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Demographic and Ecological Effects on Patterns of Parasitism in Eastern Chimpanzees (*Pan troglodytes schweinfurthii*) in Gombe National Park, Tanzania

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Abstract

From January 2006 to January 2008, we collected 1,045 fecal samples from 90 individually-recognized, free-ranging, eastern chimpanzees (*Pan troglodytes schweinfurthii*) inhabiting Gombe National Park, Tanzania to determine how patterns of parasitism are affected by demographic and ecological covariates. Seventeen parasite species were recovered, including eight nematodes (*Oesophagostomum* sp., *Necator* sp., *Probstmayria gombensis*, *Strongyloides fulleborni*, *Ascaris* sp., *Trichuris* sp., *Abbreviata caucasica*, and an unidentified strongyle), 1 cestode (*Bertiella* sp.), 1 trematode (Dicrocoeliidae), and 7 protozoa (*Entamoeba coli*, *Entamoeba histolytica/dispar*, *Iodamoeba bütschlii*, *Troglodytella abrassarti*, *Troglocorys cava*, *Balantidium coli*, and an unidentified protozoa). Significant differences were observed in interannual infection prevalence and parasite richness between 2006 and 2007. Intercommunity comparisons demonstrated higher prevalence of parasites for the Mitumba compared with Kasekela chimpanzee community. Prevalence of several parasites was strongly correlated with monthly rainfall patterns for both 2006 and 2007. Subadult chimpanzees had lower prevalence for most parasite species compared with adults in both years and also yielded a lower average parasite species richness. No significant differences were observed between males and females in prevalence in 2006. However, in 2007 the prevalence of *S. fulleborni* and *I. bütschlii* were higher in males than in females. Parasite

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prevalence and richness were substantially higher in this multiyear study compared with previous short-term studies of the gastrointestinal parasites of Gombe chimpanzees. This coupled with the significant interannual and interseasonal variation, demonstrated in this study, emphasizes the importance of multiyear monitoring with adequate sample size to effectively determine patterns of parasitism in wild primate populations.

Keywords

apes; gastrointestinal parasites; health; noninvasive analyses; zoonoses

The chimpanzees of Gombe National Park are arguably the best documented wild ape population (Pusey et al., 2007). However, despite almost 50 years of systematic collection of behavioral and life history data on these chimpanzees, there has been little standardized diagnostic data collected for baseline health monitoring. Baseline prevalence of clinical signs of ill health and patterns of parasitism are necessary to understand both individual animal health status as well as for the assessment of potential ‘outbreaks’: a level of disease or parasitism in excess of what is normally observed. Risk of immobilization for animal safety (i.e., capture myopathy, injury, and death), often precludes disease diagnosis through collection of blood and tissue samples. Therefore, most health evaluations conducted in ape conservation areas, including Gombe, are based on the analysis of noninvasively collected samples such as feces, saliva, or urine. To better characterize and manage the risk of infectious disease, a noninvasive, observational health-monitoring program was implemented in two of the three chimpanzee communities in Gombe in early 2004 (Lonsdorf et al., 2006).

The majority of health studies on wild apes have focused on fecal analysis for parasitological research. Parasites can impact host survival and reproduction directly through pathological effects and indirectly by reducing host condition (Boyce, 1990; Dobson and Hudson, 1992; Hudson et al., 1992; Coop and Holmes, 1996). Severe parasitosis can lead to blood loss, tissue damage, spontaneous abortion, congenital malformations, and death (Chandra and Newberne, 1977; Despommier et al., 1995). However, less severe infections are more common and may impair nutrition, travel, feeding, predator escape, and competition for resources or mates, or increase energy expenditure (Dobson and Hudson, 1992; Hudson et al., 1992; Coop and Holmes, 1996; Packer et al., 2003). Unfortunately, the majority of studies are not directly comparable because of methodological differences, thereby making it exceedingly difficult to assess and understand meaningful differences both within a population over time and between populations (Gillespie, 2006, 2010).

At Gombe, the first published survey of chimpanzee gastrointestinal parasites (repeated sampling of 32 chimpanzees) was performed in 1973 (File et al., 1976), followed by a three-week survey in 1989 (single sampling of 20 chimpanzees, Murray et al., 2000), another short survey in 1991 (single sampling of 39 chimpanzees, Nutter, personal communication), and finally a nine-month study in 2005 (repeated sampling of 60 chimpanzees, Bakuza and Nkwengulila, 2009). All of these studies recovered eight to twelve parasite species.

Potentially pathogenic rhabditoid and strongyle nematodes (i.e., *Oesophagostomum* sp., *Necator* sp., and *Strongyloides fulleborni*) were prevalent across all study periods, but zoonotic parasites, typically associated with humans and livestock (i.e., *Ascaris* sp.), were not confirmed although some of the studies specifically looked for them. The current health-monitoring effort at Gombe began in 2004 and includes observational assessments of clinical signs of ill health and fecal sample collection for analysis of parasites (Lonsdorf et al., 2006 for specific monitoring methodology). Our objective in this study is to characterize patterns of chimpanzee health, specifically parasitism, in relation to community, age, sex, and interannual and interseasonal variation. Such an approach provides the opportunity to determine the factors that have the strongest influence on the ecology of parasitism in wild chimpanzee populations, while guiding scientists and conservationists to differentiate between natural baseline patterns of chimpanzee parasitism and changes in patterns of parasitism that represent threats to the health of chimpanzees and other endangered apes.

