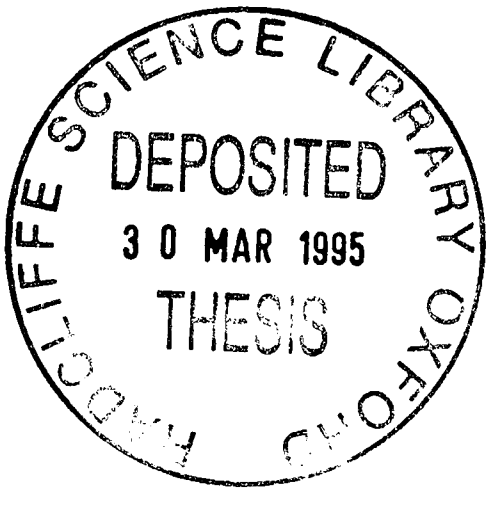
ECOLOGICAL PARASITISM OF BABOONS AND LIONS

CHRISTINE D. M. MÜLLER-GRAF

Thesis submitted for the degree of Doctor of Philosophy,

University of Oxford



The Queen's College

Trinity Term, 1994

To my family

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ABSTRACT

This thesis investigates the epidemiology of intestinal parasites in wild populations of two social animals, olive baboons (*Papio anubis*) in Gombe Stream National Park, Tanzania, East-Africa, and lions (*Panthera leo*) in the Serengeti and Ngorongoro-Crater, Tanzania. These populations have been observed for over 20 years and detailed information on individual hosts was available for analysis with the parasitological data allowing to address questions about the relationship of host genetics and social behaviour to parasite infection.

The baboons were infected with seven different helminths as well as two types of protozoans. Fifteen morphologically different parasites were found in the lions. All baboons and almost all lions were parasitized. Parasite distribution in both host species was overdispersed. Spatial differences in parasite infection in the baboons and lions emerged as the strongest effect on heterogeneity of infection. Parasites of both host species showed seasonal and temporal variation. Parasite-parasite associations did not appear to have a strong impact on overall patterns of infection in either baboons or lions. Across all parasite taxa, age (with one exception in baboons), sex, reproductive status and group size had little significant influence on parasite burden. For baboons age-prevalence and age-severity profiles resembled those for the same parasites in humans. Correlations between baboon social rank and parasite burden were equivocal. Parasite infection was not correlated with size of baboon female genital swelling. Two lion populations were compared, an inbred and an outbred. Only one parasite, *Spirometra* spp., had a significantly higher prevalence in the inbred population, contrary to expectations.

Results of this study suggest that any effects on levels of infection in these wild populations due to social behaviour, genetics, sex and reproductive effort may be masked by the stronger influence of environmental and/or behavioural components of exposure, at least in the short term. This implies that the importance of factors such as genetics or social behaviour on infection may not always be apparent and may be dependent on the details of the local ecology of both host and parasite at the time the system is studied.

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CONTENTS

CHAPTER 1

Introduction

1.1 WHY STUDY PARASITES IN WILD POPULATIONS?

Parasites do not only cause disease, but they are also considered to be an important ecological and evolutionary force (Hamilton 1982; Begon *et al.* 1986; Dobson and Hudson 1986; Gregory and Keymer 1989; Minchella and Scott 1991; Hamilton *et al.* 1990; Hamilton 1990; Hamilton 1991). Parasites may affect population dynamics and community structure of host species and also influence, for instance, host behaviour, genetics and nutrition. It has been suggested that parasites have the potential to play a major role in regulating host populations (Anderson 1978; Anderson and May 1978; Anderson 1979; Scott and Dobson 1989; Spratt 1990). They also may have a substantial influence on maintaining host genetic diversity (Hamilton 1982; Hamilton and Zuk 1982; May 1985; Anderson 1988; O'Brien and Evermann 1988; Wakelin 1988; Kennedy 1989; Keymer and Read 1990; Howard 1991). For instance, gene-for-gene associations between host and parasites can sustain polymorphism in gene frequencies of host and parasites (May 1985). To coevolve with their parasites, hosts must be able to change gene combinations at appropriate rates. This process may be responsible for the establishment of sex, as a way to quickly create new gene combinations (Hamilton *et al.* 1990). Parasites may alter host behaviour; hosts may choose their mates according to their health (Hamilton and Zuk 1982) or the host may adapt its behaviour to minimize the impact of disease (Hart 1990; Keymer and Read 1991; Hart 1992; Møller *et al.* 1993).

A considerable number of theoretical studies have explored host-parasite interactions (Anderson and May 1978; Anderson and May 1982; Anderson and May 1991) and carefully designed experimental studies (Park 1948; studies reviewed in Scott and Dobson 1989; Zuk *et al.* 1990; Barnard 1994) have tried to demonstrate these effects. This has proven difficult to do. On the one hand laboratory studies which concentrate on a limited number of factors may not reflect host-parasite interactions under natural conditions (Scott 1991). On the other hand field studies inevitably involve extraneous variables which may complicate the picture shown in the laboratory. Moreover, whereas laboratory research has concentrated mainly on one or two parasitic infections, individuals in the wild may have multiple infections which can interact with each other in various ways, changing the overall impact of parasites and host-parasite interaction for any individual parasite species. Furthermore, in the field other factors than parasites may be more important, for instance for the regulation of host populations (Holmes 1982). To understand the ecological and evolutionary role of parasites, data from the wild are therefore needed to complement the findings from experimental studies and theoretical models and to ascertain which factors are important in influencing parasite infections and how parasites interact

with their wild host. "We know very often how biotic factors can affect parasites: we need to know whether and under what conditions they actually do so in natural populations" (Kennedy 1982). Studies in which parasite assemblages in host population are monitored over some time are especially necessary (Scott and Dobson 1989). Despite numerous survey studies (see Esch *et al.* 1990), few studies of wild mammal populations have looked at the host-parasite interaction on the level of the individual in much detail (but see Meade 1983; Festa-Bianchet 1990; Gulland 1991).

Host-parasite interactions are best studied within a framework of long-term projects which monitor the demography and behaviour of individually known animals. This makes it possible to relate parasite data to age, life-history and social behaviour as well as to abiotic factors. Material such as faeces may be relatively easy to obtain within these studies and because animals are individually recognizable, they can be resampled.

Additionally, the knowledge of host-parasite interactions in wild populations is of paramount importance for conservation. Examples - such as the black-footed ferret in North America - have shown how disease can endanger the survival of a species (Thorne and Williams 1988). Increased inbreeding of host species because of partitioned ranges may also increase the danger of susceptibility to disease. As the information about host-parasite interactions in the wild is still limited, the potential conservation problems caused by diseases cannot be fully evaluated.

Three populations of large mammals, lions in the Serengeti and the Ngorongoro Crater and baboons in Gombe Stream National Park, provided an ideal opportunity to study host-parasite interactions. These populations have been observed for more than twenty years (Schaller 1972; Packer 1977; Bertram 1978; Packer 1979; Ransom 1981; Packer *et al.* 1988). Individuals could be reliably identified and extensive data on each individual were available.

1.2 THESIS OUTLINE

After a brief review of the potential factors influencing parasite distribution in a host population, chapter 2 deals with the methods used to estimate parasite infection. The advantages and disadvantages of coprological examinations and the reliability of egg counts over time and between samples are discussed. Chapter 3 describes the morphology of the eggs found in the baboon faeces and life cycles of the respective parasites, while chapter 4 deals with the general distribution of parasites in a population of baboons in Gombe Stream National Park, Tanzania, East-Africa sampled in autumn 1991. Parasite prevalences are compared to previous baboon studies and age related patterns are compared with those for the same parasite taxa in humans. The impact of social rank,

which has been observed to influence parasite egg output in previous studies (Hausfater and Watson 1976), is discussed as well as associations between parasite infection and sex, female reproductive status and female genital swelling. In chapter 5 spatial differences among the baboon troops of the population are analysed. Putative factors that may cause these differences are discussed, such as the effect of troop size and the presence of humans. Chapter 6 examines the temporal changes in parasite infection in the baboon population between spring 1991 and spring 1992 with the aim of determining whether seasonal variation occurs and, if so, how it interacts with other determinants of parasite distribution. The morphology of the parasite eggs detected in the lion faeces and their respective life cycles are described in chapter 7. Chapter 8 discusses the epidemiology of intestinal lion parasites in two populations of lions in the Serengeti and Ngorongoro Crater in Tanzania, sampled between March 1991 and November 1992 and examines the factors contributing to the overall heterogeneity of infection. In particular, infection in an inbred and an outbred lion population in different environments are compared. The thesis concludes in chapter 9 with an overall discussion of the results of both studies and directions for further research.

1.3 SOURCES OF HETEROGENEITY OF PARASITE INFECTION IN HOST POPULATIONS

Marked variation in parasite burden between individuals is typical for most host-parasite systems. The distribution of parasites among hosts is often overdispersed (Crofton 1971; Anderson and May 1991). This is important, because the fraction of a population which is most heavily infected is more likely to suffer morbidity and mortality. Furthermore, heavily infected individuals contribute most to transmission. It is clearly important to identify the proximate causes of high and low parasite burden in individual hosts in the wild. Therefore, in a large-scale field study, variations in levels of infection have to be examined for associations with the available information on the ecology and biology of the hosts. Heterogeneity in infection can occur over space and time caused, for instance, by environmental factors. Differences in levels of infection can also originate from host related and parasite related (e.g. inter- and intraspecific interactions among parasites) factors (Anderson and Gordon 1982). These factors will be briefly reviewed in the following section.

Spatial variation

When comparing parasitic infection of the same host species in several geographical areas, prevalence and intensity of infection are not uniform (Dogiel 1964; Kisielewska 1970a; Esch *et al.* 1990; Montgomery and Montgomery 1990). Spatial differences in infection can be found on smaller scales as well (Forrester *et al.* 1988; Anderson *et al.* 1993; Jaenike 1994). Variability in prevalence on a larger scale may be caused by historic events, such as local parasite immigration or extinction (Freeland 1979; Anderson and May 1991; Esch and Fernandez 1992). Moreover, the habitat, defined by numerous environmental factors such as vegetation, temperature, humidity and substrate (Esch 1982), can influence patterns of infection by determining whether, and to what extent, conditions for transmission are suitable. For direct life cycle parasites, transmission depends on these factors while transmission of indirect life cycle parasites relies additionally on the ecology of intermediate hosts. Spatial differences may be caused by different exposure to infective stages due to ecological conditions. However, environmental factors/exposure are usually confounded by host characteristics such as genetics, behaviour, group size and nutrition which can vary between different habitats of a host species. Differences on smaller spatial scales, for instance differences between helminth infections among households in a village (Anderson *et al.* 1993; Chan *et al.* 1994) or between the degree of infection of mycophagus *Drosophila* from different individual mushrooms (Jaenike 1994), have been used to attempt to disentangle environmental, genetic or behavioural factors. Studies of small-scale spatial differences in infection are important in shedding light on the relative importance of these factors for parasite distribution in a population. Differential exposure to parasites may be one of the main components causing heterogeneity of infection in host populations.

The importance of environmental factors as determinants of parasite burden has been discussed in detail in human studies. For example, hookworm is confined mainly to the tropics and subtropics because here the environmental conditions are suitable for the survival of the larvae (Schad *et al.* 1983). Even in the normal ranges of these parasites, hookworm abundance is affected by environmental conditions (Cort *et al.* 1926; Sen-hai and Wei-xia 1990). Temperature, soil type and moisture can have a significant impact on the abundance of hookworm larvae in the ground (Augustine and Smillie 1926; Kochar *et al.* 1976; Schad *et al.* 1983; Hominick *et al.* 1987). Shaded sites, for instance, provided a more favourable habitat for hookworm transmission than sunny areas (Hominick *et al.* 1987). The amount of contact with sites contaminated with infective stages can of course be an important determinant of an individual's parasite burden (Hominick *et al.* 1987). Likewise, quantity and quality of contacts with infective stages of parasites can correlate with levels of infection of parasites, such as *Ascaris* (Chandler 1954; Croll 1983),

Schistosoma mansoni (for children) (Croll *et al.* 1982; Wilkins *et al.* 1987; Bundy and Blumenthal 1990) or *Trichostrongylus* sp. (Croll 1983; Nwosu 1983).

Temporal variation

Temporal variation in infection of host populations and individuals has been observed in various host-parasite systems (Dogiel 1964; Halvorsen *et al.* 1985; Montgomery and Montgomery 1988; Anderson and May 1991; Anderson 1993). A knowledge of seasonal and annual variation is important for comparing samples within and across populations. Temporally irregular variation could indicate that the dynamics of certain host-parasite systems are unstable. Seasonal or annual fluctuations are caused by climatic changes which interact with other factors like host behaviour, intermediate host abundance, infective stage longevity, infectivity, and parasite development (Anderson 1993) and may furthermore affect individual hosts of the same species differently because of differences in, for instance, host nutrition or sex (Halvorsen *et al.* 1985). Temporal changes in parasite population dynamics can be influenced by host factors, such as variation in the population structure of the host, which in turn may or may not be directly connected with environmental factors. Host density as well as changes in the age structure of a population or in the age of an individual can have an impact on parasite dynamics (Kisielewska 1970b). Temporal changes in an individual's infection may be also caused by variation in its physiology, such as occurs with pregnancy, nutritional status and stress. Additionally, patterns of infection can vary irregularly due to stochastic events or/and local changes in environment.

Spatial and temporal variation can reflect environmental, host-related and parasite related factors. The two latter are considered explicitly in the following sections with particular reference to mammals including humans.

Host characteristics which influence parasite distribution

Immune response

The host reacts to a parasitic infection by activation of its immune system with the final aim of eliminating the parasite. The reaction of the immune system after infection can span from complete expulsion of the parasite, reduced intensity of infection, effects on parasite physiology to no effect at all (Cox 1982). Both innate and acquired immunity play

a role in challenging micro- and macroparasites (Roitt *et al.* 1989). The involvement of humoral and cellular immune responses has been identified (as reviewed in Lloyd and Soulsby 1988; Crook 1990; Maizels *et al.* 1993; Wilson 1993) but the precise mechanisms for each parasite infection are still not resolved (Wilson 1993). The antibodies IgA, IgE, IgG and IgM are produced in response to parasites and act in various ways to counteract the infection (Cox 1993; Wilson 1993) sometimes in association with specific cells, such as IgE and eosinophils in helminth infection (Roitt *et al.* 1989; Moqbel and MacDonald 1990; Wilson 1993). These associations are closely regulated by cytokines varying with parasites and between hosts, resulting in Th1 and/or Th2 responses (Cox and Liew 1992; Maizels *et al.* 1993; Wilson 1993). Experimental results showing different mechanisms for different parasites may indicate that the immune response is very much dependent on the particular host-parasite system (Maizels *et al.* 1993); for instance, mice respond to schistosome infection with Th1 production and rats with Th2 (Wilson 1993).

Immune responses to micro- and macroparasites can differ (Cox 1993). The aim of the immune system is to contain a microparasite infection and stop further multiplication. In contrast, as no multiplication of macroparasites occurs inside the definitive host, the intention is to either eliminate or shorten the life-span of the parasite and/or to reduce parasite fecundity. Macroparasite infections are normally long-lasting and cause morbidity rather than mortality and immune responses confer no complete protection against reinfection (Maizels *et al.* 1993).

Variations in immunocompetence among hosts, which result in heterogeneity of parasite distribution within a population, are influenced by the interactions of the immune mechanisms with other host attributes. Immune responses can be genetically controlled (Wakelin and Blackwell 1988). Acquired immunity is also related to intensity of infection and exposure and inevitably therefore to time and host age as well as current infection. In addition, host immunity can be modified by sex hormones. Nutrition also can play an important role in interacting with the immune system (Keusch and Farthing 1986; Keymer and Tarlton 1991).

Host genetics

Host genetics can be a major factor for resistance and susceptibility to infection (as reviewed in Wakelin 1978; Wakelin 1985a; Wakelin 1985b; Wakelin and Blackwell 1988; Wakelin 1989; Grencis 1990). Laboratory studies, such as those involving the selection of mice for resistance and susceptibility to infection by *Nematospiroides dubius* over ten generations (Brindley and Dobson 1982) and studies of several other mice strains showing genetically determined variation in susceptibility to helminth and protozoan infections

(Wakelin and Blackwell 1988), demonstrated that the genome of an individual can determine whether, and to what extent, that individual is resistant against a particular parasite (see also Behnke and Robinson 1985; Wassom *et al.* 1986; Keymer *et al.* 1990; Keymer and Tarlton 1991). Human studies have shown associations between known genetic markers and resistance to disease (Grencis 1990; Hagan 1992). A recent study in malaria has pointed to the importance of HLA-alleles for the susceptibility to severe malaria (Hill *et al.* 1991). Malaria is also an often quoted example of how overall genetic control (not only of the immune system) of variation in red blood cell expression (resulting for instance in sickle cell anaemia and β-thalassaemia) can confer resistance to the carrier or reduce the severity of the disease (Allison 1954; Miller 1988). In field studies of humans, it has been noted that some individuals are predisposed to certain infections (Bundy *et al.* 1987b; Forrester *et al.* 1990; Keymer and Pagel 1990). Although exposure has been suggested as playing an important role, genetic factors may also be significant.

Acquired immunity and/or innate resistance to infection are under genetic control (Wakelin and Blackwell 1988). Resistance against parasite infection may be under simple genetic control involving only one or few closely linked genes (Wakelin and Blackwell 1988); this is even the case in macroparasite systems, such as *Brugia malayi, Dipletalonema viteae, Nematospiroides dubius, Trichinella spiralis, Trichuris muris* and *Taenia taeniaeformis* where resistance was inherited as a dominant character when crossing resistant and susceptible parents (Wakelin 1985a). In other cases several genes are involved in the resistance against the parasite and inheritance may be complex (Behnke and Robinson 1985). The MHC plays a role in resistance, in connection with modifying influences of background genes (Behnke and Robinson 1985; Wakelin 1985a; Kennedy 1990). Clearly, differences in genotype have the potential to contribute significantly to the differences in parasite burden of individuals of the same host species.

In addition, genetic control of resistance has an influential impact on the dynamics of host populations, and also consequently for the coevolution of host and parasites. Parasites may select for certain genotypes (May 1985; Dobson and Merenlender 1991). Also, a host population which can maintain diversity in immune response modulating genes may be better equipped to deal with novel diseases (O'Brien and Evermann 1988). The classical example is the decimation of a cheetah population after an outbreak of the feline infectious peritonitis virus in comparison to the effect of the same virus on domestic cats, which was probably due to the extreme uniformity of the cheetah population, and indicates the effect parasites may have on more inbred populations (O'Brien *et al.* 1985; O'Brien and Evermann 1988). Therefore, the maintenance of genetic diversity with regard to susceptibility to disease may be important for the conservation of species (May 1988; Scott 1988).

Age

The relationship between parasite infection and host age has been documented in numerous surveys (Bundy *et al.* 1987a; Anderson and May 1991). This association has been particularly well studied in humans because of its importance for control measures. Different types of age-prevalence and -intensity curves occur. Convex curves have been observed, where infection increases in younger age-classes and then decreases in older age-classes, for example, *Trichuris trichiura, Ascaris lumbricoides* intensity and *Schistosoma haematobium* prevalence and intensity (Bundy *et al.* 1987a; Wilkins 1987; Chandiwana *et al.* 1991; Bundy and Medley 1992). Another type of curve occurs where infection increases in the younger age-classes until it reaches a plateau in the mature age-classes, such as for hookworm prevalence (Bundy *et al.* 1987a).

The effect of age on infection can result from several factors. Different age-classes may experience different degrees of exposure caused by behaviour, which will then be reflected in prevalence and intensity of infection (Bundy and Blumenthal 1990; Chandiwana *et al.* 1991). Repeated exposure to parasites can lead to the acquisition of immunity, so that older age-classes previously exposed to the parasite have more resistance (Hagan *et al.* 1991). That acquired immunity may be involved in the reduction of infection rates in older individuals is supported by findings of more pronounced decline of infection in older individuals in areas of high transmission rates compared to communities with lower transmission rates (Anderson and May 1985b; Berding *et al.* 1986; Woolhouse *et al.* 1991) and lower rates of reinfection in older age-classes (Anderson 1986). However, for some parasite species, such as hookworm, infection rates are not distinctly reduced in older individuals (Bradley *et al.* 1992; Bundy and Medley 1992). Here, acquired immunity does not seem to be an influential factor and higher infection in older individuals may indicate longer exposure to pathogens. Whilst acquired immunity may protect older individuals, old age can also be associated with a decline in function of T- and B-lymphocytes and a poorer functioning immune system in general (Behnke 1990).

The association between age and infection is not only important for an individual host, but may also influence the population dynamics of infection (as reviewed in Quinnell and Keymer 1990). A population of mixed ages may show heterogeneity of infection just because of the differential effect of age.

Sex

Sex differences in prevalence and intensity of parasite infection have been observed for several host-parasite systems (Brabin and Brabin 1992). Either male or female hosts can be more highly infected, although it has been recognised that females generally mount stronger immune responses (Weinstein *et al.* 1984; Grossman 1989; Schuurs and Verheul 1990), but on the other hand can suffer from higher levels of morbidity (Bundy 1988a). Among microparasite infections females can be more resistant to certain species of trypanosomiasis and *Giardia,* but more susceptible to *Trichomonas vaginalis* and AIDS (Alexander and Stimson 1988; Brabin and Brabin 1992; Garnett and Anderson 1993). In macroparasite infection a higher prevalence of infection has often been reported for males (Alexander and Stimson 1988), but the reverse has been observed as well (Barriya and Al-Khalidi 1991). Differences may be caused by a number of reasons as the genders vary not only in their physiology, but also in their behaviour (Brabin and Brabin 1992). Gender-related behaviour may expose one sex more to parasites than another (as reviewed in Bundy 1988a). For instance, male desert toads (*Scaphiopus couchii*) are more heavily infected with the monogenean parasite *Pseudodiplorchis americanus* than females; transmission of the parasite takes place in the water during mating and males spend much longer periods in the water to mate than females (Tinsley 1989).

The immune system interacts with hormones and this may explain some of the differences in infection found between the sexes (as reviewed in Alexander and Stimson 1988). Sex hormones (Weinstein *et al.* 1984; Grossman 1985; Grossman 1989; Schuurs and Verheul 1990) and/or stress-related hormones can play a role. Males and females can be exposed to different levels of stress (Folstad and Karter 1992), and stress may be related to susceptibility of infection (Barnard 1994). Castrated reindeer, which exhibited lower testosterone levels and were not subjected to rut stress, showed lower parasite infections with *Hypoderma tarandi* similar to those of females (Folstad *et al.* 1989). It is usually difficult to disentangle differential behaviour, immune response or other factors as the sole cause of the differences in infection between the sexes (Alexander and Stimson 1988; Bundy 1988a).

Female physiological states

Hormones may also influence infection in females during different reproductive states, such as pregnancy, menstruation or lactation. Raised levels of certain hormones (oestrogen, progesterone and corticosterone have been suggested as possible candidates) can lead to immunosuppression and increased susceptibility during pregnancy (Lloyd

1983; Alexander and Stimson 1988; Brabin and Brabin 1992). Moreover, the different nutritional requirements of a pregnant individual may also contribute to a variation in susceptibility (Brabin and Brabin 1992). Immunosuppression during lactation and an increase in helminth egg output (post-parturient rise), assumed to be related to the influence of lactogenic hormones, has been observed in several host-nematode systems (Connan 1976; Michel 1976; Lloyd 1983; Behnke 1990). In one study however, sexually cycling female baboons were found to have a higher parasite ova emission than anoestrus ones, while pregnant females had the lowest ova emission and females were observed to have the highest ova emission during the week immediately before and the one week immediately after the onset of menstruation in comparison with females in the remaining mid-cycle (Hausfater and Watson 1976). The mechanisms causing differential levels in susceptibility to parasite infections during the female reproductive stages depend on the particular host-parasite system.

Nutritional status

Nutrition interacts with parasite infection in complex ways (as reviewed in Bundy and Golden 1987; Crompton 1987; Stephenson 1987; Crompton 1991; Crompton 1993; Filteau and Tomkins 1994). Host nutritional status can affect the parasite and conversely parasite infection can influence the host's nutritional situation.

Parasites need the host for the provision of nutrients for their growth and reproduction. A malnourished host - depending on parasite and host physiology - may not be able to provide adequately for a particular parasite and hence growth or fecundity of the parasite can be stunted (as reviewed in Crompton 1987). One example is the dependence of the development of *Moniliformis dubius* on rat nutrition. Low fructose content in the host's diet was found to result in reduced growth and development and a delay in the onset of parasite reproduction (Keymer *et al.* 1983c). In laboratory experiments, protein deficient rodents showed decreased levels of malaria parasitaemia (McGregor 1988), which may be due to possible competition for essential amino acids by host and parasites (McGregor 1988). Host susceptibility to infection, and hence parasite establishment, may also be related to host nutritional status (Abbott *et al.* 1988). Malnutrition may change the gut environment and consequently alter the conditions for the establishment of parasites (Bundy and Golden 1987) or, by interaction with the immune system, may decrease humoral and cellular defences (Behnke 1990; Castro 1990) and/or induce defects in cellular immunity (Michael and Bundy 1992).

Parasite infection can also result in a change in the nutritional status of a host. Parasitic infections, especially helminth infections, may result or be accompanied by a

reduction of nutrient intake (Stephenson 1987; Crompton and Stephenson 1990), and may decrease the absorption of nutrients (Symons 1976; Crompton and Stephenson 1990; Solomons 1993) and nutrient utilization (Keymer *et al.* 1983b). Furthermore, a loss of nutrients can result from infection; for instance, blood loss through *Ancylostoma duodenale* infection amounts to 0.14 - 0.4 ml blood per worm per day (Crompton and Stephenson 1990). Infection can change the host's metabolism (Shetty and Shetty 1993; Stephenson 1993). As a consequence of all these changes brought about by infection the general host immune response can be weakened (Keusch and Farthing 1986; McGregor 1988). Effects of helminthiasis can be more severe in malnourished individuals (Keymer *et al.* 1983b; Abbott *et al.* 1985; Stephenson 1987; Slater 1988), but the reverse has also been found, for instance in schistosomiasis (as reviewed in Dale 1991), where the host immune response is weakened in malnourished individuals and less able to generate damage to the host itself, the main cause of morbidity in schistosome infections.

Differential effects of nutritional status are therefore yet another possible factor contributing to parasite overdispersion in a population. The effects of nutrition can be taken to the level of host population. In malnourished host populations, intensity of helminth infections may be higher and helminth survival increased (Bundy and Golden 1987) enhancing transmission rates. Furthermore, there may be higher parasite transmission rates in malnourished hosts because parasite fecundity may be less regulated by density-dependent constraints including acquired immunity (Keymer *et al.* 1983c; Slater and Keymer 1986). Conversely, production of normal eggs may be lower in malnourished hosts, as in the case of schistosome infection (DeWitt 1957; Knauft and Warren 1969; Akpom and Warren 1975; Dale 1991). Weakened, malnourished hosts may be more susceptible to infection in general (Slater and Keymer 1986). On the other hand, as effects of infection may be more aggravated in malnourished individuals, highly infected individuals and their parasites may be more quickly removed from a population through death, decreasing the overall number of parasites in the host population.

Behaviour

Behaviour can be related to parasitism on a group or individual level. The pressure of parasitism may influence group size, and an individual's social rank can show a correlation with levels of infection. Parasites may alter host behaviour to increase transmission and/or as a result of physiological changes and hosts may alter their behaviour to minimize the impact of disease.

Animal group size, which is determined by various factors, such as foraging, mating systems and predation (Hamilton 1972; Harvey and Green 1981; Pulliam and

Caraco 1984; Packer *et al.* 1990), may also be influenced by disease as a result of its cost to host fitness. One way to reduce infection may be to alter the group size (as reviewed in Keymer and Read 1991; Møller *et al.* 1993; Coté and Poulin 1994). Whether group size is increased or decreased should be dependent on the mode of transmission of the parasite and the host species in a particular environment.

For directly transmitted parasites, a reduced group size may lead to fewer species of parasites and lower intensities of infection. This has been observed in mammal and bird populations such as prairie dogs, horses and swallows (Hoogland and Sherman 1976; Hoogland 1979; Brown and Brown 1986; Rubenstein and Hohmann 1989; Loye and Caroll 1991). For certain rainforest primates, species richness of intestinal protozoan parasites can be a function of group size, provided that social isolation exists between groups, with smaller primate groups being more likely to lose protozoan parasites than larger groups due to random extinction (Freeland 1977b; Freeland 1979).

For vector-transmitted parasites, an increase in group size may limit disease because the number of vector bites per individual may be reduced in larger groups. Group size can be increased either by recruiting more animals of the same species or by forming mixed species groups (Møller *et al.* 1993). Several studies have reported a negative relationship between group size and bites of vectors including associations between flies and horses (Duncan and Vigne 1979; Rubenstein and Hohmann 1989) and sticklebacks and their ectoparasites (Poulin and Fitzgerald 1989). Temporal occurrence of rainforest primate polyspecific associations was correlated with activity of dipteran vectors (Freeland 1977a). However, as group size is a complex result of various factors and depends on the specific ecology of a species, even for vector-transmitted diseases, larger group sizes have also been found to be associated with higher infection rates (Davies *et al.* 1991). For other indirect life cycle parasites, no correlation between group size and parasite infection has been found (Coté and Poulin 1994).

Observations on group size and infection are in general supported by epidemiological field data of human infections and theoretical models showing that host densities can have an impact on prevalence and intensity of parasite infections (Anderson 1993). Transmission of many direct life cycle microparasites is directly proportional to the contact rate between hosts (Anderson 1993). A higher density of susceptible hosts can thus lead to a higher prevalence of infection. Likewise, a very low contact rate could drive a parasite to local extinction (Anderson 1993) as in the study of rainforest primates (Freeland 1979). For macroparasites, it has been suggested that host density times the density of infective stages is an important determinant of the net rate of infection (Anderson and May 1991). Moreover, the basic reproductive number for macroparasites, R_0, depends on host density (Anderson and May 1991). For most human macroparasitic organisms, the threshold densities are very low, therefore infections are not likely to go

extinct (Anderson and May 1991), and lower densities may only have an impact on intensity of infection rather than prevalence. An exception are vector-transmitted diseases where the principal parameters determining infection rate are 'biting rate', the number of times an individual is bitten by a vector and the ratio of vectors to hosts (Anderson 1993). Group size may therefore affect levels of parasitism, but if and how is determined by the particular host-parasite system.

Within groups, social status, mainly of males, has been found to be associated with parasite infection. In baboons, a higher male social rank was correlated with higher parasite ova emission (Hausfater and Watson 1976) and similar observations were made for male reindeer (Halvorsen 1986). In mice, parasitic infection can prevent males from becoming dominant or may lead to loss of dominance (Freeland 1981; Rau 1983; Rau 1984). High-ranking male mice can show reduced resistance to infections (Barnard 1994); therefore one possible explanation for the association between rank and parasite infection is that high levels of testosterone in higher ranking males may impair the immune system (Grossman 1985; Schuurs and Verheul 1990), leading to higher parasite ova emission. On the other hand, it has been reported that younger, subordinate animals can suffer more severely from infection than older, dominant ones (Møller *et al.* 1993). However, the effects of age and dominance may not be clearly separable in this instance, or other factors such as stress may be predominant.

Parasites can induce changes in host behaviour in order to maximize transmission (Holmes and Bethel 1972; Holmes and Zohar 1990; Milinski 1990; Moore and Gotelli 1990; Combes 1991; Ewald 1993). Selection pressure of diseases can also force hosts to change their behaviour (as reviewed in Hart 1990; Keymer and Read 1991; Hart 1992; Møller *et al.* 1993). A host may avoid sites which could provide transmission opportunities (Hart 1990; Keymer and Read 1991); for instance, baboons in Amboseli National Park, Kenya, may change their sleeping sites to avoid contact with the infective stages of direct-life cycle parasites (Hausfater and Meade 1982; Hausfater and Sutherland 1984). Hosts may medicate themselves to eliminate or prevent infections: for example, chimpanzees may use a plant with antihelminthic properties to cure infections (Rodriguez *et al.* 1985; Huffman and Seifu 1989; Ohigashi *et al.* 1994). Other behaviours whose function is to minimize the impact of parasites are grooming (Murray 1990; Hart *et al.* 1992), preening, fly repelling behaviour (Hart 1992; Hart and Hart 1994) and nest fumigation (Clark and Mason 1985), immigration and dispersal (Møller *et al.* 1993) and possibly testing the quality of immigrating animals (Freeland 1976). Within and across populations, different behaviour may therefore be associated with differences in parasite infection. Further associations between behaviour and parasites have been noted, such as an impact on life histories (Curio 1988; Keymer and Read 1991), but are not discussed here.

Over the last decade the role of parasites in sexual selection has been greatly discussed (reviewed in Møller 1990; Read 1990; Clayton 1992). Parasites are likely to select for host sexual behaviour which minimizes the effect of parasitism. The hypothesis of parasite-mediated sexual selection assumes that individuals prefer healthy, parasite-free or resistant partners, because of 'good genes', which could confer parasite resistant alleles to their offspring (Hamilton and Zuk 1982), or in order to avoid infection with sexually or directly transmitted diseases (Clayton 1990; Hamilton 1990; Møller 1990) or to acquire partners which may provide better resources, such as parental care (Read 1990; Clayton 1992). Indication of quality can be provided by the results of intrasexual competition, chemosensory cues, direct signs of infection and condition of secondary sexual characters (Hamilton and Zuk 1982; Read 1990). Predominantly, male secondary traits have been the focus of debate. However, females may also advertise their quality and health; genital swelling in female primates for instance may be an indicator of female health, including parasite infection (Hamilton 1990; Pagel 1994).

Parasite - parasite interactions

Interspecific and intraspecific competition or enhancement between parasites in the same host may have an impact on parasite distribution (Esch *et al.* 1990; Sousa 1994). However, there are other explanations for the correlations between prevalence and intensity of parasite species.

Intraspecific competition may lead to a reduction in parasite infection because parasites of the same species may compete for the same resources (Keymer 1982; Barger 1987; Michael and Bundy 1989). However, other explanations point to the host immune response as the cause of the reduction in the number of parasites (Keymer 1982; Anderson and May 1985a; Quinnell 1988; Maizels *et al.* 1993) Conversely, in a few cases, suppression of immunity can enhance the establishment and survival of more parasites of the same species (Behnke 1987). Not only survival and establishment, but also fecundity or growth of parasites can be reduced at higher densities in the host (Keymer 1982; Keymer *et al.* 1983a; Michael and Bundy 1989). Density-dependence has been suggested to play a central role in regulating parasite population growth (Keymer 1982; Anderson and May 1991).

For interspecific interactions within a host, the same ecological mechanisms that occur between free-living species are active. There has been evidence from experimental studies and quantitative data from field studies for these interactions (Holmes 1961; Lie 1973; Esch *et al.* 1990; Sousa 1994). Such interactions can reach the extent that species can compete and replace each other in the same host (see Whitfield 1979). For example,

Phyllodistomum folium is seldom found together with *Myxidium lieberkuhni* in the bladder of the pike. As the digenean is known to digest the protozoan, this has been interpreted as direct interaction (Kennedy 1975). A seminal study by Holmes (1961) demonstrated that in concurrent infection of rats with *Hymenolepis diminuta* and *Moniliformis dubius* the growth of both parasites was affected, with *Hymenolepis diminuta* being more impaired. However, the number of parasites established was not affected by double infections, so that it would be difficult to detect this kind of interaction in a field study. Overall, the importance of parasite interactions in shaping parasite communities and parasite distribution is still a point of debate (Holmes 1973; Price 1980; Dobson 1985; Esch *et al.* 1990; Sousa 1994). For many coexisting parasite species no interaction has been observed (Kennedy 1975; Price 1980; Sousa 1994). However, it seems that numerous different types of interactions can take place, and that isolationist (non-interactive species) and interactive parasite communities are two ends of a continuum (Goater *et al.* 1987). Confounding influences, such as the immune system, make it difficult to observe if and what kind of interactions take place between parasite species infecting the same host. The interpretation of field data especially has to be treated with caution because of the numerous confounding factors.

Other factors could also cause supposed parasite-parasite interactions. Correlations between parasite species may also be linked to the impact of cross-immunity, where immunity against one parasite confers immunity against another at the same time. In natural situations individuals are often infected with several parasites and cross-immunity may be important. This is more likely if parasites are closely related and thus evoke very similar immune responses. Cross-immunity has been observed in laboratory studies (Dobson 1985; Christensen *et al.* 1987; Quinnell and Keymer 1990).

Not only negative effects, but also positive interactions between parasite species have been found. These may explained by the following mechanisms. On one hand, if immunity is genetically controlled then predisposition could operate similarly for both parasite species, thus creating a synergistic effect (Keymer and Pagel 1990; Quinnell and Keymer 1990). On the other hand, if parasites are transmitted, for instance, by the same route of transmission, the same intermediate host or in the same environment, they may occur together more often than parasites which are transmitted by completely different routes (Christensen *et al.* 1987; Lotz and Font 1991; Lotz and Font 1994). Furthermore, positive interactions can result if parasites of one species suppress the host's immune response, and thus facilitate the establishment of other parasite species (Barger 1984; Behnke 1987; Christensen *et al.* 1987). Immunosuppression has been observed for helminths and protozoans (Christensen *et al.* 1987).

This review is intended to give a brief overview of what factors may be important in shaping patterns of parasite distribution and abundance. As it is impossible to address all aspects of host-parasite interactions, some factors could not be considered in the study described here, such as host immune response, host nutritional status or disease avoidance behaviour. Information on age, sex, reproductive status and group size was related to parasite infection. Furthermore, genetic data on a subset of the lion population data were used to examine correlations between parasite burden and genotype. Changes in parasite infection over time were studied and spatial differences between different groups of the studied animals were considered. Parasite-parasite interactions were investigated using the multiple infection data of the study animals.

CHAPTER 2

Materials and methods for examination of parasite infection

ABSTRACT

Faecal egg counts are used to estimate intestinal parasite burden in baboons and lions. Advantages and disadvantages of this method are discussed. The collection of faecal samples is described. From the different methods of faeces analysis which were compared for both baboon and lion samples, the formol-ether method proved the most suitable. Three slides were analysed for each faecal sample as the majority of parasite taxa could be found in three as compared to ten readings. The reliability of egg counts within one faecal sample was high, indicating that eggs may be randomly distributed in the faeces. Some fluctuations in eggs per gram of faeces were noted in baboon samples taken from the same individual during one day, although no diurnal pattern was obvious. The largest amount of variation in parasite egg output was observed in samples from the same individual over a number of days.

2.1 ESTIMATION OF PARASITE INFECTION BY FAECAL EGG COUNTS

Faecal egg counts provide an indirect measure of prevalence and intensity of helminth infection. However, this method, although a meaningful measure of parasite burden, has limitations. Prevalence can be underestimated and estimates of intensity are likely to be biased by several factors influencing the number of eggs in a faecal sample (Guyatt and Bundy 1993). Any interpretation of results based on faecal egg counts has to take these factors into account.

Several different methods of analysing faecal samples for parasite eggs exist. As the choice of method may influence the result, initial tests were performed to assess several of the available methods. Presented here are the results of this comparison and the calculation of intensity of infection. Identifications of parasite taxa are described in chapters 3 and 7.

The reliability of the method as well as the biological variation of egg output over time may influence the results. Different samples from the same stool and faecal samples over one and over several days from the same individual baboons were compared to test the consistency of the results.

Although the estimation of parasite infection by stool examination is a common procedure in parasitological surveys, this indirect method of determining parasite infections 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 faeces (Thienpont *et al.* 1979). A roughly linear positive relationship between number of eggs per gram of faeces and parasite burden has been observed for some parasite species (Stoll 1923; Sachs and Sachs 1968; Keymer and Hiorns 1986; Pritchard *et al.* 1990). Results of coprological examination have to be interpreted bearing in mind the factors which may influence estimates of prevalence and intensity of parasite infection.

There are a number of reasons why infected individuals may be diagnosed as negative by the use of faecal egg counts (Hall 1981; Pritchard *et al.* 1990; de Vlas and Gryseels 1992; Guyatt and Bundy 1993). When the intensity of infection is low and only a few eggs are excreted, eggs can be missed during the analysis. In addition, the daily egg output of female parasites may vary and infection may not be detected when the egg output is low (Thienpont *et al.* 1979). Furthermore, the consistency of faeces may influence the detectability of parasite eggs (Thienpont *et al.* 1979). Non-random

distribution of eggs in faeces will influence estimates of infection status (Guyatt and Bundy 1993). In one previous study it was found that beads ingested by volunteers were randomly dispersed in the stool and it was thus inferred that helminth eggs are randomly distributed in the faeces as well (Martin 1965). In another study, however, variations in the concentration of eggs were found in different samples from the same stool (Hall 1981). Further studies are required to examine the distribution of parasites eggs in faecal material. No eggs will be observed in the prepatent period of a primary infection by definition as the parasite only starts laying eggs in the patent period. This may cause additional problems when looking for seasonal variation in egg burden. A higher worm burden may reflect seasonal transmission, but its expression as faecal egg output may be delayed due to the prepatent period. Detection of intestinal protozoan prevalence from stool samples can also be unreliable. Some protozoan cysts, such as *Giardia* are passed out on a cyclical basis (Garcia and Bruckner 1988), and therefore cannot be detected in every faecal sample. Consequently, for accurate diagnosis of these parasites, stool samples have to be examined over an adequate length of time. For dioecious parasites, the presence of only one sex may not result in egg production; hence the parasites will not be detected. In some species such as *Ascaris* spp. only the sole presence of males will lead to the absence of eggs as the females can lay unfertilized eggs (Guyatt and Bundy 1993). However, in general chances of encountering a partner seem relatively high because of the overdispersed distribution of parasites among hosts (Anderson and May 1985a).

Faecal egg counts are not only used to estimate prevalence, but also to assess intensity of infection. Density-dependent fecundity will affect the relationship between egg counts and worm burdens. 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 been observed despite a low intensity of infection (Anderson and Schad 1985; Anderson and May 1991) and may additionally influence detectability of eggs. However, not all parasites show evidence of density-dependent fecundity (Gregory *et al.* 1990). It has been argued that some of the effects of density-dependent fecundity might be due to measurement errors and that further studies are necessary to reveal the influence of density on parasite reproduction (Quinnell 1990). Daily variation in egg output will also distort estimates of intensity of infection. The egg count can also be influenced by the nutritional status of an individual (Thienpont *et al.* 1979). Furthermore, the host immune response may depress helminth fecundity (Quinnell 1990). Acquired immunity of older hosts may lead to a depression in egg output with host age. 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).

Reliability of egg counts varies among different parasite taxa. Cestodes, for instance, may not only release eggs but also proglottids containing many eggs. Because of these clusters, the egg output for one individual host can vary considerably within a short time span. As a result, cestode eggs may not reflect parasite infection to the same degree as nematode eggs (Thienpont *et al.* 1979). Egg counts cannot be compared between different parasite species as the mean number of eggs per female varies considerably among different parasite taxa (Thienpont *et al.* 1979; Anderson and May 1991).

The above mentioned factors have to be taken into account when interpreting parasite infections using faecal egg-counts. 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. 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. Besides dissections or worm expulsions, 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 gives an estimate of the intensity of infection.

2.2 SAMPLING

Faecal samples were collected when animals were observed defaecating. Before removing part of the faeces, the stool was mixed to prevent uneven sampling due to clustering of parasite eggs. Baboon faeces were placed in 25 ml vials and lion faeces in 50 ml vials and preserved with 10% formalin. Baboon faeces were collected immediately after defecation and preserved in formalin instantaneously or within 2 - 3 hours after collection. Lion faecal samples were collected after the lion had moved away from the place of defecation, which usually took only a few minutes, and subsequently preserved *in situ* or at the research station. In both cases, date, time of defecation, name of individual and pride/troop were recorded. Baboons were identified by field assistants who observed them daily and could recognize each individual by its appearance. Lions were identified according to whisker-spot pattern and earmarks (Pennycuick and Rudnai 1970; Packer 1992; Packer and Pusey 1993).

2.3 LABORATORY METHODS

Several methods have been described for detecting and quantifying different parasite taxa according to the method by which they have been preserved. They do, however, vary in sensitivity to different types of parasite eggs (Cheesbrough 1987). The choice of an appropriate method is determined by the logistic constraints in the field and by the type of parasites involved. A suitable method should detect all the different parasite taxa and allow estimates of infection intensity as well. The method should be easily repeatable to permit standardized comparison of different samples. To determine the best method for the analysis of lion and baboon faecal samples, samples were analysed with the direct smear-test, salt-flotation method, zinc-sulphate flotation method (with zinc-sulphate solution and hypersaturated zinc-sulphate solution) and the formol-ether method.

As the weight of faeces and amount of formalin in each vial were unknown, each sample was subjected to a standard procedure before comparison. An unknown amount of faeces-formalin mixture was mixed with glass beads and shaken to break down the faecal matter. The mixture was sieved through a tea strainer to remove large contaminants, such as bones and hair. The debris from the tea strainer was then examined under a low power microscope to ensure that no eggs remained in the residue. Only for very highly infected individuals could eggs be seen in the debris. The filtrate was centrifuged twice for 5 min at approximately 1500 rpm and the supernatant fluid discarded each time. After mixing with water the sample was split into equal volumes for testing each method. The water was discarded after further centrifugation and the samples were then treated according to the respective method.

I. Direct-smear method

A direct smear was taken from the pellet on to a slide and examined under a Zeiss microscope (x 10 or x 40) completely and systematically (Cheesbrough 1987).

II. Salt-flotation method

Saturated salt-solution (NaCl) was added to the pellet and the suspension was shaken a few times. The tube was filled up to the brim with solution and a coverslip placed on top. The coverslip was removed after 20 - 30 minutes and examined under the microscope (Thienpont *et al.* 1979; Ministry of Agriculture, Food and Fisheries 1986).

III. Zinc-sulphate flotation method

A zinc-sulphate solution was prepared (331g in 1000 ml H_2O) (Levine 1985). Exactly the same procedure as with the salt flotation method was followed (Levine 1985; Cheesbrough 1987; Garcia and Bruckner 1988). Additionally, some samples were treated

with hypersaturated zinc-sulphate solution. To produce this solution 84g zinc-sulphate were put in 100 ml water and dissolved while heating to 80°C and stirring (J. McGurry, Liverpool School of Tropical Medicine, personal communication).

IV. Formol-ether method

The pellet was mixed with 12 ml 10% formalin in a 15 ml centrifuge tube. Two ml of ether were added and the solution was shaken vigorously for at least one minute, releasing the air pressure in the tube occasionally. The tube was centrifuged for 5 min at 2000 rpm. The ring of floating detritus and then the formalin were decanted (Cheesbrough 1987; Garcia and Bruckner 1988). The remaining pellet was suspended in formol-saline up to a known volume (usually 0.5 ml). From this suspension 20µl were examined on a slide under a Zeiss microscope in a complete and systematic way. The number of eggs in the known volume was calculated by multiplying the number of eggs in 20µl by the appropriate factor (usually 25). The number of eggs per gram of faeces (e.p.g.) was obtained by using one gram of the already sieved and twice centrifuged faeces with the formol-ether method.

Eggs and protozoan cysts were identified by their morphology at x10 and x40 magnification (see chapter 3 and 7). Egg size was measured with a micrometer.

2.4 COMPARISON OF METHODS FOR BABOON SAMPLES

Ten formalin preserved baboon faecal samples were used for a preliminary comparison. In this test, salt-flotation and zinc-sulphate flotation methods detected very few eggs. This may be due to the formalin which alters the properties of eggs such as weight (Garcia and Bruckner 1988). Both the formol-ether technique and the direct smear yielded good results. Differences between these two methods were investigated by using 58 samples collected in 1989 and 41 samples collected in spring 1991. The latter were used to reduce possible effects of prolonged storage. Prevalences were compared using χ^2-tests and intensities were compared using Mann-Whitney *U*-tests. Results are shown in Figure 2.1.(a and b) and Table 2.1.(a and b). Overall differences between the two methods in detection of parasite eggs were small for most taxa with the exception of hookworm in the samples from 1989, where the direct-smear technique detected more hookworm eggs. This may be due to a more pronounced effect of formalin on eggs after longer preservation. Differences between the methods were smaller for the 1991 samples than for the 1989 samples. Overall, the direct smear technique shows certain nematode eggs such as hookworm, *Strongyloides* sp. and the unidentified larvae better than the

formol-ether technique. The formol-ether technique has been found to be better at detecting trematodes and most other parasites (Cheesbrough 1987; Jewsbury *et al.* 1991). All further analyses were carried out with the formol-ether technique as it seemed better at detecting a greater number of parasite taxa. Also, the prevalence data were considered more important than intensity data, and the formol-ether method was also easier to standardize as one could calculate the volume examined without difficulty. Due to the properties of this method the prevalence and intensity of hookworm, *Strongyloides* sp. and the unidentified larvae found in the baboons may be slightly underestimated in this study.

2.5 COMPARISON OF METHODS FOR LION SAMPLES

Eleven lion samples were analysed using the same four methods detailed above. The results are shown in Table 2.2. No eggs were detected with the direct-smear technique. Only a very small number of eggs was observed when using the saturated salt-solution flotation method. The saturated salt-solution flotation did not detect *Giardia* and *Entamoeba* cysts (Cheesbrough 1987). The zinc-sulphate flotation method did not show better results in recovery of eggs than the salt-solution. A slightly improved outcome was observed when using the hypersaturated solution of zinc-sulphate. However, some eggs, such as *Spirometra* eggs, were distorted by the hypersaturated solution. The flotation methods gave reasonable results for heavy infection but proved not to be very good in detecting light infections. An additional impediment was that other material floated to the surface as well as the eggs and obscured them. The best results for the lion faecal samples were found with the formol-ether method. Light as well as heavy infections could be observed. Furthermore, all parasite species could be detected with this method. No distortion of eggs occurred. Sand was a major contamination factor for the concentration technique, but the problem could be circumvented by dilution of the suspension. The choice of formol-ether as the best overall method coincides with the result of previous studies (Moriya 1954; Cheesbrough 1987; J. Martin *et al.* unpublished manuscript). Formalin, not ethanol or PVA, was chosen to fix the faecal material, because it is more easily available in Tanzania.

