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Host density and human activities mediate increased parasite prevalence and richness in primates threatened by habitat loss and fragmentation

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Summary

1. Habitat loss and fragmentation are the principal causes of the loss of biological diversity. In addition, parasitic diseases are an emerging threat to many animals. Nevertheless, relatively few studies have tested how habitat loss and fragmentation influence the prevalence and richness of parasites in animals.

2. Several studies of nonhuman primates have shown that measures of human activity and forest fragmentation correlate with parasitism in primates. However, these studies have not tested for the ecological mechanism(s) by which human activities or forest fragmentation influence the prevalence and richness of parasites.

3. We tested the hypothesis that increased host density due to forest fragmentation and loss mediates increases in the prevalence and richness of gastrointestinal parasites in two forest primates, the Tana River red colobus (*Procolobus rufomitratus*, Peters 1879) and mangabey (*Cercocebus galeritus galeritus*, Peters 1879). We focused on population density because epidemiological theory states that host density is a key determinant of the prevalence and richness of directly transmitted parasites in animals.

4. The Tana River red colobus and mangabey are endemic to a highly fragmented forest ecosystem in eastern Kenya where habitat changes are caused by a growing human population increasingly dependent on forest resources and on clearing forest for cultivation.

5. We found that the prevalence of parasites in the two monkeys was very high compared to primates elsewhere. Density of monkeys was positively associated with forest area and disturbance in forests. In turn, the prevalence and richness of parasites was significantly associated with monkey density, and attributes indicative of human disturbance in forests.

6. We also found significant differences in the patterns of parasitism between the colobus and the mangabey possibly attributable to differences in their behavioural ecology. Colobus are arboreal folivores while mangabeys are terrestrial habitat generalists.

Key-words: Africa, conservation, endangered species, gallery forest, structural equation modelling

Introduction

Habitat loss and fragmentation appear to be the principal causes of the loss of biological diversity (Wilcox & Murphy 1985; Simberloff 1988; Caughley 1994). In addition, parasitic diseases are emerging as a serious threat to many species of animals (McCallum & Dobson 1995; Daszak, Cunningham & Hyatt 2000; Patz *et al*. 2000). However, relatively few studies have tested how habitat loss and fragmentation influence the prevalence (proportion of infected hosts; Margolis *et al.*

1982) and richness (number of different species per host) of parasites in animals (McCallum & Dobson 2002). By all indications, habitat loss and fragmentation should increase parasite prevalence and richness in vertebrate animals.

Habitat loss per se reduces the amount of habitat available, and thus reduces the amounts of food resources and thus may increase competition for food among resident animals. In addition, immediately after habitat is lost, animals may crowd into the remaining smaller patches leading to increases in rates of agonistic behaviours. The combined effects of increased food competition and agonistic interactions among animals can increase their stress levels, compromise

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immunocompetence and thereby lower their resistance to infection (Eley *et al*. 1989; Coe 1993; Friedman & Lawrence 2002).

Habitat fragmentation per se creates a larger number of smaller habitat patches in a given landscape (Bascompte & Sole' 1996; Fahrig 2003). When a landscape is so fragmented that suitable habitat patches are embedded in a matrix of unsuitable habitat, there is usually a positive correlation between animal population density and patch area (Andren 1994; Connor, Courtney & Yoder 2000; Brotons, Monkkonen & Martin 2003; Fahrig 2003). Because fragmentation also limits foraging and travel routes, high population density should lead to intensive use of the same habitat patches which would increase the levels of contamination of the environment with infective stages of parasites such as eggs and larvae (e.g. Stoner 1996). Furthermore, high host population density influences positively the probability that parasite transmission stages such as eggs or larvae contact a host (Anderson & May 1978; Anderson 1979; Morand & Poulin 1998; Poulin 1998; Packer *et al*. 1999). Consequently, parasite species that require high rates of transmission for their population persistence may exist only in high-density populations (Anderson 1979; Arneberg 2002). Thus, habitat fragmentation should lead to increased prevalence and richness of directly transmitted parasites. Clearly, host population density should play a key role in mediating the effects of habitat loss and fragmentation on the prevalence and richness of directly transmitted parasites. In fact, among nonhuman primates in general, host population density is the key determinant of parasite species richness (Nunn *et al*. 2003).

Fragmented landscapes also contain more edge habitat for a given amount of core habitat (Fahrig 2003). The increased edge effects in combination with elevated population densities in habitat fragments, should promote cross-species transfer and acquisition of novel parasites (Murcia 1995; Fagan, Cantrell & Cosner 1999; Ries *et al*. 2004). Thus, among colobus monkeys in western Uganda forests, individuals living at the edge of the forest were found more likely to be infected with multiple parasites species than individuals in the core of the forest (Gillespie, Chapman & Greiner 2005a).

