The influence of human settlements on gastrointestinal helminths of wild monkey populations in their natural habitat

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DOKTORS DER NATURWISSENSCHAFTEN
(Dr. rer. nat.)
Fakultät für Chemie und Biowissenschaften
Karlsruher Institut für Technologie (KIT) – Universitätsbereich
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DISSERTATION
von
Dipl. Biol. Alexandra Mücke
geboren in Germersheim

Dekan: Prof. Dr. Martin Bastmeyer
Referent: Prof. Dr. Horst F. Taraschewski
1. Korreferent: Prof. Dr. Eckhard W. Heymann
2. Korreferent: Prof. Dr. Doris Wedlich
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To Maya

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Abstract

Human-wildlife interactions have reached unprecedented levels in the present days and humans are changing the earth's ecosystems more rapidly and extensively than ever before. This development gives cause for serious concern, especially since disease interactions between wildlife and humans have been recognized as major conservation threats. Primates are our closest relatives, but almost half of the known species are threatened of going extinct in the future. The transmission of human pathogens to susceptible, endangered wild primates has already led to major population crashes.

The aim of my work was to determine the influence of humans on the presence of gastrointestinal parasites in wild primates. I approached this goal by studying two tamarin species of Peru (*Saguinus fuscicollis* and *Saguinus mystax*) and a macaque species in Thailand (*Macaca fascicularis*) as model animals. I screened fecal samples from primate groups differing in their intensity of contact to humans and differing in the proximity to humans and their facilities. In addition, I analyzed stool samples of the villagers living in the vicinity of the primates with contact to humans.

My parasitological investigations showed that the human population had high prevalences of different helminth species including *Ascaris lumbricoides*, *Trichuris trichiura*, *Strongyloides stercoralis*, hookworms and, in Thailand, *Opisthorchis viverinii*. The primate populations were also parasitized by several species of helminth eggs. However, parasite burden of humans and primates were different, and I could not detect evidence for anthropozoonotic parasite transmission. But my results show significant differences between the parasite communities infecting human contact groups and sylvatic groups. In Peru, the highly pathogenic acanthocephalan *Prosthenorchis elegans* and in Thailand *Strongyloides fuelleborni* and a foodborne trematode were significantly more present in human associated groups.

Evaluation of my data suggests that parasite transfer between humans and primates is limited. However, my results are indicating that human induced changes to the monkey's habitat have a significant negative impact on the parasite burdens and parasite community structure of wild monkeys. Human alteration of the habitat and the primate behavior is likely to play a major role in determining the occurrence, prevalence and intensity of helminth infestation of wild nonhuman primates.

Zusammenfassung

Menschen und Wildtiere interagieren heutzutage auf einem noch nie dagewesenen Niveau. Der Mensch verändert dabei die Ökosysteme der Welt intensiver und schneller als jemals zuvor. Diese Entwicklung gibt Anlass zu ernster Besorgnis, insbesondere da die Übertragung von Krankheiten zwischen Menschen und Wildtieren als große Bedrohung der Arterhaltung erkannt wurde. Ein gutes Beispiel dafür sind die Primaten. Diese sind unsere nächsten Verwandten, doch beinahe die Hälfte aller bekannten Arten könnte in naher Zukunft aussterben. Die Übertragung menschlicher Krankheitserreger auf anfällige oder gefährdete Tiere hat bereits zu starken Populationsseinbrüchen bei wildlebenden Primaten geführt.

Das Ziel meiner Arbeit war es, den Einfluss des Menschen auf das Vorhandensein gastrointestinaler Parasiten in wild lebenden Primaten zu bestimmen. Um dies herauszufinden, wählte ich zwei Tamarinarten in Peru (*Saguinus fuscicollis* und *Saguinus mystax*) und eine Makakenart in Thailand (*Macaca fascicularis*) als Modellorganismen. Ich analysierte Kotproben von verschiedenen Gruppen dieser Arten, welche sich durch die Intensität des Kontaktes zu Menschen und deren Einrichtungen unterschieden. Zusätzlich untersuchte ich Stuhlproben der Menschen, welche Kontakt zu den untersuchten Primatengruppen hatten.

Meine parasitologischen Untersuchungen zeigten, dass die menschliche Bevölkerung mit einer Reihe von Helminthen infiziert war, darunter befanden sich Spulwürmer, Zwergfadenwürmer, Hakenwürmer oder auch in Thailand der cacinogene Leberegel *Opisthorchis viverinii*. Die untersuchten Primaten wiesen ebenfalls einen starken Helminthenbefall auf. Allerdings konnte ich keine Übereinstimmungen zwischen den Parasitengemeinschaften von Menschen und Affen feststellen. Ich konnte keine Hinweise auf eine Parasitenübertragung von Mensch auf Affe finden.

Jedoch konnte ich signifikante Unterschiede zwischen den Parasitengemeinschaften von Primaten mit Kontakt zu Menschen und Primaten ohne menschlichen Kontakt feststellen. In Peru war der hochpathogene Acanthocephale *Prosthenorchis elegans*, in Thailand waren *Strongyloides fuelleborni* und ein durch Nahrung übertragener Trematode in höheren Prävalenzen in den menschlichen Kontaktgruppen zu finden.

Die Auswertung meiner Daten deutet darauf hin, dass die Übertragung von Parasiten zwischen Menschen und Primaten begrenzt ist. Allerdings zeigt meine Arbeit, dass der

Mensch durch Veränderungen am Lebensraum und dadurch am Verhalten der Primaten selbst Einfluss auf Vorkommen, Häufigkeit und Intensität von Helminthenbefall bei wilden Primaten ausübt.

Chapter 1

Introduction

1.1 Why study primate parasites?

The world's monkeys, apes and other primates are mankind's closest relatives, but they are disappearing from the face of the earth. Presently the International Union for Conservation of Nature (IUCN) recognizes a total of 634 primate species (IUCN 2010). Almost 50% are in danger of going extinct according to the criteria of the IUCN Red List of threatened species. In Asia, more than 70% of primates are classified on the IUCN Red List as vulnerable, endangered, or critically endangered - meaning that they could disappear forever in the near future. Since the 1970s, the academic community has recognized that many primate populations are severely threatened by human activities (Chapman and Peres 2001). The main threats are habitat destruction, particularly from the burning and clearing of the tropical rainforests for settlement and agriculture, the hunting of primates for food (bushmeat), traditional medicine, and the illegal wildlife trade (IUCN 2010). In addition, disease is becoming more and more recognized as a serious threat to endangered species due to significant outbreaks in a wide variety of endangered species (Werikhe *et al*. 1998; Daszak *et al*. 2000; Chapman and Peres 2001; Deem *et al*. 2001; Lafferty 2003; Smith *et al*. 2006; Köndgen *et al*. 2008).

The term "disease" includes both infectious and non-infectious cause of morbidity or mortality. Infectious diseases are caused by pathogenic agents such as viruses, bacteria, protozoa and helminths. They are listed among the top five causes of global species extinctions (Smith *et al*. 2006), and respect no species or geographic boundaries. Often the threat is increased by diseases that can be transmitted between closely related species such as cattle and buffalo (e.g. bovine tubercolsis (DeVos *et al*. 2001), rinderpest (Kock *et al*. 1999), foot and mouth disease (Sutmoller *et al*. 2000)) or people and nonhuman primates (e.g. scabies (Kalema-Zikusoka 2002), simian immunodeficiency virus (SIV)/human immunodeficiency virus (HIV) (Gao *et al*. 1999; Hahn *et al*. 2000). In parallel with studies of infectious disease in other mammals (Young 1994; Woodroffe 1999; Funk *et al*. 2001), major negative impacts on wild great ape populations, causing devastating mortality, including recent deaths arising from Ebola haemorrhagic fever and anthrax infection have been noticed. Such diseases represent an additional threat to wild ape populations (Wolfe *et al*. 1998; Boesch and Boesch-Achermann 2000; Walsh *et al.* 2003; Leendertz *et al*. 2004, 2006).

The anthropoid primates, which include humans and to a lesser degree simian primates, share broadly similar physiological and genetic characteristics. Thus, they share susceptibility to many viruses, bacteria, fungi, protozoa, helminths, and ectoparasites that have the potential to cross primate species boundaries (Ruch 1959; Brack 1987; Ott-Joslin 1993; Wolfe *et al*. 1998; Chapman *et al*. 2005a). The potential for disease transmission between humans and apes has long been recognized and addressed in captive settings. Similarities in pathogen susceptibility have made nonhuman primates ideal laboratory models.

Although humans have always shared habitats with nonhuman primates, the dynamics of human-primate interactions are changing radically (Chapman and Peres 2001, Wolfe *et al*. 2004). Primates are now commonly forced to live in an anthropogenically disturbed landscape comprising farmland, human settlements, forest fragments and isolated protected areas (Chapman and Peres 2001). This situation facilitates potential cross-transmission of disease. Indeed, recent work has shown that wild nonhuman primate populations harbor a variety of potentiallyzoonotic pathogens similar to those causing significant disease in humans (Leendertz 2004, 2006; Chi *et al*. 2007; Perpens *et al*. 2007). Such pathogens have already led to major epidemic problems for human health, such as the transfer of SI-viruses to the human population resulting in HIV-AIDS (Gao *et al*. 1999; Hahn *et al*. 2000). Köndgen and colleagues (2008) recently showed that pandemic human viruses caused the decline of endangered great ape populations.

Parasites play an important role in the dynamics of wildlife populations (Scott 1988; McCallum and Dobson 1995). They are an important part of the biological diversity of tropical rainforests and investigation of them can enhance our understanding of ecological and evolutionary processes and interactions. Parasites are significant sources of mortality in wild animal populations (Hudson *et al.* 2002; Moore and Wilson 2002). It is therefore of immense importance to learn more about the patterns of parasitism in wild hosts. For a parasite, closely related hosts offer new environments in which infection, maintenance, replication and transmission remain possible. A cross-transmission of parasites could have fatal consequences, especially when parasites are transmitted to new hosts that are not immunologically equipped to deal with them (Viggers *et al*. 1993).

In order to protect the health of both human and nonhuman primates in wild settings a basic understanding of some important diseases is necessary. The study of parasites in wild

primates provides us with knowledge for evaluating the health and the infection risk in populations, and it may also enhance the success of management programs.

1.2 Objectives of the study and thesis outline

The primary object of this study was to determine the influence of human contact and potential habitat modification on the presence of gastrointestinal parasites in wild nonhuman primates by using two species of New World monkeys, the saddleback tamarin (*Saguinus fuscicollis*) and the mustached tamarin (*Saguinus mystax*), and an Old World monkey species, the long-tailed macaque (*Macaca fascicularis*) as models. For convenience nonhuman primates will simply be called primates in this thesis.

Parasites represent an important component of natural communities. Understanding the factors that underlie patterns of parasite diversity is vital for primate conservation. In order to contribute to the knowledge of host-parasite interactions and especially to contribute to the knowledge of anthropozoonotic disease transmissions, this study has two major goals. The first is to collect baseline data on the intestinal parasite spectrum of three wild primate species. While most information available on the parasite spectrum in general is collected from laboratory and captured animals, data on intestinal parasites of wild primates are still limited. There have been over 400 parasite species reported from 119 primate species (Nunn and Altizer 2005). This is in contrast to over 900 parasites that have been reported in only seven livestock species (Cleaveland *et al*. 2001), and over 1400 parasites which are known to infect humans (Taylor *et al*. 2001). While it is possible that humans and livestock harbor greater parasite diversity, it is probable that we have only begun to identify the great variety that may exist in wild animals.

Second, this study explores patterns of variation in intestinal parasite infection between groups of the same species that differ in their interactions with humans. Research that investigates links between human activities and parasitic diseases in primates is just beginning and has focused mostly on African species (Sleeman *et al*. 2000; Adams *et al*. 2001; Graczyk *et al*. 2001; Lilly *et al*. 2002; Nizeyi *et al*. 2002; Gillespie and Chapman 2006). Although it is likely that human activity plays a role in primate-parasite interactions, relatively few studies have examined differences in parasite infection between populations of the same species that

differ in their interactions with humans (Eley *et al*. 1989; McGrew *et al*. 1989; Appleton and Henzi 1993; Müller-Graf 1994; Müller-Graf *et al*. 1997; Hahn *et al*. 2003; Gillespie *et al*. 2005a, b). Understanding the causes and consequences of such variation is likely to have important implications for primate conservation.

Primate groups in the same habitat with different degrees of interaction with humans were studied at the same time in order to control for confounding factors like predation pressure, resource availability, climatic conditions and other unknown geographical factors.

Several non-mutually exclusive hypotheses or predictions can be derived for the human influence on primates' gastrointestinal parasite burden:

A: Contact with humans influences the intestinal parasite spectrum of nonhuman primates.

Prediction A1: Primates are infected with intestinal parasites obtained from humans.

Prediction A2: Primate groups having contact with humans or living next to human settlements show infection with other intestinal parasites than primate groups living without human contact.

Prediction A3: Primates having contact with humans are threatened by new intestinal parasites

B: Parasite species richness (PSR), prevalence and egg/larvae output of nonhuman primates is dependent on the degree of interaction with humans.

Prediction B1: PSR is higher in primate groups having contact with humans

Prediction B2: Human altered habitat offers more diverse and conducive conditions for parasite encounter, and therefore to higher prevalences and egg/larvae output.

This study focuses helminth parasites, mainly those dwelling in the intestinal tract. Due to the use of coprological examinations (see chapter 3), the investigated parasites also include helminth parasites inhabiting other sites than the intestine which shed their propagules with the feces. These include species that inhabit the stomach, upper parts of the alimentary tract, pancreas, liver, mesenteric vessels, lungs and other tissues. For convenience the helminths investigated in this study will simply be called intestinal parasites.

After a review on the background of the study and the current state of research in chapter 2, chapter 3 describes and discusses the methods used for the research. Chapter 4 presents the first part of the study which took place in Peru where the influence of a rural human settlement on two sympatric tamarin species was investigated. Chapter 5 will deal with the second fieldsite in Thailand where the human influence in two forest parks on macaques was determined. The thesis concludes in chapter 6 with an overall discussion of the results from both studies and directions for further research.

Chapter 2

Literature Review

2.1 Parasites and host ecology

Parasites are an integral part of the life on earth, with parasite biodiversity exceeding the diversity of free-living hosts (Price 1980; Windsor 1998; Zimmer 2000). They are therefore ubiquitous in the lives of animals and humans. The manner by which parasites can influence host population dynamics was clearly demonstrated over 30 years ago in two theoretical papers published by Roy Anderson and Robert May (Anderson and May 1978; May and Anderson 1978). Parasites play a central role in ecosystems, affecting the ecology and evolution of species interactions (Esch and Fernandez 1993), host population growth and regulation (Hudson *et al*. 1998; Hochachka and Dhondt 2000), and community biodiversity (Hudson *et al*. 2002). Having evolved with and adapted to their surrounding environment, parasite induced infectious disease is natural in wild animal populations.

But how much do we know about the occurrence and prevalence of primate parasites? In general, such data were rarely collected and published unless there was a direct implication for human health, livestock production or other economically important activities (Cleaveland *et al*. 2002). Nowadays, however, with the growing recognition of the significance of disease for primates, the results of an increasing number of studies are becoming available (e.g. Muriuki *et al*. 1998; Nizeyi *et al*. 1999, 2002; Chapman and Peres 2001; Kalema-Zikusoka *et al*. 2002; Lilly *et al*. 2002; Chapman *et al*. 2005a, b, 2006).

Primate hosts are, like virtually every organism, inhabited by an incredible diversity of parasites, including sexually transmitted viruses, bacteria and protozoa, insect-borne protozoa that cause malaria and helminths responsible for schistosomiasis and tapeworm infections. However, at least in vertebrates, most of these do little or no harm most of the time. In general, there is an established ecological or evolutionary balance such that parasites are usually able to survive and to reproduce effectively without killing the host, thus maintaining their ability to continue reproducing in the future (Jones 1982; Kuntz 1982; Dobson and May 1986; Lyles and Dobson 1993). Some diseases in the wild are therefore symptomless or represent sub-clinical infections without any obvious ecological impact. In primates for instance, the simian immunodeficiency viruses (SIVs) and herpes B (simian herpes virus) are relatively benign in their natural hosts, and thus have virtually undetectable effects on primate fitness (cf. Nunn and Altizer 2006). Nonetheless, parasites can cause severe illness or even

death (e.g. *Plasmodium* spp./Malaria, *Entamoeba histolytica* (WHO 1997; WHO 1998a). Under what circumstances do they become pathogens?

It has been hypothesized that disease emergence most frequently results from a change in the ecology of the host or parasite, or both (Schrag and Wiener 1995). Anthropogenic change may alter vector dynamics, transmission rates, parasite host range and parasite virulence (Daszak *et al*. 2001; Gillespie *et al*. 2005a). Therefore, human habitat disturbance appears to disrupt the delicate balance in ecological systems which can occasionally lead to epizootic outbreaks that devastate natural host populations (Wobeser 1994).

Parasitic infection in primates can reduce fitness at both the individual and population levels. It can lead to malnutrition, impaired movement, feeding, predator escape and competition for resources and mates, or to increasing energy expenditure (Dobson and Hudson 1992; Hudson *et al*. 1992; Coop and Holmes 1996; Packer *et al*. 2003). More severe forms can cause blood loss, tissue damage, spontaneous abortion, congenital malformations, and death (Chandra and Newberne 1977; Despommiere *et al*. 1995). Parasite induced morbidity is also likely to have an effect on immunology, genetic diversity, behavior, reproductive success, fecundity, ecology, animal community structure, species diversity, and demography (Spalding and Forrester 1993). Additionally, the effects of parasitism can often be amplified when parasites are transmitted to populations that are not immunologically equipped to deal with them (Viggers *et al*. 1993). While naturally existing parasitism plays an important role in population maintenance and natural selection, introduced diseases can alter natural dynamics and become problematic for species survival (e.g. Thorne and Williams 1988; Goltsman *et al*. 1996; Bermejo *et al*. 2006).

2.1.1 Terminology

The word parasite is used in different ways. The ecological definition, which is used for this study, implies that a parasite is any organism that lives on and draws nutrients from another living organism (the host), usually to the host's detriment. Parasites therefore comprise a wide range of organisms such as viruses, and pathogenic bacteria, fungi, protozoa, helminths and arthropods. They diverge enormously in their mode of replication and transmission, generation times, elicited immune responses and diseases (Hudson *et al*. 2002). An important

distinction made by Anderson and May (1991) is that parasitic organisms can be categorized either as microparasites or macroparasites. Microparasites are often referred to as pathogens or disease-causing microbes and include viruses, bacteria, protozoa, and fungi, whereas macroparasites typically include worms (helminths) and arthropods. Disease refers to the pathology caused by infection, including physical signs and behavioral changes. Parasites are the disease-causing agents.

Parasite diversity cannot be investigated without considering parasite ecology in terms of life cycle, transmission mode, host-specificity and host and parasite habitat characteristics. In order to understand host-parasite interactions, knowledge of the parasites' life cycle is of fundamental importance.

Direct life cycle (homoxenous) parasites are those species for which transmission occurs within individuals of one host, where the adult parasite reproduces sexually and releases propagules (Bush *et al*. 2001; Eckert *et al*. 2005). However, some of the directly transmitted parasites can spend an obligatory period outside of the host, e.g. in the soil to undergo development into infective stages. Such parasites are, for instance, the so called soil-transmitted parasites *Strongyloides* spp., *Trichuris* spp., *Ascaris* spp. and hookworms (Ash and Orihel 1987; Bethony *et al*. 2006).

An indirect life cycle (heteroxenous) parasite requires at least one other host-species as so called "intermediate host" in which asexual reproduction takes place (Bush *et al*. 2001; Eckert *et al*. 2005).

The definitive host (primary host) is usually defined as the host in which sexual reproduction occurs and where the adult parasites live (Nunn and Altizer 2006). The intermediate host (secondary host) is usually defined as the host in which the parasite passes its larval or nonsexual existences, as host that harbors the parasite only for a short transition period, during which (usually) some developmental stage is comleted (Bush et al. 2001). A reservoir host comprises one or more epidemiologically connected populations in which the parasite can persist and from which infection is transmitted to the definitive target population (Haydon *et al*. 2002; Hudson *et al*. 2002).

2.1.2 Parasites and their role in population regulation

Parasites are considered to be an important ecological and evolutionary force (Gregory and Keymer 1989; Hamilton 1990; Hamilton *et al*. 1990; Minchella and Scott 1991; Dobson and Hudson 1992) and are recognized as a major component of ecosystems (Hudson *et al*. 2002). Due to the fact that a direct impact, for example in terms of occurrence of sick and moribund animals in natural populations, can seldom be observed, the influence of parasitic diseases on hosts was underestimated for a long time (Keymer and Read 1991). However, theoretical models suggest that parasites can have regulatory effects on population dynamics (Anderson and May 1978; May and Anderson 1978).

They can exert a significant impact on host population regulation by reducing fecundity and/or survival of the individuals (Scott and Dobson 1989; Hudson *et al*. 2002) and thereby affecting the population dynamics and community structure of host species (Freeland 1983; Minchella and Scott 1991). Parasites may alter host behavior in a way that infected individuals may choose their mates according to their apparent health (Hamilton and Zuk 1982), or they may adapt their behavior to minimize the impact of the disease (Hart 1990, 1992; Keymer and Read 1991; Møller *et al*. 1993). Parasites that preferentially infect a competitively dominant species can lead to increased species diversity within ecosystems by reducing the host's competitive advantage (Ayling 1981). When nutrients are scarce, parasitic infections can relax host competition by reducing their abundance (Washburne *et al*. 1991).

However, parasites can also influence their host populations by inducing mortality, especially under the influence of co-stressors, and therefore lead to rapid declines of populations or even species extinction (Daszak *et al*. 2000, Harvell *et al*. 2002). Parasite induced morbidity and mortality are difficult to demonstrate, especially in the field. Host fitness can be affected in many ways by parasites. Pathology due to parasite infection can directly reduce host survival (Goater and Ward 1992) or its reproductive potential (Hudson *et al*. 1998; Telfer *et al*. 2005). The regulation of host populations by lowering social status, mating success and reproduction rate of the infected host individuals is also known (cf. Taraschewski 2005).

Field studies by Hudson *et al*. (1998) show that the red grouse (*Lagopus lagopus scoticus*) undergoes population crashes every fourth year. These are caused by the intestinal nematode *Trichostrongylus tenuis*. This is one of the best studied host-macroparasite systems, showing the ability of parasites to regulate host populations. The study of *T. tenuis* infection in red

grouse in northern England (cf. Hudson *et al*. 1998) provides empirical support for the model based hypothesis of Anderson and May (1978). In this system, the caecal worms can drive host population cycles as a result of their low level aggregation and the time delay in their impact on the fecundity of grouse (Dobson and Hudson 1992). Pathogens can also influence reproduction in more subtle ways. Infected males may have difficulties maintaining territories and attracting females due to infection (Schall and Dearing 1987). Parasites can reduce the territorial behavior in red grouse (Fox and Hudson 2001). Treated males with reduced levels of parasitism won significantly more territorial contests than untreated individuals and showed more aggressive behavior in response to playback recordings of novel conspecific territorial intruders (Fox and Huson 2001).

Another good example for parasite impact on fecundity involves the Svalbard reindeer (*Rangifer tarandus platyrhynchus*). In the high Arctic habitat on Svalbard, there are no mammalian herbivores competing for food or mammalian predators. In addition, the parasite community is very simple, being dominated by only two species of strongyle nematodes in the abdomen, *Ostertagia gruehneri* and *Marshallagia marshalli* (Albon *et al*. 2002). These nematodes have a direct life cycle with no alternative hosts available in this area. Only *O. gruehneri* appears to be pathogenic since high intensities of this are associated with reduced reindeer pregnancy rates (Stien *et al*. 2002). Albon *et al*. (2002) found evidence for the first time in a mammalian herbivore consistent with the theory that a macroparasite can regulate a host population in the natural environment. Antihelmintic treatment showed that the parasitic nematode *O. gruehneri* decreased fecundity but not the survival of reindeer. This parasite mediated reduction in calf production was density dependent, increasing with the annual mean estimate of *O. gruehneri* abundance in the host population. In turn, the abundance of *O. gruehneri* was density dependent with a delayed positive response to changes in host declines.

A reduction in fecundity in parasitized hosts was reported in further studies on wildlife. *Chlamydia pecorum* infection, which causes reproductive-tract infections in koalas (*Phascolarctus cinereus*) may limit the reproductive potential within populations (Philips 2000). Reduced fertility rates due to the disease were thus implicated as one of the causes of local population declines. Another example for the decrease in reproduction effort can be observed in the bank vole (*Myodes glareolus*) and the wood mouse (*Apodemus sylvatius*) which were infected with cowpox virus. The infection was strongly associated with an increased age at maturity, reduced survival in winter but increased survival in summer as a

result of the suppression of costly reproductive activity (Telfer *et al*. 2002, 2005). Females infected with cowpox virus probably delayed maturation and reproduction until the following breeding season to maximize the probability of surviving infection (Telfer *et al*. 2005).

Some endemic parasites which are not pathogenic or do not influence host reproduction may become important when animals are stressed, malnourished or made more susceptible by infection with other pathogens (Telfer *et al*. 2002). An excellent example is provided by Gulland (1992). Every three to four years, the population of Soay sheep on the island of St. Kilda over-exploits its food supply and crashes. Sheep are emaciated and show signs of malnutrition and immunosuppression. In addition they have high nematode burdens. Sheep treated with antihelmintic in the field had lower mortality rates, while experimentally infected sheep, kept in the laboratory on a high level of nutrition, showed no signs of disease despite being infected with numbers of nematodes equivalent to those of sheep dying in the field. Gulland concluded that malnutrition suppressed the immune system allowing the nematodes to become pathogenic.

Reproduction seems to be disturbed due to an increase of stress hormones combined with a reduced production of sexual hormones (Dunlap and Schall 1995; Morales *et al*. 1996). For example, a field study found that fence lizards (*Sceloporus occidentalis*) infected with the malarial parasite *Plasmodium mexicanum* had higher levels of corticosterone and lower levels of testosterone as a response to stress than uninfected conspecifics (Dunlap and Schall 1995).

Parasites can also kill their hosts indirectly by increasing their susceptibility to predation (Holmes and Bethel 1972) or by reducing their competitive fitness (Park 1948). The North-American moose *Alces alces* can only exist in places where the white tailed deer *Odocoileus virginianus* does not exit. If a moose habitat has been colonized by the white-tailed deer, the moose population crashes due to morbidity mediated by *Parelaphostrongylus tenuis.* This nematode is spread by *O. virginianus*, but within this host it is less pathogenic than in the moose (Schmitz and Nudds 1994). In addition, some parasites are known to enhance their host susceptibility to pollution (Sakanari *et al*. 1984; Brown and Pascoe 1989).

Parasitosis can lead to blood loss, necrosis, spontaneous abortion, genital deformation and the death of the individual (Chandra and Newberne 1977; Despommiere *et al*. 1995). Since parasites play such an important role, it is crucial to investigate factors that shape the probability of acquiring parasites and the risk of developing pathology caused by these parasites, the so called disease-risk (Nunn and Altizer 2006). Parasite diversity in hosts is

assumed to be shaped by many different factors. The disease risk can be modulated at any stage of the potential infection: parasite encounter, transmission, parasite recruitment, colonization, parasite reproduction and establishment. It is difficult to measure in wild populations, thus indirect surrogates are needed such as parasite species richness (PSR), which describes the number of parasite species encountered per host (Morand and Harvey 2000; Nunn *et al*. 2003). Parasite intensity is the number of individuals of a particular parasite species (i.e. parasite load) within a single infected host. Parasite intensity only deals with those hosts infected and does not include a measure of uninfected hosts (Margolis *et al*. 1982; Bush *et al*. 1997). Parasite abundance is a measure of the mean parasite load of a single species of the entire host population, including uninfected individuals (Nunn and Altizer 2006). Parasite prevalence is the number of hosts infected with one or more individuals of a particular parasite species as a proportion of all hosts examined (Margolis *et al*. 1982; Bush *et al*. 1997). In summary these metrics allow us to estimate indirectly the disease risk in host populations.

2.2 Disease and wildlife conservation

As early as 1933 Aldo Leopold stated that "the role of disease in wildlife conservation has probably been radically underestimated" (Leopold 1933). Animal populations are predominantly regulated by three factors: availability of quality food, predation and infectious disease (Minchella and Scott 1991; Dobson 1995). Historically, wildlife diseases only attracted public attention when domestic animals (e.g. bovine tuberculosis) or human health (SIV/HIV, Hepatitis B-virus) were involved, many cases of which have been documented (Friedman 1971; Weigler 1992; Kennedy *et al*. 1993; Heneine *et al*. 1998; Sandstrom *et al*. 2000; DeVos *et al*. 2001).

Now, however, disease is becoming increasingly recognized as a threat to wildlife conservation. Conservation ecologists have recently shifted their attention to the role of parasites in population dynamics, because of their ability to threaten already reduced populations, to trigger catastrophic declines in otherwise robust host populations and because human activities can drive both of these processes (Dobson and Foufopoulos 2001; Lafferty and Gerber 2002). Pathogens can threaten their hosts in two major ways: first, they may be a direct cause threatening population extinction. One of the best known examples is the black-

footed ferret (*Mustela nigripes*; Thorne and Williams 1988) which is arguably the most endangered mammal in North America. In the mid 1980s outbreaks of canine distemper and sylvatic plaque effectively eliminated black-footed ferrets from the wild (Dobson and Lyles 2000). The giant panda (*Ailuropoda melanoleuca*) an endangered species endemic to China, is threatened by a parasitic infection (Zhang *et al*. 2008). Visceral larvae migrans (VLM) is the most significant cause of death. In pandas that died of VLM, nematodes identified as *Baylisacaris schroederi* were recovered from the liver, lungs, heart and brain. Individuals often exhibit heavy intestinal worm burdens leading to intestinal inflammation and metabolic disorders. The increase in the fraction of parasite-related deaths in periods where other threats have been reduced suggests that this parasite represents a significant threat to panda conservation (Zhang *et al*. 2008). Alternatively, pathogens may suppress the size or resilience of their host populations, increasing the probability of extinction due to other factors. This has been suggested for various land birds endemic to islands (van Riper III *et al*. 1986), as well for grey wolves (*Canis lupus*; Mech and Goyal 1995) and Mednyi island arctic foxes (*Alopex lagopus semenovi*; Goltsman *et al*. 1996).

Evidence of parasite-mediated mortality in wild primates is also available. Cheney and colleagues (1988) found that illness accounted for more deaths than predation in one troop of vervet monkeys (*Cercopithecus aethopis*) with lower-ranking animals suffering more from parasite infections. Mantled-howler monkey (*Alouatta palliata*) mortality increased with the intensity of botfly larvae infections (Milton 1996). Other populations of howler monkeys appear to have been exterminated by yellow fever epidemics (Galindo and Srihongse 1967). Some chacma baboon (*Papio ursinus*) individuals harbored more than 400 ticks. This led to over 50% infant mortality due to tick infestations. The infants were not able to nurse because too many ticks were attached to their muzzles (Brain and Bohrmann 1992). In Bwindi Impenetrable National Park, Uganda, four gorillas were suffering from scabies and died in 1996 (Kalema-Zikusoka *et al*. 2002). Finally, perhaps the most striking example in primates is the Ebola virus, which caused an 80% decline of gorilla and chimpanzee populations in Gabon between 2001 and 2003 (Huijbregts *et al*. 2003; Walsh *et al*. 2003; Bermejo *et al*. 2006). In Lassi Sanctuary Ebola has eliminated an entire population of 143 gorillas that have been studied for 10 years (Leroy *et al*. 2004). Similarly, *Bacillus anthracis* has led to epidemics in chimpanzee populations (Leendertz *et al*. 2004, 2006). There are other cases in which primate populations crashed as a result of disease, but the infectious agents remain unknown (e.g. siamangs (*Symphalangus syndactylus*), Palombit 1992). It remains to be

determined whether such epidemics are part of natural processes or driven by anthropogenic disturbance at local, regional and/or global scales.

The risk factors for the occurrence of disease in conservation projects are complex. A good example is toxoplasmosis in captive lemurs, squirrel monkeys, lion tamarins and Australian marsupials. These animals have evolved in the absence of *Toxoplasma gondii*. Through human intervention (translocation) they have come into contact with these parasites (Frenkel 1989; Cunningham *et al*. 1992; Pertz *et al*. 1997). Due to missing co-evolution the parasites have found naïve hosts without any protective mechanism, and infection therefore have led to the death of the animals parasites (Frenkel 1989; Cunningham *et al*. 1992; Pertz *et al*. 1997). The introduction of similar infections into naïve primate populations could also have fatal consequences, especially in small host populations (May 1988; McGrew *et al*. 1989; McCallum and Dobson 1995; Butynski and Kalina 1998; Homsey 1999).

2.2.1 Disease in the human-wildlife interface

Humans and wildlife have interacted for hundreds of thousands of years, but the level at which these two groups currently interact is unprecedented due to such factors as human population growth, changes in agricultural practices and the extraction of natural resources (Daszak *et al*. 2001; Slinghenberg *et al*. 2004). Recent research has found that over the last 50 years humans have changed the earth's ecosystems more rapidly and extensively than at any other time period in human history (Millenium Ecosystem Assessement 2005). The transmission of pathogens between humans and nonhuman species is driven by anthropogenic factors that increase contact between humans and animals (Daszak *et al*. 2000, 2001). These factors include agricultural expansion and intensification, global travel (tourism, buisness and emigration), animal trade and urbanization, all of which are likely to increase as the human population continues to grow (Daszak *et al*. 2001; Jones *et al*. 2008).

One result of these various human activities has been a greater awareness of the importance of disease interactions between domestic animals, wildlife species and humans (Deem 1998; Wolfe *et al*. 1998; Gao *et al*. 1999). In a survey of emerging pathogens in wildlife in North America, Dobson and Foufopoulos (2001) found that human involvement facilitated 55% of pathogen outbreaks. Only in 19% of the cases there was no evidence of human influence.

Although pathogens 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, pathogen host range and virulence (Daszak *et al*. 2001; Patz *et al*. 2000). Resulting changes in host susceptibility may lead to elevated morbidity and mortality and ultimately in population declines.

Patterns of parasitism in wildlife populations are considered to be influenced by characteristics of the host, such as ranging behavior, density, intra- and interspecific contacts and diet (Nunn *et al*. 2003; Nunn and Altizer 2006), all of which are altered by, for example, forest fragmentation. Habitat degradation and landscape characteristics of fragment boundaries may also influence the frequency and nature of contacts among wildlife, humans and livestock populations. Isolated populations of wild animals are surrounded by or in proximity to humans and their domestic animals. Increasing levels of interaction present greater opportunities for pathogen exchange (Bengis *et al*. 2002; Lafferty and Gerber 2002; McCallum and Dobson 2002; Woodford *et al*. 2002) and the introduction of new diseases into naïve hosts.

The number of human diseases aquired from animals (zoonoses) is large (Sedgwick *et al.* 1975; Siemering 1986; Reinquist and Whitney 1987) and has become an increasingly significant public health threat because of their potential to cause substantial and sometimes widespread disease in humans (Daszak *et al*. 2000; Jones *et al*. 2008). During the past three decades little known human diseases including AIDS, Ebola fever, hantavirus infection and dengue haemorrhagic fever, have merged from enzootic foci and led to major epidemic problems for human health (Hahn *et al*. 2000; Daszak *et al*. 2004; Rouquet *et al*. 2005).

Diseases that are transmitted from humans to animals, called anthropozoonoses, are also numerous, but less well documented and therefore an understudied aspect of global animal health (Acha and Szyfres 1987; Ott-Joslin 1993; Epstein and Price 2009). Some diseases carried by humans and their domestic animals can cross the species barrier and may cause rapid mortality in the new host (Rossiter 1990; Holmes 1996; Butynski 2001). In fact, over 60% of human micro- and macroparasites, and 80% of those reported to infect domesticated animals, are capable of infecting more than one host species (Cleaveland *et al*. 2001; Taylor *et al*. 2001; Woolhouse *et al*. 2001). Some key human-borne pathogens have been shown to infect animals and cause morbidity and mortality. These include measles virus (paramyxoviruses), herpes simplex 1 virus (herpesviruses), protozoal and helminthic parasites

and bacteria such as *Staphylococcus aureus* and *Mycobacterium tuberculosis* (Epstein and Price 2009).

However, zooanthropogenic pathogens are most commonly reported in captive animals or domestic livestock with close human contact. The potential for economic loss and human reinfection is here more apparent. Domestic animals such as cattle, goats and sheep can perpetuate cycles of infection in humans through contamination of drinking water with human enteric pathogens. *Cryptosporidium hominis* and *Giardia lamblia*, both human protozoan parasites, have been found in domestic cattle that have access to reclaimed wastewater (Epstein and Price 2009). Methicillin-resistant *Staphylococcus aureus* is a bacterial pathogen and the typical reason of many hospital based infections. A recent study by Lefebvre *et al*. (2009) showed that dogs which visited human health care facilities were more likely to become infected than those that did not. The dogs were likely to have acquired the infection by licking patients or accepting treats from them.

2.2.2 Cross-transmission between humans and primates

The threat of cross-species infection is especially relevant between humans and primates because of their similar physiology (Wolfe *et al*. 1998; Woodford *et al*. 2002). They are susceptible to many of the same infectious diseases. This fact has long been evidenced by the widespread use of primates as models in biomedical research (Brack 1987; Chapman *et al*. 2005). Primates have long been the focus of surveillance for potential zoonoses such as yellow fever, malaria and schistosomiasis (Coatney *et al*. 1971; Ghandour *et al*. 1995; Robertson *et al*. 1996). The interest in primate associated zoonoses has grown dramatically since the global HIV/AIDS pandemic was definitively traced to the transmission of SIV from chimpanzees (*Pan troglodytes troglodytes*, HIV-1) and from sootey mangabeys (*Cercocebus atys*, HIV-2) in West Africa (Gao *et al*. 1999; Hahn *et al*. 2000; Keele *et al*. 2006). Indeed, recent work has shown that wild primate populations harbor an additional variety of pathogens similar to those causing significant disease in humans (Leendertz *et al*. 2006; Chi *et al*. 2007; Perpens *et al*. 2007), which may be zoonotic. Related retroviruses (i.e. simian foamy virus) and filoviruses (i.e. Ebola) continue to pass between wild primates and people with disquieting regularity through the widespread practice of hunting and butchering wild primates (Leroy *et al*. 2004; Wolfe *et al*. 2005).

Humans have been responsible for massive irrevocable changes to primate habitats within the last several decades (Chapman and Peres 2001). The increasing contact between wild nonhuman primates and humans (researchers, tourists, and local inhabitants) is considered by many primatologists to pose a considerable threat to the wild animals (Wolfe *et al*. 1998; Adams *et al*. 1999, 2001; Homsey 1999; Wallis and Lee 1999; Butynski 2001; Whittier *et al*. 2001). In general, as levels of interaction increase so does pathogen exchange, resulting in further risks to both human and primates (Wolfe *et al*. 1998).

Pathogen transmission from humans to primates places both captive and wild animals at serious risk for diseases such as measles and tuberculosis, which are deadly in many primate species (Wolfe *et al*. 1998). There have been several cases of transmission of human diseases to gorillas and chimpanzees in zoos that are well documented but have never been published (Homsey 1999). Japanese macaques (*Macaca fuscata*) in a Korean zoo suffered from an outbreak of measles. The source of infection could not be identified, but it seemed that monkeys were infected by aerosol from infected visitors (cf. Epstein and Price 2009). Herpes simplex virus 1 is another well known example of a cross-transmitted pathogen, associated with high mortality rates (cf. Epstein and Price 2009). The most common outbreaks have also been described in zoo animals. In such an outbreak three white faced saki monkeys (*Pithecia pithecia*) died within three days of the onset of the initial signs of disease. The origin was thought to be a visitor or a zookeeper (Schrenzel *et al*. 2003). Infection with human pathogens may have fatal consequences for immunologically naïve great apes in captivity (Ruch 1959; Brack 1987; Ott-Joslin 1993) with evidence accumulating of similar effects in the wild (Wolfe *et al*. 1998; Adams *et al.* 1999).

In the wild, where humans from across the globe have increasing contact with many primate populations, the potential transmission of human infections to primates has only recently begun to attract attention (Ashford *et al*. 1990; Mudakikwa *et al*. 1998; Werikhe *et al*. 1998; Wolfe *et al*. 1998; Wallis and Lee 1999; Woodford *et al*. 2002). Transmission of such diseases at the interface of protected areas with human settlements can be exacerbated by mixing of people, wildlife and domestic animals when wild animals leave park boundaries, when domestic animals graze illegally within the parks (Bengis *et al*. 2002), and when, for example, tourists, researchers, and field staff enter protected areas to view primates (Macfie 1992; Woodford *et al*. 2002). Of the humans that come into contact with free-living apes, least control can be exerted over villagers, poachers, prospectors, miners, loggers, forest-

product gatherers and in areas of political unrests, refugees, aid workers, soldiers, and bandits. Any of these may carry a multiplicity of potential diseases that can threaten populations of primates (Wallis and Lee 1999; Adams *et al*. 2001).

The main routes of transmission of human diseases to primates are respiratory and fecal oral (Hudson 1992; Kalema and Cooper 1996; Butynski and Kalina 1998; Homsey 1999; Wallis and Lee 1999). Contact with objects contaminated by disease, such as boots, clothes, used toilet paper and tissue handkerchiefs may also play an important role in the transmission of infectious diseases, as well as biting insects. The risk of aerosol-inhalation based infection is directly proportional to the closeness of contact (Woodford *et al*. 2002). Transmission is most likely to take place where close physical contact occurs regularly, such as in sanctuaries, where newly arrived young animals need the comfort of body contact. Factors facilitating a transmission could be inadequate human waste disposal, a general lack of human hygiene, or that primates forage near human settlements in areas where human waste is not properly covered or is used to fertilize crops. (Whittier *et al*. 2001). Because the types of parasites found in primates also change as a result of exposure to humans, this may result in long-term chronic infections that could cause disability and reduced resistance to fatal diseases.

Small populations living in fragmented, unstable ecosystems may be at particular risk (May and Andersson 1978; Hudson 1992; McCallum and Dobson 1995; Holmes 1996). Large numbers of humans in close contact with such populations may result in disasters similar to that seen with canine distemper virus in African wild dogs *Lycoon pictus* and lions *Panthera leo* which came into contact with domestic dogs in the Serengeti ecosystem of Tanzania (Alexander and Appel 1994; Roelke-Parker *et al*. 1998).

In the field, disease transmission from apes to humans has been documented, and transmission from humans to primates is strongly suspected in many cases. Human herpes simplex virus is common in nonhuman primates and may reflect contact with humans (Wolfe *et al*. 1998). Wild baboons have died from tuberculosis, probably as a result of contact with human settlements (Tarara *et al*. 1985; Sapolsky and Else 1987). In Benin, Zaire and Gombe Stream National Park (Tanzania), polivirus transmission from humans to chimpanzees has been suspected and several chimpanzees died (Goodall 1986; Kortlandt 1996). In 1996, respiratory diseases led to the death of at least eleven chimpanzees in Mitumba Community, Gombe (Wallis and Lee 1999). They only had contact to researchers and park staff. In 1993 eleven chimpanzees suffered from possible human originated influenza in Mahale Mountains

National Park (Hosaka 1995). Tourists are a possible source of infection. Five Chimpanzees died of suspected pneumonia in Kasekela Community, Gombe National Park, Tanzania in 1968 (Goodall 1986), and a further nine died from a similar disease in 1987 (Wallis and Lee 1999). At Kibale National Park, Uganda *Escherichia coli* isolates from the feces of human habituated chimpanzees were found to be genetically more similar to isolates from the feces of humans employed in research and tourism than those obtained from humans in a local village who had no regular interactions with them (Goldberg *et al*. 2007).

Similar results were detected by Rwego and colleagues (2008). They could show that the habitat overlap between humans, livestock and mountain gorillas can influence patterns of gastrointestinal bacterial exchange among species. Mountain gorilla populations that overlap in their used habitat with people and livestock tend to harbor *E. coli* bacteria that are genetically similar to those from people and livestock. *E. coli* from tourist associated gorilla groups in particular were consistently most genetically similar to those from local people and livestock (Rwego *et al*. 2008).

But these results have to be interpreted cautiously with respect to transmission. Genetic similarity between bacterial populations does not necessarily imply transmission in the conventional sense (i.e. direct exchange of microbes through direct or indirect contact). Transmission in the Bwindi system, as well as in the Kibale National Park, may occur indirectly and over extended time periods, perhaps through contaminated environmental sources such as soil or water. In addition, there are only a few cases known which have conclusively demonstrated the occurence of the same parasite in humans and primates of the same habitat; one example is *Cryptosporidium parvum* in mountain gorillas and humans in Uganda (Graczyk *et al*. 2001; Nizeyi *et al*. 2002).

To date, bacterial and parasitic infections of typically low virulence have been shown to move from humans to wild apes. Köndgen *et al*. (2008), however, present a strong body of molecular, observational and epidemiological evidence that human paramyxovirus has infected chimpanzees from Taï National Park (Côte d'Ivoire) on at least three occasions, causing a decline in these endangered great apes. Several outbreaks of respiratory illness were observed in the chimpanzees between 1999 and 2006. Morbidity rates were 92% and the mortality rates up to 19%. In the dead chimpanzees human metapneumovirus (HMPV) and human respiratory syncytial virus (HRSV) were found. Both of these measles related viruses

are of human origin based on phylogenetic analysis (Köndgen *et al*. 2008). This is the first definitive proof of virus transmission from humans to wild apes.

2.3 Primates, Protozoa and Macroparasites

Besides the considerable array of communicable diseases, including the common cold, influenza, hepatitis, measles, yellow fever, and Ebola fever, nonhuman primates and humans share a broad variety of protozoan and macroparasitic diseases. These include malaria, schistosomiasis, giardiasis, filariasis and infection with *Strongyloides* spp. (Benirschke and Adams 1980; Kalter 1980, 1986; Toft 1986; Ashford *et al*. 1990, 1996; Meder 1994; Wolfe *et al*. 1998; Homsey 1999; Sleeman *et al*. 2000).

Macroparasite infections (internal) and infestations (external) may cause a variety of disease manifestations in the host. In primates, physiological disturbances, nutritional loss, pathologic lesions leading to serious debilitation, secondary infections in already compromised hosts, and sometimes death have been reported (Ratcliffe 1931; Vickers 1968; Burrows 1972; Benirschke and Adams 1980, Wilson *et al*. 1984).

The nematode *Oesophagostomum stephanostomum* is said to have been responsible for the death of many apes in zoos during the 1950s and 1960s when standards of care were lower than today and the apes were assumed to be stressed (Cousins 1972). In the 1970s the acanthocephalan *Prosthenorchis elegans* came under scrutiny. Numerous studies on captive Old and New World primates have demonstrated that these thorny-headed worms are highly pathogenic leading to morbidity and mortality (Taraschewski 2000). This species has been associated with sudden die-offs of entire colonies of monkeys, lemurs and chimpanzees in captivity, especially in zoological gardens (Moore 1970; Schmidt 1972). It does not directly cause the animals death, but rather causes lesions which enable secondary pathogens to become established, resulting in debilitation and death of the primate (Cubas 1996; Taraschewski 2000; Tantaleán *et al*. 2005).

Generalist parasites are of considerable concern as they are capable of infecting multiple hosts including human and nonhuman primates. High pathogenicity is predicted to drive a parasite extinct before its host; however, generalist parasites (capable of infecting other hosts) can overcome low host density and drive a focal host species to extinction (de Castro and Bolker

2005). Therefore animals within the same taxonomic family, with known generalist parasites, are at high risk of spreading infection to, for example congeneric species. Additionally, the frequency of non-sexual and arthropod transmission mechanisms reflects the ease with which generalist parasites can infect other species of wildlife, as well as human populations (Pedersen *et al*. 2005)

2.3.1 Protozoa

Protozoa are the second most diverse group of parasites reported from wild primates in terms of total number of species (Pedersen *et al*. 2005). Since the late 1990s, protozoa have been reorganized into 13 phyla, with seven of these containing important parasitic genera (Cox 2002). Parasitic representatives of these unicellular eukaryotes inhabit a wide variety of host organs and tissues, including the blood cells, muscles, nervous tissue, intestines, the mouth and genetalia (Bush *et al*. 2001). Although many protozoa, such as *Giardia* and *Entamoeba*, have a direct life cycle and their infective stages (cysts or oocysts) are ingested by contact with infected hosts or via contaminated water or food items (Stuart *et al*. 1998; Rothman and Bowman 2003; Eckert *et al*. 2005), dispersal via biting arthropods represents the dominant transmission strategy among protozoa infecting primates (Pedersen *et al*. 2005).Examples of these vector-borne protozoa are more than 20 species of *Plasmodium* (Garnham 1966; Deane *et al*. 1969; Coatney *et al*. 1971; Davies *et al*. 1991) and over ten species of *Trypanosoma* and *Leishmania* (Lainson *et al*. 1989; Toft and Eberhard 1998).