2.6 NUMBER OF SLIDES EXAMINED

With increasing number of slides of one faecal sample examined, the likelihood of parasite egg detection, especially of low intensity infection, increases. The choice of how many slides to examine is thus a trade-off between efficiency in reading slides from as many different individuals as possible and the detection of all parasites present. It is difficult to estimate how many slides to examine for mixed infections, as there are other factors than intensity of infection that influence detectability. For certain parasites more slides have to be examined than for others (Moriya 1954). Reading three slides was compared with reading ten slides from the same faecal sample. In four out of ten cases no more parasite taxa were discovered in ten slides than in three. In three cases one additional taxa was found and in three cases two more parasite taxa were detected. However, on average, 85% of the parasite taxa and always more than 60% were discovered on the first three slides. Only in cases when the intensity of a parasite taxon was very low were more than three slides required. All of the following analyses are based on three slides per sample.

2.7 REPEATABILITY OF EGG COUNTS

Because faecal 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 faecal sample, in different samples over one day, or over consecutive days. The repeatability for these three situations was calculated as follows: repeatability, or r, the intraclass correlation coefficient, which is based on variance components derived from a one-way analysis of variance (Sokal and Rohlf 1981; Lessells and Boag 1987), is given by

$$r = s^2_A/(s^2 + s^2_A)$$

where s^2_A is the among-groups variance component and s^2 is the within group variance component. Both values can be taken from most ANOVA tables of statistical packages (such as Statview II with $s^2 = MS_w$); r may vary between 0 and 1. If r = 1, it indicates that samples are identical. Faecal egg counts were log(x+1) transformed for analysis. If all samples from one individual were negative, this individual was excluded from the analysis as zero counts may bias the result.

Homogeneity of faecal samples

Although faeces were stirred by the field assistants before taking a faecal sample, variation within the stool was tested nevertheless. Two samples were each taken from different parts of eight faecal samples and analysed. The repeatability for each parasite was quite high (Table 2.3.). The slightly lower repeatability which was found for taxa richness than for each individual taxon is probably connected with the chances of detecting all species on the first three slides. Overall, samples taken from different areas of the faeces were very similar. This result supported the hypothesis that the distribution of eggs in the faeces is random. Given this result and the additional mixing of the faeces before collection, it was assumed that a cross-section of parasite eggs and protozoans of a whole stool could be detected in one sample and no additional sampling of the same stool was necessary.

Diurnal variation of parasite egg output

Variation in parasite egg output may be connected with the time of day an animal defaecates. For certain parasites, such as pinworm, diurnal variation in egg output has been reported (Hawking 1975). To test whether there was variation and/or a pattern of egg output during a day, several faecal samples from individual baboons were collected during one day on an opportunistic basis. Calculations of the repeatability (Table 2.4.) showed that samples of an individual over one day were fairly similar. Figure 2.2.a - d show the variation in egg output per individual over time for four of the parasite taxa. Some parasite taxa were more consistent in their egg output than others, for instance *Trichuris* sp. and *Streptopharagus* sp. were more consistent in the number of eggs emitted than hookworm or *Strongyloides* sp. Variability included some false negatives (e.g. a parasite taxon was not detected in one of the samples even though it had been found in the other samples from the same day).

In the fluctuations of egg output which were observed, no diurnal pattern of egg output was obvious. Therefore, time of collection was not controlled for in subsequent statistical analyses.

Repeatability of egg counts over several days

Variation of egg counts over a period of days was monitored. Fifty-seven samples were collected from 19 individuals whenever they were observed defecating, but never

more than one per day for a given individual, with the longest series for an individual being collected over 9 days. The repeatability of intensity over several days was considerably lower (Table 2.5.) than per stool or over one day. Some parasites, such as *Physaloptera* sp., showed hardly any repeatability. The actual number of parasites infecting the baboons is unlikely to have differed much over this relatively short period of time. Hence, a rapid change in parasite infection reflected in the parasite output is unlikely to be the reason for the variation. In a study on variation in parasite egg output of humans, considerable variation was observed over a period of five days (Hall 1981). No satisfactory explanation for these fluctuations has been put forward. One reason may be a day to day variation in egg production (Hall 1981). However, diurnal variation in samples is normally low (Table 2.4.), suggesting that if fluctuations in egg production occur, they tend to occur from one day to the next and not during one day. Another possible explanation for the variation observed is that the type of faeces - depending on the food ingested- may have an impact on the egg counts. The amount of water and fibre content will very likely have an impact on the estimate of parasite intensity. Fibre can increase the weight of faeces (Hall 1982). As the calculations refer to 1 gram of faeces, an increase of weight of faeces with the egg output constant will necessarily lead to a lower estimate of infection, while water may dilute the faeces and thus diminish faecal egg counts. On the other hand, other authors considered the correction of egg counts due to consistency of faeces unnecessary (Scott and Headlee 1938). Water content should not have presented a problem in this study, because of the centrifugation of the faecal sample before analysis.

One has to be aware of the variability of parasite egg outputs over several days when interpreting the results of this study. However, as numerous egg counts from one population are used, variations in egg counts due to different foods and female parasite behaviour should not have such a large impact. The mean egg count of faecal samples collected from the baboon population and from each troop will still give a useful indication of the parasite population dynamics of this community (Hall 1982), even though it may be more difficult to specify the exact intensity of infection of one individual from one or two samples.

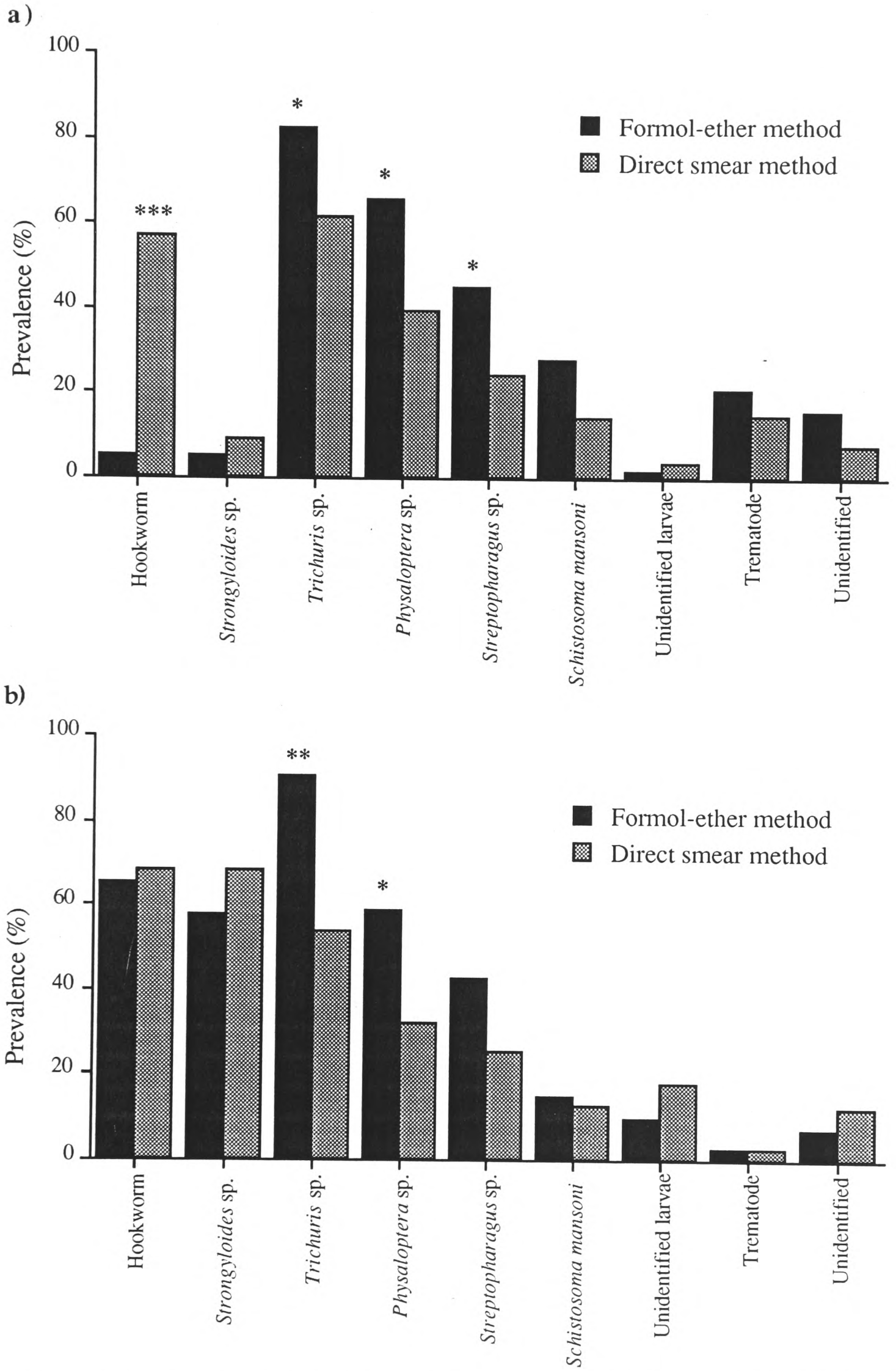


Figure 2.1. Comparison of prevalences between the formol-ether method and the direct smear method for a) samples from 1989 b) samples from spring 1991. Significant differences in χ^2 - test are indicated by * - $p< 0.05$, ** - $p<0.01$,*** - $p<0.001$

Table 2.1. Comparison of egg counts between the formol-ether method and the direct smear method using a Mann-Whitney *U* test for a) samples from 1989 and b) samples from spring 1991.
Significantly lower "-" and significantly higher "+" intensities with the formol-ether method are reported. Signs in brackets indicate non-significant direction of difference.

a)

parasite	Z-value (n = 30)	p-value	higher intensity for formol-ether
Total helminth load	- 2.53	0.012	-
Hookworm	- 6.07	0.0001	-
Strongyloides sp.	- 4.45	0.0001	-
Trichuris sp.	- 0.56	0.58	(+)
Physaloptera sp.	- 0.53	0.60	(+)
Streptopharagus sp.	- 0.86	0.39	(+)
Schistosoma mansoni	- 1.25	0.21	(+)
Trematode	- 0.72	0.72	(+)
Unidentified larvae	- 0.15	0.15	(-)
Unidentified	- 0.42	0.42	(+)

b)

parasite	Z-value (n = 22)	p-value	higher intensity for formol-ether
Total helminth load	0	1	=
Hookworm	- 2.60	0.009	-
Strongyloides sp.	- 1.67	0.096	(-)
Trichuris sp.	- 0.19	0.85	(+)
Physaloptera sp.	- 0.89	0.38	(+)
Streptopharagus sp.	- 0.15	0.89	(-)
Schistosoma mansoni	- 0.052	0.96	(+)
Trematode	*		
Unidentified larvae	- 0.50	0.55	(-)
Unidentified	- 0.33	0.97	(+)

* too few infected

Table 2.2. Comparison of methods for lion faecal samples.
Each row gives the number of parasite eggs recovered from one sample. The columns for NaCl, $ZnSO_4$ and hypersaturated $ZnSO_4$ report the total number of eggs detected. For the formol-ether method, first the total number of eggs calculated up to the volume of the pellet is reported and then in parentheses the average number of eggs per three slides.

NaCl	$ZnSO_4$	hypersaturated $ZnSO_4$	Formol-ether
1	-	1	3 (1.6)
91	-	59	69 (10.7)
-	-	-	-
-	-	-	-
7	5	89	4680 (117)
3	-	28	357 (22)
3	1	1	276 (6)
1	-	-	113 (5)
26	5	77	2970 (270)
11	1	6	48 (3)
10	5	-	2904 (33)

Table 2.3. Repeatability of egg counts from the same baboon stool.

Parasite Parameter	F-ratio	df	r
Total helminth egg count	8.98	7,8	0.80
Hookworm	*	*	*
Trichuris sp.	8.25	6,7	0.79
Strongyloides sp.	12.23	6,7	0.84
Physaloptera sp.	13.23	3,4	0.86
Streptopharagus sp.	4.28	5,6	0.62[x]
Schistosoma mansoni	*		*
Trematode	*		*
Number of species: protozoans included	6.51	7,8	0.57
Number of species without protozoans	3.65	7,8	0.50

n = 8 x 2 samples per individual

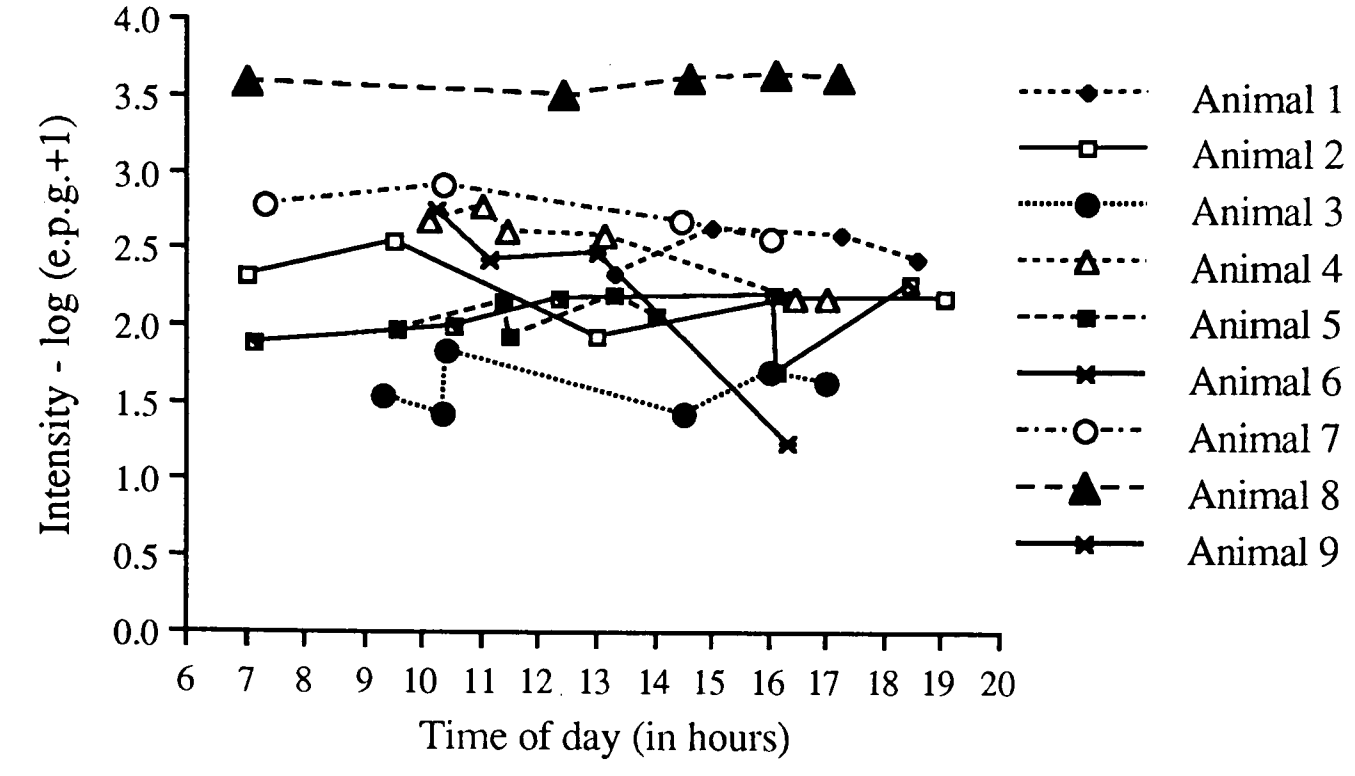
* too few infected (< 4 samples from different stools)
x with outlier, when outlier removed r = 0.94

Table 2.4. Repeatability of samples collected over a day from a single baboon.

Parasite Parameter	F-ratio	df	r
Total helminth egg count	39.48	8,36	0.89
Hookworm	9.29	8,36	0.62
Trichuris sp.	22.27	8,36	0.81
Strongyloides sp.	3.52	3,15	0.35
Physaloptera sp.	10.43	7,33	0.65
Streptopharagus sp.	58.11	5,22	0.92
Schistosoma mansoni	(53.56	1,8	0.92)
Trematode	(0.14	1,7	0.24)
Number of species: protozoans included	14.14	8,36	0.73
Number of species without protozoans	18.30	8,36	0.78

Numbers samples per individual per day differed slightly: 3 individuals were sampled x 4 & 3 x 5 & 3 x 6
Two parasite taxa, *Schistosoma mansoni* and the trematode, only occurred twice in the sampled animals, hence these results have to be treated with caution.

a)



b)

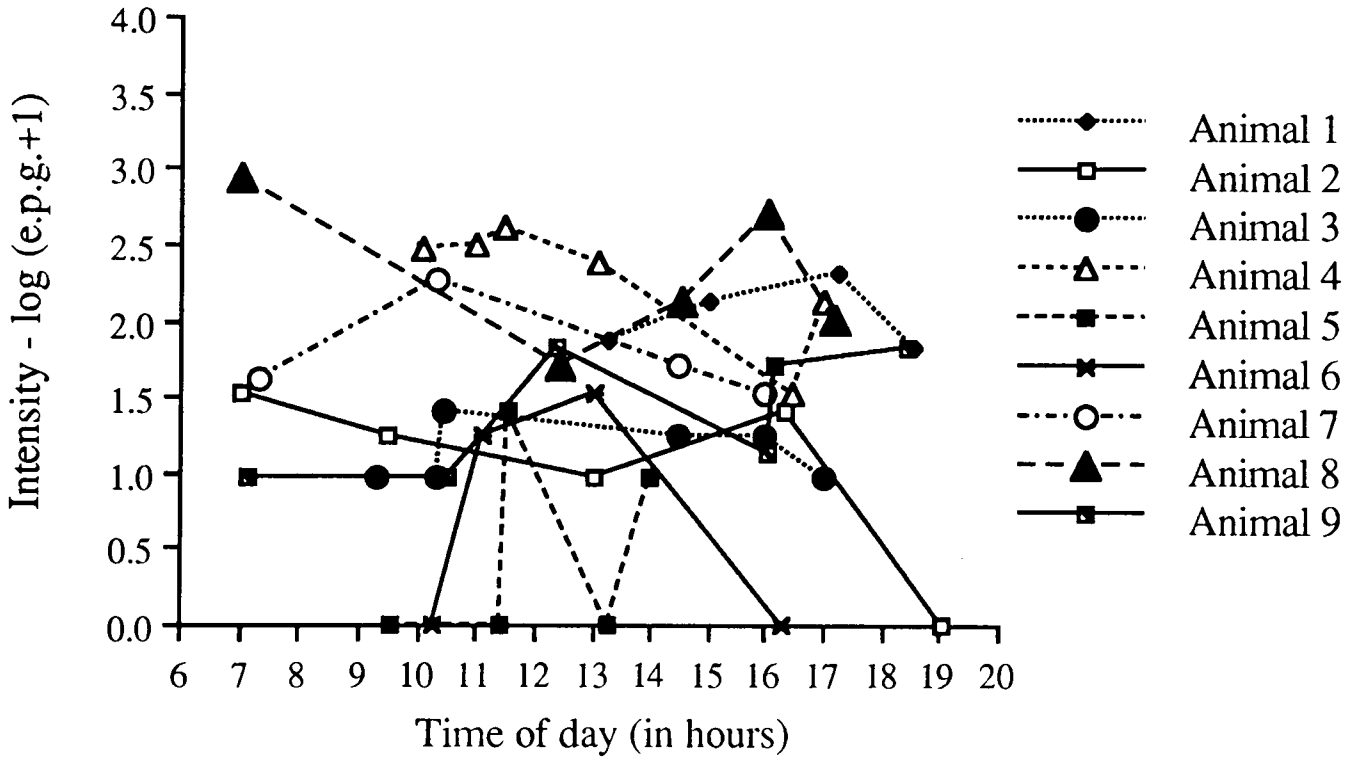
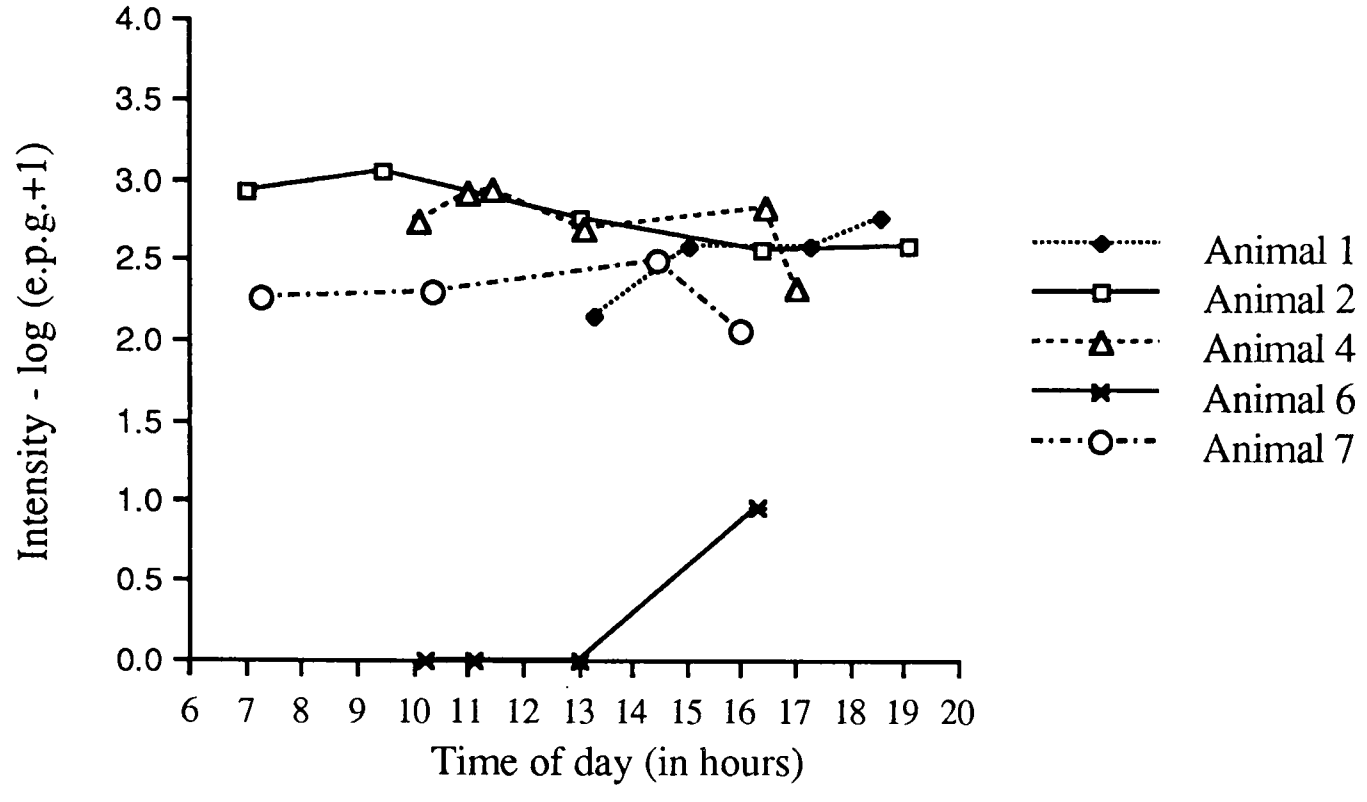


Figure 2.2. Variation of egg output over one day for a) *Trichuris* sp. and b) hookworm. E.p.g. (eggs per gram of faeces) were log(x+1) transformed. Uninfected animals were excluded from the graphs.

c)



d)

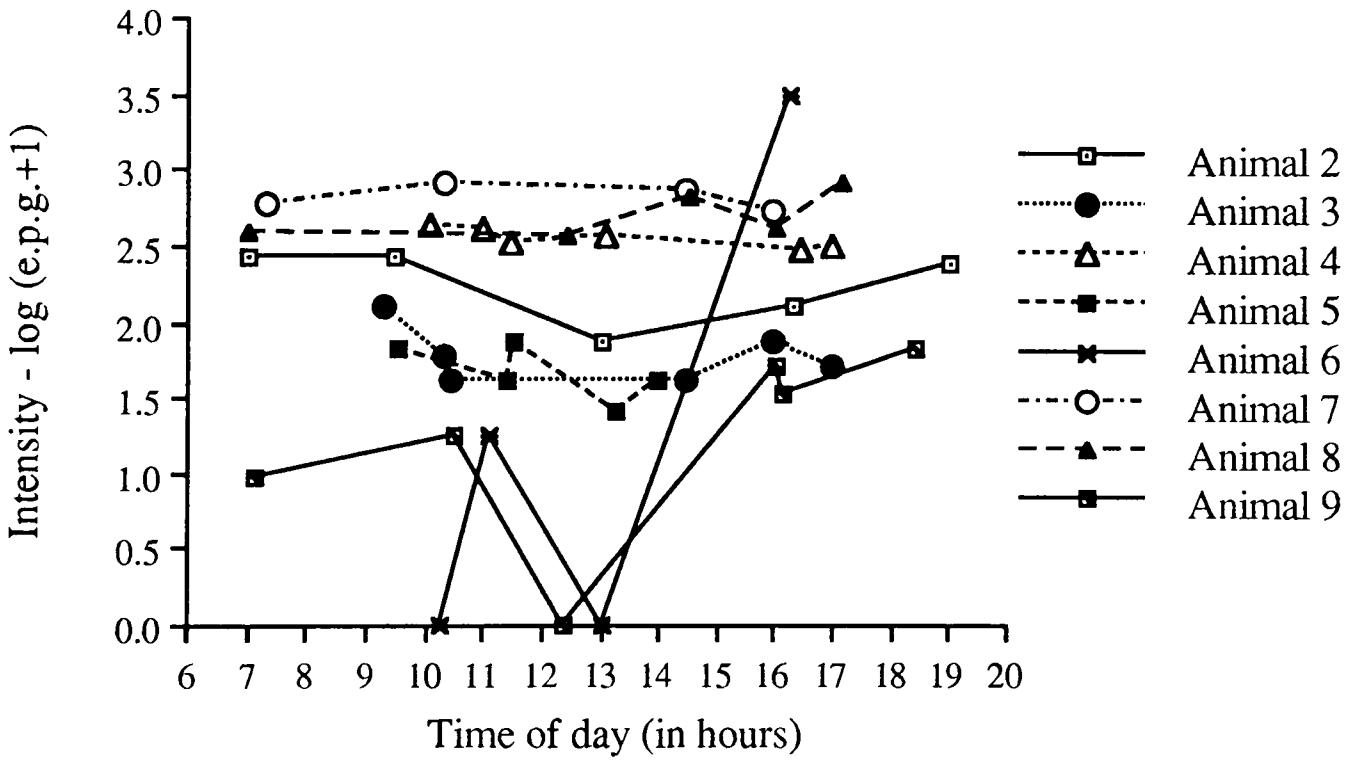


Figure 2.2. Variation of egg output over one day for c) *Streptopharagus* sp. and d) *Physaloptera* sp. E.p.g. were log (x+1) transformed. Uninfected animals were excluded from the graph.

Table 2.5. Repeatability of samples over several days.

Parasite Parameter	F-ratio	df	r
Total helminth egg count	3.17	19,38	0.43
Hookworm	0.39	13,28	0.22
Trichuris sp.	2.40	19,38	0.33
Strongyloides sp.	1.32	16,34	0.18
Physaloptera sp.	0.78	18,36	0.09
Streptopharagus sp.	3.39	18,37	0.45
Schistosoma mansoni	1.67	4,18	0.16
Trematode	2.16	4,10	0.29
Number of species: protozoans included	2.19	19,38	0.29

Numbers of repeated samples per individual differed: 8 individuals were sampled x 2 & 6 x 3 & 2 x 4 & 2 x 5 & 1 x 6

CHAPTER 3

Baboon parasites

ABSTRACT

Seven different helminth eggs were distinguished in the faeces of the Gombe baboons: *Trichuris* sp., *Strongyloides* sp., *Physaloptera* sp., *Streptopharagus* sp., *Schistosoma mansoni*, another unidentified trematode and hookworms and hookworm-like nematodes, which are combined in the category hookworm. Also, some unidentifiable helminth larvae were detected in the faeces as well as several protozoans, which could be only distinguished as two types: *Balantidium coli* and cysts. The cysts could have been from several different species of protozoans such as the amoebae *Entamoeba coli, Entamoeba histolytica* and *Iodamoeba bütschlii* and from a flagellate protozoan *Chilomastix mesnili*. The morphology of parasite eggs is reported as a guide-line for identification together with the mode of transmission.

3.1 INTRODUCTION

Whereas diseases in captive baboons have been studied extensively (Vagtborg 1965; Fiennes 1967), little is known about parasite infections in wild baboons. "In short, we know a lot about parasitism and parasitic disease in laboratory and zoo animals but amazingly little about natural parasitism in primates" (Dunn 1970). Some field studies have been undertaken (e.g. Kuntz and Myers 1967; Myers and Kuntz 1967; Myers *et al.* 1971; Goldsmid and Rogers 1978; Meade 1983; Pettifer 1984; Appleton *et al.* 1986; Eley *et al.* 1989; Appleton *et al.* 1991; for a review of previous studies see chapter 4). These studies, including two studies of primate parasites in Gombe (McGrew *et al.* 1988; Murray 1991), were used as background information about possible parasite infections in the Gombe baboons. Additionally, a list of potential parasites in the baboons in Gombe was compiled from several lists dealing with parasitic infections of primates and more specifically baboons (Yamashita 1963; Myers 1965; McSwain 1983; Owen 1992) and from the parasite catalogue of the Natural History Museum in London which contains a list of parasite species reported for the genus *Papio*.

Most literature on helminth parasites concentrates on the morphology of adult worms but lacks descriptions of egg morphology. Identification of parasites by egg morphology was made according to descriptions and photographs of baboon parasite eggs (S.File, personal communications; Jessee *et al.* 1970; Goldsmid and Rogers 1978; Grove 1989) and information from medical parasitology because of similarities between human and baboon parasites. However, no illustration of the eggs of some parasites could be found, which additionally made identification difficult.

An overview of the baboon parasite taxa found in this study is given in Table 3.1., according to taxonomies of Cheng (1986) and Kreier and Baker (1987). Unidentified larvae, categorized as a group, are not separately included in the table as they probably belong to either *Strongyloides* or hookworm taxa. Information about parasite egg morphology and possible taxonomic status is followed by a characterisation of the mode of transmission and any special features which may relate to host-parasite interactions are briefly described. Helminths with direct life cycles will be discussed first, then helminths with indirect life cycles and finally protozoans. A detailed description of the eggs according to which parasites were identified is given (Table 3.2. shows the egg measurements). A knowledge of the biology of the parasite is crucial for understanding host-parasite interactions.

3.2 BABOON PARASITES

Trichuris sp.

Trichuris sp. (order: Trichocephalida; class: Adenophorea) eggs are brownish, lemon-shaped eggs with protruding ends and transparent plugs on each pole. The wall is thick and smooth. The content is granular and unsegmented (Plate 3.1).

Trichuris trichiura has been reported from baboons (Myers 1965; Soulsby 1982; Appleton 1989; Owen 1992). *Trichuris cynocephalus* (Yamashita 1963; McSwain 1983) has also been found in *Papio cynocephalus* (conspecific with or closely related to *Papio anubis* (Haltenorth and Diller 1977), depending on the taxonomy used). Because *Trichuris trichiura* is more common, it is likely that the *Trichuris* eggs found in the Gombe baboons belong to this species.

Trichuris has a direct life cycle. Infections are acquired by ingestion of eggs. The prepatent period is approximately 3 months. An adult female's egg production ranges from 1000 to 7000 eggs per day (Soulsby 1982; Schmidt and Roberts 1989).

Hookworm

Hookworm (order: Strongylida; class: Secernentea) eggs are ovoid with rounded poles and thin smooth walls. They appear transparent. Internal segments, usually 2 - 8 blastomeres, but sometimes only a mass of cells, can be seen (Plate 3.2.).

Several nematode eggs are indistinguishable from hookworm eggs (Goldsmid and Rogers 1978; Goldsmid 1991), therefore these eggs were categorised as hookworm. In this study these might include *Necator americanus*, *Oesophagostomum* spp. and *Trichostrongylus* spp. The latter two are not hookworms taxonomically, but their eggs are so similar to hookworms' that they have been reported as hookworm in previous studies (Goldsmid 1991). It has not been established whether all the taxa in this category could actually be found in Gombe. However, an adult worm of the genus *Oesophagostomum*, which could be *Oesophagostomum stephanostomum*, was identified from the baboon faeces (L. Gibbons, International Institute of Parasitology, St.Albans, personal communication). The occurrence of *Trichostrongylus* spp. is not very likely as previous studies have not found *Trichostrongylus* spp. in Gombe. Moreover, the size and shape of *Trichostrongylus* spp. should make it possible in some cases to distinguish these eggs from eggs of other taxa in the category (Goldsmid and Rogers 1978; Goldsmid 1991).

Infection with *Necator* occurs mainly through skin-penetration by infective larval stages. Parasites mature after a prepatent period of at least 5 weeks. Female *Necator americanus* can produce up to 9000 eggs a day. Infections of *Oesophagostomum* and *Trichostrongylus* are acquired by the ingestion of infective larvae.

Strongyloides sp.

Strongyloides sp. (order: Rhabditia; class: Secernentea) eggs are slightly smaller than hookworm eggs and are elliptical with very rounded, almost square, ends. They have a thin single wall and look transparent. The egg contains a larva, quite often in the form of a figure eight (Plate 3.3.).

The eggs found in the baboon faeces are very likely to be *Strongyloides fuelleborni*. According to one survey *Strongyloides fuelleborni* is a common parasite of primates in Africa (Pampiglione and Ricciardi 1972). *Strongyloides stercoralis*, another *Strongyloides* reported from primates, shows larvae but no eggs in the faeces (Soulsby 1982). Therefore, some of the larvae may be *Strongyloides stercoralis*.

Transmission of *Strongyloides* is similar to that of hookworms. Infective larvae penetrate the skin of a host and migrate through the body to the intestines. The prepatent period lasts approximately five to seven days. Oral and lactogenic infection may occur as well. However, *Strongyloides* sp. are different in that they have completely free-living and completely parasitic forms. Parasitic females are parthenogenetic and their eggs produce either a new generation of infective larvae or free-living males and females, which are capable of generating parasitic or free-living forms.

Physaloptera sp.

Physaloptera sp. (order: Spirurida; class: Secernentea) eggs are round to oval in shape. They have a thick wall and contain a smooth coiled larva. There is a thick, rough, transparent outer layer around the wall which looks almost as if it just debris and does not belong to the egg (Plate 3.4.). Because of this appearance eggs may be mistaken for *Ascaris* eggs (Meade 1983; Murray 1991).

It was assumed that the parasite detected is the species *Physaloptera caucasica*, because it has been reported as the main species of this genus infecting baboons (Kuntz and Myers 1966; Kuntz and Myers 1967; Goldsmid and Rogers 1978; Pettifer 1984; Appleton *et al.* 1986; Owen 1992), although other species of *Physaloptera* have been reported from other primates (Yamashita 1963; Myers 1965; Windle *et al.* 1970). There

has been confusion over the nomenclature of *Physaloptera caucasica;* the same species was also reported as *Abbreviata caucasica* (Yamashita 1963; Goldsmid and Rogers 1978; Soulsby 1982; McSwain 1983). According to Soulsby (1982) *Physaloptera caucasica* is a synonym of *Abbreviata caucasica.*

The life cycle of *Physaloptera* most likely involves an insect intermediate host, such as an orthopteran or coleopteran and may possibly also entail a paratenic vertebrate host (Windle *et al.* 1970; Schmidt and Roberts 1989). It has been argued by Krupp (1962) that it may be transmitted directly as well. Baboons become infected by eating the intermediate or paratenic host (a transport host which may be infected by the parasite between intermediate or definitive host without the parasite undergoing any development).

Streptopharagus sp.

The eggs of *Streptopharagus* sp. (order: Spirurida; class: Secernentea) are oval in shape with a smooth outside and a thin shell, being filled completely by a larva inside (Plate 3.5.a and b).

Three species of *Streptopharagus* have been reported for primates: *S. armatus*, *S. pigmentatus* and *S. baylisi* (and/or *S. sandgroundi* in Yamashita (1963); Yamashita considers *S. pigmentatus* and *S. armatus* to be the same species) (Soulsby 1982; McSwain 1983). *S. pigmentatus* appears to be the species most frequently found in previous studies (Kuntz and Myers 1967; Pettifer 1984; Appleton *et al.* 1991).

The life cycle is indirect but not well known. It is believed that an arthropod is the intermediate host (Soulsby 1982).

Schistosoma mansoni

Schistosoma mansoni (order: Digenea; class: Trematoda) eggs are large with a distinctive shape. They are ovoid with one pole round and one relatively pointed. The eggs have a protruding spine on one side near the round pole. Their shell is smooth and thin and the inside filled with a miracidium (Plate 3.6.). Because of the unique shape of the egg the parasite could be identified to species level.

Schistosoma mansoni has an indirect life cycle with snails (*Biomphalaria* spp.) as intermediate hosts. The prepatent period lasts 5 - 8 weeks. In humans, females produce 100 - 300 eggs per day (Hairston 1965; Anderson and May 1991).

Unidentified Trematode

The eggs of the unidentified trematode (order: Digenea; class: Trematoda) have a smooth shell with medium thickness inside which a granular droplet-like content can be seen. The egg is brownish in colour and the shell is slightly thickened on each pole, with an indistinct operculum (Plate 3.7.).

Several trematodes other than schistosomes have been reported from baboons (Myers 1965; Cosgrove 1966). These are *Dicrocoelium lanceatum,* other Dicrocoeliidae, *Watsonius watsonius*, *W. deschiensi*, *Fasciola lanciniata, Brodenia lanciniata* and *Paragonimus africanus*. However, no photographs or measurements of eggs could be found for a comparison.

The life cycle of digenetic trematodes is indirect with at least one intermediate host, and it is very likely that at least one of the intermediate hosts is a mollusc (Dönges 1988; Mehlhorn and Piekarski 1989).

Unidentified Larvae

A number of larval helminths were detected in the faeces. They could not be distinguished because they were at a very early stage in their development. However, it is suggested that they are either early stages of hookworms or *Strongyloides stercoralis,* because these are the taxa reported from baboons which can produce larvae in the faeces.

Balantidium coli

Balantidium coli (order: Trichostomatorida; class: Kinetofragminophorasida) cysts and trophozoites were found in the faeces. Trophozoites were recognisable by the cilia surrounding an irregular, tapered cylindrical shape (Plate 3.8.a). The cyst has a grey colour and amorphous granular contents with some bubble-like features (Plate 3.8.b). Sometimes an outer transparent layer could also be observed.

Balantidium coli is directly transmitted (Schmidt and Roberts 1989) by ingestion of trophozoites or cysts.

"Amoebae"

The protozoans contained in the category "amoebae" all had similar round to oval cysts with one to several nuclei. The cysts could only in one case be identified down to species levels (Plate 3.9.). *Entamoeba coli, Entamoeba histolytica, Iodamoeba bütschlii, Endolimax nana, Entamoeba chattoni, Entamoeba gingivalis, Dientamoeba fragilis, Entamoeba polecki* and *Entamoeba hartmanni* (order: Amoebida; class: Lobosea) can all infect baboons (Myers 1965). *Chilomastix mesnili* (order: Retortamonadida; class: Zoomastigophorea) has also been reported from baboons, but does not belong to the Endamoebidae family. It is a flagellate protozoan of the family Retortamonadidae. However, because of the close resemblance to all the other cysts, it is put into this category of "amoebae", which is a morphological category rather than a taxonomic one. *Entamoeba coli* is probably among the detected cysts, based on size and morphological features (M.Anwar, University of Oxford, personal communication) as well as because of its previous discovery in Gombe. *Iodamoeba bütschlii* was also found in Gombe (McGrew *et al.* 1988).

All these protozoans are passed on directly by ingestion of cysts in contaminated matter. *Entamoeba histolytica* can also be transmitted indirectly by various diptera (Soulsby 1982).

Table 3.1. Parasite taxa and their life cycles.

Parasite	Order	Class	Life cycle	Mode of transmission
"Amoebae" *Entamoeba coli* *Entamoeba histolytica* *Iodamoeba bütschlii*	Amoebida	Lobosea	direct	through ingestion of cysts, (*E. histolytica* also can be transmitted by flies)
Chilomastix mesnili	Retortamonadida	Zoomastigophorea	direct	through ingestion of cysts
Balantidium coli	Trichostomatorida	Kinetofragminophorasida	direct	through ingestion of trophozoites and cysts
Trichuris spp.	Trichocephalida	Adenophorea	direct	through ingestion of eggs
Strongyloides spp.	Rhabiditia	Secernentea	direct	through penetration of skin by larvae or ingestion of larvae or transmammary transmission
Hookworm: *Necator americanus* *Oesophagostomum* spp. *Trichostrongylus* spp.	Strongylida	Secernentea	direct	through penetration of skin by larvae or ingestion of larvae (also transmammary transmission)
Streptopharagus spp.	Spirurida	Secernentea	indirect	infection through ingestion of intermediate host (arthropod)
Physaloptera caucasica *(Abbreviata caucasica)*	Spirurida	Secernentea	indirect	infection through ingestion of intermediate host (Orthoptera or Coleoptera) or possibly a paratenic host
Schistosoma mansoni	Digenea	Trematoda	indirect	infection through penetration of skin by cercariae; intermediate host: snail (*Biomphalaria* spp.)
Trematode unidentified	(Digenea)	Trematoda	indirect	possibly a mollusc as an intermediate host

3.2. Table of parasite egg and cyst measurements.
The range is reported and in brackets the mean and standard error.

Parasite	length in µm	width in µm	n
Trichuris sp.	47.4 - 63.8 (55.4 ± 0.4)	24.3 - 30.4 (26.7 ± 0.2)	51
Hookworm	57.8 - 82.7 (70.0 ± 1.1)	38.9 - 57.8 (45.8 ± 0.8)	39
Strongyloides sp.	45.6 - 66.9 (52.6 ± 0.7)	30.4 - 38.0 (34.1 ± 0.3)	52
Physaloptera sp.	47.1 - 69.9 (60.8 ± 0.6)	39.5 - 57.8 (48.9 ± 0.5)	52
Streptopharagus sp.	36.5 - 42.6 (40.4 ± 0.2)	21.3 - 24.3 (22.3 ± 0.2)	51
Schistosoma mansoni	124.6 - 176.3 (151.5 ± 4.3)	57.8 - 69.9 (63.4 ± 1.0)	12
unidentified trematode	80.7 - 109.6 (94.2 ± 2.2)	54.8 - 70.0 (59.0 ± 1.3)	11
Balantidium coli	diameter:	30.4 - 51.7 (39.6 ± 1.8)	10
"Amoebae" cysts	diameter:	7.0 - 10.0 (8.4. ± 0.4)	7

Plate 3.1. *Trichuris* sp. egg.

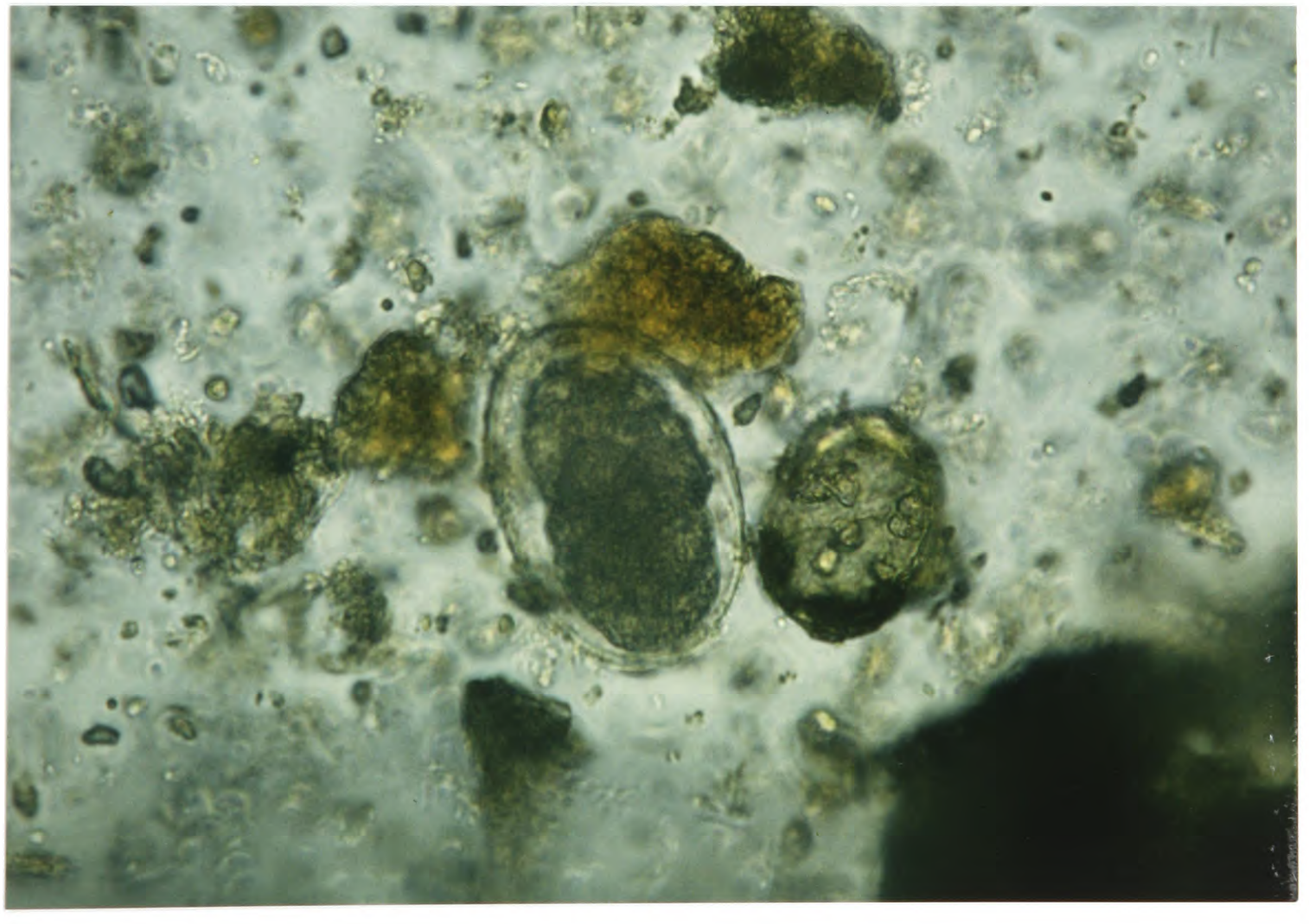


Plate 3.2. Hookworm egg.

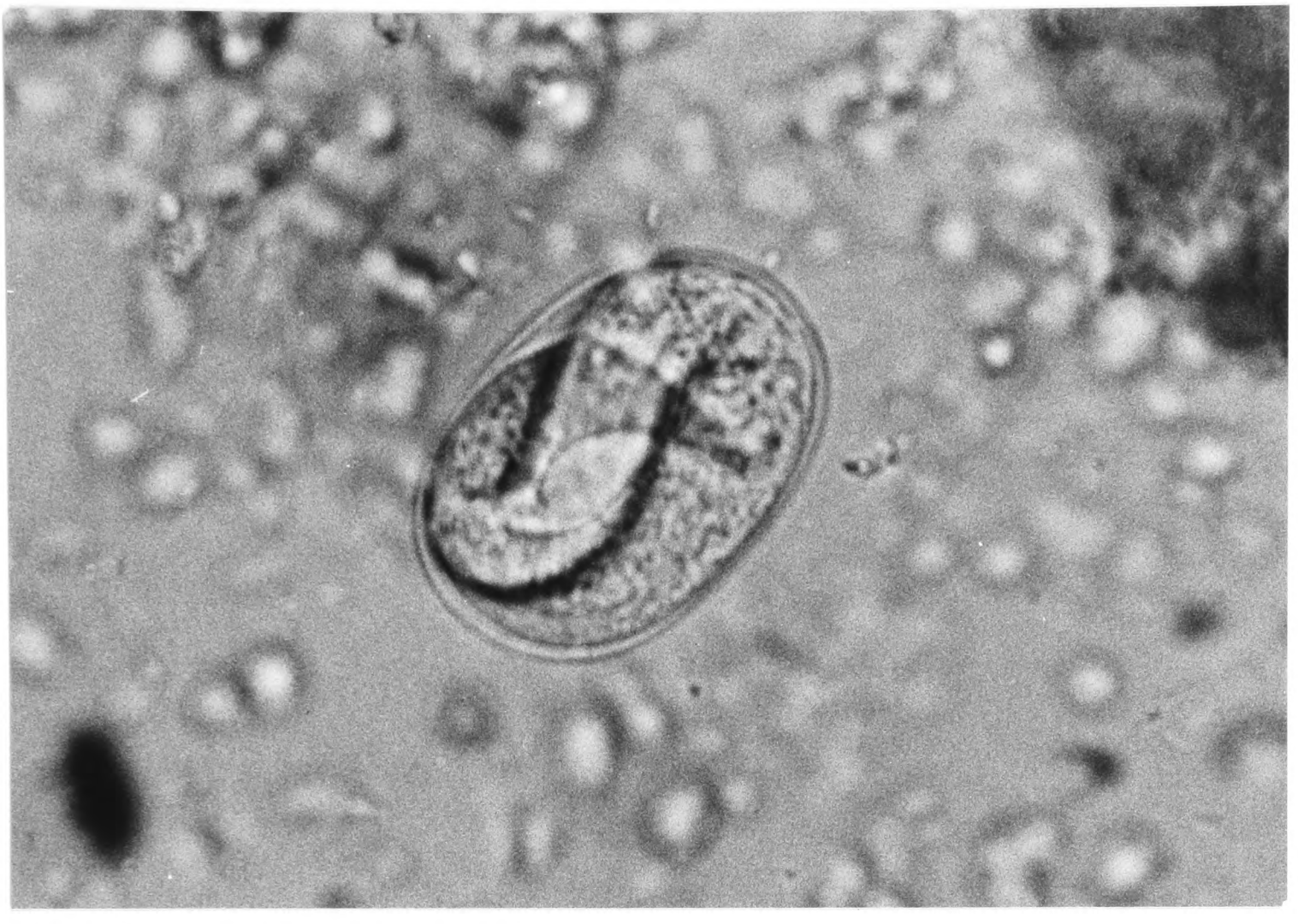


Plate 3.3. *Strongyloides* sp. egg.