In this study, we tested the hypothesis that increased population density due to forest loss and fragmentation causes increased prevalence and richness of gastrointestinal parasites in the Tana River red colobus (*Procolobus rufomitratus*, Peters 1879) and mangabey (*Cercocebus galeritus*, Peters 1879). These primates are endemic to Tana River forests in eastern Kenya (Fig. 1). The Tana River forests are an ecosystem where habitat loss and fragmentation caused by human activities may greatly influence host density and thus promote increased prevalence and richness of parasites in resident animals. Forest loss and fragmentation there is caused by a growing human population which increasingly depends on resources extracted from the forest and clearing of forest for cultivation (Wieczkowski & Mbora 2000; Mbora & Meikle 2004a). These forests appear to be remnants of a previously continuous forest that extended from Central to East Africa during the upper-Pleistocene (25 000–30 000 years BP; deMenocal 2004; Bobe & Behrensmeyer 2004). As such, the Tana forests are of great conservation value and are part of the East African Coastal forests global biodiversity hotspot (Myers *et al*. 2000). Thus, in 1976, the government of Kenya established the Tana River Primate National Reserve (TRPNR) to protect the two species of primates and the forest ecosystem. Both primates are forest dependent and critically endangered (IUCN 2007; Mittermeier *et al*. 2007).

We tested four postulates in an effort to understand how habitat loss and fragmentation influence parasite prevalence and richness in the Tana River landscape. First, we expected a positive correlation between the area of forest patches and the density of primates as has been reported for animals in other highly fragmented landscapes (Andren 1994; Connor *et al*. 2000; Brotons *et al*. 2003). In the Tana River landscape, larger forests tend to be less degraded than smaller forests thereby creating a positive correlation between area of forest patches and the abundance of important primate food trees (Medley 1993a). Because the density of monkeys is strongly correlated with abundance of important primate food trees (Mbora & Meikle 2004a) in the Tana, it seemed plausible that forest area and density of monkeys would be correlated. Second, we expected a positive correlation between monkey density and human activities that cause forest loss, for example, basal area or density of cut stems. In areas where forest is being actively cut, primates are forced to live in smaller patches relative to the area of original forest, and such forests should have elevated densities of primates in the short term (Medley 1993a). Third, following from the first and second postulates, and in accordance with epidemiological theory that host density influences positively the prevalence and richness of directly transmitted parasites (Anderson & May 1978; Anderson 1979; Morand & Poulin 1998; Poulin 1998; Packer *et al*. 1999), we predicted a positive correlation between the density of monkeys and the prevalence and richness of parasites across the landscape.

Although basal area or density of cut stems represents a direct measure of human activities, it does not measure the full range of human activities in the Tana River forests (Medley 1993b). For example, harvesting of nontimber forest products such as thatch or the tapping of wine from the *Phoenix reclinata* palm are common activities in the forests (David N. M. Mbora, personal observations). Such activities would not lead into an increase in the primate population density because they do not decrease the area of forest patches. However, these activities contribute to increased interactions between humans and the primates and the likelihood of cross-species transfers of atypical parasites species. The levels of these human activities in the forests should correspond to the demand from the community, and the demand should correlate with the abundance of the human population within the vicinity of the forests. Therefore, we surmised that human population density and proximity of forests to cultivated areas should correlate with these unmeasured activities. Consequently, as a fourth postulate, we predicted that human population density and proximity of forests to cultivated areas
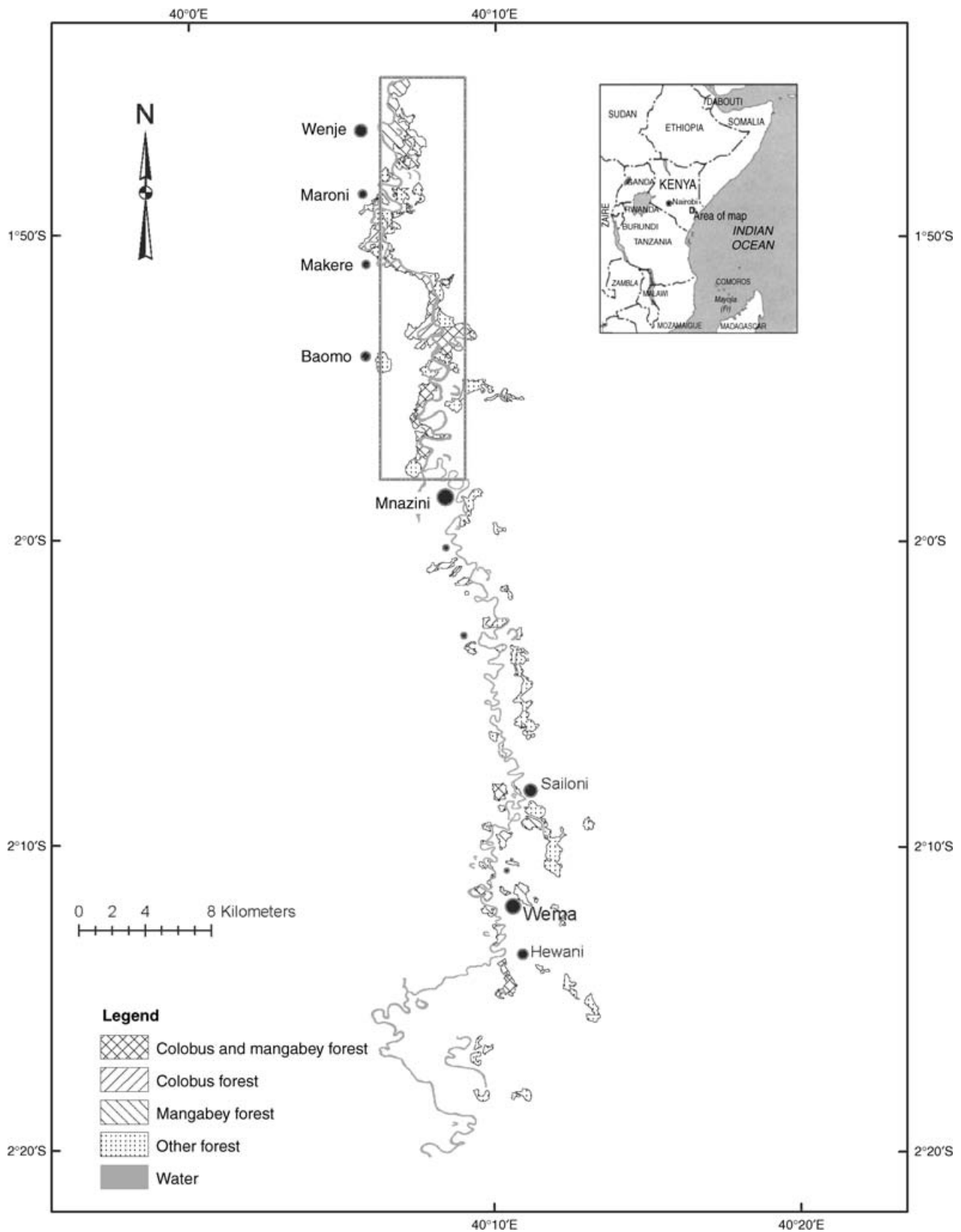
Fig. 1. Distribution of study forest patches along the Tana River, Kenya. The frame on the top half shows the approximate location of the Tana River Primate National Reserve (TRPNR), and the major villages are named.

