Infectious disease transmission is a cost of sociality in humans and other animals. Nevertheless, the mechanisms linking social behaviour to infection risk are poorly known. We conducted a field experiment to examine how host intrinsic traits, behaviour and physiology affect infection of nonhuman primates with gastrointestinal parasites. We measured rate to reinfection in a social group of red-capped mangabeys, Cercocebus torquatus, following chemotherapeutic treatment for parasite infections. By measuring behaviour, infection and glucocorticoid levels, we compared the relative effects of space sharing, directional contact and physiological stress on risk of acquiring new infections. We found that, within proximity networks, individuals that were central, well connected and had a tendency to switch groups were at increased risk of infection with helminths. Protozoan infections, however, were acquired more uniformly across the population. In general, position in the social network and, in particular, space sharing appears to be more important than the immunosuppressive effects of physiological stress or host traits in determining risk of infection. Our results suggest that future studies of disease ecology within wildlife populations should focus on measures of network association in addition to individual host traits.

In humans and other social animals, variation in behaviour and physiology can alter the risk of exposure to, and infection by, pathogens, ultimately affecting host fitness (Kappeler, Crémer, & Nunn, 2015; Nunn, Craft, Gillespie, Schaller, & Kappeler, 2015; Silk, 2014). Variation in parasitism often has direct links to host social behaviour, such that infection-related costs of sociality are considered important selective forces in human and animal evolution (Altizer et al., 2003; Kappeler et al., 2015; Moller, Dufval, & Allander, 1993). Clarifying the mechanisms whereby sociality translates to infection is important for our understanding of disease ecology and host–parasite coevolution. For example, it is currently unclear whether close proximity and high levels of contact or increased physiological stress resulting from within-group social dynamics is more important for infection in primates.

Macroparasites are generally aggregated within populations, with few hosts harbouring the majority of infections (Crofton, 1971; Poulin, 2007; Shaw & Dobson, 1995). Classic measures typically associated with infection include age, sex and dominance status (Nunn & Altizer, 2006). Behavioural and physiological mechanisms that influence encounter rates and immune status can vary with these measures, further explaining why certain individuals are at increased risk of infection. Focus on classic measures alone may therefore obscure important contributions of social connectivity and/or physiological stress (Cavigelli & Caruso, 2015; Kappeler et al., 2015).

In primates, trade-offs between sociality, encounter rates and immune function result in conflicting predictions for disease risk of individuals (Nunn & Altizer, 2006). For example, age can increase parasitism if larger-bodied individuals occupy more space, require more resources and contact contaminated foods and substrates disproportionately. Conversely, lack of acquired immunity in younger individuals may increase risk of parasitism in juveniles (Hudson & Dobson, 1997). Parasitism tends to be more common in males than in females across vertebrate taxa (Habig & Archie, 2015). However, male-biased parasitism is confounded by body size, such that the immunosuppressive effects of stress and parasitism may be more pronounced in males than in females.
testosterone are unclear (Zuk & McKeen, 1996). In primate societies with dominance hierarchies, greater access to resources and rank-mediated social contact should increase risk for high-ranking individuals (MacIntosh et al., 2012; Rushmore et al., 2013). Meanwhile, immunosuppressive effects of stress hormones can increase susceptibility in either dominant or subordinate individuals depending on species-typical dynamics and hierarchical stability (Cavigelli & Caruso, 2015; Sapolsky, 2005).

Empirically, intraspecific differences in physical contact, proximity (González-Hernández et al., 2014; MacIntosh et al., 2012; Rimbach et al., 2015) and physiological stress (Chapman, Saj, & Snait, 2007; Clough, Heistermann, & Kappeler, 2010; Muehlenbein, 2006) are associated with transmission of parasites within primate groups. In Japanese macaques, Macaca fuscata, for example, socially mediated exposure seems to be more important than the immunosuppressive effects of stress in explaining why dominant females have more infections from directly transmitted parasites (MacIntosh et al., 2012). Nevertheless, the relative importance of network connectivity versus physiological stress as mechanisms for facilitating pathogen spread is not well understood.