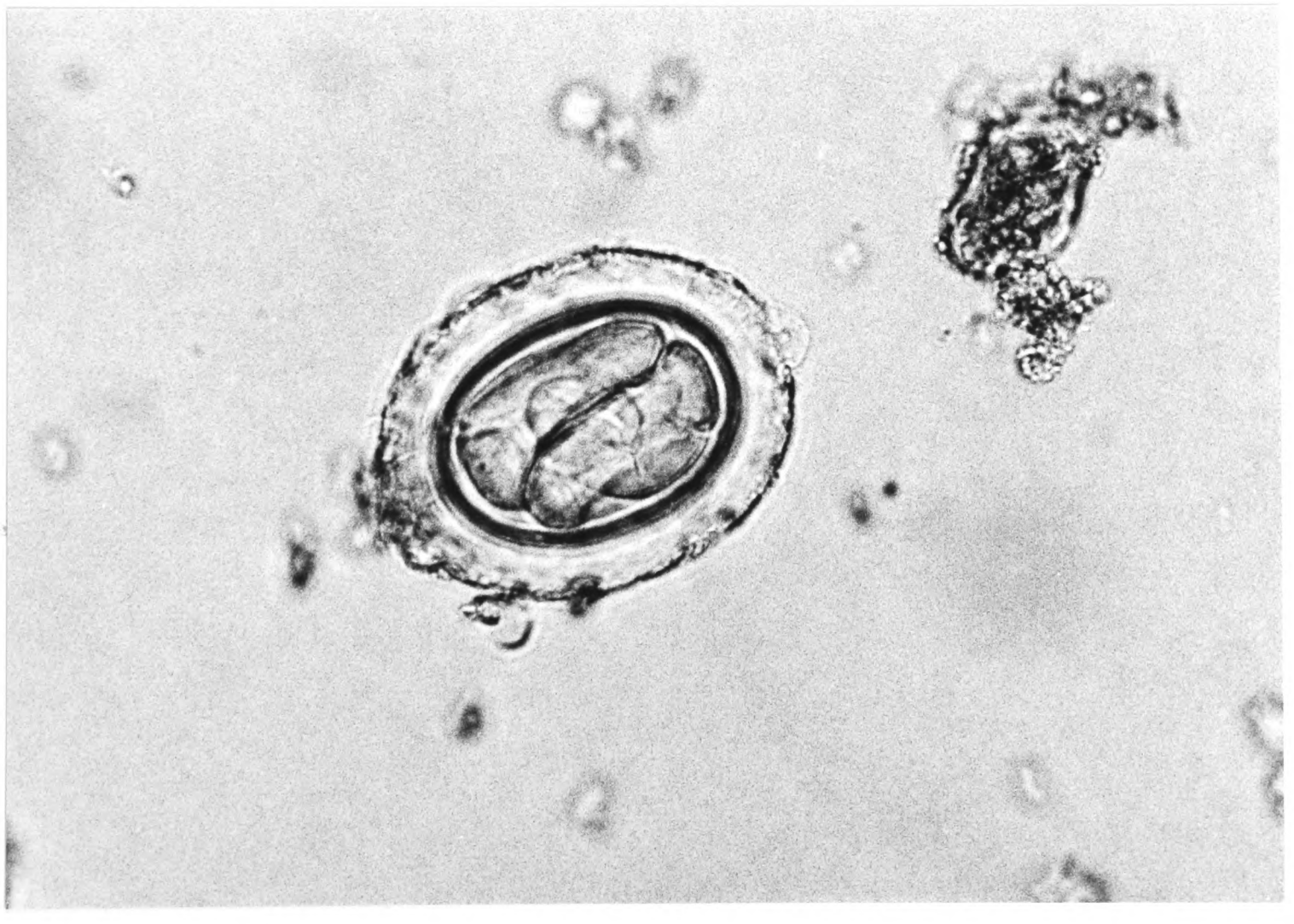


Plate 3.4. *Physaloptera* sp. egg.

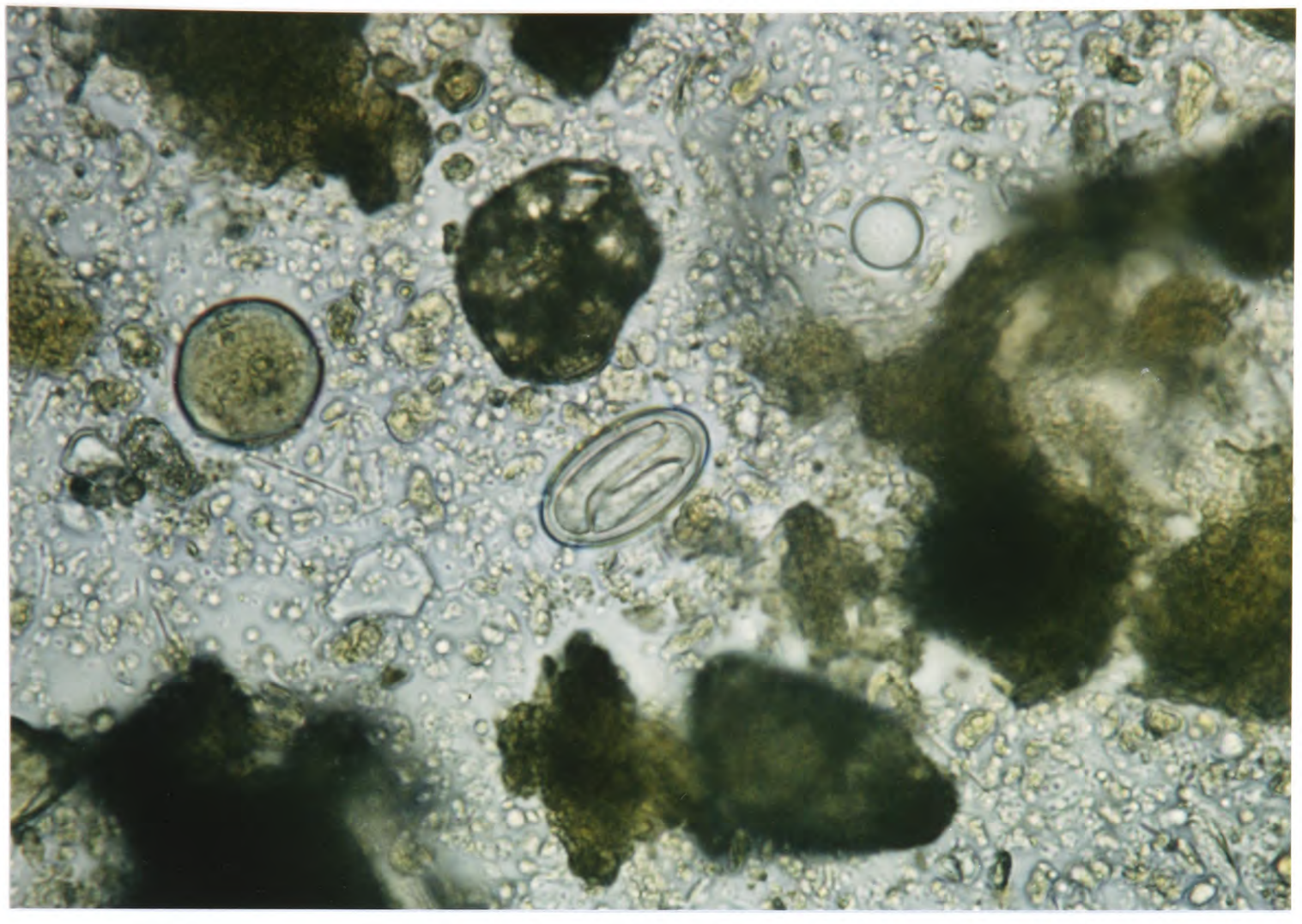


Plate 3.5. a) *Streptopharagus* sp. egg.

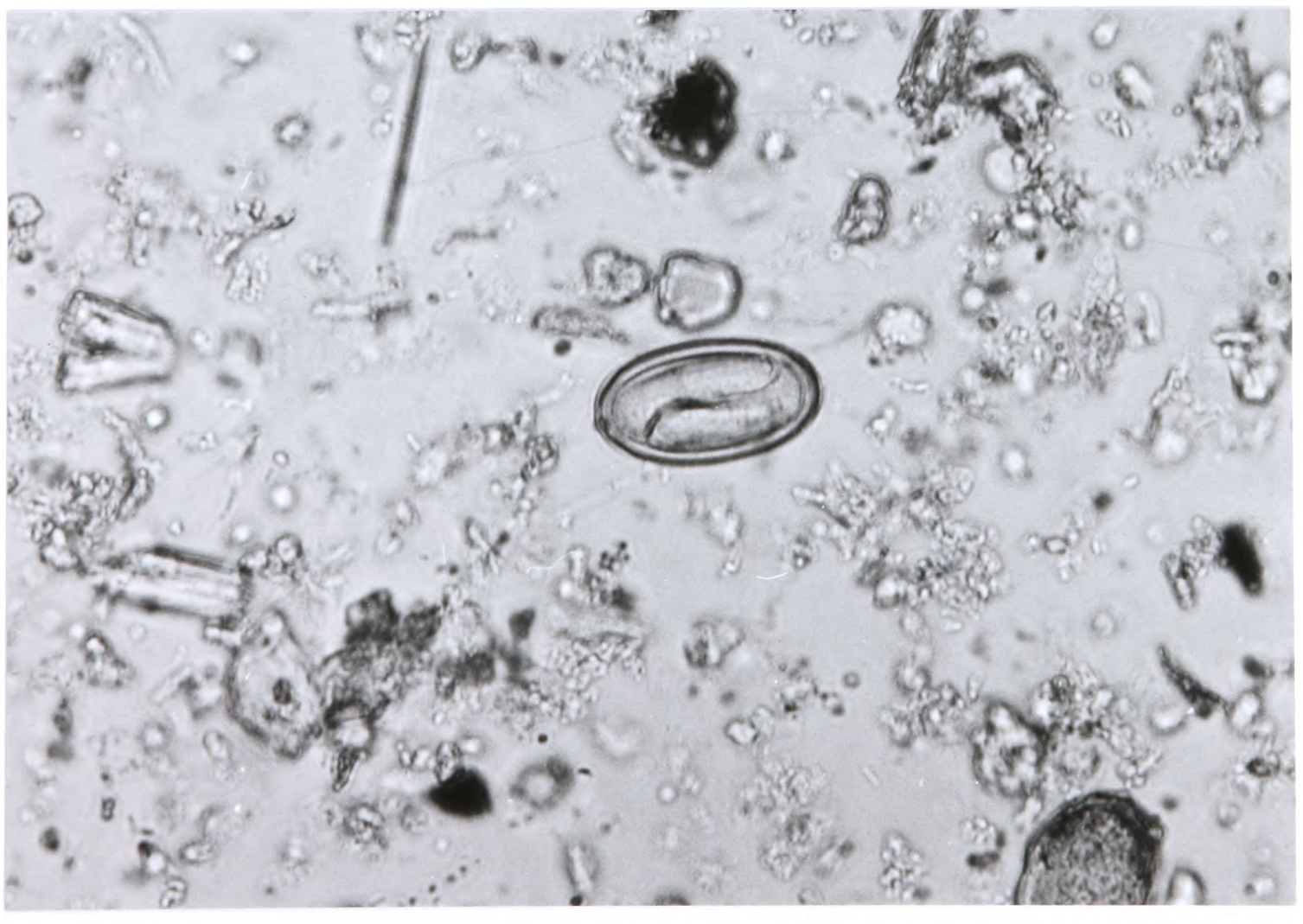


Plate 3.5. b) *Streptopharagus* sp. egg.

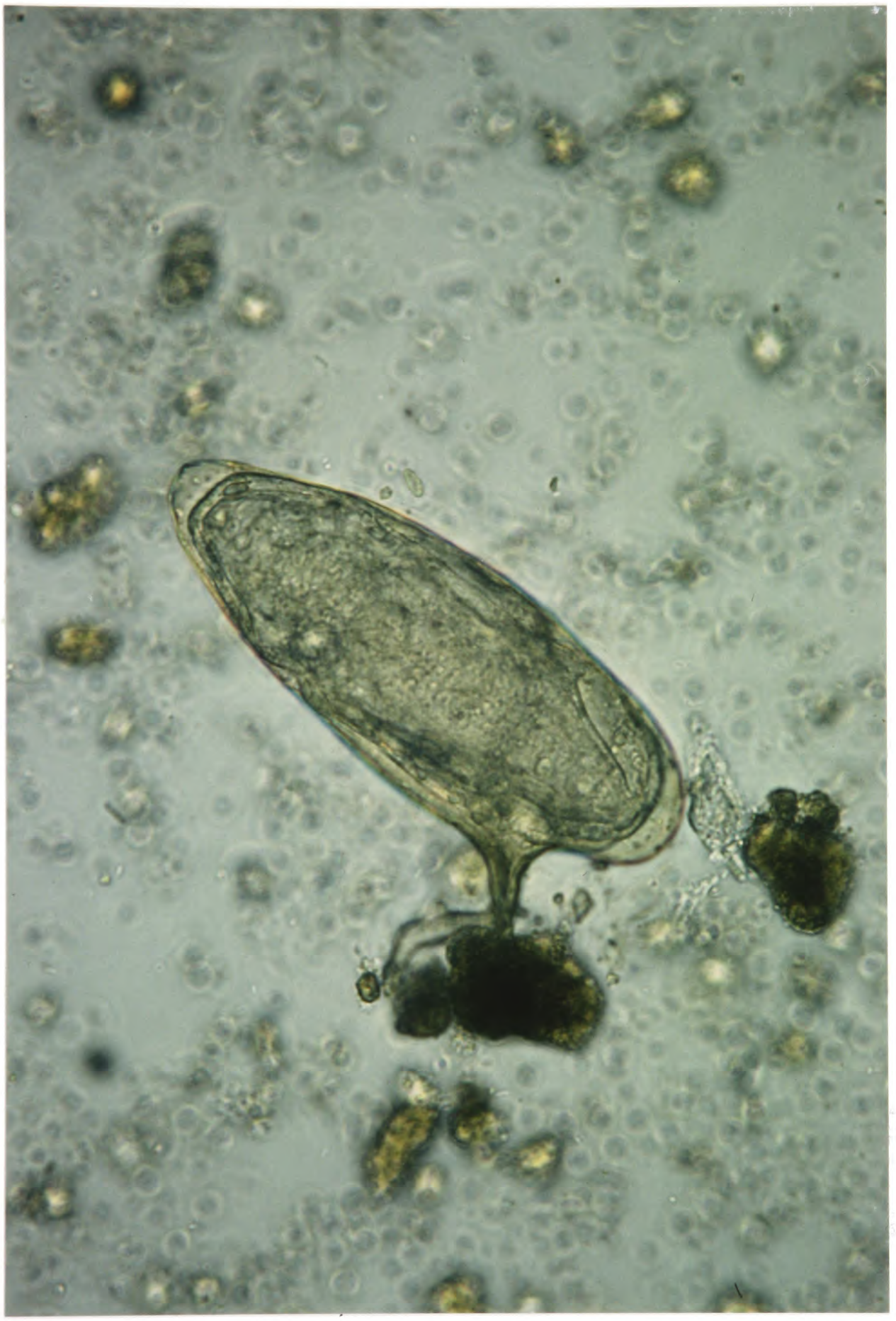


Plate 3.6. *Schistosoma mansoni* egg.

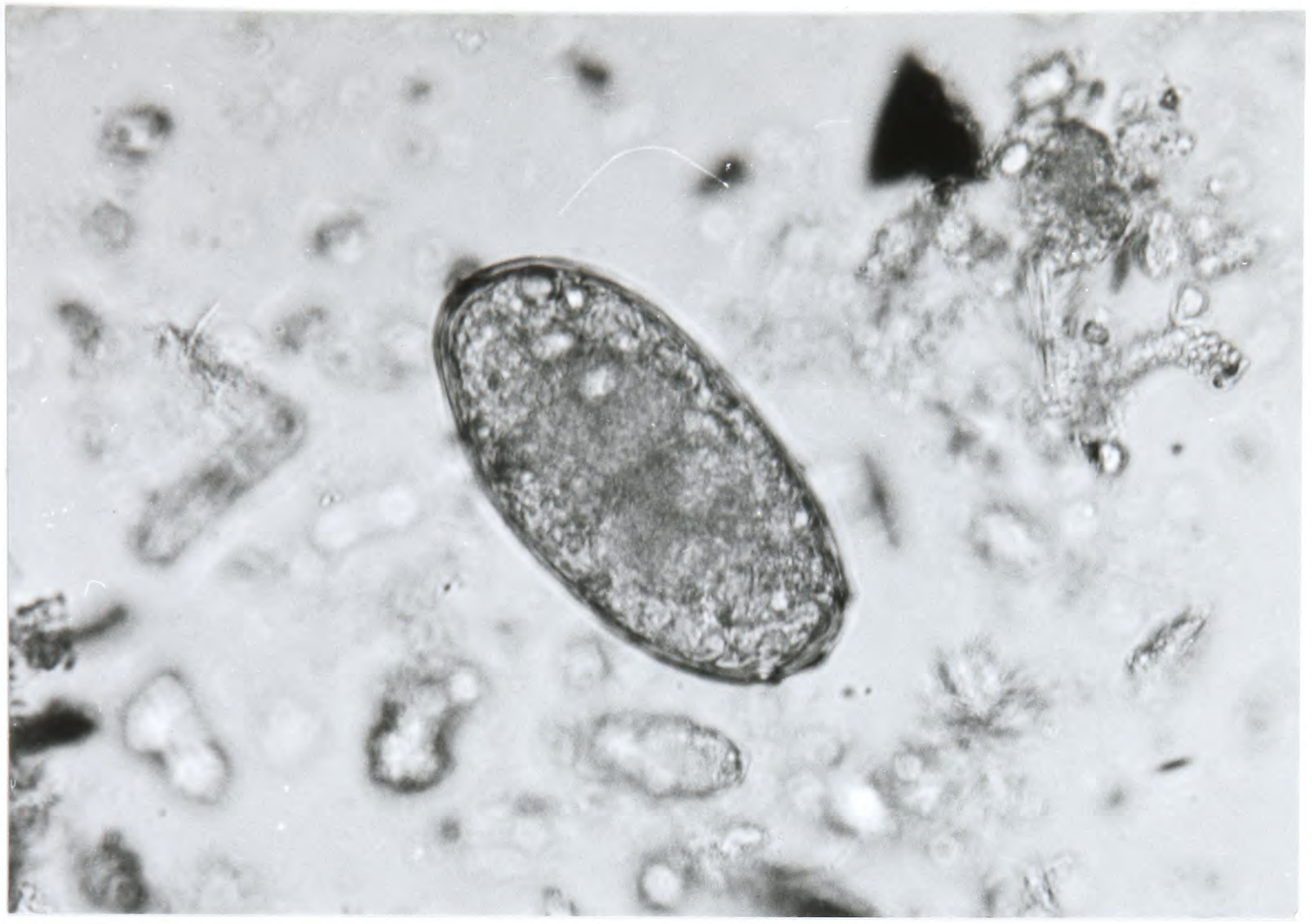


Plate 3.7. unidentified trematode egg.

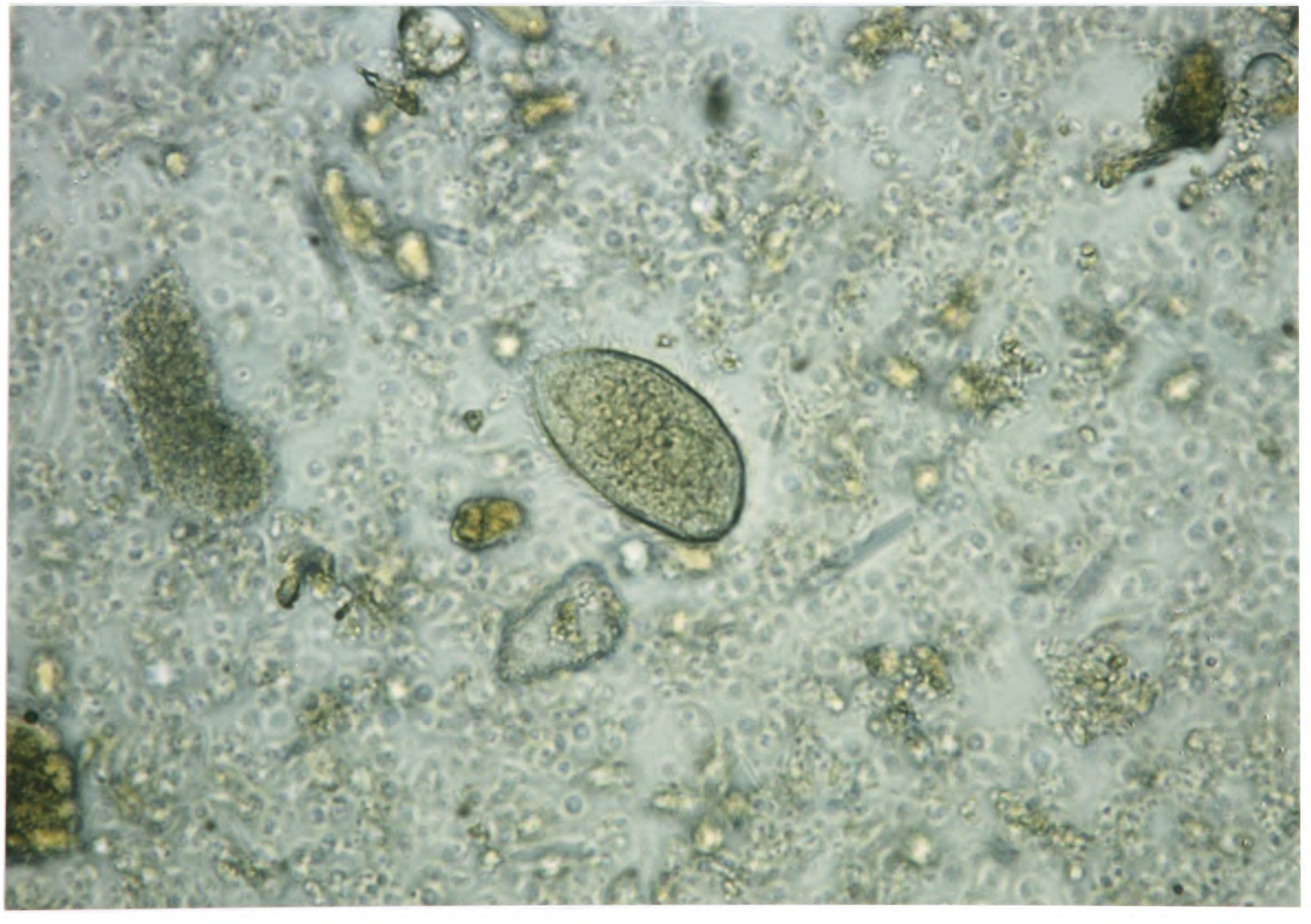


Plate 3.8. a) *Balantidium coli* trophozoite.

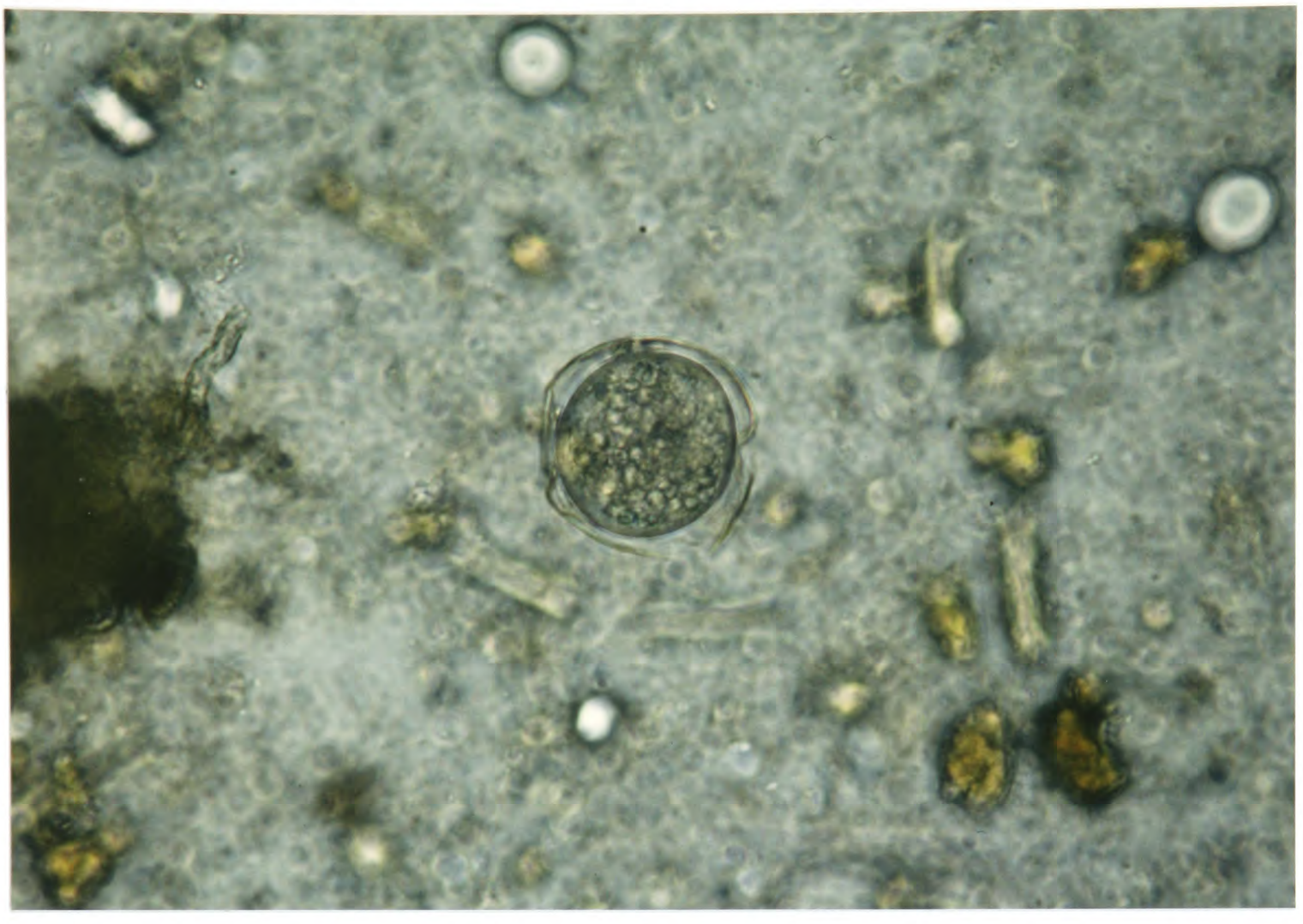


Plate 3.8. b) *Balantidium coli* cyst.

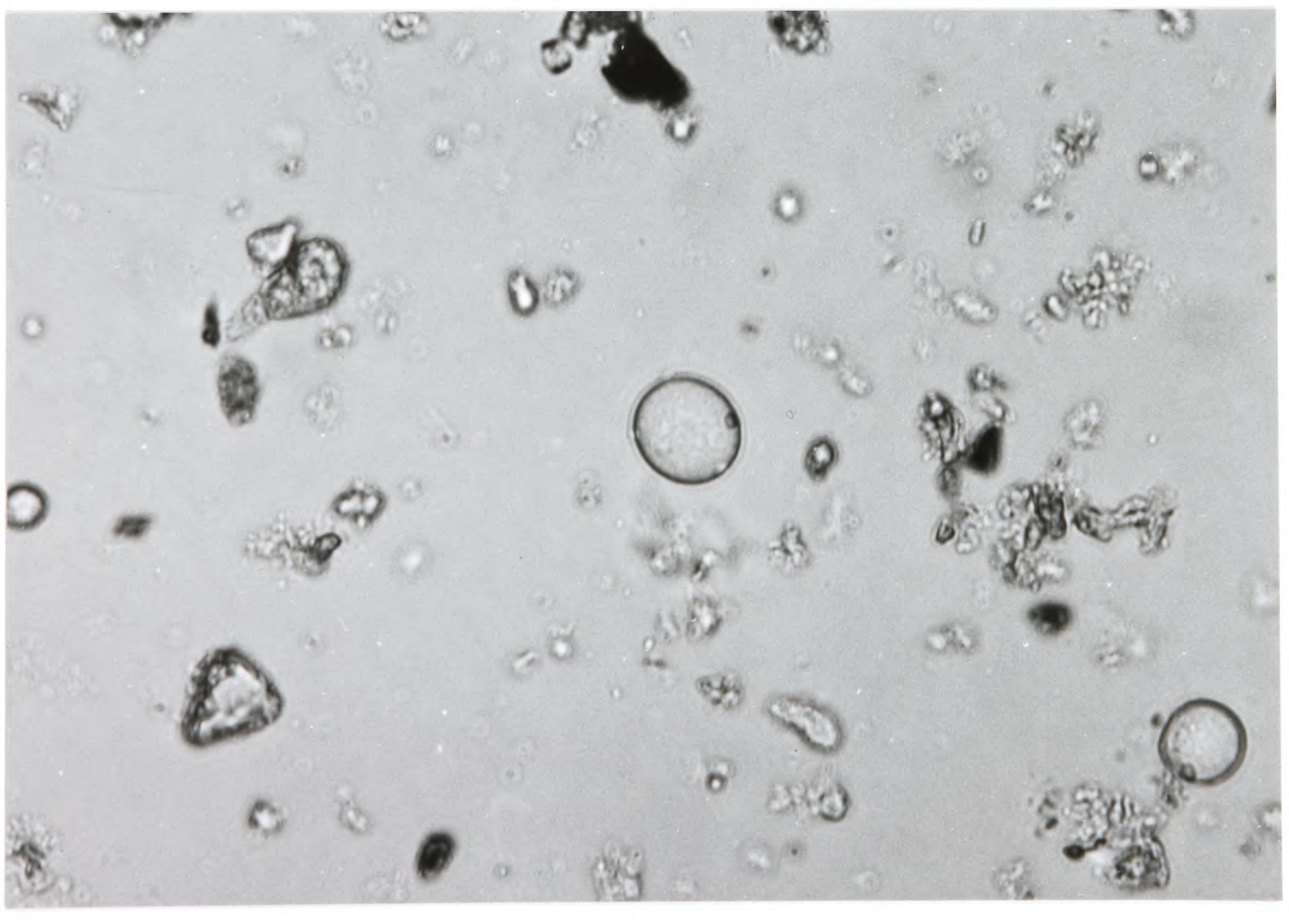


Plate 3.9. *Entamoeba* sp. cyst.

Distribution of intestinal parasites in a population of olive baboons (*Papio anubis*) in Gombe Stream National Park, Tanzania

ABSTRACT

A cross-sectional parasitological study of a population of wild olive baboons (*Papio anubis*) - consisting of five troops - was conducted in Gombe Stream National Park. The aim was to establish normative data on the prevalence and intensity of baboon parasites and to study factors influencing the distribution of infection. Individual baboons and their life histories were known and could be related to parasite infection. The presence of 7 helminth taxa and 2 protozoan taxa, some consisting of several species are reported. All baboons were parasitized and helminths showed aggregated distributions among hosts. In comparison with previous parasitological surveys of baboons, overall relatively high prevalences of infection were found in Gombe. Variation in levels of infection was examined with respect to age, sex, social status and female genital swelling. No significant sex or age effect on the prevalence of parasites could be detected, with the exception of age on *Strongyloides* sp. The severity of *Strongyloides* sp. and *Physaloptera* sp. infection also differed with age. Sex interacted significantly with severity of parasite infections for *Schistosoma mansoni* and *Streptopharagus* sp. only. Age-prevalence and age-severity profiles resembled those for the same parasite species in humans. Correlations between parasite infection and social rank were equivocal; some troops showed mainly non-significant correlation between high social rank and high parasite egg output while others did not. Parasite infection and the size of female genital swelling were

not correlated. Heterogeneity in parasite infection may be influenced by parasite-parasite associations. However, these do not appear to have an important impact on the present system, even though parasite species were not independently distributed among hosts; only four positive associations for prevalence and one negative association for severity were found between parasite species.

Plate 4.1. Olive baboons (*Papio anubis*) in Gombe Stream National Park.

4.1 INTRODUCTION

Heterogeneity of levels of infection within a host population is a characteristic feature of many parasitic infections (Anderson and Gordon 1982; Bundy 1988b; Anderson and May 1991). It may be caused by a number of factors, such as environmental and climatic factors or host characteristics, such as age, sex, behaviour, immunology, genetics and/or nutrition (see chapter 1). The relative impact of these characteristics can vary among host species, parasite taxa, different habitats and seasons. Although detailed experimental studies have been carried out to determine the effects of various host characteristics, little is known about host-parasite interactions in social groups of animals in the wild.

A well known wild population of social animals provides a good opportunity to study the impact of these factors on parasite distribution in the wild. A natural population of olive baboons (*Papio anubis*) (Plate 4.1.), consisting of five troops, in Gombe Stream National Park, Tanzania, East Africa has been monitored since 1967 (Packer 1977; Ransom 1981). Animals are individually known and information about sex, age, life-history, troop membership and family is available. A cross-sectional parasitological study was undertaken in autumn 1991 to determine parasite distribution in the baboon population.

This chapter has several aims. Firstly, the overall distribution of parasites in the population is described. Prevalence, intensity and severity of infection of the intestinal parasites across all troops are reported to provide baseline data on parasitic infection in baboons. Although baboons have been used in parasitological laboratory experiments, information on the distribution of parasite infection in the wild is limited (Dunn 1970). Some parasitological surveys have been carried out on baboons previously, reporting only prevalence of infection. The distribution of parasite species richness can be compared with a Poisson and summed binomial distribution to study whether parasite taxa are distributed independently among the hosts.

Secondly, the influence of host characteristics, such as age and sex, on infection is examined to see whether infections differ significantly between sexes or age-classes. For overall associations between age and infection, age-prevalence and age-severity profiles provide information on the dynamics of infection. As well as sex, female reproductive status, i.e. pregnancy, lactation or cycling, may have an impact on parasite infection .

It has been suggested that social behaviour can influence parasite infection (Hausfater and Watson 1976). Baboons as social animals offer a good opportunity to elucidate the relationships between social behaviour and parasite infection. In a previous

baboon field study, host social status was found to affect parasite egg emission. Higher ranking male baboons showed more eggs in their stools than lower ranking males (Hausfater and Watson 1976). Therefore, correlations between parasite infection and social rank within troops were explored. Furthermore, in line with the theory that parasites may influence sexual selection (Hamilton and Zuk 1982; Kirkpatrick 1987; Hamilton 1990; Møller 1990; Read 1990; Clayton 1992; Pagel 1994), possible correlations between size of female genital swelling and parasite infection were examined.

Finally, the study considered whether prevalence or severity of parasite infections of different taxa were correlated, to see whether parasite distribution may be shaped by interspecific competition or enhancement.

4.2 MATERIAL AND METHODS

Study Area

Gombe Stream National Park is situated on the Eastern shore of Lake Tanganyika, 16 km north of Kigoma at 4°40' S, 29° 38'E. The park covers an area of 52 km^2 and is bounded in the east by the escarpment of the eastern wall of the Rift Valley (Figures 4.1. and 4.2.). The lake shore is actually excluded from the National Park allowing fishermen and local people to use the beach. The park is dissected by 13 steep-sided valleys with streams running from east to west. In the park, the altitude rises quickly from 773 m above sea level at the lake shore to over 1500 m at the top of the escarpment. Because of this difference in altitude the vegetation is varied, ranging from evergreen forest, dry forest, to wooded grassland through to grassland near the ridges. The baboons live mainly in the evergreen forest area but their home ranges extend to the beach as well.

Apart from baboons, the park is also home to chimpanzees, red colobus monkeys, blue monkeys, red tail monkeys and vervets. A permanent research station with houses for the staff is situated within the borders of Gombe National Park, and fishermen camp along the beach for approximately three weeks every month. The beach is also used as a footpath for local people carrying food and other items from the surrounding areas (Ransom 1981; Bygott 1992).

Host species

The olive baboons (*Papio anubis*) in Gombe Stream National have been monitored since 1967 (Ransom 1981). At present, six field assistants observe the five troops every

day. Baboons can be distinguished individually. In autumn 1991, the baboon population consisted of 272 animals, forming five troops varying in size from 33 to 67 members (mean = 54) with a roughly equal sex ratio. The following names are used for the five troops throughout the study: A-troop, B-troop, C-troop, D-troop and L-troop. Three of the five troops A-, B- and D-troop are closely related (D.A. Collins, Gombe Stream Research Centre, personal communication). In 1969 A-troop split off from B-troop. The latter split again in 1978 to form B- and D-troop. Little is known about the history of C-troop and L-troop; they may have been related to these three before 1969 or be descendants from other neighbouring troops. However, all troops are related to some extent as male adult baboons migrate from the troop in which they are born and breed in other troops nearby (Packer 1979).

Females form the stable core of troops, staying in their natal troop while young males leave. Both adult males and females have a rank in the hierarchy of the baboon troop. The female rank is very stable over long periods of time whereas the male rank changes more often (Hausfater 1975; D.A. Collins, personal communication).

Baboons of a troop forage together, eating an omnivorous diet consisting of various foods: fruits, seeds, leaves, roots, flowers, stems, insects and small animals. In Gombe, more than half of their diet consists of fruit but, given the opportunity, they also scavenge on human rubbish and eat the fish which the fishermen dry on the beach (Ransom 1981; Bygott 1992).

Sampling

In autumn 1991, 256 stool samples were collected from 206 known individuals from all five troops. Time and date of collection were recorded. Information was available on troop membership (natal troop, troop of residence), sex, age, rank, reproductive status and size of female genital swelling. The genital swellings of all females in oestrous within each troop were compared by field assistants and individual females were ranked in order of size of their maximum swelling. The faecal samples were preserved in 25 ml centrifuge tubes in 10% formalin on site. They were analysed in Oxford by the formol-ether method for parasite egg and cyst content as described in chapter 2.

Parasite species

Parasite taxa identification is described in chapter 3.

Statistical Analysis

Prevalence is defined as the percentage of individuals of a host species infected with a particular parasite species. **Intensity** describes the number of larvae or eggs of one parasite species in an individual host. Mean intensity is calculated for all host individuals, infected and uninfected. Mean **severity** of infection describes the mean intensities of infection for infected hosts only. These three measures of parasitic infection are not independent and are related to each other as follows:

$$\text{Mean severity} = \frac{\text{Mean intensity x 100}}{\text{Prevalence}}$$

Richness describes the number of parasite taxa in the same host individual. The full list of baboon parasite taxa is presented in Table 3.1., excluding the unidentified larvae. **Total helminth load** refers to the sum of all helminth eggs and larvae counted in any one faecal sample. It is used as an indication of overall intensity of helminth infection.

The following analyses are based on the helminth egg counts only, which were log (x+1) transformed. Where repeated samples were available from the same individual, prevalence was assessed across all samples, richness was controlled for sampling effort by taking residuals from a linear regression line and intensity and severity were estimated by taking the arithmetic mean of all samples from the same host individual (see chapter 8). No intensity and severity data were collected for the protozoan parasites; they were not included in the analysis of richness because they consisted of too many indistinguishable species. Unidentified larvae were included in taxa richness, as they may consist of specific species, but not were considered in other analyses, unless stated, because of low prevalence.

A 'summed binomial distribution' (Lotz and Font 1991) was fitted to the parasite richness data. A summed binomial distribution generates the probability that any individual host will be infected with k different parasite species (k = 0 to S); where S is the total number of species in the parasite community. It is the result of the sum of the binomial distributions of each parasite species. The observed frequency distribution of parasite richness was tested against a Poisson and a summed binomial distribution using a χ^2- test. Each parasite species has an individual probability that it is present (p_i is the

prevalence of the *i*th parasite species in the host population) (Lotz and Font 1991). The expected variance of the summed binomial distribution if species are independently distributed among hosts is then:

$$s^2 = \sum_{i=1}^{S} p_i - \sum_{i=1}^{S} p_i^2 \quad \text{(where } \sum_{i=1}^{S} p_i \text{ equals the mean number of species per host)}$$

If parasite species are not independent in their likelihood of infecting a host, the observed variance includes covariances which account for the differences between variances:

$$\sigma^2 = \sum_{i=1}^{S} p_i - \sum_{i=1}^{S} p_i^2 + 2\sum_{ij} \text{cov}\,(p_i, p_j)$$

The ratio of the observed variance s^2 to the expected variance σ^2 (modified by N = number of hosts) is the test statistic for the independent distribution of parasite species on the basis of a χ^2 distribution $[(N - 1)s^2/\sigma^2]$ (Lotz and Font 1991). If a significantly larger variance is found than expected from the summed binomial distribution, it can be concluded that a covariance is present.

For a comparison of parasite prevalence between age-classes and sex, log-linear analysis was employed (using "SPSS"; SPSS Inc 1990) controlling for troop and interactions with troop. Differences between troops are examined in detail in chapter 5 but are controlled for in the analyses presented here. Age was treated as a categorical variable with two classes: young and old (young $\leq$ 8 years; old > 8 years). Above the age of 8 years baboons are fully grown adults (C. Packer, University of Minnesota, personal communication). The relationship of severity and intensity of infection with the age-classes and sex was analysed employing GLMs (General Linear Models) in the statistical package "Minitab"; (Minitab Inc. 1991), controlling for troop membership. More detailed age-classes were used for the age-prevalence and age-severity profiles and are reported with the graphs.

The analysis of any association between reproductive status and parasite infection involved only females which were either pregnant, lactating or cycling as only two individuals were amenorrhoeic. Analysis was carried out using GLMs with age as a covariate and controlling for troop.

Partial correlation was employed to test for an association between rank and parasite infection, controlling for age. Partial correlations were carried out separately for males and females. P-values were taken from a one-tailed test. The directional hypothesis

was that higher ranking individuals had higher parasite egg emission (Hausfater and Watson 1976). Rank data from each troop were adjusted as follows:

$$\text{adjusted rank} = \frac{\text{rank} - 1}{N - 1}$$

N = number of ranks in a particular troop at the time sampled.
Adjusted ranks therefore ranged from 0 (highest) to 1 (lowest). *Trichuris* sp. was chosen as a representative parasite taxon, because it was the most prevalent parasite among all parasite taxa found and it was also one of the parasites used in a previous study which found a correlation between social rank and parasite egg emission (Hausfater and Watson 1976). Also used were total helminth load and parasite richness.

An association between size of female genital swelling and parasite prevalence and intensity was studied using logistic regression (in "Statistix"; Analytical Software 1992) and partial correlation respectively, controlling for age (as a continuous variable), rank and troop.

To analyse the relationships between individual parasite taxa, prevalences were correlated using logistic regression controlling for troop, age (as a continuous variable), sex and sample effort. Severity was compared using partial correlation controlling for the same variables. Samples sizes <10 were excluded from the analysis.

For all tests, the level of significance was set at 0.05. If not otherwise stated, p-values from two-tailed tests are reported.

4.3. RESULTS

Prevalence and intensity of parasites within the population

Of the 206 animals sampled all were infected with at least one parasite taxon. The mode and median number of parasite taxa (excluding protozoans) per individual was 4. Parasite richness - including unidentified parasites - was distributed as shown in Figure 4.3.a. The frequency distribution was significantly different from a Poisson distribution ($\chi^2 = 28.93$, $p < 0.001$, df = 8, including categories for 0 and 8 taxa) and a summed binomial distribution ($\chi^2 = 39.52$, $p < 0.001$, df = 6, groups 1 and 2 and groups 8 and 9 were pooled to fulfil test requirements; Figure 4.3.b). The observed variance ($s^2 = 1.94$) was significantly larger than calculated from a summed binomial ($\sigma^2 = 1.28$; $\chi^2 = 321.02$, df = 205, $p < 0.0001$) with a covariance of 0.36, but not from a Poisson ($\sigma^2 = 3.59$; $\chi 2 = 114.62$, df = 205, $p < 0.50$).

Prevalence of all parasites in the population is shown in Figure 4.4. *Trichuris* sp. had the highest prevalence of all parasite taxa found. Overall, the lowest prevalence was found for the unidentified trematode, which was found mostly in C-troop. Median severities of parasite taxa are shown in Table 4.1. For each parasite taxon, intensity as measured by e.p.g. (eggs per gram of faeces) is overdispersed (Figure 4.5 a - g). Egg counts were not only overdispersed across the whole population, but also within individual troops.

The effect of age

Except for one parasite, *Strongyloides* sp., which had significantly higher prevalence in young animals (Z max = -2.041, $p < 0.05$), parasite prevalence did not differ significantly between young and old baboons. However, hookworm prevalence showed an interaction between age-classes and sex (Z-value = -2.68, $p < 0.01$). Juvenile males showed a higher hookworm prevalence than juvenile females whereas older female baboons had a higher prevalence than older males.

Two parasites showed a significant relationship between intensity and/or severity of infection and age-classes. Intensity and severity of *Strongyloides* sp. differed significantly between age-classes (intensity: $F_{1,194} = 13.6$, $p < 0.001$; severity: $F_{1,132} = 11.7$, $p = 0.001$). Severity of *Physaloptera* sp. infection was significantly higher in younger animals ($F_{1,125} = 5.25$, $p = 0.024$), but intensity of this parasite was not significantly affected by age ($F_{1,194} = 0.66$, $p = 0.42$).

A non-linear relationship between age and parasite infection can be seen in age-prevalence curves (Figure 4.6. a - j) and age-severity curves (Figure 4.7. a - h). For overall helminth load, intensity and severity were higher in the two first age-classes. *Trichuris* sp. prevalence and severity were very high from an early age onwards and remained more or less consistent, with the exception of a dip around the subadult age-classes (5 - 6 and 7 - 8 years). The age-prevalence curve for schistosomes showed that this parasite has higher prevalences in juvenile animals. The age-severity curve exhibited a similar pattern, except the decline in the curve started at a slightly older age. Age-prevalence curves for *Physaloptera* sp., *Streptopharagus* sp., the unidentified trematode and the larvae, showed that a comparatively high prevalence was reached at an early age. Hookworm showed a slow increase in prevalence, and severity peaked around 5 - 6 years of age. Severity of infection declined slightly for the unidentified trematode, *Schistosoma mansoni*, *Strongyloides* sp. and *Physaloptera* sp., while in *Streptopharagus* sp. and *Trichuris* sp. there was no change in severity across the age-classes. In contrast to the high prevalence of helminths in younger age-classes, prevalence of both protozoans was

lower in young animals, reaching a plateau in older animals for "amoebae" and exhibiting a convex curve for *Balantidium coli*.

The effects of sex and reproductive status

No significant differences were found between the sexes in the prevalence of parasites; however, differences could be detected in two parasite taxa in intensity and severity of the infections. Males showed higher intensity and severity of *Schistosoma mansoni* infection than females (intensity: $F_{1,193} = 6.46$, $p = 0.01$; severity: $F_{1,37} = 7.31$, $p = 0.01$). A significant difference in the severity of *Streptopharagus* sp. infection between males and females was also observed, with females having a higher parasite egg output ($F_{1,128} = 11.23$, $p = 0.001$). However, no significant difference was found between the sexes in the intensity of *Streptopharagus* sp. ($F_{1,194} = 1.25$, $p = 0.26$).

Reproductive status had no influence on prevalence of infection. Only intensity and severity of *Trichuris* sp. infection differed significantly among pregnant, cycling and lactating individuals (intensity: $F_{2,59} = 3.65$, $p = 0.032$; severity: $F_{2,44} = 3.74$, $p = 0.032$), with the lactating individuals showing the highest intensity, followed by cycling and then pregnant females (Figure 4.8.).

The impact of social rank on parasite egg emission

Social rank was not consistently correlated with total helminth load, taxa richness and *Trichuris* sp. intensity. For males and females within and across all troops, in some cases high social rank was correlated with higher parasite burden (negative correlation) but the opposite trend could also be observed, with high ranks associated with low parasite burdens (Table 4.2.). Only three correlations were significant: between A-troop female rank and total helminth load ($r = - 0.47$, $p = 0.02$, $df = 17$), A-troop female rank and taxa richness ($r = - 0.44$, $p = 0.03$, $df = 17$) and C-troop male rank and taxa richness ($r = - 0.73$, $p = 0.03$, $df = 5$). Across troops, male rank showed a non-significant negative correlation with *Trichuris* sp. and taxa richness, but not with total helminth load. The direction of the correlation varied for males from troop to troop. Females showed a trend across troops with 11/15 correlations being negative ($\chi^2 = 3.27$, one tailed $p < 0.05$, $df = 1$), although total helminth load and *Trichuris* sp. intensity are not fully independent.

Parasite infection and female swelling

No relationship was found between prevalence and intensity of any of the parasites and the size of the female genital swelling.

Parasite-Parasite Interactions

Tests for an interaction in prevalence between the different taxa showed four positive correlations: between *Streptopharagus* sp. and *Trichuris*. sp. ($p < 0.0001$), *Balantidium coli* and hookworm ($p = 0.005$) and *Physaloptera* sp. and the unidentified trematode ($p = 0.046$); one negative correlation was detected between the trematode and the "amoebae" ($p = 0.012$) (Table 4.3.). For severity of infection only one significant negative correlation, between hookworm and *Trichuris* sp., was found ($p = 0.03$; $n = 61$).