should correlate positively with parasite prevalence and richness. Thus, we expected that unprotected forests outside TRPNR would potentially experience higher levels of human activity than forests protected inside TRPNR, and, that forests outside TRPNR would exhibit higher parasite prevalence and richness.

We focused our analyses on gastrointestinal parasites because the species commonly found in the Tana River primates are directly transmitted and are relatively easy to sample from faeces (Mbora & Munene 2006).

Materials and methods

The study area comprised 26 km^2 of gallery forest patches of various sizes (size range, < 1–220 ha) on both sides of the lower floodplain of the Tana River in eastern Kenya (Fig. 1; Mbora & Meikle 2004b). The general topography of the area is flat, with a maximum elevation of 40 m above sea level, and a mean annual rainfall of 400 mm (Hughes 1990). The forest is maintained by groundwater and by periodic flooding, and its lateral extent is limited to about 1 km on either side of the river (Hughes 1990). Thus, there is no climate variability among study forests, and the main gradient in forest community composition is decreasing density and basal area of trees with increasing distance from the main river channel (Mbora & Meikle 2004a). The intervening matrix comprises cultivation, riparian grassland, and dry shrubs.

We mapped the gallery forest using aerial photographs, and selected 20 forest patches (size range, 1·2–220 ha) as study sites. The study forests were located within 50 km of each other, and were systematically selected to achieve representative sampling east and west of the river, inside and outside TRPNR (Fig. 1; Mbora & Meikle 2004b). Each study forest was thoroughly surveyed to determine the number of resident groups of colobus and mangabey, and a subset of groups (number range, colobus, one to three; mangabey, one to four) within each forest was identified for detailed studies of group size and composition over time. Social groups for detailed studies were systematically selected so that they were easy to locate and to identify using 'marker' animals on subsequent days. Both species were sympatric in 10 forests (area range, 18·9–220 ha), colobus were found alone in six forests and mangabeys were found alone in four forests. We periodically surveyed these forests and monitored the study groups since 2001 (Mbora & Meikle 2004a; David N. M. Mbora, unpublished data). Monkey density was calculated from these surveys as the total number of social groups and as the number of individuals found in each forest divided by the area of the forest patch (see below for how forest area was determined). Although another three species of nonhuman primates [*Papio cynocephalus* (Linnaeus, 1766) *Cercopithecus mitis*, (Wolf 1822) and *Chlorocebus aethiops*, (Linnaeus, 1766)] are found in this area, they are not forest dependent as they use the outlying savannah and matrix habitats extensively. In contrast, the endemic red colobus and mangabey are forest dependent and account for the bulk of the primate biomass in the forests (David N. M. Mbora, unpublished data). Thus, only the density of red colobus and mangabey rather than the density of all nonhuman primates was considered.