MATERIALS AND METHODS

Study site

Gombe National Park, Tanzania—a 35 km^2 park on the eastern shore of Lake Tanganyika in western Tanzania—is the site of the longest continuous field study of wild chimpanzees in the world. The majority of the park consists of hills sloping westward from a rift escarpment ~1,500 m above sea level and is home to seven species of nonhuman primates (Wallis and Lee, 1999). Two large villages flank the park on its northern and southern borders (Fig. 1). The park is restricted to researchers, eco-tourists, park management staff, local field assistants, and associated families. Local fishermen were allowed to camp temporarily on the beach during certain fishing seasons until 2003 (Wallis and Lee, 1999).

Study subjects

Between January 2006 and January 2007, fecal samples were collected from 74 individual chimpanzees (28 males and 47 females) consisting of 25 sub-adults (age 10 or younger), 39 prime, and 10 old (age over 30) individual chimpanzees from Gombe National Park, Tanzania. In the following year between January 2007 and January 2008, fecal samples were collected from 79 individual chimpanzees (29 males and 48 females), consisting of 29 sub-adults, 39 prime, and 12 old individual chimpanzees.

Within Gombe National Park, samples were taken from two communities habituated by the Jane Goodall Institute and other collaborators. Kasekela, the larger community, is situated at the center of the park and has been studied continuously since research began in 1960. Mitumba, the smaller Northern community, was habituated by the mid-1990s and is in proximity to Mwamgongo, a large village on the park's northern border (Pusey et al., 2007) (Fig 1). The 2006 sample set comprised of 52 members of the Kasekela community and 22 members from Mitumba. The 2007 sample set comprised of 59 members of the Kasekela community and 24 members from Mitumba.

Sample collection

During the course of research observation, 1,038 fecal samples were collected from known, individually identified chimpanzees. On average 6.9 samples were collected per individual over the course of each year. (N = 407 in 2006; N = 631 in 2007). All feces collected were freshly voided by known individuals. The amount of feces necessary to fill the tube to a premeasured and premarked fill line was collected in ParaPak® containers (Meridian Bioscience, Cleveland, OH) prefilled with 15 ml of 10% formalin fixative to preserve the sample. Care was taken to avoid collecting soil, foliage, or standing water contaminants. The samples were sealed with ParaFilm®, (Pechiney Plastic Packaging, Chicago, IL) and the tube was shaken to mix the sample with the formalin solution. Each tube was labeled with date of collection, observer, location, and animal identifier and then stored until shipped to the United States under appropriate regulations for analysis.

Parasitological analyses

Helminth eggs, larvae, and protozoal cysts were recovered via sodium nitrate floatation and fecal sedimentation. Both techniques utilized standardized fecal floatation and sedimentation methodologies, as previously described in detail by Gillespie (2006) and advocated by the Great Ape Survival Plan (GRASP) and the IUCN/ SSC Primate Specialist Group (Leendertz, 2010). Slides prepared by each method were examined with compound microscope and parasites were identified on the basis of egg, larvae, or cyst coloration, shape, contents, and size. Each parasite species per sample was quantified and representatives measured at 400× to the nearest 0.1 μm with an ocular micrometer. If needed, one drop of Lugol's iodine solution was added to aid in species identification. Unknown and representative parasite species were also photographed for later identification and comparison (Fig. 2).

Control for sample bias

Although a standardized health monitoring regime has been implemented for chimpanzees in Gombe, there was uneven sampling of individuals because of factors such as degree of habituation or lack of travel after defecation, resulting in anywhere from 1 to 15 samples collected for a given individual per year. Huffman et al. (1997) demonstrated that significant reporting bias is created when one uses the number of samples instead of the number of individuals to calculate the prevalence of parasitic infection in a population. To eliminate such bias, prevalence was calculated as the proportion of individual chimpanzees positive for a given parasite divided by the total number of chimpanzees examined. If a single sample was positive for a parasite species, the corresponding chimpanzee was considered positive for the collection period, the year for most analyses, and the month for analyses of seasonality. Gastrointestinal parasite prevalence and richness data were then calculated for each individual categorically by community, age, and sex. Prevalence data for select parasites was also compared with monthly rainfall patterns to determine seasonal trends. Monthly rainfall patterns were calculated as the number of days with measureable precipitation per month as reported by Weather Underground (http://www.wunderground.com/history/station/63801/) for Kigoma, the regional capital located several kilometers south of Gombe National Park. We recognize that quantity of rainfall

(i.e., cumulative rainfall per month) represents a better proxy for seasonality. Unfortunately, this data is not currently available for Gombe National Park during the years of this study.

Statistical analyses

The degree of independence between community and sex with respect to parasite prevalence was estimated by 2 × 2 contingency analyses for calculating Chi-square (significance at $P < 0.05$, DF = 1) using the Centers for Disease Control Epi-Info™ Version 3.4.3 software. Degree of independence between age with respect to parasite prevalence was estimated by a 2 × 3 contingency analysis with Epi-Info™ Version 3.4.3 software (significance at $P < 0.05$, DF = 2). Correlation coefficients between seasonality of infection and frequency of wet days per month were used to determine the strength and direction of a linear relationship (significance at $r > 0.5$ or $r < -0.5$). Parasite richness data was analyzed using a Student's *t*-test to determine if mean richness data were significantly distinct (significance at $P < 0.05$, DF = $(n_1 + n_2 - 2)$).