4.4 DISCUSSION

Overall distribution of parasites in the population

The results of the present study were compared with previous surveys of baboon parasites (Table 4.4.). The most common parasite genera, which were observed in the other baboon studies, were also found in Gombe with the following exceptions: *Bertiella* sp., *Enterobius* and *Oxyria* were not detected in Gombe. On the other hand, *Schistosoma mansoni* has only appeared at 4 other study sites: in East Africa (Kuntz and Myers 1966), in Zimbabwe (Goldsmid 1974), the Kruger National Park (McConnell *et al.* 1974) and near Mt. Assirik in Senegal (McGrew *et al.* 1988). Because of the difficulties in distinguishing between hookworm-like (or strongyle) eggs not much can be said about the composition of these genera in comparison to other studies. However, because of the comparatively large size of *Trichostrongylus* and *Ternidens* eggs it seems unlikely that these taxa are among those found in Gombe, whereas *Oesophagostomum* definitely infects the Gombe baboons. A relatively high infection with intestinal protozoans was observed in all other studies which diagnosed protozoans. *Balantidium coli* showed lower infection rates in Gombe than in other studies, but infection with other protozoans ("amoebae") was comparatively high in this study. To compare protozoan prevalences between studies is difficult as prevalence was reported in other studies according to species or genera and not just as one category. Gombe showed the highest *Strongyloides* prevalence and was among the three studies which had the highest *Trichuris* infections, the other studies being

conducted in Amboseli National Park (Meade 1983) and in the Drakensberg Mountains in South Africa (Appleton *et al.* 1986). Similarly, the infection rates of *Physaloptera* and *Streptopharagus* were comparatively higher in Gombe. Hookworm prevalence was lower than in more than half of the other studies, which may be due in part to the method used here for analysis which underestimates true hookworm infection rates (see discussion in chapter 2). Even when comparing the present study with studies where parasite infection was determined by autopsies, the prevalences of other helminths in Gombe were quite high. However, the data set in Table 4.5. can only give an approximate idea of baboon parasite prevalences; for more detailed comparisons, information on age and sex of the sampled animals, sampling effort and ecology of habitat would be necessary.

Furthermore, prevalence of infection was, in general, higher in the present study than in the two previous parasitological studies in Gombe Stream National Park, with the exception of hookworm. The differences in prevalences could be partly caused by different methods, smaller sample sizes in the two other studies in Gombe from fewer troops and/or sampling during a different season. McGrew (1988) sampled over two years from October 1993 through to January 1974 and in January 1975, whereas Murray (1991) sampled only in the dry season, during July and August 1989. A comparison of only dry season data from the present study (see chapter 6) with the results of the Murray survey, showed that prevalences of all parasites with the exception of *Streptopharagus*, hookworm and the unidentified trematode were still higher. Hence, an increase in infection could have taken place since these earlier studies.

The present study is the first to report frequency distributions of baboon parasites, therefore they cannot be compared to other studies. Intensities of infection have not been recorded previously in baboons with the exception of Pettifer (1984), who noted the ranges of the number of helminths found in autopsies, and Fenwick (1969), who reported only the number of *Schistosoma mansoni* observed in dissected baboons.

The marked aggregation of the parasites' e.p.g. found in this study, with few baboons harbouring a high number of parasites and the majority of baboons harbouring only a few or none, is characteristic of the majority of macroparasite-host associations (Anderson and May 1991). An aggregated distribution can reflect differences in exposure, innate susceptibility and acquired immunity between individual hosts.

The effect of age

Age-prevalence and age intensity/severity curves show age related changes in infection. These may result from a variety of factors, including changing behaviour,

exposure time and/or increased resistance to infection with age. Here, a significant difference between age-classes was only detected for *Strongyloides*. For the other parasite taxa, no significant changes between age-classes could be found. However, the observed age prevalence- and age severity curves do show patterns of interest for many of the recorded parasites. For those parasites which have been examined both in humans and baboons, the age curves are in most cases similar. This suggests that similar behavioural and/or physiological mechanisms may be involved in determining the characteristics of parasite infection. Unfortunately, not enough is known to attribute age-related changes in the baboons to either immunological or behavioural factors. The majority of helminths, with the exception of hookworm, showed extremely rapid infection (in prevalence and intensity) within the first year of life, sometimes even approaching peak levels during the first twelve months. This situation indicates high transmission rates and, in some cases, may be suggestive of transmammary infection. For several helminths, levels of infection fall in older individuals implying reduced exposure, lower innate susceptibility, acquired immunity or a combination of these factors. However, for some other taxa the fall in prevalence is less marked. These results are in agreement with similar patterns observed for human parasitic diseases (Wilkins 1987; Bundy 1990; Anderson and May 1991). Associations between age and baboon parasite infections have been documented in two previous studies (Meade 1983; Pettifer 1984), although, their sample sizes were not sufficiently large to draw conclusions on each parasite species. However, both also observed high infections rates in very young baboons.

In the following section age-prevalence and age-severity curves for the individual parasite taxa will be briefly discussed and, if possible, related to infections found in other species. *Strongyloides* exhibits a greater infection in younger baboons, following a similar pattern shown in humans infected with *Strongyloides fuelleborni,* who exhibit higher infection and intensity rates in younger individuals (Pawlowski 1989). These results were also supported by a previous baboon study (Meade 1983). However, the percentage of *Strongyloides* prevalence in Gombe, in contrast to human infection in areas with high prevalence of the parasite, was higher in adult baboons than in humans of a comparative age (Ashford and Barnish 1989; Ashford *et al.* 1992). A decrease of prevalence in older individuals could be due to acquired immunity, but behaviour may also be important. Juvenile male baboons show a greater preference for the underground parts of certain plants (C. Packer, personal communication; J. Oliver, unpublished manuscript). Because infective stages are transmitted by soil, the higher contact of juvenile baboons with soil may contribute to higher infection rates; however, this does not explain the infection patterns in very young animals. It has been suggested that infection with *Strongyloides* at a very young age can occur transmammarily; apparently even egg-negative mothers can transmit the parasite in this way (Loke 1983). This route of

transmission, in combination with the relatively high rate of egg-positive adults found here, may partly explain the high incidence of strongyloidiasis among the very young baboons.

Trichuris shows a similar prevalence pattern as in humans (Anderson and May 1991; Bundy and Medley 1992) with the exception of a slight decrease in the infection of subadult baboons. The curve remains the same when looked at by troop. *Trichuris* prevalence in humans and baboons rises very quickly at a young age to a plateau and then slightly declines in adult life. However, severity of infection in baboons does not show the decline typically seen in older humans, even though the same method was used, i.e. egg counts (Bundy *et al.* 1987a). Perhaps the rate of establishment of infection in humans is reduced due to mechanisms that do not operate in baboons.

The age-prevalence curve for hookworm rose initially, dropped for 7 - 8 year old individuals and then rose again to a maximum in old animals. The age-severity curve showed a similar pattern. Typically, age-prevalence curves for human hookworm infection rise with age and reach a stable plateau in early adulthood (around 15 - 20 years of age), however, hookworm age-prevalence and age-intensity curves for humans can be quite variable (Bundy 1990) and patterns similar to the baboon hookworm infections have been seen in some cases, e.g. Bradley *et al.* (1992, 1993). Hookworm infection can occur as mixed infections which may complicate patterns and comparisons. Baboons support different species other than are found in humans, e.g. *Oesophagostomum* sp. A previous baboon study showed an increase in prevalence and mean worm burden with age (Pettifer 1984).

Age-prevalence curves for *Physaloptera* and *Streptopharagus* both rose to a plateau. However, the age-severity curves showed a decrease in older age-classes (although only very slight for *Streptopharagus*). Both parasites are acquired through ingestion of arthropod intermediate hosts. No detailed observations of differences in exposure with age were made. However, adult males spent more time processing foods of animal or human origin than other age-sex classes (J.Oliver, unpublished manuscript), although adult females eat more insects (D.A. Collins, personal observation), which could result in higher parasite burden in adult animals. A lighter severity of infection in older hosts may then reflect acquired immunity, reducing the number of worms which mature, or reducing egg emission, but not conferring complete immunity to reinfection (Quinnell and Keymer 1990). In the study of baboons in the Transvaal *Physaloptera caucasica* and *Streptopharagus pigmentatus* worm burden appeared to decrease with age, whereas no apparent pattern was obvious from the age-prevalence curve, apart from fewer parasites in the first or the second youngest age-classes (Pettifer 1984).

Younger baboons have higher schistosome infections than older ones, which mirrors results of human studies (Wilkins 1987; Chandiwana *et al.* 1991). The behaviour

of baboons may explain these differences to some extent. Watercontact observations made in Gombe in 1975 showed that juvenile baboons had much higher water-contact rates in Lake Tanganyika than older baboons; no baboon older than 11 years was observed in the water (D.A. Collins, unpublished data ; Figure 4.9. a and b). Lake Tanganyika is free of snails at Gombe (D.A. Collins, personal communication) and therefore is not a likely point of *Schistosoma* infection. Although these observations at the lake shore may indicate a general pattern of behaviour, no observations of water-contact have been made in areas which harbour snails. However, young baboons sometimes swim in stagnant pools and stream beds in the dry season (D.A. Collins, personal communication). Baboons of all ages have sometimes been observed to forage for crabs in the streams, so it is clear that even older baboons have water-contact from time to time (C. Packer, personal communication). Swimming behaviour may expose younger animals more to infection, but the higher prevalence of infection in young animals might also reflect a higher innate susceptibility or acquired resistance in the older animals.

For most helminths, a high prevalence in young age-classes was observed. The age-prevalence curves of the "amoebae" and *Balantidium coli* show a different trend, with a slower acquisition of infection, and then a subsequent decline for *Balantidium coli* in adulthood, but with increased infection in the two oldest age-classes for the "amoebae". Not much is known about age-prevalence curves of these taxa in humans yet; in the United States, children under five were found to have a lower prevalence of *Entamoeba histolytica* than older children, while the greatest prevalence occurred in the age group 26 to 30 (Schmidt and Roberts 1989). Infection with *Entamoeba histolytica* may not elicit an immunological response from the host sufficient to confer immunity to reinfection (Kreier 1978; Cox 1993). If the situation is similar for other amoebae, the higher infection in older age-classes reflects changes in the force of infection with age.

The effects of sex and reproductive status

Although sex differences in infection patterns are well documented for other host species (Bundy 1988a; Brabin and Brabin 1992), sex hardly influences parasite infection in the Gombe baboons. Other baboon studies have found similar patterns, with sex differences in prevalence observed only for *Physaloptera caucasica*, with males having a higher burden (Pettifer 1984). This is surprising as Gombe adult male and female baboons, in addition to a different hormonal profile, show different feeding patterns. Females in B-troop were observed to eat more seeds than males who spend more time processing food of animal origin or human waste (J. Oliver, unpublished manuscript), although females may eat more insects than males (D.A. Collins, personal observation).

The present results suggest that the differences in feeding behaviour may not be strong enough in most cases to affect prevalence of infection or transmission of parasites. On the other hand, if the sex differences in the severity of *Streptopharagus* infection are real (because they were not reflected in the intensity of infection, they have to be treated cautiously), they could reflect feeding behaviour as the parasite is transmitted by insects. However, similar findings would be expected for *Physaloptera* sp. because of a similar route of transmission. Differences in intensity and severity of infection with *Schistosoma mansoni* may be explained by the water-contact patterns already described (Figure 4.9. a and b). Males may have higher rates of contact with water and hence greater opportunities to become infected. However, other factors such as an interaction between hormones and the immune system could be equally important (Grossman 1989; Schuurs and Verheul 1990).

The majority of parasite taxa did not appear to be affected by female reproductive status. Reproductive status seemed only to be important for the intensity and severity of *Trichuris* infection. Hausfater and Watson (1976) found the highest egg emissions in cycling females, followed by anoestrous (lactating or otherwise non-cycling) and then pregnant females. In this study, pregnant females were also found to have the lowest egg output, in contrast to expectations that they should be more susceptible to infection due to immunosuppression during pregnancy (Brabin and Brabin 1992). On the other hand lactating exerts great nutritional stress on the female baboons. Lactating females at Gombe showed the highest *Trichuris* egg output. This may be the result of the hormonal status of the lactating female, a weakened immune system - the time spent lactating appears to be the most stressful time in the adult lives of the female Gombe baboons (D.A. Collins, personal communication) - or possibly an adaptive behaviour of the parasite (Lloyd 1983). By increasing egg output during lactation, *Trichuris* could increase the probability of infecting the infant. Differences in behaviour and environment between the two study sites may explain differences between the studies; additionally, Hausfater and Watson (1976) used untransformed parasite egg data, which may be biased by a few individuals with extreme infections.

The impact of social rank on parasite egg emission

Social status has been associated with parasite infection in several host species (Freeland 1981; Rau 1983; Rau 1984; Halvorsen 1986). During periods of stable dominance relationships, high-ranking male baboons at Amboseli had a significantly higher overall parasite egg emission than low ranking males (Hausfater and Watson 1976). Few significant correlations were found between the social rank of the Gombe baboons

and parasite ova emission of overall helminth load (one significant result), *Trichuris* infection (no significant results) and parasite richness (two significant results). Generally, the relationship between infection and social rank was weak and the results equivocal. Males across troops showed non-significant correlations in the predicted direction, with higher ranking males having a higher *Trichuris* sp. egg output and taxa richness, but not total helminth load. Correlations of male rank and parasite infection differed in direction for the individual troops with species. However, more samples may be needed over a longer period of time to observe any correlations, rather than samples from one point in time. Higher ranking females showed, with two exceptions, higher egg outputs and higher taxa richness than low ranking females, except for D-troop where the correlation indicated a reversed trend.

Overall, the relationship between social rank and parasite load may hold in some troops but not in others. Associations between rank and infection may vary according to environment and other factors even within a fairly similar habitat. The different habitats of the olive baboons in Gombe and the yellow baboons in Amboseli, as well as different food sources and amounts of food available may account for different interactions with parasites.

Association of female genital swelling with parasite infection

Current theories suggest that animals can advertise their quality, including such aspects as resistance to pathogens by external features (Hamilton and Zuk 1982; Kirkpatrick 1987; Møller 1990; Read 1990; Loye and Zuk 1991; Clayton 1992). When it is beneficial for females to solicit copulations, they may need to advertise their state of health for instance by displaying their genital swelling (Hamilton 1990; Pagel 1994). In the Gombe baboons, no association between the size of female genital swelling and parasite infection could be found. If female swellings advertise quality, it could be that the colour rather than the size of the swelling is important. Colour was not measured in this study and further research may reveal whether females use oestrous swellings to advertise their quality with regard to parasite infection.

Interaction of parasite communities in the host

Parasite taxa might interact with each other within a host by competing for resources or by enhancing establishment (Esch *et al.* 1990; Sousa 1994). Significant differences of the observed parasite richness distribution from the Poisson and the

summed binomial distribution showed that data were not independently distributed. These two distributions have been suggested as the null hypothesis for the underlying distribution of parasite richness assuming random parasite distribution. Although the Poisson distribution is commonly used (Dobson 1990) recent work by Lotz and Font (1991) pointed out that a summed binomial distribution is a more accurate model of parasite richness. The present distribution shows more hosts with fewer and more parasite taxa than expected and fewer hosts with an intermediate number of parasite taxa (Figure 4.3. b) (33% of the observed variance resulted from covariance). This implies that, overall, parasites are not independently distributed in infections of individual hosts.

When correlating the prevalence of parasite taxa, a significant interaction was found between four pairs of parasite taxa (Table 4.4.). In the analysis each taxon was correlated with all other taxa, so that altogether 45 tests were executed. One would expect to obtain 2 to 3 significant results merely by chance. Therefore, these results should be treated with caution. On the other hand, all correlations were controlled for confounding variables and *Trichuris* and *Streptopharagus* still showed a positive interaction at a highly significant level, suggesting that there may indeed be an association. It cannot be assumed, however, that the results are due to interspecific interactions of parasites after invading the host. For instance, positive correlation may occur because parasite taxa have their infective stages in the same environmental area, share a same route of transmission or in some other way be ecologically correlated. Conversely, for parasites with different transmission routes this may lead to negative associations. However, positive correlations found in this study did not seem to be obviously related to route of transmission. If certain individuals are predisposed to parasite infections in general rather than to one particular species, positive correlations may result as well (Haswell-Elkins *et al.* 1987; Forrester *et al.* 1990; Keymer and Pagel 1990). So far, multi-species predisposition appears to be weak, and may also be confounded in cross-sectional studies by chance encounters of the infective stages of each species of parasites involved (Keymer and Pagel 1990). The occurrence and the type of interactions between parasite species is far from clear and more studies are needed.

4.5 CONCLUSION

Parasites constitute a substantial component of baboon ecology; all baboons were found to be parasitized. The distribution of parasites in a population of baboons was overdispersed, as is the case for most macroparasite infections. Age-prevalence and age-severity curves were often similar to curves observed for humans. Rapid infection in young individuals implies high transmission rates. The decreased infection in older

individuals found for some helminths may reflect, among other possibilities, acquired immunity. Behaviour, for instance changes in water-contact with age as in the case of schistosomes, may contribute to lower infection rates in older individuals as well. Sex differences were not marked and where they existed may reflect behavioural differences. Female reproductive states appears to have little influence on infection. In contrast to earlier work, social rank was not found to be significantly related to the distribution of parasitic infection. In this study, size of female sexual display did not reflect parasite infection, contrary to some theoretical arguments. Parasite-parasite interactions appear to have only a weak effect on the overall patterns of infection. Even though parasite distribution was aggregated in the host population, none of the host factors considered here had a strong impact on the overall distribution. However, trends were observed. Heterogeneity of infection may be shaped more by exposure within and among troops, genetic differences across troops or possibly nutritional differences among hosts than by the host factors analysed in this chapter.

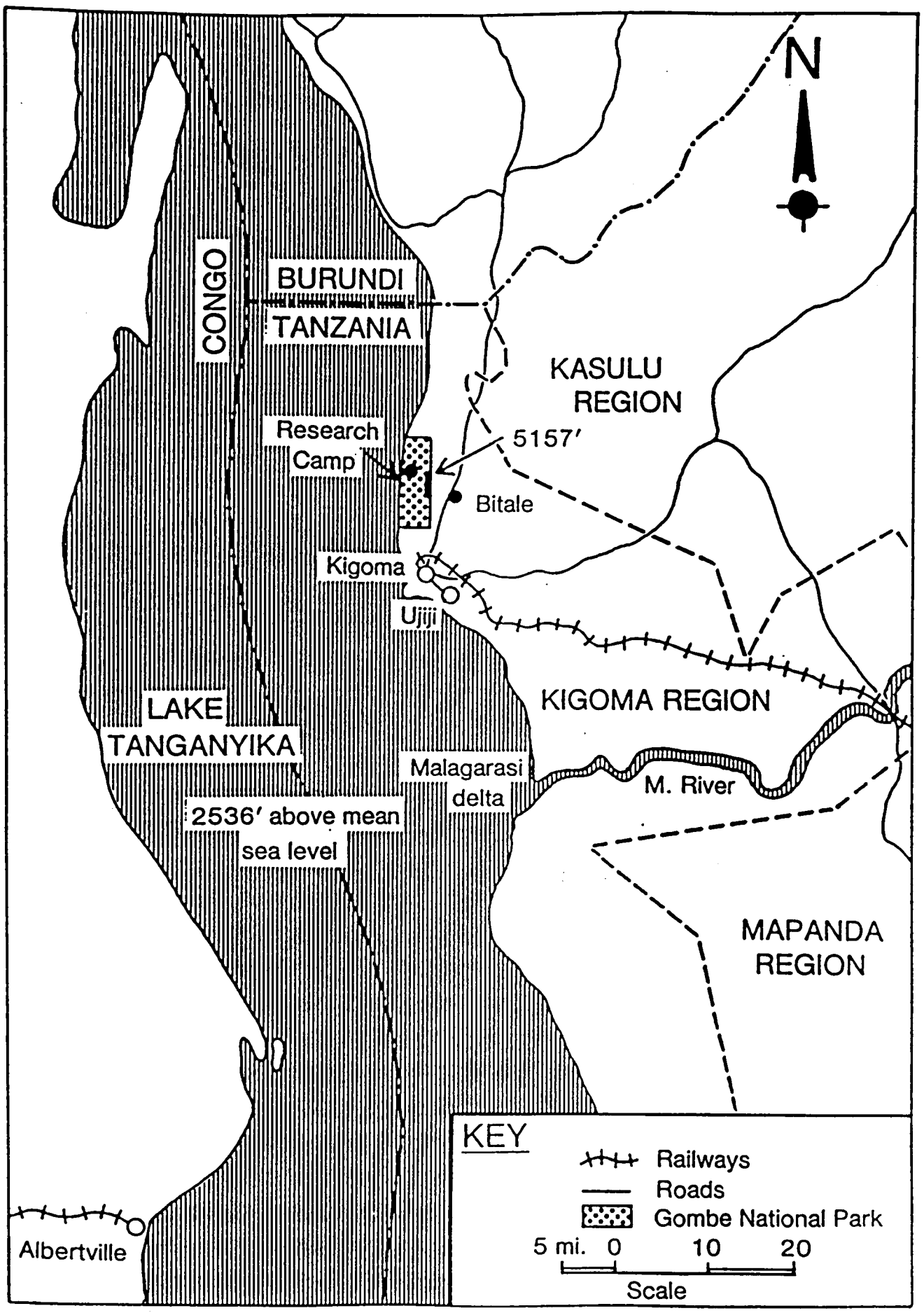


Figure 4.1. The Kigoma region of Tanzania, East-Africa, location of the Gombe Stream National Park from Ransom (1981)

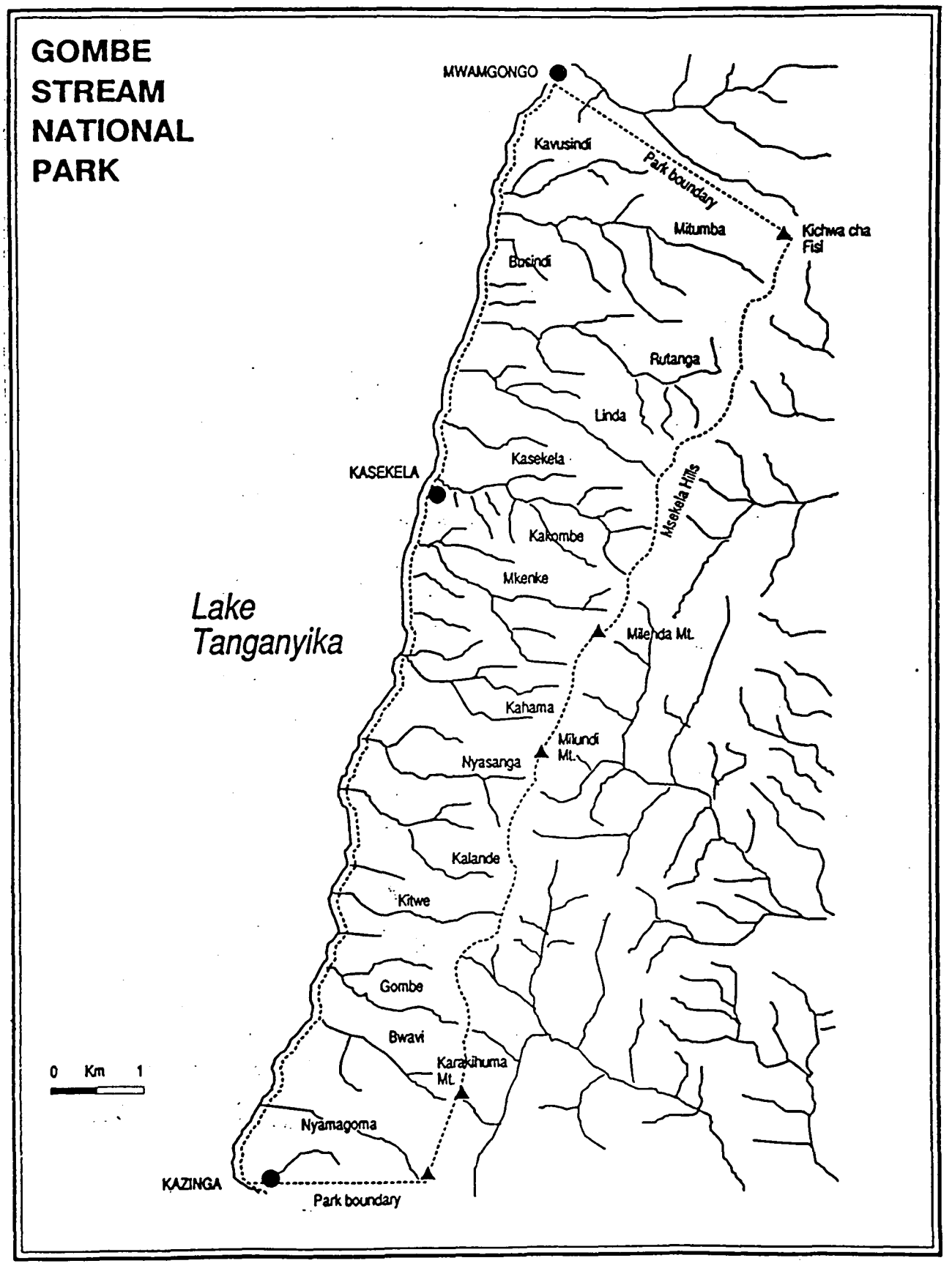
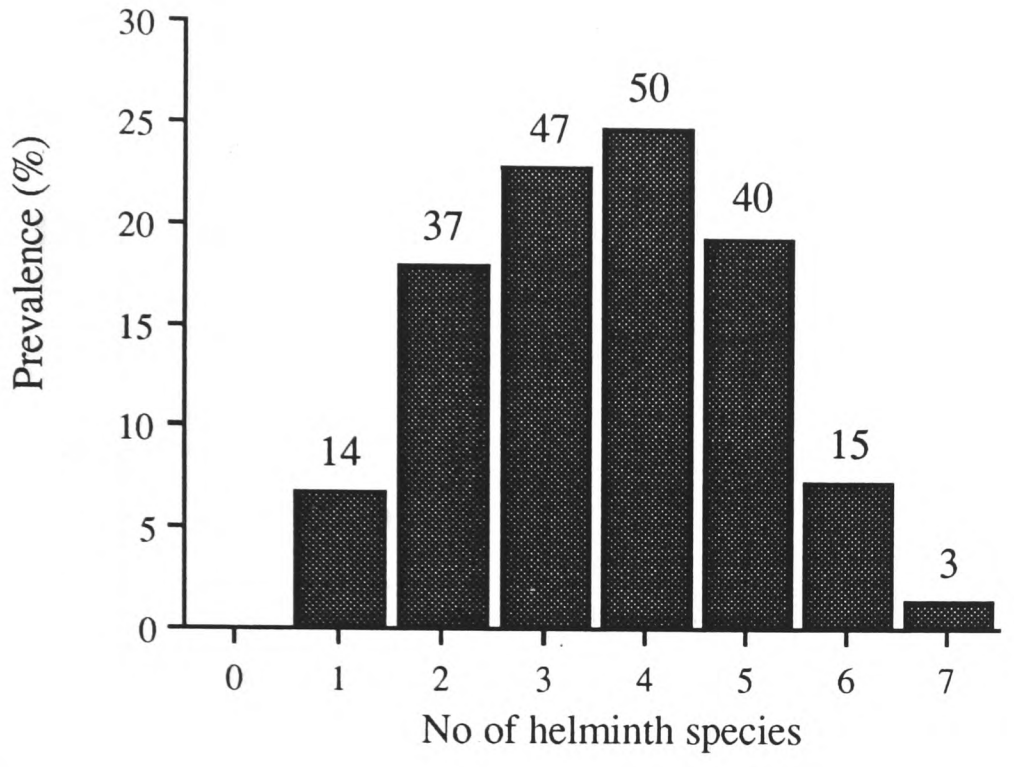


Figure 4.2. The area of Gombe Stream National Park, from Bygott (1992).

a)



b)

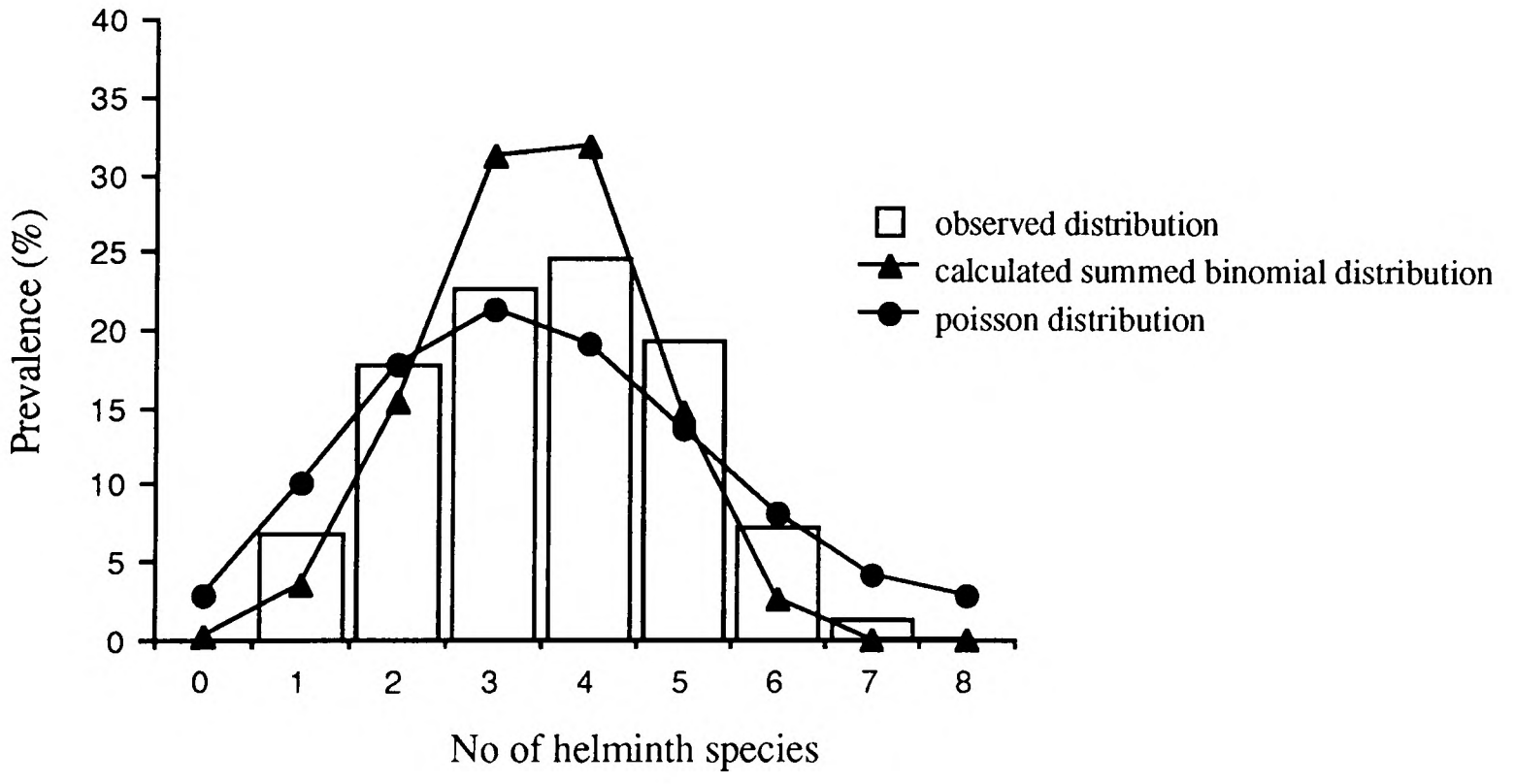


Figure 4.3. Helminth taxa richness
a) Observed richness distribution; number of individuals are reported on top of each bar.
b) Observed distribution of helminth richness compared to a fitted Poisson and the calculated summed binomial distribution.

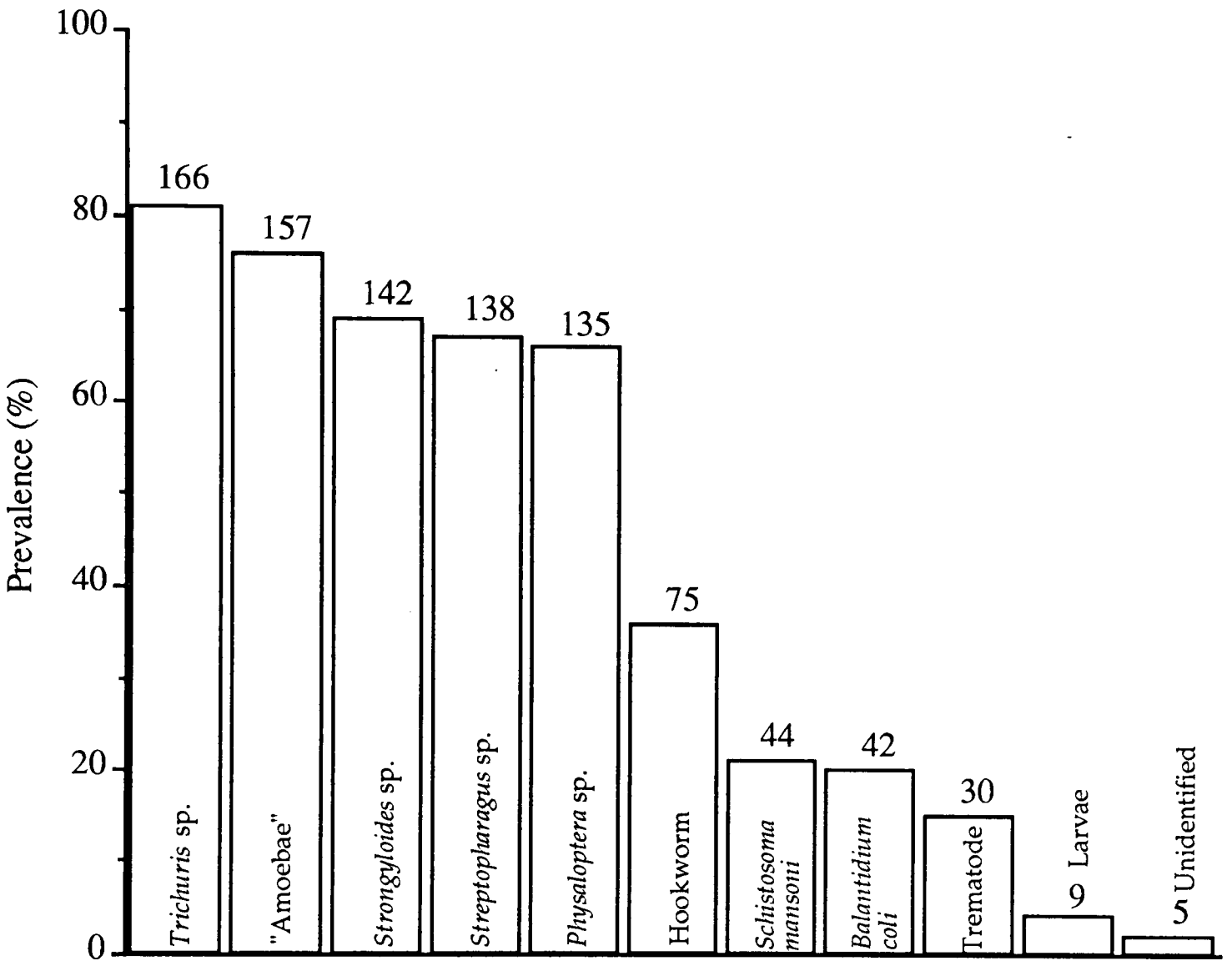


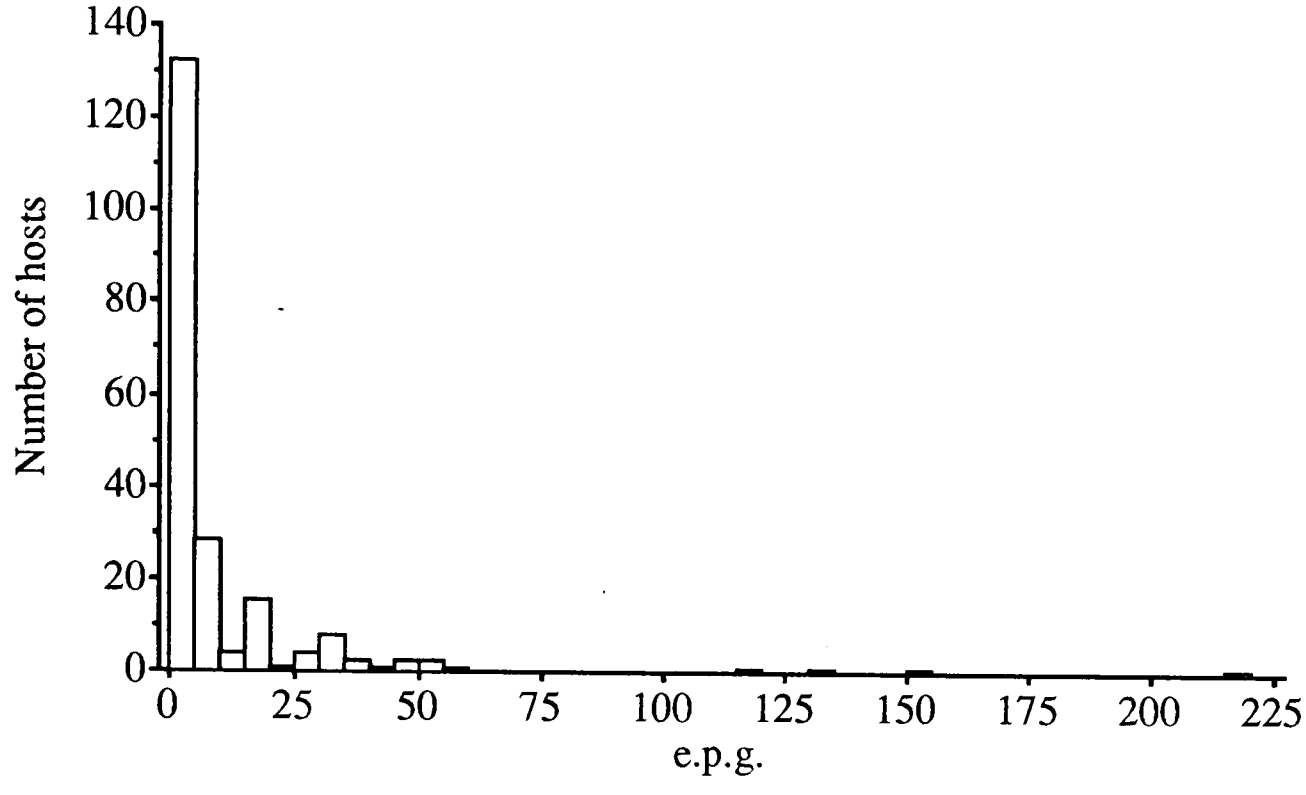
Figure 4.4. Prevalences of baboon parasites.
The number on top of each bar indicates the number of infected individuals (n = 206).

Table 4.1. Median severity and maximum helminth egg output.

Parasite Taxa	**median severity (e.p.g.)**	**maximum e.p.g.**
Unidentified Trematode	382.8	3316.7
Streptopharagus sp.	283.1	4450
Strongyloides sp.	141.6	11133.3
Physaloptera sp.	125	1383.3
Trichuris sp.	66.7	991.7
Hookworm	16.7	216.7
Schistosoma mansoni	16.7	283.3
Unidentified larvae	8.3	33.3

e.p.g. = number of eggs per gram of faeces.

a) Hookworm



b) *Strongyloides* sp.

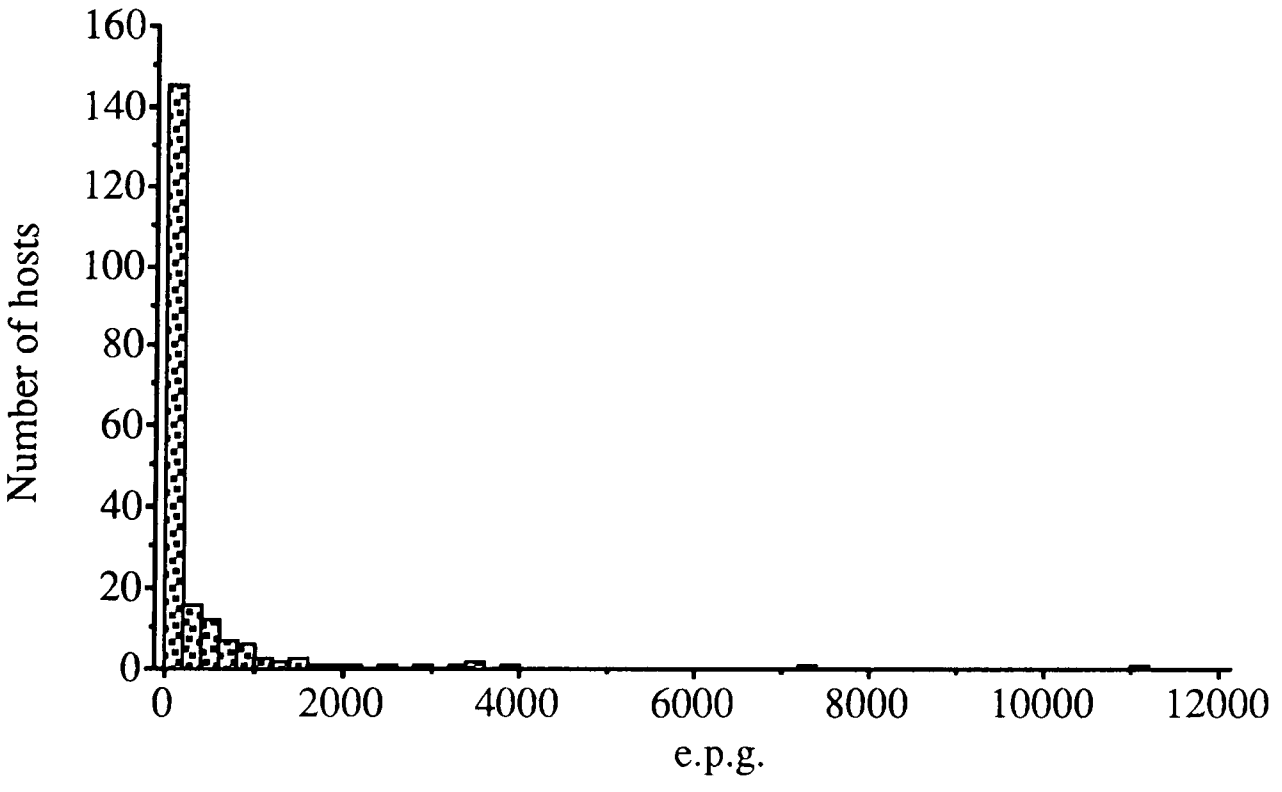
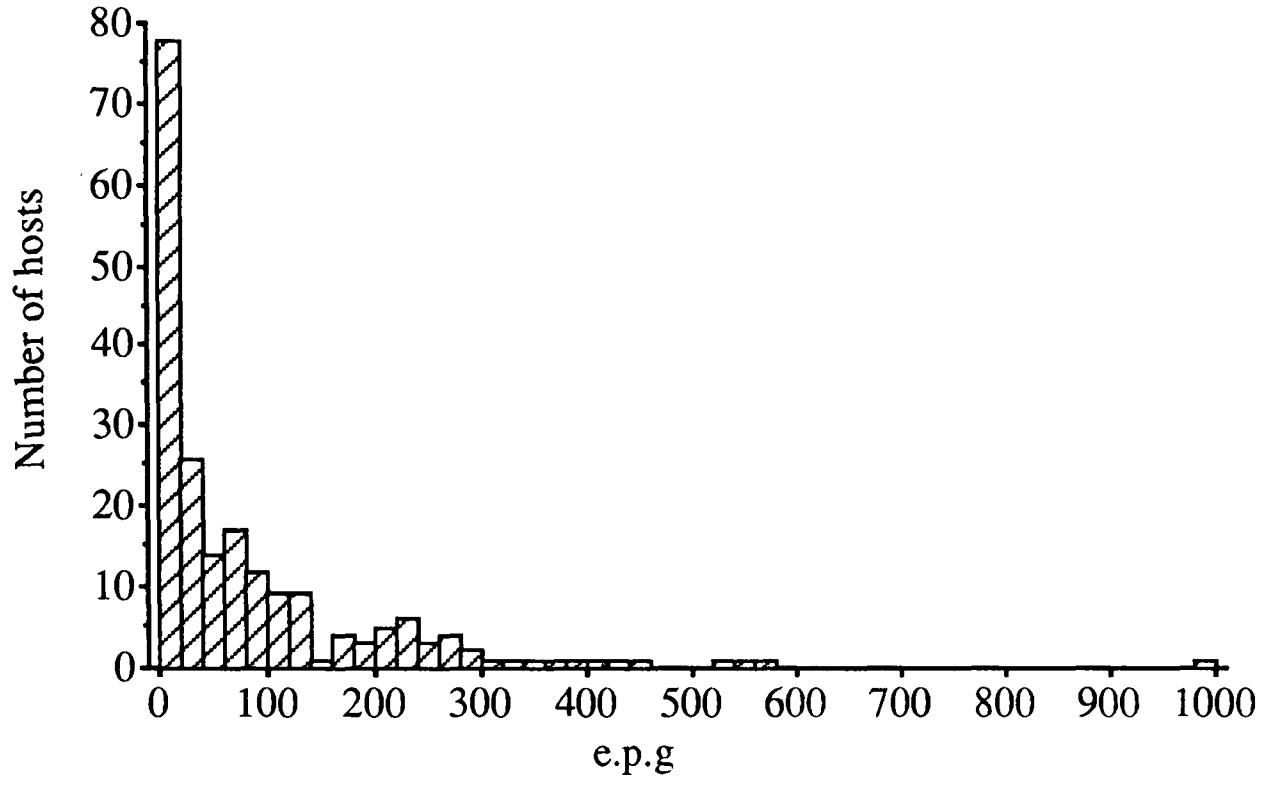


Figure 4.5. Distribution of intensity of infection as measured in e.p.g. Zero counts are included in the first bar.
a) Bar interval 5 e.p.g.
b) Bar interval 250 e.p.g.

c) *Trichuris* sp.



d) *Physaloptera* sp.