Faecal samples were collected from different sets of habituated and semi-habituated free ranging study groups in July–August 2005, and 2006. We followed the social groups from 06.00 h to 11.30 h, and then from 15.00 h until nightfall. Upon observing an animal defecating, a sample of the faeces was collected and stored in a 20-mL vial containing 10% formalin as a preservative. The vial was labelled with the date, species identity and a code identifying the troop. The number of faecal samples taken from each group and forest was based on two main considerations. First, we aimed to take only a single sample from any particular animal, but to sample as many individuals from each social group as possible. Second, we aimed to obtain enough samples from each forest fragment to allow for inter-forest fragment comparisons. We assumed that each forest fragment comprised a subpopulation, and calculated the minimum number of samples needed using the formula, $n = \ln(\alpha)/\ln(1 - p)$, where n is the sample size, α the significance level (0·05) and p is the mean parasite prevalence in the population (Gillespie 2006). Based on our preliminary work in the Tana on parasite prevalence (Mbora & Munene 2006; unpublished data), we expected mean prevalence of 15–25% for each primate in forest fragments. Thus, we aimed for 10–15 faecal samples for each primate in each forest patch.

The samples were then examined for gastrointestinal parasites at the Institute of Primate Research in Nairobi, Kenya, from August– September 2005 and 2006. A modified formalin–ether sedimentation method was used to diagnose the presence of ova, cysts, and larval parasite stages in stool samples (Long, Tsin & Robinson 1985). The detailed procedure used to diagnose and identify parasites is described in Ash & Orihel (1991) and the specific modifications are outlined in Mbora & Munene (2006). In 2005, we examined 150 samples from 10 colobus groups in 9 forests, and 81 samples from 5 mangabey groups in 6 forests. In 2006, we examined 128 samples from 9 colobus groups in 7 forests, and 124 mangabey samples from 8 social groups in 8 forests. Parasite prevalence was measured as the proportion of hosts infected by a particular parasite in each social group (Margolis *et al.* 1982), and richness as the number of different parasite species detected per host.

The area of each forest patch was measured using ARCMAP GIS. The proximity of forests to human habitation was measured using ARCMAP GIS as the straight line distance from the forest edge to the nearest edge of a cultivated area. Human use of the forest vegetation was measured in belt transects established within each forest perpendicular to the river channel. Each belt was 5 m wide, and was run for a maximum length of 100 m unless the width of the forest was less than 100 m. The number of transects sampled in each forest was based on the size of the forest; we sampled three transects in all forests less than 5 ha and added one belt transect for every 10-fold increase in area (Mbora & Meikle 2004a). In each transect, we recorded the species identity, counted and measured the girth diameter (whenever practicable) for trees cut or in any way damaged by humans as a measure of human use of forests. We then summarized human use of forests as absolute basal area of cut trees (m^2 ha^{-1}) and as density of trees cut (individual trees/ha) per forest.

We obtained information on the density of the human population from the study area from the last national census of Kenya (Kenya Central Bureau of Statistics 2001). These human population data were structured around government administrative units called sublocations. The study area comprised nine sublocations made up of clusters of one to three villages. We set the value of the density of humans associated with each forest as the value of the sublocation in which the forest was located.

Initial exploratory data analyses showed that the following sets of attributes were highly correlated (Pearson product-moment correlation), basal area and density of cut trees ($r = 0·44$, $P < 0·01$), and density of monkey groups and density of individual monkeys ($r = 0·89$, $P < 0·01$). Therefore, we used basal area of cut trees as a measure of human activity in forests, and density of individual monkeys in all subsequent analyses. We used structural equation modelling (maximum likelihood) to analyse how forest area and

human activities influenced monkey density inside and outside TRPNR; and, how parasite prevalence and richness were influenced by monkey density and by being inside or outside TRPNR.

Structural equation modelling simultaneously solves a system of linked regression equations that defines an assumed causal structure among the variables of interest (Grace 2007). In our analysis, parasite prevalence and richness were assumed to be variables that directly depended on monkey density and whether the social group was inside or outside TRPNR (a dichotomous variable). Monkey density was in turn assumed to depend directly on forest area, human density and basal area of trees cut in the forest. We used LISREL version 8·5 (Jöreskog & Sörbom 2002) to solve this system of regression equations using the maximum likelihood algorithm, to estimate partial regression coefficients and correlations, and to evaluate the overall fit of the data to this assumed causal model. We calculated separate structural equation models for each species.