RESULTS

Parasite species identified

A total of 17 species of parasites were recovered, including eight nematodes (*Oesophagostomum* sp., *Necator* sp., *Probstmayria gombensis*, *Strongyloides fulleborni*, *Ascaris* sp., *Trichuris* sp., *Abbreviata caucasica*, and an unidentified strongyle), 1 cestode (*Bertiella* sp.), 1 trematode (Dicrocoeliidae), and 7 protozoa (*Entamoeba coli*, *Entamoeba histolytica*, *Iodamoeba bütschlii*, *Troglodytella abrassarti*, *Troglocorys cava*, *Balantidium coli*, and an unidentified protozoa). This is the first documentation of *Ascaris* sp., the dicrocoeliid liver fluke, and *Bertiella* sp. in Gombe chimpanzees (Table 1, Fig. 2). Identification to the species level was not always possible because of inherent morphological similarity between certain strongyles. Annual parasite prevalence was calculated as the percentage of individuals positive for a parasite of the total number of individuals included in each year. In 2006, prevalence ranged from 95.95% for *T. abrassarti* to 1.35% for an unidentified trematode. In 2007, prevalence ranged from 100.00% for *Oesophagostomum* sp. to 5.19% for the dicrocoeliid liver fluke (Table 1).

Community, age, and sex differences by year

Comparison of parasite prevalence between chimpanzee communities demonstrated that *I. bütschlii* was significantly more prevalent in Mitumba than Kasekela for 2006. In 2007, Mitumba showed significantly higher prevalence of the dicrocoeliid liver fluke, *B. coli*, and an unidentified entodiniomorph (Table 2). In 2006, prevalence differed significantly with age for four taxa of parasite. Sub-adults were observed to have the highest prevalence for *S. fulleborni*, individuals in the prime group had the highest prevalence for *A. caucasica*, and *Ascaris* sp prevalence was highest for individuals in the old group. Sub-adults had significantly lower prevalence of *T. abrassarti* than individuals in either of the other age categories (Table 3). In 2007, prevalence differed significantly with age for five parasite taxa. Individuals in the prime age group were observed to have higher prevalence of *Necator* sp., *A. caucasica*, *Ascaris* sp., and *E. coli* (Table 3). Sub-adults were again observed to have a significantly lower prevalence of *T. abrassarti* than either of the other age categories

(Table 3). No significant differences were observed between males and females in prevalence of any of the 17 parasite taxa in 2006. However, in 2007 the prevalence of *S. fulleborni* and *I. bütschlii* were significantly higher in males than in females (Table 4).

Parasite richness

Mean parasite richness for 2007 (7.83 parasite species per individual) was significantly higher than mean parasite richness for 2006 (5.85 species per individual, $P < 0.01$). No significant differences were found in average parasite richness between males and females for either 2006 or 2007. Likewise, no significant differences were found in average parasite richness between the communities of Kasakela and Mitumba. No significant difference was found between the three age groups analyzed in 2006. However, in 2007, the average richness of old individuals (9.00 species per individual) was significantly higher than the richness of sub-adults (6.79 species per individual, $P < 0.01$).

Seasonality

Prevalence of pathogenic parasites was correlated with the percentage of wet days per month (index of seasonality). A significant positive correlation of prevalence with rainfall was observed for *Oesophagostomum* sp., *Necator* sp., and *S. fulleborni* in 2006 (Fig. 3). The same observations were found in 2007 with the exception of *Oesophagostomum* sp., which remained at a high prevalence throughout the year. A significant negative correlation of prevalence with rainfall was observed for *A. caucasica* in 2007 (Fig. 3).

DISCUSSION

This study provides the first longitudinal study of patterns of parasitism in the chimpanzee communities of Gombe National Park. The significant interannual and interseasonal variation in parasite prevalence and richness observed emphasizes the need for long-term monitoring to effectively determine patterns of parasitism in wild primate populations. In addition, parasite prevalence and richness were substantially higher in this study compared with previous short-term studies of the gastrointestinal parasites of Gombe chimpanzees, highlighting the need for long-term monitoring with adequate sample size and appropriate analyses.

The first published survey of Gombe chimpanzee gastrointestinal parasites ($n = 32$) was performed in 1973 using formalin–ether concentration and zinc sulfate flotation (File et al., 1976), followed by a three-week survey in 1989 ($n = 20$) using direct smear and zinc sulfate flotation (Murray et al., 2000), another short survey occurred in 1991 ($n = 39$) using formalin–ether concentration and direct smear (Nutter, personal communication), and finally a nine-month study in 2005 ($n = 60$) employed formalin–ether concentration and acid-fast staining (Bakuza and Nkwengulila, 2009). Unfortunately, many of the methodologies used in these earlier studies are specialized methods of clinical veterinary medicine that are simply not adequate for epidemiological parasite surveys. For example, zinc sulfate is used as a flotation solution in a clinical setting when a patient's symptoms are suggestive of giardiasis. Zinc sulfate is ideal for recovering *Giardia* sp. cysts from a fecal sample, but is far less effective at recovering the diversity of other parasites that affect wild primates (OIE,

2004; Zajac and Conboy, 2006). Likewise, the formalin–ether concentration technique was developed to investigate the parasites of carnivorous companion animals (i.e., cats and dogs) and humans eating an animal-product dominated Western diet. This technique is far less appropriate for the majority of free-ranging primates whose diets are primarily frugivorous and/or folivorous (MAFF, 1979). Lastly, although direct smear and fecal sedimentation are ideal for detection of protozoa in fresh fecal material and flukes, respectively, these techniques are inadequate for general surveys as they detect only a proportion of the parasites present in a given fecal sample (OIE, 2004; Zajac and Conboy, 2006). Thus, it is likely that the prevalence and richness values observed in the previous studies are underestimates. The methods employed in the current study were developed to provide a standardized set of protocols that maximize recovery of the broadest spectrum of gastrointestinal parasites while using equipment and supplies that allow the flexibility of field- or laboratory-based sample screening (Gillespie, 2006; Gillespie et al., 2008). Community dynamics such as immigration and emigration, increased anthropogenic habitat change, and declining chimpanzee population size within the park are additional covariates that may account for some of the differences in parasite prevalence and richness observed over time at Gombe (Pusey et al., 2007).

