



Aggression and hormones are associated with heterogeneity in parasitism and parasite dynamics in the brown mouse lemur



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Animal behaviours, like aggression, can directly affect host health by influencing exposure to parasites. Aggressive individuals may experience an increase in agonistic interactions and contact rates with conspecifics, which might increase their probability of acquiring parasites. However, aggression is not the only factor that shapes parasitism; proximate mechanisms like hormone-modulated immunosuppression can also have broad impacts. Here, we hypothesized that high levels of aggression, cortisol and testosterone would be positively associated with parasitism and that aggressive individuals would play a larger role spreading parasites to conspecifics than would docile individuals. We measured aggression using the level of aggressive response to human handling during capture. Our aim was to examine associations between aggression and hormones (cortisol and testosterone) on variation in endo- and ectoparasitism in a population of wild mouse lemurs (*Microcebus rufus*) over a 3-year period. By tracking the movement of lice (directly transmitted parasites) in the population, we also examined the effect of host aggression on population-wide parasite dynamics. We show that animals with high testosterone and cortisol were more likely to exhibit aggressive behaviours, and cortisol was associated with significantly higher ectoparasite infestations. Aggressive individuals were significantly more infested by lice, and also donated significantly more lice to conspecifics in the population. Taken together, our results offer insight into the individual and population health costs of aggression, and empirical support of a trade-off between aggression and ectoparasitism, which may have driven the evolution of aggression and interactions with conspecifics.

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The drivers of parasitism in host populations can often be complex. For example, changes in host hormone levels can trigger aggressive behaviours that may be advantageous, leading to the exploration and discovery of novel resources or mates (Wilson, Clark, Coleman, & Dearstyne, 1994); however, aggression also has the potential to decrease fitness, by increasing contacts with conspecifics and creating opportunities for exposure to parasites (Biro, Abrahams, Post, & Parkinson, 2004; Boon, Réale, & Boutin, 2008; Jonsson, Koskela, & Mappes, 2000; Sih, Bell, & Johnson, 2004). Hormones may also affect parasitism by decreasing immunity (Cox & John-Adler, 2007; Folstad & Karter, 1992; Klein, 2004; Roberts,

Buchanan, & Evans, 2004). Therefore, an understanding of the bidirectional feedback between hormones, aggression and parasitism is necessary to determine how these factors shape host populations.

Androgens such as testosterone have long been associated with behaviour (Carere, Groothuis, Möstl, Daan, & Koolhaas, 2003; Koolhaas et al., 1999; Kralj-Fišer, Scheiber, Blejec, Moestl, & Kotrschal, 2007; Sellers, Mehl, & Josephs, 2007), for example, mediating aggression, reproduction and agonistic interactions in birds (Klein, 2000; Partecke & Schwabl, 2008; Wingfield, Ball, Dufty, Hegner, & Ramenofsky, 1987). Research suggests that positive associations between androgens and aggression may also involve heightened hypothalamic–pituitary–adrenal (HPA)-axis activity and elevated glucocorticoid levels, as they can be correlated (Liptrap & Raeside, 1978). Glucocorticoids such as corticosterone and cortisol are metabolic hormones that mediate energy in the face of social or physical environmental challenge (Beehner &

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Bergman, 2017; Sapolsky, Romero, & Munck, 2000). A study by Muller and Wrangham (2004) found that elevated cortisol levels in dominant male chimpanzees, *Pan troglodytes*, were associated with aggression and likely due to the high metabolic costs of aggressive displays (although they also acknowledge that aggression may be stressful in and of itself). In turn, aggression can also increase glucocorticoid levels directly, while decreasing testosterone levels, as found in Australian water dragons, *Intellagama lesueurii* (Baird, Lovern, & Shine, 2014), possibly reflecting the immunosuppressive effects of HPA-axis energy mobilization. It has thus been suggested that the stress response (as reflected in glucocorticoid levels) and androgen levels may underlie individual variation in aggression (Korte, Koolhaas, Wingfield, & McEwen, 2005).

Hormones may also directly influence heterogeneities in parasitic infections, by altering immunity (Cox & John-Adler, 2007; Folstad & Karter, 1992; Klein, 2004; Roberts et al., 2004). The immunocompetence handicap hypothesis proposes that costly testosterone enhances the expression of sexual traits but suppresses immune function (Folstad & Karter, 1992). In several bird species it has also been shown that elevated testosterone can suppress both adaptive and innate immune responses (Müller et al., 2005; Navara, Hill, & Mendonça, 2006; Tobler, Hasselquist, Smith, & Sandell, 2010), increasing the likelihood of parasitism. Cortisol may also influence parasitism by suppressing immune function in a trade-off with other energetic needs (Elenkov & Chrousos, 1999; Webster, Tonelli, & Sternberg, 2002).

