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Predictors of helminth parasite infection in female chacma baboons (*Papio ursinus*)

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ABSTRACT

Helminth parasite infection can impose major consequences on host fitness. Several factors, including individual characteristics of hosts, environmental conditions, and patterns of coinfection, are thought to drive variation in parasite risk. Here, we report on four key drivers of parasite infection—phase of reproduction, steroid hormone profiles, rainfall, and patterns of coinfection—in a population of wild female chacma baboons (*Papio ursinus*) in South Africa. We collected data on reproductive state and hormone profiles over a 3-year span, and quantified helminth parasite burdens in 2955 fecal samples from 24 female baboons. On a host level, we found that baboons are sensitive to parasite infection during the costliest phases of the reproductive cycle: pregnant females harbored higher intensities of *Protospirura* eggs than cycling and lactating females; lactating and cycling females had a higher probability of *Oesophagostomum* infection than pregnant females; and cycling females exhibited lower *Trichuris* egg counts than pregnant and lactating females. Steroid hormones were associated with both immunoenhancing and immunosuppressive properties: females with high glucocorticoid concentrations exhibited high intensities of *Trichuris* eggs but were at low risk of *Oesophagostomum* infection; females with high estrogen and progestagen concentrations exhibited high helminth parasite richness; and females with high progestagen concentrations were at high risk of *Oesophagostomum* infection but exhibited low *Protospirura* egg counts. We observed an interaction between host reproductive state and progestagen concentrations in infection intensity of *Protospirura*: pregnant females exhibited higher intensities and non-pregnant females exhibited lower intensities of *Protospirura* eggs with increasing progestagen concentrations. At a population level, rainfall patterns were dominant drivers of parasite risk. Lastly, helminth parasites exhibited positive covariance, suggesting that infection probability increases if a host already harbors one or more parasite taxa. Together, our results provide a holistic perspective of factors that shape variation in parasite risk in a wild population of animals.

1. Introduction

Infection with parasites can be costly to host organisms (Gulland 1995; Morales-Montor et al., 2004; Cooper et al., 2012; Akinyi et al., 2019). Parasites deplete energy and nutrients required by hosts to fight infection and elicit immune defenses (Coop and Holmes 1996; Koski and Scott 2001; Colditz 2008). Consequently, parasite infections have been linked to reductions in survival and reproduction, and increased susceptibility to infections with other parasites (e.g., Jolles et al., 2008; Ezenwa et al., 2010; Nguyen et al., 2015; Schneider-Crease et al., 2017; Budischak et al., 2018; Akinyi et al., 2019). Several factors, including individual characteristics of hosts, social interactions between hosts, characteristics of the parasites themselves, and environmental conditions, are thought to shape host-parasite interactions (Seppälä et al.,

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2008; Mostowy and Engelstädter 2011; Griffin and Nunn 2012; Fairbanks and Hawley 2012; Habig and Archie 2015; Rimbach et al., 2015; Rushmore et al., 2017; Habig et al., 2018). Because infection with parasites is potentially costly, identifying key drivers of infection is important for revealing the selection pressures and constraints that shape patterns of parasitism, and for drawing inferences about the causes of individual variation in infection risk. Here, we use a uniquely detailed dataset to analyze four key drivers of gastrointestinal parasite infection in a wild population of chacma baboons (*Papio ursinus*): stage of reproduction, steroid hormone profiles, rainfall, and patterns of coinfection.

Reproduction has been shown to be an energetically costly period of life for females, characterized by tradeoffs between reproductive effort and immunity and mediated by steroid hormones (Nordling et al., 1998; Archie et al., 2014; Leivesley et al., 2019). Moreover, some stages of reproduction are associated with higher exposure and susceptibility to parasite infection (Müller-Graf et al., 1996; Ravasi 2009; East et al., 2015; Albery et al., 2020). For example, some studies of nonhuman primates have found that females harbor more parasites during their monthly ovulatory cycles compared to other phases of reproduction (e.g., yellow baboon, *Papio cynocephalus*: Hausfater and Watson 1976; Meade 1984). Likewise, research on humans suggests a shifting reproduction-immunity tradeoff: infection susceptibility is heightened during ovulatory cycling, particularly when progesterone concentrations are elevated in the host (Alvergne and Tabor 2018).

Pregnancy is another energetically costly component of a female's life because the mother is providing resources for herself and her fetus (Lloyd 1983). Because of the energetic costs associated with pregnancy, immune function may be compromised, and females may be at increased risk of parasite infection (Blackwell et al., 2015). In support of this idea, several studies have reported a positive association between pregnancy and parasite infection (e.g., yellow baboon, *P. cynocephalus*: Meade 1984 but see Hausfater and Watson 1976; mandrill, *Mandrillus sphinx*: Setchell et al., 2007; small ruminants: Mideksa et al., 2016). For example, in an experimental field study, reindeer (*Rangifer tarandus plathyrynchus*) that were provided antihelminthic treatment were more likely to be pregnant than untreated controls (Albon et al., 2002). Despite such findings, other studies have demonstrated an enhancement of female immune function during pregnancy (e.g., Mor and Cardenas 2010; Mor et al., 2011). Indeed, research suggests that pregnancy favors immune responses that promote increased levels of Th-2 cytokines such as IL-4, IL-6, and IL-10, which provide humoral immunity against helminth parasites (Mosmann and Sad 1996; Vargas-Villavicencio et al., 2009).

Lactation requires more energy than any other stage of reproduction; hence, a tradeoff exists between the reproductive effort associated with lactation and the ability to resist parasite infection (Pond 1977; Künkele 2000). Following infection, lactating females experience metabolic stress and reduced energy balance, which further exacerbates parasite proliferation (East et al., 2015). Indeed, studies of females during lactation have shown that the experimental removal of parasites results in both increased milk production and offspring survival (Hillegass et al., 2010; Patterson et al., 2013). Moreover, in an experimental study of laboratory rats, the reduction of litter size during lactation resulted in an 83% reduction in worm burdens (Jones et al., 2012). Hence, while reproduction in general imposes costs during a female's life, some stages of the reproductive cycle are more energetically costly than others and thus more likely to increase susceptibility to parasite infection and proliferation.

Evidence indicates that steroid hormones, such as glucocorticoids, progesterone, and estrogen, also influence susceptibility to and the course of parasitic infections (Romano et al., 2015). Indeed, steroid hormones have been found to mediate parasitic infections by influencing the host's immune system and by either promoting or inhibiting parasite reproduction (Klein 2004; Tait et al., 2008; Vargas-Villavicencio et al., 2009; Defolie et al., 2020). Because glucocorticoids function in part to mobilize energy in response to environmental challenges, these metabolic hormones are among the most well-studied among animals (Schwarzenberger 2007; Beehner and Bergman 2017). Like other steroid hormones, glucocorticoids exhibit both immunoenhancing and immunosuppressive effects (Sapolsky et al., 2000). When animals are exposed to adverse environmental stimuli, physiological costs are reflected in elevated glucocorticoid concentrations (Creel et al., 2013). The prolonged secretion of glucocorticoids diverts energetic resources from essential activities, such as immune function, and is thought to increase susceptibility to parasitic infection (Nava-Castro et al., 2011). In support of this causal relationship, experimental studies have shown that exogenous administration of glucocorticoids promotes the proliferation of many parasites including *Entamoeba histolytica* (Carrero et al., 2006), *Cryptobia salmonistica* (Li et al., 2013), and *Taenia crassiceps* (Hinojosa et al., 2012).

In female mammals, two sex hormones secreted by the ovaries — progesterone and estrogen — are thought to yield varying degrees of influence on the course of parasitic infection (Morales-Montor et al., 2004). Progesterone can exert both stimulatory and inhibitory effects on the immune system but is typically considered as immunosuppressive (Klein 2004). For example, experimental treatment with exogenous progesterone has been found to promote the proliferation of *Plasmodium falciparum* (Lingnau et al., 1993). Contrary to this finding, an experimental study found that in vitro administration of progesterone significantly decreased egg production of *Schistosoma mansoni* (Morrison et al., 1986). Like progesterone, estrogen can also exhibit both immunoenhancing and immunosuppressive effects on parasites (Morales-Montor et al., 2004; Foo et al., 2017). For example, experimental studies of exogenous treatment with estrogen have yielded mixed results, in some cases inhibiting the proliferation of parasites (e.g., *Strongyloides venezuelensis:* Rivero et al., 2002) and in other cases facilitating parasite proliferation (e.g., Toxoplasma gondii: Pung and Luster 1986; *Taenia crassiceps*: Morales-Montor et al., 2002). These differences are thought to be mediated by an interplay between host immune activation, host hormonal environment, and the ability of individual parasite species to alter host immune and endocrine pathways (Romano et al., 2015). Strikingly, a meta-analysis found that exogenous treatment with estrogen has mostly immunoenhancing effects as evidenced by overall reduced parasite loads; however, in this study, endoparasites and ectoparasites were not assessed separately (Foo et al., 2017). Overall, these contradictory results suggest that sex hormones vary in their capacities to yield influence on parasitic infection and that the individual traits of parasite themselves define the intensity of infection (Klein 2004). Whether progesterone and estrogen mediate parasitism in wild populations of animals remains largely unknown.

Beyond individual characteristics of hosts, environmental factors such as patterns of rainfall can influence host exposure and susceptibility to parasitism. Survival of infective helminth stages in the environment can depend on humidity and substrate, and many soil-transmitted helminths therefore show increased abundance of infective stages during rainy seasons; this may result in increased host exposure to parasites (Nunn and Altizer 2006). Accordingly, some studies of wild populations of animals have reported higher risk of parasitism during rainy periods (e.g., red fox, *Vulpes vulpes*: Miterpáková et al., 2006; mandrill, *M. sphinx*: Setchell et al., 2007; black and white colobus, *Colobus guereza*: Chapman et al., 2010). During dry periods, on the other hand, nutritional and thermal stress may reduce host immune function and in turn increase host susceptibility to parasitism (Ezenwa 2004). In support of this association, other studies have reported higher risk of parasitism during dry periods (e.g., Asian elephant, *Elephas maximus*: Vidya and Sukumar 2002; yellow baboon, *P. cynocephalus*: Akinyi et al., 2019; Habig et al., 2019; Grant's gazelle, *Nanger granti*: Shearer and Ezenwa 2020). Thus, the direction and magnitude of environmental effects on levels of parasitism vary within and between populations and species and are largely a function of host and parasite ecology.

Finally, wild animals are often infected with multiple parasite species, and interactions between parasites and their hosts can either

facilitate or inhibit coinfection with other parasites (Viney and Graham 2013; Wilcox et al., 2015). If hosts are exposed to infective stages of parasites that cooccur in the same environment contemporaneously, then hosts are more likely to experience coinfection (e.g., Mwangi et al., 2006; Viney and Graham 2013). Moreover, if infection with one parasite compromises immunity or damages host tissue, then hosts are at higher risk of coinfection (e.g., Ezenwa et al., 2010; Telfer et al., 2010). On the other hand, if infective stages of parasites differ spatially or temporally, then hosts are less likely to experience coinfection (e.g., Kelly-Hope et al., 2006; Warburton et al., 2016). Likewise, if one parasite species competitively excludes another parasite species (e.g., Holmes 1961; Stancampiano et al., 2010) or if infection with one parasite reduces host susceptibility to other parasites (e.g., Bentwich et al., 1999; Jolles et al., 2008), then hosts are expected to have reduced risk of coinfection. Thus, patterns of coinfection are driven by multiple factors including host exposure to and susceptibility to different parasite species.

Chacma baboons are an ideal species in which to study parasite-host dynamics in wild animals for several reasons. First, because of their generalist diet and occupation of a wide range of habitats, chacma baboons are exposed to a variety of parasite taxa (Appleton et al., 1991; Ravasi et al., 2012; Drewe et al., 2012). Second, because chacma baboons reside in multimale, multifemale groups where reproduction occurs year-round, and where both males and females mate with multiple partners (Weingrill et al., 2004; Alberts and Altmann 2006), their mating and social systems likely shape patterns of exposure and susceptibility to parasites (Benavides et al., 2012). Lastly, female chacma baboons, like all baboons, exhibit visible sexual swellings around the time of ovulation, making it possible to reliably determine their reproductive stage and to combine these observations with noninvasively determined fecal hormone and parasite profiles (Weingrill et al., 2004; Gillespie 2006; Gesquiere et al., 2019). Thus, by quantifying parasite infection of chacma baboons, we can make inferences about physiological, reproductive, and environmental drivers of parasite risk and elucidate selective pressures shaping reproductive ecology in these and other populations of animals.