Community, age, and sex differences

Several species of parasites were found at higher prevalence in the Mitumba chimpanzees compared with the Kasekela community, including *B. coli*, a pathogenic ciliate known to cause morbidity and mortality in humans and captive apes (Teare and Loomis, 1982). The Mitumba community's range is constrained by the patrols of the larger Kasekela group to the south and the ever-expanding human populations at the northern boundary of the park. Consequently, Mitumba chimpanzees may have a higher risk of infection because of recurrent use of a limited core habitat (Freeland, 1980, Pusey et al., 2007). In addition, unlike the Kasekela community, Mitumba chimpanzees crop raid outside the park and overlap with goats that travel into the park while grazing during the dry season each year (Shadrack Kamenya, personal communication), increasing the risk of zoonotic transmission of parasites from people and livestock to chimpanzees. This is consistent with previous studies that have demonstrated that anthropogenic disturbance can be associated with an increase in the prevalence of gastrointestinal parasites (Gillespie et al., 2005a; Gillespie and Chapman, 2006, 2008).

Unlike previous short-term studies that found no differences in parasite prevalence relative to age for eastern chimpanzees at Gombe (File et al., 1976) and Ngogo, Uganda (Muehlenbein, 2005), prevalence differed significantly with age in the current multiyear study. Subadults were more likely than others to be infected with *S. fulleborni* and less likely than others to be infected with *T. abrassarti*. Gotoh (2000) found a similar trend for *S. fulleborni* in macaques (*Macaca fuscata*). *T. abrassarti* is a symbiont involved in hindgut fermentation that likely establishes via repeated inoculation via fecal contamination. Thus it is not suprising that prevalence of this organism is higher in older age classes (Modry et al., 2009). Prime individuals had the highest prevalence for a number of parasites including several pathogenic geohelminths (i.e., *Necator* sp. and *Ascaris* sp.) and overall parasite richness was highest in old individuals. Such accumulation of infection with age is widely

noted in nature and is typically associated with development of some degree of resistance to the pathology of prevalent parasites, allowing for passive infections and opportunities for other organisms to accumulate (Anderson, 1992).

Prevalence of *S. fulleborni* and *I. bütschlii* were higher for male compared with female chimpanzees. Perhaps this reflects energy and nutrient stress associated with maintaining social dominance (Hausfater and Watson, 1976), which may result in an increased susceptibility to infection (Gulland, 1992; Milton, 1996). However, if this is the case, it is not clear why infection prevalence is not higher for other parasite species in males compared with females.

Seasonality

Evidence of general patterns of seasonal infection in primates is equivocal, with clear seasonal patterns of infection in some cases (Freeland, 1977; Huffman et al., 1997) and no clear pattern for others (Gillespie et al., 2004, 2005b). The significant positive correlation of prevalence with rainfall observed for a number of strongyle and rhabditoid nematodes (i.e., *Oesophagostomum* sp., *Necator* sp., and *S. fulleborni*) in this study largely mirrored the pattern reported for chimpanzees at Mahale to the south of Gombe (Huffman et al., 1997), aside from a significant negative correlation of prevalence with rainfall observed for *A. caucasica*. At sites with marked seasonality, such as Gombe and Mahale, it is likely that infective stages of many gastrointestinal parasites can persist longer during the wet season because of reduced dessication, resulting in higher infection risk for resident primates (Huffman et al., 1997). Our understanding of the interplay between seasonality and chimpanzee parasitism will be greatly improved as better approximations of seasonality become available for more ape research sites.

Interannual trends

Parasite richness and the prevalence of many parasites examined were significantly higher in 2007 compared to 2006 in Gombe chimpanzees. As 2007 was a substantially wetter year with less marked seasonality, these patterns may simply reflect natural, interannual variability associated with rainfall. However, the observed increases in parasite richness and prevalence may reflect other environmental or anthropogenic stressors impacting overall chimpanzee population health, and further study will be needed to explore these possibilities.

Pathogenic parasites

Rhabditoid and strongyle nematodes—The strongyle or rhabditoid nematodes that infect Gombe chimpanzees (i.e., *Oesophagostomum* sp., *Necator* sp., *S. fulleborni*, and the unidentified strongyle) have the capacity to cause substantial pathology and death in primates. Heavy infections with these parasites have been associated with mucosal inflammation, ulceration, iron deficiency anemia, protein malnutrition, dysentery, weight loss, and death in primates (McClure and Guilloud, 1971; DePaoli and Johnsen, 1978; Harper et al., 1982; Roberts and Janovy, 2009). Of these parasites, *Oesophagostomum* sp., the nodule worms, are best understood in terms of their capacity to cause pathology in humans and nonhuman primates. In humans, they cause oesophagostomosis resulting from

the formation of granulomas, caseous lesions, or abscesses in intestinal walls. Associated clinical signs of parasitism by *Oesophagostomum* spp. include diarrhea, weight loss, abdominal pain, and secondary bacterial infections (Brack, 1987). Although high *Oesophagostomum* sp. prevalence is common in wild chimpanzee populations (File et al., 1976; Ashford et al., 2000; Krief et al., 2005), evidence of the pathogenicity of this agent in wild apes has been equivocal, with chimpanzees from sites in Uganda and Cote D'Ivoire developing nodular esophagostomosis without associated severe clinical signs (Krief et al., 2008), whereas at sites in Tanzania including Gombe, chimpanzees suffer from oesophagostomosis-associated morbidity and mortality (Huffman et al., 1997; Karen Terio, personal communication). Thus the extremely high prevalence of *Oesophagostomum* sp. (95–100%) observed in this study and lack of a seasonal reduction in *Oesophagostumum* sp. prevalence during the dry season of 2007 suggests a qualitative shift in host–parasite dynamics that may reflect altered host immunity or changes in parasite virulence. Further integration of behavioral health monitoring data, patterns of infection, and information provided by opportunistic necropsies will better elucidate the role of *Oesophagostomum* sp. and the other rhabditoid and strongyle nematodes in morbidity and mortality in Gombe chimpanzees.