Results

We found 16 helminth and 5 protozoan parasites in both primates out of which 71% were directly transmitted (Table 1). All the parasites found in the colobus were also found in the mangabey, but the mangabey had four additional parasites not found in the colobus (*Anoplocephala spp.*, *Heterakis spp.*, *Streptopharagus spp.*, *Isospora spp.*). Among the helminths, *Trichuris spp.* was the most common parasite, being found in at least 60% of the forests in each of the primates. Among the protozoans, *Escherichia coli*, *E. hartmani*, and *E. histolytica*-like had the highest prevalences in both primates. Interestingly, the protozoan *Balantidium coli*-like, which was found at relatively high prevalence (32·25%) among the mangabeys, was found at relatively low prevalence (4%) among the colobus. Generally, the highest prevalence and richness of parasites in both primates was found in the same forest patches located outside TRPNR (Fig. 2a). Across the landscape, mean parasite prevalence was higher in mangabeys than in the colobus (88·4% vs. 76·6%; $\chi^2 = 6·15$, d.f. = 1, $P < 0·01$). However, the higher number of parasite species per host in the mangabey was not statistically significant ($t = 0·74$, $P = 0·50$, Fig. 2b). The prevalence and richness of parasites were strongly correlated in both primates (colobus, $r = 0·63$, $P = 0·00$, mangabey, $r = 0·83$, $P = 0·00$).

The causal structure we assumed for the structural equation models showed good overall fit to the data for each monkey species [overall tests for goodness of fit of the structural equation models (note that a nonsignificant goodness of fit test indicates that the data fit the model well) – colobus: $\chi^2 = 2·97$, d.f. = 7, $P = 0·89$; mangabey: $\chi^2 = 7·17$, d.f. = 7, $P = 0·41$]. Most of the variance in the density of monkeys was explained by forest area, basal area of cut stems and density of humans in both colobus ($R^2 = 0·67$, $F = 10$, d.f. = 2, 16, $P < 0·05$; Fig. 3a) and mangabey ($R^2 = 0·74$, $F = 12·98$, d.f. = 2, 15, $P < 0·05$; Fig. 3b). Forest area and basal area of cut stems were not significantly correlated and thus both attributes made independent contributions to the variance in host density (Fig. 3). Among colobus, a significant amount of variance in the prevalence and richness of parasites was accounted for by monkey density and being
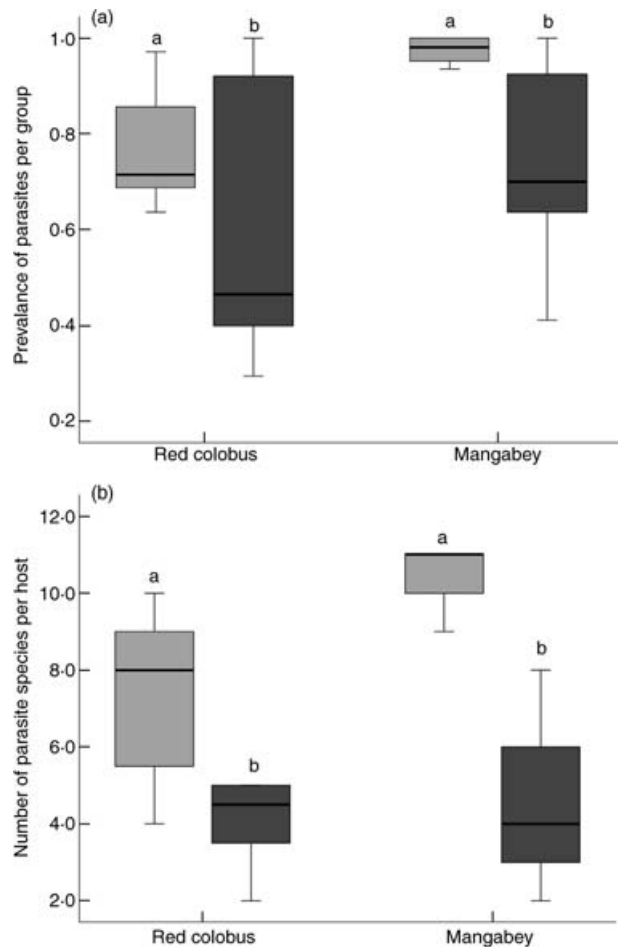

Fig. 2. (a) Boxplots‡ of the prevalence of parasites in Tana River red colobus and mangabey outside (a) and inside (b) TRPNR. (b) Boxplots‡ of the number of parasites per group in Tana River red colobus and mangabey outside (a) and inside (b) TRPNR. ‡The boxplots show the five statistics, minimum, second quartile, median, third quartile, and maximum.

outside TRPNR (Prevalence, $R^2 = 0·31$, $F = 3·61$; Richness, $R^2 = 0·44$, $F = 6·29$; d.f. = 2, 16, $P < 0·05$; Fig. 3a). Similarly for mangabeys, a significant amount of variance in the prevalence and richness of parasites was positively correlated with monkey density and being outside TRPNR (prevalence, $R^2 = 0·42$; $F = 5·39$; richness, $R^2 = 0·69$, $F = 16·43$; d.f. = 2, 15, $P < 0·05$; Fig. 3b).