In this study, we investigated how social connectivity and physiological stress compare to host intrinsic factors with respect to explaining patterns of parasite aggregation in primates. To overcome confounding heterogeneities in exposure, susceptibility and resulting infection levels over time, we experimentally removed parasites and measured rate to reinfection. To date, experimental manipulations of parasite infections in wild animals have focused primarily on behavioural, immune and fitness responses to parasitism (Coster, Neve, Martín-Gálvez, Therry, & Lens, 2010; Hillegass, Waterman, & Roth, 2010; Raveh, Neuhaus, & Dobson, 2015). Here, we investigated patterns of parasite reacquisition following chemotherapeutic treatment of red-capped mangabeys, Cercocebus torquatus, for gastrointestinal helminth and protozoan parasites. Specifically, we examined how centrality within social networks and individual stress varied within the population according to sex, age and dominance. We then compared how these mechanistic explanations (e.g. contact, proximity and/or stress) performed against classic measures in predicting rate to reinfection. We compared reinfection from helminths and protozoans separately, given their inherent differences in time to infection and aggregation within hosts (Shaw & Dobson, 1995). By focusing on gastrointestinal parasites, which can be collected noninvasively and can be treated with oral medications, we were able to compare results from our field experiment to other observational studies that also investigated gastrointestinal parasites. We predicted that following experimental manipulation of infection, centrality would augment classic measures to more powerfully explain differences in infection rates.

METHODS

Study Site and Population

The study took place at Rhoko Research and Conservation Education Centre (41.21°N, 16.16°E), the forest site of the Centre for Education, Research and Conservation of Primates and Nature (CERCOPAN). Rhoko is located in the transition zone surrounding the Oban division of Cross River National Park in Cross River State, Nigeria. The vegetation is characteristic of lowland rainforest, forming a mosaic of disturbed and relatively undisturbed forest patches. Climate includes a long wet season from April to November and a short dry season from November to March.

We studied 49 red-capped mangabeys living in a multimale–multifemale social group that either had been rescued from the bushmeat and pet trades as young juveniles, or were first- to third-generation captive born. The group was housed in a 1 ha open-topped forest enclosure with full canopy cover and within the natural home range of the species. The population was provisioned daily but also ate wild foods opportunistically and drank from a stream running through the enclosure. Animals were vulnerable to natural predators (e.g. snakes and birds of prey) and parasites. All animals were well habituated and individually recognizable to the trained observer through individual differences in size, pelage and facial characteristics; all data were collected from animals where observers had achieved 100% agreement on identification. The age of each individual at the start of data collection (range 1.08–18.5 years) was known from birth records or estimated from tooth wear, pelage characteristics and sexual maturity at date of rescue. We categorized males as adult (>6 years, N = 9), subadult (≥3 year, N = 9) and juvenile (<3 years, N = 7), and females as adult (≥4 years, N = 19) or juvenile (<4 years, N = 5). Cercocebus torquatus is currently listed as vulnerable by IUCN (Oates, Gippoliti, & Groves, 2008).

Chemotherapeutic Treatment

In June of 2012, the entire population was treated for gastrointestinal parasites via simultaneous administration of metronidazole (50 mg/kg for 7 days) for protozoans, mebendazole (50 mg/kg for 3 days) for nematodes, and praziquantel (20 mg/kg for 3 days) for cestodes and trematodes. For all drugs, a single dose was delivered in maize cereal to identified individuals to ensure that each animal received at least one dose. The remaining doses were dissolved in fruit and administered via group feeds following standard practice for the population-level treatment at CERCOPAN. The treatment period lasted 10 days in total.

Study Design

The study took advantage of a planned treatment event, providing a unique opportunity to measure patterns of infection following an intervention and minimizing additional risk. The treatment regimen was developed from standard treatment practices at CERCOPAN and in consultation with two wildlife veterinarians. Oral administration of drugs and noninvasive assessment of parasites were used to minimize adverse risks and enhance welfare. The Institutional Animal Care and Use Committee at University of Wisconsin-Madison approved all research activities (protocol V1490).