Reproduction in intestinal protozoa is usually asexual, only species of the phyla Sporozoa (syn. Apicomlexa) and Ciliophora also reproduce sexually. This involves micro- and macrogametes or conjugation (Eckert *et al*. 2000, 2005; Bush *et al*. 2001). The host spectrum of intestinal protozoa can be broad (euryxenous) or very specific (stenoxenous). For example *Giardia intestinalis* can infect a wide array of species of mammals, birds and reptiles (Toft and Eberhard 1998; Eckert *et al*. 2005). *Entamoeba histolytica (sensu latu)* and *Balantidium coli* affect diverse primate species, rats, cats and dogs (Toft and Eberhard 1998; Eckert *et al*. 2005). Others, like *Isospora callimico, I. cebi* or *I. saimiriae* affect one single host species *(*Goeldi's marmoset (*Callimico goeldii*)*,* white-fronted capuchin (*Cebus albifrons*)*,* common squirrel monkey (*Saimiri sciureus*), respectively (Duszynski *et al*. 1999).

Some of the protozoa like *B. coli* and *Entamoeba dispar* are apparently nonpathogenic. Others like *G. intestinalis* and *Cryptosporidium* spp. can cause severe diseases (e.g. gastritis or enteritis) (Toft and Eberhardt 1998). Pathogenicity can even vary highly within one species: *E. histolytica (sensu latu)* infection can be asymptomatic or produce severe diseases in primates (necroulcerative colitis, peritonitis, amoebic abscesses in liver, lung, central nervous system). The impact of such an infection is influenced by multiple factors which include host species, host body condition, environmental factors and of course the parasite strain (King 1976; Toft and Eberhard 1998).

2.3.2 Helminths

Collectively, helminths are the most commonly reported and taxonomically diverse group of parasites in wild primates (Pedersen *et al*. 2005). The major groups of these metazoan parasites include the phyla Platyhelmintha (with the digenean Trematoda, monogenean Cercomeromorpha and Cestodea), Nematoda and Acanthocephala (Schnieder and Tenter 2006). Life cycles of helminth parasites can either be extremly complicated with different hosts for different developmental stages or simple with only a single host (Eckert *et al*. 2005; Schnieder and Tenter 2006)

Trematoda

Trematodes, commonly called flukes, are dorsoventrally flattened, slug-shaped parasites that have two suckers on their bodies in the adult stage. All species exhibit complex multi-host life cycle involving at least two hosts: intermediate hosts, which are usually molluscs or crustaceans, and definitive hosts, which are vertebrates (Kuntz 1972; Eckert *et al*. 2005). The complex life cycles are commonly linked to the feeding strategy of their definitive hosts, which frequently involves contact with molluscan or crustacean intermediate hosts. Infections are considered to be rare compared to nematode and cestode infections in primates, but several prominent examples can be found (Kuntz 1972).

In African monkeys and apes, liver flukes such as *Fasciola* (Hogg 2002) and *Dicrocoelium* (Landsoud-Soukate *et al*. 1995) have been reported. These flukes are known to cause massive mortality and morbidity in domesticated animals (Bush *et al*. 2001). Schistosomes are

probably the most prominent example, as members of this genus cause serious disease in humans. Estimated 200 million humans suffer from schistosomiasis in Africa and Asia (Crompton 1999). Infections have also been reported from African primates (e.g. *Papio*, McGrew *et al*. 1989; Müller Graf *et al*. 1997). Intermediate stages of this parasite (cercariae) are released into water and spread to the definitive primate host through contact with the contaminated water (Schnieder and Tenter 2006). The definitive host sheds eggs with the feces or urine, and snails or other aquatic invertebrates become infected with an early developmental stage of the parasite (Bush *et al*. 2001). Adult flukes reside in the intestinal lumen, liver, bile duct, gall bladder, mesenteric and other abdominal veins, lungs and rarely in other organs (Toft and Eberhard 1998).

Cestoda

Cestodes are another major group within the Platyhelmintha. They are highly diverse and commonly infect nonhuman primate species (Toft and Eberhard 1998). Cestodes or tapeworms inhabit the intestinal tracts of vertebrate animals but they are usually not very pathogenic (Dunn 1968; King 1976). Adult cestodes adsorb nutrients through the surface of their bodies while they are attached to the host with their scolex. Their body is segmented and the egg-filled reproductive segments (proglottids) at the posterior end, once fully developed, are expelled with the feces. Common genera in primates are *Bertiella*, *Anoplocephala* and *Hymenolepis* (Ghandour *et al*. 1995; Ashford *et al*. 1996; Stuart *et al*. 1998).

Generally, cestodes have complex life cycles. The infections of definitive hosts are acquired through ingestion of intermediate hosts (e.g. insects and vertebrate prey). Some species, including *Hymenolepis nana*, can complete their life cycles without an intermediate host (Toft and Eberhard 1998). Gelada baboons (*Theropithecus gelada*) infected with *Taenia serialis* showed painful swellings caused by the larval stages and even death in a significant number of individuals could be observed (Dunbar 1980).

Nematoda

Nematodes are by far the most diverse group of parasitic worms among primates (Nunn *et al*. 2003; Vitone *et al*. 2004). They possess the highest variability in life cycles of all helminths (Bush *et al*. 2001). Some intestinal nematodes exhibit a direct life cycle with diverse

transmission strategies. Some species invade by penetrating the skin, others are ingested as eggs or encysted larvae, or utilize lactogenic or inauterine transmission. Pinworm eggs are even small enough to become airborne. Others have complex life cycles including free-living generations (Orihel and Seibold 1972; Toft and Eberhard 1998; Bush *et al*. 2001). Regarding their host specificity, some nematodes are stenoxenous (family Oxyuridae), while others can infect various genera of primates (*Strongyloides cebus, Longistriata dubia*) (Hugot *et al*. 1994; Toft and Eberhard 1998).

Nematodes move through the gut or live within host tissues, inhabiting the oral cavity, oesophagus, stomach, pancreas (Spiruridae), small intestine (Trichostrongyloidea, Spirurida), large intestine (Oxyurida, Trichuroidea) or lungs (Metastrongylidae) (Toft and Eberhard 1998). Their pathogenicity is also highly variable ranging from asymptomatic to severe pathologies like ulcerative enteritis or lung haemorrhages (Flynn 1973; Toft and Eberhard 1998). Infections with *Oesophagostomum* spp. are symptomless in less severe cases, but heavy infections can lead to diarrhoea, weakness and high mortality rates (Brack 1987). The species *O. stephanostomum* has been responsible for the death of gorillas and chimpanzees (Cousins 1972; Flynn 1973; Brack 1987).

Acanthocephala

Acanthocephalan infections are rarely reported in wild primate populations, although they are well known as dangerous parasites in captive primates (Schmidt 1972). Taxonomically, they are represented only by a few species in primates (Kuntz and Myers 1972; Schmidt 1972, Stuart *et al*. 1998; Toft and Eberhard 1998). Acanthocephalans are heteroxenous: insects, crustaceans and other arthropods act as intermediate hosts (Schmidt 1972; Bush *et al*. 2001). Like cestodes and some nematodes, they possess a holdfast mechanism that anchors them to the gut of the definitive host. Severe pathologies are associated with infections of *Prosthenorchis elegans* (Dunn 1968; Schmidt 1972; Toft and Eberhard 1998). This species has been associated with sudden die-offs of entire colonies of monkeys, lemurs and chimpanzees in captivity (Moore 1970; Schmidt 1972).

2.3.3 Cross-transmission of macroparasites

Worldwide, 3.5 billion people are infected with intestinal parasites and 450 million of them are clinically ill (WHO 1998b). For instance, more than 1.4 billion humans are infected with roundworms (*Ascaris lumbricoides*, Crompton 1999), a nematode that lives in the small intestine which can lead to significant pathology, including reduced growth, lower activity levels and learning disabilities among children (o'Lorcain and Holland 2000).

Cross-transmission of macroparasites to primates has been inferred in a number of African species including wild apes (e.g. Mudakikwa *et al*. 1998; Graczyk *et al*. 2001; Lilly *et al*. 2002), but without any conclusive evidence. Unfortunately, very little information is available on the extent of transfer of parasites between humans and primates. We know even less about the influence of human settlements bordering natural habitats of nonhuman primates on the primates' gastrointestinal parasites.

In Bwindi Impenetrable National Park, Uganda, parasite burdens were found to be higher in habituated gorillas than in unhabituated gorillas (Kalemna 1995). A study at Gombe National Park, Tanzania, showed that the number of parasite species isolated from chimpanzees and olive baboons *Papio anubis* was greatest in the groups that had the most contact with humans (Nutter *et al*. 1993). In different parks, wild gorillas show various degrees of parasite similarity in fecal samples compared to humans from surrounding communities (Ashford *et al*. 1990, 1993, 1996; Hastings *et al*. 1992; McCallum and Dobson 1995; Cooper 1996; Holmes 1996; Eilenberger 1997; Meader *et al*. 1997; Mudakikwa *et al*. 1998). Young Orang Utans kept in close proximity to humans are commonly infected with human malaria, transmitted by mosquitoes, whereas wild Orang Utans are infected with only two species of malarial parasites, neither of which infects humans (*Plasmodium pitheci*, *P. silvaticum* Kilbourne *et al.* 1998).

Human hookworms, *Necator americanus*, may have caused the death of a gorilla in the Volcanoes National Park, Rwanda (Fossey 1983). In Uganda, free-ranging human habituated gorillas and humans and cattle that inhabit adjacent areas, have all been found to be infected with the same assemblage of *Giardia intestinalis* (Graczyk *et al*. 2002). Primate ecotourism (e.g. gorilla watching) has been associated with the possible disease transmission from humans to primates including the human whipworm (*Trichuris trichiura*; Mudakikwa *et al*. 1998; Sleeman *et al*. 2000). Tanzanian baboons with consistent contact with people were

more likely to be infected with *Schistosoma mansoni* than baboons not associated with humans (Müller-Graf *et al*. 1997; Murray *et al*. 2000). A howler monkey that lived in close proximity to humans showed evidence for infection with *Ascaris lumbricoides* (Stuart *et al*. 1990). Nevertheless, all of these examples are circumstantial, coming without direct evidence. Humans, therefore, seem to represent a risk for wild primate populations, which has to be investigated extensively.

Chapter 3

Materials and Methods

3.1 Non-invasive study

A great deal of energy has been expended on studying parasites of free-ranging primates. However, past surveys on wild primates required blood samples from a large number of animals: These were obtained by capturing and anaesthetizing individual animals (Karesh *et al*. 1998). Samples could also be obtained from bushmeat markets (Wolfe *et al*. 2005). However, such approaches raise ethical dilemmas. The anaesthesia of wild animals entails various risk factors like animals falling from trees, over dosage, pre-existing health problems or injuries through the needle, while animals awaking from anaesthesia may become disoriented and create dangers for humans (Sleeman *et al*. 2000). Therefore, many studies have been confined to fecal surveys as it is both difficult and not practical to gain knowledge of parasites in primates by using the older and highly questionable method of studying the parasites that are recovered at necropsy. Parasitological studies based on non-invasive methods have some limitations, but they are often the only feasible and responsible method to obtain samples that can be collected and examined with reference to parasites in free-ranging primates for which capture is not an option (Stuart and Strier 1995).

Although the estimation of parasite infection by stool examination is a common procedure in parasitological surveys, this indirect method of determining parasite infection does not permit calculation of the precise size of the real infection. However, prevalence can be determined reliably in most cases, and the level of infection can be indicated by the number of eggs found in the feces (Thienpont *et al*. 1979). The number of parasite eggs in the fecal material, however, is affected by many factors and likely to be biased by several external and internal factors (Hall 1981; Pritchard *et al*. 1990; Guyatt and Bundy 1993, Stuart and Strier 1995). These factors include, for instance, the fixation and concentration technique (Foryet 1986; Warnick 1992), the consistency of feces and the variation of the daily egg output of female parasites (Thienpont *et al*. 1979). For an accurate diagnosis of these parasites, stool samples have to be collected and examined over an adequate period of time.

Nevertheless, in assessing prevalence of infection the method of coprological examination has proved a very useful tool in parasitology and is used for standard diagnostic procedures. It can be used to screen a large number of individuals quickly. It is non-invasive, uncomplicated and less expensive than methods of direct determination of infection, such as worm expulsion. It can also be repeated over time with a minimal outlay of resources. Despite the problems

described above, it enables the determination of overall parasite infection of a population or an individual with a relatively high success rate. Beside dissections or worm expulsion, which are not feasible when monitoring populations of wild animals, or circulating antigen assays (de Jonge *et al*. 1989), which require advanced skills and are expensive, it is the only method which provides an estimate of the intensity of infection.

3.2. Sampling regime

Samples were obtained from individual animals as often as possible. Sampled individuals were followed during their whole activity time (tamarins from about 6am to 4.30pm; macaques from about 6am to 6pm). When they were observed defecating, the fresh droppings were collected immediately. All sampled animals were known individually by natural markings (for further details see study animal description in chapters 4.2.2 and 5.2.2). Locality, date, group, species, sex, individual and time of defecation were recorded. After collection, the fecal samples of tamarins were immediately preserved in separate 5ml PET vials, the macaque samples in 15ml PET vials, both containing either a defined volume of 10% buffered formalin (solution of 10% formaldehyde and sodium phosphate buffer, pH 7.0) or 85% ethanol. The stool was mixed to prevent uneven sampling due to clustering of parasite eggs. After returning to the camp, samples were weighed and stored at ambient temperature. At least three samples from each of the sampled individuals were collected on non-consecutive days.

Additionally, stool samples from the villagers around the primate habitats were obtained; at least three samples per individual from different days. The samples were stored in 50ml PET vials containing either 10% formalin or absolut ethanol. Only samples with accurate identification of the defecating individual and immediate sample collection were used for parasitological analyses.

The fecal sampling regime is of great importance for measuring parasite species richness (PSR) and prevalence (Huffman *et al*. 1997; Hudson *et al*. 2002). Bias, which could occur during selection of the studied subpopulation, results in a non-representative conclusion about the sampled population (Grimes and Schulz 2002; Kreienbrock and Schach 2002). Sources of selection bias can be opportunistic or selective sampling of a subset of individuals. Such a

subset could consist for instance of captured or hunted animals, sick or injured animals or road kills. Samples should also be assigned to single individuals when studying free ranging animals. This is the only way to ensure an accurate PSR and prevalence calculation. Knowing the identity or even the age and sex of individual primates sampled improves data quality exponentially. Unless samples are from known individuals without duplication, one cannot treat them as independent data points (OIE 2004). This limits conclusions that can be drawn regarding prevalence and specific interactions for both, primates and the parasites.

To avoid these problems, either all troop members were tested equally (tamarins) or individuals for each sex, age and rank were chosen within the troop (macaques). However, in the macaque population, sampling was restricted to members of the groups which could be individually recognized by natural markings or injuries. Therefore, there could be an underestimation of PSR and an over- or underestimation of prevalence in the macaque samples. In addition, sampling effort correlates positively with parasite species richness (Nunn *et al*. 2003; Muehlenbein 2005). Therefore it is necessary to consider an equal number of fecal samples per host.

The immediate sample collection is of great importance for the interpretation of results. It ensures a correct match of the hosts with the collected fecal samples and the avoidance of further contamination by egg laying flies, soil or water. This avoids misinterpretations regarding host specificity for a given parasite taxa (MAFF 1979).

Fixation (involving emersion in a preservation agent for a certain period of time) is another important factor for the recovery and identification of parasite stages in feces. 10% neutral buffered formalin was chosen because it provides several advantages over other fixatives. Firstly, it adequately preserves helminth eggs/larvae (Ash and Orihel 1987). Secondly, it is a good long-term fixative even over several years (Ash and Orihel 1987). For formalin fixation, less distortion of the stages is reported (compared to polyvinyl alcohol for example, which accounts for a higher diagnostic efficiency especially with the performed formalin-ethylacetat sedimentation technique (Caroll *et al*. 1983). Finally, formalin is cheap and available even in developing countries and rural areas, such as the study sites. These advantages exceed the handicap of formalin fixation for the conservation of specific parasites where other fixatives are more suitable. The higher suitability of other fixatives can result in higher recovery rates or better parasite identification (e.g. 2-2.5% potassium dichromate solution for coccidian oocysts, Duszinsky *et al*. 1999).

In addition to formalin fixation, alcohol was also chosen for an additional set of fecal samples for each individual. It was chosen because of its adequacy for eventual molecular analyses of the recovered parasites. For extraction of parasite DNA from stool samples, fecal samples should be stored at -80°C. If this is not possible, potassium chromate or absolut ethanol are the best alternatives (Da Silva *et al*. 1999).

3.3. Laboratory methods

3.3.1 Sedimentation procedure and microscopic examination

Several methods are known for detecting and quantifying different parasite taxa according to the manner by which they have been preserved. They do, however, vary in sensitivity for different types of parasite eggs (Cheesbrough 1987). It was not possible to compare different preservation and concentrating techniques within this study, because the fecal sample weights were too small to be split up into various sub-samples.

For the present study a modified formalin-ethylacetate sedimentation technique was used to process the preserved fecal samples in the laboratory. This technique in combination with formalin fixed fecal samples is reported to be an excellent method for recovering helminths (Ash and Orihel 1987). Müller-Graf (1994) compared different procedures for formalin-fixed baboon stool samples. Formol-ether and direct smear yielded good results, but formol-ether seemed to be better at detecting a greater number of parasite taxa and is easier to standardize. In recent years, the concern with storage and use of ether, a potentially flammable and explosive material, has led to the use of ethylacetate as a substitute (Young *et al*. 1979; cf. Ash and Orihel 1987). Another advantage of ethylacetate over ether is that the detection of the eggs of some species (*Hymenolepis* and *Taenia* species), cysts (*Giardia*, *Entamoeba* spp.) and larvae (*Strongyloides* spp.) seems to be facilitated because they do not get trapped in the debris as often when being centrifuged (Young *et al*. 1979; Ash and Orihel 1987). For detecting even small numbers of parasitic stages in the fecal samples, it is indispensable to use a concentration procedure (Ash and Orihel 1987). Sedimentation procedures are probably the most frequently used in diagnostic laboratories because the sediment will generally contain all the parasites occurring in the stool sample.

In this study, two minor modifications were made compared to the original method (Ash and Orihel 1987; Ash *et al*. 1994). The formalin-fixed fecal solutions were homogenized well before proceeding with the extraction. After that, fecal solution was filtered through a polyamide sieve into a centrifugation tube. The polyamide sieve with standardized mesh size (400µm) was used instead of two layers of wet gauze recommended in the original method. The 15ml centrifugation tube was filled to ten ml with formalin and the solution was stirred well. Then three ml of ethylacetate were added, and the solution was shaken vigorously for at least one minute. The tube was centrifuged for ten min at 500*g*, compared to five min in the original method. The centrifugation step resulted in four layers (from top to bottom): ethylacetate, plug of debris, formalin and sediment. The ethylactetate, the detritus and after that the formalin was decanted. The remaining pellet was suspended in formalin up to volume 0.5ml. From this suspension 100µl were examined on a slide under a Zeiss microscope in a complete and systematic way at a magnification of 100 to 400 or 650. Some drops of iodine were added to facilitate species identification. Samples were microscopically scanned for the presence of eggs and larvae of different intestinal helminths. The examination time was limited to 45min per sample.

The two modifications compared to the original formalin-ethylacetate sedimentation technique were made to achieve a greater sensitivity and accuracy. The polyamide sieve has the advantage of reduced variance introduced by plant fibers or seeds in the fecal remnants. In addition, polyamide is non-adhesive, and therefore the number of recovered parasites after the filtering procedure should be increased. The centrifuging time was raised to have a sufficient concentration of parasite stages in the sediment of the small tamarin fecal samples. Because of the low weight of the samples (mean weight 1.46g, N 356, SD 0.67, range 0.18- 6.24g) and the fact that decanting the top layers after centrifugation bears the risk of loosing parasite stages, the second centrifugation was omitted in order to preserve as much fecal material as possible (Ash and Orihel 1987; Ash *et al*. 1994). In addition, to determine whether eggs or oocysyts were lost during the concentration procedure, the two parts that are routinousley discarded during the procedure were retained: the polyamidsieve and the supernatant after the centrifugation step. The sieve was washed with 15ml of formalin and the suspension was collected. After centrifugation, slides were prepared with the sediment. The supernatant was also investigated under the microscope. In both preparations, none or only sporadic parasite stages could be found.

All samples were number coded before starting the sedimentation procedure, thus the origin of the sample was unknown during each step of the parasitological analysis. An unbiased analysis of the samples was therfore ensured.

Number of slides examined

The likelihood of parasite egg detection increases with increasing number of slides examined for one fecal sample, especially when there is a low intensity infection. It is therefore a trade-off between efficiency in reading slides from as many different individuals as possible and the detection of all parasites present when choosing how many slides to examine. For certain parasites more slides have to be examined than for others (Moriya 1954). Because it is difficult to estimate how many slides to examine for mixed infections, the present study is geared to the study of Müller-Graf (1994) which found that on average 85% of parasite taxa are detected in the first three slides. More slides were only required when the intensity of infection was very low (Müller-Graf 1994). Therefore, all following analyses and results are based on three slides per sample.

In addition, multiple stool examinations of one individual are required before the presence of parasitic infections could be ruled out. Although most helminth eggs are passed on a continual basis, many protozoa are passed intermittently, and their detection is best accomplished by the examination of multiple specimens collected at two or three day intervals (cf. Ash and Orihel 1987; Garcia and Bruckner 1988). Muehlenbein (2005) validated this serial sampling method and showed that it adequately determined the parasite species richness and prevalence. The present study follows these.

Parasite identification

Eggs and larvae were identified by shape, size and other visible structures as far as possible. Measurements were made to the nearest 0.1µm using an ocular micrometer fitted to a compound microscope. For each morphotype mean egg size (length and width) was determined from different stages from one individual if the eggs were not damaged or deformed. For each morphospecies minimum and maximum size, median size and interquartile ranges (IQR) per host species were recorded. Photographs of representative specimens were taken.

In addition to morphological characteristics, reports on geographical distribution and presence of parasites in the wild species or related primates were taken into account. This information was compiled from parasitological standard references (e.g. Kuntz 1972; Orihel and Seibold 1972; Schmidt 1972; Flynn 1973; Toft and Eberhardt 1998; Eckert *et al*. 2005; Schnieder 2006) or review articles (e.g Dunn 1963; Kuntz 1982; Wolff 1990; Gozalo 2003; Lacoste 2009) and specific papers as well as the Global Mammal Parasite Database (Nunn and Altizer 2005). The cited literature on which the final identification was based are provided in Appendix A. Furthermore, helminth identification was done in collaboration with Gertrud Textor-Schneider und Petra Förster from the Universitätsklinikum Heidelberg, Germany, Sektion Klinische Tropenmedizin and with Prof. Dr. Paiboon Sithithaworn from the University of Khon Kaen, Thailand, Department of Parasitology. The taxonomic classification follows Schnieder and Tenter (2006) if not otherwise indicated.

It should be noted, however, that primate parasites are poorly understood and defined taxonomically. Parasite identification is partly presented at the level of family or genus. Identification to species level is not always possible, especially when identification is based on the microscopic examination of eggs and larvae without the adult worms (Ash and Orihel 1987; Gillespie *et al*. 2005b). Gastrointestinal parasite identification is weak by its very nature. For instance, trichostrongyloid, strongyloid, and rhabditoid nematode eggs are similar in size and appearance, making differentiation extremely difficult (OIE 2004). Depending on host species, the excreted eggs of parasites in the feces may vary in size (Faust 1967). In addition, the stage of development of parasite eggs and larvae at the time of conservation can vary with temperature and intestinal passage time, which can further complicate parasite identification (Ash and Orihel 1987). An accurate identification especially for rare parasites in wild hosts is difficult and often impossible.

3.3.2 Coproculture

To distinguish between hookworms and different *Strongyloides* species, coprocultures with the agar plate method after Koga *et al*. (1991) were made. Because of the circumstance in Peru without laboratory equipment and possibilities for adequate storage of primed agar plates, coproculture could only be applied in Thailand for macaque samples.

About 4g of fresh fecal material were placed on an agar plate. The plate was sealed with Parafilm® and maintained for four to five days at room temperature. The presence of larvae was determined by washing the plate with 10% formalin and examination of the wash-out under the microscope.

This procedure is used to provide another *look* at the types of strongylid nematodes present in an animal. Agar Plate Culture (APC) is a highly effective technique for the coprological diagnosis of strongyloides (Arakaki *et al*. 1990; Sukhvat *et al*. 1994; Jongwutiwes *et al*. 1999; Intapan *et al*. 2005). Coproculture allows the eggs found in the feces to develop into the infective L3 larvae. This facilitates the differentiation of nematodes with morphologically similar eggs.

But beside the high sensitivity there are several disadvantages of the APC. APC is expensive, laboratory equipment is necessary near the study site and one should keep in mind that a substantial portion of stool positive by formalin ethylactetat concentration technique could be negative in APC (Sukhavat *et al*. 1994; Intapan *et al*. 2005).

3.4 Estimation of intensity of infection by fecal egg counts

Since the presence of parasites in feces alone does not necessarily mean that an animal is sick, it is also important to get information on intensity of infection. Under natural epidemiological conditions, primate populations can be infested with parasites without harmful effects. Indeed, the pathogenic effects of many parasite species depend mainly on their abundance in the host (strongylids, ascarids) and only quantative coprology will assess the associated disease risks (Ancrenaz *et al.* 2003). A roughly linear, positive relationship between numbers of eggs/g of feces and parasite burden has been observed for a number of parasite species (*e.g.*, Keymer and Hiorns 1986; Pritchard *et al*. 1990; Sithithaworn *et al*. 1991). Therefore, fecal egg counts provide an indirect measure of abundance and intensity of helminth infection.

In addition to species identification of parasite stages in feces, egg and larvae output per 100µl concentrated sediment (EPS) was counted. This refers to the number of eggs or larvae of one parasite species in the fecal sediment after the removal of feeding residuals during the sedimentation procedure. All samples were taken into account. The egg output of three

different samples per individual was counted and for each sample the count of three slides was averaged.

In this study, egg and larvae output refers to facial sediment after removal of feeding residuals instead of "original" fecal mass. The contribution of feeding residuals like plant fibers, seeds or other undigested food items to the original fecal mass is very variable, especially in tamarins, since these hosts often swallow whole seeds (Garber and Kitron 1997; Knogge and Heymann 2003). To reduce the confounding effects on egg and larvae output resulting from fluctuations of undigested food components, eggs per 100µl sediment instead of the usually used eggs per gram fecal mass were counted.

Because fecal egg counts are used to estimate parasite infection, it is important to know how egg counts vary under different conditions. Egg counts may vary within a fecal sample, in different samples over one day, or over consecutive days. Müller-Graf (1994) examined this variability for baboon stool samples. Variation in egg output may be connected with the time of day at which an animal defecates. For certain parasites, such as pinworms, diurnal variation in egg output has been reported (Hawking 1975). For this reason, Müller-Graf (1994) took several fecal samples from individual baboons during one day. The samples of an individual over one day were fairly similar. No diurnal patterns of egg output were obvious; therefore time of collection was not controlled for in this study. The repeatability of intensity over several days was considerably lower than per stool or over one day, and some parasites show hardly any repeatability. One reason may be a day-to-day variation in egg production (Hall 1981).

But the mean egg output of fecal samples collected from the animal will still provide a useful indication of the parasite population dynamics of their community (Hall 1981), even though it may be more difficult to specify the exact intensity of infection of one individual from one or two samples.

Prevalence and PSR are relatively robust metrics. However, neither incorporates the pathogenical potential of the parasites found. Another problem, especially in non-invasive studies, is that PSR and prevalence bear the risk of underestimating the actual number of different species, especially if identification is based on morphological features. However, PSR (e.g. Nunn *et al*. 2003; Vitone *et al*. 2004; Ezenewa *et al*. 2006) and prevalence (e.g. Müller-Graf *et al*. 1997; Chapman *et al*. 2005a, b, 2006; Gillespie *et al*. 2005a, b) have been

used in many studies and are often the only feasible method of quantifying the disease risk to which the host is exposed.

Egg and larval output was taken as a surrogate to measure propagule excretion, which represents a parameter of parasite transmission (Festa-Bianchet 1989; Kapel *et al*. 2006). It is also used as a measure for parasite intensity (Müller-Graf 1994; Stoner 1996; Chapman *et al*. 2005b). The problems of the use are discussed earlier in this chapter (3.3).

The metrics used are considered robust measures to provide an overview of disease risk posed to host populations, however, one also has to include the individual characteristics and pathogenic effects known for the parasite species found.

3.5 Behavioral observations

In addition to fecal sample collection, behavioral data were collected while following the monkey groups the whole day using "instantaneous scan sampling" (Martin and Bateson 1993). With this method, the group was scanned every five minutes and the maintenance activity of all visible individuals, their height in the forest and their approximate location in the area were recorded during a two minute scanning period. This is an appropriate scanning period for rather dispersed groups of primates (Martin and Bateson 1993). The five minute interval between scans was used because it warranted independent data points for group positions. The host groups are able to reach every point of their home range within this period.

Definitions of maintenance activities are self evident: locomotion, feeding, drinking, grooming and resting (Table 3.1). It was differentiated between human-provided food (FH) and natural food sources (FN) if the animals were feeding, and the height classes were grouped into six different categories: ground, 0-5m, 5-10m, 10-15m, 15-20m and >20m. The height was estimated from the ground. The behavior of dependent infants was not recorded.

Due to the fact that in Peru some of the groups were habituated and some were not, no unbiased data collection was possible. Therefore, this dataset was not used for analyses.

Table 3.1 Description of activity categories applied in the "instantaneous scan sampling" (Martin and Bateson 1993)

Activity categories	Description
1. Locomotion (AM)	movements resulting in a displacement of at least two times the body length including walking, running, climbing, jumping but also movements associated with playing, scent marking and searching and hunting for food items
2. Feeding (AF)	manipulation of food items, biting, chewing, and swallowing
3. Grooming (AG)	one individual picking through the hair of another individual (allogrooming) or of itself (autogrooming) with its hands or mouth
4. Drinking (AD)	intake of liquids (f.e. from ground, leaves, rivers)
5. Resting (AR)	sitting or laying with no activity corresponding to 1.-3.

3.6 Statistical analyses

All statistical tests were carried out using SPSS 11.5 (Spss Inc.) for Windows. Parametric tests were conducted whenever possible. If tests showed that data did not meet the required assumptions, equivalent non-parametric tests were performed. All tests were two-tailed and significance levels were set to α=0.05.

PSR and parasite prevalence

In order to detect the general effects on parasite species richness (PSR) a nonparametric repeated measures analysis of variance (ANOVA) was conducted. Host species and study group entered the model as independent variables and their main effects and interaction effects were analysed. In case a significant effect was detected, a ranked t-test for unequal variances was conducted as a post-hoc test.

In order to test for differences in the prevalence of parasites between host species, sexes and groups a Fisher's exact test (extension by Freeman and Halton for more than two categories) was conducted.

To examine the relationship between the different parasite species found in the hosts, Spearman's Rank Correlation Coefficient (r_s) was calculated. In order to analyze the relationship between PSR/prevalence and behavioral variables (activity, height, diet) the Spearman's Rank Correlation Coefficient (r_s) was calculated.

Egg/larvae output

To test differences in eggs and larval excretion for each parasite species and each individual monkey sampled, the parasite count from each of the samples was summed and averaged to determine a relative egg and larval output for each parasite per sample slide. In addition, total helminth load for each individual was quantified by adding all mean helminth parasite egg counts. These were log(x+1) transformed to meet criteria of normality before performing parametric statistical tests. However, normality was not given for all parasite species, therefore Mann-Whitney U-test were performed to look for variation in parasite egg output between species and study groups.

Chapter 4

Peru

Publication: Wenz, A., Heymann, E. W., Petney, T. N. and Taraschewski, H. F. 2010. The influence of human settlements on the parasite community in two species of Peruvian tamarin. *Parasitology* **137**, 675-684.

4.1 Introduction

Parasitic infections have been identified as critical components in conservation biology as reviewed in chapter 2. Though many studies have documented the gastrointestinal parasites of wild populations of African apes (e.g. McGrew *et al*. 1989; Ashford *et al*. 1990, 1996; Lilly *et al*. 2002), baboons (e.g. Eley *et al*. 1989; Appleton and Henzi 1993; Müller-Graf *et al*. 1997; Hahn *et al*. 2003), and howlers (e.g. Stuart *et al*. 1990, 1998; Stoner 1996), the gastrointestinal parasites of other taxa remain poorly known.

In particular, we still have little information on the prevalence of intestinal parasites in feral Neotropical primates (Platyrrhini) (Michaud *et al*. 2003; Phillips *et al*. 2004). Although some basic data are available for a limited number of platyrrhine genera, including *Saimiri* spp., *Alouatta* spp. and *Ateles* spp. (Stuart *et al*. 1990; Tantaleán *et al*. 1990; Stoner 1996; Karesh *et al*. 1998), the general lack of information on parasites in Neotropical primates means that we know even less about the influence of and possible anthropozoonotic transmission from humans and about their facilities on the parasite burden of these primates. Neotropical nonhuman primates, however, are also susceptible to many human diseases such as Chagas disease (Ziccardi and Lourenço de Oliveira 1997) and malaria (Di Fiore *et al*. 2009).

There are some documented cases of suspected cross-transmission of gastrointestinal parasites from humans to nonhuman primates. In a captive common woolly monkey (*Lagothrix lagothricha*) the human hookworm *Ancylostoma brazieliensis* and the human roundworm *Ascaris lumbricoides* were observed (Michaud *et al*. 2003). In a single uakari (*Cacajao calvus rubicundus*) an infection with *Necator americanus* was found (Tantaleán *et al*. 1990). These two cases were suspected to be most likely infections acquired from humans during captivity, because the presence of hookworms and *Ascaris lumbricoides* in New World monkeys is very unusual according to previous studies conducted on Neotropical primates (Horna and Tantaleán 1990; Tantaleán *et al*. 1990; cf. Michaud *et al*. 2003). Clinically these monkeys presented with severe diarrhoea, dehydration and weakness (Michaud *et al*. 2003).

Phillips and colleagues (2004) documented *Schistosoma mansoni* in owl monkeys (*Aotus vociferans*) and *Ascaris* sp. in a capuchin (*Cebus apella*). Although these parasites were found at a location with limited human activity, the authors conclude that the results may warrant further investigations as some researchers have suggested that the presence of these parasites in wild nonhuman primate population may be indicative of anthropozoonotic exchange (Stuart e*t al*. 1990; Hahn *et al*. 2003).

In order to obtain more information on the influence of humans on the gastrointestinal parasite burden in Neotropical primates and a possible cross-transmission of these parasites, the parasite prevalences of wild Neotropical primate troops with different intensities of human contact were investigated. This chapter will present the details of a study on two tamarin species (*Saguinus mystax* and *Saguinus fuscicollis*) in Peru, which were used as model organisms to determine this influence.

4.2 Materials and Methods

4.2.1 Study animals

Saddle-back tamarins (*Saguinus fuscicollis,* Figure 4.2 (A)) and mustached tamarins (*Saguinus mystax,* Figure 4.2 (B)) are two out of 15 species of the genus *Saguinus* included in the family Callitrichidae (Rylands *et al*. 2000). Hershkovitz (1977) identified several subspecies for both species, three for *S. mystax* and 14 for *S. fuscicollis* although modern classification only lists twelve for *S. fuscicollis* (Groves 2001). The investigated groups belong to the subspecies *Saguinus mystax mystax*, and to the subspecies *Saguinus fuscicollis nigrifrons*. Mustached tamarins are the largest and saddle-back tamarins the smallest members of the genus. The body mass of *S. mystax* ranges from 360 to 650g (Soini and Soini 1990) and that of *S. fuscicollis* between 290 to 420g (Heymann 2003). The sexes are not dimorphic but females tend to be slightly larger and heavier than males (Soini and Soini 1990).

Tamarins are widely geographically distributed. They occurr in nearly the whole upper Amazonian basin from the Peruvian and Ecuadorian Andes in the west, in the Guyanas and northern Brazil in the east, as well as in northwestern Columbia, Panama, and southeastern Costa Rica (Rylands *et al*. 1993; Heymann 2003). Tamarins mainly live in high-ground primary rainforest although they can also be found in seasonally flooded forests, patches or

secondary forests. They also range into mixed fruit-plantations (Snowdon and Soini 1988; Soini and Soini 1990; Heymann 2003).

S. mystax and *S. fuscicollis* often occur sympatrically. They commonly live in stable mixed-species troops with only one other congeneric troop sharing and defending the same home range (Terborgh 1983; Smith 1997; Heymann and Buchanan-Smith 2000). In the study area, saddleback and mustached tamarins spend up to 80% of their time together (Heymann 1990). Their home range can vary between ten and 200 ha according to population (Heymann 2003). The group size of mustached tamarins varies between two to twelve individuals (Soini and Soini 1990) and that of saddle-back tamarins ranges from three to ten individuals (Snowdon and Soini 1988; Heymann 2003). The mating system is polyandrous (Heymann and Buchanan-Smith 2000) and groups consist of one to two adults of each sex and their offspring from previous years (Heymann 2003).

Both tamarin species are active for about 10hrs per day, from shortly after dawn until afternoon (Heymann 1995). They spend the night in enclosed sleeping places like palm trees and tree hollows, dense epiphyte tangles and the crotches of trees (Heymann 1995; Smith 1997).

Mustached and saddle-back tamarins are primarily frugivorous but they also ingest insects, other arthropods, small vertebrates, nectar, soil, gums and other exudates (Snowdon and Soini 1988; Garber 1993; Nickle and Heymann 1996; Smith 1997; Heymann and Buchanan-Smith 2000; Heymann *et al*. 2000; Heymann 2003). Known predators of tamarins are raptors, felids, mustelids and snakes (cf. Heymann 1990). Due to their small size they are not regularly hunted by humans for food, but infants are sometimes caught and held as pets.

Both species are listed as being of least concern on the IUCN Red List of threatened species (last assessed in 2008). Populations of wild mustached tamarins have been noted to be stable, although habitat destruction remains a threat to species living in the Amazonian rainforest: *S. fuscicollis* populations however have been found to be declining (IUCN 2010).

4.2.2 Study area

The study was conducted in the Amazonian lowlands of northeastern Peru around the Estación Biológica Quebrada Blanco (EBQB, 4°21'S and 73°09'W), a field site located on the right bank of the Quebrada Blanco, a tributary of the Rio Tahuayo (Figure 4.1). The study area comprises approximately 100ha of undisturbed primary forest and contains a trail system based on footpaths every 100m, in direction from North to South and from East to West (for further details on the study site see Heymann 1995). Additionally, samples were collected in and around the village Diamante on the opposite bank of the Quebrada Blanco. Diamante is the home of ~40-50 people. The village center of Diamante includes seven houses and a school. This population has no adequate medical treatment for intestinal parasites and sanitation is limited to the river and a pit latrine. The only other humans occurring in the area are members of the scientific establishment with access to anthelmintics.

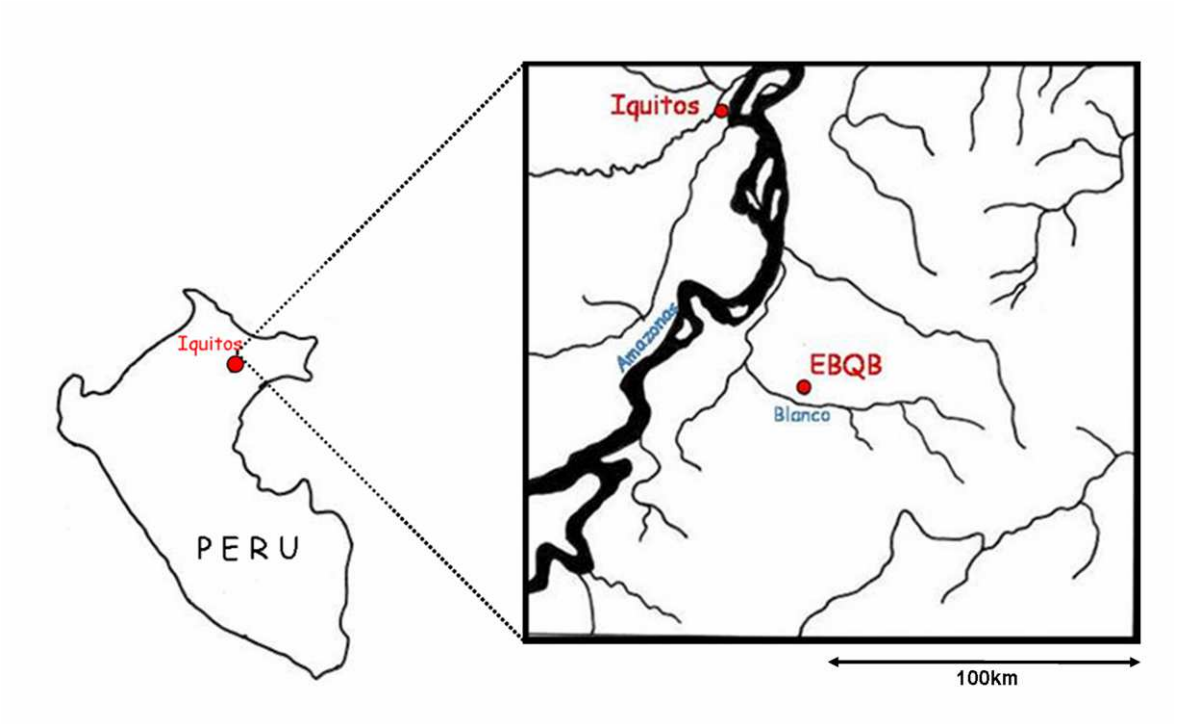

Figure 4.1 Location of the study area. EBQB= Estación Biológica Quebrada Blanco.

4.2.3 Study groups

The subjects of the study were eight wild groups from two species of tamarins: four groups of mustached tamarins (*Saguinus mystax mystax*) and four groups of saddleback tamarins (*Saguinus fuscicollis nigrifrons*). Both species lived in mixed-species troops. Some of the groups have been under observation for up to ten years and are habituated to humans (Müller 2007). All group members are known individually by natural markings (e.g. genital pigmentation, shape of the tail, etc.). The groups investigated differed in their proximity to humans and their facilities. One of the mixed-species troops lived around the village Diamante (group D). The home range of this group reached to the houses of the villagers and included the village fields (Figure 4.3). The second mixed-species troop had its home range around the EBQB with a minimum distance of five meters to the research station (group S). The other groups lived deeper in the forest with no contact to humans or their facilities except for the observers watching them for a variable number of days each month (groups F1 and F2). In all groups males and females of different age classes were present (Table 4.1).

Table 4.1 Study group composition. S.f.= *Saguinus fuscicollis*, S.m.= *Saguinus mystax*,♀ = female individuals, ♂= male individuals. Age classes are defined by Soini and Soini (1990).

Group	Human Contact	Species	Group size	Adults		Subadults		Juveniles	
			♀♂	♀	♂	♀	♂	♀	♂
D	yes	S.f.	7	2	1	3			1
		S.m.	6	2	2	1	1		
S	yes	S.f.	4	2	2				
		S.m.	6	2	1		1	2	
F1	no	S.f.	6	2	4				
		S.m.	5	2	2	1			
F2	no	S.f.	3	2	1				
		S.m.	6	2	2	2			

Table 4.2 Composition of gender and age classes of sampled humans from Diamante. ♀ = female individuals, ♂= male individuals.

Villagers	Adults		Juveniles		Children	
♀♂	♀	♂	♀	♂	♀	♂
27	7	9	2	6	1	2

Figure 4.2 Study host species. A *Saguinus mystax mystax*, B *Saguinus fuscicollis nigrifons*

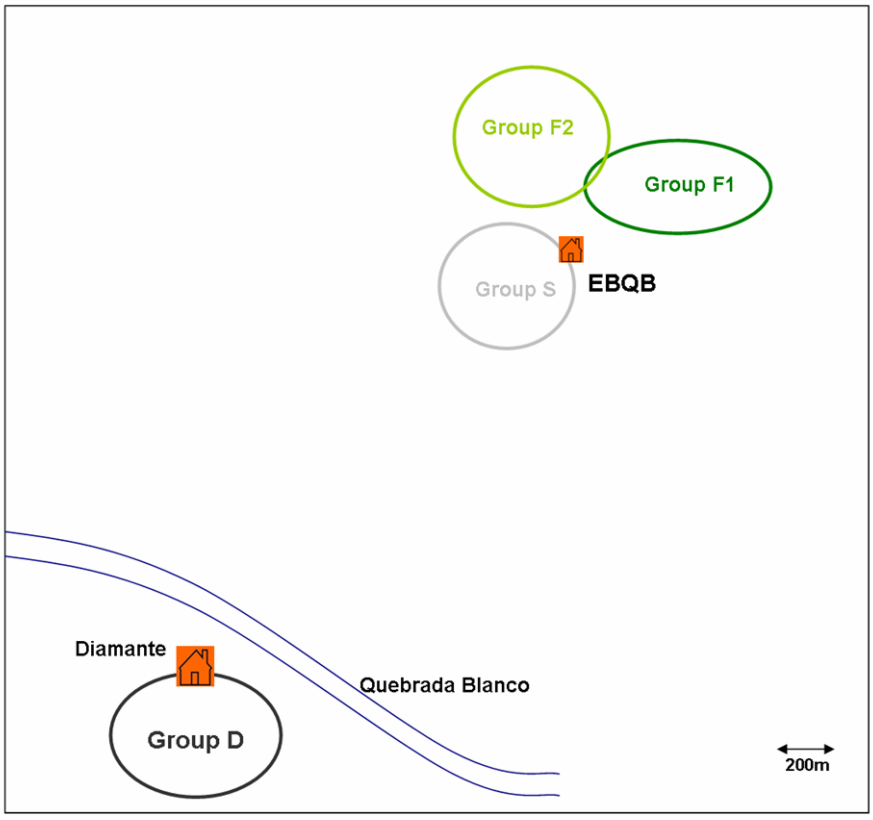

Figure 4.3 Home ranges of the study groups. In grey home ranges of groups with contact to humans (group D and group S) are presented, in green (group F1 and group F2) sylvatic groups.

4.2.4 Study period and sample collection

Collection of fresh fecal droppings from all group members took place during the dry season between July and September of 2007 by following the groups during their whole activity period (~6.00am–4.30pm).

At least three samples from each of the 43 individuals were collected on non-consecutive days. In total, 356 fecal samples from the eight study groups were obtained. Additionally, stool samples from 27 people in Diamante were collected (Table 4.2), at least three samples per individual from different days. In total, 85 human stool samples were obtained.

Fecal samples were gathered directly after defecation and locality, date, group, species, sex, individual and time of defecation were recorded. After collection, the fecal samples were immediately preserved in 10% buffered formalin (solution of 10% formaldehyde and sodium phosphate buffer, pH 7.0). Samples were stored at ambient temperature. The samples were processed following a modified formalin-ethyl acetate sedimentation method (Ash *et al*. 1994) and microscopically examined for the presence of eggs and larvae of different intestinal helminths. In addition the egg (larval) output per 100µl concentrated sediment was counted. For more information on the methods and statistical analyses see chapter 3.

4.3 Results

4.3.1 Human fecal samples

The human feces contained four different helminth species: *Ascaris lumbricoides*, *Trichuris trichiura*, *Strongyloides stercoralis* and hookworm eggs (*Necator americanus*/*Ancylostoma duodenale*). Human hookworm eggs are indistinguishable from eachother. An overview on the recovered helminths and their taxonomy can be found in Table 4.3. The descriptive statistics of length and width of each morphospecies are presented in Table 4.4. Light microscopical photographs of all parasite taxa are presented in Figure 4.5.

The prevalence of *Ascaris* in Diamante was extremely high: in total 88.9% of the tested people had *Ascaris* eggs in their stool (Figure 4.4),. *Trichuris* and hookworms were moderately represented, and *Strongyloides* infections were comparatively rare (Figure 4.4).