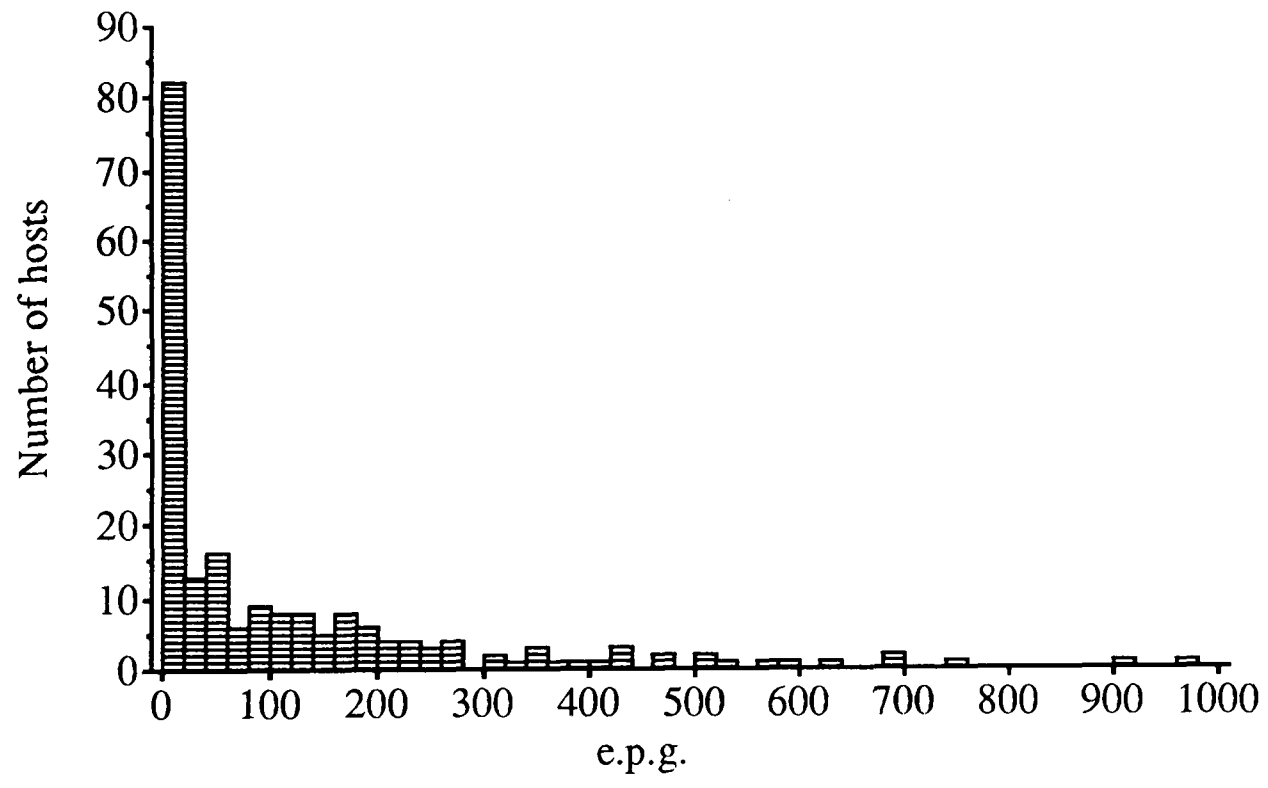
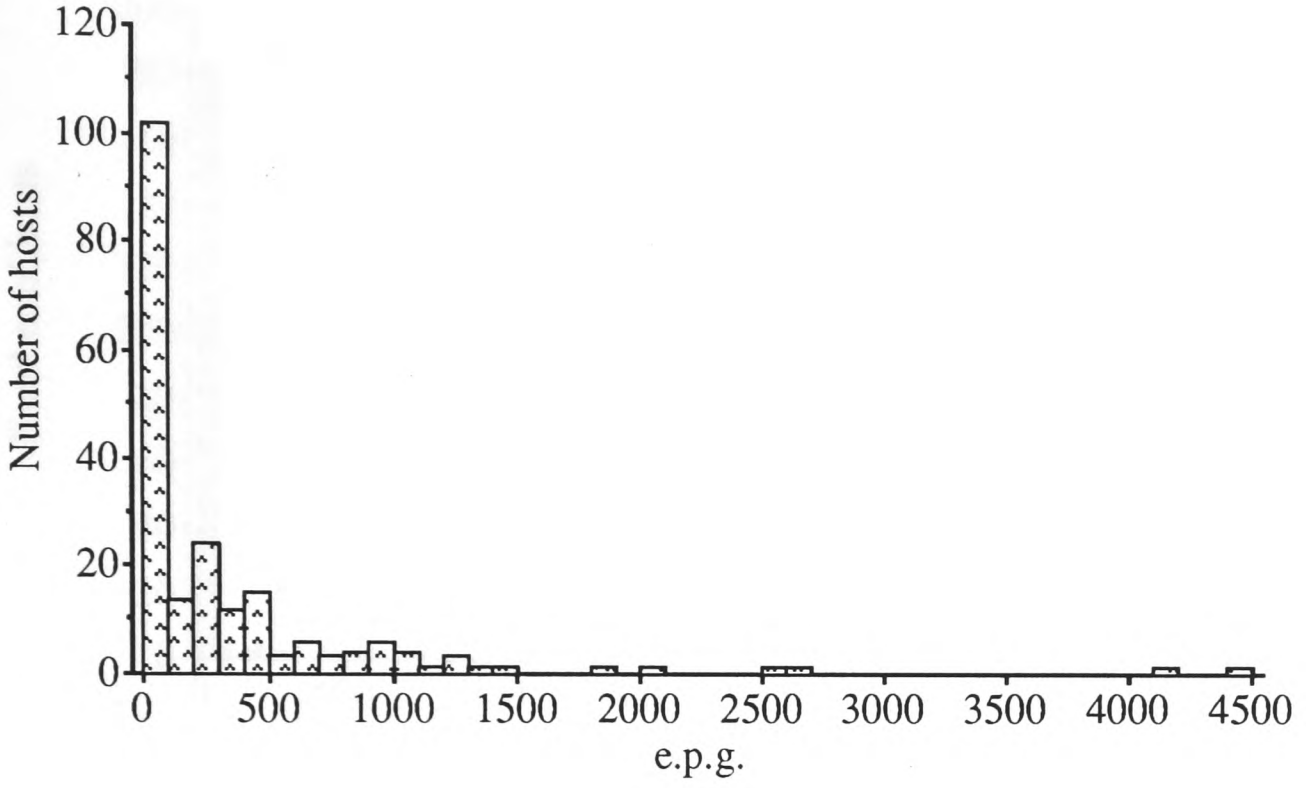


Figure 4.5. Distribution of intensity of infection as measured in e.p.g. Zero counts are included in the first bar.
c) Bar interval 20 e.p.g.
d) Bar interval 20 e.p.g.

e) *Streptopharagus* sp.



f) *Schistosoma mansoni*

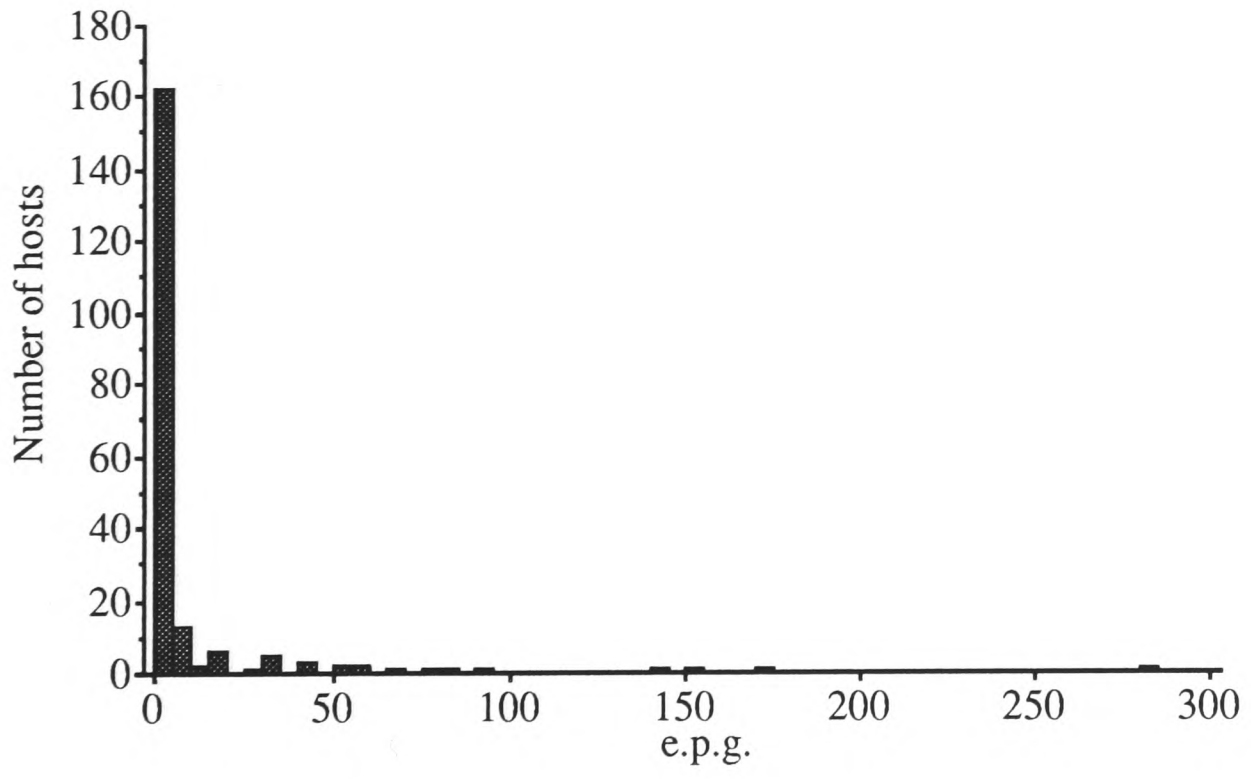


Figure 4.5. Distribution of intensity of infection as measured in e.p.g. Zero counts are included in the first bar.
e) Bar interval 100 e.p.g.
f) Bar interval 5 e.p.g.

g) unidentified trematode

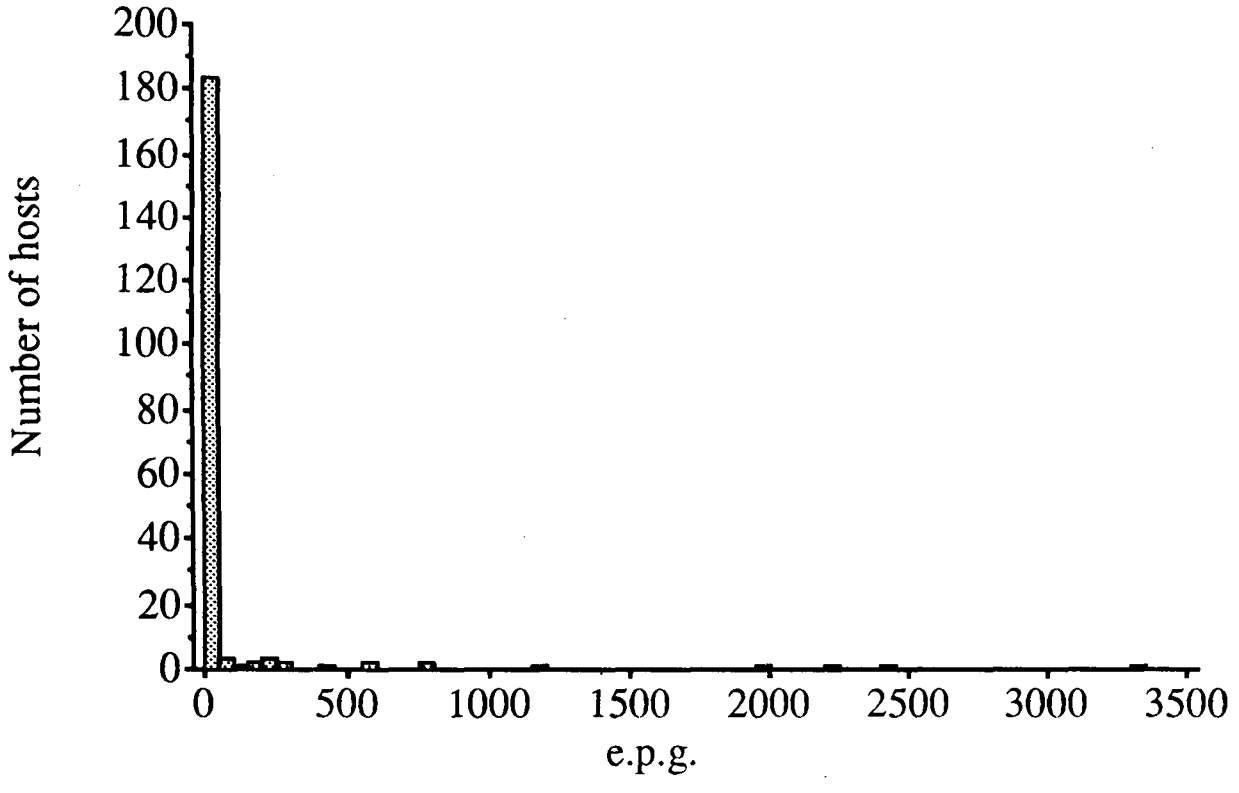
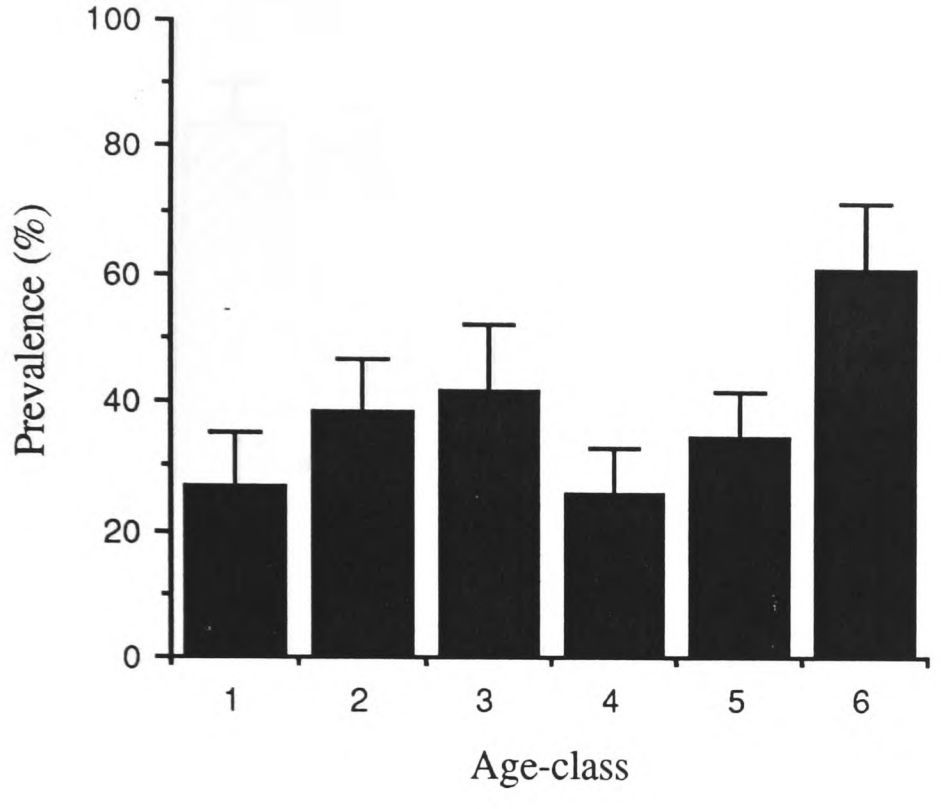


Figure 4.5. Distribution of intensity of infection as measured in e.p.g. Zero counts are included in the first bar.
g) Bar interval 50 e.p.g.

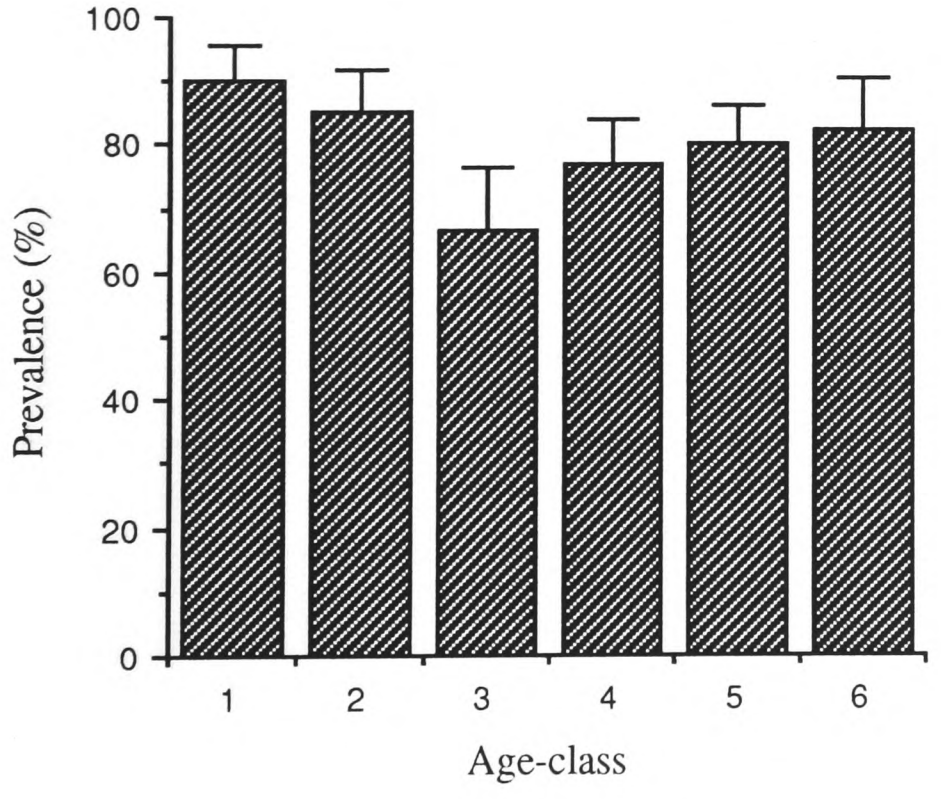
Figure 4.6. Age-prevalence profiles.

Age-prevalence profiles were calculated across the whole population. X-axes indicate the age-classes and y-axes the percentage infected. Age-classes are classified as follows: 1 = up to 2 year (infants) n = 30 , 2 = 3 - 4 years (juveniles) n = 34; 3 = 5 - 6 years (subadults) n = 24; 4 = 7 - 8 years (young adults) n = 36; 5 = 9-15 years (adults) n = 45; 6 > 15 (old individuals) n = 22. Bars represent standard errors of the mean.

a) Hookworm

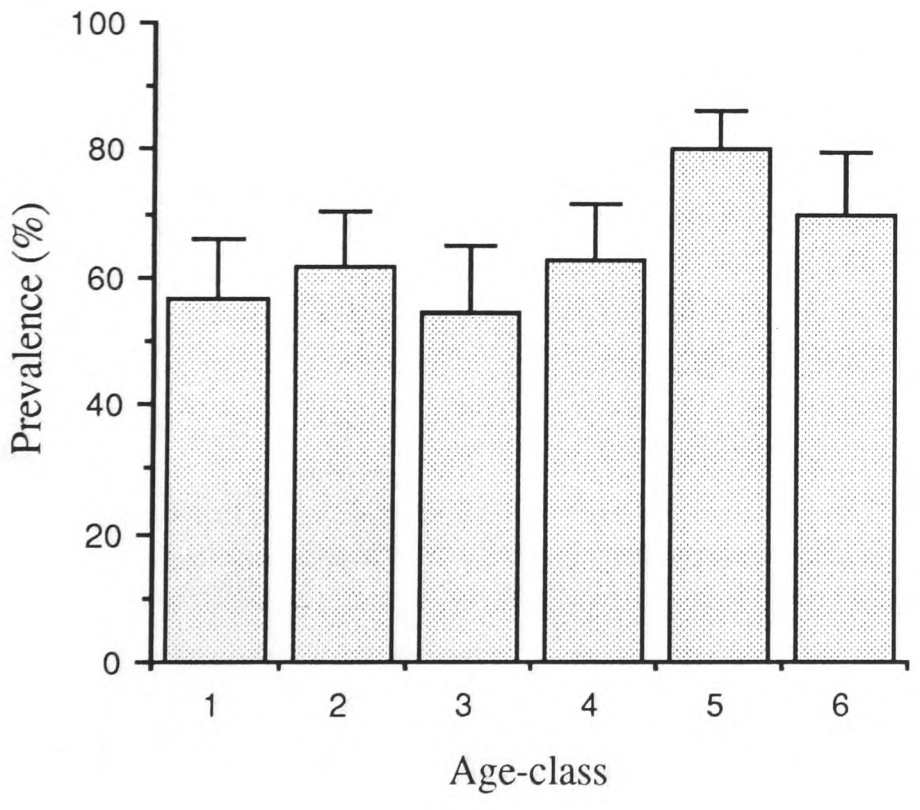


b) *Trichuris* sp.

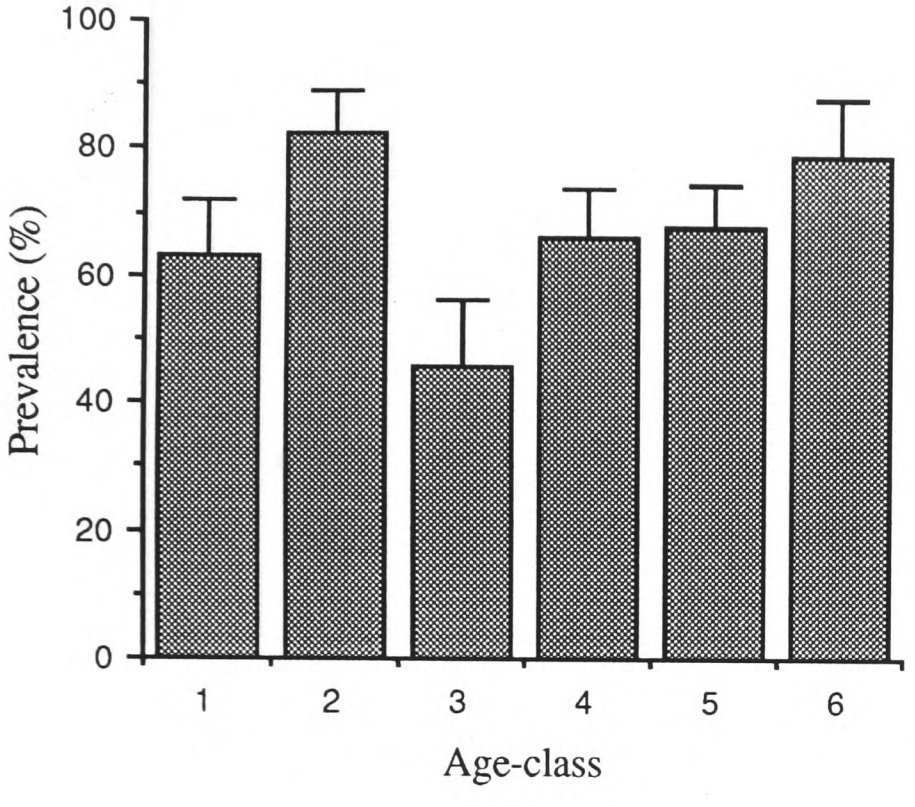


c) *Strongyloides* sp.

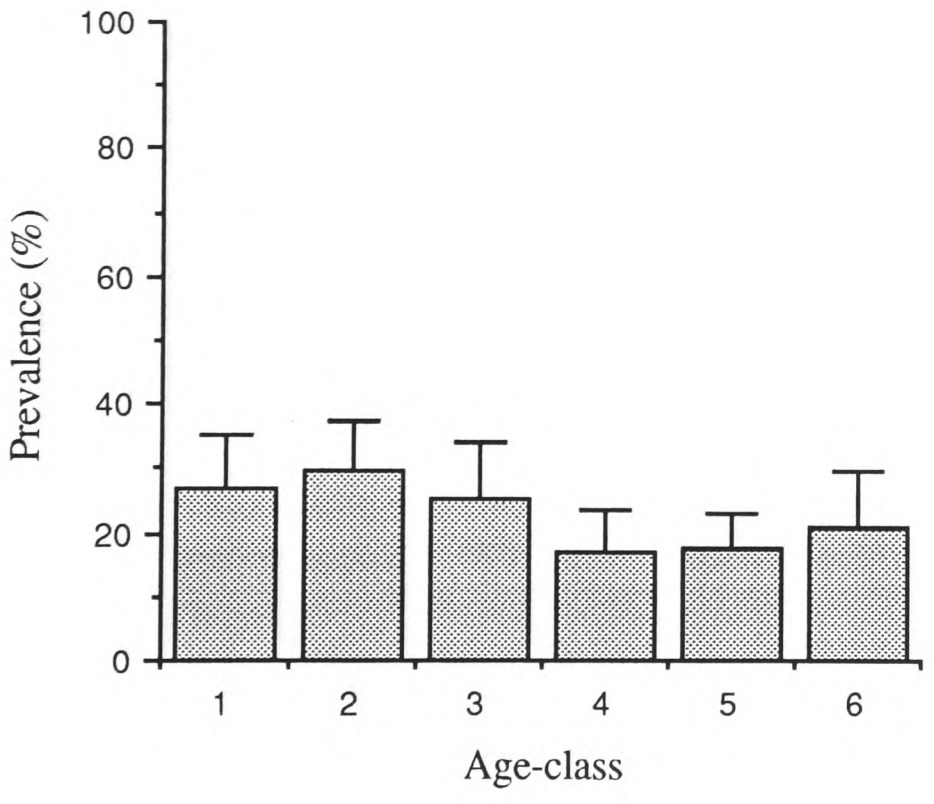
d) *Physaloptera* sp.



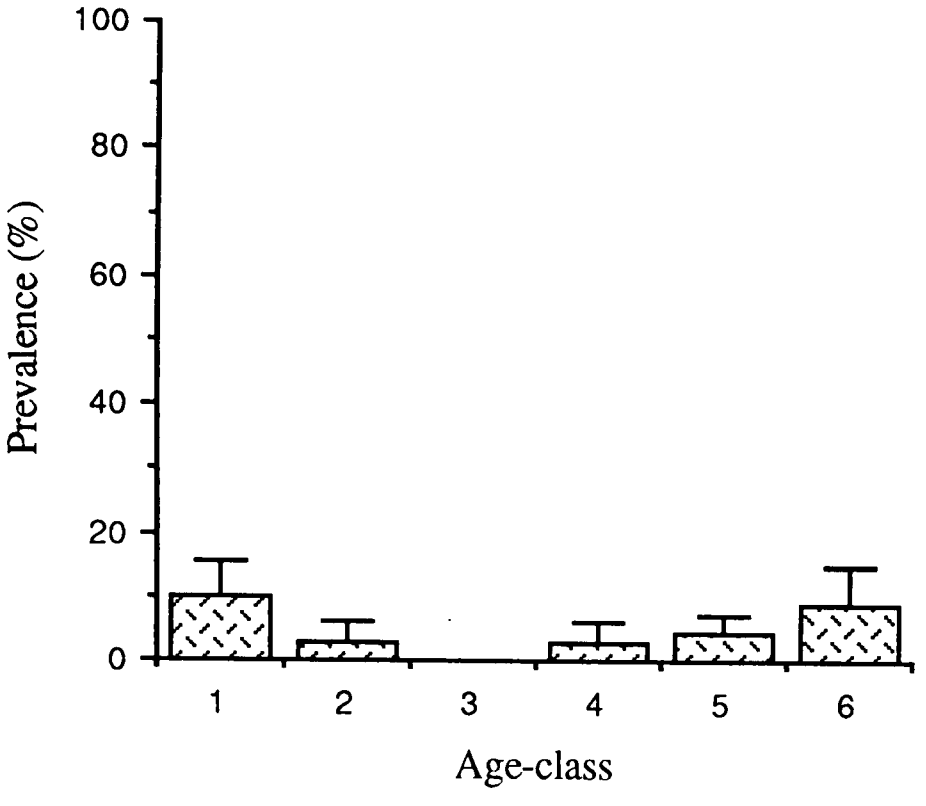
e) *Streptopharagus* sp.



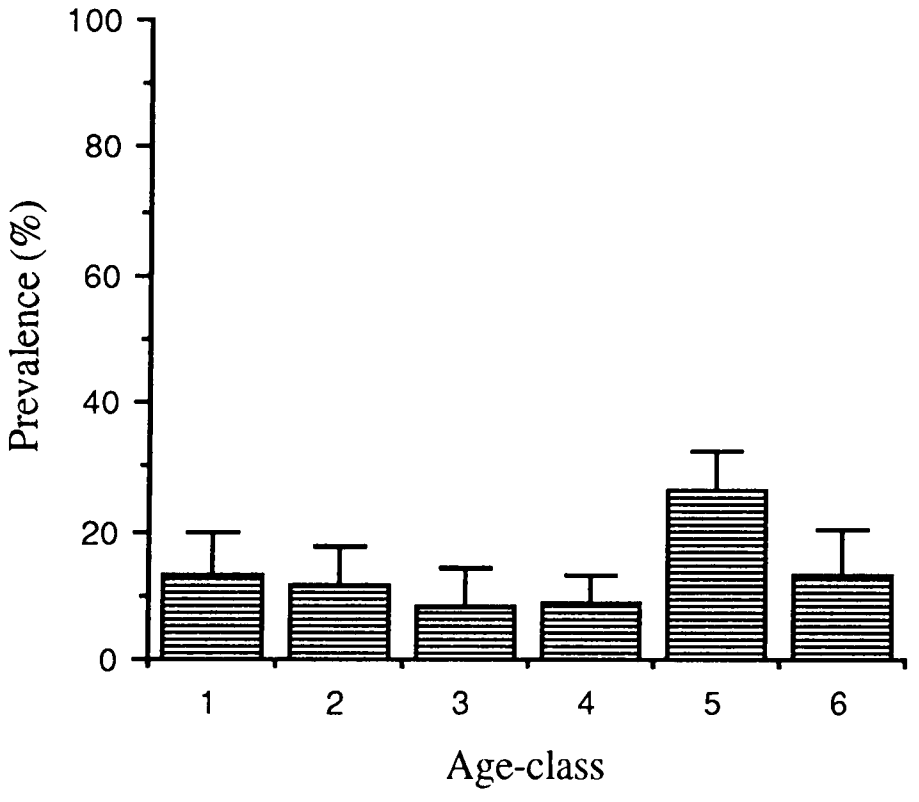
f) *Schistosoma mansoni*



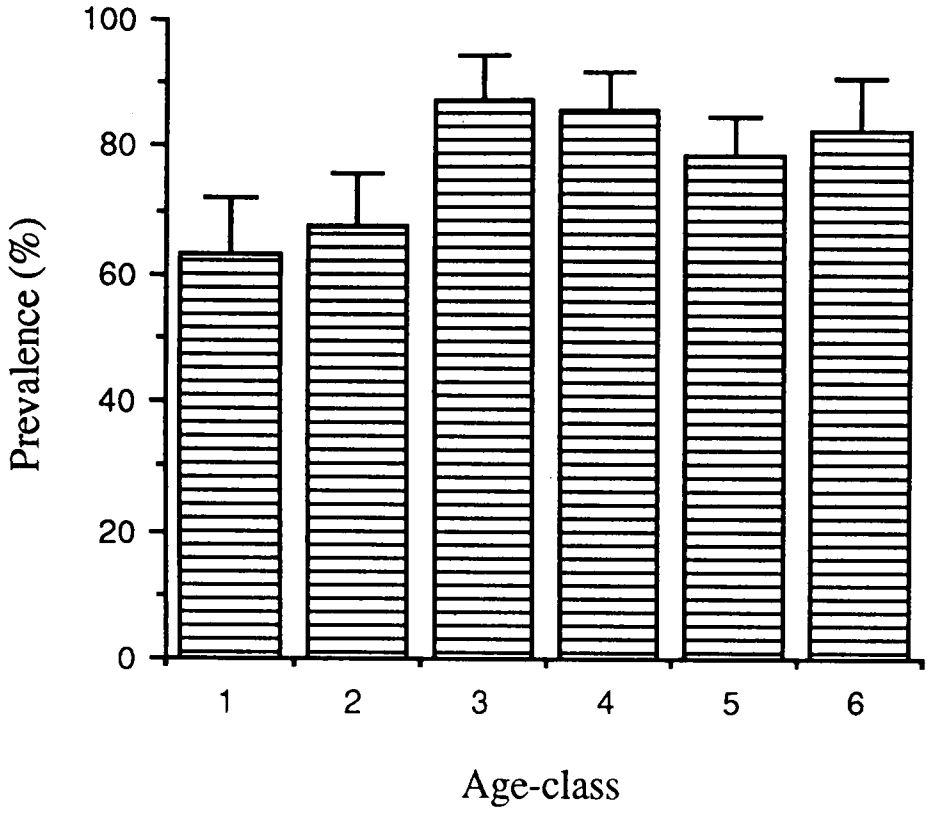
g) Unidentified larvae



h) Unidentified trematode



i) "Amoebae"



j) *Balantidium coli*

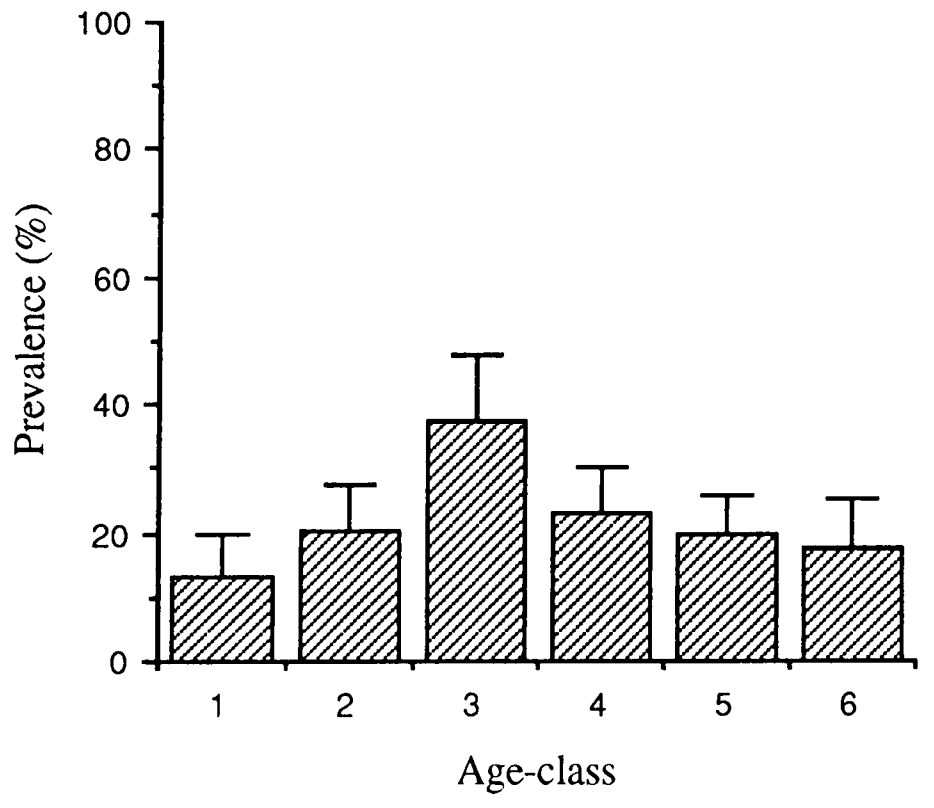
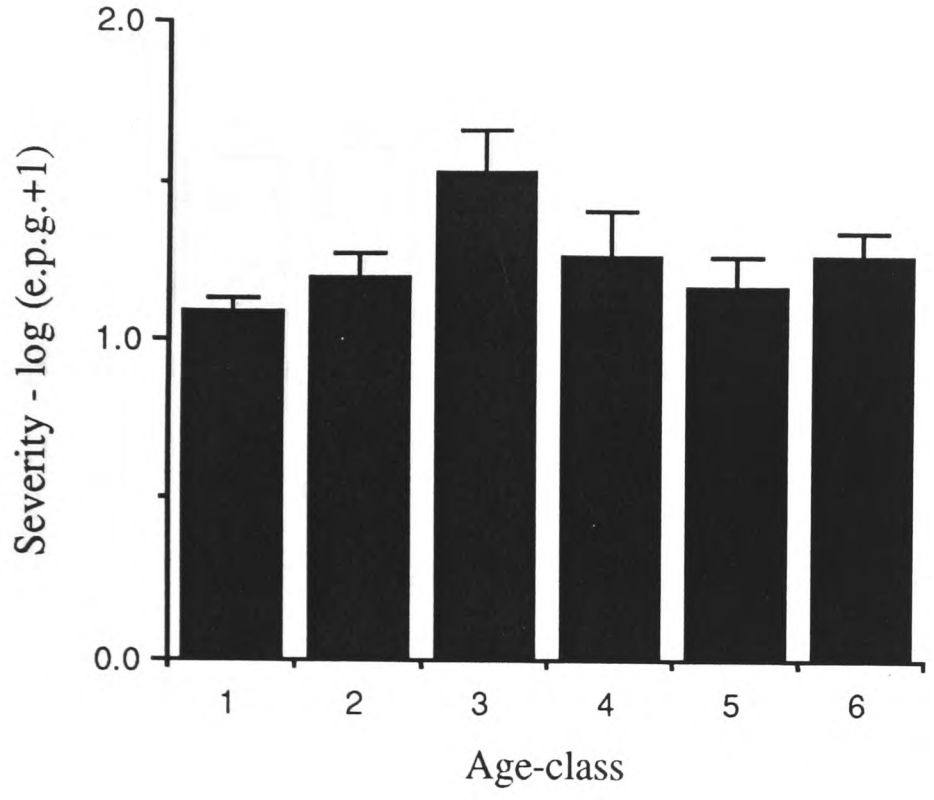


Figure 4.7. Age-severity profiles.
Age-severity profiles were calculated across the whole population. X-axes indicates the age-classes and y-axes the severity of infection in log(x+1) transformed e.p.g. Age-classes are classified as follows: 1 = up to 2 year, 2 = 3 - 4 years, 3 = 5 - 6 years, 4 = 7 - 8 years, 5 = 9 -15 years, 6 = > 15 years. Bars represent standard errors of the mean.

a) Hookworm



b) *Trichuris* sp.

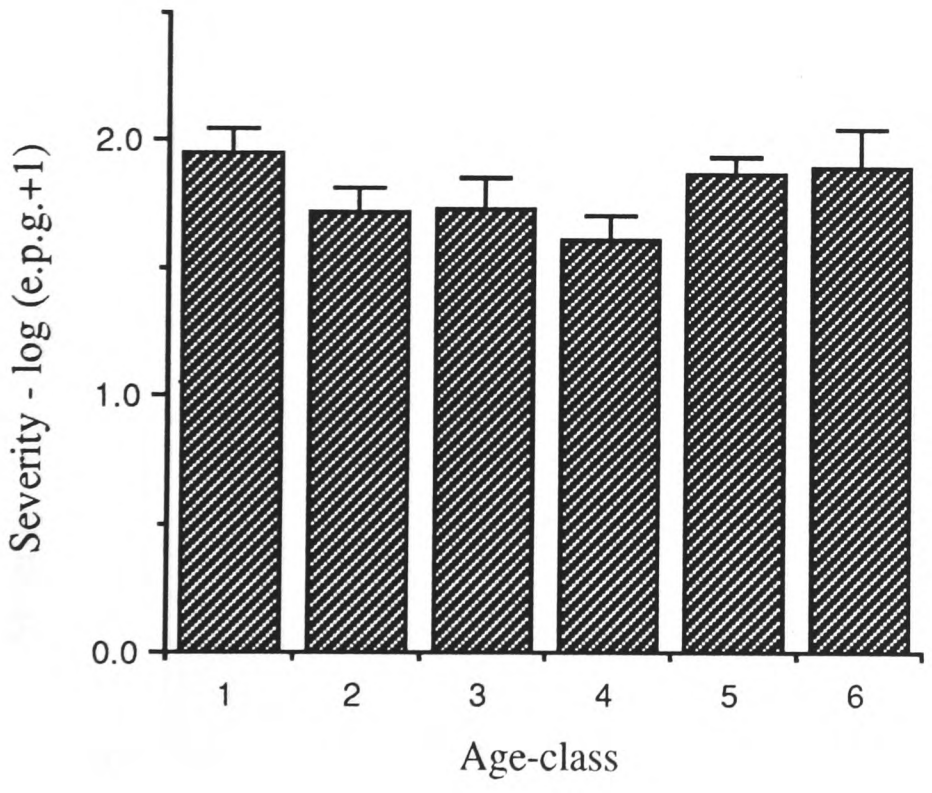


Figure 4.7. Age-severity profiles.
a) hookworm (1 n = 8, 2 n = 13, 3 n = 11, 4 n = 9, 5 n = 15, 6 n = 12)
b) *Trichuris* sp. (1 n = 27, 2 n = 29, 3 n = 16, 4 n = 27, 5 n = 36, 6 n = 18)

c) *Strongyloides* sp.

d) *Physaloptera* sp.

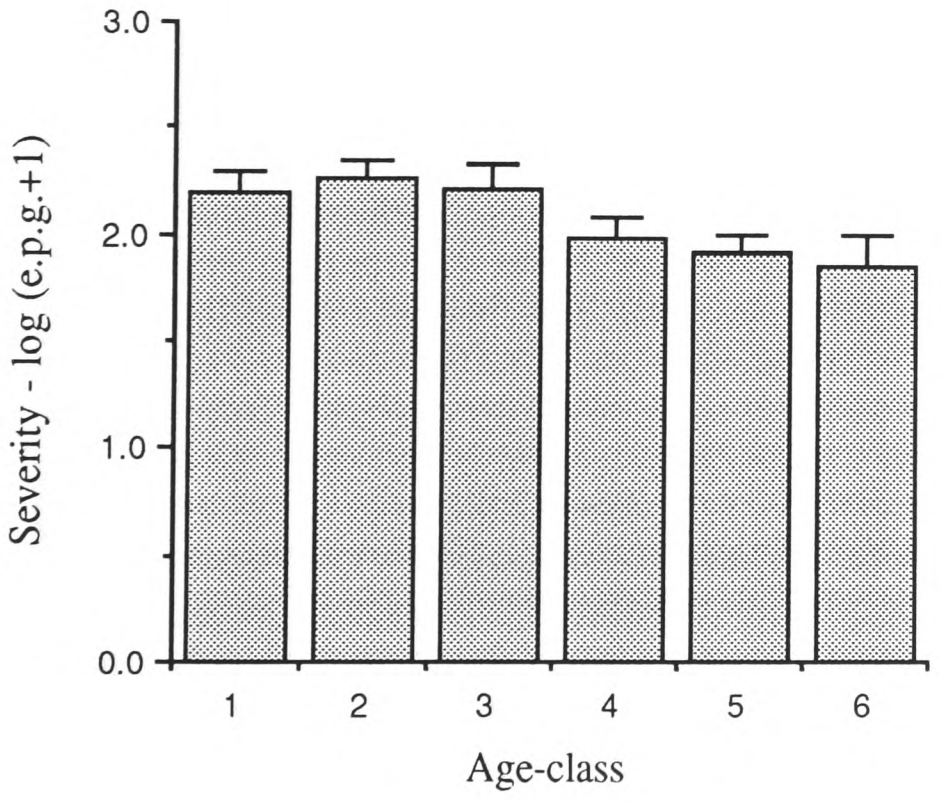


Figure 4.7. Age-severity profiles.
c) *Strongyloides* sp. (1 n = 25, 2 n = 27, 3 n = 22, 4 n = 20, 5 n = 26, 6 n = 14)
d) *Physaloptera* sp. (1 n = 17, 2 n = 21, 3 n = 14, 4 n = 22, 5 n = 35, 6 n = 15)

e) *Streptopharagus* sp.

f) *Schistosoma mansoni*

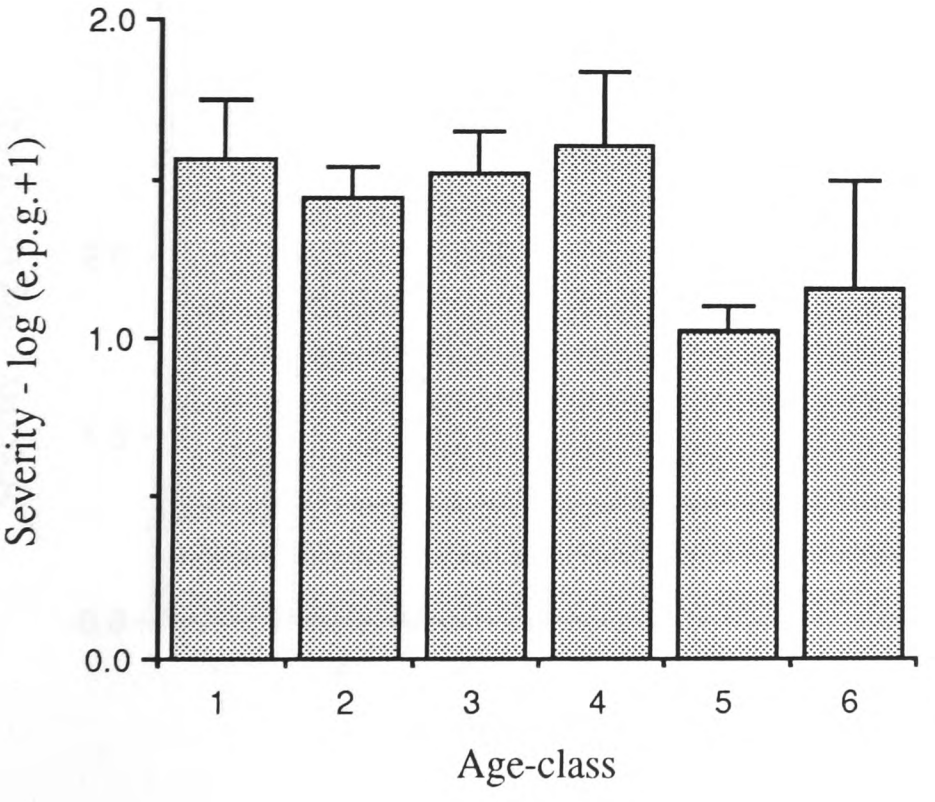
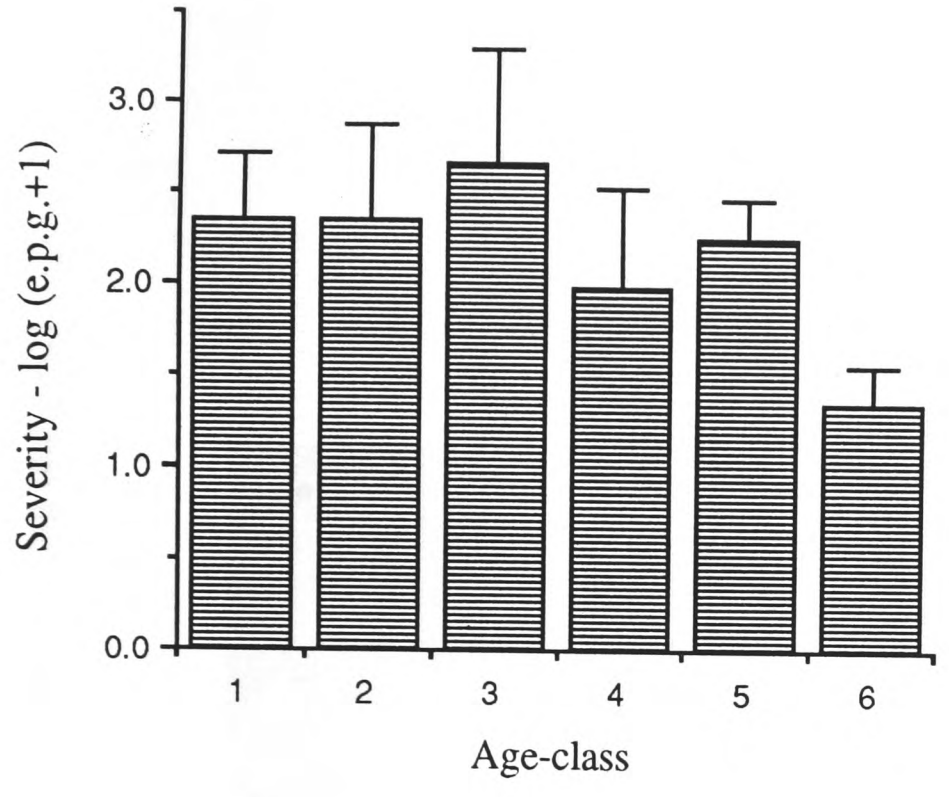


Figure 4.7. Age-severity profiles.
e) *Streptopharagus* sp. (1 n = 19, 2 n = 28, 3 n = 11, 4 n =23, 5 n = 30, 6 n =17)
f) *Schistosoma mansoni* (1 n = 8, 2 n = 10, 3 n = 6, 4 n = 6, 5 n = 8, 6 n = 4)

g) Unidentified trematode



h) Total helminth load

Figure 4.7. Age-severity profiles.
g) Unidentified trematode(1 n = 4 , 2 n = 4, 3 n = 2, 4 n = 3, 5 n = 11, 6 n = 3)
h) Total helminth load (1 n = 30, 2 n = 34, 3 n = 25, 4 n = 35, 5 n = 44, 6 n = 22)

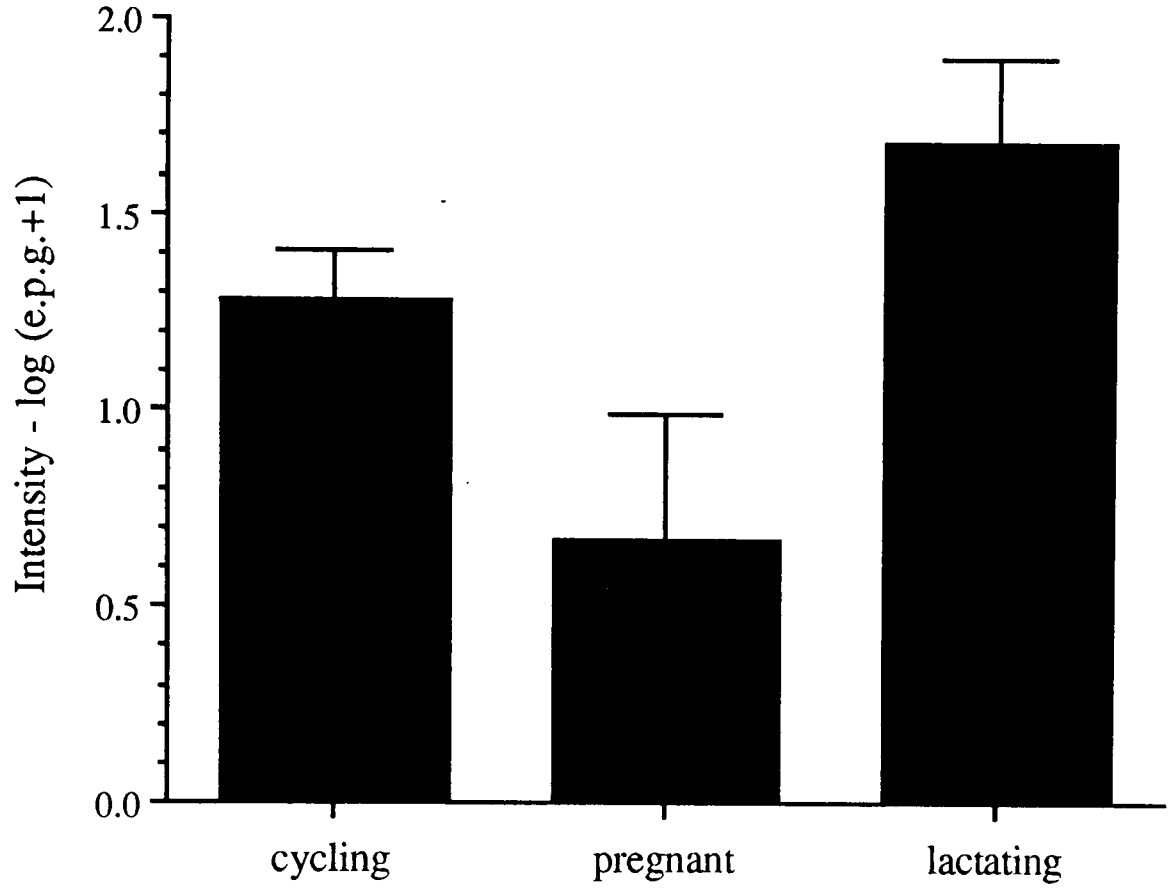


Figure 4.8. Association between *Trichuris* sp. intensity and reproductive status. Means were adjusted for age. Cycling females n = 42, pregnant n = 7, lactating n = 18. Error bars indicate the standard error.

Table 4. 2. Correlations of parasite infection with social rank.

a) Correlation of total helminth load intensity with social rank

Sex	A-troop			B-troop			C-troop			D-troop			L-troop			across troops		
	r	p	df	r	p	df	r	p	df	r	p	df	r	p	df	r	p	df
Male	.36	(N.S.)	2	-.48	0.12	6	.44	(N.S.)	5	-.39	0.18	3	-	-	-	.12	(N.S.)	27
Female	-.47	0.02	17	-.50	0.13	5	-.43	0.07	11	.36	(N.S.)	16	.74	(N.S.)	4	-.14	0.12	65

b) Correlation of *Trichuris* sp. intensity with social rank

Sex	A-troop			B-troop			C-troop			D-troop			L-troop			across troops		
	r	p	df	r	p	df	r	p	df	r	p	df	r	p	df	r	p	df
Male	.84	(N.S.)	2	-.30	0.23	6	-.38	0.20	5	-.30	0.31	3	-	-	-	-.12	0.26	27
Female	-.07	0.39	17	-.62	0.07	5	-.38	0.10	11	.19	(N.S.)	16	-.06	0.46	4	-.11	0.20	65

c) Correlation of taxa richness with social rank

Sex	A-troop			B-troop			C-troop			D-troop			L-troop			across troops		
	r	p	df	r	p	df	r	p	df	r	p	df	r	p	df	r	p	df
Male	-.70	0.15	2	.30	(N.S.)	6	-.73	0.03	5	.70	(N.S.)	3	-	-	-	-.11	0.28	27
Female	-.44	0.03	17	-.40	0.19	5	-.12	0.35	11	.17	(N.S.)	16	-.14	(N.S.)	4	-.17	0.08	65

"r" denotes the partial correlation coefficient, expressing the correlation of rank versus intensity of parasite infection allowing for variation in age. The test used was a one-tailed test, predicting a negative relationship. "(N.S.)" indicates that the relationship deviates from the predicted direction. In L-Troop samples were only available from two males of known rank and were therefore excluded from the analysis.