We collected faecal samples and behavioural and health data between May and September 2012. We conducted faecal sampling 1 month prior to treatment and parallel behavioural sampling for 3 months immediately following treatment. We collected pre-treatment faecal samples in triplicate from each individual to increase detection of parasites shed intermittently. To assess variation in protozoan infection, which are infectious upon shedding, we measured time to infection from protozoans during the period of high sampling intensity only (30 days post treatment). We gradually decreased sampling intensity in the second month (ca. every 5 days) and in the third month (ca. every 10 days) to detect new infections from helminths, which develop in the external environment and have longer prepatent periods, such that we measured helminth reinfection over this longer time period (80 days post treatment). We extracted hormones from triplicate samples directly following treatment (ca. every 10 days), with sampling intensity reduced to twice a month (ca. every 15 days) for the second and third months.
**Behavioural Data Collection**

We collected associational data by recording all grooming partners, direction of grooming and nearest neighbours (within 2 m) of focal individuals during three observation periods daily: early morning (0700–1000 hours), mid-morning (1100–1500 hours) and evening (1600–1900 hours). The mangabeys were often dispersed in dense undergrowth throughout their enclosure, making it difficult to collect data on a predetermined schedule or in a specific order. We therefore selected individuals opportunistically. We allowed a minimum of 1.5 min to elapse between observations to reduce interdependence of data. We did not sample an individual if it was an associate or nearest neighbour in the preceding observation. We collected dominance data by recording all observed agonistic interactions as well as the directionality of submissions and suppliants using structured ad libitum sampling, as conducted for similar species (Range & Noé, 2002). Three observers collected all data for 81 days over 3 months post treatment. We tested and accepted interobserver reliability by calculating Fleiss’s Kappa test for categorical agreements between multiple observers (Kappa = 0.89, P < 0.001 for observers 1 and 2; Kappa = 0.92, P < 0.001 for observers 1 and 3).

**Faecal Collection and Preservation**

We collected faecal samples immediately following defecation and fixed the samples within 2 h of collection. We used separate aliquots of each sample for preservation of gastrointestinal parasites and hormones. First, we took a 2 g aliquot from within the faecal mass and stored it in a 3:1 ratio of 10% formalin to faeces for preservation of gastrointestinal parasites (Greiner & McIntosh, 2009). We then separated a second aliquot, mixed it thoroughly, and placed 0.50–0.55 g in a 15 ml tube. We mixed equal parts (2.5 ml) of distilled water and ethanol with the sample by shaking it vigorously for 5 min for preservation of hormones (Ziegler & Wittwer, 2005).

We extracted hormones in the field via solid-phase extraction (SPE) (Ziegler & Wittwer, 2005). Samples were stored for over 24 h until faecal material naturally sedimented at the bottom of the tube and a clear supernatant was evident. We decanted supernatant into a small weighing dish, removed 2 ml and pushed it through the SPE columns (Prewitt C18, Alltech, Deerfield, IL, U.S.A.) using a 5 ml disposable syringe at a flow rate of 1 ml/min. We then washed solid-phase extraction columns by pushing 1 ml of distilled water through the cartridges at the same rate to eliminate contaminants. We capped cartridges and stored them at room temperature out of direct sunlight until transport. We transported formalin-preserved samples and SPE cartridges to the University of Wisconsin, Madison following all applicable import, export and International Air Transport Association regulations.

**Behavioural Data Analysis**

We constructed dominance matrices from post-treatment dyadic suppliants and aggressive and avoidance interactions between adults of the same sex using SOCPROG 2.6 (Whitehead, 2009). We used David’s scores (DS) to dichotomize individuals as ‘usually dominant’ (DS > 0) or ‘usually subordinate’ (DS < 0) (de Vries, Stevens, & Vervaecke, 2006). We assessed linearity of male and female hierarchies using de Vries’ test and h with 1000 permutations for dominance hierarchies containing unknown or tied relationships (de Vries et al., 2006).