Amoeba and ciliates—The majority of amoeba (i.e., *Iodamoeba butschlii*, *Entamoeba coli*) and ciliates (i.e., *Troglodytella abrassarti* and *Troglocorys cava*) found to infect Gombe chimpanzees are nonpathogenic commensals or symbionts (Tokiwa et al., 2010). The exceptions are *E. histolytica* and *B. coli*. *Entamoeba histolytica* ranks second in worldwide causes of human morbidity by parasitic infections, causing dysentery and colitis (Laughlin and Temesvari, 2005). Although humans are thought to be the primary reservoir of this pathogen, it has been described in various free-ranging primates including chimpanzees (Lilly et al., 2002; Gillespie et al., 2004; Gillespie et al., 2005b). Mortality associated with *E. histolytica* infection has been observed in a diversity of captive primates as divergent as apes (Patten, 1939), colobines (Frank, 1982; Loomis et al., 1983), and Atelines (Amyx et al., 1978). However, pathogenicity of this agent in wild primates remains to be determined. *Entamoeba histolytica* and *E. dispar* (nonpathogenic) are morphologically similar and cannot be differentiated by standard microscopic examination. Molecular confirmations will be required to determine definitively if Gombe chimpanzees are infected with *E. histolytica*.

Balantidium coli is the only species of ciliate protozoan known to be pathogenic to humans. Clinical symptoms of Balantidiasis include diarrhea, dysentery, and colitis (Roberts and Janovy, 2009). *Balantidium coli* is common in captive primates where it is rarely associated with disease (Nakauchi, 1999; Drevon-Gaillot et al., 2006). *Balantidium coli* has been observed previously in ape populations experiencing extensive overlap with humans (Lilly et al., 2002). However, pathogenicity of this agent in wild apes remains to be determined.

Ascarids—Ascariasis is the most common helminth-associated human disease worldwide. Infection can cause morbidity and death, by compromising nutritional status, affecting cognitive processes, inducing tissue reactions, such as granuloma, and provoking intestinal obstruction or rectal prolapse (Roberts and Janovy, 2009). Like, *B. coli*, *Ascaris* sp. has been

observed previously in ape populations experiencing overlap with humans (Lilly et al., 2002). Mortality associated with *Ascaris* sp. infection has been observed in a diversity of captive primates (McClure and Guilloud, 1971; Orihel and Seibold, 1972). However, pathogenicity of this agent in wild primates remains to be determined.

Chimpanzee and ecosystem health

Though parasites are a normal component of a functioning ecosystem and low-intensity infections are often asymptomatic (Anderson and May, 1979), anthropogenic change may result in altered transmission rates, parasite host range, and parasite virulence (Gillespie et al., 2005a; Gillespie and Chapman, 2006, 2008). Resultant changes in host immune status and susceptibility may influence clinical presentation of disease, resulting in elevated morbidity and mortality, and ultimately, population declines for chimpanzees and other species. The lack of a seasonal reduction in *Oesophagostumum* sp. prevalence during the 2007 dry season observed during this study may reflect such a qualitative shift in host–parasite dynamics. Determining the health status of chimpanzee populations prior to large-scale disruption or indications of population stress provides a baseline that may alert managers of potential threats early enough to maintain compromised populations when anthropogenic stressors intensify.

In addition, the presence and diversity of symbiotic protozoa, such as *Troglodytella abrassarti* and *Troglocorys cava* may provide an index of gastrointestinal function in wild ape populations. These symbionts aid in digestion and can be used as a proxy for population health in chimpanzees and help indicate the health and nutritional quality of a community (Modry et al., 2009; Tokiwa et al., 2010). Chimpanzees that inhabit pristine habitats with minimal anthropogenic impact exhibit greater prevalence and diversity of symbiotic ciliate species. For example, the remote Goualougo Triangle chimpanzee population in Congo experiences little known exposure to zoonotic pathogens and harbors eight species of symbiotic gut ciliates (Gillespie et al., unpublished data). At the other end of the spectrum, captive and semicaptive ape populations often have only one or no symbiotic gut ciliates (Petrzelkova et al., 2010). The presence of only two symbiotic protozoa in Gombe chimpanzees suggests a shift from natural patterns of parasitism in this population.

In addition to the potential role of naturally occurring parasites to great ape morbidity and mortality and commensals on health, it is important to also consider introduced parasitic risks from humans, livestock, and other species. Protocols have been implemented in recent years at Gombe to reduce the human-induced disease risk to the chimpanzees. These include increasing the minimum viewing distance for researchers, implementing a quarantine period for researchers, improving sanitation (including moving pit toilets and covering trash pits) in staff quarters, and constructing mesh-protected areas for dish and clothes washing to prevent raiding by baboons. Considering our findings of *Ascaris* sp. and *B. coli*, two human-associated zoonotic pathogens, further work is needed to elucidate the relative threat of sympatric wildlife such as baboons and resident people and livestock on patterns of parasite transmission and persistence in Gombe chimpanzees. In addition, considering recent evidence from Gombe demonstrating an AIDS-like syndrome associated with SIVcpz infection resulting in reduced health, reproduction, and lifespan of wild chimpanzees (Keele

et al., 2009), the observed increases in parasite richness and prevalence observed in this study may reflect environmental or anthropogenic stressors impacting overall chimpanzee population health. Considering the recognized health importance of parasitic coinfections and opportunistic infections associated with HIV in humans (Mwachari et al., 1998), future work will be needed to determine if similar coinfections represent a novel threat to ape health and survival.

