Valentiner-Branth P, Lanata CF, Black RE. 2008. Multi-country analysis of the effects of diarrhoea on childhood stunting. Int J Epidemiol 37:816–830.

Chittka L, Niven J. 2009. Are bigger brains better? Curr Biol 19:R995–R1008.

Cohen S, Doyle WJ, Skoner DP, Rabin BS, Gwaltney JM. 1997. Social ties and susceptibility to the common cold. JAMA 277:1940–1944.

Cooper MA, Berntein IS, Hemelrijk CK. 2005. Reconciliation and relationship quality in Assamese macaques (Macaca assamensis). Am J Primatol 65:269–282.

Cooper N, Nunn CL. 2013. Identifying future zoonotic disease threats: Where are the gaps in our understanding of primate infectious diseases? Evol Med Public Health 2013:27–36.

Corbet GB, Hill JE. 1991. A World List of Mammalian Species. New York: Oxford University Press.

Cords M. 2000. Agonistic and affiliative relationships in a blue monkey group. In: Whitehead PF, Jolly CJ, editors. Old World Monkeys. Cambridge: Cambridge University Press. p 453–479.

Craft ME, Volz E, Packer C, Meyers LA. 2010. Disease transmission in territorial populations: the small-world network of Serengeti lions. J R Soc Interface 8:776–786.

Craft ME. 2015. Infectious disease transmission and contact networks in wildlife and livestock. Philos Trans R Soc Lond B Biol Sci 370:20140107–20140107.

Crow JF. 2010. Wright and Fisher on Inbreeding and Random Drift. Genetics 184:609–611.

Csardi G, Nepusz T. 2006. igraph software package for complex network research. InterJournal Complex Systems:1965.

Côte IM, Poulin R. 1995. Parasitism and group size in social animals: A meta-analysis. Behav Ecol 6:159–165.

Day RL, Coe RL, Kendal JR, Laland KN. 2003. Neophilia, innovation and social learning: A study of intergeneric differences in callitrichid monkeys. Anim Behav 65:559–571.

Deacon RMJ. 2006. Housing, husbandry and handling of rodents for behavioral experiments. Nat Protoc 1:936–946.

Deaner RO, Barton RA, van Schaik CP. 2003. Primate brains and life histories: Renewing the connection. In: Kappeler PM, Pereira ME, editors. Primate Life Histories and Socioecology. Chicago: The University of Chicago Press. p 233–265.

Dingemanse NJ, Réale D. 2005. Natural selection and animal personality. Behaviour 142:1159–1184.

Downs CT, McDonald PM, Brown K, Ward D. 2003. Effects of *Acacia* condensed tannins on urinary parameters, body mass, and diet choice of an *Acacia* specialist rodent, *Thallomys nigricauda*. J Chem Ecol 29:845–858.

Ducatez S, Audet J-N, Rodriguez JR, Kayello L, Lefebvre L. 2016. Innovativeness and the effects of urbanization on risk-taking behaviors in wild Barbados birds. Anim Cogn 20:33–42.

Dunbar R. 1982. Structure of social relationships in a captive gelada group: A test of some hypotheses derived from studies of a wild population. Primates 23:89.

Dunbar RI, Dunbar EP. 1976. Contrasts in social structure among black-and-white colobus monkey groups. Animal Behaviour 24:84–92.

Dunbar RIM, Shultz S. 2007. Evolution in the social brain. Science 317:1344–1347.

Dunbar RIM. 1991. Functional Significance of Social Grooming in Primates. Folia Primatol 57:121.

Dunn FL. 1968. Epidemiological factors: health and disease in hunter-gatherers. In: Lee RB, DeVore I, editors. Man the hunter. Chicago, IL: Aldine. p 221–228.

Dunn JC, Cole EF, Quinn JL. 2011a. Personality and parasites: sex-dependent associations between avian malaria infection and multiple behavioural traits. Behav Ecol Sociobiol 65:1459–1471.

Dunn JC, Cole EF, Quinn JL. 2011b. Personality and parasites: sex-dependent associations between avian malaria infection and multiple behavioural traits. Behav Ecol Sociobiol 65:1459.

Ezenwa VO, Archie EA, Craft ME, Hawley DM, Martin LB, Moore J, White L. 2016. Host behaviour–parasite feedback: an essential link between animal behaviour and disease ecology: Table 1. Proc R Soc Lond B Biol Sci 283:20153078–9.

Ezenwa VO, Etienne RS, Luikart G, Beja-Pereira A, Jolles AE. 2010. Hidden consequences of living in a wormy world: nematode-induced immune suppression facilitates tuberculosis invasion in African buffalo. Am Nat 176:613–624.