We constructed adjacency matrices for social network analyses directly from observed post-treatment pairwise associations (‘proximity’ network) and directional grooming interactions (‘contact’ networks) using SOCPROG 2.6 (Whitehead, 2009). We imported matrices with attribute information into R v.3.2.2 (R Core Team, 2014), where we conducted all further analyses unless otherwise specified. We performed calculations of symmetric network metrics from proximity matrices and calculations of asymmetric metrics from directional contact matrices using the ‘sna’ package (Farine & Whitehead, 2015). For each individual in each network, we calculated node-based measures commonly used for modelling transmission of infectious diseases: (1) degree centrality: the number of associates or interactants (hereafter referred to as ‘centrality’); (2) strength: the number of associations or interactions; (3) closeness: the shortest number of paths needed to reach all other individuals; and (4) betweenness: the number of shortest paths going through an individual (Drew & Perkins, 2015). We calculated in degree and out degree for directional networks, representing groom-receive and groom-give, respectively. Given the nonindependence of data in network analyses, we compared network metrics to host characteristics using permutation tests. We built null models from data stream-based randomizations and measured significance by comparing the test statistic of the models fitted to the observed data with the test statistic calculated from 1000 permutations of the network using the ‘sna’ package (Farine, 2013; Farine & Whitehead, 2015). We calculated Cohen’s d effect size for all comparisons using the ‘compute.es’ package (Farine & Whitehead, 2015; Re, 2015). We constructed network diagrams using UCINET software’s NetDraw program (Borgatti, Everett, & Freeman, 2002), with node size representing individual centrality, weighted edges representing strength, and without filters.

**Parasitology**

We concentrated 1 g of formalin-preserved faeces via faecal sedimentation following the protocol of Greiner and McIntosh (2009) for assessment of gastrointestinal parasites of primates (Greiner & McIntosh, 2009). Briefly, we suspended 1 g of faeces in 40 ml of sedimentation solution (soapy water), mixed it gently to avoid formation of bubbles and filtered it twice through cheesecloth to remove large debris. We allowed the mixture to sediment for 10 min, after which we decanted the supernatant. We resuspended the remaining pellet in distilled water and allowed it to sediment for another 10 min. We then removed the supernatant with a transfer pipette and preserved the sediment in formalin until examination. We systematically examined the entirety of the sediment at 10× objective light magnification for helmhing eggs and larvae. We then examined one drop of sediment from each sample at 40× for identification of protozoan cysts. We measured representative parasites with a calibrated ocular micrometer and photographed them at 40× magnification. All helmhing eggs, larvae and protozoan cysts were assigned to taxa based on their size, shape, colour and contents. Parasite richness (number of different parasite taxa within a host) and prevalence (percentage of individuals infected with a particular parasite taxon) were calculated (Bush, Lafferty, Lotz, & Shostak, 1997). We evaluated the efficacy of treatment by examining changes in parasite richness over time (before and after treatment) using one-way ANOVA and Tukey HSD post hoc tests. Prior to this study, parasite communities had not been reported for this species.

**Faecal Cortisol Analysis**

We measured faecal cortisol levels via enzyme immunoassay at the Wisconsin National Primate Research Center Assay Services Unit. We eluted steroid hormones using 2 ml of 100% methanol after washing cartridges with 1 ml of 5% methanol. We then...
they had generalized variance inflation factors (GVIF) within reason \((-4)\) (O'Brien, 2007), satisfied the proportional hazards assumption (i.e. residuals were not significantly correlated with time, ZPH: Pearson's \(r: P > 0.05\)), and they explained significantly more variance than the null model (likelihood ratio test: \(P < 0.05\)) (Kleinbaum & Klein, 2012).

We report hazard ratios (HR), the ratio of the chance of parasite acquisition in one level of an explanatory variable relative to the other, for all significant predictors. We produced survival curves for significant predictors using the Kaplan–Meier method. We dichotomized centrality scores at the median because hazard ratios and Kaplan–Meier curves are more interpretable when comparing groups, and because in general, models with dichotomized variables outperformed those with continuous variables (i.e. produced higher log likelihoods and fewer ZPH violations). We performed all analyses with the 'surv', 'coxph' and 'cox.zph' functions in the 'survival' package using R v.3.2.2 (R Core Team, 2014; Therneau, 2015).

RESULTS

Dominance

We constructed dominance matrices from 628 female \((\mu + SD = 66.11 + 3.09.4\) per individual; \(\mu + SD = 3.48 + 1.63\) per dyad) and 368 male \((\mu + SD = 81.78 + 32.33\) per individual, \(\mu + SD = 9 + 3.59\) per dyad) dyadic dominance interactions. For both male and female hierarchies, deviEs test of linearity indicated that dominance was moderately linear and nonrandom for female \((H' = 0.73, P < 0.001)\) and male \((H' = 0.84, P < 0.01)\) hierarchies.