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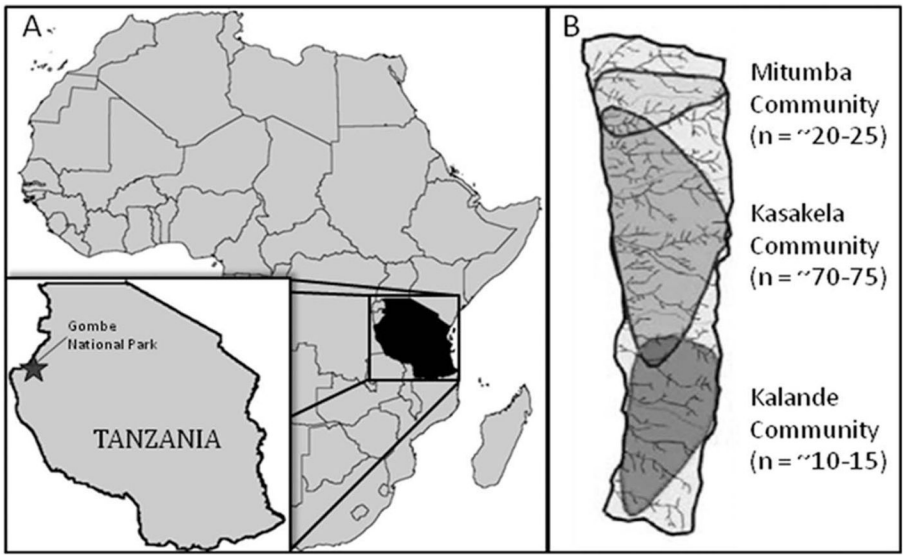

Fig. 1.

The study site, Gombe National Park. **A**: Relative location of Gombe National Park within Africa and Tanzania. **B**: Ranges of the three chimpanzee groups of Gombe; Mitumba, Kasekela, and Kalende (*n* = chimpanzee community size).

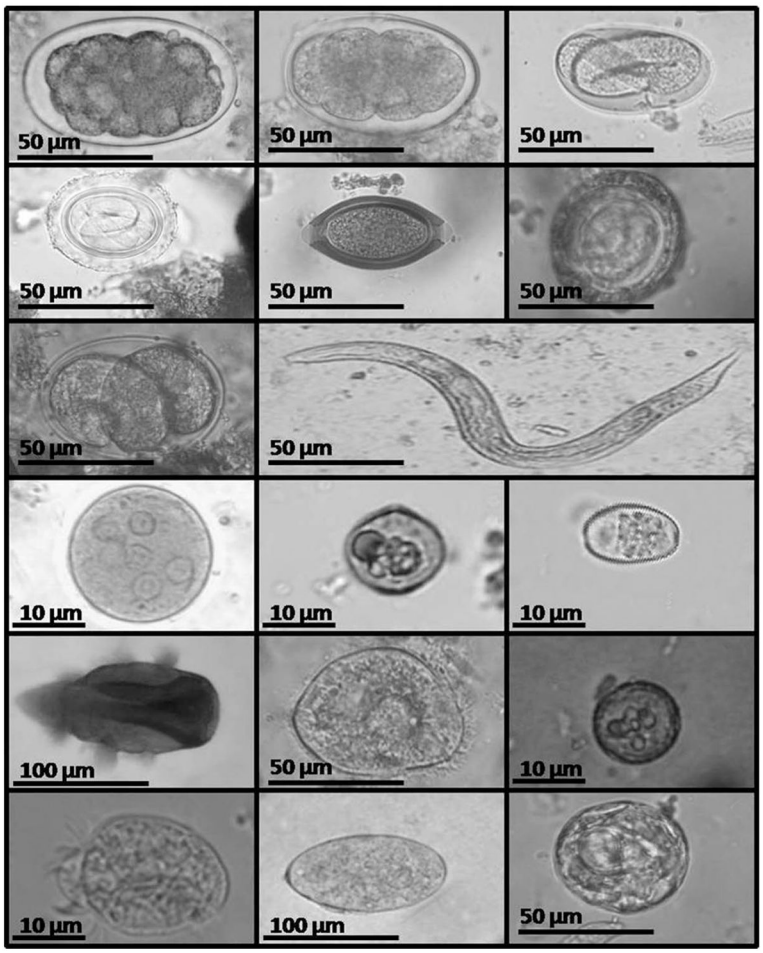

Fig. 2.

Gastrointestinal parasites recovered from fecal samples of Eastern chimpanzees (*Pan troglodytes schweinfurthii*) in Gombe National Park, Tanzania. At top from left to right: **Oesophagostomum* sp., **Necator* sp., **Strongyloides fulleborni*, *Abbreviata caucasica*, *Trichuris* sp., **Ascaris* sp., *Larvated strongyle, *Probstmayria gombensis*, *Entamoeba coli*, **Entamoeba histolytica/ dispar*, *Iodamoeba bütschlii*, *Troglodytella abrassarti*, **Balantidium coli*, Unidentified protozoan, *Troglocorys cava*, *Dicrocoeliidae* sp., *Bertiella* sp. (* *denotes* pathogenic).