Given the complexity of host-parasite interactions, as well as host and parasite biology, it is important to consider differences among helminth taxa when making inferences about the causes and consequence of helminth parasite infection. Two common genera of helminth parasites known to infect chacma baboons are *Trichuris* (whipworm) and *Oesophagostomum* (nodular worm) (Ravasi et al., 2012; Moxley 2013). These two genera of worms also commonly infect other baboon species (e.g., Guinea baboon, *Papio papio*: Ebbert et al., 2013; olive baboon: *Papio anubis*: Müller-Graf et al., 1996; Munene et al., 1998; yellow baboon, *Papio cynocephalus*: Akinyi et al., 2019; Habig et al., 2019). A third genus, *Protospirura* (stomach worm), is less common, but is known to infect olive baboons in wild populations in Uganda (Bezjian et al., 2008).

The whipworm (*Trichuris*) is a gastrointestinal parasite that mainly resides in the cecum and large intestine of the baboon host (Strait et al., 2012). This parasite has a direct life cycle: infection results when a baboon ingests an egg or larvae found on food or surfaces in its environment (Cogswell 2012; Strait et al., 2012). While light infections do not appear to cause lesions, heavy infestations are associated with elevated glucocorticoid hormones (Akinyi et al., 2019) as well as ulceration, mucoid diarrhea, and mortality (Pettifer 1984; Strait et al., 2012).

The nodular worm (*Oesophagostomum*) is regarded as the most debilitating gastrointestinal parasite for baboons (Pettifer 1984). Like *Trichuris*, *Oesophagostomum* has a direct life cycle. Transmission occurs when the baboon host ingests an infective third stage larvae found on food or surfaces in the environment (Cogswell 2012; Strait et al., 2012). Adult worms typically reside in the colon of their host (Cogswell 2012; Strait et al., 2012). Baboons infected with *Oesophagostomum* have been observed with tarry black nodules in their large intestines; severe infections are associated with lethargic behavior, weight loss, diarrhea, and mortality (Pettifer 1984; Strait et al., 2012).

The stomach worm (*Protospirura*) is a spirurid nematode that infects the gastrointestinal tract of the primate host (Foster and Johnson 1939; Toft 1986). The parasite is transmitted when the baboon host ingests an intermediate arthropod host (Anderson 2000; Smales et al., 2009). While little is known about its pathogenicity in wild populations, heavy infection is associated with mortality in captive primates (Foster and Johnson 1939; Ruch 1959).

The goal of this study was to understand the patterns and drivers of gastrointestinal parasitism in wild female chacma baboons living in the Tokai Forest in South Africa. To accomplish our goal, we collected data on female parasite burdens, reproductive states, hormone profiles, and rainfall over a three-year period. Our specific objectives were to test whether reproductive state, steroid hormone concentrations (glucocorticoids, progestagen, and estrogen), and patterns of rainfall were predictive of female infection, and whether infection with one parasite increased or decreased the probability of infection with other parasites (see Fig. 1 for specific predictions). We focus on females because we were specifically interested in the fitness consequences of parasitism in the context of female reproductive physiology and endocrinology. Together, our results provide a holistic perspective on the drivers of parasitism in the context of reproduction, physiology, and environmental conditions.

2. Methods

2.1. Study site and population

The Tokai Forest, i.e., Tokai section of Table Mountain National Park (34°03′17 S, 18°23′59 E), is located approximately 20 km south of Cape Town, South Africa. This area contains the home ranges of four groups of chacma baboons (*P. ursinus*) in a mosaic of exotic pine and eucalyptus forest, indigenous fynbos, and suburban and commercial agricultural areas, mainly vineyards (Chowdhury et al., 2020). The study subjects were 24 adult female chacma baboons residing in the largest of the four groups, the Main Tokai troop 1 or MT1 troop. Data collection spanned from August 2012 to July 2015. All methods complied with the laws of South Africa and the International Primatological Society guidelines for the ethical treatment of research subjects, and all protocols were approved by the Queens College Institutional Animal Care and Use Committee (protocol #132).

2.2. Parasitological sample collection and analyses

We opportunistically collected fecal samples (n = 2955) from 24 individually identified females in the MT1 troop. Each sample was subdivided, with one portion used for parasitological analyses and the other portion for endocrinological analyses (see below). The portion used for parasitological analyses was deposited into a pre-prepared tube with 10% buffered formalin saline solution and was stored at room temperature until transport to the laboratory. The collection of fecal samples for quantifying parasite ova has been found to be an effective and humane method for measuring parasite burdens noninvasively (e.g., Akinyi et al., 2019; Habig et al., 2019). Several studies of mammals have confirmed a positive correlation between adult worm burden and fecal egg output (Stear et al., 1995 [$r = 0.63$; $P < 0.01$]; Gassó et al., 2015 [$R^2 = 0.68$–0.86; $P < 0.001$]; Byrne et al., 2018; [$R^2 = 0.29$–0.38; $P < 0.01$]). Therefore, we estimated parasite burdens non-invasively by counting eggs in fecal samples.

All parasitology work was conducted by Colleen Archer at the Parasitology Diagnostic Laboratory in Durban, South Africa. To extract helminth parasite ova from fecal samples, we used a modified formal-ether sedimentation technique (Allen and Ridley 1970). Using a clean wooden applicator stick, we emulsified each sample in the formal-saline solution used for its collection. We then filtered them individually through a small plastic mesh tea-strainer into a 100 mL plastic beaker. The filtrate in the 100 mL beaker was decanted into a 15 mL plastic, conical test-tube (Falcon tube), appropriately labelled with the animal's

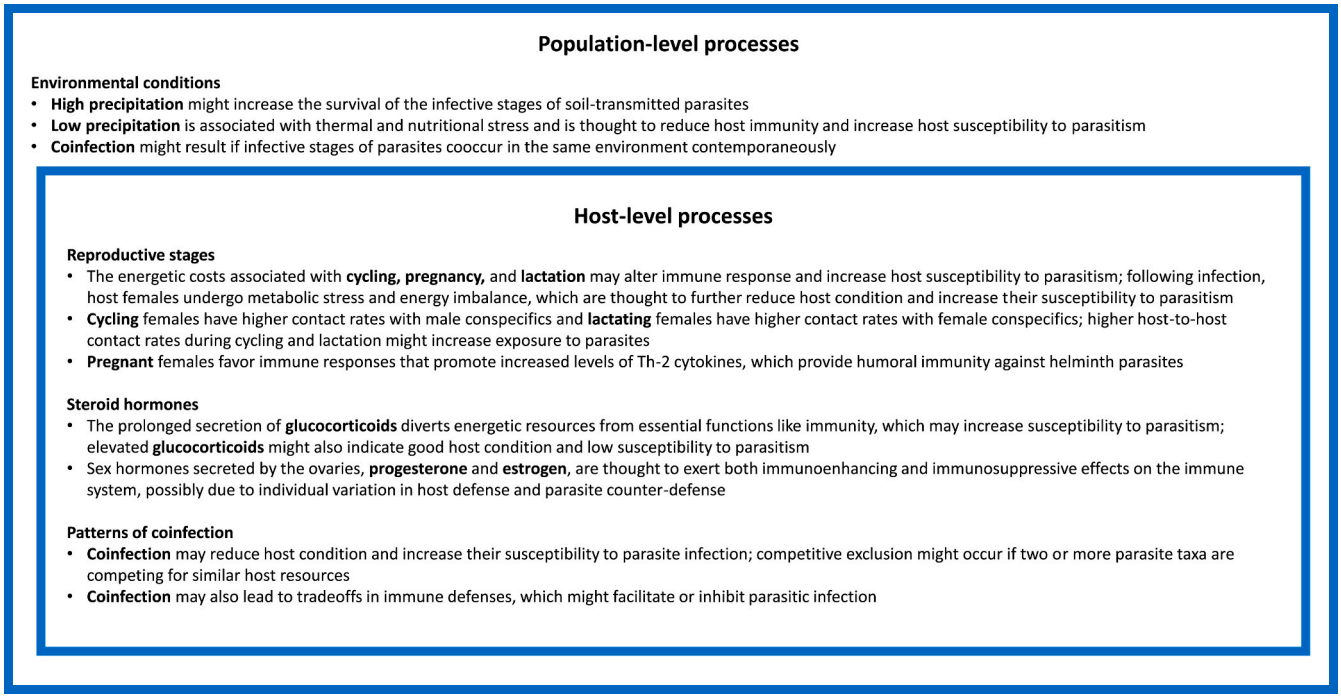

Fig. 1. Population and host level processes proposed to drive within group variation in helminth infection risk among female chacma baboon hosts (partially adapted from Akinyi et al., 2019; Habig et al., 2019). Four key drivers of parasite risk are examined: environmental conditions; reproductive stage; steroid hormones; and patterns of coinfection.

identification number, and topped up to the 7 mL mark with formal-saline. The large debris retained in the strainer was discarded and the strainer well washed for reuse. We added 4 mL diethyl-ether to the tube, which was then sealed with a rubber bung and shaken well for 60 s. Next, we removed the bung and centrifuged the suspension at 2000 rpm (675 g-force) for 6 min. The Falcon tube then contained a pellet deposited at the bottom of the tube, a layer of formalin above it, a ring of debris above that, and a layer of diethyl-ether on top. We used an applicator stick to loosen the plug of debris and poured all the layers off in one smooth movement, leaving only the pellet and a small amount of formalin in the test tube. Depending on the size of the pellet, we suspended it in one or more drops of saline (the thicker the pellet, the more it was diluted). We examined one or two drops of the pellet at a time on a microscope slide, covered with a 22 × 40 mm cover glass, until the whole pellet was examined. A compound light microscope was used to identify and quantify taxa based on their morphology, shape, and size (Gillespie 2006; Bowman 2014). We counted all nematode eggs and converted the count to eggs per gram (epg) using the recorded fecal mass of the collected sample.

2.3. Predictors of helminth parasite infection

We tested four categories of predictor variables: (1) reproductive state; (2) steroid hormone concentrations; (3) season; and (4) infection with other parasites (Table 1).

2.3.1. Reproductive states

We used field observations of behavior, sexual swellings, and changes in the perineal skin to identify specific reproductive stages of females (Shaikh et al., 1982; Dixson 2015; Gesquiere et al., 2019). (1) Cycling. We identified individual females as undergoing sexual cycling when we observed progressive inflation (turgesence) or deflation (deturgescence) of the perineal skin during daily observations (Shaikh et al., 1982; Dixson 2015; Gesquiere et al., 2019). (2) Pregnancy. We noted females as pregnant when we observed the paracallosal skin change from greyish black to pink accompanied with cessation of swelling followed by apparent weight gain (Altmann 1973). (3)

Table 1
Reproductive, endocrinological, ecological, and parasitological predictors of male parasite risk.

sample size = 2955 samples; 24 females	
Random effects	
individual identity	the identity of the subject contributing to the sample
Response variables	
helminth parasite richness	number of distinct helminth parasite taxa identified in a host sample
probability of Oesophagostomum infection	presence or absence of *Oesophagostomum* in a host sample
log Protospirura egg density	number of *Protospirura* eggs per gram identified in a host sample; log (eggs per gram + 0.5)
log Trichuris egg density	number of *Trichuris* eggs per gram identified in a host sample; log (eggs per gram + 0.5)
Predictor variables	
reproductive state	reproductive state on day of sampling (cycling, pregnant, or lactational amenorrhea)
Estrogens	fecal estrogen (ng/g) concentrations identified in a host sample
Glucocorticoids	fecal glucocorticoid (ng/g) concentrations identified in a host sample
Progestagens	fecal progestagen (ng/g) concentrations identified in a host sample
Season	*dry*: sample collected in month with ≤35 mm and ≤8 days of rainfall (November–March); *wet*: sample collected in month with >35 mm and >8 days of rainfall
Oesophagostomum infection [a]	presence or absence of *Oesophagostomum* in a host sample
log Protospirura intensity [a]	number of *Protospirura* eggs in a host sample; log (eggs per gram + 0.5)
log Trichuris intensity [a]	number of *Trichuris* eggs identified in a host sample; log (eggs per gram + 0.5)

[a] Note: presence or density of a parasite was not modeled as a predictor variable in cases where the parasite itself was modeled as the response variable.

Lactational amenorrhea. Females were identified as exhibiting lactational amenorrhea during the period following their infant's birth up to the time when they resumed cycling, characterized by the onset of sexual swelling (Gesquiere et al., 2019). When there were gaps in our observational data, we used the following protocols: gaps of <5 days with the same state before and after the missing observation were filled with the same state; gaps of <5 days with different states before and after the missing observation were filled equally with the two adjoining states (in cases where the gap was an odd number, the middle date was filled with the previous state; if the gap only included one day, the gap was filled with the previous state); gaps >5 days were coded as unknown. Of the total 2955 fecal samples collected, we could not reliably identify the female's reproductive state at the time of collection in 75 cases. Of the 2880 fecal samples for which reliable data were available for reproductive stage, 798 (27.7%) were collected from cycling females, 773 (26.8%) from pregnant females, and 1309 (45.5%) from females undergoing lactational amenorrhea.