Ezenwa VO. 2004. Host social behavior and parasitic infection: a multifactorial approach. Behav Ecol 15:446–454.

Felsenstein J. 1971. Inbreeding and variance effective numbers in populations with overlapping generations. Genetics 68:581–597.

Fichet-Calvet E, Koulémou K, Koivogui L, Soropogui B, Sylla O, Lecompte E, Daffis S, Kouadio A, Kouassi S, Akoua-Koffi C, Denys C, Meulen ter J. 2005. Spatial distribution of commensal rodents in regions with high and low Lassa fever prevalence in Guinea. Belg J Zool 135:63–67.

File SE, Wardill AG. 1975. Validity of head-dipping as a measure of exploration in a modified hole-board. Psychopharmacologia 44:53–59.

Formenty P, Boesch C, Wyers M, Steiner C, Donati F, Dind F, Walker F, Le Guenno B. 1999. Ebola virus outbreak among wild chimpanzees living in a rain forest of Côte d'Ivoire. J Infect Dis 179:S120–S126.

Franz M, Nunn CL. 2009. Network-based diffusion analysis: a new method for detecting social learning. Proc R Soc Lond B Biol Sci 276:1829–1836.

Freckleton RP, Harvey PH, Pagel M. 2002. Phylogenetic analysis and comparative data: A test and review of evidence. Am Nat 160:712–726.

Freeland WJ. 1979. Primate social groups as biological islands. Ecology 60:719–728.

Froeschke G, Matthee S. 2014. Landscape characteristics influence helminth infestations in a peri-domestic rodent - implications for possible zoonotic disease. Parasites & Vectors 7:393.

Fuehrer H-P, Igel P, Treiber M, Baumann TA, Riedl J, Swoboda P, Joachim A, Noedl H. 2012. Ectoparasites of livestock, dogs, and wild rodents in the Chittagong Hill Tracts in southeastern Bangladesh. Parasitol Res 111:1867–1870.

Galic MA, Spencer SJ, Mouihate A, Pittman QJ. 2009. Postnatal programming of the innate immune response. Integr Comp Biol 49:237–245.

Garamszegi LZ, Erritzøe J, Møller AP. 2007. Feeding innovations and parasitism in birds. Biol J Linn Soc 90:441–455.

Ghai RR, Chapman CA, Omeja PA, Davies TJ, Goldberg TL. 2014. Nodule Worm Infection in Humans and Wild Primates in Uganda: Cryptic Species in a Newly Identified Region of Human Transmission. PLoS Neglect Trop Dis 8:e2641.

Glass RJ, Glass LM, Beyeler WE, Min HJ. 2006. Targeted social distancing design for pandemic influenza. Emerg Infect Dis 12:1671–1681.

Gomez-Uchida D, Palstra FP, Knight TW, Ruzzante DE. 2013. Contemporary effective population and metapopulation size (N e and meta-N e): comparison among three salmonids inhabiting a fragmented system and differing in gene flow and its asymmetries. Ecol Evol 3:569–580.

Gratz NG. 1997. The burden of rodent-borne diseases in Africa South of the Sahara. Belg J Zool 127S:71–84.

Gratz NG. 1999a. Rodent reservoirs & flea vectors of natural foci of plague. In: WHO plague manual: epidemiology, distribution, surveillance and control. Geneva: WHO. p 63–96.

Gratz NG. 1999b. Urbanization, arthropod and rodent pests and human health. In: 3rd International Conference on Urban Pests; Robinson WH, Rettich F, Rambo GW, editors. p 51–58.

Greenberg R, Mettke-Hofmann C. 2001. Ecological Aspects of Neophobia and neophilia in birds. In: Heyes CM, Galef BG Jr, editors. Social Learning in Animals: The Roots of Culture. San Diego: Academic Press. p 119.

Greenberg R. 2003. The role of neophobia and neophilia in the development of innovative behaviour of birds. In: Reader SM, Laland KN, editors. Animal Innovation. New York: Oxford University Press. p 175–196.

Gregory RD. 1990. Parasites and Host Geographic Range as Illustrated by Waterfowl. Funct Ecol 4:645–654.

Griffin RH, Nunn CL. 2012. Community structure and the spread of infectious disease in primate social networks. Evol Ecol 26:779–800.

Groube L. 1996. The impact of diseases upon the emergence of agriculture. In: Harris DR, editor. The origins and spread of agriculture and pastoralism in Eurasia. London: UCL Press. p 101–129.

Groves CP. 2001. Primate taxonomy. Washington, DC: Smithsonian Institution Press.