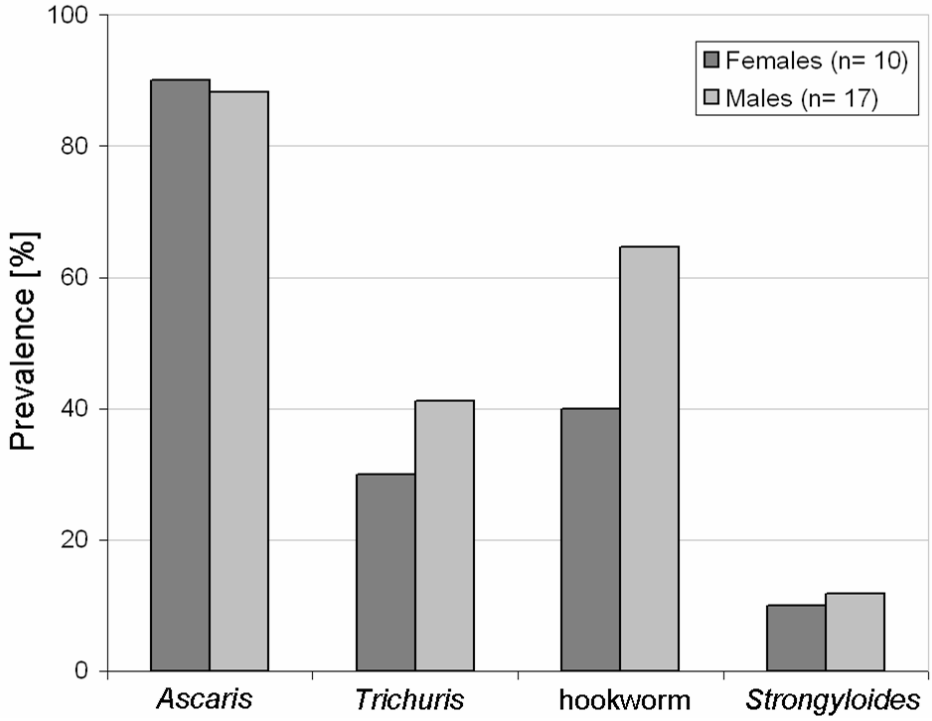

Figure 4.4 Prevalence of infection of all parasite taxa in people of Diamante. (*Ascaris*= *Ascaris lumbricoides*, *Trichuris*= *Trichuris trichiura*, hookworm= *Ancylostoma duodenale*/*Necator americanus*, *Strongyloides*= *Strongyloides stercoralis*). Bars denote prevalence per parasite for females and males of Diamante.

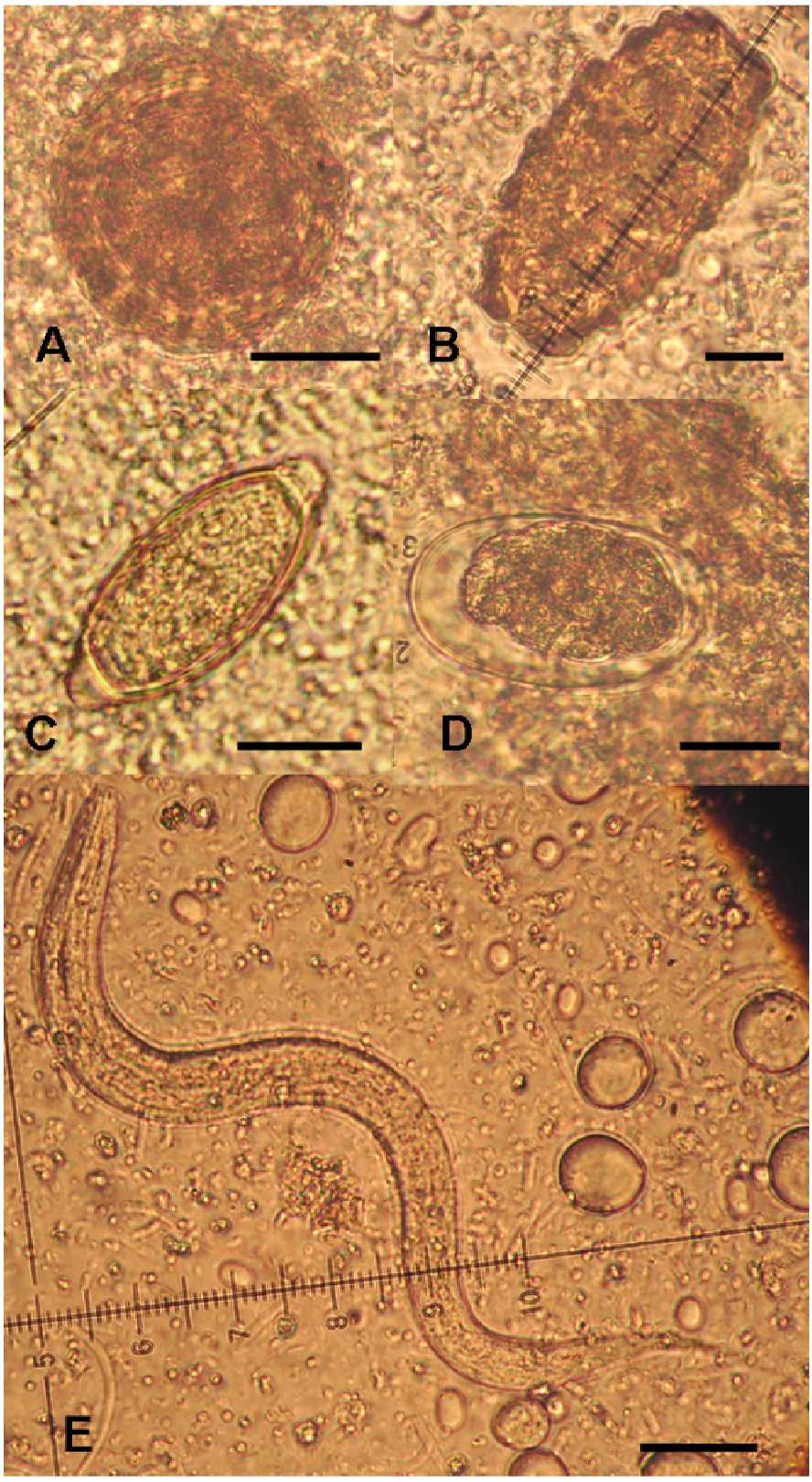

Figure 4.5 (A-E) Light microscope pictures of parasite eggs and nematode larva recovered from stool samples of humans in Diamante. A: fertile egg of *Ascaris lumbricoides*, B: unfertile egg of *A. lumbricoides*, C: *Trichuris trichiura* egg, D: hookworm egg (*Ancylostoma duodenale*/*Necator americanus*), E: *Strongyloides stercoralis* larva; scale bar = 25µm.

Table 4.3 Human parasite identification. All species found as eggs except *Strongyloides stercoralis* larvae. Letters a) and b) give the probable parasite identification. Taxonomy follows Schnieder and Tenter (2006).

Parasite morphospecies	Parasite identification		
	Phylum/Class	**Order/Familiy**	**Species or genus**
1. *Ascaris lumbricoides*	Nematoda/Chromadorea	Ascaridina/Ascarididae	*Ascaris lumbricoides*
2. *Trichuris trichiura*	Nematoda/Enoplea	Trichinellida/Trichuridae	*Trichuris trichiura*
3. hookworm	Nematoda/Chromadorea	Rhabditida/Ancylostomatidae	*a) Necator americanus*
			b) Ancylostoma duodenalis
4. *Strongyloides stercoralis*	Nematoda/Chromadorea	Tylenchida/Strongyloididae	*Strongyloides stercoralis*

Table 4.4 Descriptive statistics of length and width for eggs of four parasite morphospecies from male and female villagers from Diamante. hookworm= *Ancylostoma duodenale/Necator americanus*, N is the number of parasite stages measured. Median, minimum, maximum and IQR (interquartile ranges) are presented for each morphospecies and host gender in µm.

	males		**females**	
***Ascaris lumbricoides* (fertile)**	length	width	length	width
N	15	15	8	8
Median	62.8	52.6	65.8	55.3
Minimum	55.6	42.5	60.6	45.3
Maximum	67.3	57.9	69.9	57.6
IQR	3.3	8.9	3.9	7.0
***Ascaris lumbricoides* (infertile)**	length	width	length	width
N	4	4	5	5
Median	88.4	44.2	88.1	45.6
Minimum	87.9	43.9	85.3	44.2
Maximum	88.8	46.9	89.4	47.1
IQR	0.8	2.3	3.6	2.3
Trichuris trichiura	length	width	length	width
N	14	14	9	9
Median	67.5	39.9	69.8	38.2
Minimum	57.8	36.9	63.8	37.2
Maximum	73.8	42.3	75.3	40.3
IQR	6.0	2.8	2.9	2.3
hookworm	length	width	length	width
N	6	6	6	6
Median	54.9	23.7	54.6	24.5
Minimum	52.4	22.8	53.8	23.6
Maximum	56.1	24.9	56.7	25.8
IQR	2.0	0.8	2.4	1.5

In humans, the mean intensity was moderate except for *Ascaris* (Table 4.5). In one sample more than 6000 *Ascaris* eggs were counted. The maximum for *Trichuris* was 33 eggs, for hookworms 49 and for *Strongyloides* four larvae.

Table 4.5 Intensity of infection in stool samples from Diamante. N gives number of positive stool samples. Mean egg count, maximum egg count and standard deviation are presented for each parasite taxon.

Parasite morphospecies	**Mean**	**SD**	**Maximum**	**N**
Ascaris lumbricoides	490.37	1179.74	6183	70
Trichuris trichiura	10.73	7.76	33	30
hookworm	11.2	12.18	49	50
Strongyloides stercoralis	2.67	1.53	4	6

4.3.2 Tamarin fecal samples

4.3.2.1 Parasite diversity

None of the parasites from the human samples was found in tamarins. Seven parasite morphospecies could be detected in both tamarin species - one acanthocephalan, two cestodes and four different nematode species. Trematodes were not detected in either of the tamarin species studied.

The helminth morphospecies were identified as: the acanthocephalan species *Prosthenorchis elegans*, one of the cestodes belonged to the familiy of Hymenolepididae, probably being *Hymenolepis cebidarum*, the other cestode species could not be further identified and will be referred to as "cestode B". Four morphospecies represent the nematode taxa. The eggs of two nematode morphospecies belong to the order Spirurida: one will be referred to as "large spirurid", probably being *Gongylonema* sp. or *Trichuspirura leptostoma* and the other as "small spirurid". These "small spirurids" could not be determined further. In addition to these spirurids, the eggs of "strongylids" from the orders Rhabditida and Tylenchida, probably *Molineus* sp. or *Strongyloides cebus* were found. Furthermore, nematode larvae belonging either to the order Rhabditida (*Strongyloides*) or to the superfamily Metastrongiloidea (*i.e. Filaroides* and *Angiostrongylus*) were detected. This morphospecies will be referred to as "nematode larva".

An overview on the recovered helminths and their taxonomy can be found in Table 4.6. The descriptive statistics of length and width of each morphospecies are presented in Table 4.7. Information on parasite identification (morphological characteristics, description of potential host species and their origin, potential intermediate hosts) including references is given in Appendix A. Light microscopical photographs of the parasites are presented in Figure 4.6 and 4.7.

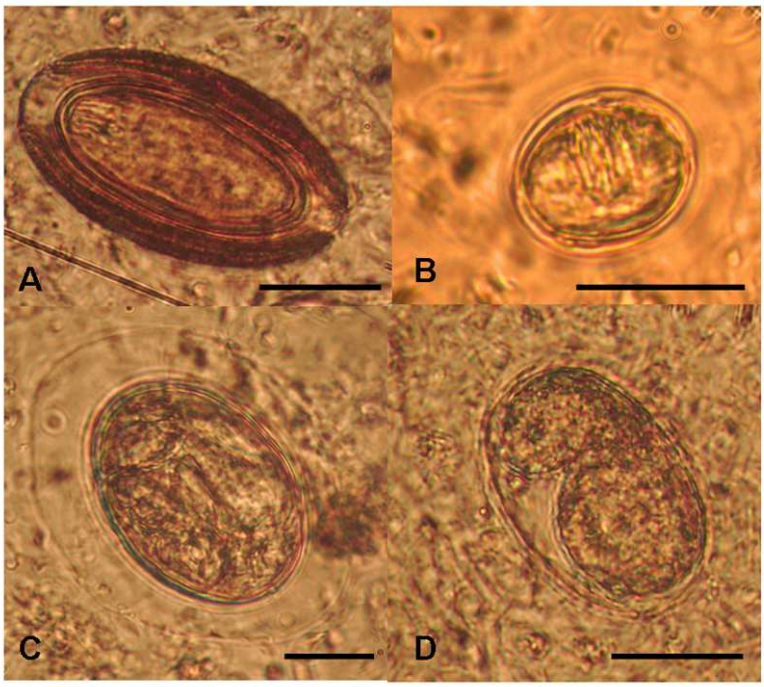

Figure 4.6 (A-D) Light microscope pictures of parasite eggs recovered from tamarin fecal samples. A: *Prosthenorchis elegans*, B: *Hymenolepis* sp., C: cestode B, D: strongylid; scale bar = 25µm

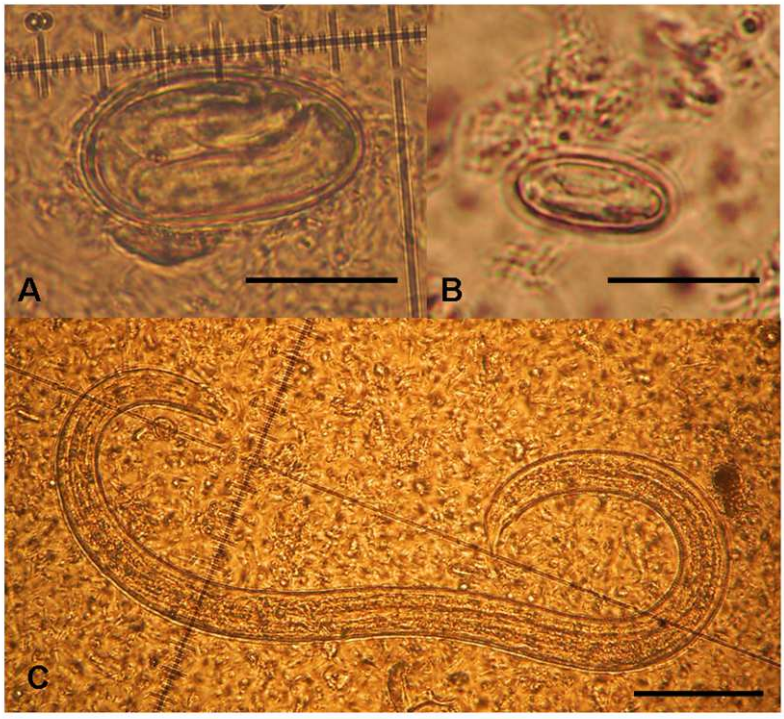

Figure 4.7 (A-C) Light microscope pictures of parasite eggs and nematode larva recovered from tamarin fecal samples. A: large spirurid, B: small spirurid, C: nematode larva; scale bar = 25µm.

Table 4.6 Tamarin parasite identification. All morphospecies found as eggs except nematode larva. Letters a)-c) give the probable parasite identification. Taxonomy follows Schnieder and Tenter (2006) except for [1]Toft and Eberhard (1998).

Parasite morphospecies	Parasite identification		
	Phylum/Class	**Order/Familiy**	**Species or genus**
1. *Prosthenorchis elegans*	Acanthocephala/Arachiacanthocephalea	Oligacanthorhynchida/Oligacanthorhynchidae	*Prosthenorchis elegans*
2. *Hymenolepis* sp.	Plathelmintha/Cestodea	Cyclophillida/Hymenolepididae	*Hymenolepis cebidarum*
3. cestode B	Plathelmintha/Cestodea	Cyclophillida/Anoplocephalidae[1]	*Paratriotaenia* sp.
4. "small spirurid"	Nematoda/Chromadorea	Spirurida/unknown	unknown
5. "large spirurid"	Nematoda/Chromadorea	Spirurida/Gongylonematidae	*a) Gongylonema* sp.
		Spirurida/Rhabdochonidae[1]	b) *Trichospirura leptostoma*
6. "strongylid"	Nematoda/Chromadorea	1. Tylenchida/Strongyloididae	a) *Strongyloides cebus*
		2. Rhabditida/Trichostrongylidae	b) *Molineus* sp.
7. nematode larva	Nematoda/Chromadorea	1. Tylenchida/Strongyloididae	a) *Strongyloides* sp.
		2. Rhabditida/Metastrongylidae	b) *Filaroides* sp.
		2. Rhabditida/Metastrongylidae	c) *Angiostrongylus costaricensis*

Table 4.7 Descriptive statistics of length and width for eggs of six parasite morphospecies found in tamarins. N gives the number of parasite stages measured. Median, minimum, maximum and IQR (interquartile ranges) are presented for each parasite taxon and host species in µm.

	Saguinus mystax		***Saguinus fuscicollis***	
Prosthenorchis elegans	length	width	length	width
N	21	21	21	21
Median	70.1	40.5	68.9	40.3
Minimum	63.9	37.9	66.1	36.9
Maximum	75.0	44.1	75.0	44.3
IQR	4.3	2.3	5.2	1.9
"large spirurid"	length	width	length	width
N	10	10	9	9
Median	57.9	28.3	57.1	30.9
Minimum	47.8	21.9	53.2	29.3
Maximum	64.3	33.6	63.2	33.8
IQR	7.9	7.8	3.9	2.9
"small spirurid"	length	width	length	width
N	65	65	60	60
Median	27.2	11.1	27.2	11.3
Minimum	21.7	10.1	20.9	9.9
Maximum	33.9	17.3	34.2	17.6
IQR	2.2	1.7	3.5	1.8
"strongylid"	length	width	length	width
N	97	97	66	66
Median	52.2	33.9	52.0	33.6
Minimum	46.8	29.3	46.8	29.6
Maximum	69.9	42.2	61.9	40.1
IQR	2.6	3.1	2.4	3.2
***Hymenolepis* sp.** (oncosphere)	length	width	length	width
N	12	12	8	8
Median	28.6	23.3	29.5	23.0
Minimum	25.3	21.8	28.1	22.3
Maximum	32.3	27.8	31.1	25.3
IQR	1.4	2.2	1.4	2.2
cestode B	length	width	length	width
N	4	4	6	6
Median	67.0	58.1	69.1	57.0
Minimum	63.2	57.8	64.8	55.1
Maximum	68.7	60.2	72.6	63.2
IQR	4.5	1.9	3.9	3.8

4.3.2.2. Parasite Species Richness (PSR)

The maximum number of intestinal parasite morphospecies per host individual was seven in *S. fuscicollis* and six in *S. mystax*. Of 43 individuals 41 had multiple parasite infections with at least two (*S. fuscicollis*) or three (*S. mystax*) morphospecies over the study period (Figure 4.8).

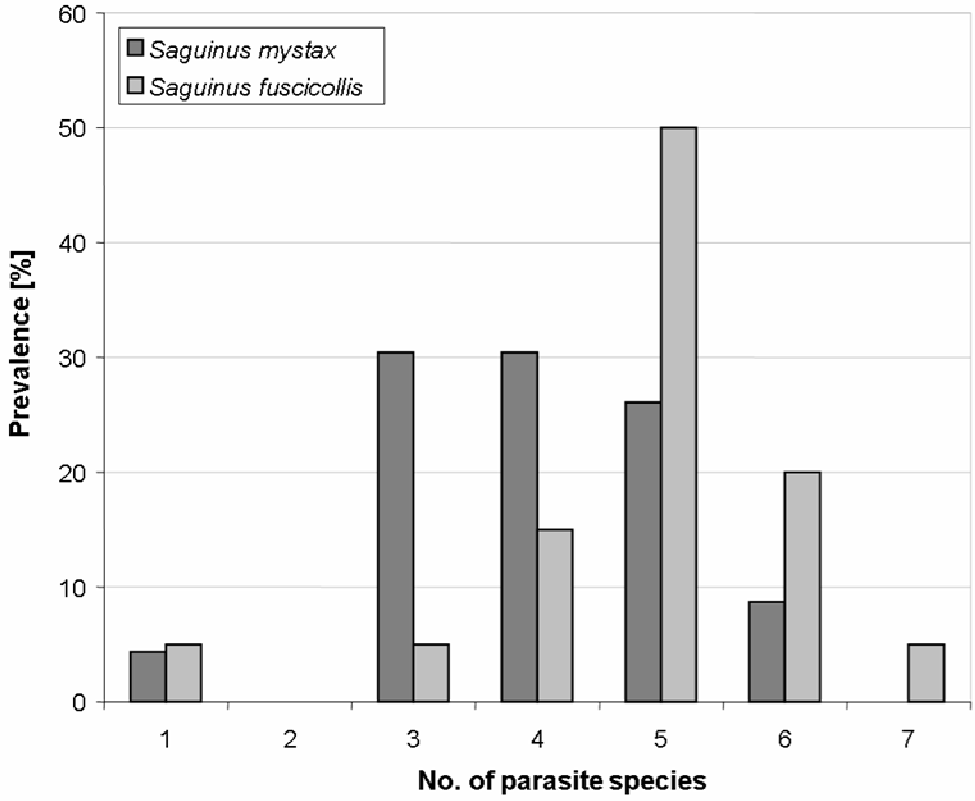

Figure 4.8 Distribution of PSR frequencies per host species. Bars denote the proportion of host individuals with a specific PSR.

The results of the ANOVA including host species and groups revealed significant effects of host species and groups on PSR. An interaction between the independent variables could not be detected to have significant influences on the PSR (Table 4.8).

Table 4.8 Effects of host species and host group on PSR. F-. p-values and degrees of freedom (df) generated by ANOVA.

Effect	df	F	p
Species	1	6.865	0.012
Group	3	5.252	0.004
Species*Group	3	1.008	0.401

Total PSR varied significantly over the host species (Table 4.8). PSR was significantly higher in *S. fuscicollis* than in *S. mystax* (Figure 4.9, PSR: ranked t-test: $t_{S.m\ vs\ S.f.}$ -2.29, df =41, p=0.027.)

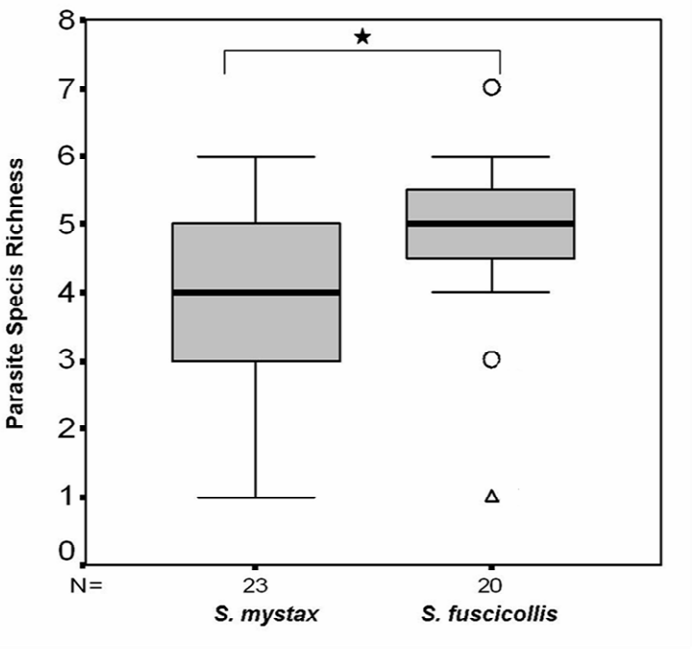

Figure 4.9 Total parasite species richness (PSR) per host species. *S. mystax*= *Saguinus mystax*, *S. fuscicollis*= *Saguinus fuscicollis*. N gives the number of individuals. The boxes show the interquartile ranges, bold horizontal bars show the median. The ends of the whiskers represent the minimum and maximum values that are not outliers. Circles represent outliers. Triangle represents an extreme value. Asterisk indicates statistical differences between species (ranked t-test: * $p \leq 0.05$).

The parasite taxa found were prevalent in all of the mixed species troops, except for *Prosthenorchis elegans*, which was absent from group F2, a non-contact group. Cestode B and the large spirurid were missing in *Saguinus mystax* from group D around the village. (Figure 4.10 for *S. mystax* and in Figure 4.13 for *S. fuscicollis*).

Total PSR varied significantly over the host groups across the different home ranges (Table 4.8). In *S. mystax* PSR was significantly lower in group D than in all other groups (Figure 4.11, PSR: ranked t-test: $t_{D\ vs\ S}$= -2.58, df 10, p= 0.028; $t_{D\ vs\ F1}$ = -4.27, df 9, p= 0.002; $t_{D\ vs\ F2}$= -4.57, df 10, p= 0.001). In *S. fuscicollis*, only members of the study group D showed infections with less than four parasite taxa. However, ranked t-test could not show any significance in PSR for *S. fuscicollis* study groups (Figure 4.12, p> 0.05).

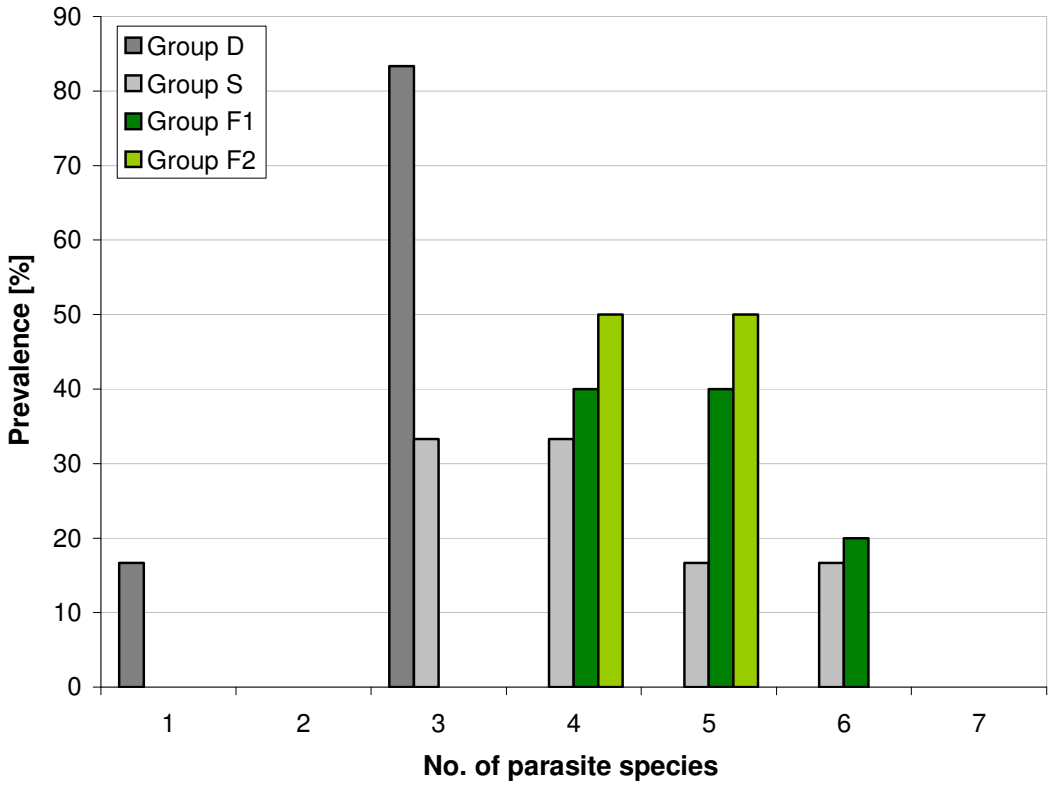

Figure 4.10 Distribution of PSR frequencies per study group in *Saguinus mystax*. Bars denote the proportion of host individuals with a specific PSR. Grey bars represent human contact groups, green bars represent sylvatic groups.

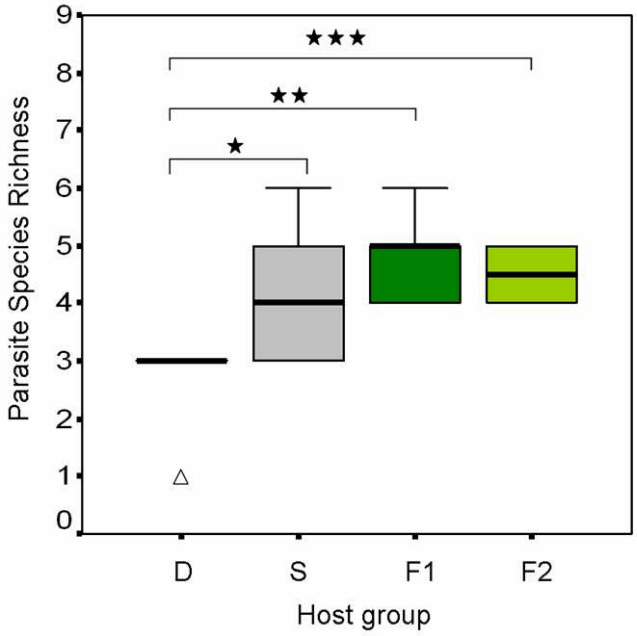

Figure 4.11 Total parasite species richness (PSR) per group in *Saguinus mystax*. Grey boxes represent human contact groups, green boxes represent sylvatic groups. Boxes show the interquartile ranges, bold horizontal bars show the median. The ends of the whiskers represent the minimum and maximum values that are not outliers. Triangle represents extreme value. Asterisks indicate statistical differences between groups (ranked t-test: * $p \leq 0.05$; ** $p \leq 0.01$, *** $p \leq 0.001$).

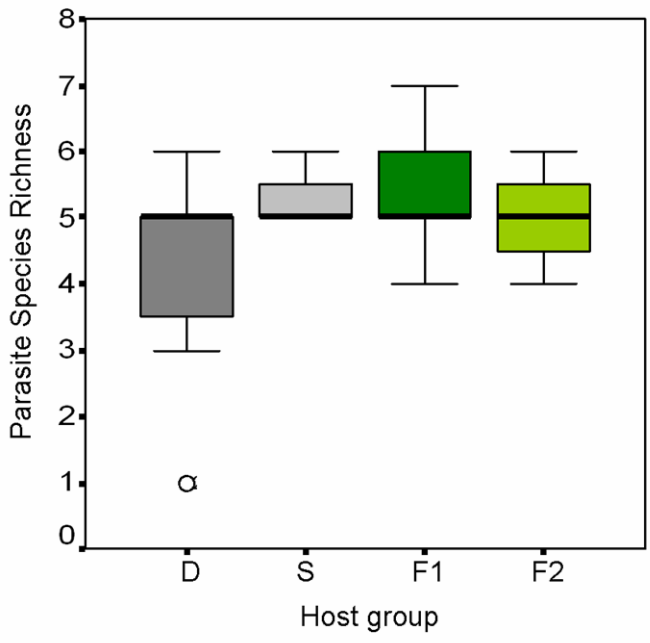

Figure 4.12 Total parasite species richness (PSR) per group in *Saguinus fuscicollis*. Grey boxes represent human contact groups, green boxes represent sylvatic groups. Boxes show the interquartile ranges, bold horizontal bars show the median. The ends of the whiskers represent the minimum and maximum values that are not outliers. Circle represents outlier.

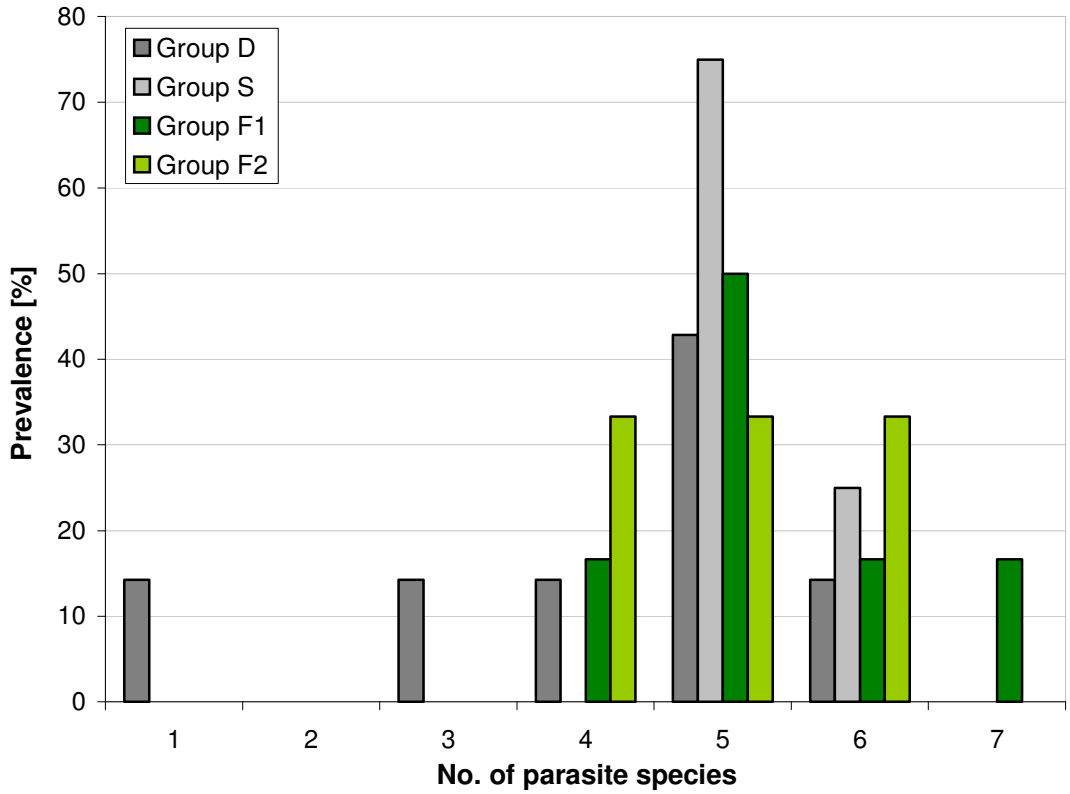

Figure 4.13 Distribution of PSR frequencies per study group in *Saguinus fuscicollis*. Bars denote the number of host individuals with a specific PSR. Grey bars represent human contact groups, green bars represent sylvatic groups.

4.3.2.3 Parasite prevalence

There were no significant differences in the prevalence of parasites between the two tamarin species within the groups (Fisher's exact test, $p>0.05$). Thus species were pooled for further analyses to increase sample sizes.

There were significant differences in the prevalence of parasites between the tamarin groups (Figure 4.14). *P. elegans* was absent in the sylvatic group F2 leading to a significant difference compared to the human contact groups D (Fisher's exact test, $p=0.017$) and S (Fisher's exact test, $p=0.03$). There was also a trend to lower prevalences in the forest group F1 than in the human contact groups D and S, but this was not significant. Nematode larva had a significantly lower prevalence in the village group D than in all other groups (Fisher's exact test D+S, $p=0.029$; D+F1, $p=0.002$; D+F2, $p= 0.006$). The sylvatic groups F1 and F2 had a significantly higher prevalence of cestode B eggs than the human contact groups D and

S (Fisher's exact test, D+F1, p=0.002; D+F2, p= 0.001; S+F1, p= 0.009; S+F2 p= 0.001). None of the other parasite taxa showed significant differences in prevalence between the tamarin groups (Table 4.9).

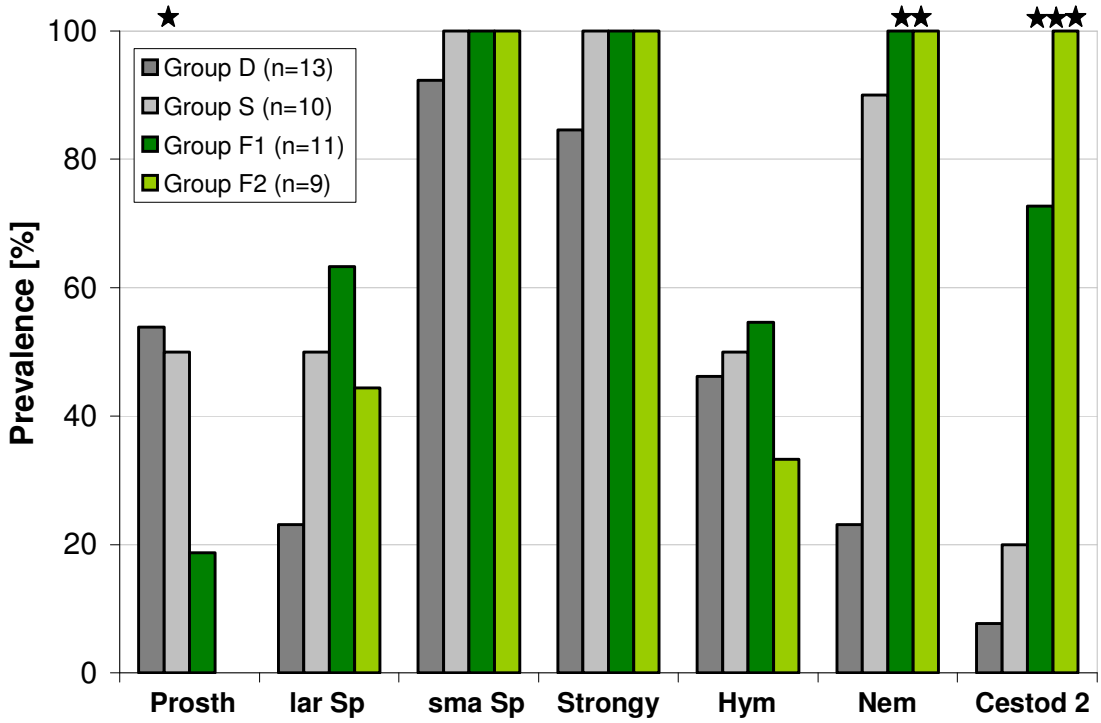

Figure 4.14 Prevalences of all parasite taxa per tamarin group. (Prosth=*Prosthenorchis elegans*, lar Spi=large spirurid, sma Sp=small spirurid, Strongy="strongylid" Hym=*Hymenolepis* sp., Nem=nematode larva, Cestod 2=cestode B). Bars denote prevalence per parasite species for all groups. Grey bars represent human contact groups, green bars represent sylvatic groups. Asterisks indicate statistical differences between groups (Extension of Fisher's exact test: * p≤ 0.05; ** p≤ 0.01, *** p≤ 0.001).

Table 4.9 P-values of Fisher's exact test comparing prevalence of parasite infection in tamarins from the different study groups. Significant results are bold printed.

		Compared Groups					
		D + S	**D + F1**	**D + F2**	**S + F1**	**S + F2**	**F1 + F2**
Parasite taxa	***Prosthenorchis elegans***	1	0.105	**0.017**	0.103	**0.033**	0.479
	large spirurid	0.221	0.095	0.376	0.670	1	0.653
	small spirurid	1	1	1	1	1	1
	strongylid	0.486	0.482	1	1	0.474	0.450
	***Hymenolepis* sp.**	1	1	0.674	0.487	0.650	0.406
	nematode larva	**0.029**	**0.002**	**0.006**	0.476	1	1
	cestode B	0.560	**0.002**	**<0.001**	**0.009**	**0.001**	0.479

4.3.2.4 Interaction of parasite communities in the host

Parasite morphospecies were prevalent in different combinations within the host individuals and some of these combinations could be detected in more than one individual. The most common combination was an multi-infection with the four morphospecies strongylid, small spirurid, nematode larva and cestode B, which occurred in six individuals. Infections with only one morphospecies could only be observed for strongylid and small spirurid. For a detailed list of all occurring single- and multi-infections see Appendix C.

When correlating the prevalence of the different parasite morphospecies, a significant interaction was found between three pairs of morphospecies: *P. elegans* and cestode B (Spearman rank correlation: r_s -0.449, p= 0.003), nematode larva and cestode B (Spearman rank correlation: r_s 0.365, p= 0.016) and large spirurid and nematode larva (Spearman rank correlation: r_s 0.343 p= 0.024). For the analysis each taxon was correlated with all other parasite taxa, so that altogether 43 tests were executed. All results of the Spearman rank correlation can be found in Appendix D.

4.3.2.5 Egg output

Eggs and larvae excreted per 100µl of fecal sediment (EPS) in tamarin samples were also measured. In general, the number of parasite stages excreted in the feces was low for all host individuals over the study period (Table 4.10). The highest EPS was measured for *Prosthenorchis* and small spirurids. The maximum count of *Prosthenorchis* eggs in 100µl of fecal sediment was 158, for small spirurids 1,506. The mean egg output was 5.6 for *Prosthenorchis* and 22.7 for small spirurids. For the other parasite taxa mean output was between 0.4 and 3.8 propagules per 100µl (Table 4.10).

There were no significant differences in the prevalence of parasites between the two tamarin species (Mann Whitney U-test, p>0.05). Thus species were pooled for further analyses to increase sample sizes.

Table 4.10 Fecal egg and larvae output in tamarin groups (refers to number of parasite stages per 100µl fecal sediment). *S. fuscicollis*= *Saguinus fuscicollis*, *S. mystax*= *Saguinus mystax*. SD is the standard deviation, mean and maximum (max) number of parasite stages per sample are shown.

Group		Individuals	*Prosthenorchis elegans*		large spirurid		small spirurid		strongylid		*Hymenolepis* sp.		nematode larva		cestode B	
			Max	Mean (SD)	Max	Mean (SD)	Max	Mean (SD)	Max	Mean (SD)	Max	Mean (SD)	Max	Mean (SD)	Max	Mean (SD)
D	***S.fuscicollis***	7	88	9.7 (SD 11.6)	7	0.4 (SD 0.6)	161	17.8 (SD 19.5)	19	2.3 (SD 2.2)	13	1.9 (SD 2.1)	11	0.8 (SD 1.1)	1	0.03 (SD 0.01)
	S.mystax	6	114	12.2 (SD 18.1)	0	0	262	16.6 (SD 34.7)	21	3.2 (SD 4.1)	21	2.1 (SD 3.2)	13	0.7 (SD 1.8)	17	0
	Total	13	114	10.9 (SD 14.5)	7	0.2 (SD 0.5)	262	17.2 (SD 26.3)	21	2.7 (SD 3.1)	21	2.0 (SD 2.6)	13	0.8 (SD 1.4)	17	0.02 (SD 0.06)
S	***S.fuscicollis***	4	44	3.7 (SD 5.2)	6	0.6 (SD 0.8)	866	33.5 (SD 60.9)	33	5.6 (SD 4.2)	16	1.7 (SD 1.9)	7	0.8 (SD 0.8)	7	0.5 (SD 0.9)
	S.mystax	6	158	19.7(SD 26.8)	1	0.1 (SD 0.01)	186	10.1 (SD 14.6)	14	2.4 (SD 1.5)	20	1.1 (SD 1.7)	17	1.9 (SD 1.3)	4	0.3 (SD 0.6)
	Total	10	158	13.3 (SD 21.5)	6	0.3 (SD 0.5)	866	19.4 (SD 23.2)	33	3.7 (SD 3.1)	20	1.3 (SD 1.7)	17	1.5 (SD 1.2)	7	0.3 (SD 0.7)
F1	***S.fuscicollis***	6	8	0.5 (SD 1.3)	5	0.7 (SD 0.8)	213	20.6 (SD 28.3)	41	6.2 (SD 3.9)	41	5.4 (SD 4.6)	6	1.4 (SD 1.0)	13	1.8 (SD 2.0)
	S.mystax	5	6	0.4 (SD 0.9)	4	0.5 (SD 0.6)	135	18.6 (SD 18.4)	12	4.9 (SD 3.0)	2	0.1 (SD 0.3)	72	6.2 (SD 10.8)	21	2.1 (SD 3.0)
	Total	11	6	0.5 (SD 1.1)	5	0.6 (SD 0.7)	213	19.7 (SD 23.2)	41	5.6 (SD 3.4)	41	3.0 (SD 4.3)	72	3.6 (SD 6.8)	13	1.9 (SD 2.4)
F2	***S.fuscicollis***	3	0	0	11	0.5 (SD 0.8)	1506	73.9 (SD 124.5)	42	4.5 (SD 3.7)	21	2.0 (SD 1.8)	51	2.4 (SD 1.9)	17	0.9 (SD 0.5)
	S.mystax	6	0	0	7	0.5 (SD 0.6)	19	4.4 (SD 3.8)	12	1.9 (SD 3.0)	18	0.7 (SD 1.6)	1	2.0 (SD 1.6)	24	2.0 (SD 2.2)
	Total	9	0	0	11	0.5 (SD 0.7)	1506	27.6 (SD 71.4)	42	2.6 (SD 2.6)	18	1.1 (SD 1.7)	51	2.1 (SD 1.6)	24	1.6 (SD 1.8)

Significant differences in the abundance of *P. elegans*, nematode larva and cestode B could be detected (Table 4.11). For *P. elegans*, the mean abundance in the human contact groups D and S was significantly higher than in the sylvatic group F2 (Mann Whitney U-test$_{D+F2}$: U=39, N= 22, p=0.011; MWU$_{S+F2}$: U=22.5, N=24, p=0.018). Additionally, the mean abundance in group D was significantly higher than in group F1 (MWU: U=39, N=24, p=0.03). The differences between group S and F1 nearly approached significance (MWU: U=32.5, N=21, p=0.059) (Figure 4.15).

For nematode larvae the mean abundance was significantly lower in the village group D than in the forest groups F1 and F2 (MWU$_{D+F1}$:U=29, N=24, p=0.012; MWU$_{D+F2}$: U=20.5, N=22, p=0.009) (Figure 4.16). For cestode B eggs the mean abundance was significantly lower in group D and group S than in groups F1 (MWU$_{D+F1}$: U= 21, N=24, p<0.001 MWU$_{S+F1}$: U= 28.5, N=21, p=0.044) and F2 (MWU$_{D+F2}$: U=0, N=22, p<0.001; MWU$_{S+F2}$: U=7, N=19, p=0.001) (Figure 4.16).

Differences were also significant for the mean abundance of strongylid eggs between the two sylvatic groups F1 and F2 (MWU: U=18, N=20, p=0.017) and between F1 and group D (MWU: U=33.5, N=24, p=0.028) (Figure 4.15). All other abundances showed no significant differences (Table 4.11).