Although the direction of the associations between the prevalence and richness of parasites, and the predictor variables were in the same direction for both primates (Fig. 3), some differences were readily apparent when the two species were compared. For example, density of monkeys was a stronger influence on the variance in the prevalence and richness of parasites in the colobus than in the mangabey (Fig. 3). Similarly, being outside TRPNR was a stronger influence on the variance in the prevalence and richness of parasites in the mangabey than in the colobus (Fig. 3). It should be noted that densities of colobus and mangabeys did not differ in forest fragments ($t = 0·50$, d.f. = 35, $P = 0·62$).

Table 1a. Parasites found in the Tana River red colobus

Parasites species	Mean* parasite prevalence (percentage)	Taxonomic group and life cycle comments (Roberts & Janovy 2005)
Abbreviata spp.	7·7 (5–11)	Nematode, indirect
Ascaridia spp.	8·0 (0–8)	Nematode, direct
Ascaris spp.	9·5 (4–15)	Nematode, direct, human pathogen
Balantidium coli-like†	4·0 (0–4)	Protozoa, direct, human pathogen
Bertiella spp.	45·0 (0–56)	Cestode, indirect, benign in primates
Capillaria spp.	28·0 (11–45)	Nematode, direct/indirect, human pathogen
Dicrocoelium spp.	16·8 (7–30)	Trematode, indirect
Entamoeba coli, (Grassi, 1879)	26·1 (7–68)	Protozoa, direct, benign in primates
Entamoeba hartmani, (Prowazek, 1912)	7·0 (1–15)	Protozoa, direct, benign in primates
Entamoeba histolytica-like†	25·33 (5–58)	Protozoa, direct, human pathogen
Oesophagostomum spp.	7·4 (3–13)	Nematode, direct/indirect, human pathogen
Physaloptera spp.	7·0 (0–7)	Nematode, Indirect, human pathogen
Strongyloides spp.	10·4 (0–25)	Nematode, direct/indirect, human pathogen
Toxascaris spp.	16·4 (7–38)	Nematode, direct, human pathogen
Toxocara spp.	14·0 (5–26)	Nematode, direct, human pathogen
Trichostrongylus spp.	7·6 (4–14)	Nematode, direct, human pathogen
Trichuris spp.	*8·44 (3–20)*	Nematode, direct, human pathogen

*Range of values among forests is in parenthesis. †These two species were identified as *–like* because of the uncertainty of species identification. We identified them based on the size, number and the morphology of the nuclei in the recovered cysts. However, it is possible that *Entamoeba histolytica*, could be *Entamoeba dispar* or a distinct species. Similarly, several different ciliates, for example, *Buxtonella,* resemble *Balantidium spp.*

Table 1b. List of parasite found in the Tana River mangabey

Parasites species	Mean* parasite prevalence (percentage)	Taxonomic group and life cycle comments (Roberts & Janovy 2005)
Abbreviata spp.	9·0 (0–9)	Nematode, indirect
Anoplocephala spp	1·8 (1–2)	Cestode, indirect, human pathogen
Ascaridia spp.	7·0 (0–7)	Nematode, direct
Ascaris spp.	8·0 (0–8)	Nematode, direct, human pathogen
Balantidium coli-like†	33·3 (7–83)	Protozoa, direct, human pathogen
Bertiella spp.	11·3 (7–25)	Cestode, indirect, benign in primates
Capillaria spp.	10·3 (7–20)	Nematode, direct/indirect, human pathogen
Dicrocoelium spp.	25·0 (6–43)	Trematode, indirect
Entamoeba coli, (Grassi, 1879)	52·7 (7–92)	Protozoa, direct, benign in primates
Entamoeba hartmani, (Prowazek, 1912)	16·8 (7–25)	Protozoa, direct, benign in primates
Entamoeba histolytica-like†	67·1 (55–83)	Protozoa, direct/indirect, human pathogen
Heterakis spp.	19·0 (7–30)	Nematode, direct, human pathogen
Isospora spp.	6·0 (0–6)	Protozoa, direct, human pathogen
Oesophagostomum spp.	5·7 (4–7)	Nematode, direct/indirect, human pathogen
Physaloptera spp.	13·8 (9–20)	Nematode, indirect, human pathogen
Streptopharagus spp.	12·7 (8–16)	Nematode, indirect
Strongyloides spp.	7·4 (4–10)	Nematode, direct/indirect, human pathogen
Toxascaris spp.	7·3 (6–9)	Nematode, direct, human pathogen
Toxocara spp.	24·0 (7–44)	Nematode, direct, human pathogen
Trichostrongylus spp.	10·8 (7–20)	Nematode, direct, human pathogen
Trichuris spp.	*18·8 (6–20)*	Nematode, direct, human pathogen