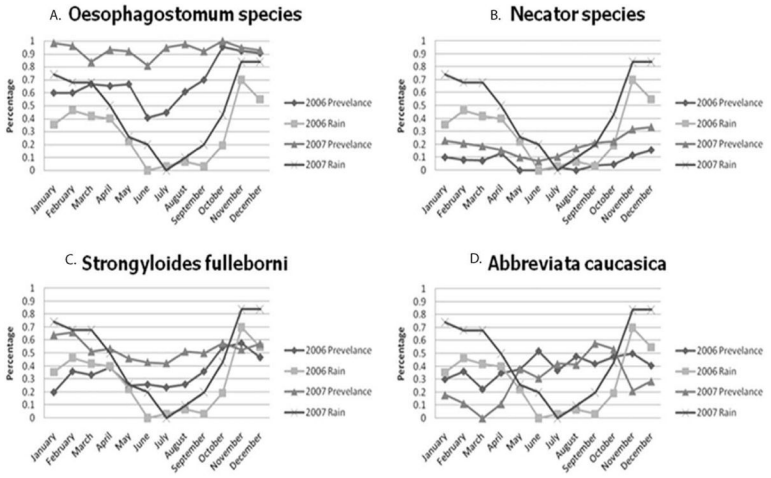

Fig. 3.

Independent comparisons of percentage of rainy days per month to monthly overall prevalence for specific gastrointestinal parasites of chimpanzees in Gombe National Park, Tanzania for 2006 and 2007. **A**: *Oesophagostomum* sp., **B**: *Necator* sp., **C**: *Strongyloides fulleborni*, and **D:** *Abbreviata caucasica. Oesophagostomum* sp., *Necator* sp., and *Strongyloides fulleborni* each showed significant positive correlation between seasonality and monthly prevalence ($r > 0.5$). *Abbreviata caucasica* showed significant negative correlation between seasonality and monthly prevalence ($r < 0.5$).

TABLE 1

Overall prevalence of parasite species in chimpanzees (*Pan troglodytes*) at Gombe National Park, Tanzania in 2006 and 2007

	Overall prevalence	
Parasite species	**2006 (*n* = 74 individuals)**	**2007 (*n* = 79 individuals)**
Nematodes		
**Oesophagostomum* sp.	94.59%	100.00%
**Necator* sp.	25.68%	71.43%
**Strongyloides fulleborni*	74.32%	92.21%
Abbreviata caucasica	79.73%	72.73%
Trichuris sp.	2.70%	15.58%
**Ascaris* sp.	9.46%	23.38%
* Unidentified strongyle	21.62%	29.87%
Probstmayria gombensis	6.76%	48.05%
Protozoa		
Entamoeba coli	51.90%	53.25%
**Entamoeba histolytica*	70.89%	72.73%
Iodamoeba bütschlii	65.82%	67.53%
Troglodytella abrassarti	95.95%	92.21%
**Balantidium coli*	0.00%	10.39%
Unidentified protozoal cyst	2.70%	9.09%
Troglocorys cava	0.00%	19.48%
Trematodes		
Dicrocoeliidae sp.	1.35%	5.19%
Cestodes		
Bertiella sp.	12.16%	0.00%

* Denotes high pathogenic potential.

TABLE 2

Comparison of gastrointestinal parasite prevalence between the Kasekela and Mitumba communities of chimpanzees (*Pan troglodytes*) at Gombe National Park, Tanzania in 2006 and 2007

	2006				2007			
Parasite species	**K (*n* = 52)**	**M (*n* = 22)**	**X^2**	**Sig.**	**K (*n* = 59)**	**M (*n* = 24)**	**X^2**	**Sig.**
Nematodes								
Oesophagostomum sp.**	96.15%	90.91%	0.83	NS	98.31%	100.00%	0.41	NS
Necator sp.**	26.92%	22.73%	0.14	NS	64.41%	70.83%	0.32	NS
*Strongyloides fulleborni***	73.08%	77.27%	0.14	NS	88.14%	87.50%	0.01	NS
Abbreviata caucasica	84.62%	68.18%	2.58	NS	72.88%	66.67%	0.32	NS
Trichuris sp.	3.85%	0.00%	0.87	NS	18.64%	4.17%	2.89	NS
Ascaris sp.**	13.46%	0.00%	3.27	NS	23.73%	16.67%	0.5	NS
Unidentified strongyle**	25.00%	13.64%	1.18	NS	30.51%	25.00%	0.25	NS
Probstmayria gombensis	7.69%	4.55	0.24	NS	44.07%	45.83%	0.02	NS
Protozoan								
Entamoeba coli	65.38%	45.45%	2.55	NS	54.24%	37.50%	1.91	NS
*Entamoeba histolytica/dispar***	44.23%	31.82%	0.99	NS	67.80%	66.67%	0.01	NS
Iodamoeba bütschlii	25.00%	59.09%	7.88	P <0.01	61.02%	70.83%	0.71	NS
Troglodytella abrassarti	94.23%	100.00%	1.32	NS	91.53%	91.67%	0	NS
*Balantidium coli***	0.00%	0.00%	NA	NA	5.08%	20.83%	4.86	P <0.05
Unidentified protozoan	3.85%	0.00%	0.87	NS	8.47%	8.33%	0	NS
Troglocorys cava	0.00%	0.00%	NA	NA	15.25%	25.00%	1.09	P <0.05
Trematodes								
Dicrocoeliidae sp.	1.92%	0.00%	0.43	NS	1.69%	12.50%	4.34	P <0.05
Cestodes								
Bertiella sp.	7.69%	22.73%	3.27	NS	0.00%	0.00%	NA	NA

Abbreviations: K, Kasekela; M, Mitumba; NS, P >0.05; NA, no X^2 performed due to 0% prevalence.