Guerra AS, Eckerlin RP, Dowling APG, Durden LA, Robbins RG, Dittmar K, Helgen KM, Agwanda B, Allan BF, Hedlund T, Young HS. 2016. Host-Parasite Associations in Small Mammal Communities in Semiarid Savanna Ecosystems of East Africa. J Med Entomol 53:851–860.

Haensch S, Bianucci R, Signoli M, Rajerison M, Schultz M, Kacki S, Vermunt M, Weston DA, Hurst D, Achtman M, Carniel E, Bramanti B. 2010. Distinct clones of *Yersinia pestis* caused the Black Death. PLoS Pathog 6:e1001134.

Hamede RK, Bashford J, McCallum H, Jones M. 2009. Contact networks in a wild Tasmanian devil (Sarcophilus harrisii) population: using social network analysis to reveal seasonal variability in social behaviour and its implications for transmission of devil facial tumour disease. Ecol Lett 12:1147–1157.

Han BA, Schmidt JP, Bowden SE, Drake JM. 2015. Rodent reservoirs of future zoonotic diseases. PNAS [Internet] 112:7039. Available from: http://dx.doi.org/10.1073/pnas.1501598112

Handcock MS, Hunter DR, Butts CT, Goodreau SM, Morris M. 2008. statnet: Software Tools for the Representation, MaVisualization, Analysis and Simulation of Network Data. Journal of Statistical Software 24:1–11.

Happold DCD ed. 2013. Mammals of Africa. Volume III: Rodents, Hares and Rabbits. London: Bloomsbury.

Hart BL. 1990. Behavioral adaptations to pathogens and parasites: Five strategies. Neurosci Biobehav Rev 14:273–294.

Hart BL. 2010. Beyond Fever: Comparative Perspectives on Sickness Behavior. In: Breed MD, Moore J, editors. Encyclopedia of Animal Behavior. Oxford: Academic Press. p 205–210.

Hart BL. 2011. Behavioural defences in animals against pathogens and parasites: Parallels with the pillars of medicine in humans. Philos Trans R Soc Lond B Biol Sci 366:3406–3417.

Hayes JP, Shonkwiler JS. 2006. Allometry, antilog transformations, and the perils of prediction on the original scale. Physiol Biochem Zool 79:665–674.

Henrich J. 2004. Demography and Cultural Evolution: How Adaptive Cultural Processes Can Produce Maladaptive Losses—The Tasmanian Case. American Antiquity 69:197.

Heroldová M, Tkadlec E, Bryja J, Zejda J. 2008. Wheat or barley? Feeding preferences affect distribution of three rodent species in agricultural landscape. Appl Anim Behav Sci 110:354–362.

Heyes CM, Galef BG Jr eds. 1996. Social Learning in Animals: The Roots of Culture. London: Academic Press.

Huffman MA. 1997. Current evidence for self-medication in primates: A multidisciplinary perspective. Yearb Phys Anthropol 40:171–200.

Hulme-Beaman A, Dobney K, Cucchi T, Searle JB. 2016. An Ecological and Evolutionary Framework for Commensalism in Anthropogenic Environments. Trends Ecol Evol 31:633–645.

Izawa K. 1980. Social behavior of the wild black-capped capuchin (Cebus apella). Primates 21:443.

Jacobs A, Petit O. 2011. Social network modeling: A powerful tool for the study of group scale phenomena in primates. Am J Primatol 73:741–747.

Jacobs A, Sueur C, Deneubourg JL, Petit O. 2011. Social Network Influences Decision Making During Collective Movements in Brown Lemurs (Eulemur fulvus fulvus). Int J Primatol 32:721–736.

Jolles AE, Ezenwa VO, Etienne RS, Turner WC, Olff H. 2008. Interactions between macroparasites and microparasites drive infection patterns in free-ranging African buffalo. Ecology 89:2239–2250.

Jones C. 1983. Social organization of captive black howler monkeys (Alouatta caraya):“Social competition” and the use of non-damaging behavior. Primates 24:25.

Jones KE, Bielby J, Cardillo M, Fritz SA, O'Dell J, Orme CDL, Safi K, Sechrest W, Boakes EH, Carbone C, Connolly C, Cutts MJ, Foster JK, Grenyer R, Habib M, Plaster CA, Price SA, Rigby EA, Rist J, Teacher A, Bininda-Emonds ORP, Gittleman JL, Mace GM, Purvis A.

2009. PanTHERIA: A species-level database of life history, ecology, and geography of extant and recently extinct mammals. Ecology 90:2648.

Jones KE, Patel NG, Levy MA, Storeygard A, Balk D, Gittleman JL, Daszak P. 2008. Global trends in emerging infectious diseases. Nature 451:990–993.

Kamilar JM, Marshack JL. 2012. Does geography or ecology best explain "cultural" variation among chimpanzee communities? J Hum Evol 62:256–260.