*Range of values among forests is in parenthesis. †These two species were identified as *–like* because of the uncertainty of species identification. We identified them based on the size, number and the morphology of the nuclei in the recovered cysts. However, it is possible that *Entamoeba histolytica*, could be *Entamoeba dispar* or a distinct species. Similarly, several different ciliates, for example, *Buxtonella*, resemble *Balantidium spp.*

Discussion

The prevalences of parasites found in the Tana River primates were very high compared to primates elsewhere. For example, in a study of the prevalence of gastrointestinal parasites in primates worldwide, Altizer, Nunn & Lindenfors (2007) found that threatened primates had a lower prevalence of parasites (mean, 15·3%) than nonthreatened species (mean, 19·1%). Both the Tana River colobus and mangabey are critically endangered (Mittermeier *et al.* 2007), yet they had mean parasite prevalences much higher than the mean for threatened species worldwide (Fig. 2a). Similarly, the Tana River red
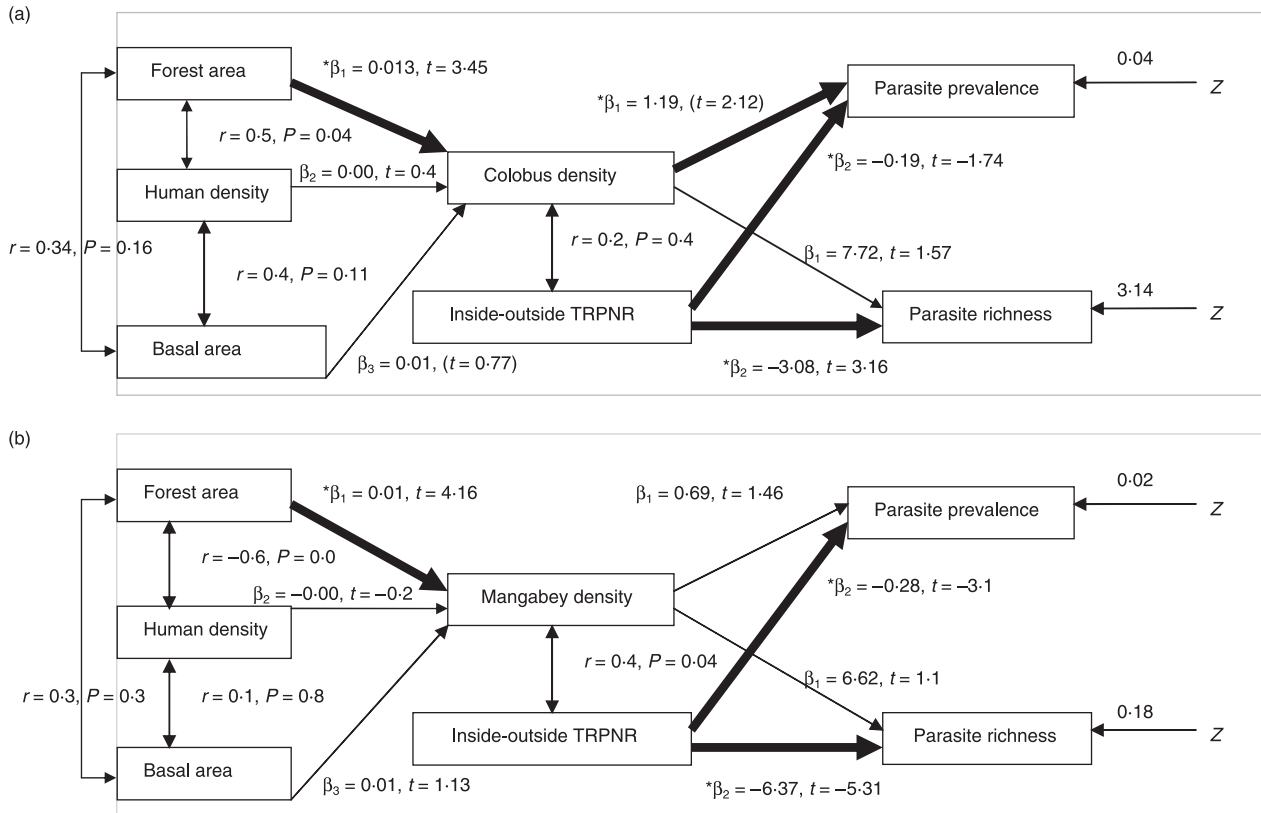
(a)

(b)

Fig. 3. Path diagram showing that parasite prevalence and richness depended directly on density of (a) colobus and (b) mangabey. In turn, monkey density depended directly on forest area, basal area of cut trees and density of humans inside and outside TPRNR. **Bold** arrows and asterisks identify model coefficients that are statistically different from 0 ($t_{16,0.95} = 1.7$). Potentially correlated independent variables are indicated with two-way arrows with associated correlation coefficients. The one-way arrows to the extreme right represent the effects of the error term Z.

colobus and mangabey had relatively high richness of parasites (Fig. 2b). Surveys of the richness of gastrointestinal parasites of seven species of primates in western Uganda found 14 species of parasites after examining 2396 faecal samples (Gillespie, Greiner & Chapman 2004, 2005b). This suggests lower parasite richness in western Uganda than in the Tana River, because we used the same sampling methods as Gillespie *et al.* (2004, 2005b), on a much smaller sample of faeces (~20%) from only two primates, but found 21 parasite species. Alternatively, it may be that the same parasites are present in Tana River and western Uganda but the higher prevalence in the Tana River made it possible to detect more species.