** Denotes high pathogenic potential and *n* denotes number of individuals.

TABLE 3

Comparison of gastrointestinal parasite prevalence among juvenile, prime, and old chimpanzees (*Pan troglodytes*) at Gombe National Park, Tanzania in 2006 and 2007

	2006					2007				
Parasite species	**Juvenile (*n* = 25)**	**Prime (*n* = 39)**	**Old (*n* = 10)**	**X^2**	**Sig.**	**Juvenile (*n* = 29)**	**Prime (*n* = 39)**	**Old (*n* = 12)**	**X^2**	**Sig.**
Nematodes										
Oesophagostomum sp.**	92.00%	94.87%	100.00%	0.91	NS	100.00%	100.00%	97.44%	1.06	NS
Necator sp.**	24.00%	25.64%	30.00%	0.13	NS	48.28%	83.33%	79.49%	8.98	*P* <0.02
*Strongyloides fulleborni***	92.00%	66.67%	60.00%	6.37	*P* <0.05	100.00%	83.33%	84.62%	5.07	NS
Abbreviata caucasica	64.00%	89.74%	80.00%	6.25	*P* <0.05	55.17%	83.33%	82.05%	6.86	*P* <0.05
Trichuris sp.	4.00%	0.00%	10.00%	3.27	NS	20.69%	25.00%	7.69%	3.31	NS
Ascaris sp.**	0.00%	10.26%	30.00%	7.57	*P* <0.05	10.34%	50.00%	23.08%	7.67	*P* <0.05
Unidentified strongyle**	24.00%	20.51%	20.00%	0.13	NS	31.03%	41.67%	25.64%	1.15	NS
Probstmayria gombensis	4.00%	10.26%	0.00%	1.78	NS	41.38%	50.00%	48.72%	0.44	NS
Protozoans										
Entamoeba coli	48.00%	64.10%	70.00%	2.17	NS	44.83%	91.67%	46.15%	8.70	*P* <0.02
*Entamoeba histolytica/dispar***	32.00%	43.59%	50.00%	1.28	NS	55.17%	75.00%	79.49%	4.85	NS
Iodamoeba bütschlii	44.00%	28.21%	40.00%	3.27	NS	58.62%	66.67%	69.23%	0.84	NS
Troglodytella abrassarti	88.00%	100.00%	100.00%	6.13	*P* <0.05	79.31%	100.00%	100.00%	11.41	*P* <0.01
*Balantidium coli***	0.00%	0.00%	0.00%	NA	NA	3.45%	16.67%	12.82%	2.32	NS
Unidentified protozoan	4.00%	0.00%	10.00%	3.27	NS	6.90%	0.00%	12.82%	2.08	NS
Troglocorys cava	0.00%	0.00%	0.00%	NA	NA	20.69%	33.33%	12.82%	2.65	NS
Trematodes										
Dicrocoeliidae sp.	0.00%	2.56%	0.00%	0.91	NS	3.45%	0.00%	7.69%	1.37	NS
Cestodes										
Bertiella sp.	8.00%	12.82%	20.00%	1.00	NS	0.00%	0.00%	0.00%	NA	NA

Abbreviations: NS, *P* >0.05; NA, no X^2 performed due to 0% prevalence.

** Denotes high pathogenic potential and *n* denotes number of individuals.

TABLE 4

Comparison of gastrointestinal parasite prevalence between male and female chimpanzees (*Pan troglodytes*) at Gombe National Park, Tanzania in 2006 and 2007

	2006				2007			
Parasite species	**Male (*n* = 28)**	**Female (*n* = 47)**	**X^2**	**Sig.**	**Male (*n* = 29)**	**Female (*n* = 48)**	**X^2**	**Sig.**
Nematodes								
Oesophagostomum sp.**	96.43%	93.48%	0.3	NS	100.00%	100.0 0%	NA	NA
Necator sp.**	32.14%	21.74%	0.99	NS	79.31%	66.67%	1.42	NS
*Strongyloides fulleborni***	85.71%	67.39%	3.06	NS	100.00%	87.50%	3.93	*P* <0.05
Abbreviata caucasica	85.71%	76.09%	1	NS	75.86%	70.83%	0.23	NS
Trichuris sp.	3.57%	2.17%	0.13	NS	24.14%	10.42%	2.59	NS
Ascaris sp.**	14.29%	6.52%	1.23	NS	31.03%	18.75%	1.52	NS
Unidentified strongyle**	17.86%	23.91%	0.38	NS	24.14%	33.33%	0.73	NS
Probstmayria gombensis	10.71%	4.35%	1.12	NS	51.72%	45.83%	0.25	NS
Protozoan								
Entamoeba coli	60.71%	58.70%	0.03	NS	55.17%	52.08%	0.07	NS
*Entamoeba histolytica/dispar***	50.00%	34.78%	1.67	NS	72.41%	72.92%	0	NS
Iodamoeba bütschlii	35.71%	34.78%	0.01	NS	79.31%	60.42%	6.91	*P* <0.01
Troglodytella abrassarti	93.33%	97.83%	1.1	NS	93.10%	91.67%	0.05	NS
*Balantidium coli***	10.00%	0.00%	NA	NA	10.34%	10.42%	0	NS
Unidentified protozoan	3.57%	2.17%	0.13	NS	6.90%	10.42%	0.27	NS
Troglocorys cava	0.00%	0.00%	NA	NA	24.14%	16.67%	0.64	NS
Trematodes								
Dicrocoeliidae sp.	6.67%	0.00%	1.67	NS	6.90%	4.17%	0.27	NS
Cestodes								
Bertiella sp.	10.71%	13.04%	0.09	NS	0.00%	0.00%	NA	NA

Abbreviations: NS, *P* >0.05; NA, no X^2 performed due to 0% prevalence.

** Denotes high pathogenic potential and *n* denotes number of individuals.