Table 4.11 Mann Whitney U-Test results for differences in parasite egg output between tamarin groups. *Prosthenorchis*= *Prosthenorchis elegans*, *Hymenolepis*= *Hymenolepis* sp., N is the sample size. Significant results are bold printed (* p≤ 0.05; ** p≤ 0.01, *** p≤ 0.001).

		compared groups					
		D+S	**D+F1**	**D+F2**	**S+F1**	**S+F2**	**F1+F2**
Prosthenorchis	U	65	**39**	**27**	32.5	**22.5**	40.5
	p	1	**0.03***	**0.011***	0.059	**0.018***	0.189
large spirurid	U	52	44	46	39	42.5	45
	p	0.342	0.075	0.313	0.24	0.825	0.72
small spirurid	U	64	66	53.5	55	42	45.5
	p	0.951	0.749	0.738	1	0.806	0.76
strongylid	U	45	**33.5**	58	39.5	30	**18**
	p	0.241	**0.028***	0.973	0.275	0.221	**0.017***
Hymenolepis	U	58	63.5	46.5	45.5	40.5	36
	p	0.639	0.62	0.368	0.479	0.682	0.262
nematode larva	U	34.5	**29**	**20.5**	51	31.5	45.5
	p	0.051	**0.012***	**0.009****	0.778	0.27	0.701
cestode B	U	56	**21**	**0**	**28.5**	**7**	24
	p	0.34	**0.001*****	**0.001*****	**0.044***	**0.001*****	0.052
N		23	24	22	21	19	20

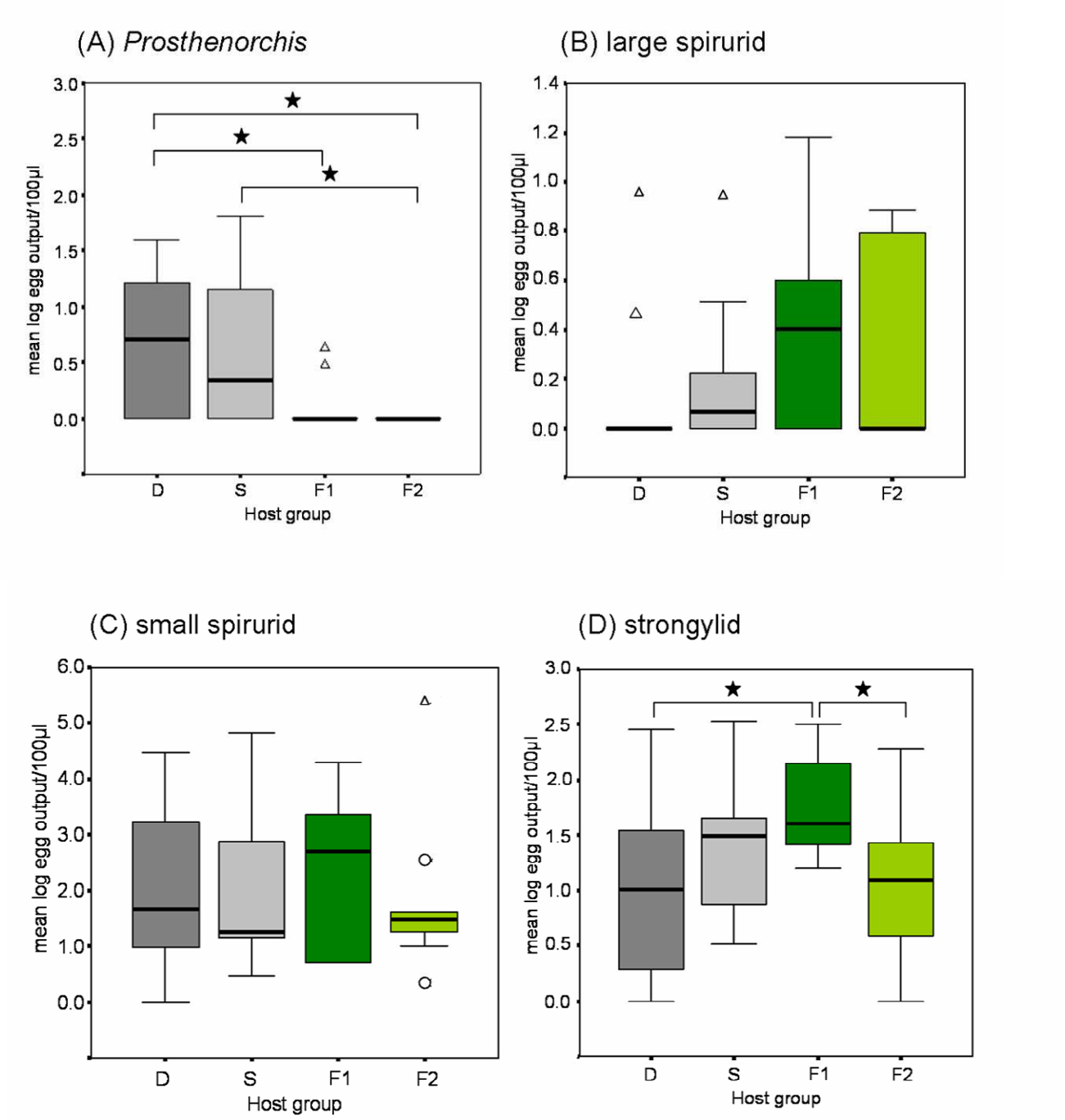

Figure 4.15 (A-D) Mean egg/larvae output for four parasite morphospecies per tamarin group. *Prosthenorchis* = *Prosthenorchis elegans*. Grey boxes represent human contact groups, green boxes represent sylvatic groups. The boxes show interquartile ranges, the bold horizontal bars give the median. The ends of the whiskers represent the largest and smallest values that are not outliners or extreme values. The circles represent outliners and the triangles extreme values. Asterisks indicate statistical differences (Mann Whitney U-test: * $p \leq 0.05$; ** $p \leq 0.01$, *** $p \leq 0.001$).

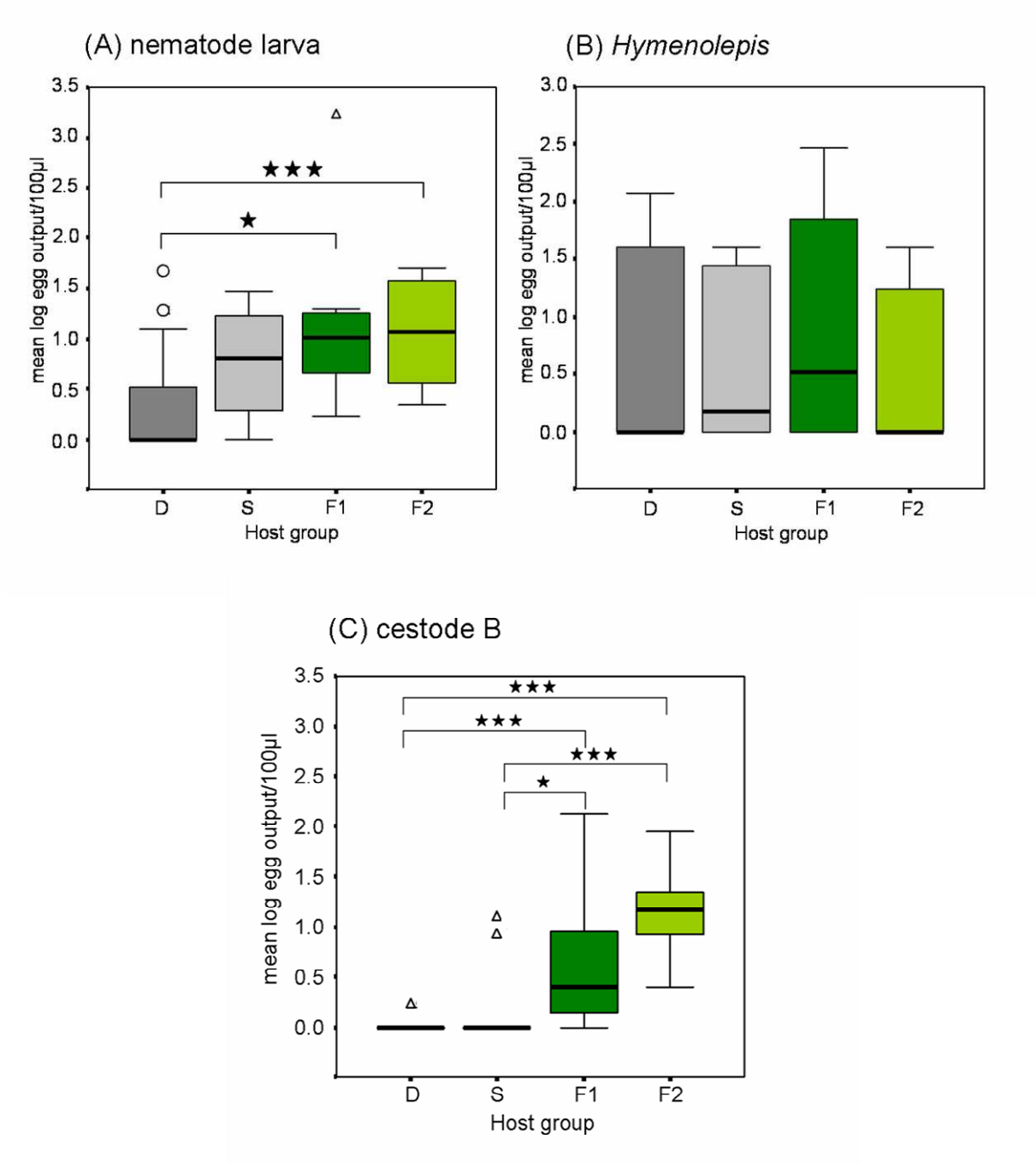

Figure 4.16 (A-C) Mean egg/larvae output for three parasite morphospecies per tamarin group. *Hymenolepis*= *Hymenolepis* sp. Grey boxes represent human contact groups, green boxes represent sylvatic groups. The boxes show interquartile ranges, the bold horizontal bars give the median. The ends of the whiskers represent the largest and smallest values that are not outliners or extreme values. The circles represent outliners and the triangles extreme values. Asterisks indicate statistical differences (Mann Whitney U-test: * $p \leq 0.05$; ** $p \leq 0.01$, *** $p \leq 0.001$).

Differences in mean intensity were also recorded for *P. elegans*. The mean outputs of the human contact groups D (21.8 eggs /100µl) and S (22.0 eggs per 100µl) were considerably higher than in the sylvatic groups F1 (2.6 eggs /100µl) and F2 (0 eggs). Strongylids could be counted slightly more commonly in group F2 than in F1. All other parasite species had similar mean egg outputs in the positive samples (Table 4.14 and Table 4.15).

Table 4.12 Mann Whitney U-Test results for differences in intensity of parasite infection between tamarin groups. *Prosthenorchis*= *Prosthenorchis elegans*, *Hymenolepis*= *Hymenolepis* sp., N is the sample size. Significant results are bold printed (* p≤ 0.05)

		compared groups					
		D+S	**D+F1**	**D+F2**	**S+F1**	**S+F2**	**F1+F2**
Prosthenorchis	U	15	**0**		0		
	p	0.684	**0.04***	/	0.053	/	/
	N	12	9		7		
large spirurid	U	3	9	6	9	5	9
	p	0.269	0.731	1	0.167	0.221	0.34
	N	8	10	7	12	9	11
small spirurid	U	54	66	44.5	55	42	45.5
	p	0.692	1	0.499	1	0.806	0.76
	N	22	23	21	21	19	20
strongylid	U	45	33.5	42	39.5	30	**18**
	p	0.481	0.076	0.869	0.275	0.347	**0.032***
	N	21	22	19	21	18	19
Hymenolepis	U	5.5	16	4.5	8	4.5	6
	p	0.082	0.749	0.243	0.201	0.368	0.439
	N	11	12	9	11	8	9
nematode larva	U	19.5	26	20.5	48	31.5	45.5
	p	0.688	0.865	0.789	0.909	0.426	0.761
	N	14	16	14	20	18	20
cestode B	U	0	0	0	5.5	7	24
	p	0.221	0.12	0.116	0.511	0.634	0.247
	N	3	10	10	10	11	17

Table 4.13 Mean intensity of parasite infection in tamarin groups (refers to number of parasite stages per 100µl fecal sediment). SD is the standard deviation, mean number of parasite stages are shown.

Group		*Prosthenorchis elegans*	large spirurid	small spirurid	strongylid	*Hymenolepis sp.*	nematode larva	cestode B
D	***S.fuscicollis***	17.0 (SD 11.0)	1.0 (SD 0.6)	17.8 (SD 19.5)	2.7 (SD 2.1)	3.3 (SD 1.7)	1.4 (SD 1.1)	0.2
	S.mystax	24.4 (SD 19.3)	0	19.9 (SD 37.7)	3.8 (SD 4.2)	6.1 (SD 1.2)	4.3	0
	Total	21.8 (SD 14.8)	0.9 (SD 0.5)	18.7 (SD 27.0)	3.2 (SD 3.1)	4.2 (SD 2.1)	2.0 (SD 1.6)	0.2
S	***S.fuscicollis***	0.9 (SD 0.1)	0.8 (SD 0.9)	33.5 (SD 60.9)	5.6 (SD 4.2)	2.2 (SD 1.8)	0.8 (SD 0.8)	1.8
	S.mystax	36.1 (SD 30.5)	0.2 (SD 0.03)	10.1 (SD 14.6)	2.4 (SD 1.5)	3.2 (SD 1.0)	2.3 (SD 1.0)	1.5
	Total	22.0 (SD 28.9)	0.6 (SD 0.6)	19.4 (SD 23.2)	3.7 (SD 3.1)	2.6 (SD 1.4)	1.6 (SD 1.2)	1.7 (SD 0.2)
F1	***S.fuscicollis***	1.25	1.1 (SD 0.8)	20.6 (SD 28.3)	6.2 (SD 3.9)	6.5 (SD 4.2)	1.4 (SD 1.0)	2.1 (SD 2.0)
	S.mystax	2	0.8 (SD 0.7)	18.6 (SD 18.4)	4.9 (SD 3.0)	0.67	6.2 (SD 10.0)	2.6 (SD 3.2)
	Total	2.6 (SD 0.9)	0.9 (SD 0.7)	19.7 (SD 23.2)	5.6 (SD 3.4)	5.5 (SD 4.4)	3.6 (SD 6.8)	2.3 (SD 2.4)
F2	***S.fuscicollis***	0	1.4	73.9 (SD 124.5)	4.5 (SD 3.7)	3.0 (SD 0.8)	2.4 (SD 1.9)	0.9 (SD 0.5)
	S.mystax	0	1.0 (SD 0.5)	4.4 (SD 3.8)	1.9 (SD 3.0)	4	2.0 (SD 1.6)	2.0 (SD 2.2)
	Total	0	1.1 (SD 0.4)	27.6 (SD 71.3)	2.6 (SD 2.6)	3.3 (SD 0.8)	2.1 (SD 1.6)	1.6 (SD 1.8)

4.4 Discussion

4.4.1 Human fecal samples

In the human samples the major intestinal geohelminths of temperate and tropical latitudes (*Ascaris lumbricoides, Trichuris trichiura, Necator americanus/Ancylostoma duodenale* and *Strongyloides stercoralis*) were present. The soil-transmitted helminths are a group of parasitic nematode worms with a direct life cycle, which involves no intermediate hosts or vectors. These parasites cause human infection through ingestion of their eggs (*A. lumbricoides* and *T. trichiura*) or by active penetration of the skin by larvae (hookworms and *S. stercoralis*) that can be found in soil, foodstuffs and water supplies contaminated by fecal matter. Eggs develop in the soil and become infective after 2-3 weeks, but can remain infective for several months or years. As adult worms, the soil-transmitted helminths live for years in the human gastrointestinal tract. Most of the geohelminth species have tissue-migratory juvenile stages.

These parasites are highly prevalent worldwide. Recent estimates suggest that *A. lumbricoides* infects over 1.4 billion people, *T. trichiura* 1 billion, and hookworms (*A.duodenale* and *N. americanus*) 1.2 billion people (Holland and Kennedy 2002). They are of worldwide importance and considered together because it is common for a single individual, especially a child living in a less developed country, to be chronically infected with all three worms (Bethony *et al*. 2006). Soil-transmitted helminths produce a wide range of symptoms including intestinal manifestations (diarrhoea, abdominal pain, and anaemia), general malaise and weakness, which may affect working and learning capacities and impair physical growth (WHO 2006). Estimates of annual deaths from soil-transmitted helminth infections vary widely, from 12000 (WHO 2004) to as many as 135000 (WHO 2002).

The presence of these parasites in the rural population of Diamante is not surprising and can be traced back to the poor sanitation and the practice of humans defecating in the forest around the village rather than in toilet facilities (Mücke, personal observation).

All of the parasites found in human fecal samples are suspected to be potentially transmittable to nonhuman primates. Ascarid eggs in feces and adult roundworms in the intestinal tract are commonly reported in a wide variety of primates. In general, they are considered an incidental finding, although fatalities have been reported in monkeys and apes (Pillers 1924; Stam 1960;

Hayama and Nigi 1963; McClure and Guilloud 1971; Orihel and Seibold 1972). Both eggs and adults found in nonhuman primates have been reported as being indistinguishable from the human roundworm, *A. lumbricoides* (Thornton 1924; Augustine 1939; Dunn and Greer 1962; Yamashita 1963; Orihel and Seibold 1972). However, finding morphologically identical nematodes in human and wild primates has to be treated with caution. For example, parasites from humans determined by morphology to be *Oesophagostomum bifurcum* were genetically distinct from those harbored by some nonhuman primates, and these genetic differences were predicted to be associated with distinct transmission patterns (de Gruijter *et al*. 2005; Gasser 2009). Cross-infection has not yet been documented for this parasite species; however, it is suspected in a howler monkey, living close to humans (Stuart *et al*. 1990).

Whipworms are also common nematodes of primates (Muriuki *et al*. 1998; Murray *et al*. 2000; Sleeman *et al*. 2000; Michaud *et al*. 2003). Usually these infections are harmless, although heavy infections may cause severe disease and even death (Ruch 1959; Flynn 1973). The whipworm stages found in primates are described to be indistinguishable from human whipworm *T. trichiura*, and thus, crosstransmission seems to be possible. Experimental transmission from monkeys to humans has been reported (Ruch 1959; McClure and Guilloud 1971; Flynn 1973) and "gorilla watching" has been associated with infections of *Trichuris* in these apes (Mudakikwa *et al*. 1998; Sleeman *et al*. 2000).

Strongyloides infections have also been reported in nonhuman primates. *S. cebus* is known from New World monkeys, and in Old World monkeys, *S. fulleborni* is a common parasite species. However, *S. stercoralis* is also known to infect chimpanzees and other apes (Murata *et al*. 2002; Gillespie *et al*. 2005b). Reports of fatal cases of Strongyloidiasis exist in chimpanzees, gibbons, orangutans, patas monkeys and woolly monkeys (Pillers and Southwell 1929; McClure *et al*. 1973; De Paoli and Johnsen 1978; Benirschke and Adams 1980; Penner 1981; Harper *et al*. 1982).

Hookworms are occasionally reported in Old World monkeys and apes but rarely in New World monkeys (Murray *et al*. 2000; Michaud *et al*. 2003). *N. americanus* is even suspected of being responsible for the death of a gorilla in Rwanda (Fossey 1983).

Even if the presence of hookworms or *A. lumbricoides* in New World monkeys is very unusual according to studies conducted in neotropical primates (Horna and Tantalean 1990; Tantaleán *et al*. 1990; Michaud *et al*. 2003), there are some suspected cases of infection with

N. americanus, *A. braziliensis*, *T. trichiura* and *A. lumbricoides* in New World monkey species (Tantaleán *et al*. 1990; Michaud *et al*. 2003; Phillips *et al*. 2004).

The high prevalences and abundances of these parasites in the human population of Diamante in combination with poor sanitation and defecating in the forest indicate that the villagers could be a potential source of parasites. The parasites could be transferred to the tamarins by ingestion of contaminated soil or food items. However, my results show that this was not the case and the reasons will be discussed in the further sections.

4.4.2 Tamarin fecal samples

4.4.2.1 Parasite diversity

The present study was able to determine the gastrointestinal parasite spectrum of four troops of wild/feral *Saguinus mystax* and *S. fuscicollis* respectively, differing in their contact to humans and their facilities. Identification of the parasites was difficult. Beside the technical problems discussed in chapter 3.2, another fact impedes the species identification even more. While there is a huge amount of literature and even taxonomic keys for the identification of parasite eggs in humans, livestock and pets, it is hard to find appropriate references for parasite identification in primates, especially in wild populations.

In addition to the lack of parasitological reports on primates, especially of Neotropical species in general, many reports lack descriptions of size, life stages, characteristic morphological features and photographic documentation. In many reports little or no information is available on the origin of the host individuals, if it is a zoo or laboratory animal, a pet, wild captured or free-ranging. Parasite diversity is amazing and many species have not yet been described. Therefore, it is probable that wild primates in outlying areas harbor parasite species we do not even know. Although some eggs or larvae may resemble morphologically those of humans or other animals, one cannot exclude the possibility that it is another species, specific for primates. In this study, only *Prosthenorchis elegans* could be identified to species level thanks to an earlier study in this area with examination of a dead animal harboring adult worms (Müller 2007).

Both species showed infection with the same seven intestinal helminth taxa. Since taxonomically related hosts are generally more prone to infections by the same parasite

species spectrum (Gregory *et al*. 1996; Bush *et al*. 2001), it is not surprising that *S. fuscicollis* and *S. mystax* had the same parasite species. Host specifity of the recovered parasite taxa is low, thus there was no reason to expect a different parasite pattern in the two host species. Both tamarin species share many morphological, behavioral and ecological traits which might cause their similarity in parasitism.

4.4.2.2 Cross-transmission of parasites

Comparing the findings for human and tamarin stool samples, none of the helminths found in the villagers, even those which were highly prevalent, could be detected in the feces of either tamarin species. Thus, no transmission of human parasites to tamarins takes place in Diamante. My data therefore do not support the prediction that primates are infected with intestinal parasites obtained from humans. Even if transmission does occur, the low suitability of tamarins as hosts for human intestinal nematodes means these worms cannot establish (short longevity, no excretion of eggs).

Two hypotheses may explain the lack of successful transfer of human parasites to tamarins. First, humans and tamarins vary in their life styles and have different habitat preferences. Tamarins are tree-dwelling primates which only spend 0.5 to 1.6% of their time on the ground (Müller 2007). The possibility that they contact contaminated soil or human feces is therefore very low. All of the recovered human parasites were geohelminths. Since infective stages of these parasites are excreted via the feces or other body fluids and accumulate on or in the ground (Holland and Kennedy 2002), terrestrial species may be at greater risk of acquiring these parasites (Nunn *et al*. 2000). Dunn et al. 1968 found that in mammals from a Malayan rain forest including primates (*Macaca* sp., *Nycetibus* sp, *Presbytis* sp, *Hylobates* sp.) the groundliving ones had the highest prevalence of cestodes and very high prevalences of nematode parasites compared to mammals using the canopy or intermediate hights. However these authors argued that higher prevalences were associated rather with food preferences than with ground use. Minette (1966) proposed that the general absence of *Leptospira* infections in New World monkeys reflects the more arboreal lifestyles of this group of primates, since this parasite is spread through contact with contaminated soil and water. Dunn (1968) even proposed that records of infection with trematodes and *Leptospira* could provide a useful proxy for the degree of arboreality for primate species, of which the behavior has not

yet been studied. The tamarins also do not feed on human food or forage in garbage dumps. Therefore, they can not acquire parasite stages from contaminated food items.

The second, non-mutually exclusive, hypothesis involves the host specificity of the parasites. Tamarins belong to the New World primate family Callitrichidae, which separated about 40 million years ago from the ancestors of humans (Kageyama 2000; Neusser *et al*. 2001), suggesting a long divergence time between the parasite communities infesting these respective hosts. Because of their phylogenetically closer relationship to humans, great apes and Old World monkey species should be more susceptible to human parasites. This is supported by the fact that captive orangutans can carry patent infections of *Giardia lamblia* and *A. lumbricoides* (Mul *et al*. 2007) as well as by many other reports of human parasites in great apes (sees chapter 2.3)

Conversly, none of the parasites found in the tamarins could be detected in human fecal samples, which is an important result for human health care. The two tamarin species are commonly kept as pet animals or occasionally eaten in some rural areas of Peru. Some of their parasites are suspected to be potentially zoonotic (Orihel 1970; Flynn 1973; Michaud *et al*. 2003). Therefore, parasite infection of *S. mystax* and *S. fuscicollis* in combination with poor sanitation, deprived economic background and crowded housing with domestic animals, could pose an important risk to human health (Michaud *et al*. 2003). In addition the recovered parasite taxa in tamarins, as far as it is possible to identify them, have a low host-specificity (see Appendix A). Infection and the manifestation of clinical symptoms in humans is likely to be possible for the parasites recovered, especially the directly transmitted species.

There are explicite records of zoonoses in the literature on the following parasite taxa: Acanthocephala, various *Hymenolepis* sp., *Gongylonema* sp., *Strongyloides* sp. and *Angiostrongylus costaricensis* (Orihel 1970; Flynn 1973; Brack 1987; Neafie and Marty 1993; Michaud *et al*. 2003). Therefore, the close contact between the host species and the rural population in Diamante was also supposed to promote transmission from primates to humans. But the results of this study show, that the presence of wild tamarins near rural human settlements does not to pose a risk to the health of humans in relation to primate gastrointestinal parasites. One factor that minimizes the risk of cross-transmission is that villagers from Diamante do not feed on primates and do not hold them as pets.

In this study at least five out of the seven parasites recovered from the tamarins have an indirect life cycle relying on arthropods or molluscs as intermediate hosts. Transmission

modes and especially obligatory intermediate hosts are key factors for understanding parasite diversity. Probably none of the parasite taxa recovered from the tamarins is transmitted by physical contact from one primate host to the other. Even the so-called directly transmitted parasites, which do not rely on intermediate hosts are closely linked to environmental factors (nematode larvae and "strongylids"). This implies that a potential human influence is not direct through transmission of parasites, but maybe an indirect one by changing the environment of the tamarins.

4.4.2.3 Variation in PSR

Although *S. mystax* and *S. fuscicollis* harbored the same parasite species, the manifested individual parasite species richness (PSR) was different between the species. On average, *S. fuscicollis* individuals were infected with one more parasite species than *S. mystax*. There are a variety of factors that may mediate the compatibility of hosts and parasites including ecology, physiology, immunity and genetics which can vary even between closely related species (Freeland 1983; Kennedy *et al*. 1986; Lile 1998; Whittington *et al*. 2000). Due to these differences, variations in PSR between the studied host groups were analyzed separately for each host species.

In both *S. mystax* and *S. fuscicollis*, the groups living around Diamante showed a significant lower PSR than the other sampled tamarin groups. This result contradicts the prediction made at the beginning of this thesis where a higher PSR was suspected for human contact groups.

One important predictor for PSR is diet composition, especially when parasites with an indirect life cycle are involved. In a meta-study dealing with the proportion of leaves in the primates' diet, the amount of consumed leaves correlated positively with PSR (Vitone *et al*. 2004). The higher proportion of leaves leads to a larger body mass, higher food uptake per se and increases the probability of consumption of contaminated food. However, if directly transmitted parasites are involved to a high degree, the influence of the diet on PSR is less pronounced (Nunn *et al*. 2003). Consumption with contaminated food is possible for the recovered parasites.

The life cycle of many parasites, especially of indirectly transmitted ones, is still poorly understood (Stuart and Strier 1995). The spectrum of intermediate hosts is often unknown and

the knowledge often based on experimental infection alone. The parasite species which were recovered in this study require insects of the orders Lepidoptera, Coleoptera, Siphonaptera, and Dictyoptera to complete their life cycles (e.g. Flynn 1973; Brack 1987; Toft and Eberhard 1998; Eckert *et al*. 2005). During other research projects in this area, the observed prey spectrum of the tamarins studied included the majority of the reported intermediate host taxa (Nickle and Heymann 1996; Heymann *et al.* 2000; Smith 2000). But of the two species, *S. fuscicollis* had a higher diversity of insect prey than *S. mystax* (Nickle and Heymann 1996). Therefore, the amount of insect prey may be a factor influencing the PSR. This would imply that the groups around Diamante ingest less insect prey than the other groups or that the spectrum of insects differs and/or their status as intermediate hosts is reduced.

In addition, differences in foraging strategy and foraging height might also account for the observed heterogeneity in PSR. *S. mystax s*cans the environment visually for prey and mostly captures exposed prey while *S. fuscicollis* manipulates substrates, enters hollow tree trunks and investigates leaf litter, capturing mainly concealed prey (Nickle and Heymann 1996; Heymann and Buchanan-Smith 2000). *S. mystax* forages usually in lower and middle canopy strata, whereas *S. fuscicollis* forages in the understory and on the ground (Nickle and Heymann 1996; Heymann and Buchanan-Smith 2000). Therefore, they might be exposed to different parasites and intermediate hosts.

Both diet composition and differences in foraging strategies are also possible explanations for the heterogeneity of PSR between the study groups. The home ranges of the groups around Diamante differ slightly from those around the EBQB. They lie on the opposite bank of the Rio Blanco and consist of forest patches and agricultural ground. Tamarins forage in the agricultural patches, therefore diet composition can vary between sites. Unfortunately, behavioral observations are limited due to the fact that the groups around Diamante are not habituated and that the objects eaten are often small, feeding activity is rapid and the foraging occurs in relatively high forest strata. Some of the possible intermediate host taxa are minute and therefore ingestion is difficult to assess (e.g. more than half of the species of land snails are smaller than 5 mm (Tattersfield 1996; Tattersfield *et al.* 2001) and coleopterans harboring *P. elegans* are around 3 mm (Stunkard 1965).

High intermediate host abundance might also have a negative influence on PSR and prevalence since a higher arthropod abundance leads to a decreasing per capita probability of encountering infected intermediate hosts (encounter reduction) (Keesing *et al*. 2006). Parasite

egg/larvae output and arthropod ingestion by the hosts is comparatively stable. Therefore, higher arthropod abundance might translate into a lower individual disease risk.

4.4.2.4 Interaction of parasite communities in the host

In this study, a negative correlation between *Prosthenorchis elegans* and cestode B was detected. Both are dependent on intermediate hosts. The intermediate hosts used by both parasites within the study area are uncertain, especially in the case of the unknown cestode species. Therefore competition within the intermediate host or within the definitive host could explain the negative correlation between these two species. However, it is more likely that both parasite species use different intermediate hosts. The composition of the arthropod fauna may be a different one between the study areas. There may also be competition between intermediate host species, leading to smaller abundances or either extinction of one species in a certain habitat. This would decrease the probability of ingestion of an infected individual by the tamarins, which might translate into a lower disease risk.

Since hosts are the habitat patches for their parasites, it is not surprising that intra-and interspecific competition, observed in other species in other habitats, can also be observed in parasites within their hosts (Begon *et al*. 2005). There are many examples of a decreasing fitness of individual parasites within a host with an increasing overall parasite abundance and an increasing overall output of parasites from a host. Many parasites have host tissues and resources in common and it is easy to see that the presence of one parasite species may make a host less vulnerable to attack by a second species (for example as a result of inducible responses in plants) or more vulnerable (simply because of the host's weakened state) (Begon *et al*. 2005). Competition between parasites is a well-known phenomenon from studies on adult helminths in their vertebrate hosts (Hesselberg and Andreassen 1975; Shostak and Scott 1993; see Poulin 1998 and references therein) and also from larval trematodes in their snail first intermediate host (Sousa 1993; Kuris and Lafferty 1994). In all these cases parasites use their host as a source of energy for their own replication, and the regulatory processes are therefore similar to the well-studied larval competition in free-living insects (Fredensbourg and Poulin 2005).

Sharing an intermediate host can also result in interspecific competition among parasites for host resources. Even when parasites share the same definitive host, competitive interactions within the intermediate host can be costly to one or both parasite species (Dezfuli *et al*. 2001; Fredensborg and Poulin 2005). For instance, the size of parasites can be influenced by the number of conspecific and heterospecific individuals sharing the same host (Dezfuli *et al*. 2001). These studies indicate that parasites may interact in hosts, where the parasitic exploitation of host resources is thought to be minimal. Density-dependent reduction in individual parasite size has been most obvious in host-parasite associations where the size of the parasite is large relative to that of the host. Examples of density-dependent effects on parasites in their intermediate host include the development of cestode cysticercoids or procercoids in arthropods, and acanthocephalan cystacanths in amphipods (Gordon and Whitfield 1985; Wedekind 1997; Wedekind *et al*. 2000; Dezfuli *et al*. 2001). Fredensborg and Poulin (2005) showed that the infracommunity of larval helminths in their intermediate host is interactive and that any density-dependent effect in the intermediate host may have lasting effects on individual parasite fitness.

4.4.2.5 Variation in Prevalence

It is widely accepted that there is a relationship between human influenced landscapes and the emergence of zoonotic diseases (Bradley and Altizer 2007; Jones *et al*. 2008), but a potential relationship between to wildlife diseases is less well understood (St-Amour *et al*. 2008). My results show that the presence of humans and an environment modified by humans can lead to substantial changes in the community structure of intestinal helminths in wild nonhuman primates with possibly fatal consequences.

In the present study, the tamarins which foraged in or near the human inhabited area differed markedly from the groups which had less overlap with humans and human-modified habitat. They showed significantly lower prevalences and abundances of a cestode species, but significantly higher levels of the pathological acanthocephalan *Prosthenorchis elegans*. The result does not really support the prediction, that human contact groups harbor other parasites than sylvatic groups. However, my results show, that the parasite burden of human influenced and sylvatic groups differs significantly.

Patterns of parasitism in wildlife populations are thought to be influenced by host ranging patterns, density, intra- and interspecific contact rates and diet, as well as increases in stress levels (Hudson *et al*. 2002; Nunn *et al*. 2003; Nunn and Altizer 2006). Studies of a variety of species have demonstrated that these characteristics can be affected by changes in forest structure (Olupot *et al*. 1994; Heydon and Bulloh 1997; Patriquin and Barclay 2003). Areas of land unaffected by human encroachment or habitat degradation are increasingly rare (Lilly *et al*. 2002).

The planting of crops is one important way in which humans modify the habitat of wildlife species and affect the behavior and diet of primates (Weyher *et al*. 2006). Comparisons of wild foraging and crop-raiding baboons have shown significant differences in the parasite burdens between the troops. Wild foraging troops showed higher prevalences in *Trichuris* sp. infections (Hahn *et al*. 2003; Weyher *et al*. 2006). Weyher *et al.* (2006) suggested that crop-raiding baboons are in better physical condition due to their increased nutrition and therefore able to "fight off" the helminth infections more readily. Alternatively, when parasites survive and reproduce in the host, heightened nutrition may provide favorable conditions for the parasite and lead to an increased burden for the host species (Chapman *et al*. 2006). Weyher (unpubl. data) found that protozoan parasites benefit from the increased starch intake associated with crop-raiding (Chapman *et al*. 2006) and Hahn *et al*. (2003) found increased prevalences of some spirurid nematodes.

Human disturbance of habitats can have indirect influences on parasitic diseases in primates by creating better conditions for vectors, allowing them to increase in abundance, or possibly introducing them into new areas. African colobine monkeys living on the edge of forest fragments were more likely to be infected with multiple species of gut parasites compared to monkeys in the interior of these fragments (Chapman *et al*. 2006). Red tailed guenons (*Cercopithecus ascanius*) in Uganda showed a higher number of intestinal parasites, including a debilitating nematode (*Oesophagostomum* sp.), among fecal samples of logged forest compared to undisturbed forest tracts (Gillespie *et al*. 2005a). Gillespie and Chapman (2006) found that the index of forest patch degradation and the presence of humans strongly influenced the prevalence of parasitic gastrointestinal nematodes in red colobus monkeys.

Differences in prevalence of *Prosthenorchis*

Prevalence of *P. elegans* in the groups around the village and the station showed a similar high level of parasite infestation (50-58%), whereas the forest groups were significantly less infected (0-18%). Therefore, humans seem to have an indirect influence on this parasitic infection in tamarins, since the parasite needs an intermediate host for transmission.

P. elegans is an acanthocephalan parasite of carnivores and primates occurring naturally in South and Central America, including tamarins in Peru (Tantaleán *et al*. 2005). Numerous studies on captive Old and New World primates have demonstrated that these thorny-headed worms are highly pathogenic leading to morbidity and mortality (Taraschewski 2000). In addition, this species has been associated with sudden die-offs of entire colonies of monkeys, lemurs and chimpanzees in captivity (Moore 1970; Schmidt 1972). The eggs of the worm begin their development in the gut of certain arthropods, which serve as intermediate hosts. The final host ingests the parasite cystacanth within the intermediate host. Inside the final host, the parasite lodges in the intestinal mucosa by boring with its proboscis. The deep attachment of the hooked proboscis may cause pain, inflammation, hemorrhaging and secondary infections. Often a complete penetration of the intestinal wall occurs (Schmidt and Roberts 1981), with clinical signs in primates including anorexia, depression and emaciation (Cubas 1996). However, most authors concluded that the parasite does not directly cause the animals death, but rather causes lesions which enable secondary pathogens to become established, resulting in debilitation and death of the primate (Cubas 1996; Taraschewski 2000; Tantaleán *et al*. 2005).

Known intermediate hosts of *P. elegans* are cockroaches (*Blatella germanica*, *Blabera fusca*, *Rhyparobia madera*) and certain coprophagous beetles (*Lasioderma serricorne*, *Stegobium paniceum*) (Schmidt 1972). Captive primates in zoological gardens acquired their infections with *P. elegans* by ingesting cockroaches (Moore 1970), which are also very common in the canopy of tropical rainforests (Basset 2001; Gurgel-Gonçalves *et al.* 2006). Their presence is related to environmental parameters among which human activity is a decisive factor (Boyer and Rivault 2006). Cockroaches frequently feed on human waste as well as on human feces (Burgess *et al*. 1973; Graczyk *et al*. 2005) and prefer to live in sugarcrane fields as well as in palms, guava and bananas (Rasplus and Roques 2010). In addition, *L. serricorne* and *S. paniceum*, two beetles of the family Anobiidae, are known to be associated with humans.

They are well known stored-product pest organisms and occur throughout the tropical and subtropical regions (Hill 1975).

As mentioned in chapter 4.2, the home range of the tamarins around Diamante included the village toilet, the rubbish dump and fields planted with yucca, plantains and other fruit. The tamarins were not observed to feed on this produce, but they did cross the fields and ingested insects on the ground. In addition, part of the tamarin homerange consists of secondary forest. The second human contact troop had its homerange around the EBQB where the influence of humans is less marked. There are no crops, but the tamarin range also included the dump and the toilet.

Currently, it is uncertain how the tamarins contact these parasites and thus far there is no information on which intermediate hosts occur in the area of study. While reasonable knowledge is available on a number of the parasite species affecting domestic animals and wildlife in temperate zones, information on potential intermediate hosts or even life cycles in complex tropical ecosystems remains extremely rudimentary. Recognizing possible intermediate hosts under natural conditions is difficult, due to methodological problems (see chapter 4.4.2.3).

The groups around the EBQB ingest cockroaches only very rarely (Heymann, personal observation). If cockroaches serve as source of infection, as they do in captive tamarins, the prevalence of *P. elegans* infection of cockroaches must be extremely high. In this case, even the very rare event of cockroach ingestion by tamarins would be sufficient for parasite transmission.

The known prey spectrum of the two host species in this area includes amphibians, arachnids, Lepidoptera, Dictyoptera, Orthoptera and Hymenoptera (Nickle and Heymann 1996; Heymann *et al*. 2000; Smith 2000; Müller 2007). In one study at the EBQB the tamarins were also reported to feed on coleopterans (Nadjafzadeh 2005). Therefore, human associated beetles could also act as a possible source of infection in the study area.

Another option would be the presence of so far unknown intermediate hosts. Basic knowledge of the intermediate host spectrum of *P. elegans* is derived from experimental studies or studies on laboratory animals (Stunkard 1965; Schmidt 1972; King 1993; Gozalo 2003). However, experimental studies cannot examine the wide array of potential intermediate hosts that may be relevant in the wild. Thus, many intermediate hosts and their diverse ecological requirements are probably unknown.

Crompton (1970) notes that acanthocephalans use crickets and other adult orthopterans as intermediate hosts. For instance, chimpanzees seem to have acquired *Protospirura muricola* after their release onto Rubondo Island, Tanzania. Rodents or the indigenous vervet monkeys might maintain this parasite naturally as final hosts and insects as intermediate hosts. Reported intermediate hosts of *P. muricola* are beetles and demopterans. However, researchers have not observed chimpanzees consuming these insects. But they are known to eat grasshoppers. Grasshoppers are a known intermediate host of *Protospirura numidica*, a parasite of the lower esophagus and stomach of Palearctic and Nearctic rodents and carnivores (Anderson 2000). Thus, *P. muricola* may also utilize grasshoppers as intermediate hosts (Petrzelkova *et al*. 2006). A similar scenario is also imaginable for *Prosthenorchis elegans*. Like other acanthocephalans, *P. elegans* may use orthopterans as intermediate hosts and tettigoniid orthopterans are the most common prey items of tamarins (Nickle and Heymann 1996; Smith 2000). However, orthopteran intermediate hosts would not explain the fact that human associated groups showed significantly higher prevalences of infection.

The prevalence of *P. elegans* infection found in the human contact tamarins reaches similar values to those in captive monkeys (Middleton 1966; Vickers 1969). This is an astonishing and alarming result. Even if *P. elegans* is natural to primates in South America, the high prevalence could have fatal consequences including mortality. Schmidt (1972, p. 145) suggests that *P. elegans* "is not only the most medically important acanthocephalan parasite of captive primates but [also] may well be the most serious of all pathogens found in this group of hosts".

It was assumed that in natural populations the severity of parasite loads is lowered or controlled through ecological barriers to infection (Dunn 1963). The tamarin habit of swallowing large numbers of sizable seeds – often as large in diameter as the tamarin gut cross section – which pass through the gut rapidly suggests a potential role in parasite expulsion (Garber and Kitron 1997). Self medication against parasitic infections may be practiced by several vertebrate species (Clayton and Wolfe 1993). The elimination of large seeds from the diet of captured and imported tamarins was used as an explanation for the high morbidity and mortality from infection of *P. elegans* in captive populations. The results of my study, however, show that high infection prevalence also occurs in wild tamarins. Therefore, it is important to determine the complete lifecycle of *P. elegans* in the study area in future

studies. It would also be important to collect data on diet composition in the troops around Diamante.

Differences in prevalence of cestode B

Beside the higher prevalence of *P. elegans*, a significantly lower prevalence of a cestode species was found in both groups living in proximity to human facilities. This cestode could not be identified to species level, highlighting the lack of taxonomic work on the parasites of tropical primates discussed earlier (chapter 3.2).

Possible intermediate hosts of cestodes are various insects including fleas (Siphonaptera) but also molluscs. Fleas and molluscs are not covered by the observed prey taxa of tamarins in the study area (Nickle and Heymann 1996; Heymann *et al*. 2000; Smith 2000; Müller 2007). This can also be due to the fact that behavioral observations of prey items are limited. The ingestion of tiny fleas responsible for cestode transmission is probably not observable. Fleas could be ingested while grooming for instance (Wade and Georgi 1988).

Even without direct human contact, human activities that disturb natural habitats create a mosaic of environments that can lead to variation in primate parasite infections. In addition to affecting the biology of hosts, anthropogenic factors can also influence the development and survival of parasites (Altizer *et al*. 2001). The influence of habitats is of paramount importance, considering the fact that almost all intestinal parasites release their propagules into the environment (Eckert *et al*. 2000; Bush *et al*. 2001). Especially indirectly transmitted parasites depend heavily on favorable conditions for intermediate hosts to complete their life cycles (Lile 1998; Arneberg 2002). For instance, cutting of climax forest may increase mosquito habitat and increase opportunities for malarial transmission.

Primate populations suffering from severe habitat disturbance may be restricted to a small area with greater opportunities for infectious transmission of other parasites as well (Stuart and Strier 1995). Vitazkova and Wade (2007) also presumed a connection with environmental factors when they found the strongest association of *Controrchis biliophilus* infection with the troop membership in *Alouatta pigra*, the Guatemalan black-howler monkey, because *C. biliophilus* has an indirect lifecycle which excludes direct infection by group members.

Hahn *et al*. (2003) found that crop-raiding baboons had a higher prevalence of *Streptopharagus* sp. and *Physaloptera* spp. As both *Streptopharagus* and *Physaloptera* spp. are spirurid nematodes with indirect life cycles and arthropod intermediate hosts, the authors suggested that in the environments used by the different troops the arthropod community was also different (Weyher *et al*. 2006).

Such a difference in arthropod community would explain both differences in prevalence of infection by *P. elegans* and cestode B. Habitat disturbance may decrease the diversity of parasites that are dependent on intermediate hosts living in tropical forests while the relative presence of parasites may increase or remain the same (Anderson and May 1982). Disruption of complex ecological relationships between primates and parasites, possibly through the elimination of intermediate hosts, may lead to a lower prevalence of infection than occurs in undisturbed populations (Stuart and Strier 1995). Unfortunately, the role of arthropod ingestion in parasite transmission has not yet been examined yet in studies dealing with habitat disturbances. Nevertheless, the findings support the hypothesis of a change in the composition of possible intermediate hosts in human-modified habitats and therefore in the diet of the different tamarin groups.

The differences in parasite composition between the groups might also be associated with aspects of multi-host systems, for example via the introduction of paratenic hosts. Paratenic hosts harbor infective stages, but the parasites undergo neither development nor reproduction in these hosts. They are not obligatory for the completion of the life cycle but may close ecological or trophic gaps (Bush *et al*. 2001). Although the tamarins do not ingest the known intermediate hosts for *P. elegans* the infection can be successful if a paratenic host is available in the area. However, in many host-parasite systems little is known about which species might serve as paratenic hosts. Furthermore, in the case of the mollusc-transmitted *Angiostrongylus costaricenis* the infection can also be elicited by ingestion of mucus contaminated plants. The primates need not to eat the snails (Sly *et al*. 1982; Brack 1987). Nevertheless, given the scarcity of knowledge on potential intermediate and paratenic host species and possibly our incomplete knowledge of the tamarin's animal prey, conclusions as to the influence of host diet can only remain speculative.

Differences in prevalence of other parasite taxa

Interestingly, of the parasite taxa that are transmitted directly, nematode larvae and "strongylids" affect almost all members of both host species (85-100%) with the exception of nematode larvae in the group around the village. This is surprising remembering the assumption that ground contact would enhance the transmission of directly transmitted parasites that accumulate in the soil and litter (Nunn *et al*. 2000). The low variation in the soil-transmitted parasites within the studied tamarin hosts leads one to assume that these parasites colonize their hosts very efficiently.

It is possible that short periods on the ground might be sufficient to infect hosts because the infective larvae are highly motile and exhibit specific behaviors to locate appropriate hosts (Hawdon and Hotez 1996). Considering the missing transmission of human soil-transmitted parasites to tamarins it is more likely that the so called soil-transmitted parasites might be less dependent on the actual soil for their development into infective larvae: the microbes on which they feed might exist not only in the soil but also in detritus material of other forest strata that the primates have more frequent contact with (Hawdon and Hortez 1996). All other parasite taxa are indirectly transmitted. In these cases the stratification of the intermediate hosts is of greater importance than the actual contact of hosts with the ground.

4.4.2.6 Variation in egg/larvae output

Living close to humans and human modified habitat also leads to differences in egg/larvae output in the studied tamarin groups, both in abundance and intensity.

Variation in Abundance

Abundance varied in three parasite species: *Prosthenorchis*, nematode larva and cestode B. Groups living in contact with humans showed a higher abundance of *P. elegans* infection and lower abundance in cestode B infections. In addition, nematode larvae were less abundant in the tamarin groups around the village than in the sylvatic groups. The result is not surprising

remembering the differences in parasite prevalence. But it highlights the differences between the human associated and sylvatic study groups.

Variation in Intensity

The number of *Prosthenorchis* eggs in the feces of infected tamarins was considerably higher in the groups with contact to human facilities (groups D and S) than in the forest groups (groups F1 and F2), suggesting that the intensities of infestation by adult *P. elegans* were higher in the groups with human contact compared with the sylvatic ones. However, the statistics for intensity of infection are weak, since only two individuals in the sylvatic groups were infected with *P. elegans.* Nevertheless, this result supports the prediction, that human altered habitat offers conducive conditions for parasite encounter.

Under natural epidemiological conditions, primate populations can be infested with parasites without harmful effects. Intensity of infection is an important factor influencing the pathology of a parasite (Ancrenaz *et al*. 2003). Intensity of a parasitic infection is relevant to the presence and severity of the parasite/parasitic disease. If the level of parasite adaptation to its host is very high, their presence usually produces little or no injury (Taraschewski 2000). If the adaptation is less complete, it can lead to more serious disturbance of the host and occasionally result in the death of both host and parasite (Tenter 2006). Similar to bacteria, not all parasites have the same virulence (Tenter 2006). Some parasitic exposures do not necessarily manifest as disease. In intestinal worms, for instance, many patients do not exhibit symptoms and some do not even know that they have been infected until the worm load becomes very high (WHO 1998b). Mostly light infections with parasites are asymptomatic, but severe infections can lead to morbidity and mortality (Flynn 1973; Eckert *et al*. 2005). Therefore, the result of my study is alarming.

Egg/larvae output was used as an indirect measure of abundance and intensity of helminth infection. However, these data come with a caveat: estimates of intensity are likely to be biased by several external and internal factors influencing the number of eggs in a fecal sample (Guyatt and Bundy 1993; Stuart and Strier 1995). Host immunity, density-dependent factors and environmental cues can depress worm ovulation (Christensen *et al*. 1995; Stear *et al*. 1995; Roepstorff *et al*. 1996). A higher parasite burden may result in lower fecundity of the individual female (Anderson and May 1991). This effect is more likely in heavily infected

individuals, but in some parasite species a density-dependent effect has also been observed despite a low intensity of infection (Anderson and Schad 1985; Anderson and May 1991).

However, not all parasites show evidence of density-dependent fecundity (Gregory *et al*. 1990). Daily variation in egg output will also distort estimates of intensity of infection. Numbers of eggs excreted on a daily basis differ due to variations in egg production, differences in stool consistency and clumping of eggs (heterogeneous mixing of eggs in stools) as shown by studies in *Schistosoma mansoni* and *S. japonicum* (cf. Engels *et al*. 1996, 1997*a,b*; Ross *et al*. 1998). In addition, some parasite species release their eggs or larvae intermittently, and prepatent adults, larvae and adult males do not excrete propagules at all (Anderson and Schad 1985; Warnick 1992; Cabaret *et al*. 1998).

The egg count can also be influenced by the nutritional status of an individual (Thienpont et al. 1979). Furthermore, the host immune response will lead to a depression in egg output with host age. But not only host but also parasite age can have an impact on the number of eggs released. Egg production may decrease as the worm grows older (Thienpont *et al*. 1979; Guyatt and Bundy 1993).