Kamiya T, O'Dwyer K, Nakagawa S, Poulin R. 2013. What determines species richness of parasitic organisms? A meta-analysis across animal, plant and fungal hosts. Biol Rev Camb Philos Soc 89:123–134.

Kasper C, Voelkl B. 2009. A social network analysis of primate groups. Primates 50:343–356.

Kavaliers M, Colwell DD. 1995. Reduced spatial learning in mice infected with the nematode, *Heligmosomoides polygyrus*. Parasitology 110:591–597.

Keeling MJ, Eames KTD. 2005. Networks and epidemic models. J R Soc Interface 2:295–307.

Keesing F. 1998. Ecology and behavior of the pouched mouse, *Saccostomus mearnsi*, in central Kenya. J Mammal 79:919–931.

Keesing F. 2000. Cryptic consumers and the ecology of an African savanna. BioScience 50:205–215.

Kendal RL, Custance DM, Kendal JR, Stoinski TS, Rakotomalala NL, Rasamimanana H. 2010. Evidence for social learning in wild lemurs (*Lemur catta*). Learn Behav 38:220–234.

Kiesecker JM, Skelly DK, Beard KH, Preisser E. 1999. Behavioral reduction of infection risk. Proc Natl Acad Sci USA 96:9165–9168.

Kimura M, Saito K, Nakano R. 2007. Extracting Influential Nodes for Information Diffusion on a Social Network. AAAI 1:1–6.

King AJ, Clark FE, Cowlishaw G. 2011. The dining etiquette of desert baboons: the roles of social bonds, kinship, and dominance in co-feeding networks. Am J Primatol 73:768–774.

Kissling WD, Dalby L, Fløjgaard C, Lenoir J, Sandel B, Sandom C, Trøjelsgaard K, Svenning J-C. 2014. Establishing macroecological trait datasets: digitalization, extrapolation, and validation of diet preferences in terrestrial mammals worldwide. Ecol Evol 4:2913–2930.

Kliethermes CL, Crabbe JC. 2006. Genetic independence of mouse measures of some aspects of novelty seeking. Proc Natl Acad Sci USA 103:5018–5023.

Koprivnikar J, Gibson CH, Redfern JC. 2012. Infectious personalities: behavioural syndromes and disease risk in larval amphibians. Proc R Soc Lond B Biol Sci 279:1544–1550.

Kotler BP, Brown JS, Smith RJ, Wirtz WO II. 1988. The effects of morphology and body size on rates of owl predation on desert rodents. Oikos 53:145–152.

Kuijt I, Finlayson B. 2009. Evidence for food storage and predomestication granaries 11,000 years ago in the Jordan Valley. Proc Natl Acad Sci USA 106:10966–10970.

Kummer H, Goodall J. 1985. Conditions of Innovative Behaviour in Primates. Philosophical Transactions of the Royal Society of London: B 308:203.

Laviola G, Macrì S, Morley-Fletcher S. 2003. Risk-taking behavior in adolescent mice: psychobiological determinants and early epigenetic influence. Neurosci Biobehav Rev 27:19–31.

Lefebvre L, Reader SM, Sol D. 2004. Brains, Innovations and Evolution in Birds and Primates. Brain Behav Evol 63:233–246.

Legendre P. 2014. lmodel2: Model II Regression. https://CRANR-projectorg/package=lmodel2.

Liebl AL, Martin LB. 2012. Exploratory behaviour and stressor hyper-responsiveness facilitate range expansion of an introduced songbird. Proc Biol Sci 279:4375–4381.

Lindenfors P, Nunn CL, Jones KE, Cunningham AA, Sechrest W, Gittleman JL. 2007. Parasite species richness in carnivores: Effects of host body mass, latitude, geographical range and population density. Global Ecol Biogeogr 16:496–509.

Loehle C. 1995. Social Barriers to Pathogen Transmission in Wild Animal Populations. Ecology 76:326.

Löttker P, Huck M, Zinner DP, Heymann EW. 2007. Grooming relationships between breeding females and adult group members in cooperatively breeding moustached tamarins (Saguinus mystax). Am J Primatol 69:1159–1172.