Table 4.3. Correlations between prevalence of parasite taxa.

	Hookworm	*Strongyloides*	*Trichuris*	*Streptopharag.*	*Physaloptera*	*Schistosoma*	Trematode	Larvae	"Amoebae"
Strongyloides	-								
Trichuris	-	-							
Streptopharag.	-	-	0.0000*** (+)						
Physaloptera	-	-	-	-					
Schistosoma	-	-	-	-	-				
Trematode	-	-	-	-	0.046* (+)	-	-		
Larvae	-	-	-	-	-	-	-		
"Amoebae"	-	-	-	-	-	-	0.012*(-)	-	
Balantidium	0.005**(+)	-	-	-	-	-	-	-	-

n = 192 Prevalences between two parasite taxa were respectively correlated, controlling for troop, age, sex and sample effort. Non significant results are indicated by " - ". Positive or negative signs in the brackets indicate whether the relationship between the two taxa is positive or negative.

Table 4.4. Prevalences of baboon parasites in previous surveys.

	Study							
	South Africa (Powell and Elsdon-Dew 1961)[1]	East Africa (Kuntz and Myers 1966)[2]	Kilifi (Kenya) (Kuntz and Myers 1967; Myers and Kuntz 1968)	Kimani (Kenya) (Kuntz and Myers 1967)[3]	Mau Narok (Kenya) (Kuntz and Myers 1967)	Tanzania (Myers and Kuntz 1967)[5]	Kenya & South Africa (Myers and Kuntz 1968; Myers *et al.* 1971)[4]	Kenya (Jessee *et al.* 1970)[6]
Worm/Egg	E	W	W	W	W	W	W&E	E
Parasites								
Nematodes:								
Trichuris spp.	18	15	17	47	17		49	33
Physaloptera spp.	0	46	0	79	0		25	16
Streptopharagus spp.	0	46	33	42	0	46	26	10
Strongyloides spp.	6						0	68
Trichostrongylus spp.	0	8				(50)	37	13
Oesophagostomum spp.	0	77	100	11	83	82	32	39
Necator spp.		8						
Ternidens spp.								5
Hookworm	76						0	
Enterobius spp.								
Oxyria spp.	0	8					2	
Trematodes:								
Schistosoma mansoni		8						
trematode unident.								14
Cestodes:								
Bertiella spp.	12	8	17	16	28	8	3	1
intest. Protozoa:								
Entamoeba histolytica	82	23	83				31	
Entamoeba coli	100	62	100			(83)	45	
Endolimax nana	47	23	16				6	
Iodamoeba bütschlii	65	31	50			(33)	18	
"Amoebae"	0	54 (unid.)	66				43 (unid.)	
Balantidium coli	0	46	100				64	
No of individuals	17	13	6	19	18	50 (6)	106	100

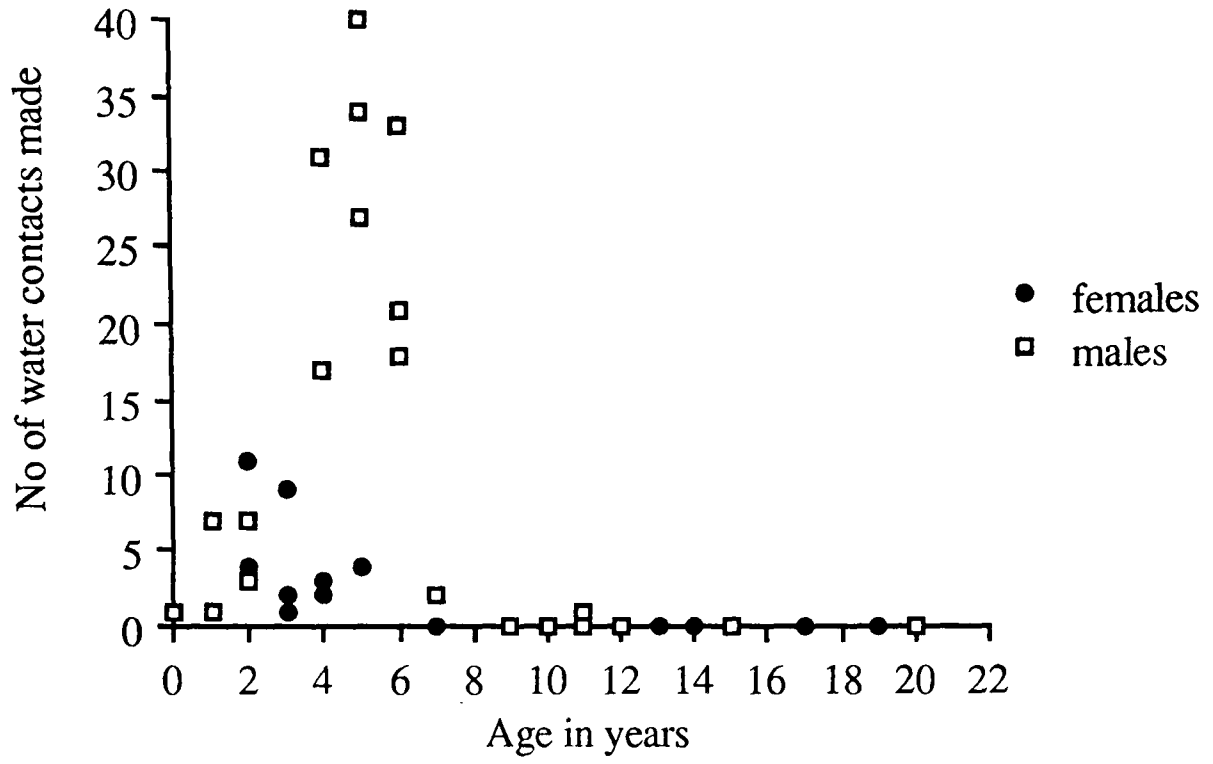
	Study							
	South Africa (Myers *et al.* 1971)[7]	Rift Valley, Kenya (Kuntz and Moore 1973)[8]	Zimbabwe (Goldsmid 1974)	Kruger National Park (McConnell *et al.* 1974)[9]	Northern Transvaal (Goldsmid and Rogers 1978)[10]	Amboseli (Meade 1983)[11]	Transvaal (Pettifer 1984)	Drakensberg Mountains, South Africa (Appleton *et al.* 1986)[12]
Worm/Egg	E	W & E	W & E	W&E	E	E	W	E
Parasites								
Nematodes:								
Trichuris spp.	22		4		4	98	1	89/24
Physaloptera spp.	12	2	8	21	4	88/37	94	47/42
Streptopharagus spp.	3	3/12/40	45	57	4	2	93	2/0
Strongyloides spp.	12		39	4	38/4	19	20	0
Trichostrongylus spp.	11	17	8	24	8	81	50	0
Oesophagostomum spp.	0	34	69	82	63	26	97	
Necator spp.			57			0		
Ternidens spp.				3	4	0		
Hookworm	0			78		0		47/51
Enterobius spp.		43/10	22			0		9/3
Oxyria spp.	0					0	14	
Trematodes:								
Schistosoma mansoni trematode unident.			2	6				
Cestodes:								
Bertiella spp.	0	27	4	26	4	2		11/8
intest. Protozoa:								
Entamoeba histolytica	3	16						
Entamoeba coli	37	42		12	54			78/62
Endolimax nana	11	9						4/16
Iodamoeba bütschlii	22	28		8	4			2/1
"Amoebae"	53(unid.)			18	4 (unid.)			
Balantidium coli	46	75		52	54			20/32
No of individuals	54	127	51	100	24	43	108	122

	Study						
	Mt. Assirik, Senegal (McGrew *et al.* 1988)[13]	Gombe, Tanzania (McGrew *et al.* 1988)	South Africa, Mkuze Reserve (Appleton 1989)[14]	Kenya (Eley *et al.* 1989)[15]	Zululand, South Africa (Appleton *et al.* 1991)[16]	Gombe (Murray 1991)[17]	Gombe (this study autumn 1991)[18]
Worm/Egg	E	E	E	E	E	E	E
Parasites							
Nematodes:							
Trichuris spp.	28	42	3	22	6	66	81
Physaloptera spp.	31	44	1		2	23	66
Streptopharagus spp.	23	35	16		30	48	67
Strongyloides spp.	26	58		19	23/6	23	69
Trichostrongylus spp.			7		1		
Oesophagostomum spp.	0	42			15	17	
Necator spp.	38	0					
Ternidens spp.					7		
Hookworm			26	96		54	36
Enterobius spp.					1		
Oxyria spp.							
Trematodes:							
Schistosoma mansoni	23	0				3	21
trematode unident.	0	2				43	15
Cestodes:							
Bertiella spp.			2		4		
intest. Protozoa:							
Entamoeba histolytica							
Entamoeba coli	87	no data collect.				34	
Endolimax nana							
Iodamoeba bütschlii	38	0					
"Amoebae"							76
Balantidium coli	72	42					20
No of individuals	39	52	123	67	191	35	206

The first line reports the study location and the study from which the data were taken. Worm (W) or Egg (E) indicates whether counts were made from worms from autopsies or eggs from faecal samples. The percentage of infection is given to the nearest whole number."Hookworm" as an extra category reports percentage of infection, where no distinction between different hookwormlike eggs could be made. The final line notes the number of individuals examined, although frequently in the literature there is no distinction made between number of individuals and number of samples taken. An empty cell indicates that this parasite was not mentioned in the study. Only studies of wild baboons in Africa were taken into account, and only the more common intestinal parasites are recorded in the table. The rare parasite genera are reported as footnotes.

1 Infection was also reported for *Entamoeba hartmanni* (58%) and *Ascaris* (35%).
2 Infection is also reported for the trematode *Brodenia laciniata* (8%) and the acanthocephalan *Nephridiacanthus* spp.(8%)
3 Infection with *Protospirus* spp. (11%) is also reported.
4 Rather confusing as this is a summary of the three previous columns with more added data. *Protospirus* spp.(1%) and protozoans *Dientamoeba fragilis* (1%) and *Entamoeba hartmanni* (6%) are also reported.
5 Additionally, infection with *Entamoeba polecki* (16%) was reported. Protozoa and *Trichostrongylus* sp. are reported from the stool samples of 6 baboons (in brackets).
6 Infection with *Physocephalus* spp. (2%) is also reported.
7 Infection was also diagnosed with *Entamoeba chattoni* (15%) and *Entamoeba hartmanni* (11%).
8 Protozoan prevalences were taken from faecal samples and helminth infection from autopsies. Infection with *Entamoeba hartmanni*(28%) *Entamoeba chattoni* (7%), *Chilomastix mesnili* (2%), *Isospora* sp. (1%), *Streptopharagus* infections were differentiated in *Streptopharagus baylisi* (3%), *Streptopharagus pigmentatus* (12%) and *Streptopharagus* sp. (40%); *Physaloptera* infections were differentiated in *Physaloptera caucasica* (2%) and *Physaloptera* sp. (2%) and *Enterobius* infections were differentiated into *Enterobius brevicauda* (43%) and *Enterobius* sp. (10%).
9 Helminths are reported from necropsies, only hookworm is documented from faecal samples of 99 individuals, all protozoans are noted from the faecal samples of 99 individual.
10 *Chilomastix mesnili* (4%), *Trichomonas* sp. (4%); *Strongyloides* spp. was also observed as 38% *Strongyloides fuelleborni* and 4% *Strongyloides stercoralis* .
11 Meade (1983) reports 88% infection with *Physaloptera* sp. and 37% with *Abbreviata* sp.
12 Data given for 2 troops but the number of troop members and how many samples per troop is not specified. 122 faecal samples were taken altogether. Additionally an unidentified taeniid (2%) was reported for the first, the 'upper' troop, and an infection with *Chilomastix mesnili*:17 % for the upper and and 9 % for the lower troop.
13 An unidentified *Stringeodea* sp. (17%) is also reported.
14 An unidentified taeniid (2%) and an unidentified acanthocephalan (1%) are reported.
15 Also *Ascaris* (2%)
16 *Strongyloides* were diagnosed as *Strongyloides fuelleborni* (23%) and *Strongyloides stercoralis* (6); also an unidentified acanthocephalan (1%), an unidentified anoplocephalid (1%), *Schistosoma haematobium* (1%) and *Schistosoma mattheei* (1%).
17 Also *Ascaris* sp. (63%)
18 Also larvae (4%)

a)



b)

Figure 4.9. Water-contact of baboons in relation to a) age and b) the previously defined age-classes. The quality of water contact is not taken into account (n = 43; f = 21, m = 22). However, females and males showed qualitatively similar kinds of water contact. Individuals with the same age and same number of water contacts appear as one data point. (D.A. Collins, unpublished data).

Parasite infection among five troops of olive baboons (*Papio anubis*) in Gombe Stream National Park, Tanzania

ABSTRACT

Parasitological data from a cross-sectional study of five troops of wild olive baboons (*Papio anubis*) in Gombe Stream National Park were used to compare parasite infection in the different troops. There were significant differences among troops in the prevalence and/or intensity/severity of all recorded helminthic parasites. Parasite prevalence in different troops may be related to some extent to environmental conditions, host genetics, the extent of interaction with neighbouring troops or to the introduction of parasites at one point in time. Baboons that were in closer contact with humans did not have more or different parasites with the exception of *Schistosoma mansoni*, which may have been introduced by humans. No positive correlation between troop size and prevalence was found. *Trichuris* sp. and *Strongyloides* sp. showed a positive and *Streptopharagus* sp. a negative correlation between intensity and troop size. Variation among troops contributed most to the explained variance in infection, with the exception of *Strongyloides* sp., where age was more important, but still did not account for most of the heterogeneity. The remaining heterogeneity of infection may be related to measurement error and individual differences in exposure, innate susceptibility or acquired immunity.

5.1 INTRODUCTION

The previous chapter showed that host-specific factors - such as sex, age and behaviour - may cause heterogeneity in parasite infections in a host population. However, these factors may not explain all the variation in infection in the host population. Environmental factors may also be important. Examining spatial variation in infection may indicate the effect of environmental factors although host-specific factors may also cause spatial heterogeneity of infection. Differences in prevalence and intensity of parasite infections between host populations on a larger geographical scale have frequently been observed (Dogiel 1964; Esch *et al.* 1990; Esch and Fernandez 1992), but spatial differences among host groups that live in close proximity to each other have received less attention (but see Forrester *et al.* 1988; Sousa 1990; Anderson *et al.* 1993; Jaenike 1994). If the causes of small-scale spatial differences in infection can be identified, they may help to shed light on factors influencing host-parasite interaction within host groups. Furthermore, differences in parasite infection among groups may contribute to the overall aggregated distributions of infection in subdivided populations (Jaenike 1994). So far, research on wild populations of large mammals has concentrated mainly on individual hosts or populations, and not much is known about parasite infections among social groups of the same host species in close proximity. Studies which deal with several groups of the same species on a small scale have been mainly on humans (Forrester *et al.* 1988; Anderson *et al.* 1993; Chan *et al.* 1994), fish (Holmes 1990) or invertebrates (Sousa 1990; Jaenike 1994).

The well-studied baboon population in Gombe National Park offered a good opportunity to study the differences in infection among five adjacent troops. These troops are not genetically distinct because of the close common ancestry of at least three troops and because of male migration between all troops. Age and sex of troop members is known and can therefore be controlled for in the analysis. Moreover, troops did not markedly differ in their age structure. Data from the cross-sectional parasitological study undertaken in autumn 1991 were used to compare the troops. The study examines whether parasite infection differs among troops and whether these also interact with other factors influencing parasite infection, such as age and sex.

Differences among troops may be related to their local environment, host genetics and aspects of host-behaviour, such as group size. If infection is influenced by environmental factors, then troops which live in closer proximity should show more similar infections. Percentage dissimilarity can be used to investigate whether contiguous troops are more similar in their parasite prevalences. Adult males migrate

between troops, if environmental factors are important, these males should, after some time, acquire the parasite profile of the troop to which they have migrated. If host genetics are important then parasite burden of closely related troops are expected to resemble each other more than other troops.

Disease has been suggested to play a part in the evolution of primate social behaviour (Freeland 1976). Group size is determined by a cost benefit trade-off that may include factors relating to disease control (Møller *et al.* 1993). With the exception of vector-transmitted parasites, larger groups may maintain more parasites, hence by limiting the number of group members, hosts might reduce their individual risk of disease. Therefore, larger baboon troops may have higher prevalences and intensities of parasitic infections.

The maintenance of a home range may also act as a disease avoidance mechanism (Freeland 1976), either by limiting the contact of conspecifics or by reducing the diversity and size of the area inhabited and thus reducing the probability of acquiring certain parasites. Differences in levels of infection among troops could indicate that maintenance of home range may be associated with parasite infection.

Baboon troops may also differ in levels of infection, depending on the extent of their contact with humans. As humans and baboons have parasites in common one would expect that closer contact with humans would result in higher prevalences and intensities of human parasites being found in the baboons, and vice versa. This possibility is also examined in this study, as the Gombe troops differ in their proximity to human settlements.

5. 2 MATERIALS AND METHODS

Study area and study organisms

Study area, host species, sampling methods and parasite species are the same as described in the previous chapter. Home ranges of the five troops are shown in Figure 5.1.

Statistical Analysis

Parasitological terms are defined as in chapter 4. The analyses are set out the same way as described in chapter 4 and are based on log(x+1) transformed helminth egg counts. Estimates of parasite richness, intensity, severity and total helminth load for

each individual were obtained as before. The level of significance was set at 0.05. P-values are two-tailed.

Parasite prevalence among troops was compared using log-linear analysis controlling for age, sex and interactions. Age was treated as a categorical variable with two classes: young and old (young $\leq$ 8 years; old > 8 years). Unidentified larvae were excluded from the comparison between troops because of very low overall prevalence.

Because interest was in differences between troops, severity was only compared among troops when there was no significant difference in prevalence, using GLMs (General Linear Models) including age, sex and interactions in the model. However, to study the contribution of troop to overall heterogeneity in intensity of infection, intensity was modelled by including age, sex and troop but no interactions terms.

To compare the dissimilarity in parasite prevalence (including all parasite taxa which are contained in taxa richness) among troops and between troops and migrating males, the percentage dissimilarity was calculated according to the following formula:

$$PD_{jk} = IA - PS_{jk}, \text{ where } PS_{jk} = \frac{200\sum_{i=1}^{I}\min(A_{ij}, A_{ik})}{\sum_{i=1}^{I}(A_{ij} + A_{ik})}$$

PD = percentage dissimilarity (between *j* and *k*)
IA = internal association (to be taken as 100)
PS = percentage similarity (between *j* and *k*)
The summations are over all helminth taxa *I*, where A_{ij} and A_{ik} are the prevalences of species *i* in samples *j* and *k* (Gauch and Hugh 1982). Adult males were excluded from the calculation of a troop's level of infection as they migrate between troops and may therefore confound a troop's parasite profile.

Troop sizes were compared to prevalence, intensity, severity and richness of infection by Spearman rank correlation coefficients, excluding the unidentified trematode which appeared mainly in one troop.

5.3 RESULTS

Differences in parasite infection among troops

Most of the parasites occurred in all troops. Only two helminths, *Schistosoma mansoni* and the unidentified larvae, were not found in all troops. In A-troop and L-troop no infection with *Schistosoma mansoni* could be detected. The unidentified

trematode predominantly infected C-troop and to a very small degree the other troops. No unidentified larvae were found in L-troop.

Even though the five troops live in close proximity, and three of them are genealogically related, the prevalence of most of the parasite taxa tested was significantly different in at least one of the troops compared to the other troops (Table 5.1.a). The only exception was *Trichuris* sp., which showed similar prevalence in all troops (Table 5.1.a), but differed significantly in severity and intensity between troops (severity $F_{4,156} = 11.96$, $p < 0.001$ in Table 5.1.b; intensity: $F_{4,194} = 5.47$, $p < 0.001$) Troop differences contributed most to the explained variation in helminth intensities (see sums-of-squares values in Table 5.2.), with the exception of *Strongyloides* sp., where age was more significant, but troop contributed to an almost equivalent extent. However, troop, age and sex together typically explained less than 25% of the total variation in intensity (with the exception of the unidentified trematode).

Troop membership not only directly influenced parasite infection, but also interacted with other factors which had an impact on parasite prevalence. For two parasites, *Schistosoma mansoni* and *Streptopharagus* sp., an age-with-troop interaction was detected (*Schistosoma mansoni* $Z = -2.75$, $p < 0.01$ *Streptopharagus* sp. Z-value = -2.05, $p < 0.05$). Prevalences of infection in some age-classes were found to be significantly different from the same age-classes in other troops (Figures 5.2. a and b and 5.3.).

Associations of percentage dissimilarity among troops with home range, relatedness and male migration

The calculation of percentage dissimilarity did not show that troops, which lived together more closely were supporting more similar parasite assemblages (Table 5.3; Spearman rank correlation coefficient corrected for ties Rho = 0.073, Z = 0.22, p = 0.83). Between closely related troops (A, B and D) the percentage dissimilarity of parasite prevalences was not lower than between troops which were more distantly related (Mann-Whitney *U* corrected for ties, $U = 9$, $Z = -0.34$, $p = 0.73$).

When comparing percentage dissimilarities of migrating males with the natal troop and troop of residence to the time the males had spent in their troop of residence, it was not found that their was a certain length of time after which an immigrant male had a more similar parasite profile to his new troop than his natal one (Table 5.4.).

The influence of group size on parasite infection

No overall relationship between group size and parasite richness was found in this study. There was almost no difference in the number of parasite taxa harboured in the five troops (Table 5.5.). Three troops were infected by eight helminth taxa, one by seven and the other one by six. Schistosomes and the unidentified larvae were the only parasites which could not be found in all troops, and their absence was not connected with troop size. There was no positive correlation between the percentage of infected individuals in a troop and group size. One negative association between group size and "amoebae" infection was found (Table 5.6.). No association between mean intensity and mean severity of infection of a troop and troop size could be found for any parasite taxon (Table 5.6.).

5.4 DISCUSSION

Inter-troop heterogeneity in parasite infection

Inter-troop differences were very marked and troop membership was an important determinant of an individual's level of infection. Several factors may account for these differences, such as environmental factors in the home range of a troop, contact with neighbouring troops, introduction of parasites at a particular point in time, troop size, troop genetics and contact with humans. However, the main determinant of inter-troop differences was not obvious in this study. It seemed to be unlikely that overall genetics was important, because more closely related troops were not supporting more similar parasite assemblages. If environmental factors play an important role, it would be expected that immigrant males would share a more similar parasite profile with their troop of residence than with their natal troop after sufficient time lapse. However, this could not be demonstrated. The result may be biased by the fact that males might have visited other troops after leaving their natal troop before they became resident in their present troop. No exact information is available on these movements. In addition, the method of calculating percentage dissimilarities may not be sensitive enough - as it was developed for determining differences between populations, rather than one individual and a population - and repeated samples of males may be necessary to determine their exact relationship with previous and present troops. The longevity of some helminths may also confound results. Contiguous troops did not show more similar parasite prevalences. On the other hand, this does not preclude the importance of environmental

factors or exposure as focal points of transmission for certain parasites may be situated in a particular troop's home range and these may not be found in neighbouring troops.

Differences in the infection of the five troops with *Schistosoma mansoni* and another unidentified trematode may be partly explained by an interaction of the environment with the parasite life-cycle and as a consequence of historical events. Both parasites rely on water and snails for transmission. Water holes or streams are reported on all troop ranges, with the exception of L-troop, where there are streams, but probably no water holes. All baboons have access to Lake Tanganyika, but this part of the lake is apparently snail- and schistosome-free. Only in the home-range of C-troop were intermediate host snails (*Biomphalaria pfeifferi*) found. However, sampling was by no means comprehensive and thus does not exclude the existence of snails in low densities in the other areas. It seems that most troop ranges could potentially maintain both parasites.

Schistosoma mansoni was found to infect only individuals of three troops: B-, C- and D-troop. Although all three troops have contiguous ranges (Figure 5.1.), no jointly shared water holes are known. This seems to suggest that independent transmission occurs in all three troop ranges. Schistosomes may have been introduced to all three ranges by humans. They were not reported in a previous paper on baboon parasites in Gombe (McGrew *et al.* 1988). *Schistosoma mansoni* is known to have been endemic in the human population since 1983. Schistosomes were not recorded in humans before 1975 in Gombe (D. A. Collins, personal communication). This suggests that they were introduced at some point between these two dates. The range of D-troop overlaps with Kakombe village. D-troop has the most contact with humans and also the highest prevalence of *Schistosoma mansoni.* B- and C-troop have limited contact with humans. A- and L-troop also have some contact with humans, possibly less than the other three troops, but the infection has not yet appeared in the baboons. Humans have greater opportunities to become infected than baboons as they are more mobile and travel more frequently between Gombe and other areas. Therefore, they may have introduced *Schistosoma mansoni* to the Gombe area. On the other hand, because the distribution of the snails is incompletely known, an absence of the snails in a troop's home range may explain the absence of the parasite.

The unidentified trematode was primarily found to infect C-troop, and several of the infected individuals in other troops had originated in C-troop. In a previous study (McGrew *et al.* 1988), only a single animal in B-troop was found to be infected. There is anecdotal evidence that the trematode may be detrimental to the health of the baboons. C-troop has been reported as looking "scruffy" and unhealthy ever since the 1970's (D.A. Collins, C. Packer, personal communication). When members of three troops, A-, C-, and D-troop, were weighed in December 1989, females were found to have a

significantly lower average weight in C-troop than in other troops (ANOVA $F_{2,29} = 5.21$; $p = 0.013$; controlled for age). Females in C-troop showed significantly longer inter-birth intervals than females in any of the other troops and young C-troop females give birth to their first offspring at a significantly greater age (reproductive success was monitored from 1970 - 1992; C. Packer, personal communication). The swamp which lies in the range of C-troop may be a good environment for the assumed intermediate host, a snail; and possibly the only place where the infection has been introduced and can be maintained. Alternatively, C-troop may be weak for other reasons, such as an unknown disease or bad nutrition, and therefore prone to additional infection.

For two parasites (*Schistosoma mansoni* and *Streptopharagus*) an interaction of troop with the effects of age on parasite prevalences was also found (Figures 5.2. a and b and 5.3.). In the case of *Schistosoma mansoni*, these differences may be related to variations in rates of infection experienced by the troops. Troops which had higher schistosome prevalences also displayed an earlier peak in infection and a more convex curve (Figure 5.2. b) as predicted by epidemiological theory if acquired immunity has significant effects (Anderson and May 1991; Woolhouse *et al.* 1991). However, the same pattern was not found for *Streptopharagus* (Figure 5.3.). Here, the pattern may be explained by different exposure in the different troops.

Influence of host interspecific associations on infection

Closer contact to humans may also affect parasite infection of baboons and vice versa. Eight of the nine parasite taxa found in these baboons can also be acquired by humans (Nelson 1965; Goldsmid 1974; Appleton 1989). Hookworm, *Strongyloides*, *Trichuris*, *Schistosoma mansoni*, *Balantidium coli* and "amoebae" are considered parasites of humans. *Physaloptera* and *Streptopharagus* only infect humans incidentally (Nelson 1965; Appleton 1989). There was no evidence that contact with humans lead to an increase in prevalence or intensity in baboons for any of the parasites investigated here, with the exception of *Schistosoma mansoni*. If contact with humans had an effect on levels of infection, then prevalence and intensity of parasite infection should have been highest in D-troop, whose range included the village, and which had the highest contact with humans. This was only the case for *Schistosoma mansoni*. On the other hand, all baboon troops in Gombe have some contact with humans, therefore, unless the effect is quite strong, it would be difficult to observe. Further studies are needed where troops with no human contact are compared with troops with human contact.

In Gombe, chimpanzees probably share four helminths with the baboons: *Trichuris*, hookworm, *Strongyloides* and *Physaloptera* (McGrew *et al.* 1988; Murray

1991). Contact with chimpanzees did not seem to affect parasite profiles of the baboon troops in this study. Chimpanzees visit the ranges of C- and D-troops more than the other troops' ranges, although they pass regularly through all the baboon ranges. In the home ranges of C- and D-troop a chimpanzee feeding station is situated, where chimpanzees and baboons tend to assemble, especially C-Troop. Here, the soil is probably more heavily contaminated with parasite eggs and larvae, but no difference in the pattern of parasite infection to the other troops was obvious. Further investigation is required to establish whether contact with humans or chimpanzees leads to higher parasite infections in baboons.

The effect of troop size on parasite infection

Larger groups may maintain more parasite species or a higher prevalence of parasites (Freeland 1976), though this relationship depends on the mode of transmission (Anderson and May 1991). In a smaller group the absence of a parasite species may result from the increased probability of random extinction of parasites (Freeland 1979). In a previous study of rain forest primates (*Cercocebus* and *Cercopithecus*) significant differences in the prevalences of protozoan parasites were positively correlated with troop size (Freeland 1979). The troops differed in size between 7 and 22 individuals. A similar argument could apply to certain helminths. However, for most directly transmitted helminths, the transmission threshold is so low that host density is not a limiting factor for parasite transmission and parasites can persist even at very low host densities (Anderson and May 1991; Anderson 1993). On the other hand a larger group may also maintain a higher intensity of infection, because greater environmental contamination with parasite infective stages by infected group members could increase the risk of further infection. Models suggest that the net rate of infection is directly proportional to the density of infective stages times the density of the host (Anderson and May 1991).

In this study overall parasite richness was not evidently related to troop size. There were no positive correlations of prevalence of any infections with troop size in accordance with the prediction for macroparasites. Contrary to expectations, there was one negative association with troop size, for "amoebae" (Table 5.6.). As the result was only marginally significant, it should be treated with caution. Furthermore, no correlation between intensity and severity and troop size were observed in contrast to predictions. These results must be treated with caution due to the small number of troops studied, the limited range of troop size. On the other hand, they do suggest that troop size may not be important in influencing levels of infection in the Gombe baboons.

Parasite infection and baboon home range

Freeland (1976) proposed that the maintenance of a home range or territory is in itself an effective disease avoidance mechanism. He suggested that a home range would limit the contacts with other conspecifics which did not belong to the same troop. However, a maintenance of a home range may also be important because it could limit the number of different habitats a host has contact with and thus reduce the probability of acquiring different parasites or reduce the area of the same habitat containing a patchily distributed parasite. On the other hand, a small home range may lead to higher intensities of infection and increased transmission, whereas in large home ranges contact with disease contaminated areas (such as soil with nematode eggs) can be more easily avoided.

Freeland's hypothesis is difficult to test, because of the many confounding variables. However, the observations in Gombe Stream National Park could not reject the hypothesis. Troops differed in their parasite load, this observation hints that home range may have an influence on parasitic infection. Although home ranges do overlap, baboon troops tend not to be together in the areas often. However, encounters between neighbouring troops take place. Troop size did not appear to be positively correlated with levels of infection, but one consequence of home range may be that very high densities of baboons where host density may be noticeably positively correlated with rate of infection do not occur. It is not known whether the "healthiness" of a home range has an effect on how home ranges are established. It would be interesting to know if the healthier home ranges are taken up by more dominant troops.

5. 5 CONCLUSION

Of all the variables tested troop membership constituted the predominant factor contributing to heterogeneity. The only exception was *Strongyloides* sp. where age had an equivalent impact. This still explained only a small fraction of the total variation. Although measurement error may account for much of the remainder, it is likely that within troop differences in exposure, innate susceptibility and acquired immunity are also important. This result was also supported by the finding (in the previous chapter) that parasite distribution among hosts was aggregated.

All directly transmitted parasites with the exception of the unidentified larvae were found in all troops, but varied in levels of infection among troops. Some indirect

life cycle parasites showed a focal distribution, possibly related to intermediate host ecology or introduction at some point in time. Humans may introduce parasites to baboon populations- such as *Schistosoma mansoni* - but other effects for baboons living in close proximity to humans could not be detected in Gombe Stream National Park. Troop size appears not to be associated with levels of infection in the Gombe baboons.

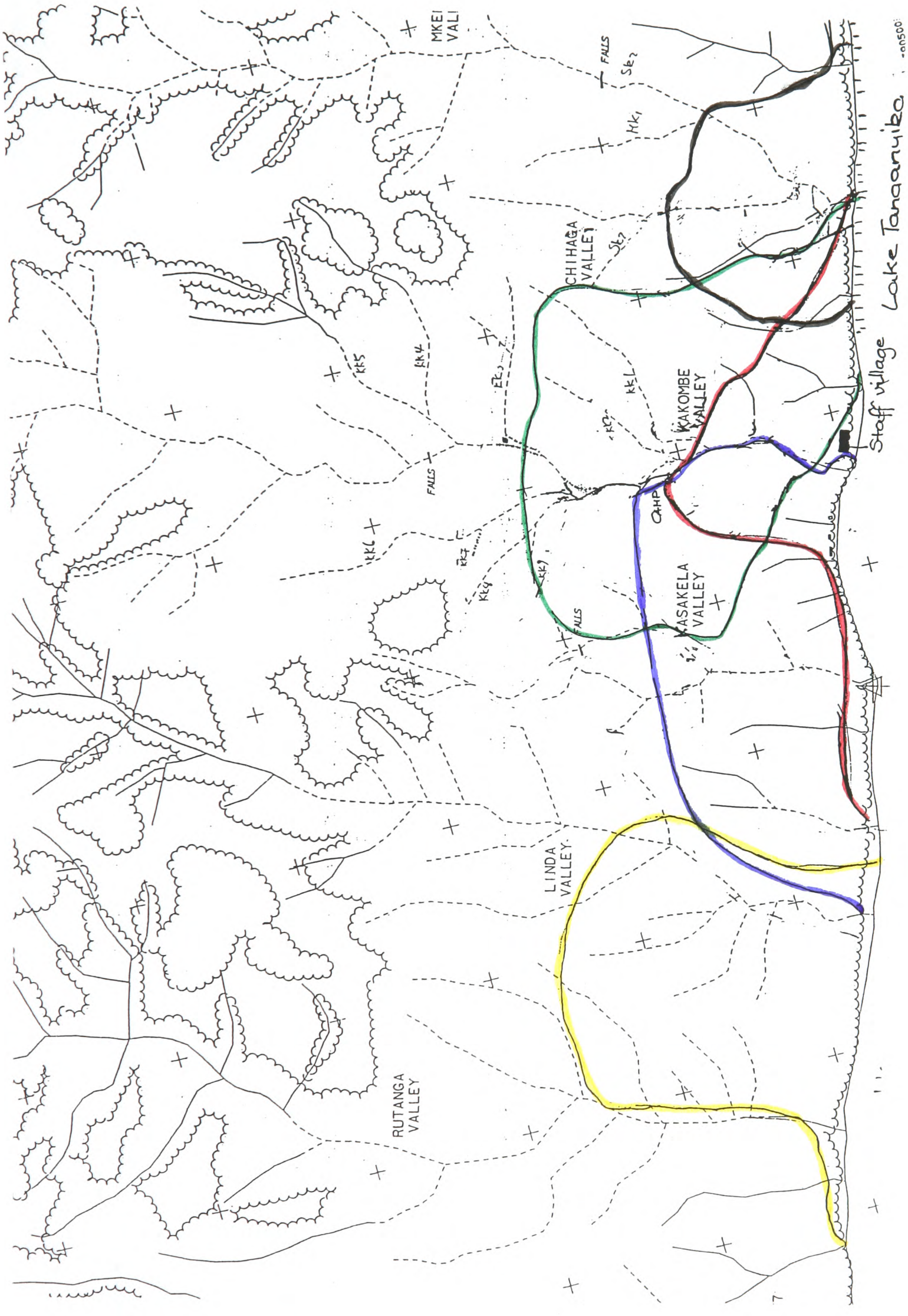


Figure 5.1. Map showing the core home ranges of each troop. The troops' sightings were recorded daily. These resulted in monthly records of home ranges. The monthly home ranges from over two years (1990 - 1992) were combined for this map. Maximum ranges of all troops were larger than the main troop ranges shown. Mean home ranges are the most commonly used home ranges when superimposing all home ranges recorded from one troop. A-troop = blue, B-troop = black, C-troop = green, D-troop = red, L-troop = yellow.

in that troop. Troops are ordered according to home ranges. Age and sex were controlled for in a log-linear analysis. Z- and p-values indicate the greatest variation found between troops.

Parasites	L-troop	A-troop	D-troop	C-troop	B-troop	Z-max	p-value
Hookworm	54	54	20	24	26	-2.68	<0.01
Strongyloides sp.	56	85	74	61	65	-2.22	<0.05
Physaloptera sp.	73	72	62	82	36	2.92	<0.005
Streptopharagus sp.	68	85	42	66	81	3.66	<0.0005
Schistosoma mansoni	0	0	45	26	36	-3.09	<0.005
Trematode	2	2	4	66	3	-6.50	<0.0001
Trichuris sp.	83	96	74	76	71	-1.90	>0.05
Size of Troop	64	66	67	42	33		

Table 5.1.b. Variation of *Trichuris* sp. severity among troops.
The columns for each troop show the mean value of log (x+1) transformed *Trichuris* sp. severity. Age and sex were not significant in GLM analysis.

Parasite species	L-troop	A-troop	D-troop	C-troop	B-troop	F-value	p-value
Trichuris sp.	1.47	1.91	1.94	1.99	1.45	$F_{4,156} = 11.96$	<0.001

Table 5.2. Intensity among troops compared for the helminth taxa.

a) ANOVA-Table for hookworm

Source	DF	Seq SS	Adj SS	Adj. MS	F	p
Troop	4	11.23	11.25	2.81	7.72	<0.001
Age	1	0.36	0.34	0.34	0.94	0.33
Sex	1	0.00	0.00	0.00	0.01	0.91
Error	193	70.28	70.28	0.36		
Total	199	81.87				

b) ANOVA-Table for *Strongyloides* sp.

Source	DF	Seq SS	Adj SS	Adj. MS	F	p
Troop	4	18.39	16.30	4.07	3.31	0.012
Age	1	18.74	16.79	16.79	13.62	<0.001
Sex	1	1.85	1.85	1.85	1.50	0.22
Error	194	239.18	239.18	1.23		
	200	278.16				

c) ANOVA-Table for *Trichuris* sp.

Source	DF	Seq SS	Adj SS	Adj. MS	F	p
Troop	4	13.55	13.84	3.46	5.47	<0.001
Age	1	0.66	0.93	0.93	1.47	0.23
Sex	1	1.25	1.25	1.25	1.98	0.16
Error	194	122.64	122.64	0.63		
Total	200	138.10				

d) ANOVA-Table for *Physaloptera* sp.

Source	DF	Seq SS	Adj SS	Adj. MS	F	p
Troop	4	27.67	26.96	6.74	6.61	<0.001
Age	1	0.56	0.68	0.68	0.66	0.42
Sex	1	0.32	0.32	0.32	0.31	0.58
Error	194	197.88	197.88			
Total	200	226.43				

e) ANOVA-Table for *Streptopharagus* sp.

Source	DF	Seq SS	Adj SS	Adj. MS	F	p
Troop	4	34.01	34.18	8.54	6.39	<0.001
Age	1	0.62	0.36	0.36	0.27	0.61
Sex	1	1.68	1.68	1.68	1.25	0.26
Error	194	259.55	259.55	1.34		
Total	200	295.85				

f) ANOVA-Table for *Schistosoma mansoni*

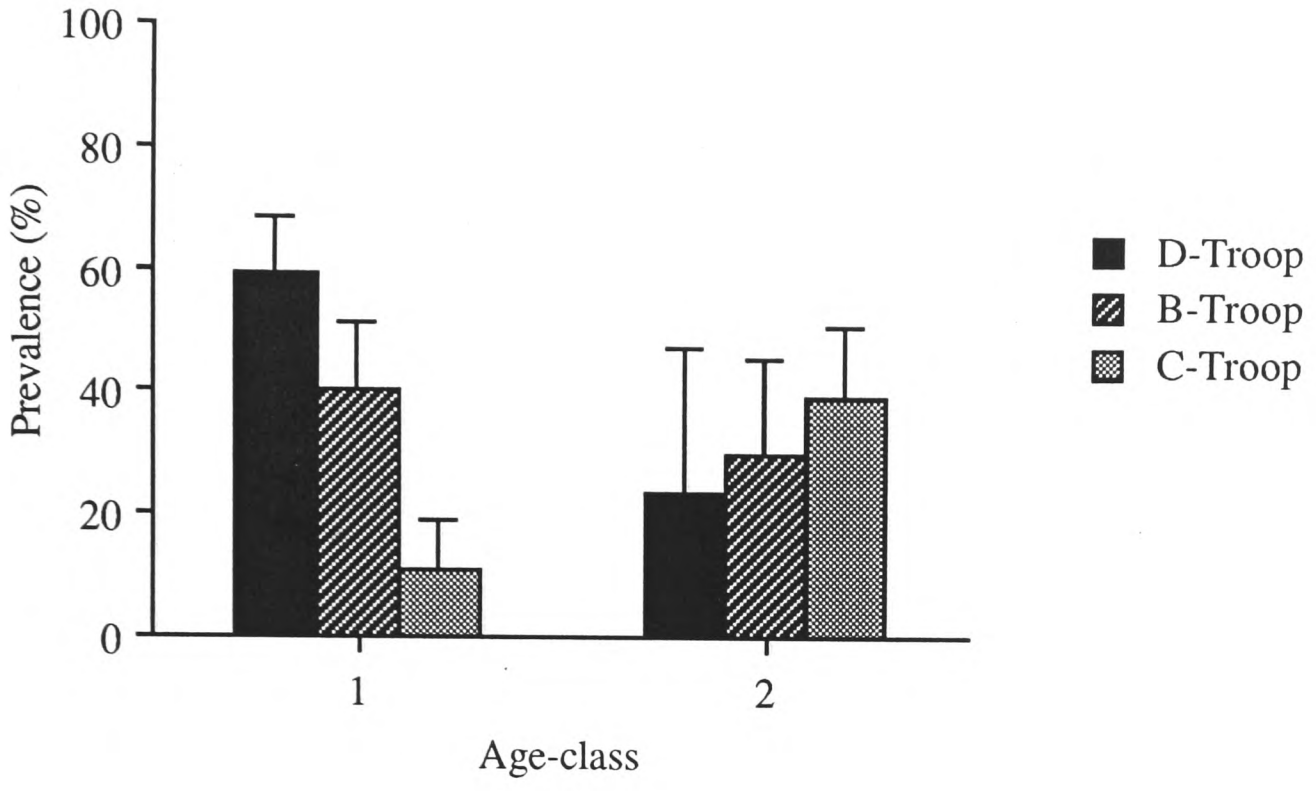
Source	DF	Seq SS	Adj SS	Adj. MS	F	p
Troop	4	17.18	16.26	4.06	14.41	<0.001
Age	1	0.86	0.51	0.51	1.82	0.18
Sex	1	1.82	1.82	1.82	6.46	0.01
Error	193	54.46	54.46	0.28		
Total	199	74.32				

g) ANOVA-Table for unidentified trematode

Source	DF	Seq SS	Adj SS	Adj. MS	F	p
Troop	4	65.00	64.21	16.05	50.77	<0.001
Age	1	0.00	0.00	0.00	0.00	1
Sex	1	0.00	0.00	0.00	0.00	1
Error	194	61.34	61.34			
Total	200	126.33				

Fewer DF for hookworm and *Schistosoma* are reported because in each case one sample had to be excluded from the analysis.

a)



b)

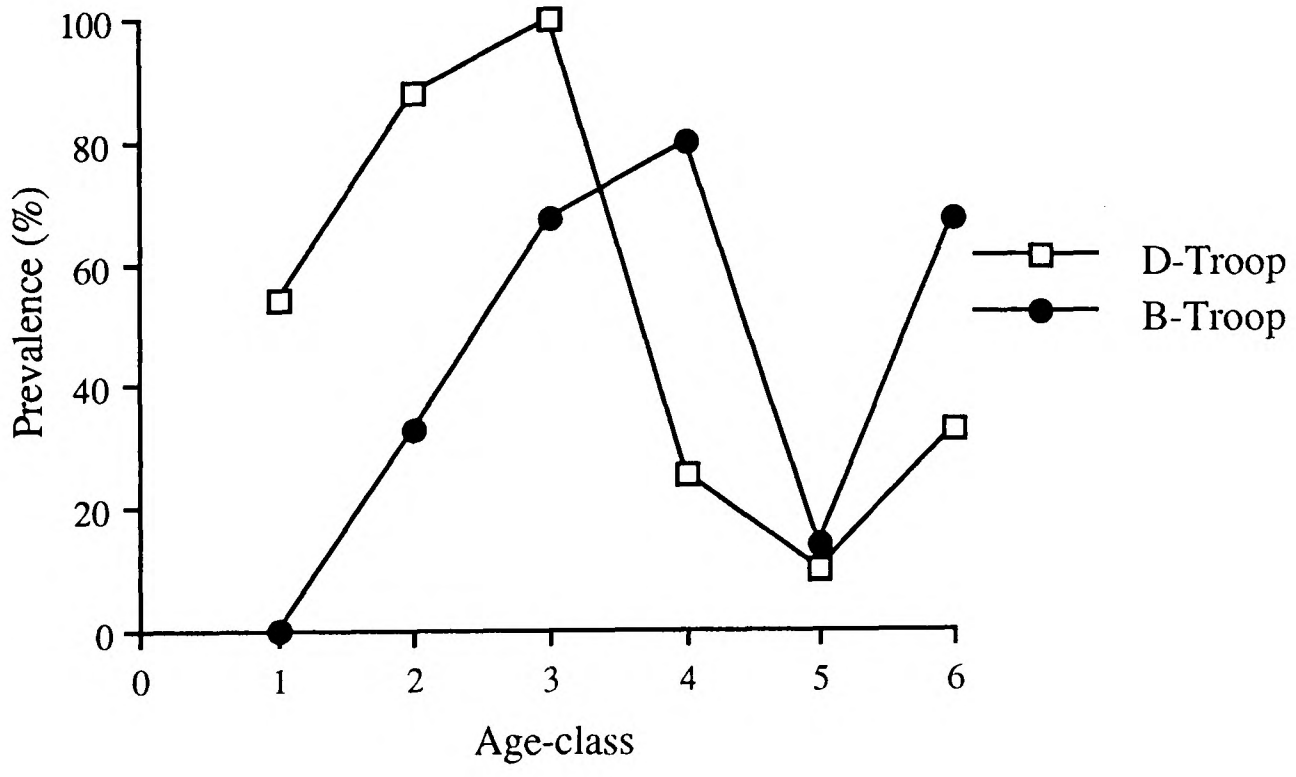


Figure 5.2. a) Differences in prevalence of *Schistosoma mansoni*.in the two age-classes in different troops. (1 = young, ≤ 8 years, 2 = old, > 8 years; A-troop, 1 n = 29, 2 n =17; B-troop 1 n = 20, 2 n = 10; C-troop 1 n = 18, 2 n = 18; D-troop 1 n = 32, 2 n = 17; L-troop 1 n = 24, 2 n = 6). Error bars indicate one standard error.
b) Age-prevalence profile of *Schistosoma mansoni* over six age-classes. Age-classes are categorized as in chapter 4 (B-troop 1 n = 6, 2 n = 6, 3 n = 3, 4 n = 5, 5 n = 8, 6 n = 2; D-troop 1 n = 13, 2 n = 8, 3 n = 4 , 4 n = 8, 5 n = 10, 6 n = 6); C-troop was not included as data for age-classes 4 and 5 were missing.

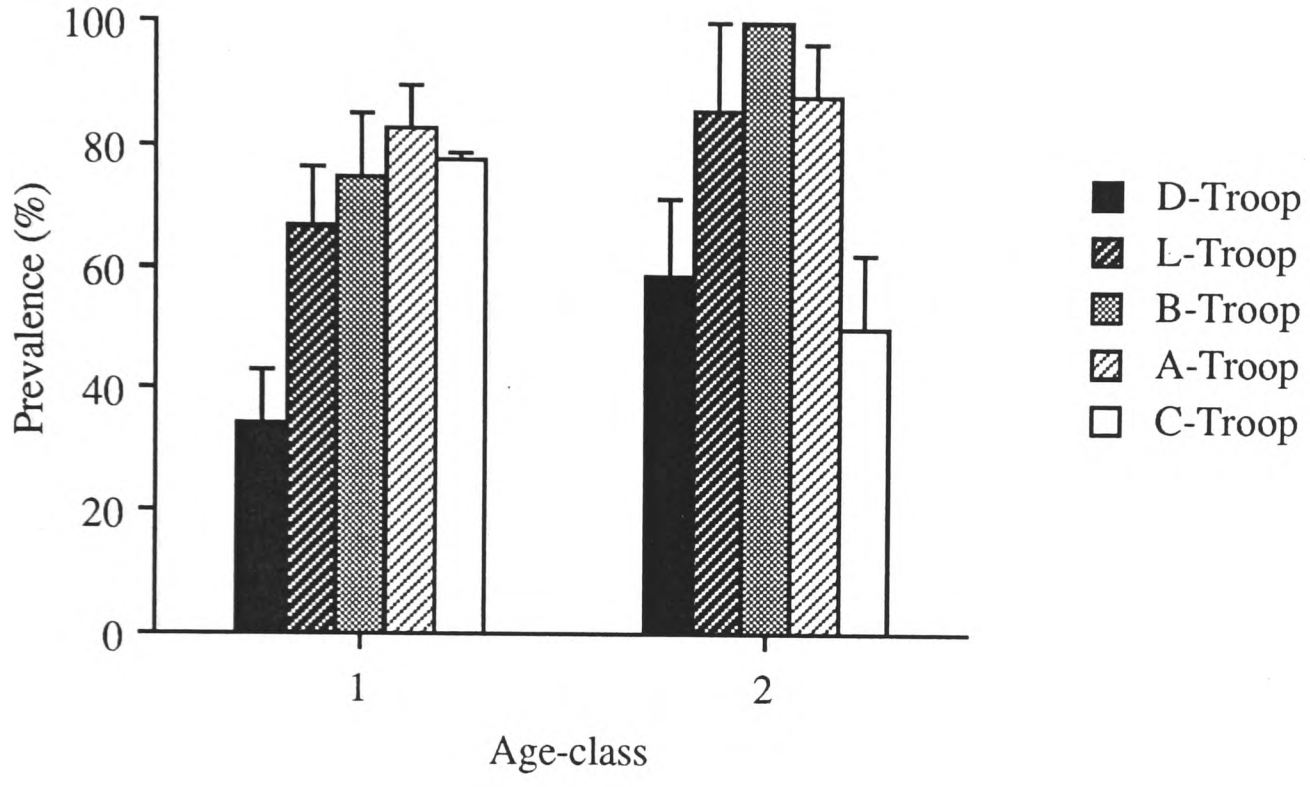


Figure 5.3. Differences in prevalence of *Streptopharagus* sp. in the two age-classes (1 = young, ≤ 8 years, 2 = old, > 8 years) in different troops. Error bars indicate one standard error.