4.4.2.7 Summary

In the present study, significant differences could be shown between wild tamarin groups living in human influenced areas and groups living in undisturbed forest patches. The divergences observed in parasite community structure in the tamarins are not related to a direct transfer of parasites between humans and primates. However, the tamarins foraging in an area of human altered habitat showed substantial changes in their parasite communities with potentially pathogenic consequences. In fact, human alteration of the habitat seems to have an influence on arthropod composition and the foraging strategies of the tamarins, which lead to the observed changes in parasite community structure. In the special case of the hosts examined in my study, the key factor seems to be the intermediate hosts. The results of egg/larvae output variation between the study groups completes the picture given by the other measures, showing significant differences between human contact groups and sylvatic groups. To date, however, there is no information available on the potential pathogenic effect of these parasites on their host species in the wild.

Chapter 5

Thailand

5.1 Introduction

While the first part of this study was carried out on two arboreal New World monkey species, the hypothesis should also be tested on an Old World primate species. Although the parasitological status of many Old World primates is well studied (for examples see chapter 2 and chapter 4.1), the gastrointestinal parasites of other wild Old World primates, especially of Asian origin, remain poorly known. Old world monkeys are phylogenetically more closely related to humans than New World monkey species (Kageyama 2000; Neusser *et al*. 2001) and therefore probably more susceptible to human infections. It is no coincidence that Old World monkeys, especially macaque species like *Macaca mulatta* and *M. fascicularis*, belong to the most common animals studied in biomedical research, where they are critically needed for vaccine testing (Gardner and Luciw 2008). Experimental studies have shown them to be susceptible to a wide variety of human diseases including bacteria, viruses, fungi, parasites and prions (reviewed in Gardner and Luciw 2008).

Gastrointestinal parasite infections are less investigated, especially with the background of environmental and human influence on parasite burdens in wild populations. In chapter 2 a review on suspected parasite transmission from humans to great apes and other Old World primates with partly fatal consequences has been done. From Asian primates only two studies are known dealing with this problem in Orang Utans. Mul and colleagues (2007) compared the intestinal parasites of captice, semi-captive and free-ranging Sumatran Orang Utans. They could only detect *Ascaris* sp. and *Giardia* sp. in captive individuals, and prevalence of *Strongyloides* sp. infection was significantly higher in captive Orang Utans. Transmission from humans was suspected for all three parasite species, although they could not be determined to species level (Mul *et al*. 2007). Young Orang Utans kept in close proximity to humans are commonly infected with human malaria, transmitted by mosquitoes, whereas wild Orang Utans are only infected with *Plasmodium pitheci* and *P. silvaticum*, neither of which infects humans (Kilbourne *et al*. 1998).

To deepen the findings of the study described in chapter 4 and to obtain more information on the influence of humans on the gastrointestinal parasite burden and a possible cross-transmission in general, the parasite prevalence of wild macaque troops with different intensities of human contact were investigated. This chapter will present the details of a study on long-tailed macaques (*Macaca fascicularis*) to determine the influence of humans on the

parasite burden of partly ground-living primates in Thailand. This species was chosen as model organism, because of its suitability as biomedical study object and the fact that they often live in high risk interfaces like temples, monkey forests and tourist attraction sites in Asia.

It is estimated that the number of humans who come in contact with nonhuman primates at monkey temples around the world is probably seven million per year (Jones-Engel *et al*. 2006). Extensive, unregulated and often close contact between humans and monkeys occurr at these sites, and there is great body of evidence documenting extensive human-monkey interactions at monkey temples (Fuentes *et al*. 2005; Engel *et al*. 2006; Jones-Engel *et al*. 2006). Roving bands of monkeys quickly snatch up any offerings of food made by devoters and tourists, in addition the monkeys climb on the heads and shoulders of vistors. People who live and work in and around temples or forest parks share common water sources with the monkey inhabitants and report that monkeys frequently invade their homes and gardens in search of food (Jones-Engel *et al*. 2006). It was therefore decided that long-tailed macaques are an excellent model to test the hypotheses.

5.2 Materials and Methods

5.2.1 Study animals

The long-tailed or crab-eating macaque (*Macaca fascicularis*, Figure 5.1) is on out of 22 species of the genus *Macaca* included in the family of Cercopithecidae (Fleagle 1998). Macaques are the only genus of the Cercopithecidae which can be naturally found outside Africa (Fleagle 1998). *M. fascicularis* is one of the world's most numerous and widespread nonhuman primates (Wheatley 1999), second only to *M. mulatta*. Synonymous names of the long-tailed macaque are *M. cynomolgus* and *M. irus*.

Within the species of *M. fascicularis*, the fur colour varies from light brown, yellow or greyish to brown, covering backs, legs and arms. The undersides are much lighter (Rowe 1996; Groves 2001). Infants have a natal coat and are born black. The colour is changing to the adult pelage when they mature (Rowe 1996).

Their defining characteristic for which they are named, is their extraordinarily long tail that is almost always longer than their height from head to rump. The tail length ranges from 40 to

65cm (Fa 1989; Groves 2001). Males have moustaches and cheek whiskers, while females have beards as well as cheek whiskers. Like other macaques, they also exhibit sexual dimorphism in size (Dittus 2004). The body length of males, not including the tail, is 41 to 65cm and their average weight is 4.7 to 8.8kg. The average weight for females is 2.5 to 5.7kg with a height of 38 to 50cm (Fa 1989). Males also possess much larger canine teeth than females (Dittus 2004).

M. fascicularis is a diurnal species, periodically active from dawn to dusk. Long-tailed macaques are primarily arboreal, moving quadrupedically through the canopy, but they also come to the ground regularly (Rodman 1991).

Like most of the primate species, macaques live in social groups. Depending on the habitat and the availability of resources, the groups typically consist of 20 to 50 individuals with adults of both genders and their infants (Bercovitch and Huffman 1999), but there are also reports about troops from six to 100 individuals (Nowak 1995). The macaque troop size is likely a function of the availability of food and pressure from predators, as well as susceptibility to disease (Bercovitch and Huffman 1999). The troops are generally larger in disturbed areas due to greater abundance of food (Sussman and Tattersall 1986).

The troops have a dominant male (alpha male) and several dominant females. Female long-tailed macaques remain in their natal groups and exhibit strong dominance hierarchies in which rank is passed on from mother to daughter (de Jong *et al*. 1994; van Noordwijk and van Schaik 1999). Males also exhibit strong dominance hierarchies. Aggressive interactions between males result in serious injuries, especially lacerations. Males are frequently driven out of their natal troops before sexual maturity, usually between four and six years of age (de Jong *et al*. 1994). Females become sexually productive at about four years (Jones 1982) and they usually give birth to singletons. The life span of macaques is about 30 years, in captivity up to 37 years (Jones 1982).

Long-tailed macaques are omnivorous, but mainly vegetarian. They consume preferentially fruits and plants (approximately 60-90%; Yaeger 1996; Wich *et al*. 2002), but during times of the year when fruits are unavailable they focus on other food sources including insects and other invertebrates, stems, leaves, flowers, seeds, grass, mushrooms, bird eggs, clay and bark (Yaeger 1996; Bercovitch and Huffman 1999; Son 2003). Where they forage near Mangroves, long-tailed macaques also consume crabs – therefore they are also called crab-eating macaques - and have been observed eating frogs, shrimp, octopus, shellfish and other littoral

animals (Sussman and Tattersall 1986; Son 2003). Predators include pythons, monitor lizards, raptors, large cats and in some areas feral dogs (van Noordwijk and van Schaik 1999).

Long-tailed macaques inhabit tropical Southeast Asia, from Burma to the Philippines and southward through Indochina, Malaysia, and Indonesia. They are found as far east as the Timor Islands (Fittinghof and Lindburg 1980; Groves 2001). Their habitats are various, they are found in primary, disturbed and secondary forests, in coastal, mangrove, swamp and riverine forests, from sea level up to 2000m (Rowe 1996). They preferentially utilize secondary forest, especially if it borders human settlements, where they have access to gardens and farms to crop-raid (Crockett and Wilson 1980; Sussmann and Tattersall 1986).

In Thailand, the distribution pattern of long-tailed macaques at present seems to be similar to that determined 30 years ago, from the lower northern region (ca. 16 °N) to the southernmost part (ca. 6°N) (Malaivijitnond *et al*. 2005); however, because of the invasion and disturbance by humans to their natural habitats, their habitats have been greatly changed from natural forests to be temples or parks close to human settlements. In addition to population explosion of the macaques, foraging behavior for natural foods has also changed to raiding gardens, begging humans and searching garbage for foods (Lucas and Corlett 1991). They have also been known to enter houses and steal food if humans are not there to frighten them (Gurmaya *et al*. 1994). Therefore, they have been identified as pest or "weed-species" that depend on and compete with humans, resulting in regular contact with humans and domesticated animals in the urban matrix (Richard *et al*. 1989; Cowlishaw and Dunbar 2000).

M. fascicularis is of least concern on the IUCN Red List of threatened species (last assessed in 2008). Populations, however, have been noted to be declining (IUCN 2010).

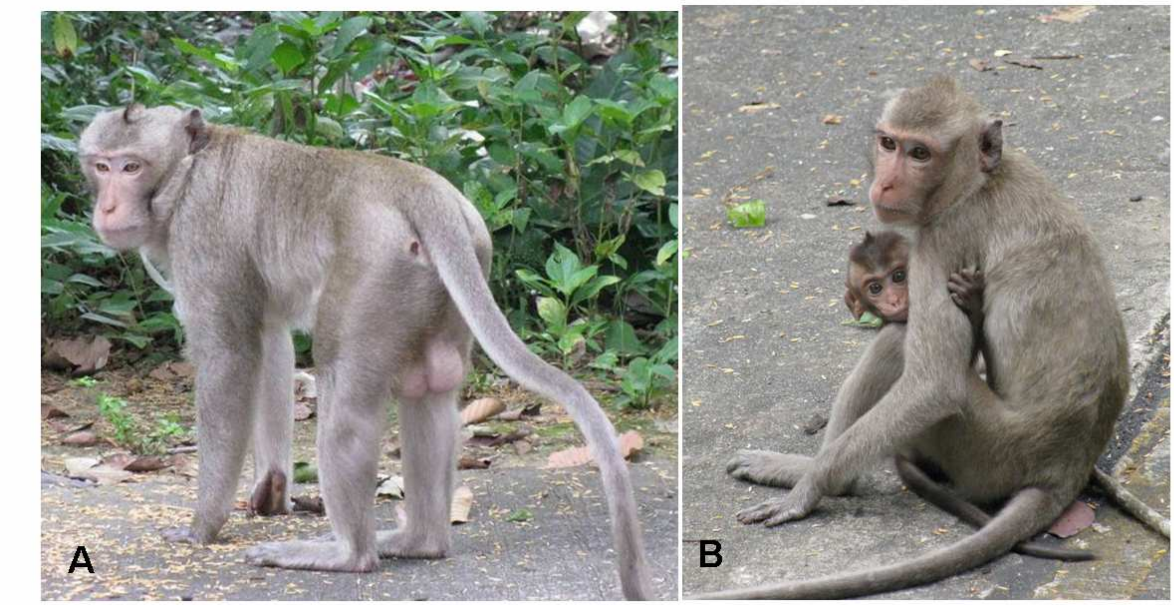

Figure 5.1 Study species *Macaca fascicularis*. A: male individual, B: female individual with infant.

5.2.2 Study area

The study was conducted in the northeastern part of Thailand. One part of the research was carried out in the Kosumpee Forest Park, Mahasarakham Province (Figure 5.2). This park comprises an area of approximately 0.2km^2 and is located next to the Chi River inmidden the city of Kosum Phi Sai. Kosum Phi Sai is a city of about 11000 people. The main office of the park lies at 16°15'N and 103°04'E as determined by GPS. According to the staff of the park, the Kosumpee macaque population has been isolated from other conspecific populations by extensive agricultural areas and human settlements since at least 1966, when the park was established by the Royal Forest Department. The park is a popular rest and recreation site in the province and visited by about 100000 tourists per year. Local pilgrims and tourists along with local residents use areas near water as open toilets, for having picnicks and for disposal of food refuse. Directly in front of the park there are a school, a temple and villagers houses. The park is home of about 400 to 500 grey and golden long-tailed macaques. However, this number may be radically undererstimated.

The second field site was located in the Don Chao Poo Forest Park in Pha Na, Amnat Charoen Province, 60km away from Ubon Rachathani (Figure 5.2). Pha Na is a village of about 2000

people. The entrance of the park lies at 15°67'N and 104°86'E as determined by GPS. This park comprises an area of approximately 1km^2 of mixed deciduous forest. It is located outside the village institutions and houses with the exception of two or three small stalls selling bananas, peanuts, green beans and some other foods on the roadside opposite the main gate. The park exists since more than 50 years. Although it is a designated animal sanctuary it has seen some encroachment. It is visited mostly by people from surrounding areas and it is also used for several municipal and religious occasions. Villagers come into the park to visit a sacred shrine and four Buddha images inside the park. At the end of December, the park is taken over for ten days by hundreds of monks for meditation and Buddhist teachings. Local farmers also graze their cattle inside the park. However, there are also some nearly unaffected parts of forests inside and next to the park. Parts of the wildlife live exclusively inside the forest which is nearly free from human use. They have year round access to clean water and natural food resources. The park is home of about 300 to 400 long-tailed macaques, with some break-away colonies around the park. However, the population seems to be increasing.

Both study areas represent semi-natural settings with a high amount of human-wildlife interaction.

5.2.3 Study groups

The subjects of the study were members of seven wild groups of long-tailed macaques (*Macaca fascicularis*), three groups in the Kosumpee Forest Park and four groups in the Don Chao Poo Forest Park. All sampled group members are recognized individually by individual markings (e.g. small injuries, scars, size or fur color). The groups investigated differed in their contact and proximity to humans.

In Kosumpee Forest Park, all of the investigated groups had very intensive contact to humans and consumed daily large amounts of human provided food. The monkeys climb on the heads and shoulders of visitors. They are reported to enter cars and scooters to enter the village in search of food. Bites and scratches have been reported from encounters between park monkeys and their human visitors. In addition they have access to the school and the houses in front of the park. People who live and work there share common water sources with the monkey inhabitants and report that the monkeys frequently invade their homes and gardens in

search of food and that they are fighting with dogs. All groups studied at Kosumpee Forest Park had contact to humans and will be referred to as K1, K2 and K3. The home ranges of the groups are illustrated in Figure 5.3.

In the Don Chao Poo Forest Park in Pha Na four groups were investigated. Two of these groups had regularly close contact to humans and consumed human provided food every day. The contact, however, was less intense than in Kosumpee Forest Park. The two human contact groups in Pha Na are the designated groups P1 and P2. The two other sampled groups were sylvatic groups referred to as P3 and P4. These sylvatic groups had practically no contact to humans and were feeding on natural food sources mainly. The home ranges of the Pha Na groups are illustrated in Figure 5.4.

Table 5.1 Study group composition of macaques. ♀ = female individuals, ♂= male individuals. Age classes are defined by Groves and Harris (2003).

Group	Human Contact	Group size	Sampled Individuals	Adults		Subadults		Juveniles	
				♀	♂	♀	♂	♀	♂
K1	yes	51	20	6	2	4	4	2	2
K2	yes	49	20	6	2	2	6	1	3
K3	yes	52	20	7	2	5	3	2	1
P1	yes	46	21	8	2	3	4	2	2
P2	yes	42	20	7	2	4	3	3	1
P3	no	33	16	7	2	3	1	1	2
P4	no	34	18	6	2	5	2	2	1

Table 5.2 Composition of gender and age classes of sampled humans from Thailand. ♀ = female individuals, ♂= male individuals.

Study area	Villagers	Adults		Juveniles		Children	
	♀♂	♀	♂	♀	♂	♀	♂
Kosum Phi Sai	105	17	22	23	21	12	10
Pha Na	65	17	16	12	8	4	8

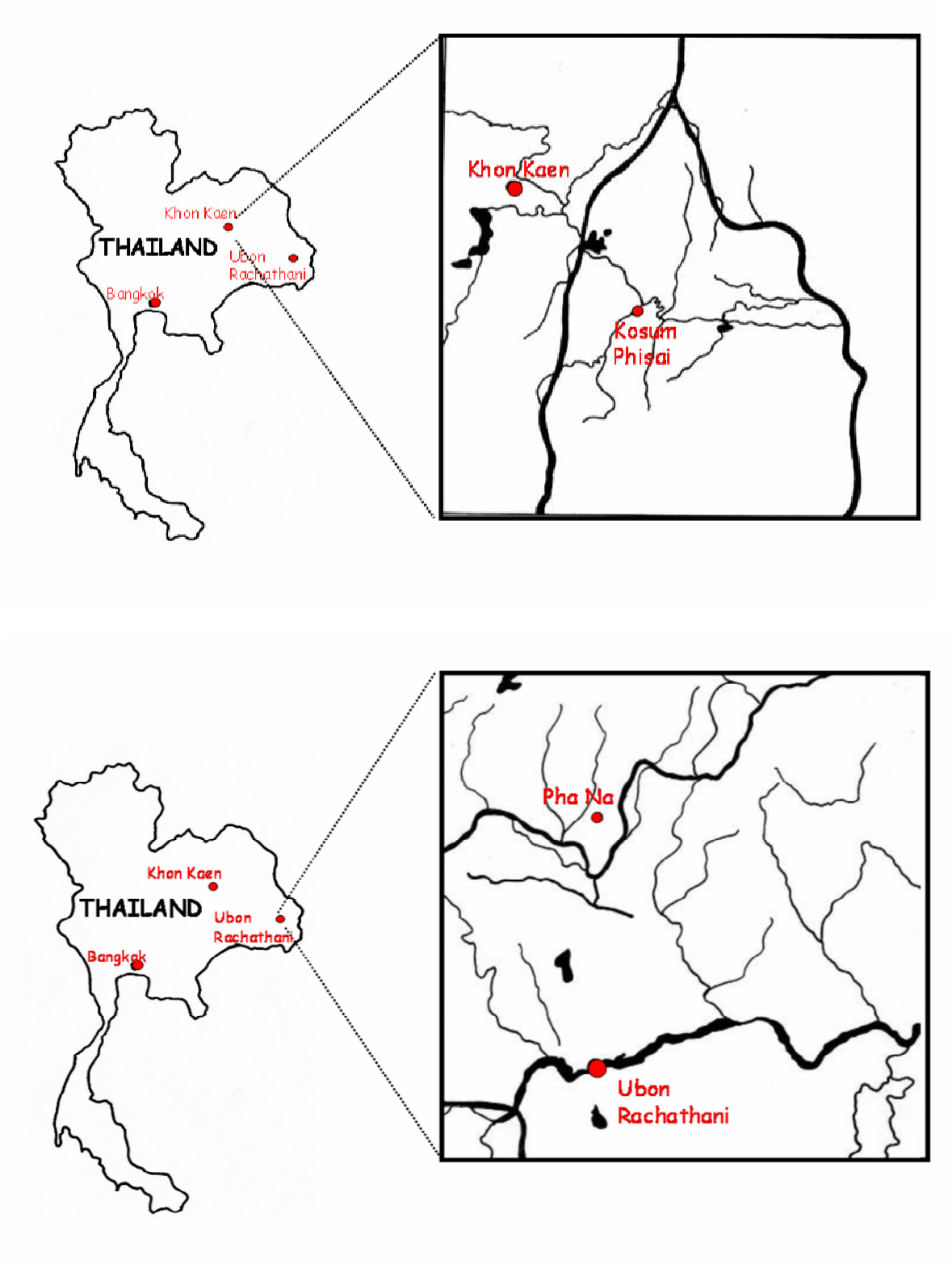

Figure 5.2 Study areas in northeastern Thailand. Kosum Phi Sai including the Kosumpee forest park and Pha Na including the Don Chao Poo forest park.

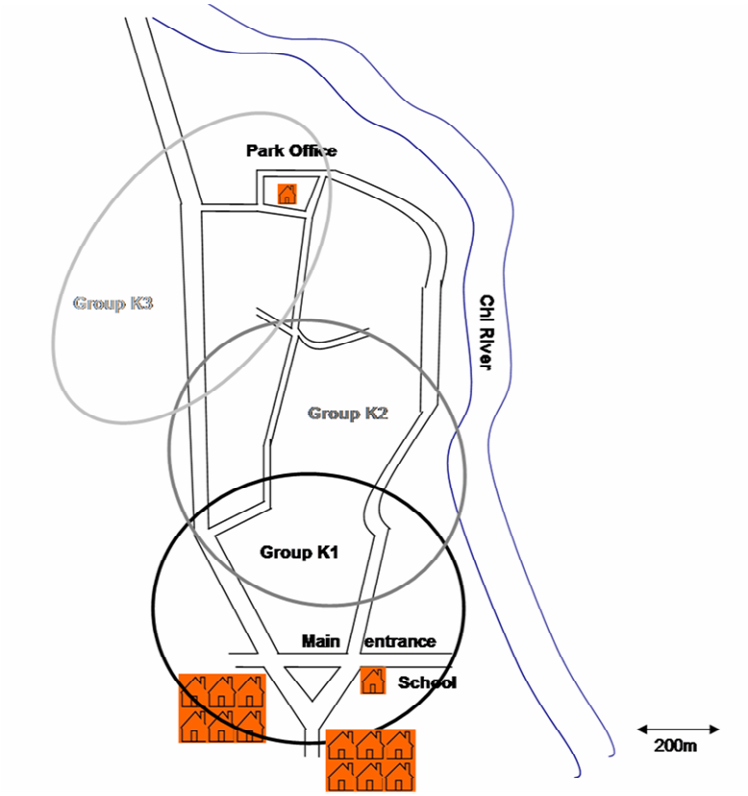

Figure 5.3 Home ranges of the study groups in Kosumpee Forest Park. Grey circles indicate the home ranges of the study goups. All study groups (K1, K2 and K3) had contact to humans

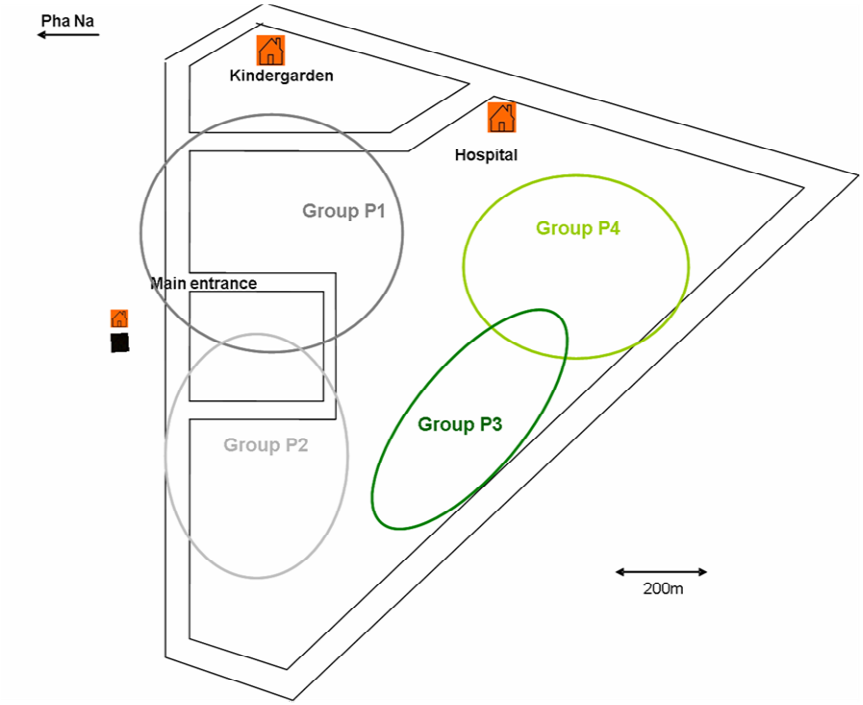

Figure 5.4 Home ranges of the study groups in Don Chao Poo Forest Park. Grey circles represent the home ranges of the stufdy groups with contact to humans (P1 and P2), green circles represent the home ranges of the sylvatic study groups (P3 and P4).

5.2.4 Study period and sample collection

The study was carried out from July to December 2008. Behavioral data and fecal sample collection took place in this period. Fecal sample collection and behavioral observations were carried out during the whole activity period of the primates: from the time they left their sleeping trees (around 5:30 to 6am) to the moment they entered the next sleeping tree in the evening (6 to 6:30pm). The study period included the dry season only. With the beginning of the wet season, both study sites were flooded. Fecal sample collection was therefore nearly impossible.

From each of the 135 individuals at least three samples were collected on non-consecutive days. In total, 605 fecal samples from the seven study groups could be obtained. Additionally, fecal samples from 170 people between the ages of one and 87 years in Kosum (105 individuals) and Pha Na (65 individuals) were taken (Table 5.2), at least three samples per individual from non-consecutive days. In total, 510 human stool samples were collected.

Fecal samples were gathered directly after defecation and locality, date, group, species, sex, individual and time of defecation were recorded. After collection, the fecal samples were immediately preserved in 10% buffered formalin (solution of 10% formaldehyde and sodium phosphate buffer, pH 7.0). Samples were stored at ambient temperature. Samples were proceeded after a modified formalin-ethyl acetate sedimentation (Ash *et al*. 1994) and microscopically examined for the presence of eggs and larvae of different intestinal helminths. In addition the egg (larvae) output per 100µl concentrated sediment was counted.

To distinguish between hookworms and different *Strongyloides* species, coprocultures with the agar plate method after Koga *et al*. 1991 were done. For more information on the methods and statistical analyses see chapter 3.

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5.2.5 Behavioral data collection

Each macaque group was followed for seven consecutive days. Observations were carried out by instantaneous scan sampling on 49 days yielding a total of 572 contact hours (Table 5.3) Unfortunately, no unbiased behavioral data could be collected because the study period was too short for habituation, especially in the case of the sylvatic groups. Behavior of dependent infants was not recorded. For details on data collection see chapter 3.

Table 5.3 Contact times for behavioral data sampling.

Group	Overall contact time (in h)
K1	80.3
K2	82.1
K3	81.2
P1	82.0
P2	81.8
P3	82.1
P4	82.4

5.3 Results

5.3.1 Human fecal samples

The human feces of both villages, Kosum Phi Sai and Pha Na contained six different helminth morphospecies: *Opisthorchis viverinii*, *Taenia spp.*, *Strongyloides stercoralis, Trichostrongylus spp.*, MIF (minute intestinal fluke), probably *Haplorchis* sp. and hookworm eggs (*Necator americanus/Ancylostoma duodenale*). An overview on the recovered helminths and their taxonomy can be found in Table 5.4. The descriptive statistics of length and width of each morphospecies are presented in Table 5.5. Light microscopical photographs of all parasite taxa are presented in Figure 5.7.

The prevalence of *Strongyloides*, *Opisthorchis* and hookworms in Kosum Phi Sai was similar and varied between 18 and 34%. Females showed highest prevalence in *Strongyloides* infection, while males showed the highest prevalence in hookworm infection. MIF, *Taenia* and *Trichostrongylus* infections were only moderately represented (Figure 5.5).

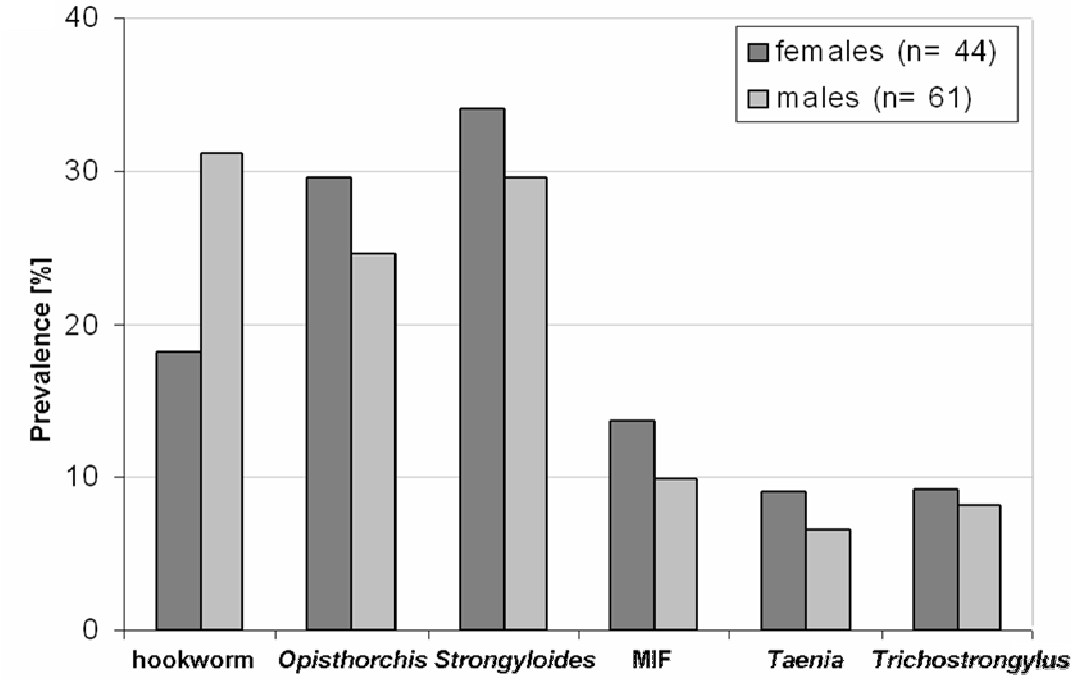

Figure 5.5 Prevalence of infection with different parasite species in people of Kosum Phi Sai. Bars denote proportion of male and female individuals infected with a parasite species. hookworm= *Ancylostoma duodenale/Necator americanus*, *Opisthorchis*= *Opisthorchis viverrini*, *Strongyloides*= *Strongyloides stercoralis*, MIF= minute intestinal fluke, *Taenia*= *Taenia* spp., *Trichostrongylus*= *Trichostrongylus* spp.

The prevalences of *S. stercoralis*, *O. viverrini* and hookworms in Pha Na were slightly higher (26 to 42%), showing maximum prevalences of *S. stercoralis* infection in females and hookworm and *Opisthorchis* infections in males. MIF and *Taenia* infections were moderately represented. *Trichostrongylus* was absent in the samples from Pha Na (Figure 5.6).

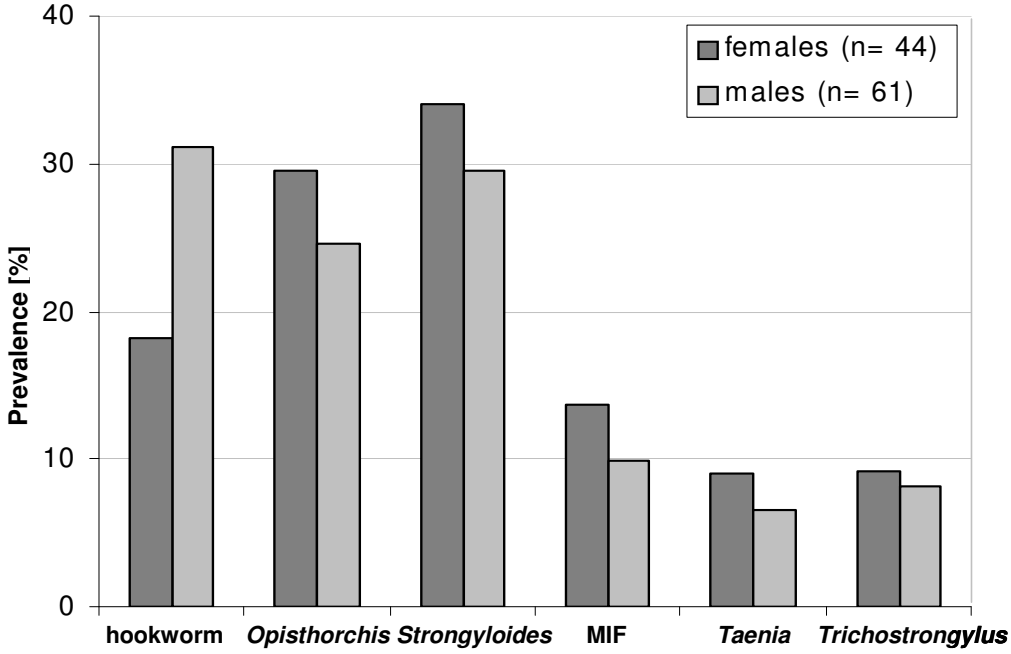

Figure 5.6 Prevalence of infection with different parasite species in people of Pha Na. Bars denote proportion of male and female individuals infected with a parasite species. hookworm= *Ancylostoma duodenale/Necator americanus*, *Opisthorchis*= *Opisthorchis viverrini*, *Strongyloides*= *Strongyloides stercoralis*, MIF= minute intestinal fluke, *Taenia*= *Taenia* spp., *Trichostrongylus*= *Trichostrongylus* spp.

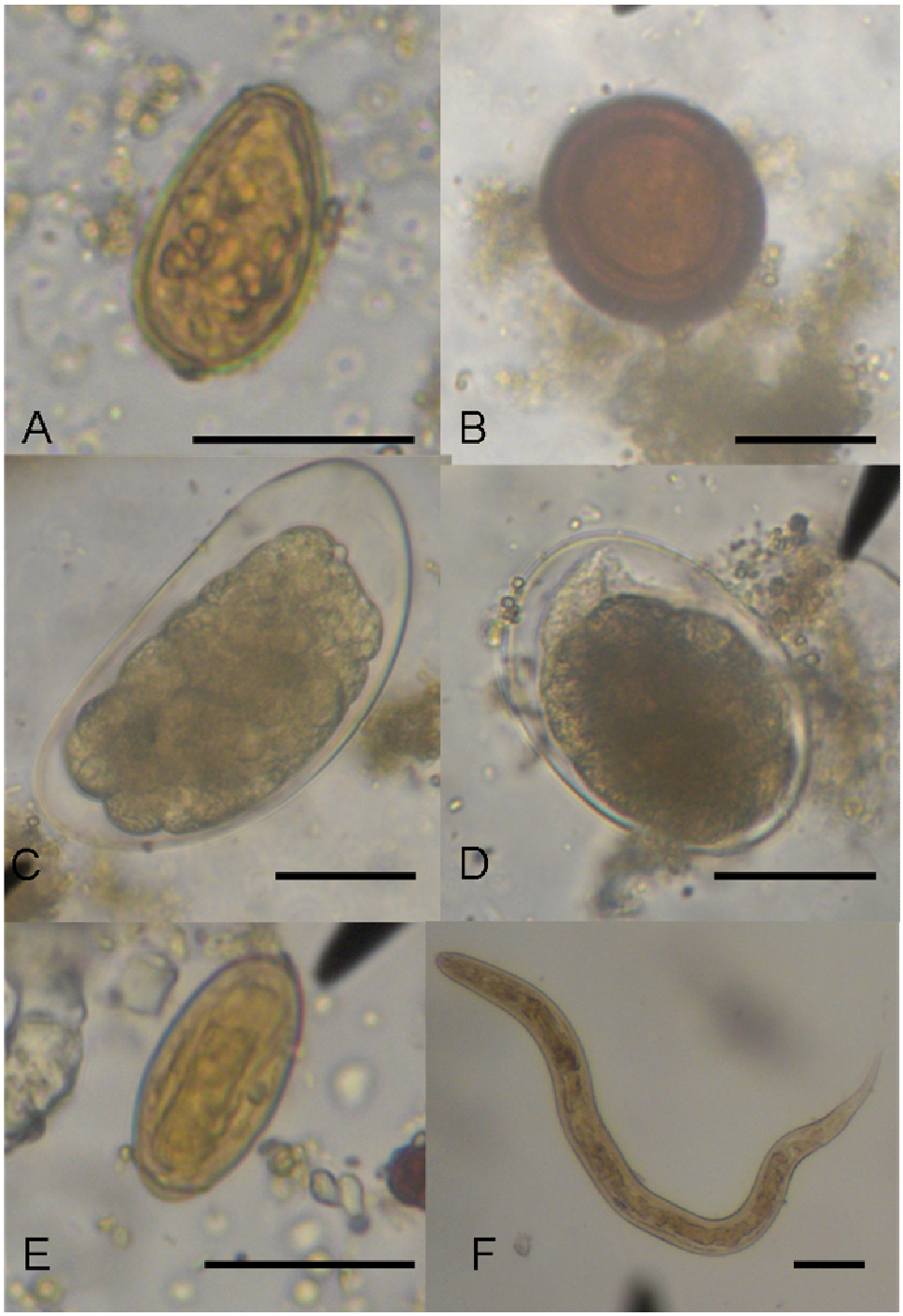

Figure 5.7 (A-F) Light microscope pictures of parasite eggs and nematode larva recovered from stool samples of humans. A: *Opisthorchis viverrini*, B: *Taenia* spp., C: *Trichostrongylus* spp., D: hookworm egg (*Ancylostoma duodenale*/*Necator americanus*), E: minute intestinal fluke (MIF), F: *Strongyloides stercoralis*; scale bar = 25µm.

Table 5.4 Human parasite identification. All species found as eggs except *Strongyloides stercoralis* larvae. MIF= minute intestinal fluke. Letters a) –c) give the probable parasite identification. Taxonomy follows Schnieder and Tenter (2006).

Parasite morphospecies	Parasite identification		
	Phylum/Class	**Order/Familiy**	**Species or genus**
1. hookworm	Nematoda/Chromadorea	Rhabditida/Ancylostomatidae	*a) Necator americanus*
			b) Ancylostoma duodenalis
2. *Opisthorchis viverrini*	Nematoda/Chromadorea	Opisthorchiida/Opisthorchiidae	*Opisthorchis viverrini*
3. MIF	Nematoda/Chromadorea	Opisthorchiida/Heterophyidae	*a) Haplorchis* sp.
			b) Metogonimus yokogawai
			c) Heterophyes sp.
3. *Strongyloides stercoralis*	Nematoda/Chromadorea	Tylenchida/Strongyloididae	*Strongyloides stercoralis*
4. *Taenia* spp.	Plathelmintha/Cestodea	Cyclophyllida/Taeniidae	*Taenia* spp.
5. *Trichostrongylus* spp.	Nematoda/Chromadorea	Rhabditida/Trichostrongylidae	*Trichostrongylus* spp.

Table 5.5 Descriptive statistics of length and width for eggs of five different parasite morphospecies found in humans in Thailand. hookworm= *Ancylostoma duodenale/Necator americanus,* MIF= minute intestinal fluke. N gives number of parasite stages measured. Median, minimum, maximum and IQR (interquartile ranges) are presented for each parasite taxon and host species in µm.

	males		**females**	
hookworm	length	width	length	width
N	8	8	9	9
Median	53.9	23.8	53.9	23.6
Minimum	52.7	22.9	51.9	22.9
Maximum	56.4	24.3	54.6	24.2
IQR	3	0.9	1.6	1.3
Opisthorchis viverrini	length	width	length	width
N	8	8	9	9
Median	29.2	16.5	29.7	16.6
Minimum	28.5	15.7	27.3	14.3
Maximum	31.1	16.9	32.9	16.9
IQR	2.2	1	2.4	1.4
MIF	length	width	length	width
N	8	8	6	6
Median	30.5	14.25	30.5	13.75
Minimum	28.25	12.5	28.75	12.75
Maximum	32.5	15.25	32.25	14.5
IQR	2.1	1.1	1.9	1.0
***Trichostrongylus* spp.**	length	width	length	width
N	4	4	4	4
Median	88.6	44	88.1	44
Minimum	87.1	43.2	87.6	43.8
Maximum	89.3	45.4	89.1	44.4
IQR	1.8	1.7	1.2	0.5
***Taenia* spp.**	length	width	length	width
N	7	7	6	6
Median	37.9	37.9	38.4	38.4
Minimum	36.9	36.9	36.8	36.8
Maximum	39.6	39.6	39.1	39.1
IQR	1.9	1.9	0.9	0.9

Intensity of infection was slightly higher in Kosum Phi Sai with the exception of *Strongyloides* infection. In none of the samples a heavy parasite infection could be observed (Table 5.6 and Table 5.7).

Table 5.6 Intensity of infection in stool samples from people from Kosum Phi Sai (refers to the number of parasite stages per 100µl fecal sediment). hookworm= *Ancylostoma duodenale/Necator americanus,* MIF= minute intestinal fluke. N is number of positive stool samples. Mean egg count, maximum egg count and standard deviation (SD) are presented for each parasite taxon.

Parasite taxa	Mean	SD	Maximum	N
hookworm	8.7	8.7	42	62
Opisthorchis viverrini	25.6	39.2	198	68
Strongyloides stercoralis	1.1	0.7	4	58
MIF	9.1	4.5	21	16
Taenia spp.	42.9	28.5	103	20
Trichostrongylus spp.	2.8	1.7	7	20

Table 5.7 Intensity of infection in stool samples from people from Pha Na (refers to the number of parasite stages per 100 µl fecal sediment). hookworm= *Ancylostoma duodenale/Necator americanus,* MIF= minute intestinal fluke. N gives number of positive stool samples. Mean egg count, maximum egg count and standard deviation are presented for each parasite taxon.

Parasite taxa	Mean	SD	Maximum	N
hookworm	8.5	7.7	33	58
Opisthorchis viverrini	7.9	6.8	25	43
Strongyloides stercoralis	3.9	4.1	17	54
MIF	8.9	4.8	16	15
Taenia spp.	25.8	12.7	54	11

5.3.2 Macaque fecal samples

5.3.2.1 Parasite diversity

Six helminthic parasite morphospecies could be recovered - one trematode and five different nematode species. Cestodes and acanthocephalans could not be detected. I found: the trematode morphospecies MIF, probably *Haplorchis* sp., and one of the nematodes could be identified as *Strongyloides fuelleborni*. This was confirmed by coproculture (Figure 5.9). Another nematode belonged to the Hymenolepididae, probably being *Oesophagostomum sp.*, the third nematode species was identified as a *Trichuris* species, probably *Trichuris trichiura*. I could also identify the eggs of a nematode from the order Rhabditida, probably *Globocephalus* sp. and referred to as hookworm B. Furthermore, I found nematode larvae belonging either to the order Rhabditida (*Strongyloides* sp.) or to the superfamily Metastrongiloidea (i.e. *Filaroides* sp. and *Angiostrongylus* sp.).

An overview on the recovered helminthes and their taxonomy can be found in Table 5.8. The descriptive statistics of length and width of each morphospecies are presented in Table 5.9. Information on parasite identification (morphological characteristics, description of potential host species and their origin, potential intermediate hosts) including references is given in Appendix A. Light microscopical photographs are presented in Figure 5.8.

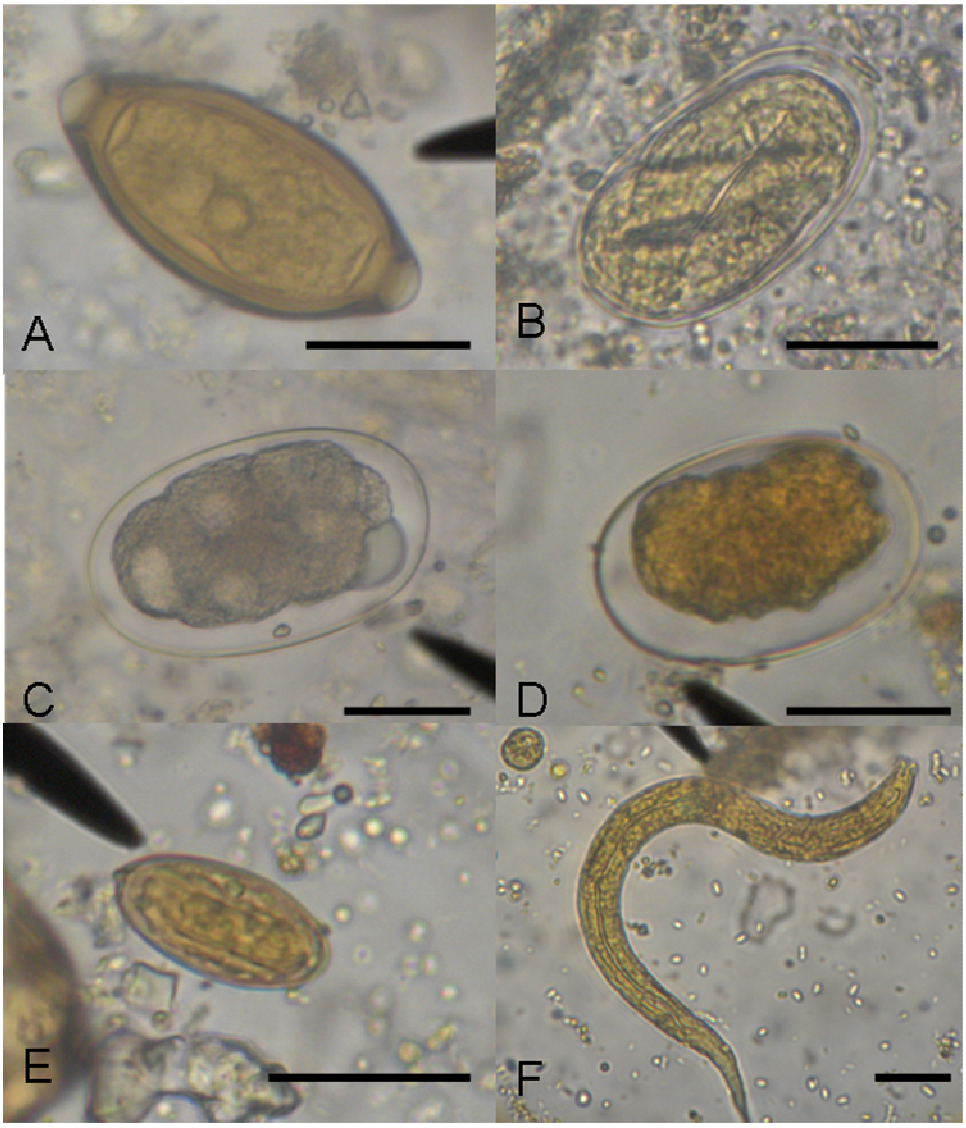

Figure 5.8 (A-F) Light microscope pictures of parasite eggs and nematode larva recovered from fecal samples of macaques. A: *Trichuris* sp., B: *Strongyloides fuelleborni*., C: *Oesophagostomum* sp., D: hookworm B, E: minute intestinal fluke (MIF), F: nematode larva; scale bar = 25µm

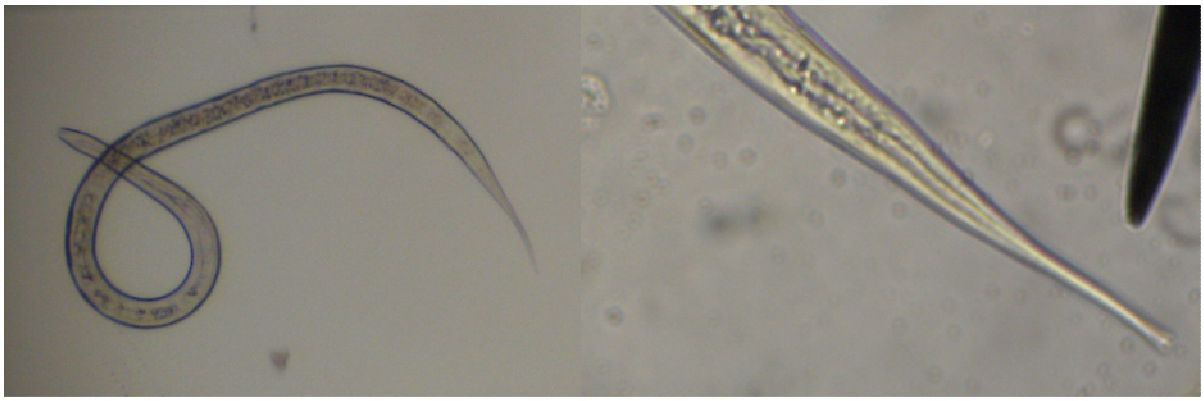

Figure 5.9 Light microscope pictures of L3 larvae from *Strongyloides fuelleborni* obtained from coproculture.

Table 5.8 Macaque parasite identification. All species found as eggs except nematode larva. MIF= minute intestinal fluke. Letters a) –c) give the probable parasite identification. Taxonomy follows Schnieder and Tenter (2006).