2.3.2. Steroid hormone sample collection and analyses

Three endocrinological predictors of parasite risk were included in our analyses: (1) fecal glucocorticoid concentrations (ng/g); (2) fecal progestagen concentrations (ng/g); and (3) fecal estrogen concentrations (ng/g). For hormone analyses, we used a portion of the same fecal sample collected for parasitological analyses (see above). During sample collection, we removed excess debris from the sample, deposited the sample in a collection bag, and homogenized the sample by thoroughly kneading the sealed sample bag by hand. Samples were transferred to our field lab within about 5 h and dried on aluminum foil in an electric oven at ~95 °C for approximately 3 h. Following the drying process, samples were crushed and stored in airtight sample tubes in a −20 °C freezer following protocols previously validated by Foerster and Monfort (2010).

Processing of steroid hormone samples was conducted by Morgan Jackson in the Endocrine Research Laboratory at the Smithsonian Conservation Biology Institute in Front Royal, VA. Hormones were extracted by adding 5 mL of 90% ethanol to 0.2 ± 0.02 g of each fecal sample and then boiled in a 95 °C water bath for 20 min. Following the boiling process, extracts were centrifuged for 20 min at 2500 rpm. Following centrifugation, the extracts (supernatant) were transferred to new tubes. The precipitates were reconstituted in another 5 mL of 90% ethanol, vortexed for 30 s and then centrifuged for 15 min at 2500 rpm. The two resulting supernatants were combined and then dried under forced air. The resulting extracts were reconstituted in 1 mL of 100% ethanol, dried under forced air, and then reconstituted in 1 mL of preservative-free phosphate buffer (0.2 M NaH2PO4, 0.2 M Na2HPO4, 0.15 M NaCl; pH 7.0) via immersion in an ultrasonic water bath for 15 min. The average extraction efficiency, based on 1939 samples, was 77% (SD = 0.032).

(1) Glucocorticoids. To measure fecal glucocorticoid concentrations, we made 1:16 dilutions by adding 100 μL of neat sample to 1.5 mL of dilution buffer. We conducted assays with a ^{125}I double-antibody radioimmunoassay (MP Biomedicals, Orangeburg, NY) following protocols described in Wasser et al. (2000). Inter-assay coefficients of variation for high and low radioimmunoassay controls were 8.8% and 5.0% (n = 25). (2) Progestagens. For fecal progestagens, we made 1:250 dilutions by adding 250 μL of neat sample to 1 mL of dilution buffer. Samples were analyzed via enzyme immunoassay using a monoclonal progesterone antibody (Quidel CL425, C.J. Munro, University of California, Davis, CA). We used flat-bottomed, high-binding 96-well microtitre plates to adsorb antibodies in coating buffer (0.015 M Na2CO3; 0.035 M NaHCO3; pH 9.6) and incubated at 4 °C for 8 h. We washed the plates (0.05% Tween 20 in 0.15 M NaCl solution) five times, and then we loaded the plates with standards (0.05 mL progesterone) in triplicate, controls, and diluted samples in duplicate. We added 0.05 mL of horseradish peroxidase (HRP) solution to each well. The plates were then incubated at room temperature for 2 h. Following incubation, we washed the plates five times by adding 0.1 mL ABTS solution (0.04 M ABTS diammonium salt; 0.5 M H2O2 in 0.05 M citric acid buffer; pH 4.0) to each well. We read assays using a microplate reader (MRX, Dynex Technologies, Chantilly, VA) at 405 nm (ref. 490 nm) to an optical density (OD) of 1.0 (range 0.9–1.1) for the 0 ng/mL standard. The progesterone inter-assay coefficients of variation (CV) for high and low enzyme-immunoassay controls were 13.3% and 2.5% (n = 58). (3) Estrogens. To measure fecal estrogens, we made dilutions by mixing 30.6 μL of each neat sample to 0.20 mL of dilution buffer. We conducted enzyme immunoassays using a polyclonal antibody (R4972; C.J. Munro, University of California, Davis, CA) in coating buffer (0.015 M Na2CO3; 0.035 M NaHCO3; pH 9.6) adsorbed to flat-bottomed, high-binding 96-well microtitre plates and incubated at 4 °C for 8 h. We washed the plates (0.05% Tween 20 in 0.15 M NaCl solution) five times, and then loaded the plates with standards (0.02 mL β-Estradiol) in triplicate, controls, and diluted samples in duplicate. We added HRP solution, incubated and washed the plates, and read the assays using a microplate reader following the protocols described above for progestagen. The estrogen (estradiol) inter-assay coefficients of variation (CV) for high and low enzyme-immunoassay controls were 8.6% and 7.1% (N = 133).

The mean concentration per sample (n = 2955) for fecal glucocorticoids was 77.54 ng/g (range = 26.76–310.68 ng/g); for fecal progestagens 553.69 ng/g (range = 7.35–9801.81 ng/g), and for fecal estrogens 214.70 ng/g (range = 0.32–2084.40 ng/g).

2.3.3. Season

The Cape Peninsula of South Africa, where Tokai Forest is located, is characterized by a Mediterranean-like climate with cool, rainy winters and hot, dry summers (Cowling et al., 1996; Lawal 2015). For our analyses, we calculated average monthly precipitation and days of rainfall during the study period based on data obtained from a Cape Town weather station (Weather Station 688160; Latitude: −33.96; Longitude: 18.6). From these data, we defined a priori the dry season as months with ≤35 mm of rainfall and ≤8 days of rainfall (November to March) and the wet season as months with greater than 35 mm of rainfall and more than 8 days of rainfall (April to October). These "seasons" do not strictly adhere to the summer and winter seasons typically used to describe the Cape Town climate because our analyses span all months (including spring and autumn) instead of only the months with climatic extremes (summer and winter). Thus, for the 2955 samples used for this study, 1437 were collected during the "dry season" and 1518 were collected during the "wet season".

2.3.4. Infection with other parasites

Because existing infections with other taxa might influence individual parasite infection risk and parasite burden, we included the most common parasites (other than the taxon that was being modeled) as predictor variables in a given model (see below for more details): (1) *Oesophagostomum* (presence or absence of eggs); (2) *Protospirura* (eggs per gram fecal matter); and (3) *Trichuris* (eggs per gram fecal matter).

2.4. Statistical analyses

We used random intercept mixed effects models to explore predictors of specific helminth parasite infection in our study subjects. All statistical analyses were conducted using R version 4.03 (R Core Team 2020), and our response variables, predictor variables, and random effects are described in Table 1. For each model, female identity was included as a random effect. Four measures of parasitism were included as response variables (Table 1): (1) log-transformed *Trichuris* egg counts, (2) log-transformed *Protospirura* egg counts, (3) presence/absence of *Oesophagostomum*, and (4) helminth parasite richness. We log-transformed two of our response variables, *Trichuris* and *Protospirura*, to keep the residuals more normally distributed (Habig et al., 2019). Two response variables, log-transformed *Trichuris* egg counts and log-transformed *Protospirura* egg counts, were modeled using Gaussian error

distributions. One of our response variables, presence/absence of *Oesophagostomum*, was modeled using binomial error distributions because only 32.08% of the samples were infected with this parasite (Table 2). Finally, we modeled helminth parasite richness using Poisson error distributions. When reporting our results, we use the terms "risk" or "probability of infection" to describe binary results (i.e., *Oesophagostomum* presence/absence), and we use the terms "burden" or "intensity" when reporting egg density (epg) results (i.e., *Protospirura* and *Trichuris* egg density).

Because the effects of hormonal predictors on helminth infections may vary across reproductive stages, we also tested interactions between individual hormones and reproductive stage. Similarly, seasonal variation may interact with reproductive state to influence helminth infections, if being in a specific reproductive state (e.g., pregnancy or lactation) makes females more susceptible to infection, which could exacerbate infections at times of high helminth prevalence. In cases in which these interactions did not improve model fit (i.e., decrease in Akaike Information Criterion (AICc) by less than two units), we removed this parameter from our models.

To perform all mixed models, we used the packages *lme4* (Bates et al., 2014) and *lmerTest* (Kuznetsova et al., 2015). We performed multiple comparison tests of all possible parameter combinations using the *MuMIn* package (Bartoń 2009). In cases in which there were two or more parameter combinations with an AICc difference <2 from the best model, we performed model averaging using the summed weight method in Burnham and Anderson (2002, 2004). The model-averaged coefficients were calculated by conditional R^2 (Nakagawa and Schielzeth 2013). From this process, we generated four models, one for each of the four measures of parasitism. We calculated marginal effect sizes for these four models using the *ggeffects* package (Lüdecke 2018) holding covariates at their mean values. When calculating marginal effect sizes based on these four models, we modeled predictor variables as quantiles (e.g., yes vs. no; presence vs. absence for binary variables; low vs. medium vs. high for continuous variables). We used these categories for the purpose of visualization and to compare percent differences among subgroups in the results. Lastly, to diagnose multicollinearity, we quantified generalized variance inflation factors (GVIFs) for model predictor variables using the package *car*; this allowed for the assessment of both categorical and continuous variables (Fox and Weisberg 2011). Because all GVIFs were <2.5, we found no evidence of problematic multicollinearity (Fox 2015).

3. Results

3.1. Helminth parasite species, prevalence, and abundance

Of the 2955 fecal samples we collected from 24 females (mean of 123 samples per individual; range: 15–170) in the MT1 troop, we identified seven helminth taxa that varied in their prevalence and intensity (Table 2). The most common parasite taxon identified was *Trichuris*, which occurred in 74.1% of samples and 100% of females; other common parasite taxa included *Protospirura* (occurring in 67.9% of samples and 100% of females) and *Oesophagostomum* (occurring in 32.1% of the samples and 100% of the females). Four rarer taxa were identified in less than one percent of the samples (Table 2). The number of helminth parasite taxa per sample ranged from 0 to 4; the median parasite richness in each sample was two taxa. Overall, we identified at least one helminth taxon in 89.9% of the samples.

Table 2
Parasite prevalence in infected individuals, proportion of samples infected, and median egg count in infected samples (N = 2955 samples).

Parasite	Prevalence of infected females	Proportion of infected samples	Median Intensity (eggs per gram fecal matter)
Trichuris	100% (n = 24)	74.1% (n = 2189)	6 (1–230)
Protospirura	100% (n = 24)	67.9% (n = 2006)	7 (1–448)
Oesophagostomum	100% (n = 24)	32.1% (n = 948)	3 (1–402)
Strongyloides	16.7% (n = 4)	0.17% (n = 5)	1 (1–3)
Ternidens	12.5% (n = 3)	0.20% (n = 6)	1 (1–2)
Unidentified Strongyle-like	8.3% (n = 2)	0.14% (n = 4)	1 (1–8)
Ascaris	4.2% (n = 1)	0.034% (n = 1)	6 (one sample)

3.2. Reproductive cycle

Pregnancy and lactational amenorrhea were associated with higher risk of parasitism or parasite burden (Table 3). Pregnant females exhibited 40.6% higher *Protospirura* egg counts than females who were not pregnant, holding other predictors at their mean values (Fig. 2A). Specifically, pregnant females harbored higher *Protospirura* egg counts than cycling and lactating females (Tukey multiple comparison test: [pregnant vs. cycling: estimate: 0.246 or 1.8 eggs per gram, $P < 0.001$; pregnant vs. lactating: estimate: 0.360 or 2.3 eggs per gram; $P < 0.001$]). Lactating females were significantly more likely to be infected with *Oesophagostomum* than pregnant females (Tukey multiple comparison test [lactating vs. pregnant: estimate: 0.526; $P = 0.016$]). Lastly, we found that non-cycling females exhibited 131% higher *Trichuris* egg counts than cycling females (Fig. 3A). Specifically, cycling females exhibited significantly lower *Trichuris* egg counts than lactating and pregnant females (Tukey multiple comparison test [lactating vs. cycling: estimate: −0.102 or 0.79 eggs per gram; $P = 0.006$; pregnant vs. cycling: estimate: −0.105 or 0.79 eggs per gram; $P = 0.019$]).

While pregnant and lactating females showed elevated risk of parasitism or parasite burden in the results presented above, cycling females were 27.3% more likely to be infected with *Oesophagostomum* than females who were not cycling. Moreover, cycling females were significantly more likely to be infected with *Oesophagostomum* than pregnant females (Tukey multiple comparison test [cycling vs. pregnant: estimate: 0.772; $P < 0.001$]).