Centrality

We constructed proximity networks from 2374 observed pairwise associations \((\mu + SD = 96.50 + 48.54\) per individual; \(\mu + SD = 197 + 0.98\) per dyad) and contact networks from 555 dyadic grooming interactions \((\mu + SD = 11.32 + 9.37\) per individual; \(\mu + SD = 0.23 + 0.19\) per dyad). Directed contact networks were more heterogeneous (varied across sex, age and dominance status) compared to proximity networks (Fig. 1). Females had higher closeness \((t_{27} = 4.66, P < 0.001, d = 1.33)\) and betweenness \((t_{27} = 3.64, P < 0.001, d = 1.04)\) in the contact networks compared to males, and higher centrality \((t_{27} = 5.83, P < 0.001, d = 1.66)\) and strength \((t_{27} = 6.17, P < 0.05, d = 1.76)\) measures in the groom-give network. Adults had significantly higher closeness \((t_{27} = 3.63, P < 0.001, d = 1.05)\) in the contact networks and higher centrality \((t_{27} = 6.15, P < 0.001, d = 1.78)\) and strength \((t_{27} = 4.08, P < 0.05, d = 1.18)\) in groom-receive network. High-ranking males were more central in the groom-give network \((t_{7} = 2.97, P < 0.001, d = 1.99)\) and had stronger connections in both proximity \((t_{7} = 2.19, P < 0.05, d = 1.47)\) and groom-receive networks \((t_{7} = 2.61, P < 0.01, d = 1.75)\). Dominant females had higher closeness \((t_{17} = 2.58, P < 0.01, d = 1.2)\) and betweenness \((t_{17} = 2.02, P < 0.001, d = 0.94)\) in contact networks and higher centrality \((t_{17} = 2.14, P < 0.01, d = 0.99)\) and stronger connections in groom-give network \((t_{17} = 3.05, P < 0.001, d = 1.42)\).

Faecal Cortisol

We collected a total of 343 post-treatment faecal samples (7 per individual) for hormone analyses. Faecal cortisol levels ranged from 0.57 ng/g to 49.39 ng/g among individuals. Cortisol levels were positively related to age \((r_{t} = 0.55, P < 0.001)\), and low-ranking individuals had higher levels \((mean = 12.77 ng/g)\) than high-ranking individuals \((mean = 8.77 ng/g)\) \((U = 50, N_{1} = 16, N_{2} = 12, P < 0.05)\).
P < 0.05). Females (mean = 9.95 ng/g) had marginally higher cortisol values than males (mean = 8.07 ng/g) \((U = 215, N_1 = 24, N_2 = 25, P < 0.1)\). There was no relationship between centrality within social networks and average faecal cortisol levels.

**Parasitism**

We collected a total of 982 faecal samples, for an average of 20 samples per individual. Mangabeys were infected with six protozoan and nine helminths prior to treatment (Table 1). Average protozoan \((F_{5,287} = 39.22, P < 0.01)\) and helminth \((F_{7,383} = 13.18, P < 0.01)\) richness differed significantly over time. Post hoc comparisons showed significant reductions in parasite richness between pre-treatment (helminth: \(\mu + \text{SD} = 1.55 + 1.29, P < 0.001;\) protozoa: \(\mu + \text{SD} = 2.77 + 1.36, P < 0.001\)) and post-treatment samples (helminth: \(\mu + \text{SD} = 0.67 + 0.87;\) protozoa: \(\mu + \text{SD} = 0.27 + 0.86\)). Protozoan infections were acquired more quickly than helmint infections. There was no significant difference between pre-treatment richness and richness at the final sample for helmints or protozoans, demonstrating return to baseline levels (Fig. 2a, b). Prevalence of five protozoans and four helmints was reduced by at least 50% and contributed to subsequent reinfections (Table 1). These nine parasites were therefore included in Cox proportional hazard models. Protozoans and helminths showed different distributions at the final time points, further supporting our decision to model infection separately for each group of parasites (Fig. 2c, d). A right-skewed distribution of helmint richness showed that a small number of individuals harboured many parasite taxa whereas most individuals were infected with only a few taxa (Fig. 2d).

Following treatment (and prior to the end of the study), 81 new helmint infections (48 in adults) and 189 new protozoan infections (111 in adults) were detected. High centrality, closeness and betweenness in proximity networks were associated with rate to infection with helmint parasites in adult mangabeys (Fig. 3, Table 2). When all age classes were included, these patterns were in the same direction but marginal. No centrality measures explained time to reinfection with protozoans. Neither host traits (e.g. sex, age or dominance) nor average post-treatment cortisol levels were associated with rate to infection under either model.