Maclean EL, Hare B, Nunn CL, Addessi E, Amici F, Anderson RC, Aureli F, Baker JM, Bania AE, Barnard AM, Boogert NJ, Brannon EM, Bray EE, Bray J, Brent LJN, Burkart JM, Call J, Cantlon JF, Cheke LG, Clayton NS, Delgado MM, DiVincenti LJ, Fujita K, Herrmann E, Hiramatsu C, Jacobs LF, Jordan KE, Laude JR, Leimgruber KL, Messer EJE, de A Moura AC, Ostojić L, Picard A, Platt ML, Plotnik JM, Range F, Reader SM, Reddy RB, Sandel AA, Santos LR, Schumann K, Seed AM, Sewall KB, Shaw RC, Slocombe KE, Su Y, Takimoto A, Tan J, Tao R, van Schaik CP, Virányi Z, Visalberghi E, Wade JC, Watanabe A, Widness J, Young JK, Zentall TR, Zhao Y. 2014. The evolution of self-control. PNAS 111:E2140–E2148.

Martin JGA, Réale D. 2008. Temperament, risk assessment and habituation to novelty in eastern chipmunks, *Tamias striatus*. Anim Behav 75:309–318.

Massen JJM, Sterck EHM. 2013. Stability and Durability of Intra- and Intersex Social Bonds of Captive Rhesus Macaques (Macaca mulatta). Int J Primatol 34:770–791.

McCabe CM, Reader SM, Nunn CL. 2014. Infectious disease, behavioural flexibility and the evolution of culture in primates. Proc R Soc Lond B Biol Sci 282:20140862.

McCormack JB, Webb PA, Krebs JW, Johnson KM, Smith ES. 1987. A prospective study of the epidemiology and ecology of Lassa fever. J Infect Dis 155:1–9.

McLennan IS, Taylor-Jeffs J. 2004. The use of sodium lamps to brightly illuminate mouse houses during their dark phases. Lab Anim 38:384–392.

Mery F, Kawecki TJ. 2003. A fitness cost of learning ability in *Drosophila melanogaster*. Proc R Soc Lond B Biol Sci 270:2465–2469.

Mettke-Hofmann C, Winkler H, Leisler B. 2002. The significance of ecological factors for exploration and neophobia in parrots. Ethology 108:249–272.

Milton K. 1996. Effects of bot fly (*Alouattamyia baeri*) parasitism on a free-ranging howler monkey (*Alouatta palliata*) population in Panama. J Zool 239:39–63.

Moore J. 2002. Parasites and the behavior of animals. Oxford: Oxford University Press.

Morand, S. 2000. Wormy world: Comparative tests of theoretical hypotheses on parasite species richness. In: Poulin R, Morand S, Skorping A, editors. Evolutionary Biology of Host-parasite Relationships. Amsterdam: Elsevier. p 63–79.

Morand S, Poulin R. 1998. Density, body mass and parasite species richness of terrestrial mammals. Evol Ecol 12:717.

Muggeo VMR. 2003. Estimating regression models with unknown break-points. Stat Med 22:3055–3071.

Mundry R. 2014. Statistical Issues and Assumptions of Phylogenetic Generalized Least Squares. In: Garamszegi LZ, editor. Modern Phylogenetic Comparative Methods and Their Application in Evolutionary Biology. Berlin: Springer-Verlag. p 131.

Nagy KA. 1987. Field metabolic rate and food requirement scaling in mammals and birds. Ecol Monogr 57:111–128.

Navarrete A, van Schaik CP, Isler K. 2011. Energetics and the evolution of human brain size. Nature 480:91–93.

Nunn CL, Altizer SM, Jones KE, Sechrest W. 2003. Comparative tests of parasite species richness in primates. Am Nat 162:597–614.

Nunn CL, Altizer SM, Sechrest W, Cunningham AA. 2005. Latitudinal gradients of parasite species richness in primates. Diversity Distrib 11:249–256.

Nunn CL, Altizer SM. 2005. The global mammal parasite database: An online resource for infectious disease records in wild primates. Evol Anthropol 14:1–2.

Nunn CL, Altizer SM. 2006. Infectious Disease in Primates: Behavior, Ecology and Evolution. New York: Oxford University Press.

Nunn CL, Dokey AT-W. 2006. Ranging patterns and parasitism in primates. Biol Lett 2:351–354.

Nunn CL, Jordán F, McCabe CM, Verdolin JL, Fewell JH. 2015. Infectious disease and group size: more than just a numbers game. Philos Trans R Soc Lond B Biol Sci 370:20140111–20140111.

Nunn CL, van Schaik CP. 2002. A comparative approach to reconstructing the socioecology of extinct primates. In: Plavcan JM, Plavcan JM, Kay RF, Kay RF, Jungers WL, Jungers WL, van Schaik CP, editors. Reconstructing Behavior in the Primate Fossil Record. Boston: Springer US. p 159–215.

Nunn CL. 2002. Spleen size, disease risk and sexual selection: A comparative study in primates. Evol Ecol Res 4:91–107.

Nunn CL. 2012. Primate disease ecology in comparative and theoretical perspective. Am J Primatol 74:497–509.