3.3. Hormones

The association between hormone metabolites and parasitism varied across parasite taxa. First, females with higher fecal glucocorticoid concentrations exhibited significantly higher *Trichuris* egg counts (Table 3), but were at lower risk of *Oesophagostomum* infection, compared to females with lower fecal glucocorticoid concentrations (Table 3). Holding the other predictors at their mean value, females in the highest tertile of fecal glucocorticoid concentrations harbored 86.4% more *Trichuris* eggs than females in the bottom tertile (Fig. 3B). However, females in the bottom tertile of fecal glucocorticoid concentrations were 37.5% more likely to be infected with *Oesophagostomum* than females in the highest tertile. There was no significant association between glucocorticoid concentrations and the intensity of *Protospirura* infection.

Second, females with higher fecal progestagen concentrations exhibited significantly lower *Protospirura* egg counts (Fig. 2B; Table 3), but exhibited higher helminth parasite richness and were at greater risk of *Oesophagostomum* infection (Table 3). Holding the other predictors at their mean values, females in the lowest tertile of fecal progestagen concentrations harbored 105.7% more *Protospirura* eggs than females in the bottom tertile. However, females with fecal progestagen concentrations in the highest tertile had 8.3% higher helminth parasite richness and were 66.7% more likely to be infected with *Oesophagostomum* than females with fecal progestagen concentrations in the lowest tertile. There was no significant association between progestagen concentrations and the intensity of *Trichuris* infection.

Lastly, females with higher fecal estrogen concentrations exhibited higher helminth parasite richness than females with lower fecal estrogen concentrations (Table 3). Holding other predictors at their mean values,

Table 3
Best supported models based on averaging of parameter estimates for each measure of parasitism in female baboons (n = 2955 samples from 24 females). Model-average coefficients (conditional average), standard error, *z*-value and *P* value of the averaged models are shown.

parasite taxa					
Trichuris	random effects	Variance	SD		
	Female ID	0.069	0.263		
	fixed effects	estimate	SE	z-value	*P*
	Cortisol	0.002	0.0003	8.170	<0.001
	Cycling	−0.198	0.075	2.655	0.008
	Season (wet)	0.195	0.015	13.366	<0.001
	Protospirura	0.182	0.022	8.098	<0.001
	Oesophagostomum	0.187	0.022	8.358	<0.001
Protospirura	random effects	Variance	SD		
	Female ID	0.049	0.222		
	fixed effects	estimate	SE	z-value	*P*
	Progestagen	−0.225	0.102	−2.211	0.027
	Pregnant	−1.155	0.565	−2.045	0.041
	Progestagen x Pregnant	0.231	0.106	2.176	0.030
	Season (wet)	−0.123	0.025	−4.819	<0.001
	Oesophagostomum	0.150	0.027	5.497	<0.001
	Trichuris	0.305	0.021	14.232	<0.001
Oesophagostomum	random effects	Variance	SD		
	Female ID	0.250	0.500		
	fixed effects	estimate	SE	z-value	*P*
	Cortisol	−0.599	0.098	6.097	<0.001
	Estrogen	0.110	0.076	1.447	0.148
	Progestagen	0.411	0.057	7.157	<0.001
	Cycling	0.909	0.350	2.597	0.009
	Lactational amenorrhea	0.667	0.349	1.911	0.056
	Season (wet)	0.544	0.088	6.179	<0.001
	Protospirura	0.345	0.063	5.466	<0.001
	Trichuris	0.575	0.078	7.394	<0.001
Helminth richness	random effects	Variance	SD		
	Female ID	0.086	0.294		
	fixed effects	estimate	SE	z-value	*P*
	Estrogen	0.042	0.025	1.692	0.091
	Progestagen	0.035	0.016	2.270	0.023
	Season (wet)	0.086	0.032	2934.0	0.006

females in the highest tertile of fecal estrogen concentrations had 5.9% higher helminth parasite richness than females in the bottom tertile. However, there was no significant association between estrogen concentrations and the intensity of *Protospirura* infection, the intensity of *Trichuris* infection, or the probability of *Oesophagostomum* infection.

3.4. Steroid hormones and pregnancy

We found evidence that the intensity of *Protospirura* infection was mediated by an interaction between progestagen concentrations and reproductive state (Table 3). Specifically, as progestagen concentrations increased in pregnant females, the intensity of *Protospirura* infection increased correspondingly (Fig. 4). Conversely, as progestagen concentrations increased in non-pregnant females, the intensity of *Protospirura* infection decreased accordingly (Fig. 4).

3.5. Season

During dry periods, females exhibited 57.5% higher *Protospirura* and 77.3% higher *Trichuris* egg counts than during wet periods (Fig. 2C; Fig. 3C; Table 3). However, during wet periods, females exhibited 4.1% higher helminth parasite richness and 38.3% higher risk of *Oesophagostomum* infection than during dry periods (Table 3).

3.6. Patterns of coinfection

For all three common parasite taxa, infection intensity or risk for one parasite was always predicted by the other two parasites. Samples that contained *Oesophagostomum* had significantly higher *Protospirura* and *Trichuris* egg counts (Table 3). Likewise, *Protospirura* infection risk was predicted by *Oesophagostomum* and *Trichuris* (Fig. 2D and E; Table 3); and *Trichuris* infection risk was predicted by *Oesophagostomum* and *Protospirura* (Fig. 3D and E; Table 3).

4. Discussion

Female chacma baboons in this study exhibited individual variation in helminth richness and infection intensity that appeared to be driven by four factors: reproductive state, steroid hormone profiles, patterns of rainfall, and coinfection dynamics. Our study adds to a growing body of research indicating that reproduction is a costly stage of life for female mammals (e.g., Archie et al., 2014; Leivesley et al., 2019 but see Ołdakowski et al., 2012). Specifically, we found helminth parasite infection intensity to be associated with two of the most energetically costly stages of the reproductive cycle: pregnancy and lactation. Moreover, in support of previous research showing that steroid hormones can sometimes facilitate and sometimes inhibit the proliferation of parasites (Klein 2004; Tait et al., 2008; Vargas-Villavicencio et al., 2009), we found that helminth infection intensity of some parasites was associated with high concentrations of certain steroid hormones whereas infection intensity of other parasites was associated with low hormone concentrations. This inconsistent relationship between helminth infection intensity and steroid hormone concentrations probably reflects both the immunoenhancing and immunosuppressive capacities of steroid hormones as well as individual characteristics of different parasite taxa, among other factors. We also found evidence that the interaction between sex

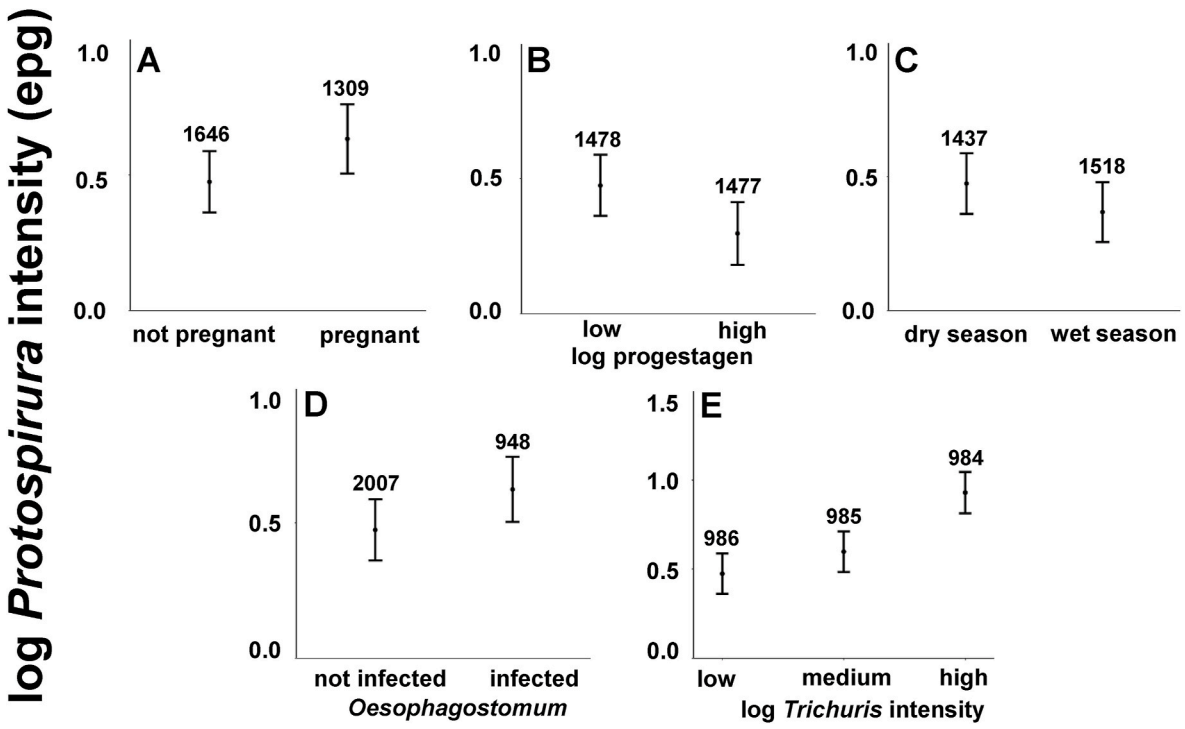

Fig. 2. Plots showing associations between log *Protospirura* intensity (eggs per gram; epg) in female baboons and marginal effects of each predictor variable. Plots are (A) pregnant (no or yes); (B) log progestagen concentrations (low = below median; high = above median; ng/g); (C) season (dry or wet); (D) presence/absence of *Oesophagostomum*; and (E) log *Trichuris* intensity (epg). Points and whiskers on the plot represent the mean and confidence intervals. For Fig. 2E, the values of each fixed effect are divided into tertiles. Numbers above each bar indicate sample size.

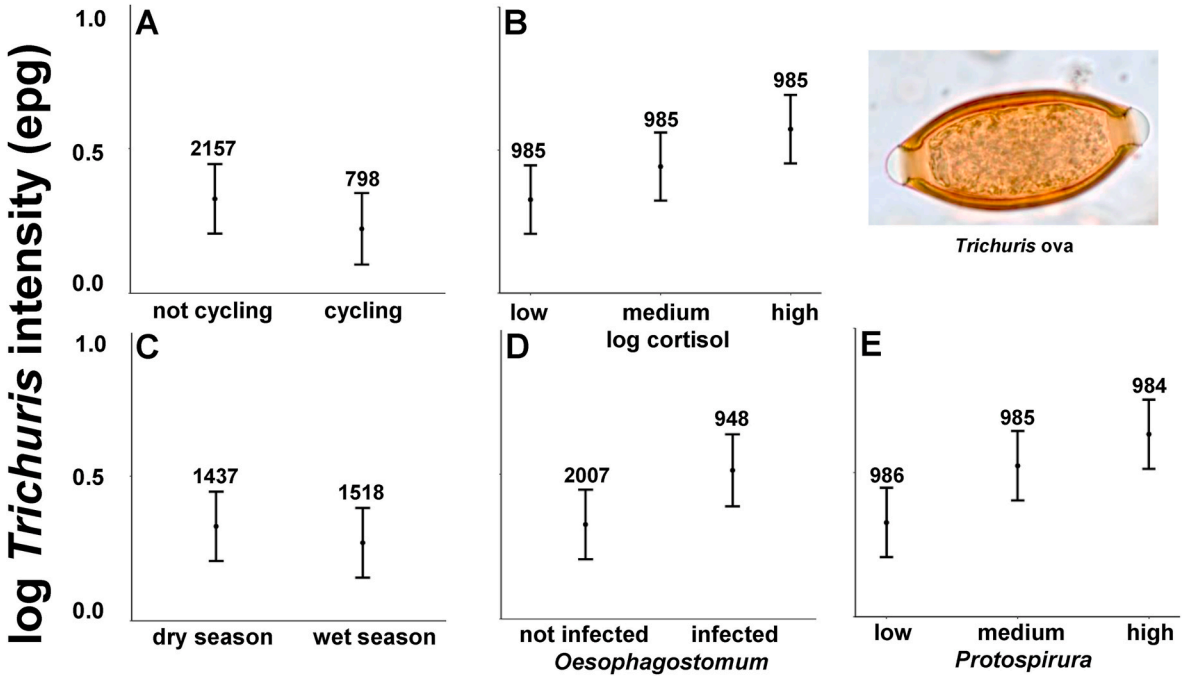

Fig. 3. Plots showing associations between log *Trichuris* intensity (eggs per gram; epg) in female baboons and marginal effects of each predictor variable. Plots are (A) cycling (no or yes); (B) log fecal glucocorticoid concentrations (ng/g); (C) season (dry or wet); (D) presence/absence of *Oesophagostomum*; and (E) log *Protospirua* intensity (epg). Points and whiskers on the plot represent the mean and confidence intervals. For Fig. 3B and E, the values of each fixed effect are divided into tertiles. Numbers above each bar indicate sample size. Photograph by Bobby Habig.