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Parasite morphospecies	Parasite identification		
	Phylum/Class	**Order/Familiy**	**Species or genus**
1. *Strongyloides fuelleborni*	Nematoda/Chromadorea	Tylenchida/Strongyloididae	*Strongyloides fuelleborni*
2. *Trichuris* sp.	Nematoda/Enoplea	Trichinellida/Trichuridae	*Trichuris trichiura*
3. hookworm B		Rhabditida/Ancylostomatidae	*Globocephalus* sp.
4. MIF	Nematoda/Chromadorea	Opisthorchiida/Heterophyidae	*a) Haplorchis* sp.
			b) Metogonimus yokogawai
			c) Heterophyes sp.
5. *Oesophagostomum* sp.	Nematoda/Chromadorea	Rhabditida/Strongylidae	*a) Oesophagostomum* sp.
			b) Ternidens sp.
7. nematode larva	Nematoda/Chromadorea	1. Tylenchida/Strongyloididae	a) *Strongyloides* sp.
		2. Rhabditida/Metastrongylidae	b) *Filaroides* sp.
		2. Rhabditida/Metastrongylidae	c) *Angiostrongylus cantonensis*

Table 5.9 Descriptive statistics of length and width for eggs of five different parasite morphospecies found in macaques. Data are presented for macaques from Kosumpee forest park and from Don Chao Poo forest park. MIF= minute intestinal fluke, N gives number of parasite stages measured. Median, minimum, maximum and IQR (interquartile ranges) are presented for each parasite taxon and host species in µm.

	Kosumpee forest park		**Don Chao Poo forest park**	
Strongyloides fuelleborni	length	width	length	width
N	18	18	18	18
Median	50	32	50	32
Minimum	50	30	50	30
Maximum	53	35	53	35
IQR	2.1	3.6	2.1	3.4
Trichuris trichiura	length	width	length	width
N	15	15	9	9
Median	58.75	28.5	60	27.5
Minimum	55	27.5	55	27.5
Maximum	65	32.5	65	32.5
IQR	7.5	1.2	7.5	1.2
hookworm B	length	width	length	width
N	6	6	6	6
Median	50	30	50.5	30.62
Minimum	48.75	28.75	50	30
Maximum	52	32.25	52.25	32.25
IQR	1.2	0.9	1.5	1.9
MIF	length	width	length	width
N	9	9	5	5
Median	30.5	14.5	30.5	13.75
Minimum	28.75	12.5	28.5	13
Maximum	32.5	15.25	32	14,5
IQR	1.9	1.1	2.5	1.0
***Oesophagostomum* sp.**	length	width	length	width
N	/	/	13	13
Median	/	/	72.75	45
Minimum	/	/	65	40
Maximum	/	/	75	47.5
IQR	/	/	4.3	2.7

5.3.2.2 Parasite Species Richness (PSR)

The maximum number of intestinal parasite morphospecies per individual was five in Don Chao Poo forest park (Figure 5.11) and four in Kosumpee forest park (Figure 5.10). Of 135 Individuals 66 had multiple parasite infections with at least two parasite taxa over the study period.

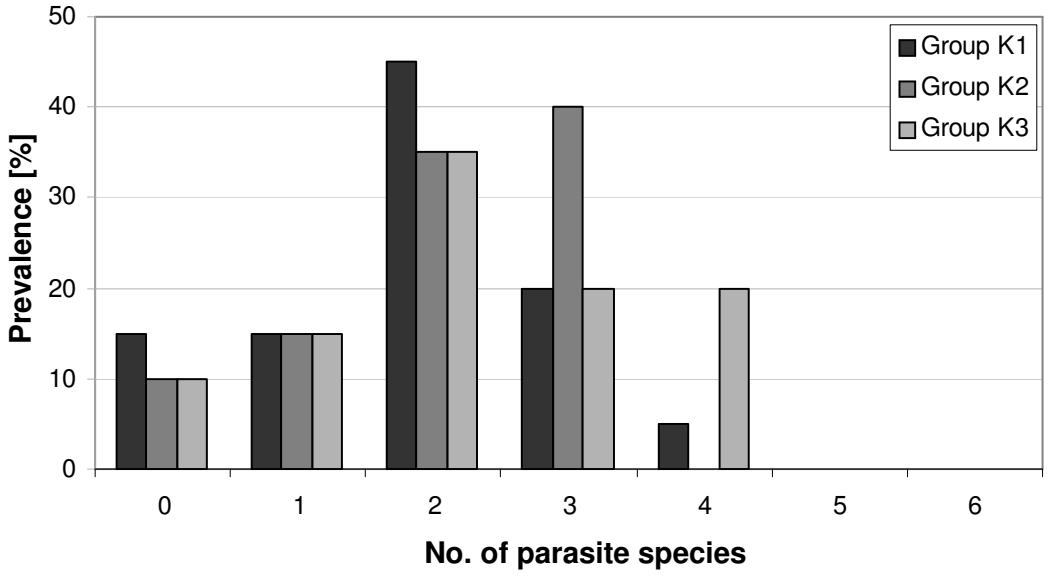

Figure 5.10 Distribution of PSR frequencies in macaques of Kosumpee forest park. Bars denote the proportion of host individuals with a specific PSR.

Total PSR varied significantly over the host groups across the different sample areas (Table 5.10). In Kosumpee forest park PSR was significantly higher than in groups P2, P3 and P4 from Don Chao Poo forest park (Figure 5.12).

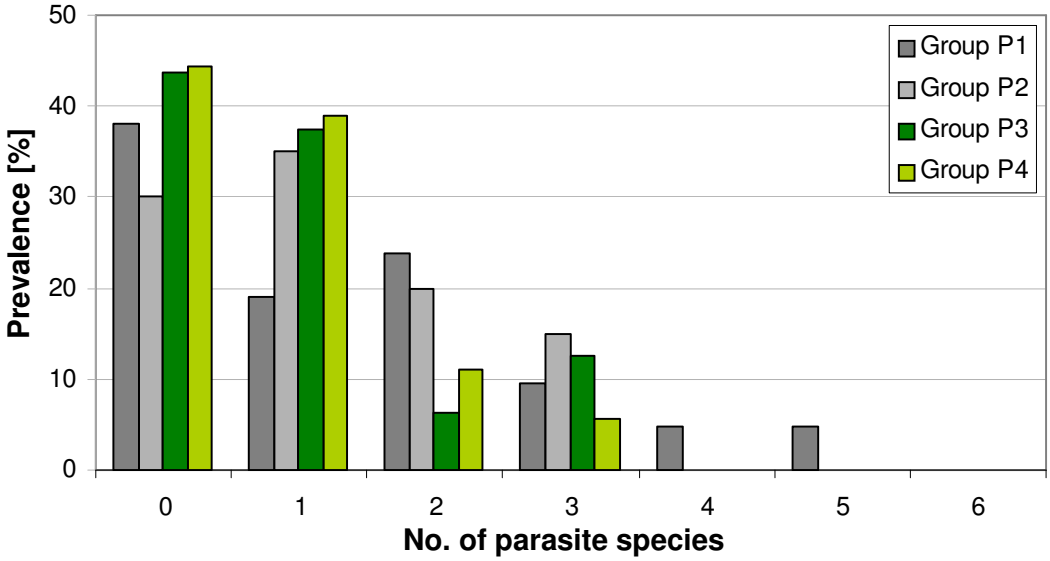

Figure 5.11 Distribution of PSR frequencies in macaques of Don Chao Poo forest park. Grey bars represent human contact groups, green bars represent sylvatic groups. Bars denote the proportion of host individuals with a specific PSR.

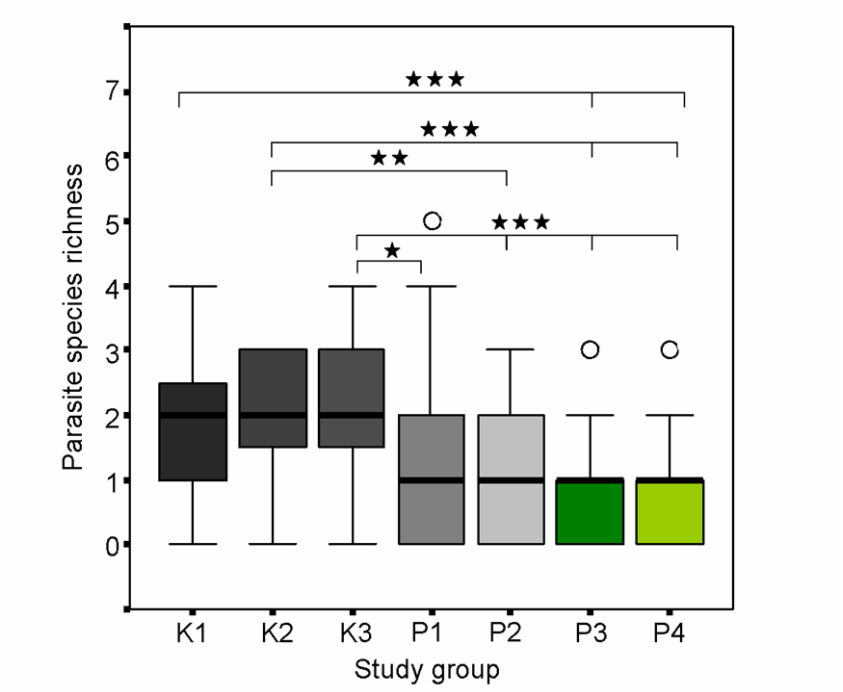

Figure 5.12 Total parasite species richness (PSR) per group in macaques. Groups K1-K3 are study groups from Kosumpee forest park, groups P1-P4 are groups from Don Chao Poo forest park. Grey boxes represent human contact groups, green boxes represent sylvatic groups. Boxes show the interquartile ranges, bold horizontal bars show the median. The ends of the whiskers represent the minimum and maximum values that are not outliers. Circles represent outliers. Asterisks indicate statistical differences between groups (ranked t-test: * $p \leq 0.05$; ** $p \leq 0.01$, *** $p \leq 0.001$).

Table 5.10 Results ranked t-test for differences in parasite species richness (PSR) between the macaque groups. Groups K1-K3 are study groups from Kosumpee forest park, groups P1-P4 are groups from Don Chao Poo forest park. Asterisks indicate statistical differences between the groups (*p≤ 0.05; ** p≤ 0.01, *** p≤ 0.001).

	compared groups					
	K1+K2	**K1+K3**	**K2+K3**			
T	-0.61	-1.08	-0.56			
df	38	38	38			
p	0.549	0.280	0.580			
	K1+P1	**K1+P2**	**K1+P3**	**K1+P4**		
T	1.16	1.92	2.74	3.32		
df	39	38	34	36		
p	0.254	0.063	0.01**	0.002**		
	K2+P1	**K2+P2**	**K2+P3**	**K2+P4**		
T	1.70	2.62	3.47	4.42		
df	39	38	34	38		
p	0.097	0.013*	0.001***	<0.001***		
	K3+P1	**K3+P2**	**K3+P3**	**K3+P4**		
T	2.04	2.87	3.54	4.15		
df	39	38	34	36		
p	0.048*	0.007**	0.001***	<0.001***		
	P1+P2	**P1+P3**	**P1+P4**	**P2+P3**	**P2+P4**	**P3+P4**
T	0.45	1.18	1.53	0.93	1.30	0.30
df	39	35	37	34	36	32
p	0.654	0.247	0.136	0.359	0.192	0.768

5.3.2.3 Parasite prevalence

Trichuris sp., hookworm B and the nematode larva were present in all of the investigated groups. *Oesophagostomum* sp. could only be detected in the groups from Don Chao Poo forest park. MIF was absent in the sylvatic groups from Don Chao Poo, P3 and P4. Additionally *Strongyloides fuelleborni* was not detectable in P4.

There were no significant differences in parasite prevalence between the three groups from Kosumpee forest park (Figure 5.13).

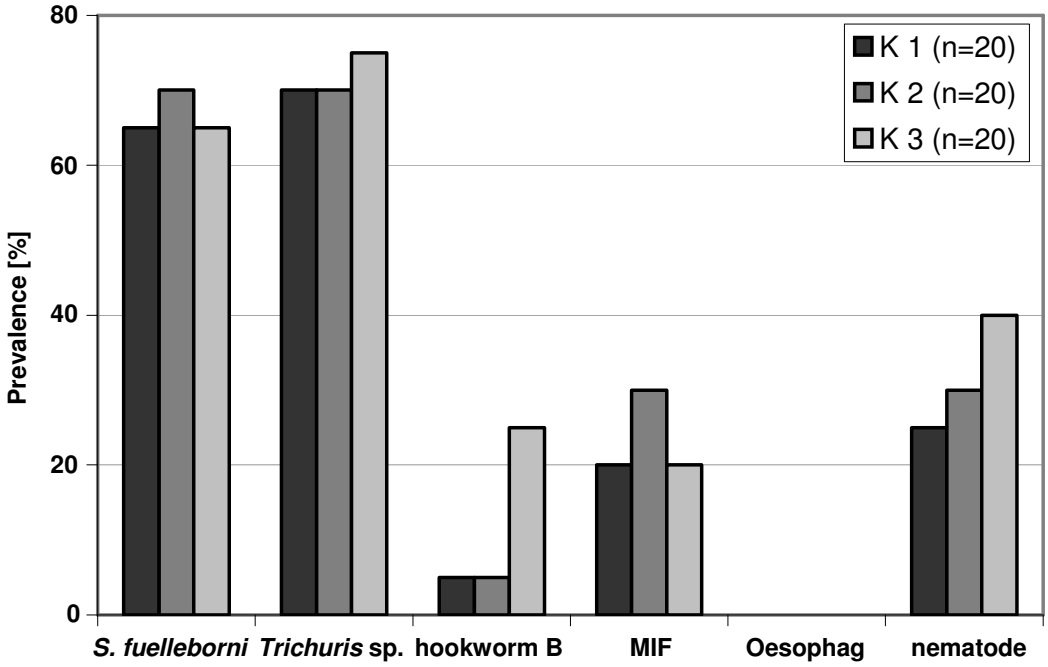

Figure 5.13 Prevalence of all parasite taxa per study group in macaques from Kosumpee forest park (*S. fuelleborni*=*Strongyloides fuelleborni*, *Trichuris*=*Trichuris* sp., hookworm B=hookworm B, MIF=minute intestinal fluke, Oesophag=*Oesophagostomum* sp., nematode=nematode larva). Bars denote prevalence per parasite species for all groups.

There were significant differences in parasite prevalence between the four groups from Don Chao Poo forest park (Figure 5.14). *S. fuelleborni* was absent in the sylvatic group P4 leading to significant differences with all human contact groups. In the second sylvatic group, P3, only one of the investigated members was infected. The human contact group P1 showed significantly higher infection prevalence than both forest groups P3 and P4 (Fisher's exact test, P1+P3, p=0.05, P1+P4, p=0.004) and the second human contact group P2 was higher infected than P4 (Fisher's exact test, p=0.021).

The minute intestinal fluke (MIF) was completely absent in both sylvatic groups, P3 and P4, whereas in all human contact groups prevalence ranged between 14.3% and 20% (Figure 5.14).

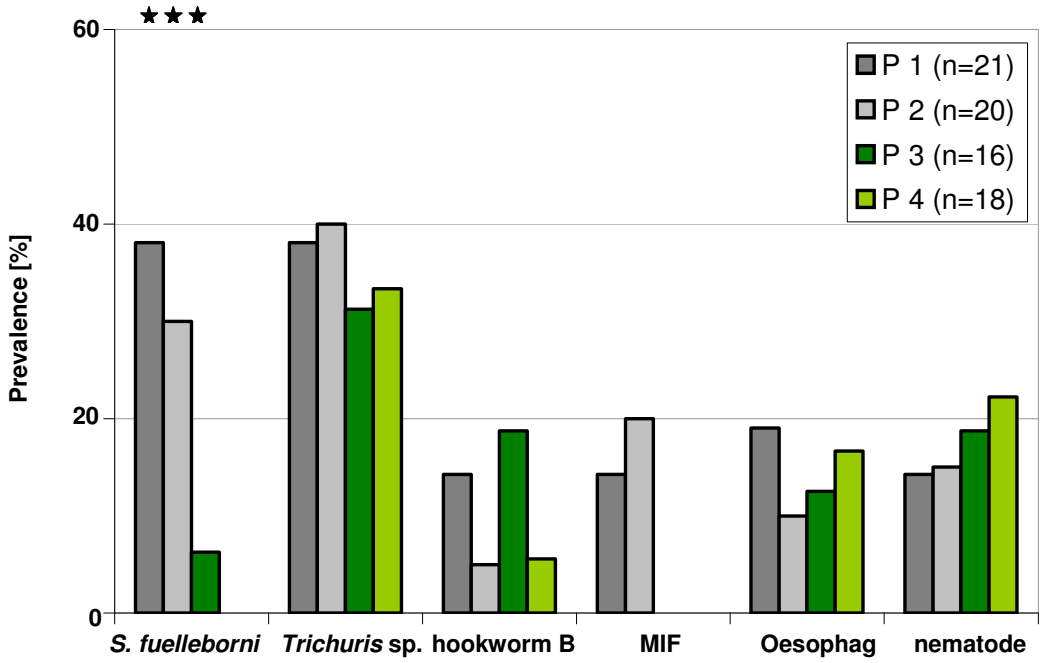

Figure 5.14 Prevalence of all parasite taxa per study group in macaques from Don Chao Poo forest park (*S. fuelleborni*=*Strongyloides fuelleborni*, *Trichuris* sp.=*Trichuris* sp., hookwom B=hookworm B, MIF=minute intestinal fluke, Oesophag=*Oesophagostomum* sp., nematode=nematode larvae). Grey bars represent human contact groups, green bars represent sylvatic groups. Bars denote prevalence per parasite species for all groups. Asterisks indicate statistical differences between groups (Extension of Fisher's exact test: *** $p \leq 0.001$).

In addition, there were significant differences in prevalence of infection between the groups from Kosumpee forest park and Don Chao Poo forest park (Figure 5.15). All groups from Kosumpee forest park had a highly significant higher prevalence of *S. fuelleborni* infection than the forest groups P3 and P4 from Don Chao Poo (Fisher's exact test, $p<0.001$). In addition, the prevalence was significantly higher in group K2 than in group P2 (Fisher's exact test, $p=0.026$) (Table 5.11).

While the MIF was absent in the forest groups P3 and P4, human contact groups showed prevalences of infection between 14.3% and 30% (Figure 5.15).

Trichuris sp. showed significantly lower prevalence in the sylvatic groups P3 and P4 than in all human contact groups from Kosumpee forest park (Fisher's exact test, $p \leq 0.05$; Table 5.11). In addition group P1 from Don Chao Poo forest park showed significantly lower prevalence than K3 (Fisher's exact test, $p=0.028$).

Oesophagostomum sp. could only be detected in the macaque groups from Don Chao Poo and was completely absent in samples from Kosumpee forest park (Figure 5.15).

None of the other parasite taxa showed significant differences in prevalence between the macaque groups (Table 5.11)

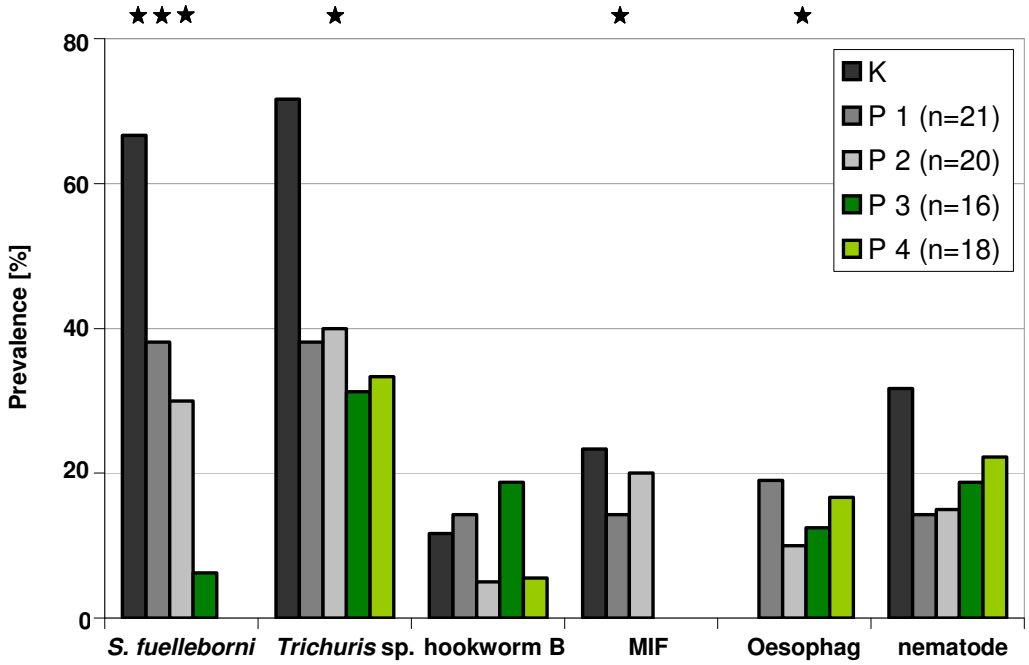

Figure 5.15 Prevalence of all parasite taxa from per study group from macaques from Thailand. (*S. fuelleborni*=*Strongyloides fuelleborni*, *Trichuris* sp.=*Trichuris* sp., hookwom B=hookworm B, MIF=minute intestinal fluke, Oesophag=*Oesophagostomum* sp., nematode=nematode larvae). Grey bars represent human contact groups, green bars represent sylvatic groups. Study groups from Kosumpee forest park (K) are pooled for a better overview, P1-P4 are study groups from Don Chao Poo forest park.. Bars denote prevalence per parasite species for all groups. Asterisks indicate statistical differences between groups (Extension of Fisher's exact test: * $p \leq 0.05$, *** $p \leq 0.001$).

Table 5.11 Results of Fisher's exact test comparing prevalence of parasite infection in macaques of the different study groups. K1-K3 are groups from Kosumpee forest park, P1-P4 are groups from Don Chao Poo forest park. Asterisks indicate statistical differences between the groups (*p≤ 0.05; ** p≤ 0.01, *** p≤ 0.001).

		Compared Groups						
		K1+K2	**K1+K3**	**K1+P1**	**K1+P2**	**K1+P3**	**K1+P4**	**K2+K3**
Parasite taxa	***Strongyloides fuelleborni***	1	1	0.121	0.056	<0.001***	<0.001***	1
	***Trichuris* sp.**	1	1	0.062	0.111	0.042*	0.050*	0.731
	hookworm B	1	0.182	0.606	1	0.303	1	0.182
	minute intestinal fluke (MIF)	0.716	1	0.697	1	0.113	0.107	0.716
	***Oesophagostomum* sp.**	/	/	0.107	0.487	0.190	0.097	/
	nematode larva	1	0.501	0.454	0.695	0.709	1	0.741
		K2+P1	**K2+P2**	**K2+P3**	**K2+P4**	**K3+P1**	**K3+P2**	**K3+P3**
	Strongyloides fuelleborni	0.062	0.026*	<0.001***	<0.001***	0.121	0.056	<0.001***
	***Trichuris* sp.**	0.121	0.205	0.092	0.103	0.028*	0.054	0.017*
	hookworm B	0.606	1	0.303	1	0.454	0.182	0.709
	minute intestinal fluke (MIF)	0.277	0.716	0.024*	0.021*	0.697	1	0.113
	***Oesophagostomum* sp.**	0.107	0.487	0.190	0.097	0.107	0.487	0.190
	nematode larva	0.277	0.451	0.700	0.719	0.085	0.155	0.277
		K3+P4	**P1+P2**	**P1+P3**	**P1+P4**	**P2+P3**	**P2+P4**	**P3+P4**
	Strongyloides fuelleborni	<0.001***	0.744	0.050*	0.004**	0.104	0.021*	0.471
	***Trichuris* sp.**	0.021*	1	0.739	1	0.731	0.745	1
	hookworm B	0.184	0.606	1	0.609	0.303	1	0.323
	minute intestinal fluke (MIF)	0.107	0.697	0.243	0.235	0.113	0.107	/
	***Oesophagostomum* sp.**	0.097	0.663	0.680	1	1	0.653	1
	nematode larva	0.307	1	1	0.682	0.613	0.687	1

5.3.2.4 Interaction of parasite communities in the host

Parasite morphospecies were prevalent in different combinations within the host individuals and some of these combinations could be detected in more than one individual. The most common combination was an double-infection with *S. fuelleborni* and *Trichuris* sp, which occurred in 20 individuals. Infections with only one morphospecies could be observed for all of the morphospecies. Infections with more than three parasite morphospecies could only be seldom detected. For a detailed list of all occurring single- and multi-infections see Appendix C.

When correlating the prevalence of parasite taxa, a significant interaction was found between six pairs of parasite morphospecies: *S. fuelleborni* and *Trichuris* sp. (Spearman rank correlation: r_s 0.527, $p < 0.001$). For the analysis each morphospecies was correlated with all other parasite morphospecies. Altogether 135 tests were executed. The complete results of the Spearman rank correlation can be found in Appendix D.

5.3.2.5 Egg output

Eggs and larvae excreted per 100µl of fecal sediment (EPS) in macaque samples were also measured. In general, the number of parasite stages emitted in feces was low regarding all host individuals over the study period (Table 5.12). The highest EPS was measured for *S. fuelleborni* and *Trichuris* sp.. The maximum count of *S. fuelleborni* eggs in 100µl of fecal sediment was 3152, for *Trichuris* sp. 154. For the other parasite taxa mean output was less than one propagule per 100µl (Table 5.12).

Significant differences in the abundance of *S. fuelleborni*, *Trichuris* sp., MIF, nematode larva and *Oesophagostomum* sp. could be detected (Figure 5.16 and Figure 5.17).

For *S. fuelleborni*, the mean abundance in the human contact groups was significantly higher than in the sylvatic groups P3 and P4 (Mann Whitney U-test: $p<0.05$, Table 5.13). Additionally, the mean abundance of the groups from Kosumpee forest park was higher than the abundance in the human contact groups from Don Chao Poo forest park (Mann Whitney U-test: $p<0.05$, Table 5.13).

All Kosumpee groups had higher *Trichuris* sp. egg outputs than the Don Chao Poo groups (Mann Whitney U-test: $p<0.05$, Table 5.13). The egg output of K3 was also significantly higher than in group K2.

Egg output for MIF was significantly lower in the sylvatic group P4 than in all Kosumpee groups (Mann Whitney U-test: $p<0.05$, Table 5.13) and in the human contact group P2 (Mann Whitney U-test: $p<0.05$, Table 5.13). The sylvatic group P3 showed lower egg output than group K2 (Mann Whitney U-test: $p<0.05$, Table 5.13) and was nearly significant for all other groups from Kosumpee forest park and P2 (Mann Whitney U-test: $p<0.05$, Table 5.13).

For nematode larva the mean abundance was significantly lower in the two Don Chao Poo forest park groups P2 and P3 than in K3 (Mann Whitney U-test: $p<0.05$, Table 5.13).

For *Oesophagostomum* sp. eggs the mean abundance was significantly lower in all Kosumpee forest park groups than in P1 (Mann Whitney U-test: $p<0.05$, Table 5.13).

Table 5.12 Fecal egg and larvae output in macaques from Thailand (refers to number of parasite stages per 100 µl fecal sediment). K1-K3 are groups from Kosumpee forest park, P1-P4 are groups from Don Chao Poo forest park. MIF= minute intestinal fluke, SD is the standard deviation, mean and maximum number of parasite stages per sample are presented.

Group	Individuals	*Strongyloides fuelleborni*		*Trichuris* sp.		hookworm B		MIF		*Oesophagostomum* sp.		nematode larva	
		Max	Mean (SD)	Max	Mean (SD)	Max	Mean (SD)	Max	Mean (SD)	Max	Mean (SD)	Max	Mean (SD)
K1	20	3152	82.8 (SD 281.6)	116	6.2 (SD 9.7)	25	0.4 (SD 1.9)	20	0.1 (SD 0.2)	0	0	16	0.4 (SD 1.3)
K2	20	322	28.6 (SD 44.6)	101	4.3 (SD 8.3)	1	0.01 (SD 0.07)	5	0.2 (SD 0.5)	0	0	56	1.7 (SD 7.0)
K3	20	3001	73.0 (SD 245.4)	154	10.5 (SD 15.6)	26	0.8 (SD 2.1)	2	0.1 (SD 0.3)	0	0	35	1.5 (SD 3.5)
P1	21	56	2.3 (SD 6.5)	11	0.7 (SD 1.4)	6	0.2 (SD 0.5)	5	0.2 (SD 0.5)	3	0.1 (SD 0.2)	12	0.2 (SD 0.8)
P2	20	16	0.8 (SD 1.7)	68	2.6 (SD 5.6)	1	0.02 (SD 0.07)	1	0.1 (SD 0.2)	1	0.03 (SD 0.09)	8	0.3 (SD 1.0)
P3	16	1	0.03 (SD 0.1)	23	1.0 (SD 2.2)	2	0.1 (SD 0.2)	0	0	6	0.2 (SD 0.5)	32	0.8 (SD 2.7)
P4	18	0	0	20	0.7 (SD 1.6)	1	0.03 (SD 0.1)	0	0	1	0.04 (SD 0.1)	14	0.4 (SD 1.1)

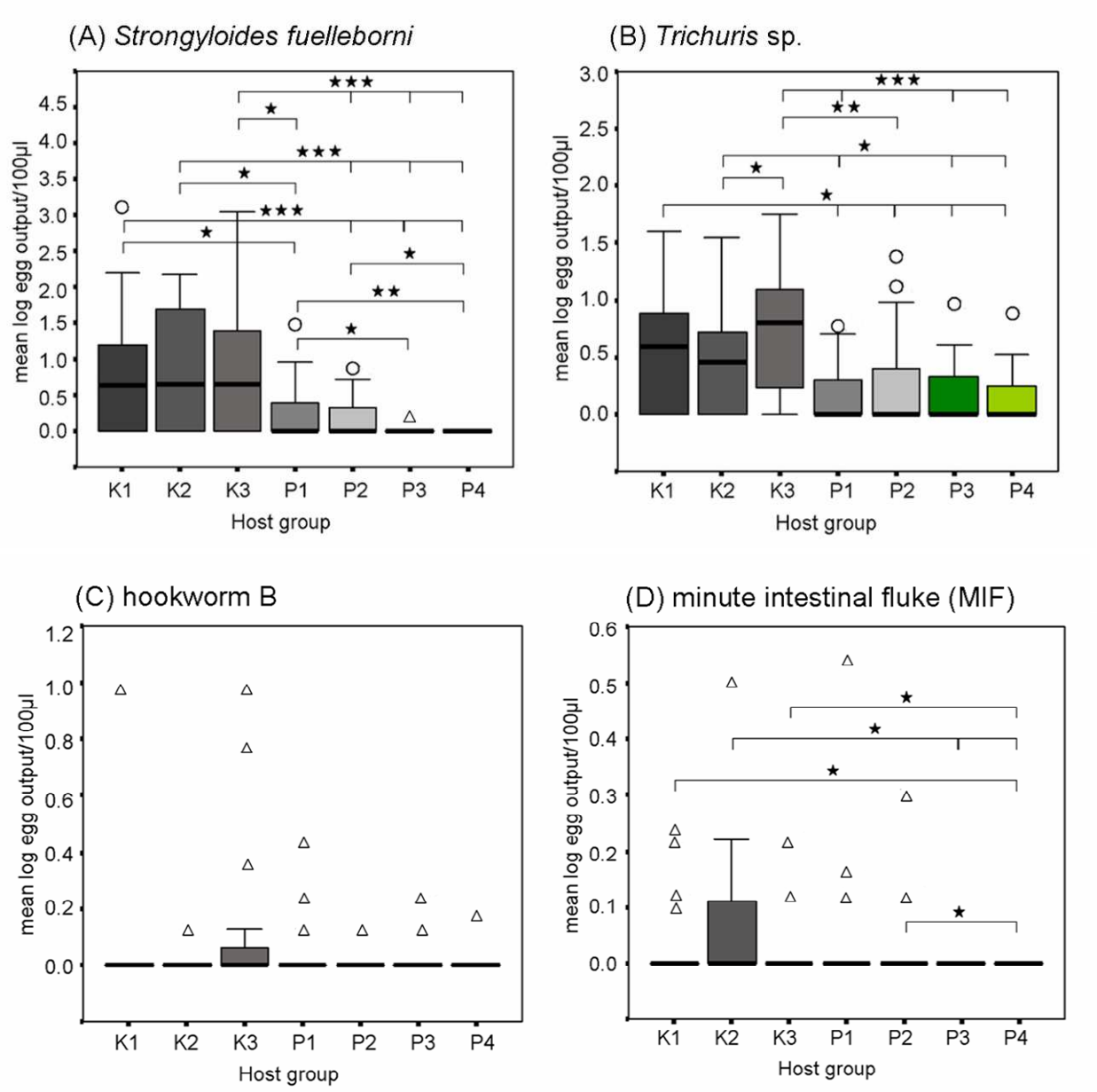

Figure 5.16 (A-D) Mean egg output for four parasite morphospecies per host group in macaques from Thailand. K1-K3 are groups from Kosumpee forest park, P1-P4 are groups from Don Chao Poo forest park. Grey boxes represent human contact groups, green boxes represent sylvatic groups. The boxes show interquartile ranges, the bold horizontal bars give the median. The ends of the whiskers represent the largest and smallest values that are not outliners or extreme values. The circles represent outliners and the triangles extreme values. Asterisks indicate statistical differences (Mann Whitney U-test: * $p \leq 0.05$; ** $p \leq 0.01$, *** $p \leq 0.001$).

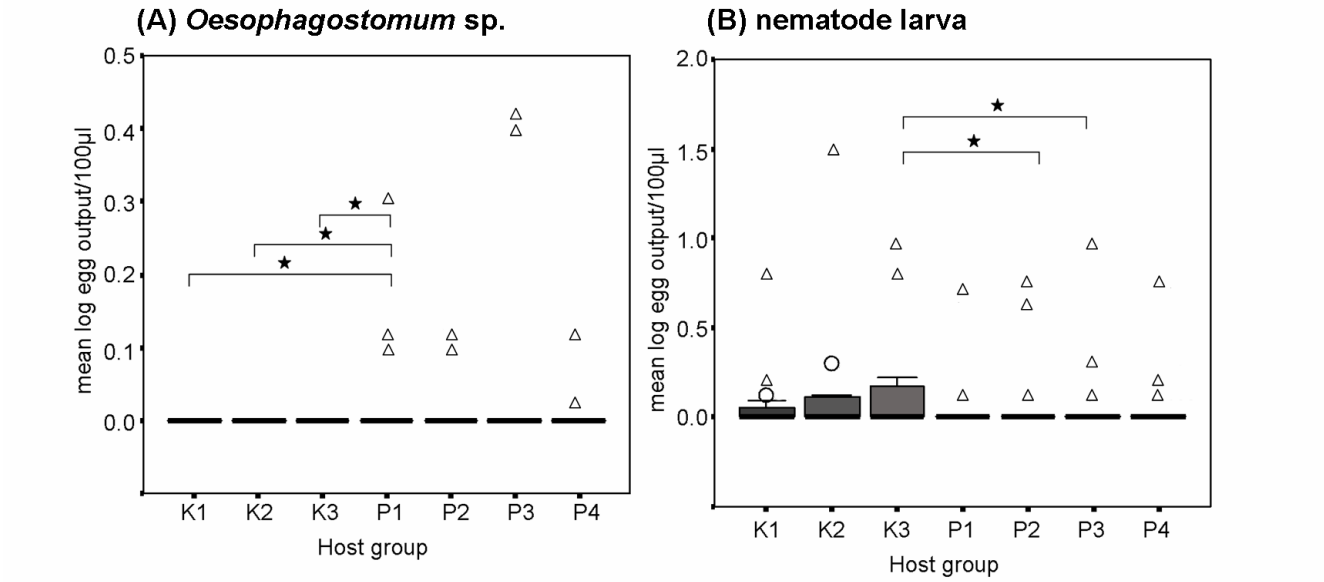

Figure 5.17 (A-B) Mean egg/larvae output for *Oesophagostomum* sp. and nematode larva per host group macaques from Thailand. K1-K3 are groups from Kosumpee forest park, P1-P4 are groups from Don Chao Poo forest park. Grey boxes represent human contact groups, green boxes represent sylvatic groups. The boxes show interquartile ranges, the bold horizontal bars give the median. The ends of the whiskers represent the largest and smallest values that are not outliners or extreme values. The circles represent outliners and the triangles extreme values. Asterisks indicate statistical differences (Mann Whitney U-test: * $p \leq 0.05$).

Table 5.13 Mann Whitney U-Test results for differences in abundance of parasite infection between macaque groups. K1-K3 are groups from Kosumpee forest park, P1-P4 are groups from Don Chao Poo forest park.*Prosthenorchis*= *Prosthenorchis elegans*, *Hymenolepis*= *Hymenolepis* sp., N is the sample size. Significant results are bold printed (* $p \leq 0.05$).

			Compared Groups										
			K1+K2	**K1+K3**	**K1+P1**	**K1+P2**	**K1+P3**	**K1+P4**	**K2+K3**	**K2+P1**	**K2+P2**	**K2+P3**	**K2+P4**
Parasite taxa	***Strongyloides fuelleborni***	**U**	192	195.5	122.5	87	59.5	63	196.5	117	83.5	54	54
		p	0.826	0.901	0.015**	0.001***	<0.001***	<0.001***	0.923	0.011*	0.001***	<0.001***	<0.001***
	***Trichuris* sp.**	**U**	167	158	104.5	125	79.5	85.5	125	123	145	47	102
		p	0.363	0.251	0.004**	0.033*	0.007**	0.003**	0.040*	0.016*	0.115	0.032*	0.015*
	hookworm B	**U**	199.5	162	192	199.5	139.5	179.5	158.5	189.5	200	137	178.5
		p	0.971	0.098	0.362	0.971	0.232	0.970	0.071	0.299	1	0.179	0.910
	minute intestinal fluke	**U**	180.5	199	199.5	199.5	128	144	184	180.5	180.5	112	126
		p	0.488	0.969	0.676	0.985	0.062	0.048*	0.569	0.288	0.487	0.019*	0.012*
	***Oesophagostomum* sp.**	**U**	200	200	170	180	140	150	200	170	180	140	150
		p	1	1	0.043*	0.152	0.109	0.061	1	0.043*	0.152	0.109	0.061
	nematode larva	**U**	189.5	163	178.5	173	132.5	178.5	173.5	168.5	164	124	169.5
		p	0.718	0.228	0.210	0.270	0.177	0.953	0.399	0.117	0.163	0.097	0.692
		N	40	40	41	40	36	38	40	41	40	36	38
			K3+P1	**K3+P2**	**K3+P3**	**K3+P4**	**P1+P2**	**P1+P3**	**P1+P4**	**P2+P3**	**P2+P4**	**P3+P4**	
	Strongyloides fuelleborni	**U**	121	86	59.5	63	165.5	113	117	137.5	144	135	
		p	0.014*	0.001***	<0.001***	<0.001***	0.149	0.025*	0.004**	0.233	0.048*	0.289	
	***Trichuris* sp.**	**U**	79.5	103	61.5	68	189	164	178	142.5	154	142	
		p	<0.001***	0.006**	0.001***	0.001***	0.533	0.886	0.718	0.517	0.379	0.934	
	hookworm B	**U**	186	158.5	147.5	144.5	189.5	162.5	172	137	178.5	124.5	
		p	0.365	0.071	0.584	0.102	0.299	0.793	0.363	0.179	0.910	0.229	
	minute intestinal fluke	**U**	197.5	197.5	128	144	200.5	144	162	128	144	144	
		p	0.619	0.923	0.062	0.048*	0.705	0.120	0.100	0.062	0.048*	1	
	***Oesophagostomum* sp.**	**U**	170	180	140	150	190.5	161	183.5	154	168	141	
		p	0.043*	0.152	0.109	0.061	0.408	0.738	0.817	0.762	0.550	0.867	
	nematode larva	**U**	144.5	142	108.5	147.5	208	163.5	164.5	155	160	123	
		p	0.023*	0.039*	0.031*	0.249	0.919	0.771	0.272	0.740	0.357	0.240	
		N	41	40	36	38	41	37	39	36	38	34	

Differences in mean intensity were also recorded for *S. fuelleborni* and *Trichuris* sp. For *S. fuelleborni* the mean output of infected animals from Kosumpee forest park group K3 were significantly higher than from groups P1 and P2. Statistics for *S. fuelleborni* infection intensity was not useful with both forest groups, due to the fact that in group P3 only one individual was infected and in P4 no infection occurred. *Trichuris* sp. infection intensity was higher in all Kosumpee groups than in the groups P1, P3 and P4. In addition K3 showed higher egg outputs than group K2 (Table 5.14 and Table 5.15).

All other parasite species had similar mean egg outputs in the positive samples (see Table 5.14 and Table 5.15).

Table 5.14 Mean intensity of parasite infection in macaques (refers to number of parasite stages per 100 µl fecal sediment). K1-K3 are groups from Kosumpee forest park, P1-P4 are groups from Don Chao Poo forest park. MIF= minute intestinal fluke, mean egg output is provided, SD is standard deviation.

Group	*Strongyloides fuelleborni*	*Trichuris* sp.	hookworm B	MIF	*Oesophagostomum* sp.	nematode larva
K1	127.4 (SD 345.6)	8.8 (SD 10.6)	8.7	0.5 (SD 0.3)	0	1.5 (SD 2.4)
K2	41.1 (SD 48.5)	6.2 (SD 9.4)	0.3	0.7 (SD 0.7)	0	5.6 (SD 12.6)
K3	112.3 (SD 300.9)	14.0 (SD 16.7)	3.0 (SD 3.6)	0.6 (SD 0.2)	0	3.8 (SD 4.9)
P1	6.1 (SD 9.6)	1.9 (SD 1.7)	1.0 (SD 0.9)	1.0 (SD 1.1)	0.5 (SD 0.4)	1.5 (SD 2.0)
P2	2.6 (SD 2.2)	6.6 (SD 7.8)	0,3	0.5 (SD 0.3)	0.3 (SD 0.1)	2.2 (SD 1.8)
P3	0.5	3.1 (SD 3.1)	0.6 (SD 0.2)	0	1.6 (SD 0.1)	4.1 (SD 6.0)
P4	0	2.0 (SD 2.4)	0,5	0	0.2 (SD 0.2)	1.6 (SD 2.1)

Table 5.15 Mann Whitney U-Test results for differences in intensity of parasite infection between tamarin groups. K1-K3 are groups from Kosumpee forest park, P1-P4 are groups from Don Chao Poo forest park. N is the sample size. Significant results are bold printed (* p≤ 0.05)

			Compared Groups										
			K1+K2	**K1+K3**	**K1+P1**	**K1+P2**	**K1+P3**	**K1+P4**	**K2+K3**	**K2+P1**	**K2+P2**	**K2+P3**	**K2+P4**
Parasite taxa	***Strongyloides fuelleborni***	**U**	89	80	27.5	17.5	0	/	84.5	35.5	23.5	3	/
		N	27	26	21	19	13	/	27	22	20	15	/
		p	0.923	0.817	0.065	0.059	0.107	/	0.752	0.161	0.162	0.353	/
	***Trichuris* sp.**	**U**	68.5	73	17.5	41	16.5	13.5	42.5	26.5	54.5	27.5	21
		N	28	29	22	22	19	20	29	22	22	19	20
		p	0.174	0.162	0.008**	0.305	0.086	0.018*	0.006**	0.043*	0.918	0.487	0.082
	hookworm B	**U**	0	0.5	0	0	0	0	1	0.5	0.5	0.5	0
		N	2	6	4	2	4	2	6	4	2	4	2
		p	0.317	0.228	0.180	0.317	0.157	0.317	0.351	0.346	1	0.317	0.317
	minute intestinal fluke	**U**	11.5	7	4.5	7.5	/	/	8	5.5	11.5	/	/
		N	10	8	7	8	/	/	10	9	10	/	/
		p	0.912	0.757	0.593	0.878	/	/	0.363	0.345	0.904	/	/
	***Oesophagostomum* sp.**	**U**	/	/	/	/	/	/	/	/	/	/	/
		N	/	/	/	/	/	/	/	/	/	/	/
		p	/	/	/	/	/	/	/	/	/	/	/
	nematode larva	**U**	14.5	13	6	6	5	6.5	17.5	7	7	7.5	8.5
		N	11	13	8	8	8	9	14	9	9	9	10
		p	0.924	0.289	0.651	0.653	0.453	0.381	0.382	0.598	0.599	0.692	0.441
			K3+P1	**K3+P2**	**K3+P3**	**K3+P4**	**P1+P2**	**P1+P3**	**P1+P4**	**P2+P3**	**P2+P4**	**P3+P4**	
	Strongyloides fuelleborni	**U**	25	12	0	/	18	0	/	0	/	/	
		N	21	19	14	/	14	9	/	7	/	/	
		p	0.050*	0.018*	0.107	/	0.435	0.120	/	0.130	/	/	
	***Trichuris* sp.**	**U**	7	33	9	8	15	12.5	22	16.5	10	10	
		N	23	23	20	21	16	13	14	13	14	11	
		p	0.001***	0.081	0.013*	0.004**	0.071	0.270	0.794	0.607	0.069	0.360	
	hookworm B	**U**	6	1	5	2	0.5	2.5	1	0.5	0	1	
		N	8	6	8	6	4	6	4	4	2	4	
		p	0.647	0.351	0.442	0.766	0.346	0.369	0.655	0.317	0.317	0.637	
	minute intestinal fluke	**U**	5.5	5.5	/	/	3.5	/	/	/	/	/	
		N	7	8	/	/	7	/	/	/	/	/	
		p	0.853	0.429	/	/	0.329	/	/	/	/	/	
	***Oesophagostomum* sp.**	**U**	/	/	/	/	3.5	0	5	0	3	0	
		N	/	/	/	/	6	6	7	4	5	5	
		p	/	/	/	/	0.803	0.060	0.711	0.121	1	0.076	
	nematode larva	**U**	5	10	11.5	15.5	3	3	3	4.5	6	5	
		N	11	11	11	12	6	6	7	6	7	7	
		p	0.147	0.680	0.918	0.931	0.487	0.487	0.285	1	1	0.724	

5.3.2.5 Behavioral data

The behavior of human contact groups differed significantly from non-contact groups by spending more time being fed by humans and on the ground (Figure 5.16 and Figure 5.17), while the non-contact groups spent significantly more time 15 to 20 meters above the ground. Time spent moving, resting, feeding, drinking and grooming were not significantly different between these groups. Activity budgets for each group can be found in Appendix E.

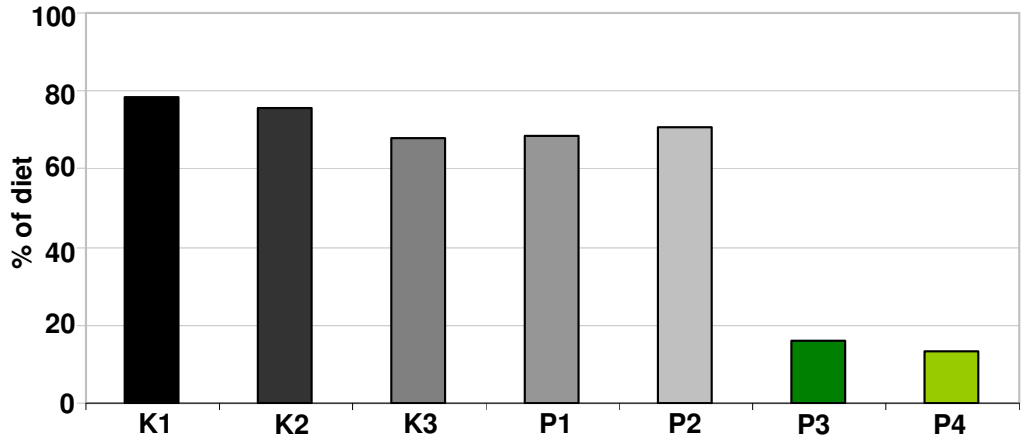

Figure 5.18 Daily time budget macaques spent on the ground for each study group. K1-K3 are study groups from Kosumpee forest park, P1-P4 are study groups from Don Chao Poo forest park Grey bars represent human contact groups, green bars sylvatic groups.