Table 5.3. Percentage dissimilarity among troops. Percentage dissimilarity of parasite prevalences were calculated for all combination of troops, given in the left column. The right column indicates the distance between the compared troops.

Troops	adjacent troops	troops one removed	troops further removed	midpoint distances (in m)
DB	15.61			900
BC	19.95			1100
DC	23.94			525
LA	11.04			700
AC	24.50			1200
AD	26.61			1000
CL		18.40		1750
DL		24.50		1650
BA		22.28		1900
BL			19.78	2550

Table 5.4. Percentage dissimilarity of migrated males. Percentage difference was calculated using information on all helminths, comparing the "prevalence" of infection (as 0 or 1) of one individual male with the parasite profile of the respective troops, excluding resident males.

Name of male	Troop of residence	Natal troop	time after migration in months	similarity to troop of residence
Jason	38.06	56.95	129	+
Bofu	13.07	20.57	75	+
Nakuru	39.85	30.96	72	-
Bombay	30.99	15.69	71	-
Macbo	44.71	51.69	67	+
Ethopia	38.73	32.23	67	-
Horlix	24.17	35.58	56	-
Sangara	15.75	22.97	56	+
Patrick	44.84	42.74	56	-
Savana	25.20	40.53	45	+
Paulo	40.53	21.18	40	-
Keke	36.80	35.14	28	-
Devota	50.23	44.82	26	-
Yabassi	32.89	15.75	23	-
Sukari	17.79	37.02	20	+
A.Pacake	47.76	50.23	9	+
Kunira	32.23	38.74	8	+
Bubango	58.51	39.48	5	-
Dahomey	31.96	23.40	3	-
Dagaa	12.79	20.57	1	+

"+ "indicates that similarity of a males parasites profile is more similar to his troop of residence than to his natal troop, "-" shows the opposite.

Table 5.5. Parasite richness among troops.
Taxa richness was calculated excluding protozoans and the unidentified parasites including multiple samples. Numbers in brackets indicate if there was a difference when using only single samples from individual baboons.

Troop	A	B	C	D	L
no. of troop members	66	33	42	67	64
maximum no. of taxa in one individual	7 (5)	6 (5)	7 (6)	7 (5)	5
minimum no. taxa richness in one individual	2 (1)	1	1	1	1
median	4	5 (3)	4	3	3
total no. of taxa	7	8	8	8	6

Table 5.6. Prevalences and intensities correlated with troop size. Prevalences and intensities of each troop were correlated with troop using the Spearman rank correlation coefficient corrected for ties.

	Prevalence			Severity		
Parasites	Rho	Z	p	Rho	Z	p
Hookworm	-0.1	-0.2	0.84	0.6	1.2	0.23
Strongyloides sp.	0.5	1	0.32	0.9	1.8	0.07
Trichuris sp.	0.4	0.8	0.42	0.2	0.4	0.69
Physaloptera sp.	0	0	1	0	0	1
Streptophara-gus sp.	-0.3	-0.6	0.55	-0.7	-1.4	0.16
Schistosoma mansoni	0.05	0.10	0.92	-0.5	-0.71	0.48
"Amoebae"	-1	-2	0.046*	-	-	-
Balantidium coli	-0.9	-1.8	0.07	-	-	-

* = $p < 0.05$

CHAPTER 6

Temporal changes in parasite infection of a population of olive baboons (*Papio anubis*) in Gombe Stream National Park, Tanzania

ABSTRACT

Patterns of parasitic infection in a population of olive baboons (*Papio anubis*) in Gombe Stream National Park were studied for three consecutive seasons (dry, wet and dry) from spring 1991 until spring 1992. Seventy individual baboons from five different troops were sampled during each season. Prevalence data were compared to samples collected in 1988/89 and to a previous parasitological study in 1974. Between 1974 and 1989, the population acquired only one new parasite, *Schistosoma mansoni,* while the occurrence of all other nine parasite taxa remained the same. From spring 1991 to spring 1992 temporal but not seasonal variation was observed in levels of infection for three parasite taxa: *Trichuris* sp., *Strongyloides* sp. and unidentified protozoans ("amoebae"). Seasonal variation was found for the prevalence of *Physaloptera* sp., *Streptopharagus* sp. and *Balantidium coli* and intensity/severity of hookworm. Four parasite taxa, *Trichuris* sp., *Strongyloides* sp., *Physaloptera* sp. and *Balantidium coli,* showed different seasonal patterns in different troops, indicating that spatial and temporal patterns can interact in a host-parasite system and contribute considerably to the overall heterogeneity of host-parasite associations. Change of female reproductive status over the three seasons did not appear to influence parasite egg output in the same individual. Change in social rank of males and females was not reflected in egg emissions.

6.1 INTRODUCTION

Variability in host-parasite interactions can occur on several levels: among groups on a spatial scale and within groups due to genetic variation, behavioural differences and environmental factors. Heterogeneity in a host-parasite system can also be due to temporal variation: between seasons, years or over longer time spans. Temporal changes in prevalence, intensity and severity of parasite infection have to be taken into account when studying the epidemiology and dynamics of disease. Temporal changes in parasite component communities (all parasite species infecting a population of a given host species) (Sousa 1994) are well reported in a number of field studies (Dogiel 1964; Kisielewska 1970b; Montgomery and Montgomery 1988, Montgomery and Montgomery 1990). Seasonal variation of infection patterns has been found for a number of parasite species (Dogiel 1964; Langley and Fairley 1982; Montgomery and Montgomery 1988; Montgomery and Montgomery 1990; Gulland 1991; Esch and Fernandez 1992).

In the following chapter, temporal and seasonal variation of parasite infection are investigated in the population of olive baboons (*Papio anubis*) in Gombe Stream National Park. Changes were examined in the structure of the baboon parasite community between 1989 and 1992. Comparison is made with previous field studies in the same area. Samples from the same individuals over three consecutive seasons from spring 1991 to spring 1992 were compared to detect seasonal differences. Interactions between season and other putative factors influencing patterns of parasite infection were investigated.

Repeated samples from the same individual over time were also used to study associations of parasite infections and female reproductive status or social behaviour.

6.2 MATERIALS AND METHODS

Materials

Host species, parasite taxa and the study area were described in chapter 4.

Seasons

The climate in Gombe is divided into wet and dry seasons. The wet season is usually from mid-October to mid-May and the dry season lasts for the other five months.

During the study period, daily rainfall data collected in the staff camp indicated that the dry season in 1991 started in June and lasted until the beginning of October whereas the dry season in 1992 started in mid-May (Figure 6.1.). From 1988 until 1992 inclusive a mean rainfall of 1405 mm per year was observed with a range between 1312 mm and 1547 mm. The mean rainfall for the years of the study amounted to 1509 mm in 1991 and 1343 mm in 1992. These observations are similar to the data from 1973 and 1974 (McGrew *et al.* 1988). However, a higher annual mean of 1600 mm and a maximum of 2600 mm have been reported (Bygott 1992). Temperatures in the shade vary during the wet season on average from 21°C to 27°C and in the dry season from 18°C to 32°C (Bygott 1992).

Methods

Sampling

Baboon faecal samples were collected in 1988 - 1989 (7.5.1988 - 19.10.1989) by D.A. Collins and field assistants without previous training in parasitological techniques. In spring 1991 (1.6.1991 - 17.7.1991), autumn 1991 (10.11.1991 - 27.11.1991) and spring 1992 (14.5.1991 - 2.6.1991) faecal samples were collected according to the methods described in chapter 2. Samples in each season were obtained from known individuals from approximately the same number of individuals per troop (Table 6.1.).

Statistical Methods

The definitions of the terms prevalence, intensity, severity, richness and total helminth load are the same as in chapter 4. The statistical analysis is based on the helminth egg and larvae counts and prevalence of protozoan parasites. The egg and larvae counts were log (x+1) transformed and the level of significance set at 0.05.

Prevalence for all sampling periods was calculated from all data, including individuals which were not considered in the seasonal analysis because they were not sampled in all three seasons. Prevalence for samples from 1988/89 was calculated both from the formol-ether and direct smear method. Repeated samples from the same individual in the same period were dealt with as described in the chapter 4. Samples used in the seasonal analysis belonged to 70 baboons which were sampled in each of the three seasons, dry (spring) 1991, wet (autumn) 1991 and dry (spring) 1992. Data on reproduction and social rank included samples from the two dry seasons, only from individuals that were sampled in both spring 1991 and spring 1992.

To determine temporal and/or seasonal variation in prevalence, data were analysed by the log-linear method across all three seasons controlling for sex, age and troop. In a second step, interaction terms between season and the other variables were included. Baboons were divided into two age classes: juvenile baboons from 0 to 8 years and the adult baboons older than 8 years. The mean age for each individual during the three seasons is used for the seasonal comparison. Season was entered as a contrast variable for adjacent comparisons, so that each season was compared with the previous season and not with the overall mean. Intensity and severity were analysed by the General Linear Model (GLM), controlling for the same independent variables as above. To study whether the variation observed was due to long-term temporal rather than seasonal variation, only the two dry seasons, spring 1991 and spring 1992 were compared. In order to be considered as seasonally variable, parasite taxa had to vary both between the first dry and wet season and then between this wet season and the second dry season but not between the two dry seasons.

Parasite infection with regard to change in female reproductive status was limited to the dry seasons to eliminate a seasonal effect and analysed by taking the difference between the two seasonal samples for the total helminth load and *Trichuris* sp., which was the only parasite found to change with reproductive status (chapter 4). The difference was then correlated with the type of change in the reproductive status in two ways: first as a non-directional change (e.g. a change between two different reproductive states: from cycling to pregnant and vice versa were put into the same category) and as a directional change (e.g. a change from pregnant to lactating and a change from lactating to pregnant were two different categories) and analysed in a GLM. Only reproductively active individuals were included.

All changes in social rank of males and females were tested for accompanying changes in intensity of total helminth load and *Trichuris* sp. infection. The difference in intensity of infection between the two dry seasons was calculated and correlated with the change in rank. Only one animal did not change rank during the study period and was thus excluded from the analysis. Results were scored according to whether the change was to a higher or lower rank and whether this was associated with higher or lower parasite infection. These four groups were analysed in a χ^2-test with continuity correction.

6.3 RESULTS

Temporal variation in parasite infection

The same 10 parasite taxa were observed in samples from all sampling periods (Table 6.2.). When comparing samples of the same individuals over the three consecutive seasons, significant variation in prevalence was found for the following parasite taxa (Table 6.3.): *Strongyloides* sp., *Physaloptera* sp. (Figure 6.2.), *Streptopharagus* sp. (Figure 6.3.), *Balantidium coli* (Figure 6.4.) and "amoebae". Because *Strongyloides* sp., *Physaloptera* sp., *Streptopharagus* sp. and *Balantidium coli* showed no significant variation in prevalence between the two dry seasons alone, the overall variation was due to differences between the dry seasons and the wet season. Of the parasite taxa only *Trichuris* sp. and "amoebae" differed significantly between the two dry seasons (Table 6.3.).

Across all seasons intensity and severity differed (Table 6.4.) for hookworm (Figure 6.5.) and the total helminth load. Severity alone differed for *Trichuris* sp., and intensity alone differed for *Physaloptera* sp.and *Streptopharagus* sp. (Table 6.4.). When comparing the intensity and severity of all parasites between the two dry seasons, only the severity of *Trichuris* sp. varied significantly (Table 6.5.).

For the parasite taxa which showed seasonal variation, the pattern of parasite infection across troops and seasons, however, was not always reflected in the pattern of parasite infection of each of the troops. Different patterns showed up for the infection of each troop in response to season (as shown in Figures 6.2. - 6.5.). The overall pattern of *Physaloptera* sp. prevalence showed higher parasite infection in the wet and lower parasite infection in the dry season. However, for the individual troops *Physaloptera* sp. prevalence decreased in B-troop over the study period and did not decrease in D-Troop and L-Troop between the wet and the last dry season. For C-Troop, no considerable difference between spring 1991 and autumn 1991 could be detected. Only A-Troop exhibited the same pattern as the overall variation. For *Streptopharagus* sp. three troops, A-, B- and C-Troop, showed the same seasonal variation as the overall prevalence with an increase of infection in the dry season. On the contrary, D- and L-Troop displayed a slight decrease in the wet season. In the case of *Balantidium coli* , the prevalence in C-Troop did not follow the overall pattern of variation over the seasons, and prevalence also failed to recover during the second dry season in B-Troop. Each of the five troops showed a different pattern for the variation of hookworm intensity over the study period. Only L-troop displayed the same kind of variation as the overall change in infection.

These observations were supported when analysing the interactions between the seasons, troop membership, sex and age. Significant interactions between troop

membership and season were found for *Physaloptera* sp. ($Z = -2.38$, $p < 0.05$; Figure 6.2.b) and *Strongyloides* sp. ($Z = 2.22$, $p < 0.05$) in one instance and for *Streptopharagus* sp. several times ($Z = 2.67$, $p < 0.01$; $Z = 2.23$, $p < 0.05$; $Z = 2.24$, $p < 0.05$; $Z = 2.96$, $p < 0.01$; Figure 6.3.b). An interaction between sex and season for hookworm prevalence ($Z = 2.22$, $p < 0.05$) was observed as well. Interactions between season and troop were found for intensity and severity of *Trichuris* sp. infection ($F_{8,187} = 5.38$, $p < 0.001$; $F_{8,148} = 4.28$, $p < 0.001$) and *Streptopharagus* sp. infection ($F_{8,189} = 3.30$, $p = 0.002$; $F_{8,77} = 2.47$, $p = 0.020$) and for intensity of *Physaloptera* sp. infection ($F_{8,189} = 2.62$, $p = 0.010$) and for intensity of the trematode infection ($F_{8,188} = 2.25$, $p = 0.026$). An interaction between season and troop was also observed for the overall helminth load ($F_{8,189} = 3.74$; $p < 0.001$).

Changes in reproductive status in association with changes in parasite burden

No significant correlation was found for either non-directional changes (total helminth load: $F_{5,34} = 0.42$, $p = 0.83$, $n = 40$; *Trichuris* sp.: $F_{5,32} = 0.88$, $p = 0.50$, $n = 38$) or directional changes (total helminth load: $F_{8,31} = 0.65$, $p = 0.73$; *Trichuris* sp.: $F_{8,29} = 0.83$, $p = 0.58$) in female reproductive status over the period between spring 1991 and spring 1992.

Correlations between social rank and parasite infection

Changes in the total helminth load and *Trichuris* sp. infection were not significantly associated with changes of social rank in the period between spring 1991 and spring 1992. Individuals that acquired higher social status did not show an increase in parasite infection, and individuals with decreased status did not exhibit a lower parasite egg count for the total helminth load or *Trichuris* sp. (Table 6.6.).

6.4 DISCUSSION

Temporal variation

Temporal changes of parasite component communities and the degree of parasite prevalence are factors contributing to heterogeneity in a host-parasite system. In some

previous studies on mammal - parasite systems, the occurrence of potentially infective parasite species for a particular host has been found to vary over different areas and time (Montgomery and Montgomery 1990; Radomski and Pence 1993); local populations of hosts occasionally lost and reacquired parasite species.

Although spatial differences were found for the baboon - parasite interactions (chapter 5), the presence of the parasite taxa over the study period seemed to be stable. All parasite taxa were found at every sampling period during this study (Table 6.2.). Comparing these data with previous Gombe baboon parasite studies (McGrew et al. 1988: A-Troop, B-Troop and C-Troop were sampled; Murray 1991: B-Troop was sampled), the same parasite species were found in 1974 and 1989 with the exception of *Schistosoma mansoni*. *Schistosoma mansoni* was not noticed in Gombe before 1975 in humans and the first record of an infected person originates from 1983 (D.A. Collins, personal communication). It seems that this parasite did not occur in the area before that time but, once introduced, established itself permanently. Another parasite, *Ascaris*, was found in Gombe baboons in 1989 (Murray 1991). However, this taxon is highly likely to have been *Physaloptera* sp. as *Ascaris* has not been reported in baboons thus far, and no *Ascaris* was detected in the samples of the first Gombe parasite study or these samples. The parasite community has varied in only one parasite species, *Schistosoma mansoni*, over a period of approximately 18 years. Thus in terms of species present, the parasite community of the Gombe baboons is a relatively stable system. The reason may be a fairly stable environment disturbed only by humans. However, as has been pointed out in chapter 4, an increase in prevalence of most of the parasite taxa compared with the previous studies was observed.

A change in abundance was recorded for several parasite taxa over the sampling period. In general, two types of temporal change have to be distinguished. One type is cyclic or seasonal variation in parasite infection as determined for instance, by the mode of life of the host, feeding patterns, food availability and changes in transmission rate, connected with climatic changes. Cyclic variation may also appear as a result of population regulation processes over a larger time span (Hudson *et al.* 1985). The other type of temporal change is non-cyclic variation influenced by different biotic and abiotic factors. In this study, seasonal variation was reported for four parasites. *Physaloptera* sp., *Streptopharagus* sp. and *Balantidium coli* showed seasonal variation in their prevalence and although hookworm prevalence did not differ significantly between seasons, intensity and severity did. This may indicate that hookworms are either producing fewer eggs throughout the wet period or that the number of infective hookworm larvae is reduced, but the probability of becoming infected was not significantly reduced. "Amoebae", *Trichuris* sp and *Strongyloides* sp. prevalences were found to differ with time, but not according to season and hence provide an example of temporal variation.

If a significant reduction in parasite ova emission is equal to a significant reduction in parasites between seasons, this decrease in parasite ova suggests a fairly short life expectancy of the parasites concerned. The loss of parasites in the next season should be due to the parasites' death. On the other hand, seasonal changes in egg production cannot be ruled out.

Life cycle, mode of transmission and host behaviour influence seasonal variation in parasite infection. An interpretation of the seasonal variation here is difficult, because the animals were sampled at the beginning of each season. Therefore, it cannot be clearly distinguished whether the difference found reflects the previous or the new season.
The prepatent period of each parasite taxon determines how long it takes until a seasonal effect can be noticed. Prepatent periods for the relevant parasite taxa, with the exception of hookworm and *Physaloptera* sp., are not known.

Balantidium coli cysts are able to survive better in a wet environment (Soulsby 1982; Cheesbrough 1987), and hence the probability of transmission would be expected to be higher in the wet season. Therefore, better transmission opportunities in the wet season may occur, but express themselves in terms of faecal cysts late in the wet season and at the beginning of the next dry season as seen here. The same argument applies to hookworm, which should be more easily transmitted when the soil is moist. Eggs of hookworm have certain moisture requirements and the survival of the larvae is also dependent on moisture and temperature (Schmidt and Roberts 1989). Furthermore, studies of temporal variation of hookworm infections in humans in areas with marked seasonal differences have shown higher levels of infection during the wet season (Chandiwana 1990). The results presented here may be due to the prepatent period of the hookworm, so that the effect of the wet season would be expressed in the early dry season data. Prepatent periods of hookworm have been reported as 15 -18 days in dogs and between 38 and 162 days in man (Soulsby 1982).

Physaloptera sp. and *Streptopharagus* sp. both showed the same seasonal effect - higher levels of infection in the wet season - and both are reported to have a similar indirect life cycle. If *Physaloptera* sp. and *Streptopharagus* sp. are transmitted by intermediate hosts, the seasonal difference in infection may reflect seasonal differences in the abundance of these intermediate hosts. For instance, dung beetles which may transmit *Physaloptera* sp. and *Streptopharagus* sp. are very active during the wet season, but scarce during drier months (Meade 1983). Nevertheless, if the prepatent period of *Physaloptera* sp. is 79 - 156 days as has been reported (Olson 1974), a higher parasite infection in the wet season should not be reflected in the wet season data, but should appear in the dry season data. Changes in egg counts could also be due to the regulation of egg production according to climate; the parasite might react to season determined stimuli, such as food and/or hormones (Hawking 1975; Kerboeuf 1987) and transmission opportunities, for

instance, increasing the egg output in the wet season when more arthropods can become infected.

Other factors may also influence parasite egg output. Host behaviour may play a role, as feeding behaviour of the baboons changes with season (J. Oliver, unpublished manuscript). It has been observed in that Gombe baboons spent more time foraging, feeding and chewing in the dry season, which would explain higher parasite infections in the dry season, and this could possibly be observed in faecal egg counts in the subsequent wet season. In the wet season the food is more plentiful and of higher quality. The nutritional status of the host may also be reflected in the immunological responses to the parasites (Nesheim *et al.* 1978; Crompton 1987; Stephenson 1987; Filteau and Tomkins 1994). The influence of nutritional status on parasite infection will vary according to the parasite and host species. Seasonal differences in baboon parasites have also been noticed in previous baboon parasite studies. Meade (1983) observed higher parasite egg output of *Streptopharagus* sp. and *Physaloptera* sp. during the dry season, which she interpreted as a higher chance of infection in the wet season. The higher likelihood of infection during the wet period could only be observed as higher egg outputs in the dry season due to a long prepatent period of the parasites. She also noticed a higher prevalence of *Strongyloides* sp., *Trichostrongylus* sp. and *Oesophagostomum* sp. during the wet season. Meade (1983) assumed this was caused by a higher chance of transmission in the wet period in connection with a short prepatent period, so that the effect was expressed in the same season. Her results directly oppose the observations here. It seems that the point of time at which the animals were sampled within each season differed from the present study. Her samples were collected throughout three months of the wet and dry season, unfortunately not indicating the overall length of the seasons and the position of the months sampled in each season. Here at Gombe, samples were collected roughly at the beginning of each season. The time of sampling within a season is probably crucial in determining the apparent effect of the season. Pettifer (1984), in another study of baboon parasites, found no difference between dry and wet season for *Streptopharagus* sp. and *Physaloptera* sp. Adult *Oesophagostomum bifurcum* burden was observed to be slightly higher during the wet season, which supports the interpretation of the present results. Pettifer dissected baboons and counted the actual helminths and did not use faecal egg counts as in the other studies. If a modulation of parasite egg output is the cause for a seasonal variation, then it is also important when comparing studies to take into account the way the data were collected. However, the difference in parasite infection over seasons may also indicate that seasonal effects vary from place to place.

Seasonal variation in parasite infection did not only differ across season but also among troops. A significant interaction was found between troop and season for four parasite taxa and for the overall helminth load. Heterogeneity on a spatial scale therefore

also extends to the effect of seasons on individual troops. Furthermore, the variation of seasonal effects on a larger geographical scale and on a smaller local scale, as in the case of inter-troop heterogeneity, shows that different levels of variability within a host-parasite system can interact. The interaction of different factors influencing variability make prediction of the overall epidemiology of a parasitic disease more complex and very much dependent on the specific parameters of each population or host-group.

Changes in reproductive status in association with changes in parasite burden

Lactating females had been found to have the highest *Trichuris* sp. egg counts (chapter 4). This result could not be replicated in the present analysis when comparing samples from females sampled repeatedly over time. The same females did not differ significantly in their parasite infection with regard to a change in their reproductive status. The interaction of parasites with their host reproductive status may be mediated by other factors, such as the genetic and immunological status of the individual. Thus at certain times the effect may become obvious and at other times will be confounded by other effects. Moreover, the changes in parasite infection due to reproductive status may be very subtle and may need a larger sample size to be statistically significant.

Correlations between social rank and parasite infection

Changes in the social status of the host were not found to be correlated with a change in parasite infection, either positive or negative. This result stands in contrast to the findings in a previous study in Amboseli (Hausfater and Watson 1976). The association of the baboons' social rank with parasite infection may be influenced by other factors, such as feeding ecology or general health of the baboons. If the higher parasite egg output of high ranking baboons is reflecting the effects of stress on the immune system, then perhaps the Gombe baboons are in general more immunocompetent (because they are, for example, better fed) and the effect does not show up. Therefore, a correlation may be observed in one place but not in another.

6.5 CONCLUSION

The parasite component community in terms of presence of individual parasite taxa was stable over the sample period. However, seasonal (cyclic), and temporal changes in the parasites' egg output were observed for several parasite taxa. The seasonal differences varied also on a spatial scale, indicating that interaction of several factors causes variability within a host-parasite system. Overall helminth load and *Trichuris* sp. infection over time were not found to be significantly influenced by an individual's social or reproductive status. Spatial and temporal factors are in this current study more important than social or reproductive status in shaping population dynamics of the parasites.

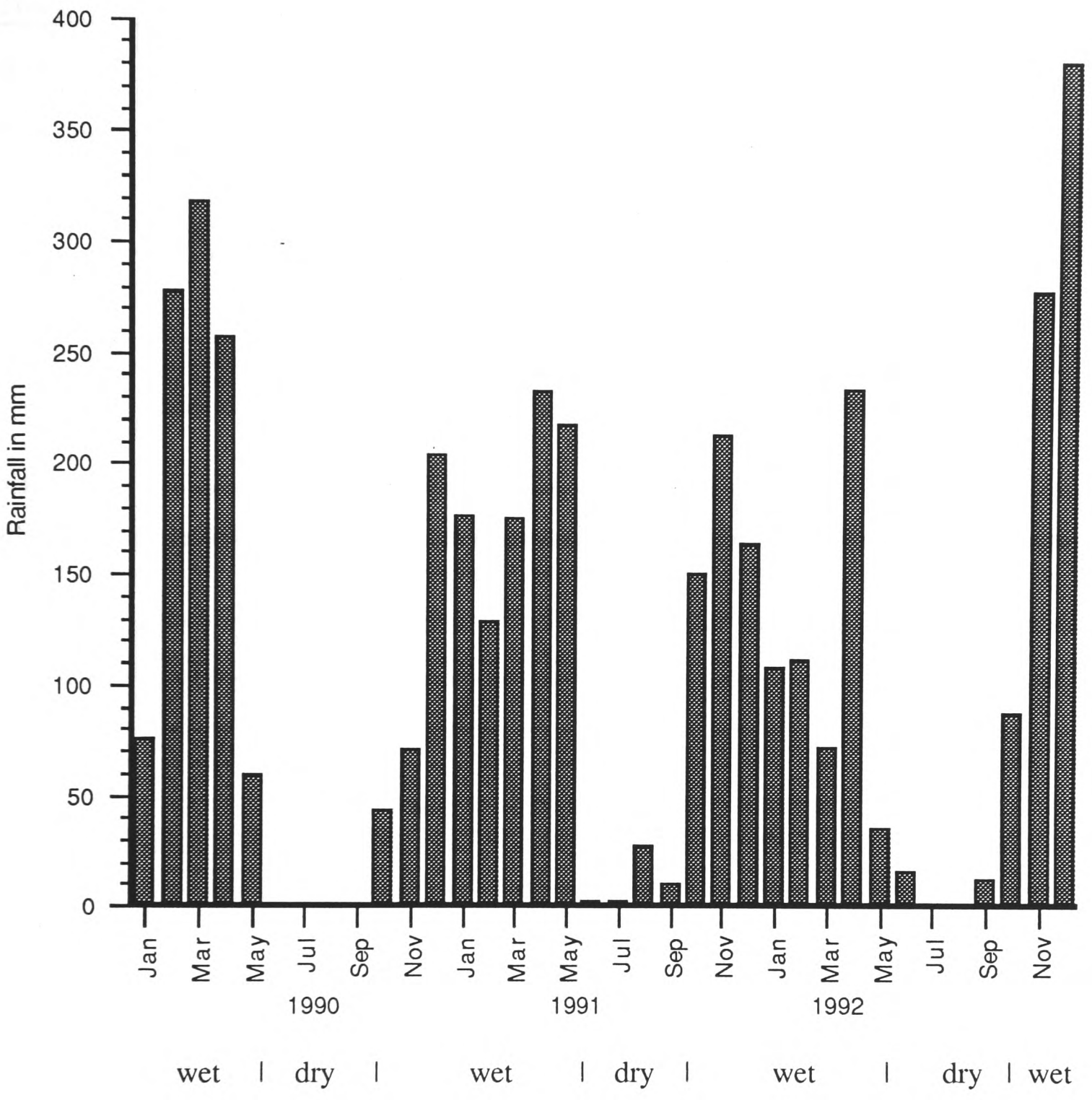


Figure 6.1. Monthly rainfall in Gombe Stream National Parks for the years 1990 1992. Rainfall was measured in mm.

Table 6.1. Distribution of samples for all three seasons across troops.

Troop	Number of individuals
A-Troop	14
B-Troop	13
C-Troop	13
D-Troop	17
L-Troop	13

Table 6.2. Prevalence (%) of parasite taxa across all sampling periods.

Parasite	1988/1989 (n = 41)	spring 1991 dry season (n = 97)	autumn 1991 wet season (n = 206)	spring 1992 dry season (n = 80)
Trichuris sp.	82.9	85.6	80.6	74.4
"Amoebae"	80.0	93.6	76.2	85.0
Strongyloides sp.	53.7	46.4	68.9	56.3
Streptopharagus sp.	41.5	45.4	67.0	33.8
Physaloptera sp.	70.0	53.6	65.5	46.3
Hookworm	68.3	50.5	36.4	46.3
Schistosoma mansoni	26.8	21.7	21.4	21.3
Balantidium coli	x	56.3	20.4	52.5
Unidentified trematode	24.4	7.2	14.6	7.6
Unidentified larvae	9.8	5.2	4.4	8.8
Unidentified	33.3	8.4	2.4	1.3

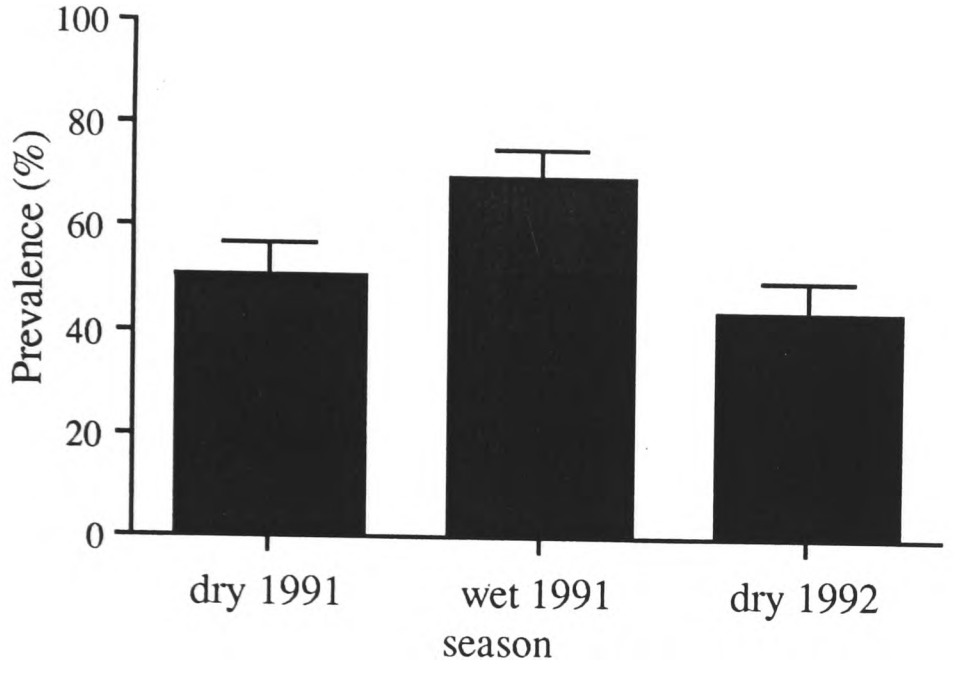
x not checked for

Table 6.3. Variation in prevalence across the seasons for individuals sampled in each season.
Data were analysed with log-linear method. The first Z-value column gives the differences across the three seasons: in the first row between the first dry season (spring 1991) and the wet season (autumn 1991) and in the second row between the wet season (autumn 1991) and the second dry season (spring 1992). The second Z-value column reports the differences between the dry seasons only.

Parasite	Z-value	p value	Z-value	p value
Hookworm	-1.72	n.s.	-0.04	n.s.
	1.30	n.s.		
Trichuris sp.	-0.74	n.s.	-2.01	*
	-1.29	n.s.		
Strongyloides sp.	2.74	**	1.32	n.s.
	-1.47	n.s.		
Physaloptera sp.	2.38	**	-0.82	n.s.
	-3.15	***		
Streptopharagus sp.	2.26	*	-1.46	n.s.
	-3.64	***		
Schistosoma mansoni	-0.12	n.s.	0.34	n.s.
	0.48	n.s.		
Unidentified trematode	1.28	n.s.	0.02	n.s.
	-1.26	n.s.		
Balantidium coli	-4.58	***	-0.66	n.s.
	4.07	***		
"Amoebae"	-3.39	***	-2.34	**
	1.50	n.s.		

for first two columns n = 3 x 70; for the other two columns n =2 x 70.
* = $p < 0.05$
** = $p < 0.01$
*** = $p < 0.001$
n.s. = not significant

a)



b)

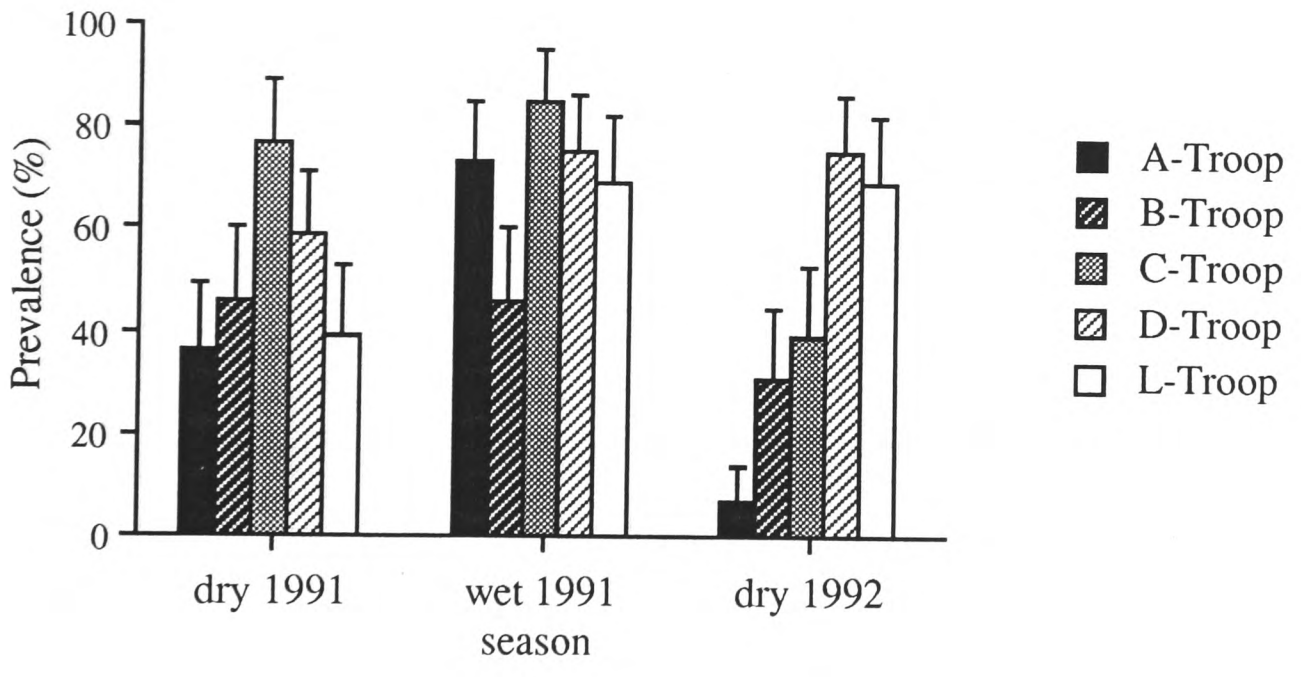
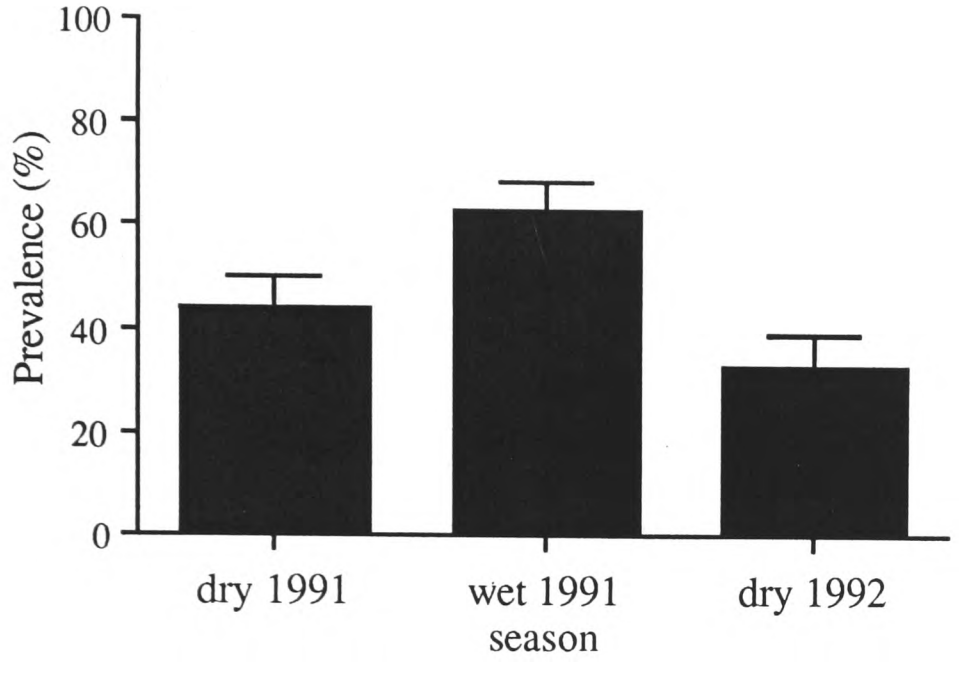


Figure 6.2. Seasonal variation of *Physaloptera* sp. prevalence.
Prevalence of infection is plotted for each season, in graph a) for the whole population and in graph b) for each troop separately. Error bars indicate one standard error.

a)



b)

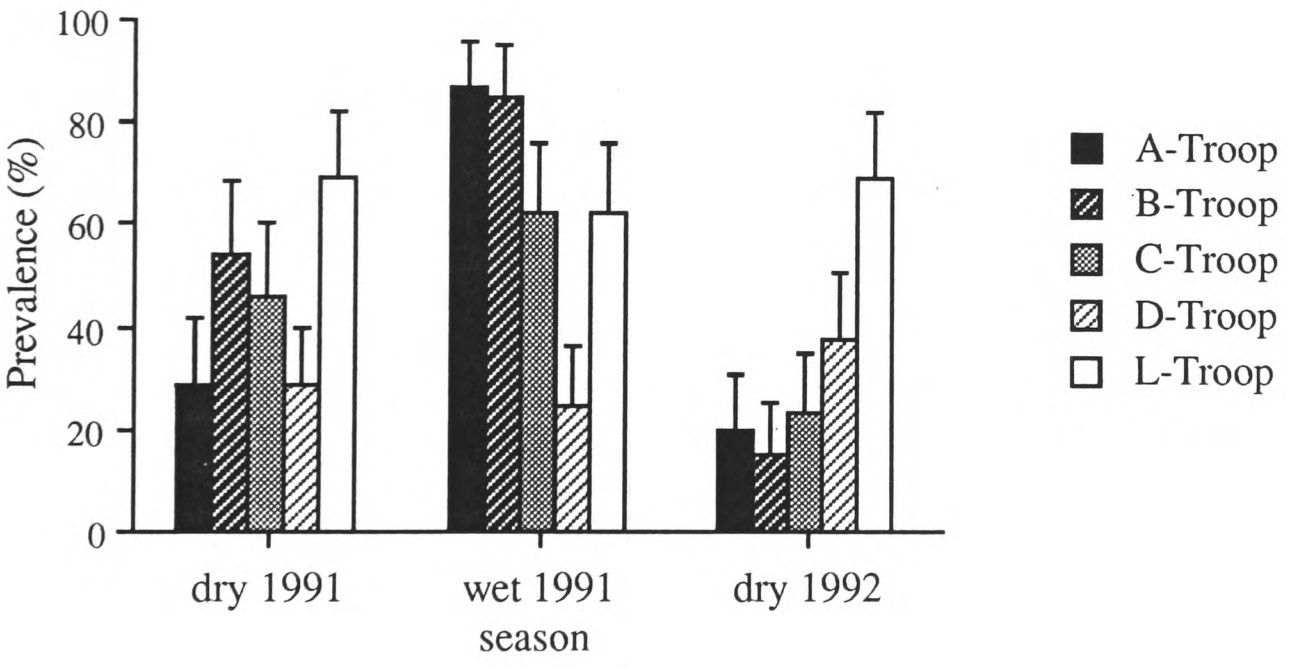
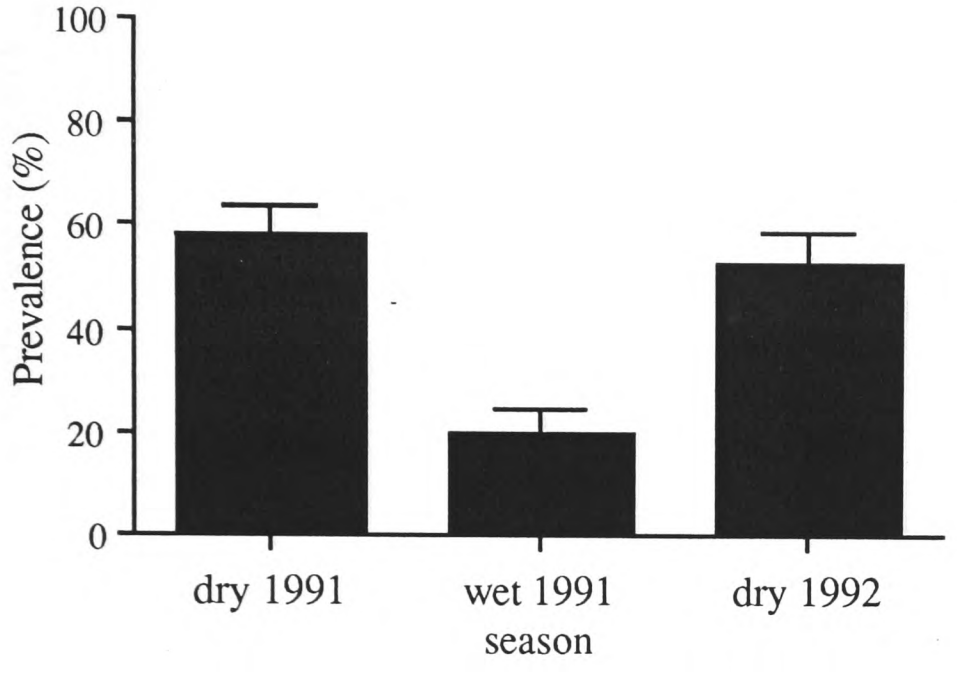


Figure 6.3. Seasonal variation of *Streptopharagus* sp. prevalence.
Prevalence of infection is plotted for each season, in graph a) for the whole population and in graph b) for each troop separately. Error bars indicate one standard error.

a)



b)

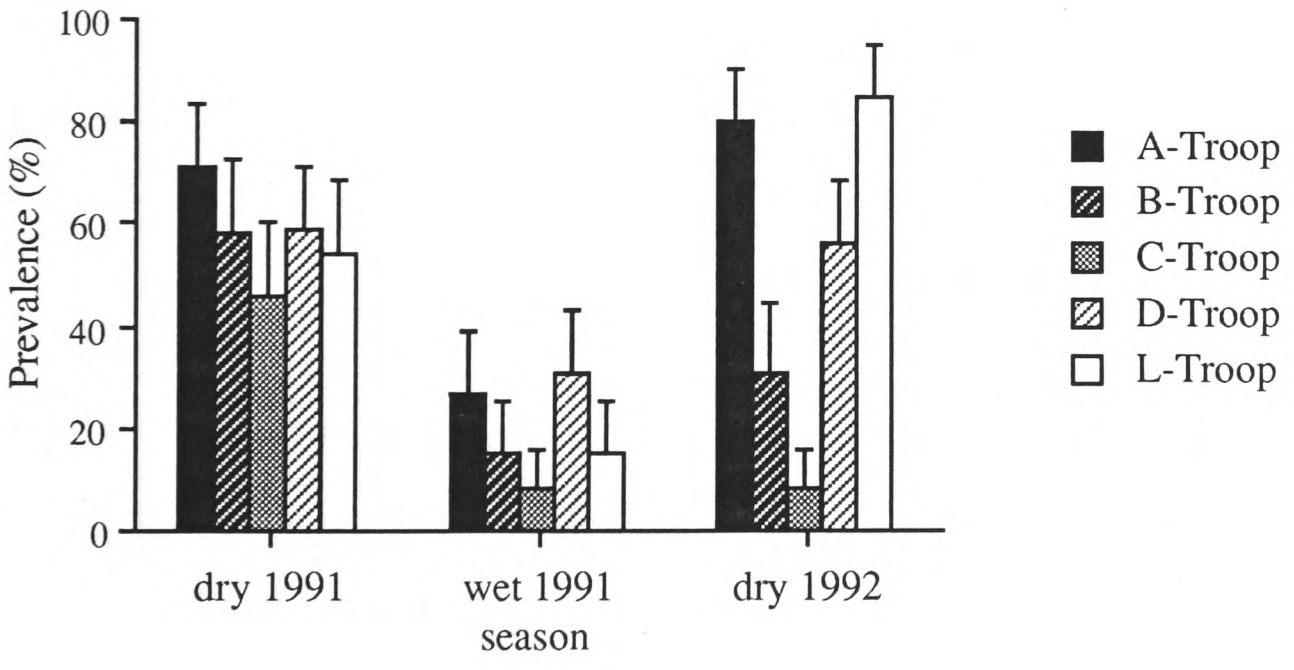
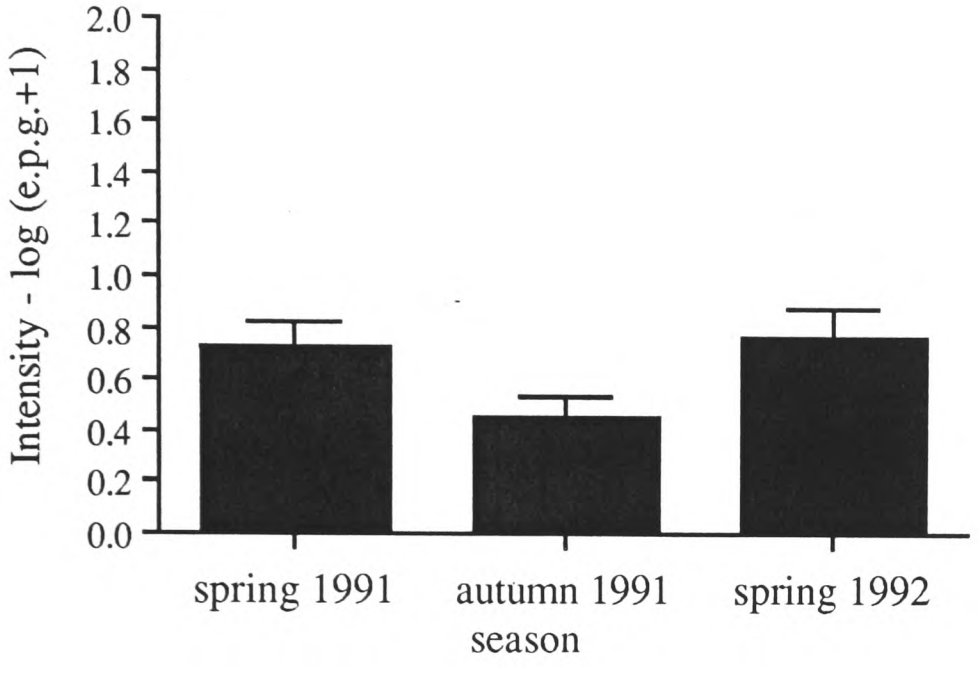


Figure 6.4. Seasonal variation of *Balantidium coli* prevalence.
Prevalence of infection is plotted for each season, in graph a) for the whole population and in graph b) for each troop separately. Error bars indicate one standard error.

a)



b)

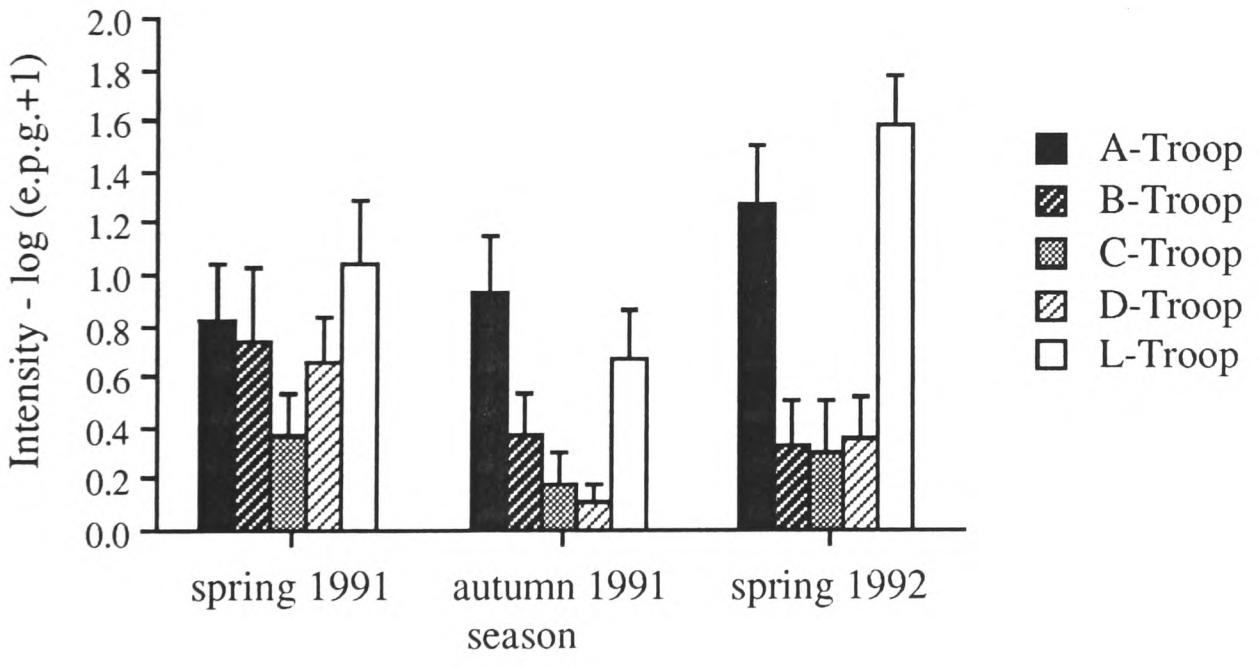


Figure 6.5. Seasonal variation of hookworm intensity.
Intensity of infection is plotted for each season, in graph a) for the whole population and in graph b) for each troop separately. Error bars indicate one standard error.