hormones and reproductive state is associated with differential risk of parasite infection. Specifically, for pregnant females, as progestagen concentrations increased, *Protospirura* infection also increased, while for non-pregnant females we observed the opposite pattern. On a broader scale, patterns of rainfall were important predictors of parasite prevalence and intensity. Lastly, helminth parasites exhibited positive covariance: infection with one parasite was positively associated with infection with another parasite. Collectively, these results provide key

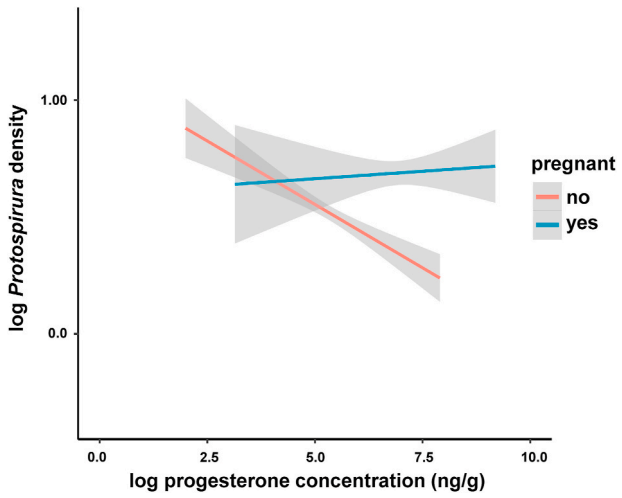

Fig. 4. An interaction between host reproductive state and progestagen concentrations in the infection intensity of *Protospirura*. Pregnant females (blue) exhibit increased infection intensity of *Protospirura* with rising progestagen concentrations. Non-pregnant females (red) exhibit decreased infection intensity of *Protospirura* with rising progestagen concentrations. Confidence intervals are in gray. (For interpretation of the references to colour in this figure legend, the reader is referred to the Web version of this article.)

insights into the drivers of parasite infection and are helpful for drawing inferences about the causes of individual variation in host infection.

4.1. Reproductive state is a significant predictor of parasite risk

We found that for two of the three most common helminth taxa in our study, both pregnancy and lactation were associated with higher parasitism; however, we found conflicting evidence that sexual cycling was associated with increased parasite risk. First, pregnant females exhibited significantly higher intensities of stomach worm (*Protospirura*) egg counts than cycling and lactating females. Because the stomach worm infects the gastrointestinal tract and siphons nutritional resources from its host (Foster and Johnson 1939; Toft 1986), pregnant females might be especially vulnerable to this parasite. Moreover, if females ingest more arthropods (intermediate hosts of stomach worms) during pregnancy, then this pattern might be explained by differences in parasite exposure. Second, we found that lactating females had a higher probability of nodular worm (*Oesophagostomum*) infection than non-lactating females. The nodular worm is considered the most debilitating gastrointestinal parasite of baboons (Pettifer 1984). Lactating females might be more susceptible to this parasite than non-lactating females because lactational amenorrhea is the most energetically costly component of reproduction (Pond 1977; Künkele 2000). Alternatively, because lactating females are highly attractive grooming partners, that is, other females groom lactating conspecifics to gain access to their infants (Silk et al., 2010), increased social interaction might contribute to higher exposure resulting in higher infection rates. In terms of susceptibility, these two results are consistent with the hypothesis that the energetic and nutrient demands of pregnancy and lactation reduce host condition and increase susceptibility to parasitism (Pond 1977; Lloyd 1983; Speakman 2008; Blackwell et al., 2015). Interestingly, lactating females were significantly more likely to be infected with *Oesophagostomum* than pregnant females. In contrast to our findings above, this result supports the hypothesis that pregnant females are less susceptible to worm infections because they favor immune responses that promote Th-2 cytokine production (Roberts and Horsnell 2015), important for providing humoral immunity against helminth parasites (Mosmann and Sad 1996; Vargas-Villavicencio et al., 2009). In support of the idea that lactation is more costly than pregnancy, several studies have reported increased parasite risk in lactating but not pregnant females (e.g., domestic sheep, *Ovis aries*: González-Garduño et al., 2014; bank vole, *Myodes glareolus*: Grzybek et al., 2014; spotted hyaena, *Crocuta crocuta*: East et al., 2015; red deer, *Cervus elaphus*: Albery et al., 2020). Lastly, we found conflicting evidence that sexual cycling is associated with parasite risk: cycling females exhibited higher risk of nodular worm (*Oesophagostomum*) infection but exhibited lower whipworm (*Trichuris*) egg counts than non-cycling females. Our contrasting results—higher *Protospirura* egg counts in pregnant than lactating females; higher risk of *Oesophagostomum* infection in lactating than pregnant females; and higher risk of *Oesophagostomum* infection but lower intensity of *Trichuris* infection in cycling than non-cycling females—may reflect individual variation in host immune response to different genera of parasites and variation in the ability of different genera of parasites to alter immune defenses during the reproductive cycle (Klein 2004).

4.2. The association between steroid hormone concentrations and parasitism varies across parasite taxa

For some parasites, steroid hormone concentrations were associated with increased parasitism even after controlling for reproductive state, and for other parasites, steroid hormone concentrations were associated with decreased parasitism. First, in support of the idea that glucocorticoids are associated with immunosuppression (Nava-Castro et al., 2011), we found that females with high fecal glucocorticoid concentrations harbored high quantities of *Trichuris* eggs. This result is consistent with other studies of nonhuman primates that have also found positive correlations between glucocorticoid concentrations and helminth parasite loads (e.g., red colobus monkey, *Procolobus rufomitratus*: Chapman et al., 2006; red-capped mangabey, *Cercocebus torquatus*: Friant et al., 2016; yellow baboon, *P. cynocephalus*: Akinyi et al., 2019; Barbary macaque, *Macaca sylvanus*: Müller-Klein et al., 2019). However, contrary to this result, we also found that females with high fecal glucocorticoid concentrations were at low risk of *Oesophagostomum* infection. This result is consistent with a study of Yakushima macaques (*Macaca fuscata yakui*), where the authors found a negative correlation between *Oesophagostomum* egg counts and fecal glucocorticoid concentrations (Broche et al., 2017). While it is unclear why females with high glucocorticoid concentrations harbor more *Trichuris* eggs yet are at low risk of *Oesophagostomum* infection, one possible explanation may relate to variation in the ability of parasite genera to evade the host's immune system (Schmid-Hempel 2009). For example, mice experimentally infected with *Trichuris muris* downregulate Th-2 immune cytokines, suppressing a primary pathway for fighting extracellular parasites (Bancroft et al., 1994). Hence, in some cases, the mobilization of glucocorticoids might reflect an adaptive metabolic response that helps facilitate immune response and maintain homeostasis following the physiological challenge of parasite infection, whereas in other cases, the mobilization of glucocorticoids might reflect poor health and the inability to maintain homeostasis, especially during more physiologically challenging periods such as when the immune system fails to elicit an effective response to parasite infection (Beehner and Bergman 2017). Moreover, glucocorticoids and steroid hormones in general might serve dual roles, both as potential drivers of and potential responses to helminth burdens (Nava-Castro et al., 2011; Lafferty and Shaw 2013). Second, our finding that females with high progestagen concentrations exhibited high helminth parasite richness and were at high risk of *Oesophagostomum* infection is consistent with previous research documenting the immunosuppressive qualities of progesterone (Klein 2004). In support of this relationship, experimental studies have shown that progesterone acts as an immunosuppressant in humans (Wyle and Kent 1977). However, we also found that females with high fecal progestagen concentrations harbored low quantities of *Protospirura* eggs, which is consistent with research demonstrating that progesterone can also have immunoenhancing qualities (Cabrera-Muñoz et a. 2010). For example, in an experimental study of golden hamsters (*Mesocricetus*

aureatus) infected with *Schistosoma haematobium*, exogenous administration of progesterone was associated with reduced parasite worm and egg loads (Soliman and Ibrahim 2005). While our results are conflicting, they are consistent with other studies showing that progesterone exerts both immunoenhancing and immunosuppressive effects (Romano et al., 2015). Whether these dualities can exist in the same individual or whether they represent higher level variations across hosts or systems is largely unknown and beyond the scope of the present study. Lastly, our finding that females with high concentrations of estrogen exhibited high helminth parasite richness is consistent with previous experimental research of mice that found that exogenous treatment of estrogen causes dramatic reduction in CD4 T cell development important for eliciting adaptive immune defenses against helminth parasites (Rijhsinghani et al., 1996). Overall, our results support the idea that the protective and permissive effects of steroid hormones are dependent on the identity of both the parasite and the host, among other factors (Morales-Montor et al., 2004; Cabrera-Muñoz et al., 2010).

4.3. An interaction between fecal progestagen concentrations and reproductive state

Our results also suggest an interaction between host reproductive state and progestagen concentrations in the infection intensity of *Protospirura* (Fig. 4). Specifically, females who were not pregnant exhibited lower intensities of *Protospirura* eggs with increasing progestagen concentrations. Conversely, pregnant females exhibited higher intensities of *Protospirura* eggs with increasing progestagen concentrations. The former result (lower *Protospirura* egg counts with increasing progestagen concentrations for non-pregnant females) provides evidence of the immunoprotective effects of progesterone (Tait et al., 2008). Indeed, several studies have found that experimental treatment of exogenous progesterone inhibits the proliferation of many gastrointestinal parasites including *Taenia crassiceps* (Escobedo et al., 2004), *Taenia solium* (Escobedo et al., 2011) and *Trichinella spiralis* (Nunez et al., 2005; Hernández-Bello et al., 2011). The latter result (higher *Protospirura* egg counts with increasing progestagen concentrations for pregnant females) is consistent with research on humans, which has found that the progesterone receptor is blocked during late pregnancy so that it is possible to induce labor (Allport et al., 2001). Moreover, while it is well established that progesterone is essential for the maintenance of pregnancy (Siiteri et al., 1977), localized immunosuppression is required to prevent the loss of the fetus (Tait et al., 2008). Thus, one possible explanation for the interaction we found is that the immunoenhancing properties of progesterone are suppressed during pregnancy, reflecting a possible tradeoff between reproductive effort and the ability to resist parasitic infection (Lee 2006).

4.4. Patterns of rainfall are associated with variation in parasite risk

For all four of our outcome variables, season was a significant predictor of parasitism. For two parasites, *Protospirura* and *Trichuris*, infection intensity was greater during the dry summer months compared to the wet winter months. These results support the hypothesis that during hot and dry periods, inadequate nutrition and heat stress reduce hosts' abilities to mount effective immune responses against helminths (Dowell 2001; Koski and Scott 2001; Mignatti et al., 2016). As an alternative explanation, because this troop of chacma baboons was found to consume significantly more grasses, roots, and bulbs during the dry season (Hoffman and O'Riain, 2011), they might come into contact with more infective *Trichuris* ova during these times. Consistent with our findings, two studies of savanna-dwelling yellow baboons (*P. cynocephalus*) also reported higher *Trichuris* egg intensities in dry versus wet conditions (Akinyi et al., 2019; Habig et al., 2019). Our results also, however, point to greater helminth parasite richness and probability of *Oesophagostomum* infection during the wet winter months compared to the dry summer months, supporting the hypothesis that rainfall and high humidity promote the survival of infectious stages of some parasites in the environment (Altizer et al., 2006; Nunn and Altizer 2006). Indeed, consistent with these findings, a study of a population of chacma baboons in Namibia reported a positive correlation between parasite species richness and rainfall (Benavides et al., 2012). The Cape Peninsula is a highly seasonal environment in which winter, despite greater rainfall, appears to impose higher physiological constraints on the baboons than summer due to the low temperatures and shorter day lengths (van Doorn et al., 2010; Chowdhury et al. in review). Hence, in temperate climates, a combination of inadequate nutrition due to reduced foraging time and thermal stress from low temperatures may reduce hosts' capacities to elicit effective immune defenses against helminths (Dowell 2001; Koski and Scott 2001). Supporting this notion is a study by Chowdhury et al. (in review), who found, after controlling for reproductive state and rank, that physiological stress in this population, as measured by elevated fecal glucocorticoid concentrations, was associated with lower temperatures, higher rainfall, and shorter day lengths of the wet season; these findings support the idea that females are more susceptible to parasitism during this more energetically challenging period. Moreover, Hoffman and O'Riain (2011) found that chacma baboons consume significantly more items in the soil and leaf litter during the cold rainy season, which might contribute to increased contact with more helminth taxa during this time. Taken together, our results suggest that two mechanisms – (1) seasonal patterns of host susceptibility and (2) seasonal patterns of parasite exposure – are both key drivers of variation in parasite risk in this population.