Orme D, Freckleton R, Thomas G, Petzoldt T, Fritz S, Isaac N, Pearse W. 2013. caper: Comparative Analyses of Phylogenetics and Evolution in R. https://CRANR-projectorg/package=caper.

Pagel M. 1999. Inferring the historical patterns of biological evolution. Nature 401:877–884.

Paradis E, Claude J, Strimmer K. 2004. APE: Analyses of Phylogenetics and Evolution in R language. Bioinformatics 20:289.

Patterson JEH, Ruckstuhl KE. 2013. Parasite infection and host group size: A meta-analytical review. Parasitology 140:803–813.

Pedersen AB, Altizer SM, Poss M, Cunningham AA, Nunn CL. 2005. Patterns of host specificity and transmission among parasites of wild primates. Int J Parasitol 35:647–657.

Perry S. 1996. Female-female social relationships in wild white-faced capuchin monkeys, Cebus capucinus. Am J Primatol 40:167–182.

Petraitis PS, Dunham AE, Niewiarowski PH. 1996. Inferring multiple causality: The limitations of path analysis. Funct Ecol 10:421–431.

Potterat JJ, Rothenberg RB, Muth SQ. 1999. Network structural dynamics and infectious disease propagation. Int J STD AIDS 10:182–185.

Poulin R. 1995. Phylogeny, ecology, and the richness of parasite communities in vertebrates. Ecol Monogr 65:283–302.

Poulin R. 1997. Species Richness of Parasite Assemblages: Evolution and Patterns. 28:341.

Powell A, Shennan S, Thomas MG. 2009. Late Pleistocene demography and the appearance of modern human behavior. Science 324:1298–1301.

R Core Team. 2016. R: A language and environment for statistical computing. https://wwwR-projectorg/.

Read JM, Eames KTD, Edmunds WJ. 2008. Dynamic social networks and the implications for the spread of infectious disease. J R Soc Interface 5:1001–1007.

Reader SM, Biro D. 2010. Experimental identification of social learning in wild animals. Learn Behav 38:265–283.

Reader SM, Hager Y, Laland KN. 2011. The evolution of primate general and cultural intelligence. Philos Trans R Soc Lond B Biol Sci 366:1017–1027.

Reader SM, Laland KN eds. 2003. Animal Innovation. New York: Oxford University Press.

Reader SM, Laland KN. 2001. Primate innovation: Sex, age and social rank differences. Int J Primatol 22:787–805.

Reader SM, Laland KN. 2002. Social intelligence, innovation, and enhanced brain size in primates. Proc Natl Acad Sci USA 99:4436–4441.

Reader SM, MacDonald K. 2003. Environmental Variability and Primate Behavioral Flexibility. In: Laland KN, Reader SM, editors. Animal Innovation. Oxford: Oxford University Press. p 83–116.

Reader SM. 2003. Innovation and social learning: individual variation and brain evolution. Anim Biol 53:147–158.

Rendell L, Fogarty L, Hoppitt WJE, Morgan TJH, Webster MW, Laland KN. 2011. Cognitive culture: Theoretical and empirical insights into social learning strategies. Trends Cogn Sci 15:68–76.

Rich KM, Denwood MJ, Stott AW, Mellor DJ, Reid SWJ, Gunn GJ. 2013. Systems approaches to animal disease surveillance and resource allocation: methodological frameworks for behavioral analysis. PLoS ONE 8:e82019.

Rico JLR, Ferraz DB, Ramalho-Pinto FJ, Morato S. 2010. Neonatal exposure to LPS leads to heightened exploratory activity in adolescent rats. Behav Brain Res 215:102–109.

Rieucau G, Giraldeau LA. 2011. Exploring the costs and benefits of social information use: an appraisal of current experimental evidence. Philos Trans R Soc Lond B Biol Sci 366:949–957.

Rifkin JL, Nunn CL, Garamszegi LZ. 2012. Do animals living in larger groups experience greater parasitism? A meta-analysis. Am Nat 180:70–82.

Roberts WW, Mooney RD, Martin JR. 1974. Thermoregulatory behaviors of laboratory rodents. J Comp Physiol Psych 86:693.

Romano V, Duboscq J, Sarabian C, Thomas E, Sueur C, MacIntosh AJJ. 2016. Modeling infection transmission in primate networks to predict centrality-based risk. Am J Primatol 78:767.

Rowe N, Myers M. 2011. All the World's Primates. http://www.alltheworldsprimates.org

Rowe N. 1996. The pictorial guide to the living primates. East Hampton, NY: Pogonias Press.

Rubenstein DI, Hohmann ME. 1989. Parasites and Social Behavior of Island Feral Horses. Oikos 55:312.