**Table 1**

<table>
<thead>
<tr>
<th>Classification</th>
<th>Genus</th>
<th>Species</th>
<th>Life cycle</th>
<th>Mode of transmission</th>
<th>Pre</th>
<th>Post</th>
<th>Final</th>
</tr>
</thead>
<tbody>
<tr>
<td>Protozoa</td>
<td>Entamoeba*</td>
<td>hartmanni</td>
<td>Direct</td>
<td>Ingestion of trophozoites and cysts</td>
<td>0.80</td>
<td>0.02</td>
<td>0.51</td>
</tr>
<tr>
<td></td>
<td>Entamoeba*</td>
<td>histolytica/dispar</td>
<td>Direct</td>
<td>Ingestion of trophozoites and cysts</td>
<td>0.58</td>
<td>0.02</td>
<td>0.78</td>
</tr>
<tr>
<td></td>
<td>Entamoeba*</td>
<td>coli+</td>
<td>Direct</td>
<td>Ingestion of trophozoites and cysts</td>
<td>0.69</td>
<td>0.02</td>
<td>0.67</td>
</tr>
<tr>
<td></td>
<td>Iodamoeba*</td>
<td>bütschlii</td>
<td>Direct</td>
<td>Ingestion of trophozoites and cysts</td>
<td>0.69</td>
<td>0.02</td>
<td>0.61</td>
</tr>
<tr>
<td></td>
<td>Chilomastix*</td>
<td>mesnili</td>
<td>Direct</td>
<td>Ingestion of trophozoites and cysts</td>
<td>0.47</td>
<td>0.00</td>
<td>0.55</td>
</tr>
<tr>
<td></td>
<td>Balantidium</td>
<td>coli</td>
<td>Direct</td>
<td>Ingestion of trophozoites and cysts</td>
<td>0.88</td>
<td>0.02</td>
<td>0.04</td>
</tr>
<tr>
<td></td>
<td>Trichuris</td>
<td>trichura</td>
<td>Direct</td>
<td>Ingestion of eggs</td>
<td>0.02</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>Capillaria</td>
<td>sp.</td>
<td>Unknown</td>
<td>Unknown</td>
<td>0.06</td>
<td>0</td>
<td>0.04</td>
</tr>
<tr>
<td></td>
<td>Strongyloides*</td>
<td>sp.</td>
<td>Direct</td>
<td>Ingestion of eggs/penetration of skin by larvae</td>
<td>0.22</td>
<td>0.06</td>
<td>0.35</td>
</tr>
<tr>
<td></td>
<td>Enterobius</td>
<td>sp.</td>
<td>Direct</td>
<td>Ingestion of eggs</td>
<td>0.16</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>Hookworm*</td>
<td>sp.</td>
<td>Direct</td>
<td>Penetration of skin by larvae</td>
<td>0.12</td>
<td>0.02</td>
<td>0.12</td>
</tr>
<tr>
<td></td>
<td>Unknown*</td>
<td>sp.</td>
<td>Unknown</td>
<td>Unknown</td>
<td>0.06</td>
<td>0</td>
<td>0.12</td>
</tr>
<tr>
<td></td>
<td>Abbreviata*</td>
<td>sp.</td>
<td>Indirect1</td>
<td>Ingestion of arthropod</td>
<td>0.87</td>
<td>0.28</td>
<td>0.59</td>
</tr>
<tr>
<td>Nematoda</td>
<td>Capillaria</td>
<td>sp.</td>
<td>Unknown</td>
<td>Unknown</td>
<td>0.04</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>Trichuris</td>
<td>trichura</td>
<td>Direct</td>
<td>Ingestion of eggs</td>
<td>0.02</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>Strongyloides*</td>
<td>sp.</td>
<td>Direct</td>