4.5. Helminth parasites exhibit positive covariance

Finally, we found that one of the key correlates of parasite risk was the presence of other parasite taxa. For instance, *Trichuris* egg counts were higher in baboons that were also infected with *Protospirura* and/or *Oesophagostomum*. Indeed, for our three most prevalent parasite taxa, infection intensity or risk was always predicted by the other two parasites. These findings are consistent with the results of two recent studies of yellow baboons (*P. cynocephalus*) that also reported positive covariance among parasite taxa (Akinyi et al., 2019; Habig et al., 2019). The patterns of coinfection we observed in our study can be explained by at least three non-mutually exclusive mechanisms. First, coinfection might occur based on shared transmission mode (e.g., Fleming et al., 2006). For instance, both *Trichuris* and *Oesophagostomum* transmission occurs when the host ingests an egg or larvae found on food or surfaces in its environment (Cogswell 2012; Strait et al., 2012). Second, patterns of coinfection might occur when infection with one parasite suppresses host immunity and increases the probability of infection with another parasite (e.g., Ezenwa et al., 2010; Telfer et al., 2010). Lastly, there might be a positive feedback loop in which a host in poor condition gets infected with one parasite, thereby further impairing the host's condition and subsequently reducing the host's ability to resist subsequent infections (Beldomenico et al., 2008; Griffiths et al., 2011).

In contrast with our findings, a study of chacma baboons in Kruger National Park found evidence of competitive exclusion between two stomach worm species (Pettifer 1984). Specifically, high infestation with one stomach worm (*Abbreviata caucasica*) was associated with low infestation of another stomach worm (*Streptopharagus pigmentatus*). Interestingly, we found no evidence of negative covariance among the helminth parasite species in the present study, which suggests that these parasites were not competing for the same infection sites or that the density of these parasites was not at levels warranting competitive exclusion. Future research incorporating the experimental removal of a target parasite and the monitoring of a potential competitor parasite could help to elucidate the extent to which helminth parasites in baboon hosts undergo competitive exclusion.

4.6. Conclusions and future directions

Our results add to a limited body of literature on the drivers of individual variation in helminth infection risk in wild populations of animals. By testing predictors of parasite risk at multiple scales—from individual characteristics of the host to environmental conditions and patterns of coinfection—we provide an especially holistic perspective on factors that influence infection risk. At the host level, our study revealed that both reproductive state and hormone profiles are associated with parasite risk and infection intensity as estimated through egg counts. Indeed, we found that baboon hosts are especially sensitive to parasitism during the costliest phases of the reproductive cycle: pregnancy and lactation. Moreover, while most studies of wild animals have focused on the relationship between glucocorticoid concentrations and parasitism (e.g., Friant et al., 2016; Akinyi et al., 2019; Müller-Klein et al., 2019), our study supplements this body of literature by additionally testing the association between sex hormones (progesterone and estrogen) and parasitism. Interestingly, our findings reveal that the interaction between sex hormones, reproductive state, and parasite infection is quite complex, and that baboon hosts are differentially vulnerable to different genera of parasites, which suggests that individual characteristics of parasites and different hormonal and reproductive environments of the host mediate the course of infection. On a broader scale, we found that patterns of rainfall are significant drivers of parasite infection, and we think this will be an important area of future research particularly in the context of climate change (Mignatti et al., 2016). Lastly, parasites exhibited positive covariance: 100% of baboon hosts were infected with at least three parasite taxa simultaneously at some point during the study, and 64.3% of all samples exhibited coinfection (infection with two or more helminth taxa). We recommend that future studies incorporate additional parasite taxa, including eukaryotic and prokaryotic microparasites (e.g., Wilcox et al., 2015). Additionally, we recommend that future studies of wild populations of animals incorporate additional methods including controlled field experiments (e.g., Budischak et al., 2018) and longitudinal survival analyses (e.g., Schneider-Crease et al., 2017) to help further elucidate the drivers of individual variation in parasite infection as well as its impact on host fitness.

Declaration of competing interest

On behalf of all the co-authors, the corresponding author declares no conflict of interest.

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References

Akinyi, M.Y., Jansen, D., Habig, B., Gesquiere, L.R., Alberts, S.C., Archie, E.A., 2019. Costs and drivers of helminth parasite infection in wild female baboons. J. Anim. Ecol. 88 (7), 1029–1043.
Alberts, S.C., Altmann, J., 2006. The evolutionary past and the research future: environmental variation and life history flexibility in a primate lineage. Reproduction and Fitness in Baboons: Behavioral, Ecological, and Life History Perspectives. Springer, Boston, MA, pp. 277–303.
Albery, G.F., Watt, K.A., Keith, R., Morris, S., Morris, A., Kenyon, F., et al., 2020. Reproduction has different costs for immunity and parasitism in a wild mammal. Funct. Ecol. 34 (1), 229–239.
Albon, S.D., Stien, A., Irvine, R.J., Langvatn, R., Ropstad, E., Halvorsen, O., 2002. The role of parasites in the dynamics of a reindeer population. Proc. Roy. Soc. Lond. B Biol. Sci. 269 (1500), 1625–1632.
Allen, A.V., Ridley, D.S., 1970. Further observations on the formol-ether concentration technique for faecal parasites. J. Clin. Pathol. 23 (6), 545.
Allport, V.C., Pieber, D., Slater, D.M., Newton, R., White, J.O., Bennett, P.R., 2001. Human labour is associated with nuclear factor-κB activity which mediates cyclo-oxygenase-2 expression and is involved with the 'functional progesterone withdrawal'. Mol. Hum. Reprod. 7 (6), 581–586.
Altizer, S., Dobson, A., Hosseini, P., Hudson, P., Pascual, M., Rohani, P., 2006. Seasonality and the dynamics of infectious diseases. Ecol. Lett. 9 (4), 467–484.
Altmann, S.A., 1973. The pregnancy sign in savannah baboons. J. Zoo Anim. Med. 4 (2), 8–12.
Alvergne, A., Tabor, V.H., 2018. Is female health cyclical? Evolutionary perspectives on menstruation. Trends Ecol. Evol. 33 (6), 399–414.
Anderson, R.C., 2000. Nematode Parasites of Vertebrates. Their Development and Transmission. CABI International, Wallingford, UK.
Appleton, C.C., Henzi, S.P., Whitehead, S.I., 1991. Gastro-intestinal helminth parasites of the chacma baboon, *Papio cynocephalus ursinus*, from the coastal lowlands of Zululand, South Africa. Afr. J. Ecol. 29 (2), 149–156.
Archie, E.A., Altmann, J., Alberts, S.C., 2014. Costs of reproduction in a long-lived female primate: injury risk and wound healing. Behav. Ecol. Sociobiol. 68 (7), 1183–1193.
Bancroft, A.J., Else, K.J., Grencis, R.K., 1994. Low-level infection with *Trichuris muris* significantly affects the polarization of the CD4 response. Eur. J. Immunol. 24 (12), 3113–3118.
Bartoń, K., 2009. MuMIn: R Package for Model Selection and Multi-Model Inference version 0.12.2.
Bates, D., Mächler, M., Bolker, B., Walker, S., 2014. Fitting linear mixed-effects models using lme4. J. Stat. Software 67, 1–48.
Beehner, J.C., Bergman, T.J., 2017. The next step for stress research in primates: to identify relationships between glucocorticoid secretion and fitness. Horm. Behav. 91, 68–83.
Beldomenico, P.M., Telfer, S., Gebert, S., Lukomski, L., Bennett, M., Begon, M., 2008. Poor condition and infection: a vicious circle in natural populations. Proc. Biol. Sci. 275 (1644), 1753–1759.
Benavides, J.A., Huchard, E., Pettorelli, N., King, A.J., Brown, M.E., Archer, C.E., et al., 2012. From parasite encounter to infection: multiple-scale drivers of parasite richness in a wild social primate population. Am. J. Phys. Anthropol. 147 (1), 52–63.
Bentwich, Z., Kalinkovich, A., Weisman, Z., Borkow, G., Beyers, N., Beyers, A.D., 1999. Can eradication of helminthic infections change the face of AIDS and tuberculosis? Immunol. Today 20 (11), 485–487.
Bezjian, M., Gillespie, T.R., Chapman, C.A., Greiner, E.C., 2008. Coprologic evidence of gastrointestinal helminths of forest baboons, *Papio anubis*, in Kibale National Park, Uganda. J. Wildl. Dis. 44 (4), 878–887.
Blackwell, A.D., Tamayo, M.A., Beheim, B., Trumble, B.C., Stieglitz, J., Hooper, P.L., et al., 2015. Helminth infection, fecundity, and age of first pregnancy in women. Science 350 (6263), 970–972.
Bowman, D.D., 2014. Georgis' Parasitology for Veterinarians, tenth ed. Elsevier, St. Louis, Missouri.
Broche Jr., N., Itoigawa, A., Kawaguchi, Y., Kawamoto, Y., Tanaka, M., Ueno, K., Xu, Z., Zhang, J., 2017. Testing the trade-off between parasite resistance and the immunosuppressive hormones cortisol and testosterone. Yakushima Field & Lab Report.
Budischak, S.A., O'Neal, D., Jolles, A.E., Ezenwa, V.O., 2018. Differential host responses to parasitism shape divergent fitness costs of infection. Funct. Ecol. 32 (2), 324–333.
Burnham, K.P., Anderson, D.R., 2002. Model Selection and Multimodel Inference: A Practical Information-Theoretic Approach, second ed. Springer, New York.
Burnham, K.P., Anderson, D.R., 2004. Multimodel inference: understanding AIC and BIC in model selection. Socio. Methods Res. 33 (2), 261–304.
Byrne, R.L., Fogarty, U., Mooney, A., Marples, N.M., Holland, C.V., 2018. A comparison of helminth infections as assessed through coprological analysis and adult worm burdens in a wild host. Int. J. Parasitol.: Parasites and Wildlife 7 (3), 439–444.
Cabrera-Muñoz, E., Escobedo, G., Guzmán, C., Camacho-Arroyo, I., 2010. Role of progesterone in HIV and parasitic infections. Open Neuroendocrinol. J. 3, 137–142.
Carrero, J.C., Cervantes, C., Moreno-Mendoza, N., Saavedra, E., Morales-Montor, J., Laclette, J.P., 2006. Dehydroepiandrosterone decreases while cortisol increases in vitro growth and viability of *Entamoeba histolytica*. Microb. Infect. 8 (2), 323–331.
Chapman, C.A., Wasserman, M.D., Gillespie, T.R., Speirs, M.L., Lawes, M.J., Saj, T.L., Ziegler, T.E., 2006. Do food availability, parasitism, and stress have synergistic effects on red colobus populations living in forest fragments? Am. J. Phys. Anthropol. 131 (4), 525–534.
Chapman, C.A., Speirs, M.L., Hodder, S.A., Rothman, J.M., 2010. Colobus monkey parasite infections in wet and dry habitats: implications for climate change. Afr. J. Ecol. 48 (2), 555–558.