Rushmore J, Caillaud D, Matamba L, Stumpf RM, Borgatti SP, Altizer SM. 2013. Social network analysis of wild chimpanzees provides insights for predicting infectious disease risk. J Anim Ecol 82:976–986.

Sallis JF, Owen N, Fotheringham MJ. 2000. Behavioral epidemiology: A systematic framework to classify phases of research on health promotion and disease prevention. Annals of Behavioral Medicine 22:294.

Sandel AA, Miller JA, Mitani JC, Nunn CL, Patterson SK, Garamszegi LZ. 2016. Assessing sources of error in comparative analyses of primate behavior: Intraspecific variation in group size and the social brain hypothesis. J Hum Evol 94:126–133.

Schaller M. 2006. Parasites, behavioral defenses, and the social psychological mechanisms through which cultures are evoked. Psychol Inq 17:96.

Seyfarth R. 1980. The distribution of grooming and related behaviours among adult female vervet monkeys. Anim Behav 28:798.

Shaw MT, Keesing F, Ostfeld RS. 2002. Herbivory on *Acacia* seedlings in an East African savanna. Oikos 99:385–392.

Sih A. 2013. Understanding variation in behavioural responses to human-induced rapid environmental change: a conceptual overview. Animal Behaviour 85:1077–1088.

Smith RJ. 1993. Logarithmic transformation bias in allometry. Am J Phys Anthropol 90:215.

Sol D, Duncan RP, Blackburn TM, Cassey P, Lefebvre L. 2005. Big brains, enhanced cognition, and response of birds to novel environments. PNAS 102:5460–5465.

Soler JJ, Peralta-Sánchez JM, Martín-Vivaldi M, Martín-Platero AM, Flensted-Jensen E, Møller AP. 2011. Cognitive skills and bacterial load: comparative evidence of costs of cognitive proficiency in birds. Naturwissenschaften 99:111–122.

Speicher MR, Antonarakis SE, Motulsky AG eds. 2010. Vogel and Motulsky's Human Genetics. 4th ed. Berlin: Springer.

Spence KW. 1960. Behavior Theory and Learning: Selected Papers. Englewood Cliffs, NJ: Prentice-Hall.

Springer A, Kappeler PM, Nunn CL. 2016. Dynamic vs. static social networks in models of parasite transmission: predicting Cryptosporidium spread in wild lemurs. J Anim Ecol 86:419.

Sueur C, Petit O, De Marco A, Jacobs AT, Watanabe K, Thierry B. 2011. A comparative network analysis of social style in macaques. Animal Behaviour 82:845–852.

Sugiyama Y. 1971. Characteristics of the social life of bonnet macaques (Macaca radiata). Primates 12:247.

Sugiyama Y. 1988. Grooming interactions among adult chimpanzees at Bossou, Guinea, with special reference to social structure. Int J Primatol 9:393.

Taylor KD, Green MG. 1976. The influence of rainfall on diet and reproduction in four African rodent species. J Zool 180:367–389.

Taylor-Seehafer M, Rew L. 2000. Risky sexual behavior among adolescent women. J Soc Pediatr Nurs 5:15–25.

Tchernov E. 1984. Commensal animals and human sedentism in the Middle East. In: Clutton-Brock J, Grigson C, editors. Animals in archaeology: Early herders and their flocks. Oxford: British Archaeological Reports. p 91–115.

Terborgh J, Janson CH. 1986. The socioecology of primate groups. Annu Rev Ecol Syst 17:111–136.

Thornton A, Clutton-Brock TH. 2011. Social learning and the development of individual and group behaviour in mammal societies. Philos Trans R Soc Lond B Biol Sci 366:978–987.

Thrall PH, Antonovics J. 1997. Polymorphism in sexual versus non-sexual disease transmission. Proc R Soc Lond B Biol Sci 264:581.

Tinbergen N. 1963. On aims and methods of ethology. Z Tierpsychol 20:410–433.

Tomasello M, Call J. 1997. Primate Cognition. New York: Oxford University Press.

VanderWaal KL, Atwill ER, Hooper S, Buckle K, McCowan B. 2013. Network structure and prevalence of Cryptosporidium in Belding's ground squirrels. Behav Ecol Sociobiol 67:1951–1959.

Vas Z, Lefebvre L, Johnson KP, Reiczigel J, Rózsa L. 2011. Clever birds are lousy: Co-variation between avian innovation and the taxonomic richness of their amblyceran lice. Int J Parasitol 41:1295–1300.

Vitone ND, Altizer SM, Nunn CL. 2004. Body size, diet and sociality influence the species richness of parasitic worms in anthropoid primates. Evol Ecol Res 6:183–199.