Our study suggests that the high parasite prevalence and richness in the Tana River is due to the extensive forest fragmentation and loss mediated primarily by differences in host density and human activities inside and outside TRPNR. First, basal area of cut stems, which was positively associated with monkey density, is a direct measure of human activities that cause habitat loss and fragmentation. Second, in accordance with our expectations, forest area was positively associated with monkey density, probably because of the positive correlation between habitat quality and forest area (Medley 1993a; Mbora & Meikle 2004a).

In accordance with epidemiological theory, increasing host density should increase the probability that a given infective

parasite egg or larva will contact a host (Anderson & May 1978; May & Anderson 1978). Therefore, the prevalence of directly transmitted parasites should correlate positively with the density of hosts in the population (Arneberg *et al.* 1998); – a prediction that was clearly supported in this study. Epidemiological theory also predicts that parasite species richness should correlate positively with host population density (Anderson & May 1978; May & Anderson 1978; Arneberg 2002; Nunn *et al.* 2003). Thus, the finding that monkey density was positively correlated with parasite richness further supports the hypothesis that high host density mediates increased parasite prevalence and richness due to habitat fragmentation and loss among these primates.

The differences observed between the two primates are quite interesting and are probably due to their divergent behavioural ecology. Colobus are forest habitat specialists whose abundance should be more closely linked to attributes of the forest habitat (Mbora & Meikle 2004a). Thus, density of monkeys was a stronger influence on the variance in the prevalence and richness of parasites in the colobus than in the mangabey. In addition, in 10 of the 20 forests where colobus and mangabeys were sympatric, mangabeys tended to have a higher prevalence of parasites than colobus did. Mangabeys live in relatively larger social groups that range much more widely than colobus do (Homewood 1978; Marsh 1981). Therefore, the

probability that a mangabey will be exposed to infective parasite stages should be much higher than for colobus, favouring increased parasite prevalence and richness in mangabeys.

It was also quite interesting that we found several parasite species that are typically human pathogens. However, it would be unwise to conclude that cross-species infections were occurring between humans and primates in this area without further genetic and morphological analyses of these parasites. For example, (De Gruijter *et al.* 2005) found that the population genetics of *Oesophagostomum bifurcum* that infect sympatric humans and monkeys in Ghana were strongly structured according to host species. Thus, even though there was ample opportunity for *O. bifurcum* to exhibit cross-species infections, it seems that this was not occurring in this location in Ghana.

Our study stands to make important contributions to basic ecological theory and provides important information to guide the conservation of nonhuman primates. First, we showed that the increased parasite prevalence and richness in primates in a highly fragmented landscape is mediated primarily by changes in host density. Previous studies that have shown a correlation between human activities and parasite prevalence have not identified the mechanism(s) by which human activities lead to increased prevalence and richness of parasites (e.g. Gillespie & Chapman 2006). Second, our study suggests that habitat fragmentation and loss may have different effects on parasite prevalence and richness in arboreal compared to terrestrial nonhuman primates. Thus, even though the two species were largely sympatric, the mangabey, a terrestrial habitat generalist, had a higher prevalence and richness of parasites than the colobus, an arboreal folivore.

It is estimated that at least 90% of all primate species are endangered by forest fragmentation and loss (IUCN 2007; Mittermeier *et al.* 2007). Therefore, it is important to know that such forest primates may face an additional threat from increases in the prevalence and richness of directly transmitted parasites. In the specific case of the two Tana River primates, both of which are critically endangered, it should be of concern that they harbour such high levels of parasite prevalence and richness. Clearly, the high prevalence and richness of parasites does not necessarily mean that these animals are sick. However, severe infections by helminths and protozoa can reduce host population abundance by lowering the efficiency of normal host activities (Hudson, Dobson & Newborn 1992; Gregory & Hudson 2000). Furthermore, given the close taxonomic relatedness of humans and nonhuman primates, it is reasonable to assume that the various parasite species identified in the Tana River primates as pathogenic in humans (Table 1; Polderman *et al.* 1991; Roberts & Janovy 2005), are also potentially pathogenic in primates. Thus, we recommend further studies to test the possible health effects of the high levels of parasite prevalence and richness found in these primates. Such studies will help to clarify if parasites play any role in causing the declining population abundance and the endangered conservation status of these and other forest primates.

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