Chowdhury, S., Brown, J., Swedell, L. (In Review). Costs of seasonality in a southern latitude: baboon behavioral endocrinology in the Cape Peninsula of South Africa. Submitted to Hormones and Behavior.
Chowdhury, S., Brown, J., Swedell, L., 2020. Anthropogenic effects on the physiology and behavior of chacma baboons in the Cape Peninsula of South Africa. Conservation Physiology 8 (1). https://doi.org/10.1093/conphys/coaa066.
Cogswell, F., 2012. Parasites of non-human primates. In: Baker, D.G. (Ed.), Flynn's Parasites of Laboratory Animals Ames. Blackwell, pp. 693–743.
Colditz, I.G., 2008. Six costs of immunity to gastrointestinal nematode infections. Parasite Immunol. (Oxf.) 30, 63–70.
Coop, R.L., Holmes, P.H., 1996. Nutrition and parasite interaction. Int. J. Parasitol. 26 (8/9), 951–962.
Cooper, N., Kamilar, J.M., Nunn, C.L., 2012. Host longevity and parasite species richness in mammals. PloS One 7, e42190.
Cowling, R.M., Macdonald, I.A.W., Simmons, M.T., 1996. The Cape Peninsula, South Africa: physiographical, biological and historical background to an extraordinary hot-spot of biodiversity. Biodivers. Conserv. 5 (5), 527–550.
Creel, S., Dantzer, B., Goymann, W., Rubenstein, D.R., 2013. The ecology of stress: effects of the social environment. Funct. Ecol. 27 (1), 66–80.
Defolie, C., Merkling, T., Fichtel, C., 2020. Patterns and variation in the mammal parasite–glucocorticoid relationship. Biol. Rev. 95 (1), 74–93.
Dixson, A., 2015. Primate Sexuality. *The International Encyclopedia of Human Sexuality*. Oxford University Press, Oxford, UK, pp. 861–1042.
Dowell, S.F., 2001. Seasonal variation in host susceptibility and cycles of certain infectious diseases. Emerg. Infect. Dis. 7 (3), 369.
Drewe, J.A., O'Riain, M.J., Beamish, E., Currie, H., Parsons, S., 2012. Survey of infections transmissible between baboons and humans, Cape Town, South Africa. Emerg. Infect. Dis. 18 (2), 298.
East, M.L., Otto, E., Helms, J., Thierer, D., Cable, J., Hofer, H., 2015. Does lactation lead to resource allocation trade-offs in the spotted hyaena? Behav. Ecol. Sociobiol. 69 (5), 805–814.
Ebbert, M.A., McGrew, W.C., Marchant, L.F., 2013. Community composition, correlations among taxa, prevalence, and richness in gastrointestinal parasites of baboons in Senegal, West Africa. Primates 54 (2), 183–189.
Escobedo, G., Larralde, C., Chavarria, A., Cerbón, M.A., Morales-Montor, J., 2004. Molecular mechanisms involved in the differential effects of sex steroids on the reproduction and infectivity of *Taenia crassiceps*. J. Parasitol. 1235–1244.
Escobedo, G., Camacho-Arroyo, I., Nava-Luna, P., Olivos, A., Pérez-Torres, A., Leon-Cabrera, S., et al., 2011. Progesterone induces mucosal immunity in a rodent model of human taeniosis by *Taenia solium*. Int. J. Biol. Sci. 7 (9), 1443.
Ezenwa, V.O., 2004. Interactions among host diet, nutritional status and gastrointestinal parasite infection in wild bovids. Int. J. Parasitol. 34 (4), 535–542.
Ezenwa, V.O., Etienne, R.S., Luikart, G., Beja-Pereira, A., Jolles, A.E., 2010. Hidden consequences of living in a wormy world: nematode-induced immune suppression facilitates tuberculosis invasion in African buffalo. Am. Nat. 176 (5), 613–624.
Fairbanks, B., Hawley, D.M., 2012. Interactions between host social behavior, physiology, and disease susceptibility: the role of dominance status and social context. In: Demas, G.E., Nelson, R.J. (Eds.), Ecoimmunology. Oxford University Press, London, pp. 440–467.
Fleming, F.M., Brooker, S., Geiger, S.M., Caldas, I.R., Correa-Oliveira, R., Hotez, P.J., Bethony, J.M., 2006. Synergistic associations between hookworm and other helminth species in a rural community in Brazil. Trop. Med. Int. Health 11 (1), 56–64.
Foerster, S., Monfort, S.L., 2010. Fecal glucocorticoids as indicators of metabolic stress in female Sykes' monkeys (*Cercopithecus mitis albogularis*). Horm. Behav. 58 (4), 685–697.
Foo, Y.Z., Nakagawa, S., Rhodes, G., Simmons, L.W., 2017. The effects of sex hormones on immune function: a meta-analysis. Biol. Rev. 92 (1), 551–571.
Foster, A.O., Johnson, C.M., 1939. A preliminary note on the identity, lifecycle, and pathogenicity of an important nematode parasite of captive monkeys. Am. J. Trop. Med. Hyg. 1 (3), 265–277.
Fox, J., 2015. Applied Regression Analysis and Generalized Linear Models. Sage, Thousand Oaks.
Fox, J., Weisberg, S., 2011. An R Companion to Applied Regression. Sage, Thousand Oaks.
Friant, S., Ziegler, T.E., Goldberg, T.L., 2016. Changes in physiological stress and behaviour in semi-free-ranging red-capped mangabeys (*Cercocebus torquatus*) following antiparasitic treatment. Proc. Biol. Sci. 283 (1835), 20161201.
Gassó, D., Feliu, C., Ferrer, D., Mentaberre, G., Casas-Díaz, E., Velarde, R., et al., 2015. Uses and limitations of faecal egg count for assessing worm burden in wild boars. Vet. Parasitol. 209 (1–2), 133–137.
Gesquiere, L.R., Altmann, J., Archie, E.A., Alberts, S.C., 2019. Interbirth intervals in wild baboons: environmental predictors and hormonal correlates. Am. J. Phys. Anthropol. 166 (1), 107–126.
Gillespie, T.R., 2006. Noninvasive assessment of gastrointestinal parasite infections in free-ranging primates. Int. J. Primatol. 27, 1129–1143.
González-Garduño, R., Torres-Acosta, J.F.J., Chay-Canul, A.J., 2014. Susceptibility of hair sheep ewes to nematode parasitism during pregnancy and lactation in a selective anthelmintic treatment scheme under tropical conditions. Res. Vet. Sci. 96 (3), 487–492.
Griffin, R.H., Nunn, C.L., 2012. Community structure and the spread of infectious disease in primate social networks. Evol. Ecol. 26 (4), 779–800.
Griffiths, E.C., Pedersen, A.B., Fenton, A., Petchey, O.L., 2011. The nature and consequences of coinfection in humans. J. Infect. 63 (3), 200–206.
Grzybek, M., Bajer, A., Behnke-Borowczyk, J., Al-Sarraf, M., Behnke, J.M., 2014. Female host sex-biased parasitism with the rodent stomach nematode *Mastophorus muris* in wild bank voles (*Myodes glareolus*). Parasitol. Res. 114 (2), 523–533.
Gulland, F.M.D., 1995. The impact of infectious diseases on wild animal populations – a review. In: Grenfell, B.T., Dobson, A.P. (Eds.), Ecology of Infectious Diseases in Natural Populations: 20–51. Cambridge University Press, Cambridge.
Habig, B., Archie, E.A., 2015. Social status, immune response, and parasitism in males: a meta-analysis. Phil. Trans. Biol. Sci. 370 (1669), 20140109.
Habig, B., Doellman, M.M., Woods, K., Olansen, J., Archie, E.A., 2018. Social status and parasitism in male and female vertebrates: a meta-analysis. Sci. Rep. 8 (1), 1–13.
Habig, B., Jansen, D., Akinyi, A., Gesquiere, W., Alberts, M., Archie, M., 2019. Multi-scale predictors of parasite risk in wild male savanna baboons (*Papio cynocephalus*). Behav. Ecol. Sociobiol. 73 (10), 1–16.
Hausfater, G., Watson, D.F., 1976. Social and reproductive correlates of parasite ova emissions by baboons. Nature 262 (5570), 688–689.
Hernández-Bello, R., Ramirez-Nieto, R., Muñiz-Hernández, S., Nava-Castro, K., Pavón, L., Sánchez-Acosta, A.G., Morales-Montor, J., 2011. Sex steroids effects on the molting process of the helminth human parasite *Trichinella spiralis*. J. Biomed. Biotechnol. 625380, 2011.
Hillegass, M.A., Waterman, J.M., Roth, J.D., 2010. Parasite removal increases reproductive success in a social African ground squirrel. Behav. Ecol. 21 (4), 696–700.
Hinojosa, L., Valdez, R.A., Salvador, V., Rodriguez, A.G., Willms, K., Romano, M.C., 2012. The effect of glucocorticoids on sex steroid synthesis in cultured *Taenia crassiceps* Wake Forest University (WFU) cysticerci. J. Helminthol. 86 (4), 465–469.
Hoffman, T.S., O'Riain, M.J., 2011. The spatial ecology of chacma baboons (*Papio ursinus*) in a human-modified environment. Int. J. Primatol. 32 (2), 308–328.
Holmes, J.C., 1961. Effects of concurrent infections on *Hymenolepis diminuta* (Cestoda) and *Moniliformis dubius* (Acanthocephala). I. General effects and comparison with crowding. J. Parasitol. 47 (2), 209–216.
Jolles, A.E., Ezenwa, V.O., Etienne, R.S., Turner, W.C., Olff, H., 2008. Interactions between macroparasites and microparasites drive infection patterns in free-ranging African buffalo. Ecology 89 (8), 2239–2250.
Jones, L.A., Sakkas, P., Houdijk, J.G.M., Knox, D.P., Kyriazakis, I., 2012. Amelioration of the periparturient relaxation of immunity to parasites through a reduction in mammalian reproductive effort. Int. J. Parasitol. 42 (13–14), 1127–1134.
Kelly-Hope, L.A., Diggle, P.J., Rowlingson, B.S., Gyapong, J.O., Kyelem, D., Coleman, M., et al., 2006. Negative spatial association between lymphatic filariasis and malaria in West Africa. Trop. Med. Int. Health 11 (2), 129–135.
Klein, S.L., 2004. Hormonal and immunological mechanisms mediating sex differences in parasite infection. Parasite Immunol. 26 (6-7), 247–264.
Koski, K.G., Scott, M.E., 2001. Gastrointestinal nematodes, nutrition and immunity: breaking the negative spiral. Annu. Rev. Nutr. 21 (1), 297–321.
Künkele, J., 2000. Energetics of gestation relative to lactation in a precocial rodent, the Guinea pig (*Cavia porcellus*). J. Zool. 250 (4), 533–539.
Kuznetsova, A., Brockhoff, P.B., Christensen, R.H.B., 2015. lmerTest: Tests in Linear Mixed Effects Models. R package version 2.0.
Lafferty, K.D., Shaw, J.C., 2013. Comparing mechanisms of host manipulation across host and parasite taxa. J. Exp. Biol. 216, 56–66.
Lawal, K.A., 2015. Understanding the Variability and Predictability of Seasonal Climates over West and Southern Africa Using Climate Models. University of Cape Town, Cape Town.
Lee, K.A., 2006. Linking immune defenses and life history at the levels of the individual and the species. Integr. Comp. Biol. 46 (6), 1000–1015.
Leivesley, J.A., Bussière, L.F., Pemberton, J.M., Pilkington, J.G., Wilson, K., Hayward, A. D., 2019. Survival costs of reproduction are mediated by parasite infection in wild Soay sheep. Ecol. Lett. 22 (8), 1203–1213.
Li, M., Leatherland, J.F., Woo, P.T., 2013. Cortisol and dexamethasone increase the in vitro multiplication of the haemoflagellate, *Cryptobia salmositica*, possibly by interaction with a glucocorticoid receptor-like protein. Int. J. Parasitol. 43 (5), 353–360.
Lingnau, A., Margos, G., Maier, W.A., Seitz, H.M., 1993. The effects of hormones on the gametocytogenesis of *Plasmodium falciparum* in vitro. Appl. Parasitol. 34 (3), 153–160.
Lloyd, S., 1983. Effect of pregnancy and lactation upon infection. Vet. Immunol. Immunopathol. 4 (1–2), 153–176.
Lüdecke, D., 2018. ggeffects: tidy data frames of marginal effects from regression models. Journal of Open Source Software 3 (26), 772.
Meade, B.J., 1984. Host Parasite Dynamics Among Amboseli Baboons (*Papio cynocephalus*). Ph.D. thesis, Virginia Polytechnic and State University, Blacksburg, VA.
Mideksa, S., Mekonnen, N., Muktar, Y., 2016. Prevalence and burden of nematode parasites of small ruminants in and around Haramaya University. World Appl. Sci. J. 34 (5), 644–651.
Mignatti, A., Boag, B., Cattadori, I.M., 2016. Host immunity shapes the impact of climate changes on the dynamics of parasite infections. Proc. Natl. Acad. Sci. Unit. States Am. 113 (11), 2970–2975.
Miterpáková, M., Dubinsky, P., Reiterová, K., Stanko, M., 2006. Climate and environmental factors influencing *Echinococcus multilocularis* occurrence in the Slovak Republic. Ann. Agric. Environ. Med. 13 (2), 235–242.
Mor, G., Cardenas, I., 2010. The immune system in pregnancy: a unique complexity. Am. J. Reprod. Immunol. 63 (6), 425–433.
Mor, G., Cardenas, I., Abrahams, V., Guller, S., 2011. Inflammation and pregnancy: the role of the immune system at the implantation site. Ann. N. Y. Acad. Sci. 1221 (1), 80.