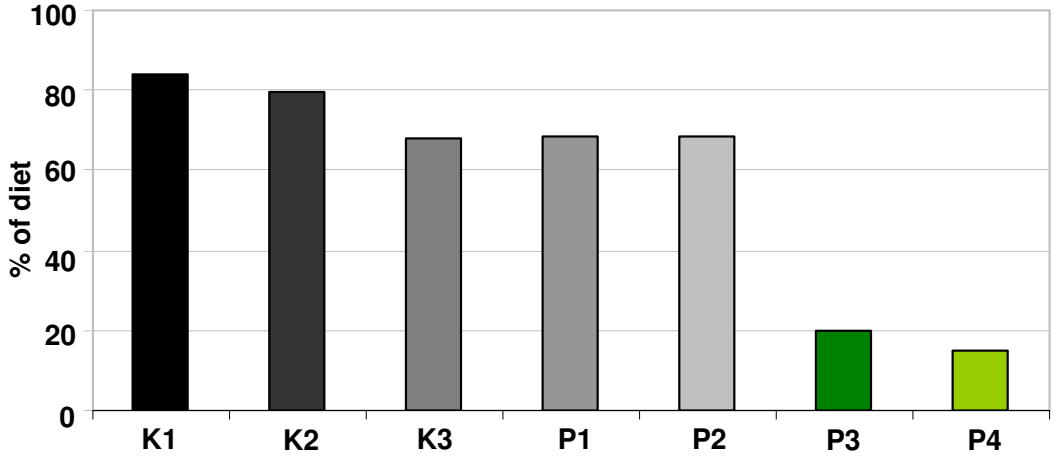

Figure 5.19 Daily budget of food provided by humans in the diet of macaques for each study group. K1-K3 are study groups from Kosumpee forest park, P1-P4 are study groups from Don Chao Poo forest park. Grey bars represent human contact groups, green bars sylvatic groups.

There were positive correlations between the prevalence of *S. fuelleborni*, *Trichuris* sp. and nematode larva and accepting human food while the correlation with *Oesophagostomum* sp. was significantly negative. The corresponding correlations with time spent eating fruit were also highly significant but in the opposite direction (Table 5.16). Similarly, the prevalence of *S. fuelleborni*, *Trichuris* sp., MIF and nematode larva was highly negatively correlated with the total time spent on feeding while for *Oesophagostomum* sp. the correlation was positive and highly significant (Table 5.16).

For intensity of infection, there were strong negative correlations between *S. fuelleborni*, *Trichuris* sp., hookworm B, MIF and nematode larva and the time spent on eating fruit, of which correlation for *Oesophagostomum* was highly significant positive (Table 5.17). The converse was true for time spent on taking human food. There were significant positive correlations between the intensity of infection and the time spent on the ground for *S. fuelleborni*, *Trichuris* sp., hookworm B and MIF while the correlation was negative and significant for *Oesophagostomum* (Table 5.17).

Table 5.16 Results of Spearman rank correlation of parasite prevalence and behavioural data of macaques. N= 135, *Strongyloides*= *Strongyloides fuelleborni*, *Trichuris*= *Trichuris* sp., hookworm B= hookworm B, MIF= minute intestinal fluke, *Oesophagostomum*= *Oesophagostomum* sp., nematode larva= nematode larva. FF= natural food sources, FT= human provided food, Ground=being on the ground, 1+= being between ground an 5m height, 5+= being between 5m and 10m height, 10+=being between 10m and 15m height, 15+= being between 15m and 20m height, 20* being higer than 20m, AM= locomotion, AR= resting, AF, feeding, AD= drinking, AG= grooming.

	FN		**FH**									
	r_s	p	r_s	p								
Strongyloides	-0.44	**0.002**	0.362	**0.01**								
Trichuris	-0.461	**0.001**	0.397	**0.005**								
hookworm B	-0.099	0.499	0.195	**0.18**								
MIF	-0.336	**0.018**	0.263	**0.068**								
Oesophagostomum	0.66	**<0.001**	-0.593	**<0.001**								
nematode larva	-0.838	**<0.001**	0.821	**<0.001**								
	Ground		**1+**		**5+**		**10+**		**15+**		**20+**	
	r_s	p	r_s	p	r_s	p	r_s	p	r_s	p	r_s	p
Strongyloides	0.364	**0.01**	0.188	0.196	-0.291	**0.043**	-0.386	**0.006**	-0.045	0.76	-0.359	**0.011**
Trichuris	0.382	**0.007**	0.194	0.181	-0.226	0.119	-0.423	**0.002**	-0.03	0.838	-0.376	**0.008**
hookworm B	0.291	0.131	0.039	0.789	-0.196	0.177	-0.171	0.239	-0.082	0.574	0.006	0.969
MIF	0.254	**0.078**	0.156	0.284	-0.162	0.267	-0.273	**0.058**	0.034	0.814	-0.257	**0.075**
Oesophagostomum	-0.589	**<0.001**	-0.267	**0.064**	0.333	**0.019**	0.51	**<0.001**	0.3	**0.036**	0.453	**0.001**
nematode larva	0.811	**<0.001**	0.187	0.198	-0.422	**0.003**	-0.68	**<0.001**	-0.537	**<0.001**	-0.55	**<0.001**
	AM		**AR**		**AF**		**AD**		**AG**			
	r_s	p	r_s	p	r_s	p	r_s	p	r_s	p		
Strongyloides	-0.076	0.605	0.028	0.849	-0.649	**<0.001**	0.101	0.49	0.536	**<0.001**		
Trichuris	-0.045	0.76	0.072	621	-0.691	**<0.001**	0.177	0.223	0.53	**<0.001**		
hookworm B	-0.042	0.775	0.2	0.169	0.011	0.939	-0.014	0.922	0.043	0.771		
MIF	-0.078	0.593	0.001	0.995	-0.6	**<0.001**	0.112	0.442	0.456	**0.001**		
Oesophagostomum	-0.133	0.364	-0.168	0.249	0.672	**<0.001**	-0.216	0.137	-0.31	**0.03**		
nematode larva	0.219	0.131	0.141	0.334	-0.645	**<0.001**	0.44	**0.002**	0.335	**0.019**		

Table 5.17 Spearman rank correlation of parasite intensity and behavioural data of macaques. . N= 135, *Strongyloides*= *Strongyloides fuelleborni*, *Trichuris*= *Trichuris* sp., hookworm B= hookworm B, MIF= minute intestinal fluke, *Oesophagostomum*= *Oesophagostomum* sp., nematode larva= nematode larva. FF= natural food sources, FT= human provided food, Ground=being on the ground, 1+= being between ground an 5m height, 5+= being between 5m and 10m height, 10+=being between 10m and 15m height, 15+= being between 15m and 20m height, 20* being higer than 20m, AM= locomotion, AR= resting, AF, feeding, AD= drinking, AG= grooming.

	FN		**FH**									
	r_s	p	r_s	p								
Strongyloides	-0.798	**<0.001**	0.762	**<0.001**								
Trichuris	-0.518	**<0.001**	0.448	**0.001**								
hookworm B	-0.197	0.176	0.164	0.26								
MIF	-0.579	**<0.001**	0.568	**<0.001**								
Oesophagostomum	0.604	**<0.001**	-0.537	**<0.001**								
nematode larva	-0.294	**0.039**	0.244	0.091								
	Ground		**1+**		**5+**		**10+**		**15+**		**20+**	
	r_s	p	r_s	p	r_s	p	r_s	p	r_s	p	r_s	p
Strongyloides	0.748	**<0.001**	0.211	0.145	-0.387	**0.006**	-0.648	**<0.001**	-0.51	**<0.001**	-0.563	**<0.001**
Trichuris	0.464	**0.001**	0.326	**0.022**	-0.35	**0.014**	-0.411	**0.003**	-0.235	0.104	-0.389	**0.006**
hookworm B	0.131	0.371	0.083	0.569	0.004	0.976	-0.215	0.138	-0.023	0.875	-0.237	0.102
MIF	0.545	**<0.001**	-0.037	0.799	-0.196	0.178	-0.403	**0.004**	-0.632	**<0.001**	-0.393	**0.005**
Oesophagostomum	-0.542	**<0.001**	-0.201	0.167	0.403	**0.004**	0.528	**<0.001**	0.264	0.067	0.487	**<0.001**
nematode larva	0.249	0.085	0.186	0.202	-0.161	0.27	-0.205	0.159	0.006	0.966	-0.162	0.266
	AM		**AR**		**AF**		**AD**		**AG**			
	r_s	p	r_s	p	r_s	p	r_s	p	r_s	p		
Strongyloides	0.241	0.095	0.16	0.273	-0.612	**<0.001**	0.382	**0.007**	0.281	**0.051**		
Trichuris	0.121	0.401	0.273	0.058	-0.499	**<0.001**	-0.025	0.864	0.152	0.296		
hookworm B	0.068	0.64	0.069	0.639	-0.262	0.069	0.139	0.342	0.163	0.264		
MIF	0.344	**0.016**	-0.081	0.58	-0.151	0.299	0.488	**<0.001**	-0.025	0.864		
Oesophagostomum	-0.04	0.783	-0.078	0.595	0.613	**<0.001**	-0.174	0.231	-0.471	**0.001**		
nematode larva	-0.044	0.766	0.081	0.578	-0.499	**<0.001**	0.038	0.795	0.276	**0.055**		

5.4 Discussion

5.4.1 Human fecal samples

In the human samples some of the most important parasites from South-East Asia were present. They include geohelminths (hookworms, *Strongyloides stercoralis* and *Trichostrongylus* spp.) and foodborne parasites (*Taenia* spp., *Opistorchis viverrini* and minute intestinal flukes). Accurate identification to species level could be made for the eggs of *O. viverrini* and *S. stercoralis*, as no other species with similar egg morphologies are found in the study area. The hookworms were not identified to species level but *Necator americanus* is likely to have predominated. Hinz (1996) indicates that 99% of all hookworms in Thailand belong to this species in this area. Minute intestinal flukes comprise the families Heterophyidae, Plagiorchiidae and Lecithodendriidae, the eggs of which are difficult to distinguish (Kaewkes *et al*. 1991; Tesana *et al*. 1991). However, with the knowledge of distribution and morphological features it was possible to determine them as heterophyids in this study. The specific status of the Southeast Asia *Taenia* species involved is uncertain (Ito *et al*. 2003). Prevalence data of *Taenia* are likely to be underestimates as stool samples were examined for eggs but not for proglottids.

The geohelminths found have a direct life cycle without intermediate hosts or vectors. They are causing human infection by active penetration of the skin by the larvae, which can be found in fecal contaminated soil, foodstuffs and water supplies. These geohelminths are highly prevalent worldwide, with billions of people being infected, particularly in tropical areas and developing countries (Holland and Kennedy 2002). They cause a wide range of symptoms including intestinal manifestations (diarrhoea, abdominal pain), and general malaise and weakness, which may affect working and learning capacities and impair physical growth (WHO 2006). Heavy infections can also cause death (WHO 2002, 2004; see chapter 4.4.1).

Hookworms are considered to be a major public health problem in Thailand (Hinz 1996). Humans acquire them when third-stage infective larvae in soil either penetrate the skin (for both *N. americanus* and *A. duodenale*) or when they are ingested (*A. duodenale* only). *Trichostrongylus* spp. do not belong taxonomically to the hookworms, but the eggs are so similar to those of hookworms that they have been reported as such in previous studies (Goldsmid 1991). The size and shape however make it possible in some cases to distinguish

these eggs from those of other taxa (Goldsmid 1991). I could also identify them due to egg morphology and coproculture could proof this identification. Infections occurs when larvae are ingested.

Strongyloides stercoralis is one of the most common parasitic infection in north-eastern Thailand with prevalences of about 30% (Sithithaworn *et al*. 2003; Jongsuksuntigul and Imsonboon 2003). This correlates with the findings of my study. *Strongyloides* infections occur in the tropical and subtropical regions worldwide, mainly in areas where fecal contamination of the soil and water is high (Keiser and Nuttman 1994). Therefore, rural areas in developing countries are mostly affected. This fits the much lower prevalences of infection in Thailand compared to the villagers of the Peruvian study (see chapter 4).

S. stercoralis can cause both respiratory, dermatological and gastrointestinal symptoms. Many of the people infected are initially asymptomatic at first. Gastrointestinal symptoms include abdominal pain and diarrhoea (Roberts and Janovy 2005). Dermatologic manifestations include itching urticarial rashes (larva currens migrans), and mild hemorrhage at the site where the skin has been penetrated. If the parasite reaches the lungs, the chest may feel as if it is burning, and wheezing and coughing may result, along with pneumonia-like symptoms (Löffler's syndrome) (Roberts and Janovy 2005). Eventually, the intestines can be invaded, leading to burning pain, tissue damage, sepsis and ulcers. In severe cases, edema may result in obstruction of the intestinal tract as well as the loss of peristaltic contractions (Roberts and Janovy 2005). In immunocompromised individuals strongyloidiasis can cause a hyperinfective syndrome (also called disseminated strongyloidiasis) due to the reproductive capacity of the parasite inside the host (Viney and Lok 2007). This hyperinfective syndrome has a mortality rate of close to 90% (WHO 1998b). *Strongyloides stercoralis* infections are also reported in dogs and cats (Nolan 2001).

Opistorchis viverrini is of special interest in northeastern Thailand. It is endemic to Thailand, Laos, Vietnam and Cambodia (WHO 1995). Currently more than 600 million people are at risk of infection with these parasites (Keiser and Utzinger 2005). An estimated six to seven million people suffer from “opisthorchiasis” in Thailand alone (Jongsuksuntigul and Imsonboon 2003).

This trematode is one of a trio (*O. viverrini, O. felineus, Clonorchis sinensis*) of closely-related medium-sized liver flukes that inhabit the bile ducts of fish-eating animals, which are recognized as a carcinogen (Sripa *et al*. 2007), and causes many deaths from bile duct cancer

(Chloangiocarcinoma) in Southeast Asia (Sithithaworn *et al*. 2007; Sripa *et al*. 2007). The association between the parasite and liver cancer is so strong that the parasite has been accepted as a known carcinogen even though the mechanism is not fully understood (Sithithaworn *et al*. 2007; Sripa *et al*. 2007). Cholangiocarcinoma accounts for more than two thirds of liver cancers in Khon Kaen province, Thailand, where more than one in three residents are infected with *O. viverrini*. (Sripa *et al*. 2007). The results of my study totally match to the data from literature, I could also detect prevalences of about 30% in people from Kosum Phi Sai and Pha Na.

Most people who have *O. viverrini* flukes in their bile ducts have no symptoms, but the more flukes there are, the more likely it is that symptoms will appear. Abdominal discomfort, flatulence and fatigue are typical (WHO 1995; Murrel and Fried 2007) Nonetheless, heavy, long-standing infection is associated with a number of hepatobiliary diseases, including cholangitis, obstructive jaundice, hepatomegaly, fibrosis of the periportal system, cholecystitis and cholelithiasis (WHO 1995; Murrel and Fried 2007).

Infection with *O. viverrini* begins with the ingestion of raw or undercooked freshwater fish in dishes such as *koi-pla* that harbor metacercariae — the larval stage of the parasite — encysted in its tissues (Sripa *et al*. 2007). *O. viverrini* flukes produce eggs that are washed out with the bile, mix with bowel contents, and pass in the stool. In addition, aquatic snails of the genus *Bythinia* are needed as first intermediate host in which cercariae develop. Free swimming cercariae leave the snail and invade the tissues of freshwater fish, developing to metacercariae in the fish muscle (Sripa *et al*. 2007). This indirect life cycle excludes the possibility of a cross-transmission between humans and primates.

Human stool samples of my study harbored further trematode eggs, referred to as minute intestinal flukes (MIF) due to the small size of the eggs. About 40 to 50 million people are estimated to be infected with foodborne trematodes worldwide (Keiser and Utzinger 2005), but this is certainly an underestimate of the true number of people infected. Like *O. viverrini*, MIF infection is acquired by ingestion of raw or undercooked fish (Dorny *et al*. 2009). Most of the infected people live in Southeast Asia. Abdominal discomfort, flatulence, and fatigue are typical clinical infestations (WHO 1995; Murrel and Fried 2007).

I could also detect another foodborne parasite in people from Kosum Phi Sai and Pha Na. Infection with *Taenia* spp. is acquired by eating raw or undercooked meat (Dorny *et al*. 2009).

Taeniid eggs are indistinguishable from each other and could belong two any of the three species infecting *Taenia solium, T. saginata* and *T. asiatica* (Craig and Ito 2007).

Prevalences of infection in both villages, Kosum Phi Sai and Pha Na are limited due to the possibility of regular medical treatment. The *Opisthorchis*-hazard in north-eastern Thailand described previously and the high prevalences of *Strongyloides* in many rural areas of Thailand led to the establishment of a system of regular screenings and treatment in hospitals (Sithithaworn *et al*. 2003) resulting in the moderate prevalences of infection (up to 35%) found in my study. In addition, people coming into contact with macaques are living in urban areas. They do not have the poor sanitation standards typical for rural areas or inmidden tropical forests (as it was the fact in the Peruvian village Diamante). They do have toilet facilities and supermarkets for buying food supplies, which also lowers the infection risk with parasites enormously.

However, all of the parasites found in the humans in Kosum Phi Sai and Pha Na are potentially infectable to primates and some are already suspected to be responsible for deaths in primate populations. As already described in chapter 4.4.1 hookworm infections are known from nonhuman primates and even fatal cases are even known (Fossey 1983). Pet macaques in Sulawesi were found to be infected with hookworms and a possible cross-transmission was not excluded (Jones-Engel *et al*. 2004). Reports about infections with *S. stercoralis* in captive individuals are available (Murata *et al*. 2002; Jones-Engel *et al*. 2004; Gillespie *et al*. 2005b). Infections of primates with *O. viverrini* are unknown so far, however, the original nonhuman host also remains unknown (Petney, pers. comm.). Primates harbour MIF and even reports on several species of minute intestinal fluke in macaques are known (Lacoste 2009).

However, since *Taenia*, *Opisthorchis* and MIF have indirect life cycles involving intermediate hosts, cross-transmission is not expected for these parasite taxa. Nevertheless, the situation in and around the study areas of Kosum Phi Sai and Pha Na poses a high risk interface where the macaques can come into contact with contaminated soil and food.

5.4.2 Macaque samples

5.4.2.1 Parasite diversity

The present study determined the gastrointestinal parasite spectrum of several troops of *Macaca fascicularis* in the wild, differing in their contact to humans and their facilities. Identification of the parasites was difficult, due to our general lack of knowledge of wild nonhuman primate parasites and the of reference material (see chapter 3.2 for discussion).

Macaques harbored six different parasite morphospecies, including both nematodes and trematodes. Most of these morphospecies could be identified to genus level. Only *Strongyloides fuelleborni* could be identified to species level with the help of agar plate culture. All of the parasite taxonomic groups detected are also known to infect humans, which supports the hypothesis that macaques are potentially susceptible for human-transmitted parasites.

The most prevalent morphospecies I found in macaques was *Trichuris* sp., the eggs resembled those of the human whipworm *T. trichiura*, although the dimensions of the eggs (55-65µm by 27.5-32.5µm) were greater than those of *T. trichiura* usually reported from humans (50-55µm by 20-25µm). Depending on host species, the eggs of parasites excreted in the feces may vary in size (Faust 1967). Morphological studies on adult parasites identified parasites as *T. trichiura* showed morphological differences between specimens collected from nonhuman primates and from humans (Ooi *et al*. 1993). Infections occur through the ingestion of embryonated eggs. They are normally asymptomatic (Flynn 1973), but heavy infections can lead to severe enteritis, anorexia, mucoid diarrhoea and even death (Flynn 1973; Brack 1987).

Strongyloides fuelleborni could also be detected in many macaque individuals. It is a typical parasite of the small intestine of Old world monkeys and apes (Flynn 1973) and occasionally also infects humans in Africa and Asia (Viney and Lok 2007). In contrast to *S. stercoralis*, the eggs can be found in fecal samples and larvae hatch several hours after being passed (Ashford and Barnish 1989). The infective larval stage enters a suitable host via penetration of the hosts skin or oral mucosa. The larvae can cause intense burning and itching when entering the host, and pulmonary symptoms are limited to a nonproductive cough. Heavy infections cause diarrhoea, weight loss, debilitation and increased mortality (Flynn 1973; Brack 1987).

Oesophagostomum spp. are the most common nematodes of Old World monkeys and apes (Toft 1982; Brack 1987; Sleeman *et al*. 2000). Identification however, often ends at genus level as in this study. For macaques, infections with *O. apiostomum*, *O. acuelatum* and *O. bifurcum* have been reported (Brack 2007; Lacoste 2009). *O. stephanostomum* is thought to be responsible for the death of gorillas and chimpanzees (Cousins 1972; Flynn 1973, Brack 1987). These "nodular worms" are infective when larvae are ingested or penetrate the skin. The larvae pass directly through the colon and penetrate deeply into the mucosa. Heavy infections cause diarrhoea, weight loss, debilitation and an increased mortality, especially together with co-stressors (Cousins 1972; Brack 1987). They are not highly specific which enlarges their potential for cross-transmission. Humans are also susceptible to this parasite (Ziem *et al*. 2004). Each of the three species known to infect humans has a large distribution in monkeys as reservoir hosts. In my study, however, I could only detect them in few individuals from Don Chao Poo forest park. Therefore it seems unlikely that infection with these parasites has fatal consequences in the study animals, since the infection risk seems to be low.

The trematode eggs found in macaque stool samples were designated as minute intestinal fluke eggs because of their small size. As already mentioned before, this class of parasites comprises the families Lecithondendriidae, Plagiorchiidae and Heterophyidae (Kaewkes *et al*. 1991; Tesana *et al*. 1991) distinction between *Opisthorchis* and MIF eggs was possible because the eggs were stained with iodine (Kaewkes *et al*. 1991). Due to the shape and size of the eggs, I could identify MIF in macaques as members of the Heterophyidae. However, the specific diagnosis of heterophyid eggs is impossible in areas with mixed infections (Chai *et al*. 2009) and in both study areas several species of Heterophyids occur (Sithithaworn pers. comm.). The infections are foodborne obtained while eating raw or undercooked fish. Known representatives of heterophyids in macaques are *Haplorchis taichui*, *H. pumillio* and *H. yokogawai* (Brack 2007; Lacoste 2009), which are all known to infect humans in northeastern Thailand and Laos (Chai *et al*. 2009). Since a specific identification was not possible, neither in human stool samples nor in macaque samples, it is possible, that humans and primates harbor the same parasite species or that they harbour different of heterophyids. The following section will discuss this result more in detail.

5.4.2.2 Cross-transmission of parasites

Comparing the findings from human and macaque stool samples, only one of the helminth morphospecies found in the villagers could be detected in the feces of macaques. However, this is an indirectly transmitted parasite, the minute intestinal fluke. Since MIF require an intermediate host for infection of macaques, no direct transmission of human parasites to these primates takes place in either of the study areas in northeastern Thailand. My data do not support the prediction that primates are infected with intestinal parasites cross-transmitted by humans.

Macaque samples contained parasites which can occur in humans. The eggs identified as *Trichuris* sp. in this study resembled those of the human whipworm *T. trichiura*. *T. trichiura* is commonly recorded from primates and cross-transmissions are considered possible (Ruch 1959; Müller-Graf 1994; Mudakikwa *et al*. 1998; Sleeman *et al*. 2000). However, *T. trichiura* could not be detected in any of the humans and both sylvatic and human contact groups had very similar prevalences of infection. Therefore, infection with *Trichuris* seems to be natural in macaques and not the result of contact with humans. The same seems to be the case for *Oesophagostomum* spp.. Neither could I detect any cross-transmission nor double infections of both *Strongyloides* species. The villagers were only infected by *S. stercoralis*, whereas the macaques harbored only *S. fuelleborni*. This result was confirmed by coproculture. Thus, no transmission of human parasites to macaques takes place in the two investigated areas.

Humans harbored only three species of directly transmitted parasites. Only these geohelminths have a direct lifecycle and infective stages are excreted *via* feces or other body fluids and accumulate on or in the ground (Holland and Kennedy 2002). But none of these geohelminths was found in macaques. The results of my study do not exclude the possibility of a cross-transmission of parasites from humans to nonhuman primates. It is possible that the probability of coming into contact with human parasites was too low.

Although in both parks, extensive, unregulated and often close contact between humans and monkeys occurs, primates and humans did not share the habitat for living. Bodily contact ocurrs regularly between monkeys and human visitors, while monkeys quickly snatch up any offerings of food and people who live and work there share common water sources with the monkey inhabitants and report that they frequently invade their homes and gardens in search of food. However, since people only rarely use the forest parks as open toilets, the highest risk

was excluded in this area. The possibility that they contact contaminated soil or human feces is therefore low.

Bites and scratches which have been reported during encounters between the forest monkeys and their human visitors and the fact that primates climb on the heads and shoulders of visitors, which may bring them into contact with tourist's conjunctiva, nasal and oral mucosa may still represent potential portals of entry for infectious agents. However, gastrointestinal helminths are not transmitted in this way.

The other parasitic infections found in humans and macaques were foodborne infections, transmitted through contaminated food. As already mentioned, infections of primates with *O. viverrini* are unknown so far. A certain suitability of macaques as hosts of *O. viverrini* is imaginable, especially since another foodborne trematode could be found in the feces of macaques. However, in 1963 macaques were tested as experimental laboratory hosts for *O. viverrini* infection (Wykoff 1964). After infection with 400 metacercariae, no eggs could be found in the fecal samples. In addition, only reports on infections of other *Opisthorchis* species, *O. felineus* and *C. sinensis* are known for macaques (Lacoste 2009).

Macaques, however, harbored MIF, which are transmitted by ingestion of raw or undercooked such food in both areas. In Kosumpee forest park, the Chi River crosses the home range of the macaques. However, the macaques have not yet been observed to fish in the river. Another explanation could be that macaques steal fish from fishermen or from houses around the park. This, however, can be excluded in the case of Don Chao Poo forest park (Pha Na), where there are no fisherman and only a few houses close to the park. The most probable explanation is that visitors provide the fish with the food they bring to the macaques. In both parks, complete meals could be observed being offered to the macaques (see Appendix B). The successful transmission of MIF from the same kind of food supports the hypothesis, that macaques are not susceptible for *O. viverrini* infection. Food items macaques select out of the offered food by humans, have to be investigated in detail, to clarify the infection source of MIF and to clarify, if macaques really come into contact with *O. viverrini*.

Although one can exclude a direct cross-transmission of parasites from humans to monkeys and vice versa in the investigated study areas, humans seem to be responsible for MIF infection in macaques by providing them with contaminated food.

5.4.2.3 Variation in PSR

Some differences were found between the two study areas for PSR. Macaques in Kosumpee forest park showed higher PSR than three groups from Pha Na. This result agrees with the prediction made at the beginning of my thesis, where a higher PSR was suspected for human contact groups. However, if only Don Chao Poo groups are considered, no significant differences could be detected between the two human contact and the two sylvatic groups. Therefore, human contact does not influence the PSR significantly. Habitat and related factors seem to be more important for the variation in PSR.

In general, diet composition seems to be an important predictor for PSR especially when parasites with an indirect life cycle are involved (see chapter 4.4.2.3 for discussion). Diet composition may be different between the two parks. However, since the parasite diversity is the same in both areas and only one indirectly transmitted parasite could be found in the macaques, diet is not the decisive factor here.

PSR may also bei influenced by group size and host density. Contact rates between individual hosts are higher in larger groups, which may lead to a higher parasite transmission rate. Positive correlations have been found in insects, birds, primates and other mammals (Côte and Poulin 1995; Morand and Poulin 1998; Arneberg 2002; Nunn *et al.* 2003). However, Vitone and colleagues (2004) could not support this hypothesis when they examined the influence of host density on indirectly transmitted parasite diversity. Most studies that found correlations between host density, group-size and higher PSR worked mainly with parasites transmitted by physical contact (Côte and Poulin 1995; Arneberg 2002) or paid no attention to the differences in the parasites' life cycles (Morand and Poulin 1998; Nunn *et al*. 2003). When indirectly transmitted or soil-transmitted parasites are involved, as in the study presented here, no support for these hypotheses could be found (Vitone *et al*. 2004). Unfortunately, the results are often based on estimates of population density or group size, which is also the case here. This can distort the actual correlation between the parameters.

Kosumpee forest park population is estimated to be bigger in size than told from the park rangers and since the habitat is smaller host density seems to be higher. The Kosumpee groups also harbor more multi-species infections with directly transmitted parasites (*Strongyloides*, *Trichuris*, hookworm and nematode larvae). Smaller home-range sizes and higher contact rates could favour this.

Another possible explanation can be found in the monkey's behavior. Since most of the directly transmitted parasites are soil transmitted, more frequent ground contact would also increase the risk of infection with these parasites. Terrestrial species may be at greater risk of acquiring parasites with a direct life cycle through fecal contamination of soil (Nunn *et al*. 2000). In chapter 4.4.2.2 the example *Leptospira* sp. infections in New World monkeys (Minette 1966) was already discussed. Primate arboreal ranging patterns were linked to avoidance of fecally contaminated pathways (Freeland 1980)

Since monkey groups in Kosumpee forest park spent most of their time on the ground, their contact rates with contaminated soil would be increased.

5.4.2.4. Interaction of parasite communities in the host

I could only found one correlation between parasite species in the present study. *Strongyloides fuelleborni* and *Trichuris* sp. were positively correlated with eachother. Positive correlations may occur because parasite taxa have their infective stages in the same environmental area, share the same route of transmission or in some other way be ecologically associated. *Strongyloides* and *Trichuris* are some of the most abundant parasites worldwide and are highly infective. Often they occur together in an individual; therefore the positive correlation of these directly transmitted parasites is not surprising. The indirectly transmitted intestinal fluke also correlates positively with both of these species. Since they normally neither share the same environmental area, nor the same route of transmission and are not otherwise ecologically correlated, there must be another reason for the observed interaction. If certain individuals are predisposed to parasite infections in general rather than to one particular parasite species, positive correlation may also result (Forrester *et al*. 1990; Keymer and Pagel 1990).

However, the occurrence and the type of interactions are far from clear and more studies are needed in this area.

5.4.2.5 Variation in prevalence

In the present study, the macaques which foraged near the human inhabited area and ingested human provided food differed markedly from the groups which had less overlap with humans and human-modified habitat. Human contact groups showed significantly higher prevalence and abundance of *Strongyloides fuelleborni* and of a minute intestinal fluke. The prevalence of *Strongyloides* in the human contact groups showed high levels of parasite infestation (30-70%), whereas the forest groups were much less infected (0-6.25%). The intestinal fluke was absent in the sylvatic groups, whereas in the human contact groups 15-30% of the individuals were infected.

My results support the prediction, that human contact groups harbor other parasites than sylvatic groups and that the parasite burden of human influenced and sylvatic groups differs significantly.

Differences in prevalence of *Strongyloides*

S. fuelleborni belongs to a genus of widely distributed nematodes parasitic in the intestine of humans and other mammals. It relies mainly on nonhuman primates for its hosts. Ashford and Barnish (1989) recommended that *S. fuelleborni* is the name to be used for *Strongyloides* infecting wild and captive Old World primates and humans from the same locations. Its life history pattern is basically the same as that of *S. stercoralis*, but the eggs are carried out of the host in the feces as opposed to *S. stercoralis*. Infection is assumed to occur when the infective L3 larvae penetrates the skin.

In my study, behavioral observations could show a positive correlation of *S. fuelleborni* infection with the time the macaques spent on the ground. As a consequence of foraging on human provided food, the daily activity and ranging patterns were different from those of the completely wild foraging macaques in the same area. The human contact macaques of my study spent more time on the ground than the wild foraging groups and, therefore, they seem to acquire the infective L3 larvae of *S. fuelleborni* more easily. Similar observations have been made in baboons. As a consequence of garbage foraging, daily activity and ranging patterns were different from those of completely wild foraging baboons (Altmann and

Muruthi 1988). Semiprovisioned baboons were resting significantly more and their home-ranges were significantly smaller (Altmann and Muruthi 1988).

S. fuelleborni infections can have deadly consequences being implicated in the deaths of rhesus monkeys (Remfry 1978). The pathological effects of *S. fuelleborni* (Flynn 1973) are divided into three phases. Firstly, the invasive phase when infective larvae penetrate the skin or the buccal mucosa. This phase does not appear to cause irritation (Remfry 1978). Secondly the migratory phase, when infective larvae are carried with the blood to the heart and lungs, break into the alveoli, enter the bronchi and are coughed up and swallowed, causing symptoms varying from a cough to bronchopneumonia. In the final and intestinal phase swallowed larvae penetrate the glandular epithelium of the small intestine, showing symptoms ranging from diarrhoea to peritonitis (Flynn 1973).

Ulcerations of the intestine were found in monkeys. Post mortem findings in a young rhesus male were a small abscess on the caecum, blood staining of omentum and peritoneal fluid and ulceration of the mucosal surface of the small and large intestines. In this case it was suggested that the peritonitis was caused by perforation of a *Strongyloides* induced ulcer (Remfry 1978). Although *S. fuelleborni* infections are natural to nonhuman primates, high prevalence and intensities may have negative consequences for a population of wild animals.

Prevalences of more than 70% in the macaques from Kosumpee forest park are therefore an alarming result, especially since the intensity of infection was also significantly higher than in sylvatic groups (for discussion see chapter 5.4.2.6).

Differences in prevalence of minute intestinal fluke

The second parasite of interest in my study is the minute intestinal fluke, probably *Haplorchis* sp., which was only prevalent in human contact groups. This parasite also infects humans in Thailand (as shown in this study), Indonesia and Laos (Kumar 1999). *Haplorchis* belongs to the so called foodborne trematode infections. Freshwater snails are suspected to be the first intermediate hosts of several human heterophyids and freshwater fish are the second intermediate hosts. Metacercariae are encysted in their tissues and human infection occurs via the consumption of raw fish which are infected with the metacercariae (Kumar 1999).

As already discussed earlier in this chapter, my observations of this parasite in macaques from both study areas implie that macaques come into contact with raw or undercooked food. My results lead to the conclusion that human provided food is the decisive factor which influences primate infection with this parasite. Behavioral observations could show a positive correlation between this trematode infection and the amount of human provided food in the diet of the macaques. Since the wild foraging macaques were not infected with this parasite, one has to conclude that within the human provided food intermediate hosts of the minute intestinal fluke can be found. Similarly, infections of baboons with tuberculosis from eating contaminated meat are also known (Keet *et al*. 2000; Sapolsky and Share 2004).

To decrease the chance of long-tailed macaques becoming agricultural pests forest parks, such as the two study areas, were established, where tourists are allowed to feed the primates (Son 2004). Where long-tailed macaques come into contact with tourists at nature reserves, up to 22% of their diet can be from provisioned food (Lucas and Corlett 1991; Son 2003). But this strategy is not without risk for both humans and macaques. When humans feed long-tailed macaques, both contact and non-contact aggression increases within and between groups at the same feeding sites, which can result in serious injuries or death (Wheatly 1991). Study animals in both study areas showed a considerable number of clearly visible scars which could be evidence for this (pers. observation). Barbary macaques at Gibraltar, for instance, had 44 contacts with tourists per hour (O'Leary and Fa 1993). This not only led to attacks on and severe laceration in tourists, but also to a viral epidemic which hit the macaque population in 1987, resulting in the death of all infants (O'Leary and Fa 1993).

Knowledge of the possible pathogenicity of the minute intestinal flukes in nonhuman primates is intermittent. But the knowledge of pathological effects of intestinal flukes in humans leads to the conclusion that there is a possible health risk for the monkeys. Sukontason and colleagues (2005) presented the pathology in the small intestine for three humans, caused by *Haplorchis taichui*. Microscopic examination revealed mucosal ulceration, mucosal and submucosal haemorrhages, fusion and shortening of the villi, chronic inflammation, and fibrosis of the submucosa. This finding clearly shows that *H. taichui* is pathogenic. It should be noted that several species of heterophyid flukes, including *Haplorchis* spp., cause erratic parasitism in humans, which is often fatal (Africa *et al*. 1940). The three most frequently affected sites are the heart valve, brain and spinal cord, where eggs and adult flukes originating from the intestinal mucosa embolized in the blood vessels (Africa *et al*. 1940).

Eggs of *Heterophyes heterophyes* were found encapsulated in the brain of patients with neurological symptoms (Zhang and Fan 1990).

Conclusions from variations in prevalence

My results from the study in Thailand, as with the results from Peru discussed above, lead to the conclusion that the presence of humans and an environment modified by humans can lead to substantial changes in the community structure of intestinal helminths in wild macaques. Humans play a role in parasite transmission not necessarily by introducing novel pathogens directly into wild host populations or by transmitting their parasites to primates, but because human activities can alter the ecology of wildlife and environmental parameters in ways that increase the probability of an infection.

Human encroachment and habitat degradation are increasing (Lilly *et al*. 2002) and with the modification of wildlife habitat humans may also alter the behavior and diet of primates (Weyher *et al*. 2006). The expansion of human populations affects the behavioral ecology of a number of primate species with many changes attributed to the availability of anthropogenic nutrition in the form of crops, waste food in garbage dumps and tourist feedings (Box 1991; Else and Lee 1986).

Primates living in close proximity to humans have been observed to forage in garbage dumps, and this could expose them to infected food items or human fecal matter. Changing foraging strategies may alter the relationship primates have with the parasites already present in their environment. Consuming human foods can have significant epidemiological costs due to an increased risk of disease transmission for both, humans and primates. The transmission can occur both directly from the other species and indirectly via contact with their bodily waste, food, rubbish and domesticated animals (Dittus 1974; Eley *et al*. 1989; Hahn *et al*. 2003; Wallis and Lee 1999).

Some studies have documented the costs to baboons from foraging in human garbage dumps, including infections with antibiotic-resistant bacteria, probably acquired from humans (Rolland *et al*. 1985; cf. Routman *et al*. 1985), infections with tuberculosis from eating contaminated meat (Keet *et al.* 2000; Sapolsky and Share 2004), and a greater incidence of cavities and periodontal diseases (Phillips-Conroy *et al*. 1993). In contrast, Eley *et al*. 1989

found no increased risk of acquiring gut parasites from garbage dump foraging and also Hahn and colleagues (2003) could not find any evidence that baboons foraging daily in human garbage pits acquired parasites that exhibit a high prevalence in local human populations.

Long-tailed macaques are already established outside their natural range and quickly adapt to human modified landscapes. Their distribution pattern in Thailand at present is similar to that determined 30 years ago (Malaivijitnond *et al*. 2005). However, because of the invasion and disturbance by humans, their natural habitats have changed from natural forests to temples or parks close to human settlements. In addition to the macaque population explosion in these areas, the monkeys foraging behavior has also changed. In disturbed areas near human settlements, they quickly learn to raid gardens or crops and beg for food from humans or search through garbage for food (Lucas and Corlett 1991). They are also known to enter houses and steal food if humans are not there to frighten them (Gurmaya *et al*. 1994).

Such a changed foraging behavior seems to be responsible for the observed differences in parasite burden in my study animals. Human contact groups in both parks are used to human provided food, they seem to prefer human provided food instead of their natural food sources. And, as already discussed, these changes seem to have costs for the macaques by acquiring potentially pathogenic parasites.

5.4.2.6 Variation in egg/larvae output

Living close to humans and human modified habitat also leads to differences in egg/larvae output in the studied macaque groups. The prediction was that the egg/larvae output of parasites is higher in primate groups living next to human settlements, since human altered habitat offers conducive conditions for the parasite encounter. Like in the peruvian study, the results of my work support this hypothesis.

Egg/larvae output was used as an indirect measure of abundance and intensity of helminth infection. However, the number of parasite eggs in host fecal material is affected by many factors (discussed in chapter 3.3.).

Variation in abundance

Abundance varied in four parasite morphospecies: *S. fuelleborni*, *Trichuris* sp., minute intestinal fluke and *Oesophagostomum* sp.. Groups living in contact with humans showed a higher abundance of *S. fuelleborni* and intestinal fluke infection. *Trichuris* had a higher abundance in Kosumpee forest park macaques. These macaques showed lower abundance in *Oesophagostomum* infections. The results are not surprising remembering the differences in parasite prevalence, especially since intestinal fluke and *Oesophagostum* were absent in some of the investigated groups. However, the results highlight the differences between human influenced and sylvatic study groups.

Variation in intensity

The number of *Strongyloides* and *Trichuris* eggs in the feces of infected macaques was considerably higher in the groups from Kosumpee forest park, the groups with the most intense contact to humans. In the case of *Strongyloides*, one has to remember that only one individual in the studied sylvatic groups was infected and this individual only harbored one egg in 100µl fecal sediment. Compared to more than 3000 eggs in individuals from Kosumpee forest park: this is a highly interesting result.

Both, *Strongyloides* and *Trichuris* are soil transmitted and higher ground contact would also increase the risk of infection with these parasites (see chapter 5.4.2.3 and 4.4.2.2 for discussion). The meaning of intensity for parasite pathology was already discussed in the Peru section. Heavy infections of *Strongyloides* sp. are associated with mucosal inflammation, ulceration, dysentery, weight loss and death (Chapman *et al*. 2006). Secondary bacterial infections of mucosal lesions resulting in ulceration and fatal septicaemia are frequent complications (Soulsby 1982).

My results contradict the hypothesis that consuming human foods provides free-ranging primates with significant nutritional benefits (Else and Lee 1986; Fa and Southwick 1988), which in turn may lead to a decrease in intensities of parasitic infections (Eley *et al*. 1989).

5.4.2.7 Summary

In the present study, significant differences could be shown between wild macaque groups living in human influenced areas and groups living in undisturbed forest patches. The divergences observed in parasite community structure in the macaques are not related to the transfer of parasites between humans and primates.

My results provide a clear evidence that human alteration of the habitat results in changes in monkey foraging behavior in such a way that leads to acquisition of other parasites than in a sylvatic environment. Increased ground contact and human provided food seem to be the main factors influencing the differences in parasite diversity and intensity between contact and non-contact groups. These substantial changes in the parasite communities may have potential pathogenic consequences.

Chapter 6

General Discussion

Various studies have suggested that parasite transmission may be occurring between nonhuman primates and humans. Based on coproscopic examination, they report nematode stages consistent with *Trichuris trichuria*, *Strongyloides fuelleborni* and *Oesophagostomum* spp. from humans (Muriuki *et al*. 1998; Rothman and Bowman 2003; Jones-Engel *et al*. 2004; Legesse and Erko 2004; Philipps *et al*. 2004). But such interpretations can be misleading. Parasites are only identified and distinguished on the basis of morphological features, the host they infect, their pathological effects on the host and their geographical origin. These criteria are frequently insufficient for species identification and diagnosis as shown in the example of *Oesophagostomum bifurcum*, which was genetically different between primates and humans (de Gruijter *et al*. 2005; Gasser 2009).

Despite previous findings suggesting parasite transmission, my results suggest this is not common. Both studies presented in my thesis could not detect a parasite transmission from humans to nonhuman primates or vice versa. Although all study areas presented high risk interfaces including body contact between humans and primates, the parasite communities of humans and monkeys were different. However, both studies show that the presence of humans influences the parasite community in nonhumans primates significantly. My work shows clear evidence that humans alter the habitat and therefore the behavior of primates. These substantial changes lead to the acquisition of other parasites than in the natural sylvatic environment of the monkeys.

Humans therefore pose a risk to primate health by changing their environment. Habitat changes can alter the species composition within the area, which can have significant effects on parasite species, especially when their life cycles depend on intermediate hosts. My studies showed that variations between human contact and sylvatic groups were mainly based on indirectly transmitted parasites. Human presence may change the intermediate host composition in an area and may also introduce new vectors. Living close to humans often causes changes in the behavior of the monkeys, which can lead to new risks for the primates, for instance by increasing contact with contaminated soil or contaminated food.

The substantial changes found in the parasite communities may have potential pathogenic consequences which are, at the moment, unclear. However, in both study areas human contact increased the prevalence of parasite infections, which are known to have severe pathological effects either in monkeys or in humans. These parasites, *Prosthenorchis elegans* in Peru, *Strongyloides fuelleborni* and minute intestinal flukes in Thailand, are even known to cause

death. In combination with the elevated intensities of *P. elegans* and *S. fuelleborni* infections, negative consequences are probable.

6.1 Disease risk and anthropogenic influence

Many factors may contribute to the ability of infectious agents to cross the species barrier, including characteristics of the host, the pathogen and the environment. Transmission may be facilitated by proximity, the degree of physical contact between animals and humans and the ability to be inoculated in the new host (Woodford *et al*. 2002; Conly and Johnston 2008). It is also well known that changes in pathogen incidence and pathogen range expansions can result from natural processes, such as seasonal and long-term climatic cycles (Patz *et al*. 2008). In the present day, however, evidence for a human role in causing disease outbreaks has increased substantially. Humans can exert influence on wildlife health in many different ways and recent studies have directly associated a number of human behaviors with the emergence of zoonotic diseases, directly or indirectly. They can directly introduce novel pathogens into wild host populations but alter environmental parameters in ways that increase the probability of disease emergence (Schrag and Wiener 1995; Holmes 1996; Daszak *et al*. 2000; Chapman *et al*. 2005a).

For instance, pet and domestic animals can transport pathogens into wildlife populations. It is known that parasites can be transported by game bird releases from farms to field (Millán *et al*. 2004) and eventually affect endangered bird species. This has been suggested in the case of a capillary nematode (*Eucoleus contordus*) normally infecting a tropical partridge but now found in a little bustard in central Spain (Villanúa *et al*. 2007). Diseases shared between wild caprine species (such as chamois and ibex) and domestic sheep and goats have important consequences for wildlife numbers and animal welfare. Sarcoptic mange (*Sarcoptes scabei*) and *Mycoplasma conjunctrae* affect several populations of ungulates in Europe and are suggested to spread from domestic livestock to wildlife(e.g. Rossi *et al*. 2007).

Although in both of my study areas, pet (Thailand) or domestic animals (Peru, Thailand) lived within the home ranges of the study animals, parasitic infections seemed not to be influenced by them. None of the parasites found to be different between human contact and sylvatic groups is known to be associated with pet or domestic animals (Schnieder and Tenter 2006).

However, in both study areas, contact between monkeys and these animals is quite limited and a cross-transmission was not suspected at all.

Human habitat modification, however, outranks the direct introduction of novel pathogens by far. Habitat modification is the leading global cause of species extinction and alterations in population dynamics, and is a particular threat to biodiversity in extremly diverse tropical ecosystems (Foley *et al*. 2005). Physical changes in landscapes may alter the abundance and distribution of species (Saunders *et al.* 1991). Higher order effects can occur when modifications in ecological processes result in altered interspecific interactions and subsequent changes in a species' abundance or persistence (e.g. Taylor and Merriam 1996). These effects may be direct, with one species affecting another through predation, herbivory or parasitism or indirectly as a result of the persistence of a third species (Strauss 1991; Wootton 1994). Within fragmented landscapes, predation is not the only higher order effect that might influence the abundance and distribution of resident species. Parasite host relations can also be altered, especially when complex life-cycles are involved.

The dynamics of parasite life-cycles may be affected by landscape alterations, especially if interactions between parasites and hosts are changed. Encounter rates and the prevalence of resulting parasitic infections may change, or potential hosts may have altered their behavior and movement patterns in fragmented landscapes (Taylor and Merriam 1996). When host species differ in behavior or movements patterns as a result of landscape modifications, interactions between species of defintive hosts and intermediate hosts change, and rates of parasite transmission may be diverse (Taylor and Merriam 1991).

My results show, that altered behavior patterns led to higher prevalences of *Strongyloides fuelleborni* infection in macaques from Thailand. While wild foraging macaques lived mainly arboreal, the human associated groups spent significantly more time on the ground, where infective stages of *S. fuelleborni* accumulate. Similarly, landscape alteration may be responsible for high prevalences of *P. elegans* infections in Peru, by creating an environment favoring the intermediate host.

Global conversion of natural habitats to agriculture has led to marked changes in species diversity and composition (Tylianakis *et al*. 2007; Tilman *et al*. 2001). However, it is less clear how habitat modification affects interactions among species (van der Putten *et al*. 2004). Tylianakis *et al*. (2007) analyzed quantitative food webs for cavity nesting bees and their parasitoids. In modified habitats there was a higher ratio of parasitoid to host species and

increased parasitism rates with implications for the important ecosystem services such as pollination and biological control that are performed by host bees and wasps (Losey and Vaughan 2000). The most abundant parasitoid species (*Melittobia acasta*) was more specialized in modified habitats (on *Anthidium* sp.), with reduced attack rates on alternative hosts (Tylianakis *et al*. 2007).

Such changed species interactions may be responsible for the differences in *P. elegans* and cestode B prevalences found in Peru. Human altered landscape may favor the intermediate host species of *P. elegans* and therefore lead to smaller abundances or even extinction of the intermediate host species from cestode B due to competition.

Changes in agricultural practices have been associated with the incidence of haemorrhagic fevers (reviewed by Morse 1995). For instance, conversion of grasslands to maize cultivation favored a rodent that was natural host for Junine virus, the cause of Argentine haemorrhagic fever and human cases increased with the expansion of maize agriculture (reviewed by Morse 1995). Urbanization favouring the rodent hosts has been linked to outbreaks of Lassa fever in humans (reviewed by Morse 1995). Road building, tree felling, reduced shade and increased pooling of water were shown to promote breeding and more rapid development of the vector mosquito larvae (Afrane *et al*. 2005, de Castro *et al*. 2006). The development of water control systems has increased the transmission of Rift-Valley fever, which is transmitted by a mosquito vector. In the outbreaks of Rift Valley fever in Mauritania in 1987, the human cases occurred in villages near dams on the Senegal River (reviewed by Morse 1995). The same effect has been documented with other infections that have aquatic hosts, such as schistosomiasis. (reviewed by Morse 1995).