<td>Ingestion of eggs/penetration of skin by larvae</td>
<td>0.22</td>
<td>0.06</td>
<td>0.35</td>
</tr>
<tr>
<td></td>
<td>Enterobius</td>
<td>sp.</td>
<td>Direct</td>
<td>Ingestion of eggs</td>
<td>0.16</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>Hookworm*</td>
<td>sp.</td>
<td>Direct</td>
<td>Penetration of skin by larvae</td>
<td>0.12</td>
<td>0.02</td>
<td>0.12</td>
</tr>
<tr>
<td></td>
<td>Unknown*</td>
<td>sp.</td>
<td>Unknown</td>
<td>Unknown</td>
<td>0.06</td>
<td>0</td>
<td>0.12</td>
</tr>
<tr>
<td></td>
<td>Abbreviata*</td>
<td>sp.</td>
<td>Indirect1</td>
<td>Ingestion of arthropod</td>
<td>0.87</td>
<td>0.28</td>
<td>0.59</td>
</tr>
<tr>
<td></td>
<td>Trichuris</td>
<td>trichura</td>
<td>Direct</td>
<td>Ingestion of eggs</td>
<td>0.02</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>Strongyloides*</td>
<td>sp.</td>
<td>Direct</td>
<td>Ingestion of eggs/penetration of skin by larvae</td>
<td>0.22</td>
<td>0.06</td>
<td>0.35</td>
</tr>
<tr>
<td></td>
<td>Enterobius</td>
<td>sp.</td>
<td>Direct</td>
<td>Ingestion of eggs</td>
<td>0.16</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>Hookworm*</td>
<td>sp.</td>
<td>Direct</td>
<td>Penetration of skin by larvae</td>
<td>0.12</td>
<td>0.02</td>
<td>0.12</td>
</tr>
<tr>
<td></td>
<td>Unknown*</td>
<td>sp.</td>
<td>Unknown</td>
<td>Unknown</td>
<td>0.06</td>
<td>0</td>
<td>0.12</td>
</tr>
<tr>
<td></td>
<td>Abbreviata*</td>
<td>sp.</td>
<td>Indirect1</td>
<td>Ingestion of arthropod</td>
<td>0.87</td>
<td>0.28</td>
<td>0.59</td>
</tr>
<tr>
<td></td>
<td>Trichuris</td>
<td>trichura</td>
<td>Direct</td>
<td>Ingestion of eggs</td>
<td>0.02</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>Strongyloides*</td>
<td>sp.</td>
<td>Direct</td>
<td>Ingestion of eggs/penetration of skin by larvae</td>
<td>0.22</td>
<td>0.06</td>
<td>0.35</td>
</tr>
<tr>
<td></td>
<td>Enterobius</td>
<td>sp.</td>
<td>Direct</td>
<td>Ingestion of eggs</td>
<td>0.16</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>Hookworm*</td>
<td>sp.</td>
<td>Direct</td>
<td>Penetration of skin by larvae</td>
<td>0.12</td>
<td>0.02</td>
<td>0.12</td>
</tr>
<tr>
<td></td>
<td>Unknown*</td>
<td>sp.</td>
<td>Unknown</td>
<td>Unknown</td>
<td>0.06</td>
<td>0</td>
<td>0.12</td>
</tr>
<tr>
<td></td>
<td>Abbreviata*</td>
<td>sp.</td>
<td>Indirect1</td>
<td>Ingestion of arthropod</td>
<td>0.87</td>
<td>0.28</td>
<td>0.59</td>
</tr>
<tr>
<td></td>
<td>Trichuris</td>
<td>trichura</td>
<td>Direct</td>
<td>Ingestion of eggs</td>
<td>0.02</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