Morales-Montor, J., Hallal-Calleros, C., Romano, M.C., Damian, R.T., 2002. Inhibition of p-450 aromatase prevents feminisation and induces protection during cysticercosis. Int. J. Parasitol. 32 (11), 1379–1387.
Morales-Montor, J., Chavarria, A., De Leon, M.A., Del Castillo, L.I., Escobedo, E.G., Sanchez, E.N., et al., 2004. Host gender in parasitic infections of mammals: an evaluation of the female host supremacy paradigm. J. Parasitol. 90 (3), 531–546.
Morrison, D.D., Waa, E.A.V., Bennett, J.L., 1986. Effects of steroids and steroid synthesis inhibitors on fecundity of *Schistosoma mansoni* in vitro. J. Chem. Ecol. 12 (8), 1901–1908.
Mosmann, T.R., Sad, S., 1996. The expanding universe of T-cell subsets: Th1, Th2 and more. Immunol. Today 17 (3), 138–146.
Mostowy, R., Engelstädter, J., 2011. The impact of environmental change on host–parasite coevolutionary dynamics. Proc. Biol. Sci. 278 (1716), 2283–2292.
Moxley, C., 2013. Infection of Two Distinct *Trichuris* Sp. Genotypes within and Among Baboon (*Papio ursinus)* Troops on the Cape Peninsula, South Africa. Doctoral dissertation, University of Cape, Town.
Müller-Graf, C.D.M., Collins, D.A., Woolhouse, M.E.J., 1996. Intestinal parasite burden in five troops of olive baboons (*Papio cynocephalus anubis*) in Gombe Stream National Park, Tanzania. Parasitology 112 (5), 489–497.
Müller-Klein, N., Heistermann, M., Strube, C., Morbach, Z.M., Lilie, N., Franz, M., et al., 2019. Physiological and social consequences of gastrointestinal nematode infection in a nonhuman primate. Behav. Ecol. 30 (2), 322–335.
Munene, E., Otsyula, M., Mbaabu, D.A.N., Mutahi, W.T., Muriuki, S.M.K., Muchemi, G. M., 1998. Helminth and protozoan gastrointestinal tract parasites in captive and wild-trapped African non-human primates. Vet. Parasitol. 78 (3), 195–201.
Mwangi, T.W., Bethony, J.M., Brooker, S., 2006. Malaria and helminth interactions in humans: an epidemiological viewpoint. Ann. Trop. Med. Parasitol. 100 (7), 551–570.
Nakagawa, S., Schielzeth, H., 2013. A general and simple method for obtaining R^2 from generalized linear mixed-effects models. Methods in Ecology and Evolution 4 (2), 133–142.
Nava-Castro, K., Muñiz-Hernández, S., Hernández-Bello, R., Morales-Montor, J., 2011. The neuroimmunoendocrine network during worm helminth infections. Invertebr. Surviv. J. 8 (2), 143–152.
Nguyen, N., Fashing, P.J., Boyd, D.A., Barry, T.S., Burke, R.J., Goodale, C.B., et al., 2015. Fitness impacts of tapeworm parasitism on wild gelada monkeys at Guassa, Ethiopia. Am. J. Primatol. 77 (5), 579–594.
Nordling, D., Andersson, M., Zohari, S., Lars, G., 1998. Reproductive effort reduces specific immune response and parasite resistance. Proc. Roy. Soc. Lond. B Biol. Sci. 265 (1403), 1291–1298.
Nunez, G.G., Gentile, T., Costantino, S.N., Sarchi, M.I., Venturiello, S.M., 2005. In vitro and in vivo effects of progesterone on *Trichinella spiralis* newborn larvae. Parasitology 131 (2), 255–259.
Nunn, C.L., Altizer, S., 2006. Infectious Diseases in Primates: Behavior, Ecology, and Evolution. Oxford University Press, Oxford.
Ołdakowski, Ł., , Piotrowska, Ż., Chrząścik, K.M., Sadowska, E.T., Koteja, P., Taylor, J.R., 2012. Is reproduction costly? No increase of oxidative damage in breeding bank voles. J. Exp. Biol. 215 (11), 1799–1805.
Patterson, J.E., Neuhaus, P., Kutz, S.J., Ruckstuhl, K.E., 2013. Parasite removal improves reproductive success of female North American red squirrels (*Tamiasciurus hudsonicus*). PloS One 8 (2).
Pettifer, H.L., 1984. The helminth fauna of the digestive tracts of chacma baboons, *Papio ursinus*, from different localities in the Transvaal. Onderstepoort J. Vet. Res. 51, 161–170.
Pond, C.M., 1977. The significance of lactation in the evolution of mammals. Evolution 177–199.
Pung, O.J., Luster, M.I., 1986. *Toxoplasma gondii*: decreased resistance to infection in mice due to estrogen. Exp. Parasitol. 61 (1), 48–56.
R Core Team, 2020. R: A Language and Environment for Statistical Computing. R Foundation for Statistical Computing.
Ravasi, D.F.C., 2009. Gastrointestinal Parasite Infections in Chacma Baboons (*Papio ursinus*) of the Cape Peninsula, South Africa: the Influence of Individual, Group, and Anthropogenic Factors. Doctoral dissertation, University of Cape, Town.
Ravasi, D.F., O'Riain, M.J., Adams, V.J., Appleton, C.C., 2012. A coprological survey of protozoan and nematode parasites of free-ranging chacma baboons (*Papio ursinus*) in the southwestern Cape, South Africa. African Journal of Wildlife Research 42 (1), 35–44.
Rijhsinghani, A.G., Thompson, K., Bhatia, S.K., Waldschmidt, T.J., 1996. Estrogen blocks early T cell development in the thymus. Am. J. Reprod. Immunol. 36 (5), 269–277.
Rimbach, R., Bisanzio, D., Galvis, N., Link, A., Di Fiore, A., Gillespie, T.R., 2015. Brown spider monkeys (*Ateles hybridus*): a model for differentiating the role of social networks and physical contact on parasite transmission dynamics. Phil. Trans. Biol. Sci. 370 (1669), 20140110.
Rivero, J.C., Inoue, Y., Murakami, N., Horii, Y., 2002. Androgen-and estrogen-dependent sex differences in host resistance to *Strongyloides venezuelensis* infection in Wistar rats. J. Vet. Med. Sci. 64 (6), 457–461.
Roberts, C.W.W., Horsnell, W.G.C.G., 2015. Effects of sex and maternal immunity on protozoan and helminth infections. Sex and Gender Differences in Infection and Treatments for Infectious Diseases. Springer International Publishing, pp. 361–388. https://doi.org/10.1007/978-3-319-16438-0_13.
Romano, M.C., Jiménez, P., Miranda, C., Valdez, R.A., 2015. Parasites and steroid hormones: corticosteroid and sex steroid synthesis, their role in the parasite physiology and development. Front. Neurosci. 9, 224.
Ruch, T.C., 1959. Diseases of Laboratory Primates. Saunders, Philadelphia.
Rushmore, J., Bisanzio, D., Gillespie, T.R., 2017. Making new connections: insights from primate–parasite networks. Trends Parasitol. 33 (7), 547–560.
Sapolsky, R.M., Romero, L.M., Munck, A.U., 2000. How do glucocorticoids influence stress responses? Integrating permissive, suppressive, stimulatory, and preparative actions. Endocr. Rev. 21 (1), 55–89.
Schmid-Hempel, P., 2009. Immune defence, parasite evasion strategies and their relevance for 'macroscopic phenomena' such as virulence. Phil. Trans. Biol. Sci. 364 (1513), 85–98.
Schneider-Crease, I., Griffin, R.H., Gomery, M.A., Bergman, T.J., Beehner, J.C., 2017. High mortality associated with tapeworm parasitism in geladas (*Theropithecus gelada*) in the Simien Mountains National Park, Ethiopia. Am. J. Primatol. 79 (9), e22684.
Schwarzenberger, F., 2007. The many uses of non-invasive faecal steroid monitoring in zoo and wildlife species. Int. Zoo Yearbk. 41 (1), 52–74.
Seppälä, O., Liljeroos, K., Karvonen, A., Jokela, J., 2008. Host condition as a constraint for parasite reproduction. Oikos 117, 749–753.
Setchell, J.M., Bedjabaga, I.B., Goossens, B., Reed, P., Wickings, E.J., Knapp, L.A., 2007. Parasite prevalence, abundance, and diversity in a semi-free-ranging colony of *Mandrillus sphinx*. Int. J. Primatol. 28 (6), 1345–1362.
Shaikh, A.A., Shaikh, S.A., Celaya, C.L., Gomez, I., 1982. Temporal relationship of hormonal peaks to ovulation and sex skin deturgescence in the baboon. Primates 23 (3), 444–452.
Shearer, C.L., Ezenwa, V.O., 2020. Rainfall as a driver of seasonality in parasitism. Int. J. Parasitol.: Parasites and Wildlife 12, 8–12.
Siiteri, P.K., Febres, F., Clemens, L.E., Chang, R.J., Gondos, B., Stites, D., 1977. Progesterone and maintenance of pregnancy: is progesterone nature's immunosuppressant? Ann. N. Y. Acad. Sci. 286, 384–397.
Silk, J.B., Beehner, J.C., Bergman, T.J., Crockford, C., Engh, A.L., Moscovice, L.R., et al., 2010. Female chacma baboons form strong, equitable, and enduring social bonds. Behav. Ecol. Sociobiol. 64 (11), 1733–1747.
Smales, L.R., Harris, P.D., Behnke, J.M., 2009. A redescription of *Protospirura muricola* Gedoelst, 1916 (Nematoda: spiruridae), a parasite of murid rodents. Syst. Parasitol. 72 (1), 15.
Soliman, M.F., Ibrahim, M.M., 2005. Antischistosomal action of atorvastatin alone and concurrently with medroxyprogesterone acetate on *Schistosoma haematobium* harboured in hamster: surface ultrastructure and parasitological study. Acta Trop. 93 (1), 1–9.
Speakman, J.R., 2008. The physiological costs of reproduction in small mammals. Phil. Trans. Biol. Sci. 363 (1490), 375–398.
Stancampiano, L., Gras, L.M., Poglayen, G., 2010. Spatial niche competition among helminth parasites in horse's large intestine. Vet. Parasitol. 170 (1), 88–95.
Stear, M.J., Bishop, S.C., Duncan, J.L., McKellar, Q.A., Murray, M., 1995. The repeatability of faecal egg counts, peripheral eosinophil counts, and plasma pepsinogen concentrations during deliberate infections with *Ostertagia circumcincta*. Int. J. Parasitol. 25 (3), 375–380.
Strait, K., Else, J.G., Eberhard, M.L., 2012. Parasitic diseases of nonhuman primates. In: Abee, C., Mansfield, K., Tardif, S., Morris, T. (Eds.), Nonhuman Primates in Biomedical Research. Academic Press, Oxford, pp. 197–297.
Tait, A.S., Butts, C.L., Sternberg, E.M., 2008. The role of glucocorticoids and progestins in inflammatory, autoimmune, and infectious disease. J. Leukoc. Biol. 84 (4), 924–931.
Telfer, S., Lambin, X., Birtles, R., Beldomenico, P., Burthe, S., Paterson, S., Begon, M., 2010. Species interactions in a parasite community drive infection risk in a wildlife population. Science 330 (6001), 243–246.
Toft, J.D., 1986. The pathoparasitology of nonhuman primates: a review. Primates. Springer, New York, NY, pp. 571–679.
van Doorn, A.C., O'Riain, M.J., Swedell, L., 2010. The effects of extreme seasonality of climate and day length on the activity budget and diet of semi-commensal chacma baboons (*Papio ursinus*) in the Cape Peninsula of South Africa. Am. J. Primatol. 72 (2), 104–112.
Vargas-Villavicencio, J.A., De Leon-Nava, M.A., Morales-Montor, J., 2009. Immunoendocrine mechanisms associated with resistance or susceptibility to parasitic diseases during pregnancy. Neuroimmunomodulation 16 (2), 114–121.
Vidya, T.N.C., Sukumar, R., 2002. The effect of some ecological factors on the intestinal parasite loads of the Asian elephant (*Elephas maximus*) in southern India. J. Biosci. 27 (5), 521–528.
Viney, M.E., Graham, A.L., 2013. Patterns and processes in parasite co-infection. In: Advances in Parasitology, vol. 82. Academic Press, pp. 321–369.
Warburton, E.M., Kohler, S.L., Vonhof, M.J., 2016. Patterns of parasite community dissimilarity: the significant role of land use and lack of distance-decay in a bat–helminth system. Oikos 125 (3), 374–385.
Wasser, S.K., Hunt, K.E., Brown, J.L., Cooper, K., Crockett, C.M., Bechert, U., Milsspaugh, J.J., Larson, S., Monfort, S.L., 2000. A generalized fecal glucocorticoid assay for use in a diverse array of nondomestic mammalian and avian species. Gen. Comp. Endocrinol. 120, 260–275.
Weingrill, T., Gray, D.A., Barrett, L., Henzi, S.P., 2004. Fecal cortisol levels in free-ranging female chacma baboons: relationship to dominance, reproductive state and environmental factors. Horm. Behav. 45 (4), 259–269.
Wilcox, J.J.S., Lane-Degraaf, K.E., Fuentes, A., Hollocher, H., 2015. Comparative community-level associations of helminth infections and microparasite shedding in wild long-tailed macaques in Bali, Indonesia. Parasitology 142 (3), 480–489.
Wyle, F.A., Kent, J.R., 1977. Immunosuppression by sex steroid hormones. The effect upon PHA-and PPD-stimulated lymphocytes. Clin. Exp. Immunol. 27 (3), 407.