Vogt JL. 1978. The social behavior of a marmoset (Saguinus fuscicollis) group II: Behavior patterns and social interaction. Primates 19:287.

WD Kissling, L Dalby, C Flojgaard, J L, B S, C S, K T, J-C S. 2014. Data from: Establishing macroecological trait datasets: digitalization, extrapolation, and validation of diet preferences in terrestrial mammals worldwide. Ecol Evol.

Walsh PD, Abernathy KA, Bermejo M, Beyers R, De Wachter P, Akou ME, Huljbregts B, Mambounga DI, Toham AK, Kilbourn AM, Lahm SA, Latour S, Maisels F, Mbina C, Mihindou Y, Obiang SN, Effa EN, Starkey MP, Telfer P, Thibault M, Tutin CEG, White LJT, Wilkie DS. 2003. Catastrophic ape decline in western equatorial Africa. Nature 422:611–614.

Walsh RN, Cummins RA. 1976. The open-field test: a critical review. Psychol Bull 83:482–504.

Weissman DB, Barton NH. 2012. Limits to the rate of adaptive substitution in sexual populations. PLoS Genet 8:e1002740.

White LA, Forester JD, Craft ME. 2015. Using contact networks to explore mechanisms of parasite transmission in wildlife. Biol Rev Camb Philos Soc 92:389–409.

Whiten A, Hinde RA, Stringer CB, Laland KN eds. 2012. Culture Evolves. New York: Oxford University Press.

Whiten A. 2000. Primate culture and social learning. Cognitive Sci 24:477–508.

Wilson DS, Coleman K, Clark AB, Biederman L. 1993. Shy-bold continuum in pumpkinseed sunfish (Lepomis gibbosus): An ecological study of a psychological trait. Journal of Comparative Psychology 107:250.

Wolfheim JH. 1977. A Quantitative Analysis of the Organization of a Group of Captive Talapoin Monkeys *(Miopithecus talapoin)*. Folia Primatol 27:1.

Wong TW, Chan YC, Joo YG, Lee HW, Lee PW, Yanagihara R. 1989. Hantavirus infections in humans and commensal rodents in Singapore. Trans R Soc Trop Med Hyg 83:248–251.

Wood CL, Lafferty KD, DeLeo G, Young HS, Hudson PJ, Kuris AM. 2014. Does biodiversity protect humans against infectious disease? Ecology 95:817–832.

Wrangham RW, Chapman CA, Clark-Arcadi AP, Isabirye-Basuta G, Goodall J, Itani J. 1996. Social ecology of Kanyawara chimpanzees: implications for understanding the costs of great ape groups. In: McGrew WC, editor. Great Ape Societies. p 45.

Wright S. 1931. Evolution in Mendelian populations. Genetics 16:97–159.

Wright TF, Eberhard JR, Hobson EA, Avery ML, Russello MA. 2010. Behavioral flexibility and species invasions: the adaptive flexibility hypothesis. Ethology Ecology & Evolution 22:393.

Wyncoll G, Tangri D. 1989. Of mice and men: is the presence of commensal animals in archaeological sites a positive correlate of sedentism? Paléorient 15:85–94.

Yom-Tov Y. 2003. Body sizes of carnivores commensal with humans have increased over the past 50 years. Funct Ecol 17:323–327.

Young HS, Dirzo R, Helgen KM, McCauley DJ, Billeter SA, Kosoy MY, Osikowicz LM, Salkeld DJ, Young TP, Dittmar K. 2014. Declines in large wildlife increase landscape-level prevalence of rodent-borne disease in Africa. Proc Natl Acad Sci USA 111:7036–7041.

Young HS, Dirzo R, McCauley DJ, Agwanda B, Cattaneo L, Dittmar K, Eckerlin RP, Fleischer RC, Helgen LE, Hintz A, Montinieri J, Zhao S, Helgen KM. 2015a. Drivers of Intensity and Prevalence of Flea Parasitism on Small Mammals in East African Savanna Ecosystems. J Parasitol 101:327–335.

Young HS, McCauley DJ, Dirzo R, Goheen JR, Agwanda B, Brook C, Otarola-Castillo E, Ferguson AW, Kinyua SN, McDonough MM, Palmer TM, Pringle RM, Young TP, Helgen KM. 2015b. Context-dependent effects of large-wildlife declines on small-mammal communities in central Kenya. Ecol Appl 25:348–360.

Zylberberg M, Klasing KC, Hahn TP. 2014. In house finches, Haemorhous mexicanus, risk takers invest more in innate immune function. Animal Behaviour 89:115.

van Schaik CP, Deaner RO, Merrill MY. 1999. The conditions for tool use in primates: Implications for the evolution of material culture. J Hum Evol 36:719–741.