Not only can host hormones play a role in parasite infestation, but parasites can affect hormone signalling within the host. Studies suggest that protozoan parasites can alter hormone concentrations in their hosts by disrupting reproductive cycles or suppressing androgen concentrations (Aina, Folashade, & Modupe, 1990; Barthelemy, Gabrion, & Petit, 2004). Whether these hormonal changes following infection are mediated by the parasite or the host remains widely unconfirmed (Klein, 2004). Host hormone manipulation by parasites is reported in both vertebrate and invertebrate hosts and is hypothesized to increase the availability of host resources for parasite growth and development (Baudoin, 1975; Larralde, Morales, Terrazas, Govezensky, & Romano, 1995; Morales et al., 1996; Romano, Valdéz, Cartas, Gómez, & Larralde, 2003).

The feedback between hormones, aggression and parasitism are complex, and an understanding of the interplay between these components is crucial (Boyer, Réale, Marmet, Pisanu, & Chapuis, 2010; Easterbrook, Kaplan, Glass, Pletnikov, & Klein, 2007; Natoli et al., 2005; Wilson et al., 1994). Here, we aim to examine the associations between hormones, aggression and parasitism in the hopes of better elucidating these feedbacks, which may be used to determine the directionality of these variables in future studies. One unique component here is that we also examine how individual aggression can affect parasitism in conspecifics in the population.

In this study, we evaluated associations between aggression, testosterone and cortisol levels and ecto- and endoparasite infestation, to test the hypothesis that aggression and hormones are positively associated with an increase in parasitism. We also hypothesized that aggressive individuals would spread more parasites to conspecifics than would docile individuals. We conducted this study in a wild population of brown mouse lemurs, *Microcebus rufus*, small, arboreal, trappable primates, in the southeastern rainforests of Madagascar. Previous studies on captive and wild mouse lemur populations have used detailed assays to classify mouse lemur behaviour (Dammhahn & Almeling, 2012; Dammhahn, 2012; Verdolin & Harper, 2013). However, those methods are often time-intensive and therefore limit the number of individuals that can be tested. In this study, we developed a rapid

assay to classify behavioural scores from all captured lemurs during all capture events, allowing us to parse out temporal or seasonal trends in aggression in recaptured individuals. Such rapid assessments are a fairly common practice in the animal behaviour literature and their results are often informative (Blumstein, Petelle, & Wey, 2012; Keiser & Pruitt, 2014; Réale & Festa-Bianchet, 2003). Our aim in this study was to assess the potential fitness consequences of aggression, through its associations with hormones and parasitism and parasite dynamics.

METHODS

Ethical Note

All international, national and institutional guidelines for the care and use of animals were followed. Research adhered to the ASAB/ABS Guidelines for the use of animals in research. All research protocols were approved by the government of Madagascar. Sample collection in Ranomafana National Park was approved by Madagascar National Parks under permit numbers 115/10 MEF/SG/DGF/DCB.SAP/SCBSE 96 and 215/08 MEFT/SG/DGEF/DSAP/SSE. Research protocols were also reviewed and approved by the University of Helsinki's institutional animal use rules and regulations board, and the Stony Brook University Institutional Animal Care and Use Committee (IACUC ID 2009-1608 and IACUC 2007-1597). Animals were captured only briefly and returned to the wild immediately after data collection.

Study Species, Site and Trapping

We established a long-term trapping system in the southeastern rainforests of Madagascar to study brown mouse lemurs at Ranomafana National Park (47°18'–47°37'E, 21°02'–21°25'S) (Atsalis, 1999). From 2008 to 2010 we used a systematic trapping grid, where 50 Sherman traps (XLR, Sherman Traps, Inc., Tallahassee, FL, U.S.A.) were set in pairs along transects in two sites, A and B. Transects were 1.5–2 km long and were separated by the Namorona River. Traps were set 2 m off the ground, baited with banana and checked 4 h later for captures. Our sampling period spanned the end of the cold season to the beginning of the rainy season, including the mouse lemur breeding season (August–December), for a total of 239 trap-nights (177 in 2008 and 2009, and 62 in 2010). Captured mouse lemurs were individually scanned (using an AVID Powertracker VI) for a microchip (FECAVA Eurochips, Vetcare, FI), sexed, weighed, aged (Zohdy et al., 2014) and measured under red light conditions, and then released into the forest the same evening at their capture location. Individuals without a microchip were given one (FECAVA Eurochips, Vetcare, FI). Nonprimate captures were released on site.

From 2008 to 2010, aggression assays were conducted on all captured individuals at every capture event to determine scores of aggression and docility.

We examined hormones, behavioural scores and parasitic infestation in 104 unique mouse lemurs, of which 63 were males (60.6%). We recorded 549 successful capture events, of which 445 (81.1%) were recaptures. The animal with the highest number of recaptures was a male who was captured 32 times during the study period (Fig. 1). The lowest trapping success occurred in August (46 captures, 8.3%), and October had the highest trapping success (246 captures, 44.8%).

Behavioural Assay

We developed a behavioural assay to quantitatively assign each lemur a score from 0 to 4, according to their behavioural response

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