*Indicates that the parasite met the criteria for incorporation into Cox proportional hazards models.

\* Prevalence was calculated from triplicate samples collected prior to treatment, a single post-treatment sample (protozoa; \(N = 45\)) and triplicate samples collected at the end of the study.

\* Species identifications are putative based on size and morphological characteristics of cysts.
Measures of social centrality were more important than individual host traits (e.g. sex, age and dominance) or physiological stress for explaining rates of infection with helminth parasites in mangabeys. Specifically, we found that individuals with more associates (centrality) that were well connected (closeness) and that had a tendency to switch groups (betweenness) were at higher risk of acquiring helminth parasites. Number of associations (strength) and measures of connectivity within contact networks were not associated with infection risk. Acquisition of protozoan infections, which do not tend to aggregate in populations, did not vary according to centrality, stress or host traits. Together, our results show that parasite aggregation was determined primarily by host associations and that social connectivity, rather than the immunosuppressive effect of stress, may explain enhanced infection risk.

The probability of acquiring new helminth infections was higher in animals that were central in the proximity network. This finding demonstrates a potential cost associated with social connectivity. A growing body of evidence suggests that social centrality increases risk of infection from macroparasites. In primates, centrality within grooming networks has been indirectly associated with nematode infections in female Japanese macaques, *M. fuscata yuki*, and directly associated with parasite species richness in brown spider.
monkeys, *Ateles hybıridus* (MacIntosh et al., 2012; Rimbach et al., 2015). Our findings differ from studies of brown spider monkeys, however, in which centrality in contact networks was more important than proximity for explaining elevated infection risk (Rimbach et al., 2015).

Helminths must develop in the external environment for days to months before they become infective. Risk associated with connectivity in proximity networks, rather than contact networks, may therefore reflect increased exposure to infectious life stages persisting on forrnites and food items (Freeland, 1980). We cannot rule out the possibility that spatial associations serve as proxies for contact (Farine, 2015). However, this is unlikely given that grooming networks showed a more heterogeneous structure (Fig. 1) despite being based on fewer observations. Indeed, space sharing has been identified as an important predictor of risk for infection from macroparasites in other taxonomic groups (Fenner, Godfrey, & Bull, 2011; Godfrey, Moore, Nelson, & Bull, 2010; Perkins, Cagnacci, Stradiotto, Arnoldi, & Hudson, 2009).

We found that protozoans, which are immediately infective once shed into the environment, were acquired uniformly across the population. This result aligns with our current understanding of the transmission biology of protozoan parasites, which tend not to be important than proximity for explaining elevated infection risk (Rimbach et al., 2015).

We did not find an effect of glucocorticoid level on acquisition of parasites. Similarly, in female Japanese macaques, social contact was determined to be more important than the immunosuppressive effects of stress in mediating the relationship between dominance and parasite infection (MacIntosh et al., 2012). Parasitism in white-handed gibbons, *Hylobates lar*, and black howler monkeys, *Alouatta pigra*, is also not affected by faecal cortisol (Gillespie, Barelli, & Heistemann, 2013; Martinez-Mota, 2015), although positive associations between parasitism and faecal cortisol have been documented in other primate species (Arlet et al., 2015; Foerster, Kithome, Cords, & Monfort, 2015; Muehlenbein, 2006).

Our results show that the nature of connections within a network affect infection risk. In our study, dominant males had stronger relationships in proximity networks, and females, dominant animals and adults tended to have higher connectivity in grooming networks. However, none of these centrality measures were associated with parasite acquisition. Attempts to link transmission of pathogens to certain host traits based on centrality metrics alone should therefore be interpreted with caution. Furthermore, risk associated with social connectivity is not likely to be static, but rather can vary over time (Rushmore et al., 2013). Heterogeneities in social connectivity, and resulting changes in infection over time, may therefore obscure the relationship between social network position and individual infection risk in cross-sectional studies.

### Conclusions

In our study population of Nigerian red-capped mangabeys, animals central in the social network had a higher probability of acquiring macroparasites than did peripheral individuals. Individuals with high centrality may facilitate transmission throughout the population, perhaps acting as ‘super spreaders’ (Lloyd-Smith, Schreiber, Kopp, & Getz, 2005). We found no direct associations between intrinsic host traits and time to infection, which concords with the result of previous studies. In fact, individual predictors of parasite aggregation (e.g. sex, age and dominance) documented in previous studies may be confounders of the direct relationship between social connectivity and infection risk. Overall, our results suggest that being central in a social network confers costs in terms of infection risk. Variation in social networks structure and dynamics should be considered in studies of infection risk in social species.

### Acknowledgments

We thank Festus Onajde, Rosemary Chegbage and CERCOPAN staff for their assistance with data collection and support in the field, and Mason Saari, Kelsey Brown, Nicholas Segel and Julia Slezak for their assistance in the lab. We also thank Dan Wittwer for his help with hormone assays, Dr Tim Yoshino for his assistance with parasite identifications, Dr Dorte Dopfer for her input on statistical analyses, and Bill Rohde for his careful edits to this manuscript. This research was funded by the Fulbright International Educational Exchange, the National Science Foundation's.
References