The results of my study fit very well to the previous examples. In Peru, human alteration of the habitat seems to be favouring intermediate hosts of *Prosthenorchis elegans* leading to increased parasite prevalences in tamarins. Since, however, the intermediate host of *P. elegans* in this area is unknown, the manner by which these intermediate hosts are favored is unclear. Food supply or not properly covered waste may favour human associated beetles and cockroaches (Burgess *et al*. 1973; Graczyk *et al*. 2005). But also the conversion of forest for agricultural use may increase populations of some arthropod species (Rasplus and Roques 2010).

Land-use change, as described before, also implies habitat destruction. In the early stages of habitat destruction, the number of fragments increases and their size decreases (Bascompte

and Solé 1996). This can result in both positive and negative outcomes for infectious disease risk. On the negative side, habitat fragmentation increases edge effects (Cowlishaw and Dunbar 2000). A recent field study of two African colobine monkeys found that the individuals on edges of forest fragments were more likely to be infected with multiple species of gut parasites compared to monkeys in the interior of these fragments (Chapman *et al*. 2006). Goldberg *et al*. (2008) found that forest fragmentation increases bacterial transmission between primates, humans and their livestock. Bacteria from humans and livestock in three forest fragments were more similar genetically to bacteria from primates in these fragments than to bacteria from nearby undisturbed forest locations. Moreover, the degree of disturbance paralleled the degree of similarity. Transmission of *Escherichia coli* from primates to humans was as likely as transmission the other direction. Similarly, Gillespie and Chapman (2006) found that the degree of disturbance of a fragment (measured as the density of tree stumps) was an accurate predictor of prevalence of infection of red colobus monkeys with nematodes.

Beside the introduction of novel pathogens and creating better conditions for vector species, human activities also have an influence on endangered and unmanaged wildlife, as the loss of certain habitats or food resources cause different species to exploit alternative options (e.g. Routman *et al*. 1985; Lucas and Corlett 1991; Tortosa *et al*. 2002; Blanco *et al*. 2007). Mechanisms such as habitat loss or climate warming can directly influence patterns of biodiversity (e.g. Forister *et al*. 2010), but little is known about their indirect consequences for host-pathogen dynamics. Habitat loss can change the behavior and abundance of wildlife in ways that influence parasite spread; human activities that crowd and subdivide populations influence patterns of disease risk and host susceptibility (Chapman *et al*. 2005a, b, 2006). The Iberian lynx, for instance, was found to be feeding on tubercolosis infected carnivores (Perez *et al*. 2001), storks and kites foraging on rubbish dumps (Tortosa *et al*. 2002; Blanco *et al*. 2007).

Macaques have the ability to adapt quickly to human changed landscapes (Crockett and Wilson 1980; Sussmann and Tattersall 1986). When they live in human influenced habitats, they often change their foraging behaviour. Instead of natural foods they are used to human associated food (e.g. from raiding crops, begging humans; Lucas and Corlett 1991). In my study, alternative food sources led to the infection of macaques with the minute intestinal fluke. Since intermediate hosts of these parasite species are not available within the natural habitat of the macaques, they could only be obtained from the human provisioned food. This

result clearly shows that primates can acquire parasites from humans, although cross-transmission did not occur.

My study highlights the risk which is indirectly posed by humans to wild animals. Small changes in natural habitats can have unpredictable outcomes influencing much more than the small area which has been changed.

6.2 Parasites and their impact on the host's survival

It is difficult to measure the actual impact of parasites on the survival of wild hosts. When disease occurs in free-ranging animal populations it is often hard to diagnose the illness, let alone identify the origin of the problem (Wallis and Lee 1999). This is also the case in wild nonhuman primates. Signals of disease are rarely observed in free-ranging primates. Infected individuals often mask their weakness to maintain social position and avoid the attacks of predators (Alados and Huffmann 2000; Boesch and Boesch-Achermann 2000; Lonsdorf *et al*. 2006).

Nevertheless, diseases are an important population regulation factor and human activities play an important role in disease epidemiology (Anderson and May 1991). The negative effects of parasites on host fitness from an area with a high density of grazing domestic sheep could be shown in red deer (Zaffaroni *et al*. 1997). Several empirical and theoretical studies suggest that either a parasitic nematode or a virus of low to intermediate pathogenicity might be used as a control agent for rats, goats and other introduced species (cf. Dobson 1988). For example, *Entamoeba invadens* may be a biological control agent for the invasive brown tree snake *Boiga irregularis* on Guam (cf. Dobson 1988). In a laboratory study of mouse population dynamics (Scott 1987) the parasitic nematode *Heligmosomoides polygyrus* considerably reduce the density of its host population.

There is a large body of empirical evidence demonstrating the negative fitness consequences of parasitic infections (reviewed in Nunn and Altizer 2006), which include sickness, compromised nutritional status, suppressed immunity, decreased fecundity and death. Although mild infections may have little effect on the host, negative effects increase with the intensity of infection or with the parasite species richness (Nunn and Altizer 2006).

Despite dramatic examples such as Ebola (see chapter 2.) the majority of primate pathogens probably only have chronic, sub-lethal effects on primates in the wild (Nunn and Altizer 2002). Macroparasite infections are often long-lasting and cause morbidity rather than mortality, and immune responses do not confer complete protection against reinfection (Maizels *et al*. 1993). Severe infections with these or other gut parasites are likely to cause diarrhoea, emaciation and malaise (Orihel and Seibold 1972; Flynn 1973).

Morbidity and mortality are often associated with multiple-species infections. For example, in humans *Schistosoma mansoni* has an increased effect on the development of malnutrition in the presence of *Trichuris trichiura* (Parraga *et al*. 1996) and a range of parasites demonstrated elevated pathogenic effects in the presence of HIV (cf. Chapman *et al*. 2006). Most deaths and extreme pathology resulting from parasitic worms occur when hosts harbor large numbers of parasites (Flynn 1973; WHO 1998b) or when parasites migrate to an organ outside their usual habitat within the host (Orihel and Seibolt 1972). Constant exposure to parasites may have lasting consequences on the survival of wild primates (Stoner 1995; Oluput and Chapman 2004; Gillespie 2006; see Kowalewski and Gillespie 2008).

In the present study different metrics, namely egg/larvae output, parasite species richness (PSR) and prevalence were used for evaluating the disease risk for host individuals or troops. Prevalence is related to the probability of infection for the host individuals, while the number of different parasite species (PSR), while the egg/larvae output are more strongly associated with the outcome of infection and the probability of morbidity or mortality (Chapman *et al*. 2005b). However, to evaluate the actual impact of parasites on the host's fitness the potential pathogenicity of each single parasite species and their interactive effects should be taken into account (Petney and Andrews 1998). Infection with one highly virulent parasite species may have a more detrimental effect on the host than the infection with several other, less virulent ones. Since the manifestation of disease symptoms depends on the interaction of many host- and parasite-intrinsic factors (Bush *et al*. 2001) evaluating parasite pathogenicity is particularly difficult.

Our knowledge of the pathological impacts of parasites is often based on laboratory studies. Results obtained from these, however, can not easily be transferred to wild populations (Randolph and Nuttall 1994). Laboratory animals may suffer from additional stress, crowding and lack of appropriate nutrition and genetic similarity. Therefore pathologies might be more severe. On the other hand, hosts in captivity might present less severe pathologies because

inter- and intraspecific competition for food resources is reduced. This is important since the same parasite species might be almost non-virulent or can cause severe pathologies, depending on the conditions influencing host and parasite (Brack 1987; Toft and Eberhard 1998). This can result in an enormous variation of the impact on host fitness and the regulation of host populations.

The three host species studied in my thesis are not endangered and no unusual outbreaks of any particular disease were observed during the study period or are known from previous years. However, events such as the unexpected incidence of canine distemper in the Serengeti in 1994, which is known to have killed approximately 85 of the 250 monitored lions (Miller 1994; Morell 1994a, b), show unmistakably how suddenly the occurrence of disease can change host population structure and also that disease must be considered in any conservation effort. As long as the monkeys are in otherwise normal health, most of the parasites are not severely pathogenic, but with a lowered resistance due to stress or other infections, parasites can then cause disease and sometimes even death. For instance, *Oesophagostomum* spp. and *Strongyloides fuelleborni* have both been implicated in the deaths of rhesus monkeys (Remfry 1978). The effects would be particularly serious for endangered species, for example the highly endangered relatives of the tamarins studied here, such as the cotton-top tamarind (*Saguinus oedipus*) or the pied tamarind (*Saguinus bicolour*), which are classified as critically endangered on the IUCN Red List of threatened species (IUCN 2010).

So how do studies such as the present one relate to conservation? The host species in this study illustrate a conservation issue, the problem of human contact. Primates, because of their phylogenetical relatedness to humans can be endangered by human diseases as illustrated by polio epidemics in Gombe Stream National Park (Goodall 1986) and others (see chapter 2 for review). However, in this study there was little indication that tamarins and macaques suffer from infections as a direct result of living close to humans. Instead it highlights the importance of considering both the direct and the indirect influences of human communities, including communities of researchers, on primate conservation in the context of disease. In terms of conservation, this indicates that areas designated for the protection of primate species should be placed far enough from human settlements to prevent foraging on disturbed land and that food provisioning of humans should be controlled more effectively in areas like forest parks.

6.3 Seasonality of the results

Although the study was only conducted during the dry season, results may also be valid for the rainy season. A previous research in the Peruvian study area showed that primates were infected with a similar spectrum of parasites during dry and wet seasons (Müller 2007). In addition, many intestinal parasites that infect primates are known to have a life span or a persistence of infection of over one year up to 25 years, e.g. hookworms, *Strongyloides stercoralis*, *Trichostrongylus* spp. and *Hymenolepis nana* (Ash and Orihel 1987; Bethony *et al*. 2006). Once the infection is established and the host's defences or self-medication do not expel the parasites, the effects of seasonally varying exposure risk might not be measurable in terms of PSR and prevalence.

The nutritional status of primates is often thought to have an influence on the immune status and therefore on parasite infection rates in primates (Holmes 1993; Coop and Holmes 1996; Koski and Scott 2001). The overall nutritional status of the studied hosts should be better in the rainy season because of higher food availability. Müller (2007), however, could not show that the inferred better nutritional status in this season actually coincides with a lower PSR, prevalence or egg/larvae output. Müller suggested that "the lack of seasonality in PSR and prevalence in the study species might be due to less pronounced immunosuppression in the dry season. The degree of malnutrition might be lower than expected because the primates can circumvent bottlenecks in fruit scarcity by shifting their diet to other food plants or components to fulfil their energy demands (Müller 2007). In addition, overall immune status in peruvian tamarins might be less affected because energetically costly periods like gestation, lactation and infant carrying fall into the period of maximum fruit availability (Soini and Soini 1990; Löttker *et al*. 2004)

Infective parasite stages in the environment can be cleared away by heavy rain (Nunn and Altizer 2006). In case of the study areas in Thailand, both parks were flooded during the rainy season (Mücke, pers. observation, and the infective fecal material was washed away with the floods. Accumulation on the ground, as during the dry season, was therefore not possible. In addition, during the rainy season, food provisioning from humans was limited. Furthermore, the activity of egg destroying organisms is enhanced in humid and warm seasons (Hausfater and Meade 1982; Larsen and Roepstorff 1999).

Nevertheless, possible variation in parasite infection between wet and dry seasons should be investigated further.

6.4 Directions for further research

Wildlife and humans will increasingly interact in the future as the human population grows and people continue to explore and colonize previously uninhabited habitats. Since it is unlikely that this kind of expansion will be stopped, it is important to determine the impact of human activities on wildlife and to take steps to minimize this impact.

Increased monitoring and field data are badly needed to evaluate the mechanisms by which human activity changes the dynamics of primate parasite interactions. Although there are several studies known on parasites in wild primates (see chapter 2), it is still important to conduct more research to establish baseline data. Especially coprological surveys of wildlife near human settlements are needed to monitor unusual outbreaks of parasitic infections. In this context, periodic coprological surveys of human populations living near wildlife are also important, to control for parasitic diseases and to ensure treatment in a timely manner.

As mentioned above possible variation in parasite infection between wet and dry seasons should be investigated further. My results can not exclude parasite cross-transmission between humans and primates completely. Since interactions can occur in such different ways (sharing the same habitat, food and water sources, having body contact, holding primates as pets, providing food etc.) more field studies are needed to obtain an more exact picture of the risk of parasite cross-transmission.

Long-term field studies are needed to quantify the relative impact of parasites on wild populations. The work on grouse in Scotland is one of the few good examples of such a project (Hudson 1986; Hudson and Dobson 1990; Dobson and Hudson 1992; Hudson *et al*. 1998; see chapter 2). Such studies could yield more information if individually known animals were monitored, making it possible to relate parasite burden to reproductive success, for instance, and to shed additional light on the parasite's role in controlling host abundance. Continuing to monitor parasite infection in the tamarin and macaque populations studied here could provide more information on the fluctuations of parasites in relation to changes in host population.

In order to investigate the important questions in the field of host and parasite biology, collaborative efforts from different biological disciplines are necessary. To gain a better understanding of host-parasite interactions more genetical studies are needed. The problems of helminth identification described in this study (chapter 3) were tackled by exact measuring of representative numbers of parasite stages and by a detailed literature review. For future studies it would be desirable to apply molecular techniques to confirm and enhance morphological parasite identification. So far, a problem in determining helminth genotypes within or among host-populations is the acquisition of sufficient adult worms from several hosts. For certain parasite species techniques like the Polymerase chain reaction (PCR) make it possible to distinguish between different genotypes of parasites by the analysis of their eggs (de Gruijter *et al*. 2005; Gasser *et al*. 2009). This technique can be employed to show whether the genotype of a parasite species varies within one area or between host populations. For instance genetical analysis can show whether the parasite found in primates and humans is the same strain or whether different species are involved. This would greatly enhance our understanding of human-wildlife disease ecology. However, the accurate identification especially of rare parasites in wild hosts will remain a challenging task in the future.

Based on the results of this study, gaining a better understanding of possible intermediate hosts is essential. In this context, for example, the life cycle of *Prosthenorchis elegans* in and around the EBQB should be investigated, and in Thailand, detailed studies on human provided food would be helpful.

In addition, educating people, especially children, about proper hygiene and the risk of disease transmission from and to wildlife could reduce the risk of zoonoses passing between humans and nonhuman animals. The control of potential anthropozoonotic transmission represents a serious management challenge. Avoidance of human contact, education in hygiene and the medical treatment of local communities around park boundaries limit the possibility of pathogen exchange.

As the rate of emerging infectious diseases (in humans and animals) is likely to increase with anthropogenic pressure (Epstein and Price 2009), it is important that we develop a better understanding of the potential for and occurrence of both cross-species pathogen transmission, as well as the effect of land use change on parasite ecology, in order to successfully protect humans, animals and ecosystem health.

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Appendices

1. TREMATODA

Parasite species	Size in µm	Morphology	Host species	Host origin	Intermediate Hosts	Pathogenicity	References
A. Diplostomidae							
Neodiplostomum tamarini			*S. nigricollis* *S. fuscicollis* *Saguinus* sp.		not known, molluscs insects, amphibians, reptiles or fish	unknown	13;25;42;61
Neodiplostomum sp.			*Saguinus* sp.				11;48;61
B. Echinostomatidae							
Artyfechinostomum sp.			*Macaca* sp.			enteritis	7;11;25;46
Echinostoma aphylactum			*S. geoffroyi* *Saguinus* sp.				47;49;74;75
Echinostoma ilocanum	88-111x53-74		*Macaca* sp.				7;49;53
Echinostoma malayanum	120-130x80-90		*M. fascicularis*		freshwater snails (*Indoplanorbis exustus, Gyraulus convexiusculus)* gastropods *(Pila scutata, Lymnaea cumingiana, Digionostoma pulchella*)	diarrhea, vomiting, anorexia	7;10;49;53
Reptiliotrema primata			*M. fascicularis*				11;25;46
C. Dicrocoeliidae							
Athesmia foxi	17-21x27-34	ovoid, thick shell, operculum	*S. oedipus* *S. sciureus* *S. fuscicollis* *S. nigricollis* *S. geoffroyi* *Saguinus* sp.	c, w	not known, molluscs insects, amphibians, reptiles or fish	non-pathogenic, obstruction and inflammation of bile ducts	11;13;22;25; 32;46;61;68; 71;74;75;81
Athesmia heterolecithodes	17-21x27-34	ovoid, thick shell, operculum	*S. mystax* *S. labiatus* *S. geoffroyi*	c	not known, molluscs insects, amphibians, reptiles or fish	non-pathogenic	22;25;32;54; 61;68;71;74;75
Athesmia sp.			*Saguinus* sp.				11;46;48

Parasite species	Size in µm	Morphology	Host species	Host origin	Intermediate Hosts	Pathogenicity	References
Brodenia sp.	42-46x 24	elipsoid, operculum	*M. nemestrina*	l			7;11;25;30;46; 49
Dicrocoelium colobusicola	42-46x 24	elipsoid, operculum	*Macaca* spp.	l			7;11;30;46;49
Dicrocoelium dendriticum	36-45x22-30		*Macaca* sp.	c, l	cholangitis		11;25;65;70
Eurytrema pancreaticum			*M. fascicularis*		*Bradybaena,* grasshoppers		7;49;49
Eurytrema satoi			*Macaca* spp.				7;11;25;46;53
Platynosomum amazonensis	20-35x34x50	ovoid, thick shell, operculum	*S. nigricollis* *S. mystax* *S. fuscicollis* *Saguinus* sp.	w, i	not known, molluscs insects, amphibians, reptiles or fish	unknown	11;13;25;32; 42;46;61;68; 71;75
Platynosomum marmoseti	20-35x34x50	ovoid, thick shell, operculum	*S. fuscicollis* *S. nigricollis* *Saguinus* sp.	i	not known, molluscs insects, amphibians, reptiles or fish	unknown	13;25;32;42;68
Platynosomum sp.			*Saguinus* sp.				11;61
Zoonorchis goliath	22-26x34-41	operculated	*S. geoffroyi* *Saguinus* sp.				35;42;46;74;75
D. Lecitodendriidae							
Phaneropsolus aspinosus			*M. fascicularis*	w			60
Phaneropsolus bonnei	23-33x13-18		*Macaca* sp.		dragonflies, damselflies		10;60
Phaneropsolus orbicularis	40	operculated	*S. fuscicollis* *S. mystax* *Saguinus* sp.	w, l	not known, molluscs insects, amphibians, reptiles or fish	unknown	11;13;25;32; 46;61;68;74; 75
Phaneropsolus oviforme			*Macaca* sp.				11;25;46;62
Phaneropsolus sp.			*Saguinus* sp. *S. fuscicollis*		not known, molluscs insects, amphibians, reptiles or fish		13;48;61
Primatotrema macacae			*M. fascicularis* *Macaca* sp.	c,l			7;11;25;46; 49;63;83
E. Plagiorchidae							
Plagiorchis multiglandularis			*M. mulatta*				7;46;49
F. Fasciolidae							
Fasciola hepatica			*M. irus* *Macaca* sp.				11;25;34;46

Parasite species	Size in µm	Morphology	Host species	Host origin	Intermediate Hosts	Pathogenicity	References
G. Opistorchiidae							
Clonorchis sinensis	27-35x11-20	oval operculated	*Macaca* sp. *M. fascicularis*	l	snails (*Bithynia* spp.), fish	inflammatory reaction, epithelial hyperplasmia	26;45-47;80
Opisthorchis felineus	30x11	oval operculated	*M. fascicularis*		snails (*Bithynia* spp.), fish	proliferaion of the biliary tract	46;80
H. Heterophyidae						cirrhosis	
Haplorchis pumilio	28-31x16-18		*M. fascicularis*		freshwater snails, fish	diarrhea, ulceration, inflammatory reaction	9;18;27
Haplorchis yokogawai	29-30x15-17		*Macaca* sp.		freshwater snails, fish	diarrhea, ulceration, inflammatory reaction	5;7;18;27;53
Metagonimus yokogawai	27-28x16-18		*Macaca sp.*		freshwater snails, fish	diarrhea, ulceration, inflammatory reaction	11;18;23;27
Pygidiosis summa			*M. fascicularis*				46
I. Microphallidae							
Spelotrema breviacaeca			*M. fascicularis*				46
J. Paragonimidae							
Paragonimus westermanii		partly flattened operculum, oval	*M. fascicularis*		snails, crabs, crayfish		11;25;34;68
K. Schistosomatidae							
Schistosoma japonicum	114-180x45-60	lateral spine	*M. fascicularis* *M. mulatta*	c	snails	hemorrhagig diarrhea, ascites	11;35;46;
L. Paramphistomidae							
Chiorchis noci			*Macaca* sp.				11;25;46
Gastrodiscoides hominis	150x60-70		*M. mulatta* *Macaca* sp.		snail	diarrhea, colitis, enteritis	11;25;34;46; 56;68;81
Watsonius watsoni	122-130x75-80		*Macaca* sp.		probably snails	diarrhea, enteritis	11;25;46;68
Watsonius macaci			*Macaca* sp.				11;25;46;68

2. CESTODA

Parasite species	Size in µm	Morphology	Host species	Host origin	Intermediate Hosts	Pathogenicity	References
A. Anoplocephalidae							
Atriotaenia megastoma	egg: 25-41x 33-45 oncosphere 24-33x30-41		*S. nigricollis* *Saguinus* sp.	w, c	insects, coleoptera	enteritis	20;25;32;38; 42;48;57;75
Bertiella studeri	egg: 46x50 oncosphere 18-20	thin shell, irregular ovoid contour	*Macaca spp.*	w	oribatid mites experimentally: *Galumma, Scholoribates laevigatus*	unknown	25;31;37;41; 44;68
Bertiella mucronata	egg: 46x50 oncosphere 18-20	thin shell, irregular ovoid contour	*S. leucopus*		oribatid mites	unknown	25;53
Bertiella okabei			*M. fascicularis*				66
Bertiella sp.			*M.fuscata*				34;53
Mathevotaenia sp.	egg: 25-41x 33-45 oncosphere 24-33x30-41		*Saguinus* sp.		coleoptera, lepidoptera *Tribolium* sp.		27;42;48;57; 75
Paratriotaenia sp.			*S. fuscicollis* *S. leucopus* *S. oedipus* *Saguinus sp.*	w, i, l	insects	unknown	6;13;25;42;48; 57;69;75
B. Hymenolepididiae							
Hymenolepis cebidarum	egg: 68 oncosphere: 30x27 hooks 16		*S. nigricollis* *Saguinus* sp.	c	unknown	unknown	5;20;25;42 48;75
Hymenolepis diminuta	60-83x52-81	sphaerical	*S. geoffroyi* *Saguinus* sp.	c	beetles, fleas, mealmoth arthropods	rarely enteritis and abscessation of lymph nodes	6;25;32;48;54; 57;68
Hymenolepis nana	30-35x44-62	oval	*M. mulatta*	w	flour beetles, fleas or no intermediate hosts		25;68;78

3. NEMATODA

Parasite species	Size in µm	Morphology	Host species	Host origin	Intermediate Hosts	Pathogenicity	References
A. Trichinelloidea							
Trichuris sp.	22x54		*M.fuscata*	c	no intermediate host	typhlitis, colitis, enterits secondary infections	2;25;59;68;75
B. Gnathostomatidae							
Gnathostoma weinberg			*Saguinus* sp.				61
Gnathostoma sp.			*Saguinus* sp.				61
C. Oxyuridae							
Enterobius vermicularis	50-60x20-30	slightly flattened at one side	*M. fascicularis*		no intermediate host	anal pruritus, irritation	7;25;75
Enterobius bipallatus			*Macaca* sp.		no intermediate host		7
Trypanoxyuris callithricis	45x90	thin shelled, symmetrical	*Saguinus* sp.	w, l, c	no intermediate host	unknown	39;75
Trypanoxyuris tamarini	35x75	thin shelled, symmetrical	*S. fuscicollis* *S. nigricollis* *Saguinus sp.*	w, i	no intermediate host	unknown	13;15;25;37; 59;61;75
Trypanoxyuris oedipi	21x40	thick shelled, symmetrical	*S. oedipus*	w	no intermediate host	unknown	17;25;59;61; 75;64
Probstmayria nainitalensis			*M. mulatta*				4;75;77
Subulura jacchi	53		*Saguinus* sp.	w, c	cockroaches	unknown	13;25;32;38;48; 54;61;71;74;75
Subulura malayensis			*M. fascicularis*				7;25;75;82
D. Physalopteroidae							
Physaloptera spp.	30-40x40-60	oval, smooth thick shell, embryonated	*S. geoffroyi* *Saguinus sp.*		insects, beetles, katydids cockroahes (*Blatella* sp.)	gastritis oesophagitis ulceratice enteritis	6;25;42;48; 74;75;81
Physaloptera tumifaciens			*Macaca* sp.				25;34;47;59;82

Parasite species	Size in µm	Morphology	Host species	Host origin	Intermediate Hosts	Pathogenicity	References
E. Rhabdochonidae							
Trichospirura leptostoma	23-30x50x55	thickshelled embryo with tooth	*S. fuscicollis* *S. oedipus* *Saguinus sp.*	i, w	roaches	pancreatitis cholangitis fibrosis obstructive jaundice	13;14;25;32; 42;48;59;75;81
F. Rictulariidae							
Pterygodermatites alphi			*Saguinus* sp.	l, z	cockroaches, arthropods	enteritis	25;64;75
Pterygodermatites nycticebi	26-36x39-45	thickshelled embryonated	*Saguinus* sp.	z	cockroaches	enteritis	75;81
G. Gongylonematidae							
Gongylonema macrogubernaculum			*Macaca* sp.		cockroaches, dung beetles		25;34;68;76
Gongylonema pulchrum	34-40x18-22	thick shelled	*Macaca* sp.	w			36;50;51;76;
Gongylonema spp.	23-26x40-50	oval, thick transparent shell larvated	*Macaca* sp. NWP		cockroaches, beetles	non pathogenic	31;25;28;75
H. Spiruridae							
Protospirura muricola	40x55	larvated	*S. fuscicollis*	c, l	cockroach (*Leucophaea madera*)	non pathogenic	8;61;74;75
Spirura guianensis	58x33		*S. nigricollis* *S. fuscicollis* *S. geoffroyi* *Saguinus* sp.		arthropods, locusts (experimentally *Locusta migratoria*)	oesophagitis, death	13;25;58;59; 61;74;75
Spirura tamarini	30-40x54-60	subglobular egg thick, hyalin, smooth shell embryonated	*S. nigricollis* *S. mystax*	c, i, l	locusts		12;58;74;75
Streptopharagus pigmentatus	28-38x17-22	thick shell embryonated	*M. fuscata*	c, l	beetles	non pathogenic	25;31;34;40

Parasite species	Size in µm	Morphology	Host species	Host origin	Intermediate Hosts	Pathogenicity	References
I. Ascarididae							
Ascaris lumbricoides			*Macaca* sp.		no intermediate host	lesions	25;59;68;75;82
Ascaris sp.	35-45x45-70	rounded, thick shell	*S. geoffroyi*		no intermediate host	non pathogenic in large numers occlusion of bowl	6;25;59;61; 68;74;75;81
J. Strongyloididae							
Strongyloides cebus	eggs:20-35 x40-70 larva: 150-190	ovoid, thin shell, embryonated or with larva	*S. fuscicollis* *Saguinus* sp.	c, p	no intermediate host	enterocolitis, dermatitis, pulmo-nary hemorrhages bronchopneumonia	6;12;25;42;48; 59;61;68;75;81
Strongyloides fuelleborni		ovoid, thin shell, embryonated or with larva	*M. mulatta* *M. fascicularis* *Macaca* sp.	w, c, i, l	no intermediate host	enterocolitis, dermatitis, pulmo-nary hemorrhages bronchopneumonia	24;25;40;59 64;68;75
K. Ancylostomatidae							
Ancylostoma duodenale	50-60x40-45	thin shell	*Macaca* sp.		no intermediate host		7
Ancylostoma sp			*Saguinus* sp.			enteritis with hemorraghes	6;48;61;71; 74;75
Necator americanus	50-60x40-45	thin shell	*Macaca* sp.	c	no intermediate host		25;68;84
Globocephalus simiae	70x40		*M. fascicularis*		no intermediate host		25;72;73;75
Characostomum asmilium			*M. nemestrina*		no intermediate host		25;75;
L. Metastrongylidae							
Angiostrongylus costaricensis	larva: 14-15x 260-280	larva:notch close to the tip, slender esophagus, nervering	*S. mystax*	c, i	slugs (*Vaginus plebius*), molluscs (*Biomphalaria*, *Achatina* sp.), freshwater snails	granulomatous appendicitis, intestinal ulcera, peritonitis, arteritis, thrombosis	6;61;75; 81
Angiostrongylus cantonensis			*M. fascicularis*	c	*Veronicella*, *Achatina* sp.		43
Filaroides barretoi	larva: 14-15x 260-280		S. mystax *S. labiatus* *Saguinus* sp.	w	unknown	non-pathogenic, atelectasis, pulmonary hemo-rrhages	25;32;42;54; 75;79

Parasite species	Size in µm	Morphology	Host species	Host origin	Intermediate Hosts	Pathogenicity	References
Filaroides gordius	larva: 14-15x 260-280		*S. fuscicollis* *S. nigricollis*		unknown	non-pathogenic, atelectasis, pulmonary hemo-rrhages	25;42;75;79
Filaroides sp.	larva: 14-15x 260-280		*S. fuscicollis* *Saguinus* sp. *M. fascicularis*	c	unknown	unknown	1;13;25;48;58; 61;75;81;
M. Trichostrongylidae							
Longistriata dubia	31-41x62-79		*S. nigricollis* *S. fuscicollis* *Saguinus* sp.	w, c	no intermediate host	unknown	13;25;29;42; 59;61;68;75
Longistriata sp.			*Saguinus* sp.				48;61
Molineus elegans	30-37x59-63	ellypsoid, thin shell, slightly tapered at one end	*S. mystax* *S. midas*	w	no intermediate host	non pathogenic	25;32;38;42; 61;68
Molineus midas	30x50	morula stage	*S. midas*	c	no intermediate host		26
Molineus vexillarius	20-29x40-52		*S. fuscicollis* *S. leucopus* *S. oedipus* *Saguinus* sp.	w, c	no intermediate host	non pathogenic	13;19;25;42;61
Trichostrongylus colubriformis			*Macaca* sp.	l	no intermediate host		25;47;75;
Nochtia nochti	60-80x35-42	thin shell, ellipsoid	*Macaca* sp.	c, l	no intermediate host	formation of benign tumors	1;34;59;75
N. Oesophagostomidae							
Oesophagostomum apiostomum	60-63x27-40	typical strongyl egg	*Macaca spp.*	w, l	no intermediate host	granulomatous lesions, ulcers	24;33;25;68;75
Oesophagostomum aculeatum	69-86x35-55		*Macaca spp.*	w, c	no intermediate host		16;25;40;68;75
Oesophagostomum bifurcum	60-75x35-40		*Macaca spp.*	l	no intermediate host		21;25;47;68;75
Ternidens deminutus	57-65x36-45	short axis	*Macaca spp.*		no intermediate host	mucosal dammage, blood loss	25;33;59;68;75

4. ACANTHOCEPHALA

Parasite species	Size in µm	Morphology	Host species	Host origin	Intermediate Hosts	Pathogenicity	References
A. Oligacanthorhynchidae							
Prosthenorchis elegans	egg: 25-41x 33-45 oncosphere 24-33x30-41	thickwalled eggs, fine reticular sculpturing in outer shell, raphe of middle shell	*S. nigricollis* *S. oedipus* *S. fuscicollis* *S. mystax* *S. geoffroyi*	w, c	cockroaches (*Blatella germanica*, *Blabera fusca*, *Rhyparobia madera*), insects, beetles (*Lasioderma serricorne*, *Stegobium paniceum*),	granulomatous ulcerative enteritis, perforation of the intestine, peritonitis	10;13;20;25;32; 38;42,48;51; 54;61;67;68; 71;74;75;81
Prosthenorchis spirula		outer shell lightly sculptured, no raphe	*S. oedipus* *M. nemestrina*	w, c l	cockroaches		7;13;25;61;67; 74;75
Prosthenorchis sp.			*S. fuscicollis* *S. mystax* *S. oedipus* *S. geoffroyi*		cockroaches		61;74

Legend:

A.-N= families of the cited parasite species, taxonomy follows Schnieder and Tenter (2006) if not otherwise noted in the references. Grey shaded cells= recovered parasite taxa of this study.

Host Species:
Saguinus sp.: *S. nigricollis, S. fuscicollis, S.mystax, S. Oedipus, S. geoffroyi, S. labiatus, S. leucopus, S. midas*

Macaca sp.: *M. mulatta*, *M. fascicularis, M. irus, M. fuscata, M. nemestrina*

Host origin: w= wild, c= captured wild animals, i= imported, l= laboratory, p= pet, z= zoo animals.

REFERENCES (LIST OF HELMINTH PARASITES OF STUDY SPECIES)

1. Abott and Majeed (1984); **2.** Allen (1960) **3.** Anderson (2000); **4.** Arya (1981); **5.** Baer (1927); **6.** Brack (1987); **7.** Brack (2007); **8.** Campos and Vargas-Vargas (1978); **9.** Chai *et al*. (2007); **10.** Chai *et al*. (2009); **11.** Cosgrove (1966); **12.** Cosgrove *et al*. (1963); **13.** Cosgrove *et al*.(1968); **14.** Cosgrove *et al*. (1970); **15.** Deinhardt *et al*. (1967); **16** Dewit *et al*. (1991); **17.** Diaz-Ungria (1964); **18.** Ditrich *et al*. (1992); **19.** Dunn (1961); **20.** Dunn (1963); **21.** Eberhard *et al*. (2001); **22.** Faust (1967); **23.** Fiennes (1967); **24.** File and Kessler (1989); **25.** Flynn (1973); **26.** Fuchun (1987); **27.** Gallati (1959); **28.** Gebauer (1933); **29.** Gibbons and Kumar (1980); **30.** Gillespie *et al*. (2005a); **31.** Gotoh (2000); **32.** Gozalo (2003); **33.** Habermann and Williams (1957); **34.** Hashimoto and Honjo (1966); **35.** He *et al*. (2001); **36.** Hernandez *et al*. (2009); **37.** Hori *et al*. (1982); **38.** Horna and Tantaleán (1990); **39.** Inglis and Cosgrove (1965); **40.** Itoh *et al*. (1988); **41.** Kagei and Hasegawa (1974); **42.** King (1976); **43.** Kodama (1981); **44.** Krishnasamy *et al* (1988); **45**. Kumar (1999); **46.** Kuntz (1972); **47.** Kuntz *et al*. (1968); **48.** Kuntz and Myers (1972); **49.** Lacoste (2009) **40.** MacIntosh *et al*. (2008); **51.** MacIntosh *et al*. (2010); **52.** Malaivijitnont *et al*. (2005); **53.** Malek (1980); **54.** Michaud *et al*. (2003); **56.** Murphy *et al*. (1979) **57.** Myers (1972); **58.** Nelson *et al*. (1966); **59.** Orihel and Seibold (1972); **60.** Palmieri and Krishnasamy (1978); **61.** Potkay (1992); **62.** Premvati (1959); **63.** Prosl and Tamer (1979); **64.** Rowland and Vandenbergh (1965) **65.** Samuel *et al*. (2001) ; **66.** Sawada and Kifune (1974); **67.** Schmidt (1972); **68.** Shadduk and Pakes (1978); **69.** Stunkard (1965); **70.** Tang (1950); **71.** Tantaleán *et al*. (1990); **72.** Tate and Rubin (1973) **73.** Teixeira de Freitas and Lent (1936); **74.** Thatcher and Porter (1968); **75.** Toft and Eberhard (1998); **76.** Uni *et al* (1994); **77.** van Waerebeke *et al*. (1988); **78.** Waggie *et al*. (1994) ; **79.** Webster (1978); **80.** WHO (1995); **81.** Wolff (1990); **82.** Yamashita (1963); **83.** Yoshimura (1970); **84.** Young et al. (1957)

Appendix B: Picture of a meal including fish provisioned to macaques.

Appendix C: Composition of parasite taxa in the individual tamarins from Peru. The table shows the combination of parasite taxa within the host individuals and the number of individuals that harbor these combination.

Parasite combination	Number of individuals				
	Total	Group D	Group S	Group F1	Group F2
strongylid	1	1			
small spirurid	1	1			
*Prosthenorchis**strongylid*small spirurid	3	3			
strongylid*small spirurid*nematode larva	2		2		
strongylid*small spirurid**Hymenolepis*	1	1			
small spirurid**Hymenolepis** nematode larva	1	1			
strongylid*small sprirurid*cestode B	1	1			
*Prosthenorchis**strongylid*small spirurid**Hymenolepis*	1		1		
strongylid*small sprirurid**Hymenolepis**nematode larva	2	1		1	
strongylid*small spirurid*nematode larva*cestode B	6			2	4
strongylid*small spirurid*nematode larv*large spirurid	1		1		
*Prosthenorchis**small spirurid*strongylid*nematode larva**Hymenolepis*	3	1	2		
*Prosthenorchis**small spirurid*strongylid*large spirurid**Hymenolepis*	1	1			
*Prosthenorchis**small spirurid*strongylid*large spirurid*nematode larva	3	1	1	1	
strongylid*small spirurid*nematode larva*large spirurid*cestode B	4			2	2
strongylid*small spirurid*nematode larva*large spirurid**Hymenolepis*	2		1	1	
strongylid*small spirurid*nematode larva*cestode B**Hymenolepis*	2			1	1
small spirurid*large spirurid**Hymenolepis**nematode larva*cestode B	1				1
*Prosthenorchis**small spirurid*strongylid*nematode larva*cestode B*large spirurid	1		1		
*Prosthenorchis**small spirurid*strongylid*nematode larva*large spirurid**Hymenolepis*	1			1	
strongylid*small spirurid*nematode larva*cestode B*large spirurid**Hymenolepis*	4		1	2	1
all seven parasite taxa	1			1	
Total number of individuals	43	12	10	12	9

Appendix C: Composition of parasite taxa in the individual macaques from Thailand. The table shows the combination of parasite taxa within the host individuals and the number of individuals that harbor these combination.

Parasite combination	Number of individuals							
	Total	Group K1	Group K2	Group K3	Group P1	Group P2	Group P3	Group P4
Strongyloides	4		1		2	1		
Trichuris	12	1		3		2	3	3
hookworm	3			1			1	1
MIF	6	1	1		1	3		
Oesophagostomum	3				1	1		1
nematode larva	7	1	1	1			2	2
Strongyloides**Trichuris	20	6	6	4	2	2		
***Strongyloides**hookworm**	3			2	1			
***Strongyloides**nematode larva**	2	2						
***Trichuris**hookworm**	1					1		
***Trichuris**MIF**	1	1						
Trichuris**Oesophagostomum	1							1
***Trichuris**nematode larva**	3				2			1
hookworm*Oesophagostomum	1						1	
MIF*nematode larva	2		1			1		
***Strongyloides**Trichuris**hookworm**	3		1	1	1			
***Strongyloides**Trichuris**MIF**	5	2	3					
Strongyloides**Trichuris**Oesophagostomum	3				1	1	1	
***Strongyloides**Trichuris**nematode larva**	9	2	3	2		2		
***Trichuris**hookworm*nematode larva**	2			1			1	
***Trichuris**MIF*nematode larva**	1		1					
***Trichuris**Oesophagostomum**nematode larva**	1							1
***Strongyloides**Trichuris**hookworm*MIF**	1	1						
***Strongyloides**Trichuris**MIF*nematode larva**	4			4				
***Trichuris**IF*Oesophagostomum**nematode larva**	1				1			
***Strongyloides**Trichuris**hookworm*MIF*Oesophagostomum**	1				1			
Total number of individuals	100	17	18	19	13	14	9	10

Appendix D: Correlation of parasite prevalence between all morphospecies found in tamarins in Peru.

Spearman Rank Correlation N=43

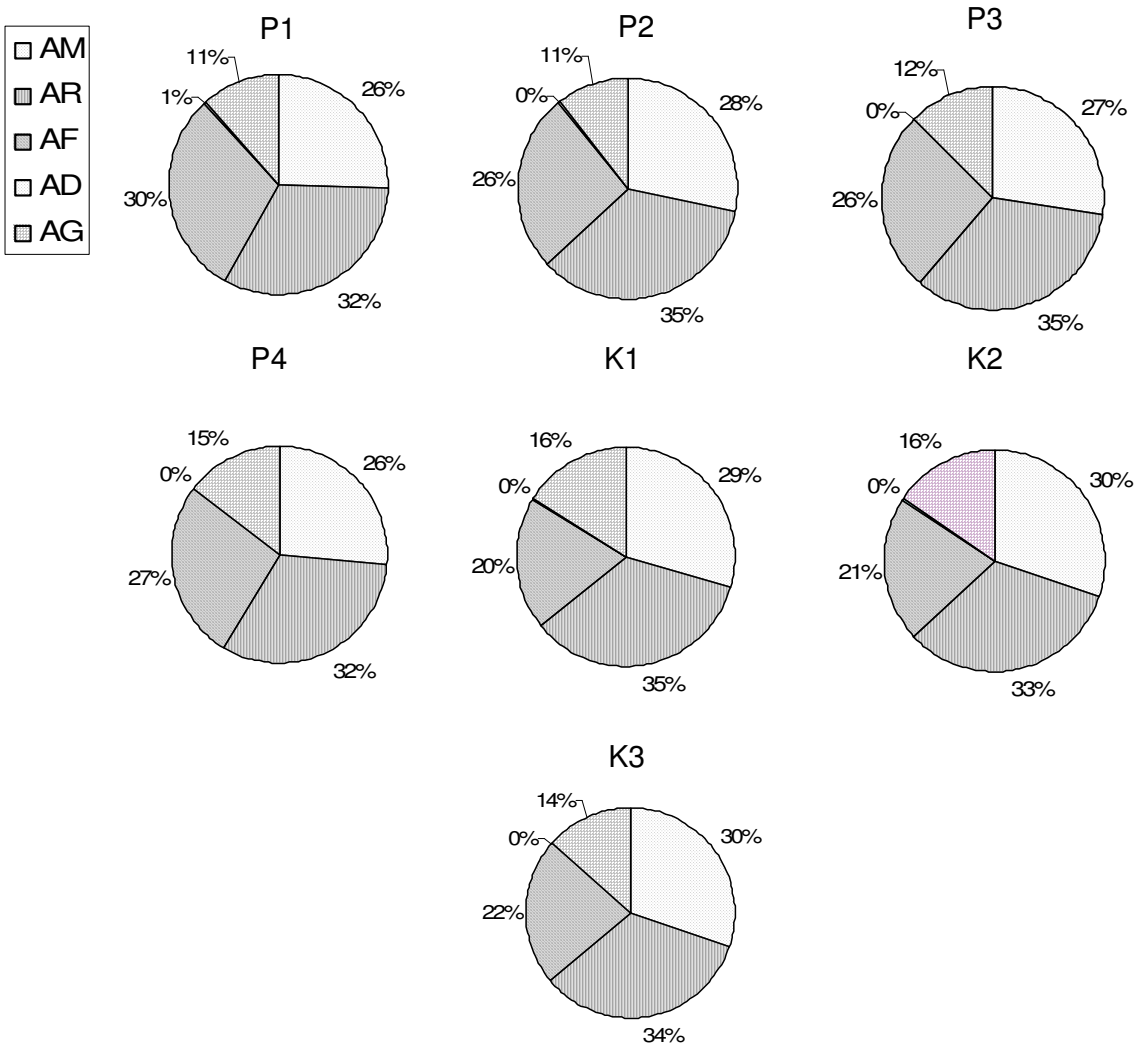
variable		*Prosthenorchis*	large spirurid	small spirurid	strongylid	*Hymenolepis*	nematode larva	cestode B	
Prosthenorchis	r_s		0.604	0.494	0.222	0.757	0.102	**0.003**	p
large spirurid		0.081		0.380	0.703	0.486	**0.024**	0.192	
small spirurid		0.107	0.137		0.788	0.357	0.051	0.357	
strongylid		0.109	0.060	-0.042		0.480	0.595	0.645	
Hymenolepis		0.049	0.109	0.144	-0.111		0.385	0.437	
nematode larva		-0.253	0.343	0.300	0.083	0.136		**0.016**	
cestode B		-0.449	0.203	0.144	0.072	-0.122	0.365		

Correlation of parasite prevalence between all morphospecies found in macaques in Thailand.

Spearman Rank Correlation N=135

variable		*Strongyloides*	*Trichuris*	MIF	hookworm B	*Oesophagostomum*	nematode larva	
Strongyloides	r_s		<0.001	0.296	0.241	0.760	0.423	p
Trichuris		0.527		0.904	0.141	0.481	0.075	
MIF		0.091	0.010		0.803	0.440	0.723	
hookworm B		0.102	0.127	-0.022		0.804	0.093	
Oesophagostomum		-0.027	0.070	0.067	0.022		0.656	
nematode larva		0.070	0.154	-0.031	0.145	-0.039		

Appendix E: Activity budgets of macaque study groups.



Legend: AM= Moving; AF= Feeding; AR= Resting; AD= Drinking; AG= Grooming. Groups P1-P4 from Don Chao Poo forest park; Groups K1-K3 from Kosumpee forest park.

Curriculum vitae

Personal details

Name	Alexandra Mücke
Birth name	Wenz
Date of birth:	February 21st, 1980
Place of birth:	Germersheim, Germany
Nationality:	German

Education

1999	Abitur (matriculation), Copernicus – Gymnasium, Philippsburg
2002	Intermediate Diploma, University of Karlsruhe (KIT)
2006	Diploma, Technical University Munich Main focus: vegetation ecology, soil science, animal ecology, behavioural ecology and botany Thesis: Behavioural observations on reintroduced Sumatran Orang Utans – a comparison of mothers and childless females Supervisors: Prof. Dr. Roland Gerstmeier, Dr. Peter H. Pratje
2006	Begin Doctoral studies, University of Karlsruhe (KIT) Thesis: The influence of human settlements on gastrointestinal helminths of wild monkey populations in their natural habitat Supervisor: Prof. Dr. Horst Taraschewski

Grants

2007-2009 Doctoral grant from the Federal State Foundation for Graduate Students

2007 Grant from the German Academic Exchange Service (Deutscher Akademischer Austauschdienst) for research in Peru

2008 Grant from the German Academic Exchange Service (Deutscher Akademischer Austauschdienst) for research in Thailand

International experience

2005 Research trip to Indonesia

Research aim: Differences in behavior between female Sumatra Orang Utans with child and childless Sumatran Orang Utans after reintroduction in the Bukit Tigapuluh National park

Collaboration partner: Dr. Peter H. Pratje

2007 Research trip to Peru

Research aim: Differences in gastrointestinal parasite burden of wild tamarins (*Saguinus mystax* and *Saguinus fuscicollis*) with different intensities of human contact.

Collaboration partner: Prof. Dr. Eckhard Heymann

2008 Research trip to Thailand

Research aim: Differences in gastrointestinal parasite burden of wild macaques (*Macaca fascicularis*) with different intensities of human contact.

Collaboration partner: Prof. Dr. Paiboon Sithithaworn

Languages: German (first language), English (fluent), French (good), Spanish (good), Bahasa Indonesia (basic), Latin (qualification in Latin after 7 years)

Congresses and publications

2008

The German Society for Parasitology, 23rd Annual Meeting. Hamburg, March 05-07.

Talk: Wenz, A., Heymann, E., Petney, T. and Taraschewski, H. (2008). The impact of human settlements on primate parasite burden: saddle-back and mustached tamarins in Peru

2009

*2nd European Congress of Conservation Biology.*Prague, September 01-05.

Poster: Wenz, A., Heymann, E., Sithithaworn, P., Petney, T. and Taraschewski, H. (2009). The impact of human settlements on primate parasite burden

2010

The German Society for Parasitology, 24th Annual Meeting. Düsseldorf, March 16-20.

Talk: Wenz, A., Heymann, E., Sithithaworn, P., Petney, T. and Taraschewski, H (2010). The impact of human settlements on primate parasite burden: saddle-back and mustached tamarins in Peru and long-tailed macaques in Thailand

2006

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