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Primate reinfection with gastrointestinal parasites: behavioural and physiological predictors of parasite acquisition

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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.
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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, Cremer, & 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; Møller, 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

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testosterone are unclear (Zuk & McKean, 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, & Snaith, 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 and have short prepatent periods, we collected post-treatment faecal samples at the highest frequency for the month immediately following treatment (ca. every 3 days/individual). 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.

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