Table 6.4. Variation in intensity and severity across three seasons. Data were analysed using GLM.

	Intensity		Severity	
Parasite	F - value	p- value	F- value	p-value
total helminth load	$F_{2,201} = 4.17$	0.017 *	$F_{2,196} = 3.08$	0.048 *
Hookworm	$F_{2,200} = 3.82$	0.024*	$F_{2,81} = 4.33$	0.016 *
Strongyloides sp.	$F_{2,201} = 1.60$	0.21	$F_{2,110} = 3.04$	0.052
Trichuris sp.	$F_{2,199} = 0.08$	0.92	$F_{2,160} = 8.16$	<0.001 ***
Physaloptera sp.	$F_{2,201} = 7.33$	0.001***	$F_{2,107} = 1.82$	0.168
Streptopharagus sp.	$F_{2,201} = 10.80$	<0.001***	$F_{2,89} = 3.06$	0.052
Schistosoma mansoni	$F_{2,200} = 0.26$	0.770	$F_{2,37} = 1.71$	0.194
Unidentified trematode	$F_{2,200} = 1.92$	0.15	$F_{2,14} = 0.27$	0.77

For intensity n = 210 for all taxa but *Trichuris* sp. (n = 207) and hookworm (n = 209)

Table 6.5. Variation in intensity and severity between two dry seasons.

	Intensity		Severity	
Parasite	F - value	p- value	F- value	p-value
total helminth load	$F_{1,132} = 0.17$	0.68	$F_{1,127} = 0.74$	0.39
Hookworm	$F_{1,132} = 0.07$	0.79	$F_{1,58} = 1.24$	0.27
Strongyloides sp.	$F_{1,132} = 1.94$	0.17	$F_{1,103} = 1.22$	0.27
Trichuris sp.	$F_{1,130} = 0.18$	0.68	$F_{1,103} = 15.75$	<0.001***
Physaloptera sp.	$F_{1,132} = 0.42$	0.52	$F_{1,59} = 0.68$	0.44
Streptopharagus sp.	$F_{1,132} = 1.71$	0.19	$F_{1,46} = 1.08$	0.31
Schistosoma mansoni	$F_{1,132} = 0.01$	0.93	$F_{1,25} = 1.53$	0.23
Unidentified trematode	$F_{1,131} = 0.00$	0.961	$F_{1,6} = 1.07$	0.34

x too few data

Table 6.6. χ^2-test (with continuity correction) for correlations between parasite infection and rank.

	all		male		female	
	χ^2	p	χ^2	p	χ^2	p
total helminth load	0.09	0.77	0.25	0.62	0.02	0.90
Trichuris sp.	0.42	0.51	0.004	0.95	2.03	0.15

df = 1;

all n = 26, males n = 11, females n = 15

CHAPTER 7

Lion parasites

ABSTRACT

Fifteen morphologically different parasites were found in the lion faeces, 11 different helminth taxa: hookworm, *Toxocara*, *Spirometra*, Taeniidae, *Physaloptera*, a spiruid parasite, a paramphistome-like parasite, a trichostrongyloid parasite, *Trichuris* and 2 types of larvae (*Ollulanus tricuspis* and *Aelurostrongylus*) and 4 protozoan taxa: *Giardia*, Coccidia, *Sarcocystis* and an unidentified cyst. Three of the helminth parasites are assumed to be spurious; the eggs probably belong to parasites of the prey rather than parasites of the lions, as they have not been reported in lions before. Morphology of the eggs, larvae and cysts found in the lions faeces is described together with the life cycle of the parasite taxon and previous observations in lions.

7.1 INTRODUCTION

Little information is available on the parasites of free-ranging lions. Lion parasites have been recorded from dead wild lions anecdotally (Leroux 1958; Rodgers 1974; Bwangamoi *et al.* 1990). In a few notable exceptions scientists have either looked at a particular group of parasites in lions, such as tapeworms (Dinnik and Sachs 1972) or blood parasites (Averbeck *et al.* 1990), or reported the results of veterinary observations on diseases of lions in a National Park (Young 1975), but not the prevalence of infection in a population. Knowledge of lion parasites stems mainly from zoo lions (Dittrich 1958; Mandal and Ray 1960; Bhatavdkar and Purohit 1963; Christensen 1964; Dosza 1964; Nickel 1964; Pande *et al.* 1970; Patnaik *et al.* 1971; Riemann *et al.* 1974; Tscherner 1974; Roth 1968; Freire and Massard 1979; Gaur *et al.* 1979; Arya 1980; He and Liu 1981; Prescott 1981; Ippen and Henne 1982; Sharma *et al.* 1983; Ogungbade and Ogunrinade 1984; Barutzki *et al.* 1985; Movsessian *et al.* 1987; Ghoshal *et al.* 1988; Tang *et al.* 1988; Ocholi *et al.* 1989). However, the findings in zoos do not necessarily reflect the parasitological fauna of wild lions.

Here the findings are reported of a study of lion parasites in the Serengeti and the Ngorongoro Crater in Tanzania. Between Spring 1991 and Spring 1993, 168 faecal samples from 112 lions (*Panthera leo*) were collected and analysed for the presence of parasite eggs. Fifteen morphologically different parasites occurred in the faeces as indicated by eggs, larvae and cysts. A brief description of the egg, larvae and cyst morphology and the life cycles of the parasites will be presented. In several cases only the genus will be reported if the identification is possible only to that taxonomic level. It is useful to distinguish parasites according to mode of transmission; first the parasites with direct life cycles are described, followed by the parasites with indirect life cycles, and finally spurious parasites which were found in the lion faeces (these on the basis of previous findings and descriptions of the parasites of the lions' prey, are presumed not to be genuine lion parasites but parasites of their prey). A summary of the lion parasites and their life cycles is presented in Table 7.1 following the taxonomies of Cheng (1986) and Kreier and Baker (1987). As a reference list for lion parasites, the "Checklist of Helminth Parasites of African Mammals" by Round (1968), "A Checklist of the Helminth Parasites of the Larger Domestic and Wild Mammals of Zimbabwe" by Jooste (1990) and "A Checklist of Helminth Parasites of Animals in Tanzania" by Bwangamoi (1970) were consulted.

7.2 PARASITES OF LIONS

Measurements of parasite eggs are presented in Table 7.2.

Hookworm

Hookworm eggs were characterized by rounded poles, barrel shaped side-walls and a thin and smooth shell. The eggs contained several blastomeres.

Hookworm (order: Strongylida; class: Secernentea) species previously reported from lions are *Ancylostoma paraduodenale* and *Ancylostoma tubaeformae* (Round 1968).

Hookworms have a direct life cycle. Although penetration of the skin is the normal infective route of hookworms, oral and transmammary transmission have also been reported for some *Ancylostoma* (Soulsby 1982; Schmidt and Roberts 1989). Such detailed information is not available for the hookworms of lions. *Ancylostoma* also may be transmitted by hosts in which the parasite does not undergo any development (paratenic hosts) (Schmidt and Roberts 1989).

Toxocara sp.

Medium sized eggs with a round or oval shape were found in the lion faeces. A thick, rough and pitted shell contained a dark-brown granular mass which filled the whole egg. The eggs fitted the description of *Toxocara* (order: Ascaridida; class: Secernentea) eggs.

Both *Toxocara canis* and *Toxocara cati* have been reported from lions (Round 1968; Gaur *et al.* 1979). The eggs were of a size corresponding to *Toxocara cati.*

Infection occurs by ingestion of eggs. Transmission also can take place transmammarily. Paratenic hosts also play an important role (Soulsby 1982), with lions possibly acquiring the infection from ruminants or rodents (Sprent 1956).

Giardia sp.

Giardia sp. (order: Diplomonadida; class : Zoomastigophorea) cysts were found in the lion faeces. Cysts were pear-shaped. They had a comma-shaped body, quite often looking like a smiling face, a thin wall and contained two nuclei.

Giardia has been observed once before in lions (G. Averbeck, University of Minnesota, personal communication).

Infection is passed on by ingestion of cysts, for instance through contaminated water (Soulsby 1982).

Coccidia

Coccidia (order: Eucoccidiida; class: Sporozoea) oocysts which had an ovoid shape with a smooth double layered wall were found (Plate 7.1 a and b). They were in most cases not sporulated and only occasionally did they contain two sporocysts. Polar granules could be observed as well. Coccidia reported from lion so far are *Isospora felis* (Wenyon 1923; Soulsby 1982), *Isospora leonina* (Mandal and Ray 1960), *Isospora* sp. (Pande *et al.* 1970; Peters *et al.* 1977) and *Eimeria felina* (Pellerdy 1974). The sizes measured here correspond with previously measured sizes for the three *Isospora*, therefore it could have been any of the *Isospora* or a mixed infection.

Coccidia are transmitted by ingestion of an infective oocyst or ingestion of a cyst in a paratenic host (Soulsby 1982). In most samples levels of infection with coccidia seemed to be relatively low as only few oocyst per slide could be discovered.

Helminth larvae

Two different helminth larvae (order: Strongylida; class: Secernentea) were observed in the lion faeces. They could be distinguished by tail morphology. One showed from three to five small cusps at the tail, whereas the other one had one big spike surrounded by smaller spikes, one on each side. However, most of the time the tail was not clearly visible because the larvae lay coiled, making it impossible to say to which genus the larvae belonged to. Therefore they were grouped as "helminth larvae".

The helminth larvae so far reported from lions and whose tails correspond with these observations are *Ollulanus tricuspis* (Chauvier and Chabaud 1964; Hasslinger *et al.* 1982; Soulsby 1982) for the larva described first and *Aelurostrongylus* spp. (G. Averbeck, personal communication) for the second larva.

Ollulanus tricuspis has a direct life cycle. The worm is viviparous and the larvae develop in the uterus of the female to the third larval stage. A host acquires the parasite when eating the vomit of an infected animal. The life-cycle may also be completed endogenously. In cats faecal examination may not indicate the whole extent of infection and diagnosing infection by analysing the vomit has been shown to be more effective

(Hasslinger *et al.* 1982; Soulsby 1982). For this reason, infection of lions may be underestimated by stool examinations. *Aelurostrongylus* has an indirect life cycle. Intermediate hosts are snails and slugs. Infection can also be passed on by paratenic hosts, such as rodents, frogs, lizards and birds.

Spirometra spp.

Both *Spirometra* (order: Pseudophyllidea; class: Eucestoda) eggs and proglottids were found in the lion faeces. Eggs were roughly oval shaped and slightly pointed at one end. They had an operculum on one side. The egg shell was fairly thin and had a pinkish colour under the microscope. No worms could be detected inside the eggs. They were filled with cells (Plate 7.2. a and b). Identification of this parasite was made on the basis of egg morphology. Eggs appeared identical to samples of eggs from a mature, gravid and identified *Spirometra pretoriensis*. This observation was supported by the identification of the proglottids as belonging to *Spirometra* sp. (A. Jones of the International Institute of Parasitology, St. Alban's, personal communication).

Spirometra was the parasite most frequently found in the lion faeces and with the highest numbers of eggs per gram of faeces. In a previous study Dinnik and Sachs (1969) observed infection of lions and hyaenas with *Spirometra*, sometimes to a considerable extent. Nelson *et al.* (1965), on the other hand, discovered heavy infections of *Spirometra* in hyaenas (14 out of 22), but none in the 4 lions examined. A high number of *Spirometra* eggs was also detected in faecal samples from three lions from the Gir Forest in India (personal observation). According to Round (1968), the species in lion is *Spirometra theileri* (previously *Diphyllobothrium theileri* and mentioned under that name in Round's list). The species parasitic in hyaenas is generally accepted to be *S. pretoriensis*. However, the morphology of one fragment of *Spirometra* found in the faeces was consistent with *Spirometra pretoriensis* but not *S. theileri* (A. Jones, personal communication). This suggests that both species may occur in lions. A third species, *Spirometra okumurai*, was also reported from a lion in the Selous Game Reserve in southern Tanzania (Rodgers 1974). It has been argued that identification of *Spirometra* species only according to morphological characters is not very reliable (Opuni and Muller 1974). *Spirometra* was first mentioned under the name *Lüheella* and *Diphyllobothrium* by Jean G. Baer (1925; 1926). There has been some confusion over the years in the use of its different names and in the description of this parasite. However, the use of *Spirometra* as the correct generic name has been proposed by Schmidt (1974).

Not much is known about the life cycles of *Spirometra theileri* and *Spirometra pretoriensis*. They have an indirect life-cycle involving two intermediate hosts. Baer

(1971) reported that upon hatching from eggs in water the coracidia infect cyclops. The second intermediate host which becomes infected with the larval stage (procercoid) is a mammal. It acquires the infection by drinking water contaminated with infected cyclops. Amphibians and reptiles seem to play no role in the transmission cycle (Schmid and Watschinger 1972; Opuni and Muller 1974) as in other species of the genus *Spirometra*. Plerocercoids (spargana) of *Spirometra* have been recovered from big game animals like antelope, buffalo, zebra, wildebeest and wart hogs (Sachs and Sachs 1968; Dinnik and Sachs 1969) in Tanzania and Kenya. The final host, a carnivore, picks up the parasite when feeding on these mammals. Man can act as an intermediate host as well (Nelson *et al.* 1965; Khalil 1991). During laboratory studies of *Spirometra theileri* a cyclic fluctuation in daily egg production was observed (Opuni and Muller 1974). This was attributed to the occurrence of a process of apolysis when a chain of proglottids, producing a large number of eggs, break free and disintegrate in the gut.

Taeniidae

Taeniid eggs were identified from the lion faeces (Thienpont *et al.* 1979). The eggs were a round to ellipsoid and had a thick smooth shell with a radially striated embryophore (Plate 7.3). Their colour varied from yellow to brown.

Because taeniid eggs (order: Cyclophyllidea; class: Eucestoda) are indistinguishable from each other, they could belong to any of the following species: *Taenia regis, Taenia simba, Taenia gonyamai* and *Echinococcus granulosus* which have been found in lions in East-Africa (Dinnik and Sachs 1972; Young 1975). Apart from these species, *Taenia hydatigena, Taenia pisiformes, Taenia taeniaeformis, Taenia jaipurensis* (Sharma *et al.* 1983) and *Taenia* sp. have been reported from lions elsewhere (Abuladze 1964). Cestode fragments from the lion droppings were identified as *Taenia* spp. and most likely *Taenia regis* (A. Jones, personal communication).

The taxonomic status of the *Echinococcus* found in lions is not completely resolved yet. Felids are not normally the definitive host of *Echinococcus*. However, adult *Echinococcus* have been reported from African lions (Dinnik and Sachs 1972; Rodgers 1974; Macpherson and Craig 1991) and other African wild cats (Macpherson and Craig 1991). The species has been reported as *Echinococcus granulosus* (Nelson and Rausch 1963; Nelson *et al.* 1965), *Echinococcus granulosus felidis* (subspecies) (Dinnik and Sachs 1972) or *Echinococcus felidis* (Macpherson 1986).

Taeniids have an indirect life-cycle with only one intermediate host, a mammal. Intermediate hosts for lion taeniids are most of the lions' prey such as antelopes, buffalo, wildebeest and other herbivores (Nelson *et al.* 1965; Dinnik and Sachs 1969). Among the

more than 18 species of wild animals that have been reported as intermediate hosts of *Echinococcus* were the most common prey species of lions (Sachs and Sachs 1968; Dinnik and Sachs 1969; Sinclair 1977; Macpherson and Craig 1991).

Because *Echinococcus* eggs are indistinguishable from *Taenia* eggs, immunodiagnostic tests were used to detect adult *Echinococcus* antigen in lion faecal samples (Allan *et al.* 1992). Tests were performed by P.S. Craig on faecal samples, which had been preserved in 5% formalin. They showed a low infection of 4 % (3 positive in a sample of 75, of which 33 were tapeworm positive) with *Echinococcus*. The test has a sensitivity of 80% (P. S. Craig, University of Salford, personal communication). The prevalence of infection is much lower than has been reported from dogs in East-Africa (Macpherson and Craig 1991).

Physaloptera sp.

Eggs which were identified as *Physaloptera* sp. (order: Spirurida; class: Nematoda) were found in the stool of one animal only. They were round to oval shaped eggs with a thick wall. Around the wall was a thick rough transparent outer layer which looks almost as if it was just detritus sticking to the egg. A smooth coiled larva lay inside the egg. These eggs may look similar to those of *Ascaris*.

So far only one species has been reported for lions: *Physaloptera praeputialis* (Leroux 1958; Round 1968; Bwangamoi 1970).

Physaloptera has an indirect life cycle and insects are assumed to be the intermediate hosts. However, it has been argued that some *Physaloptera* species may also be transmitted directly (Krupp 1962). For *Physaloptera praeputialis,* Orthoptera and Coleoptera have been suggested as intermediate hosts (Soulsby 1982). The life cycle can also entail paratenic (transport) hosts. Snakes have been found to be naturally infected, and frogs and mice can be experimentally infected (Soulsby 1982).

Spiruid egg

Unidentified spiruid eggs were detected in the lion faeces (Plate 7.4). Parasites of the super-family Spiruiodea usually have an indirect life-cycle (Soulsby 1982).

Sarcocystis spp.

Another coccidian parasite was recorded, whose sporocysts differed from the rest of the other coccidian oocysts found (Plate 7.5.). The cysts were ovoid and held four banana-shaped sporozoites with granules present. The wall was double-layered, smooth and about 0.5 - 0.7µm thick. The inner layer seemed colourless while the outer layer was light brown.

Because of its size and similarity to cysts observed in felines the cyst probably is *Sarcocystis* spp. (order: Eucoccidiida; class: Sporozoea) (Levine and Ivens 1981; Levine 1984; Levine 1985; Levine 1986; Kreier and Baker 1987). Two sporocysts were never seen together in one oocyst; there was always just one sporocyst containing the sporozoites, a characteristic of *Sarcocystis* spp. However, *Sarcocystis* sporocysts generally break out of the oocysts in the predator's intestine so that sporulated sporocysts are observed in the faeces (Levine 1985). It is difficult to decide unambiguously the taxonomic status of the observed parasite on the basis of sporocyst morphology alone. Only observation of the life cycle would allow for accurate identification. Possible sarcosporidiosis, detected by cysts in faecal samples, has been mentioned in lions (G. Averbeck, personal communication; cited by Young 1975), but without description of the parasite. *Sarcocystis* has also been observed during lion dissections (Bhatavdkar and Purohit 1963; Bwangamoi *et al.* 1990).

Sarcocystis is heteroxenous. The sexual stage occurs in a predator and the asexual in intermediate hosts, the prey animals. The intermediate host becomes infected by ingestion of sporocysts. The definitive host acquires the infection when eating prey and at the same time ingesting mature sarcocysts (Levine 1985).

Unidentified cysts

Unidentified cysts were found in 13% of the lions. The cysts were round with a light brown colour and unsporulated.

7.3 SPURIOUS PARASITES

Several of the parasite eggs observed in the lion faeces are believed to belong to species which are not genuine lion parasites, but parasites of lion prey. These parasites have not been recorded from lions previously, but they are known to infect lion prey to a considerable extent.

Paramphistome-like egg

Fluke-like eggs were found in the faeces. The eggs were oval with one end more pointed, transparent with a thin shell, and containing visible clusters of yolk or cell material (Plate 7.6) An operculum was visible at one end. Eggs looked similar to the eggs reported as liver and stomach-fluke eggs (Dinnik 1958; Ogambo-Ongoma and Goodman 1972). Liver and stomach flukes are both parasites of lion's prey (Sachs and Sachs 1968; Sinclair 1977). Although sizes for both parasites fall in the range of the eggs measured above, it seems more likely that the parasites were paramphistomes because of their thin colourless shell and because *Fasciola* eggs have sizes only in the upper range of the sizes measured. Furthermore, *Fasciola* is only infrequently found in herbivores of the Serengeti whereas almost all of them were found to be infected with paramphistomes (Sachs and Sachs 1968).

However, a trematode parasite which has been reported from lions, *Pharyngostomum cordatum,* may also have similar eggs. Unfortunately, no photographs of the eggs could be found and no eggs from adult worms could be obtained for comparison. Reported *Pharyngostomum cordatum* egg measurements ranged from 89 - 132 x 70 - 88µm (Dubois 1970) and 120 x 70 µm (Baer and Dubois 1951), falling in the lower ranges of the ones observed.

Thirty-four per cent of lion faeces contained these eggs. The prevalence of these eggs is considerably higher than for the other spurious parasites. On the one hand this may be because most of the prey animals are heavily infected with flukes; *Pharyngostomum cordatum* produces few eggs (Dubois 1970), therefore the eggs' prevalence may indicate that the eggs originate from the highly infected prey. On the other hand, the eggs may be found to a higher level in the lions than the other eggs of spurious parasites because these eggs are indeed those of a genuine lion parasite, *Pharyngostomum cordatum*. Another possibility is that, because eggs of *Pharyngostomum* and the eggs of flukes cannot be distinguished easily, there may be mixed infections. Eggs of this morphology were categorized as spurious parasites, because of the size of eggs and the frequent occurrence of paramphistomes in prey animals.

Trichostrongyloid egg

Trichostrongyloid eggs (order: Strongylida; class: Secernentea) were observed in the lion faeces. Different species of trichostrongyles frequently occur in the prey animals of the lions (Sachs and Sachs 1968; Bwangamoi 1970; Sinclair 1977; Horak *et al.* 1983; Jooste 1990), but have not been reported from lions.

Trichuris sp.

Some *Trichuris* (order: Trichocephalida; class: Adenophorea) eggs were detected in the faeces. So far *Trichuris* has not been reported from lions. Some of the lion's prey, such as zebras (L. Gibbons, personal communication), are heavily infected with *Trichuris* (Round 1968; Sachs and Sachs 1968; Bwangamoi 1970; Sinclair 1977; Jooste 1990). Therefore, it seems reasonable to assume that these can be considered as spurious parasites as well.

Table 7.1. Lion parasite taxa and their life cycles.
Spurious parasites are not included. "( )" indicate that identity of parasite and route of transmission are not known, but are suggested. "[ ]" indicate the parasite taxa which are categorized under one name.

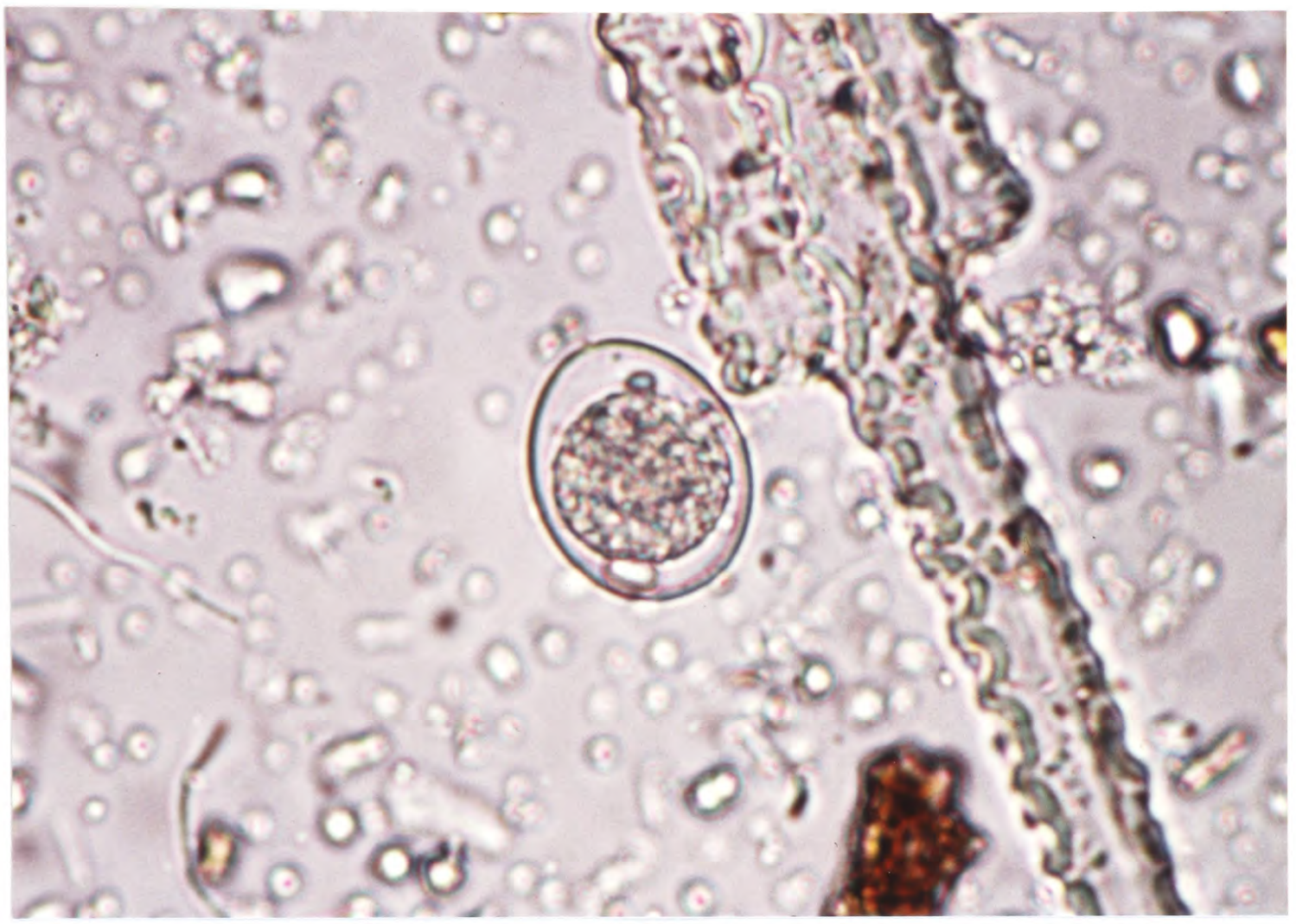
Parasite	Order	Class	Life Cycle	Route of Transmission
Hookworm [*Ancylostoma* spp.]	Strongylida	Secernentea	direct	penetration of host skin by larvae
Helminth Larvae [*Ollulanus tricuspis*	Strongylida	Secernentea	direct	ingestion of gastric fluids of infected animal
Aelurostrongylus spp.]			indirect	ingestion of intermediate host (snails or slugs), or paratenic host (rodents, frogs, lizards, birds)
Toxocara sp.	Ascaridida	Secernentea	direct (indirect)	ingestion of eggs (or through paratenic hosts)
Giardia sp.	Diplomonadida	Zoomastigophorea	direct	by cysts (e.g. in water)
Coccidia	Eucoccidiida	Sporozoea	direct	by cysts (e.g. in water)
Physaloptera sp.	Spirurida	Secernentea	indirect	ingestion of intermediate host Orthoptera and Coleoptera
Spiruid egg	(Spirurida)	(Secernentea)	(indirect)	(ingestion of intermediate host)
Taeniidae [*Taenia* spp. *Echinococcus* sp.]	Cyclophyllidea	Eucestoda	indirect	ingestion of intermediate host (lion's prey)
Spirometra spp.	Pseudophyllidea	Eucestoda	indirect	ingestion of 2nd intermediate host; two intermediate hosts: 1st copepod, 2nd mammal.
Sarcocystis spp.	Eucoccidiida	Sporozoea	indirect	ingestion of intermediate host (lion's prey)

7.2. Table of parasite egg and cyst measurements.
First the range is reported and afterwards the mean and standard error in brackets.

Parasite	length in µm	width in µm
Hookworm	48.2 - 78.3 (64.3 ± 0.97) n = 60	36.2 - 51.7 (41.1 ± 1.0) n = 20
Toxocara sp.	60.0 - 72.5 (64.9 ± 2.4) n = 5	60.0 n = 2
Spirometra spp.	54.7 - 76.0 (64.9 ± 1.0) n = 31	30.4 - 44.4 (37.5 ± 0.6) n = 30
Taeniidae	35.2 - 51.7 (41.1 ± 1.0) n = 17	30.0 - 42.6 (5.2 ± 0.7) n = 17
Spiruid egg	60.3 - 91.8 (84.2 ± 2.0) n = 18	36.2 - 57.8 (52.8 ± 1.4) n = 14
Paramphistome-like egg	120 - 168.7 (143.5 ± 1.0) n = 33	66.6 - 84.4 (76.6 ± 1.6) n = 7
Trichostrongyloid egg	81.9 - 96.4 (88.3 ± 1.2) n = 21	60.8 n =1
Giardia sp.	12.2 n = 3	9.1 n = 3
Coccidia	24.3 - 54.7 (33.3 ± 0.9) n = 78	21.3 - 45.6 (27.6 ± 0.6) n = 73
Sarcocystis spp.	12.2 - 13.7 (12.7 ± 0.1) n = 23	9.1 - 9.7 (9.2) n = 23
cysts	24.3 - 30.4 (25.8 ± 1.8) n = 5	

Physaloptera sp. and *Trichuris* sp. were not measured.

a)



b)

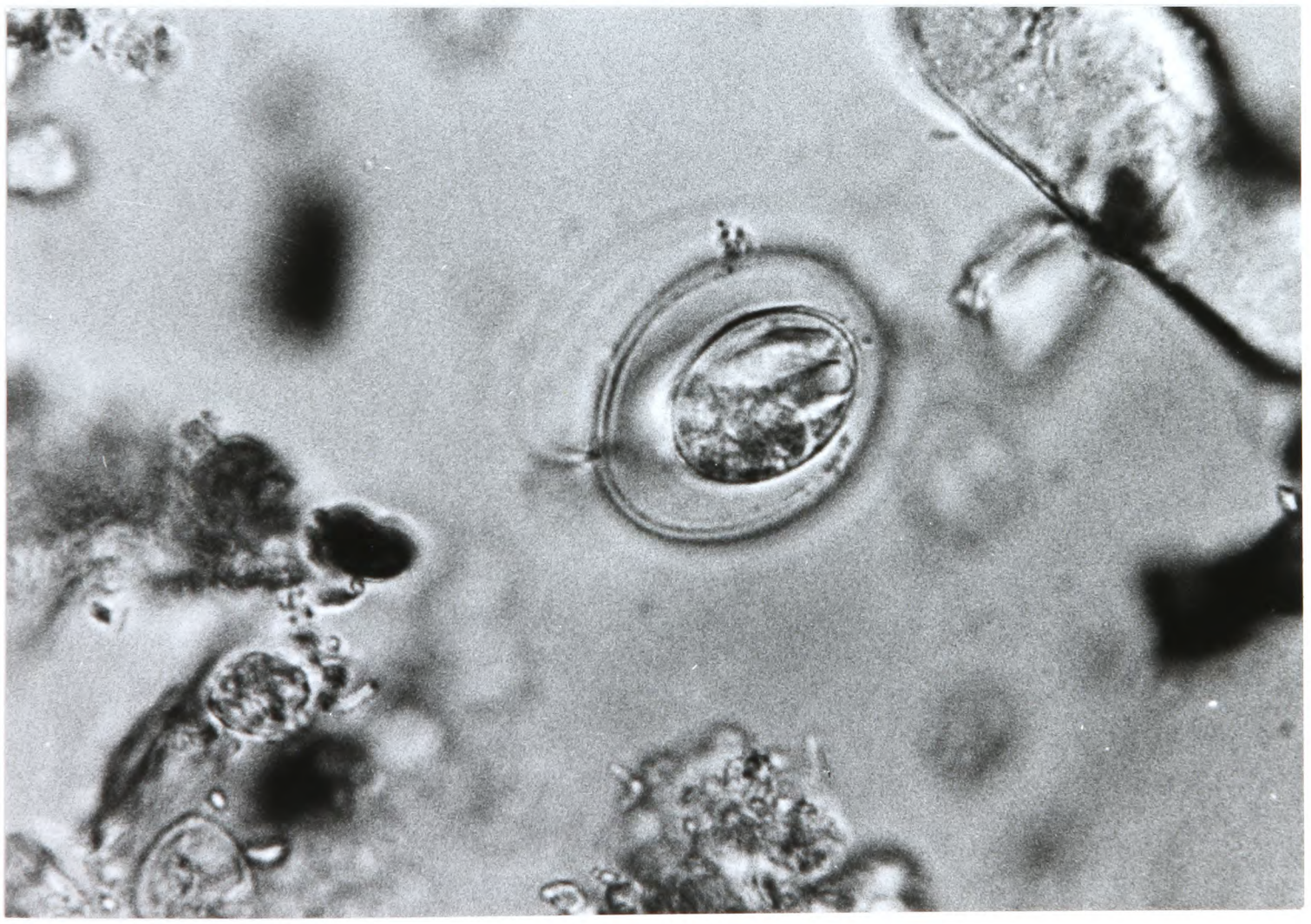


Plate 7.1. Coccidia cyst a) and b).

a)

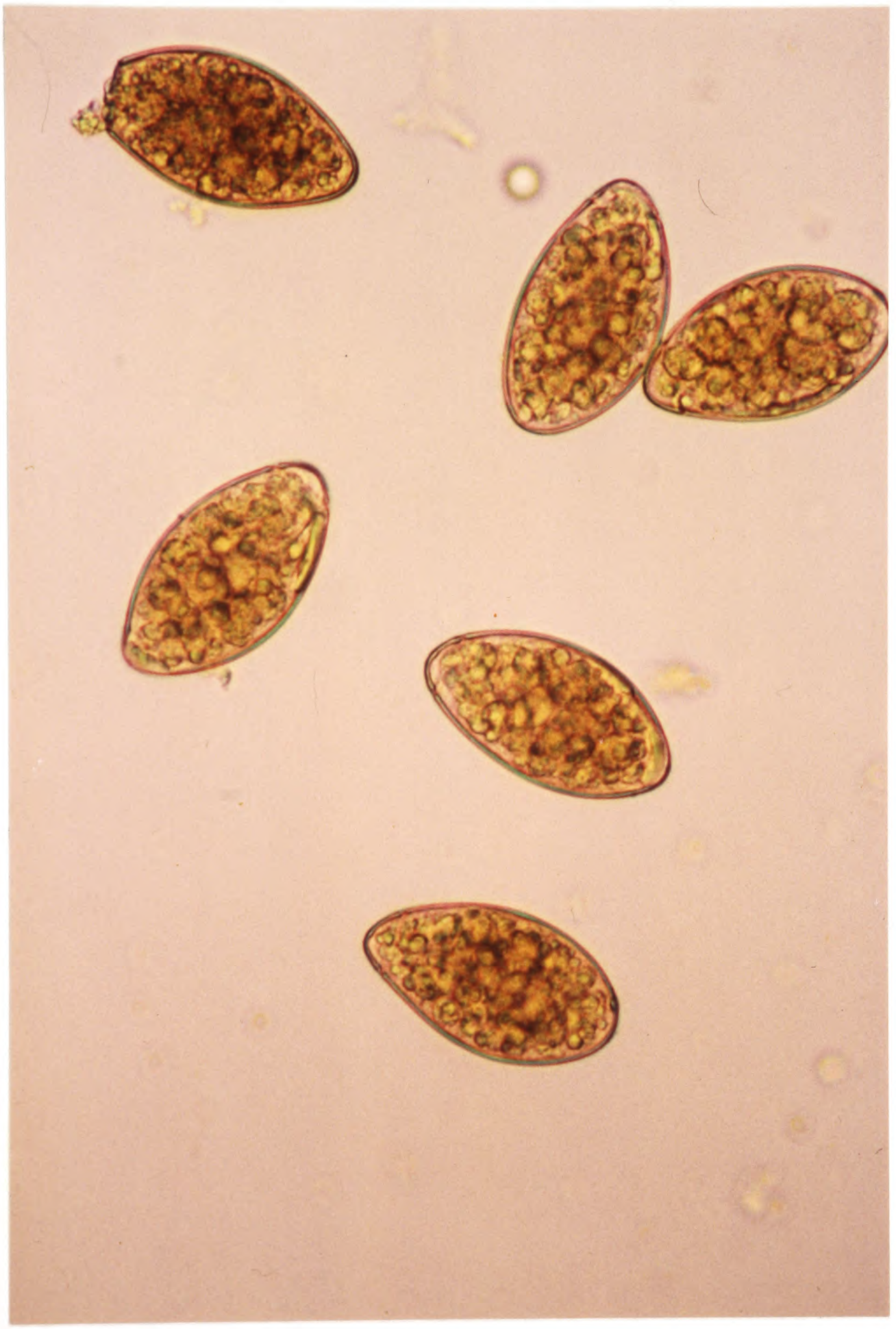
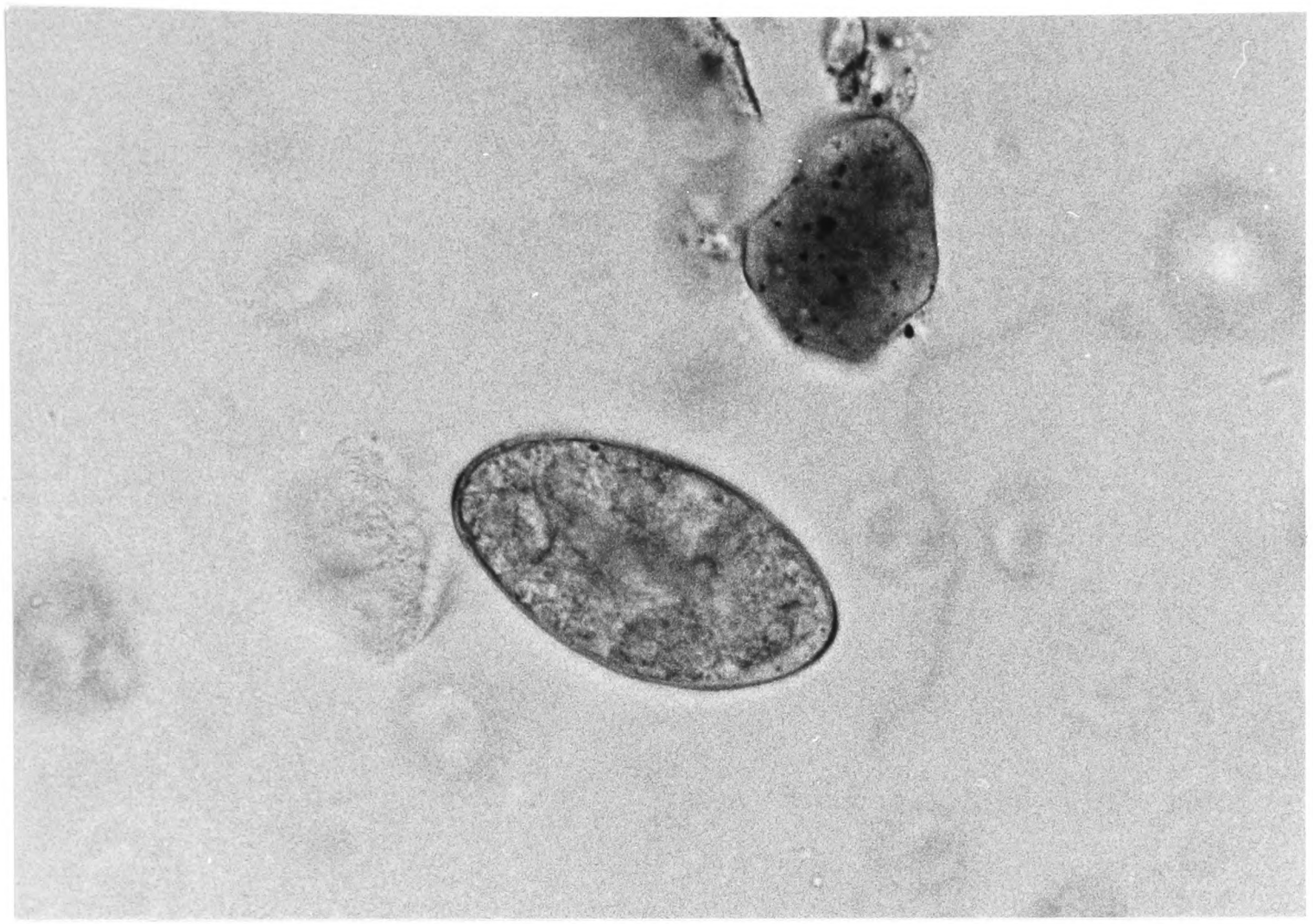


Plate 7.2. a) *Spirometra* sp. egg.

b)



c)

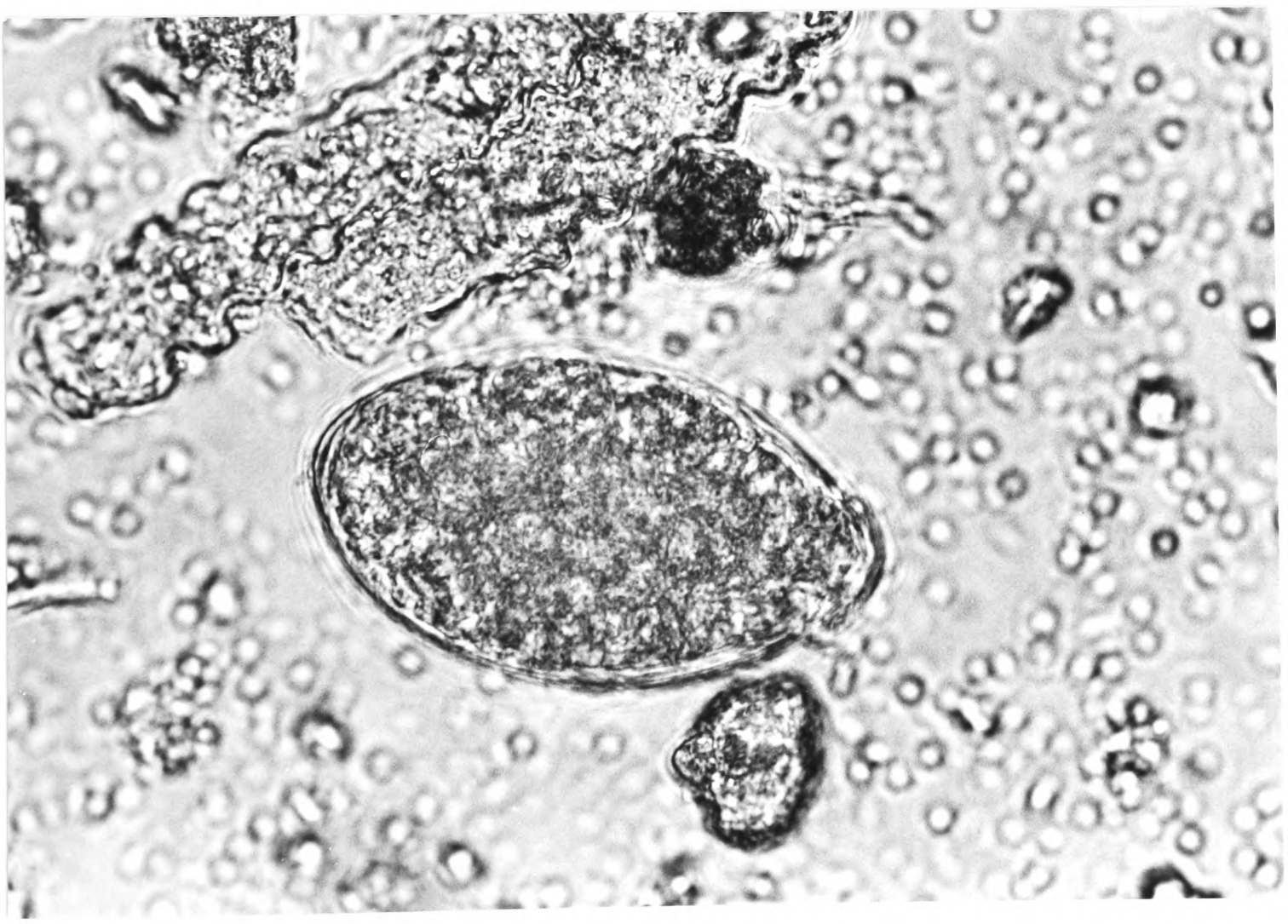


Plate 7.2. *Spirometra* spp. egg b) and c).

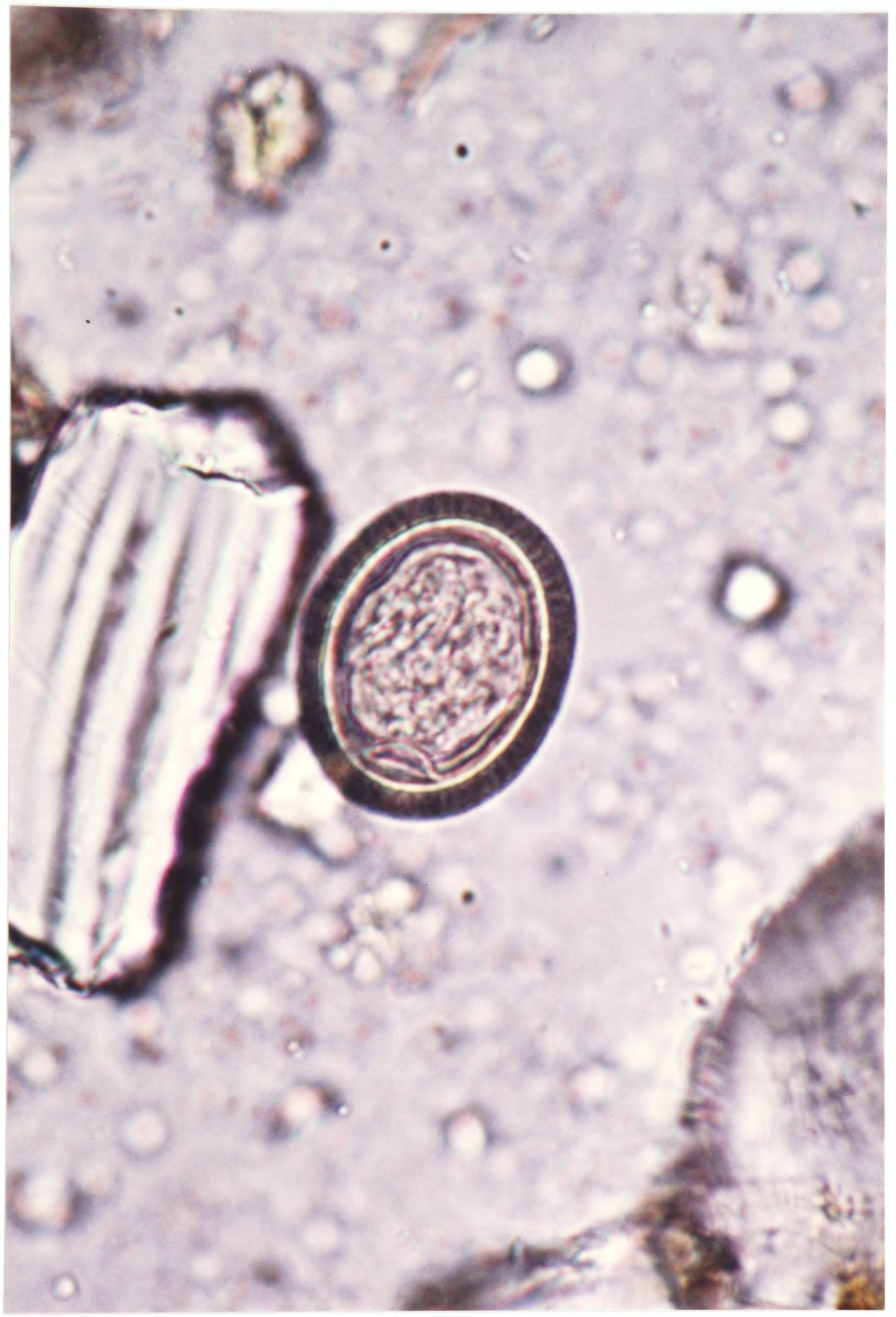


Plate 7.3. Taeniid egg.

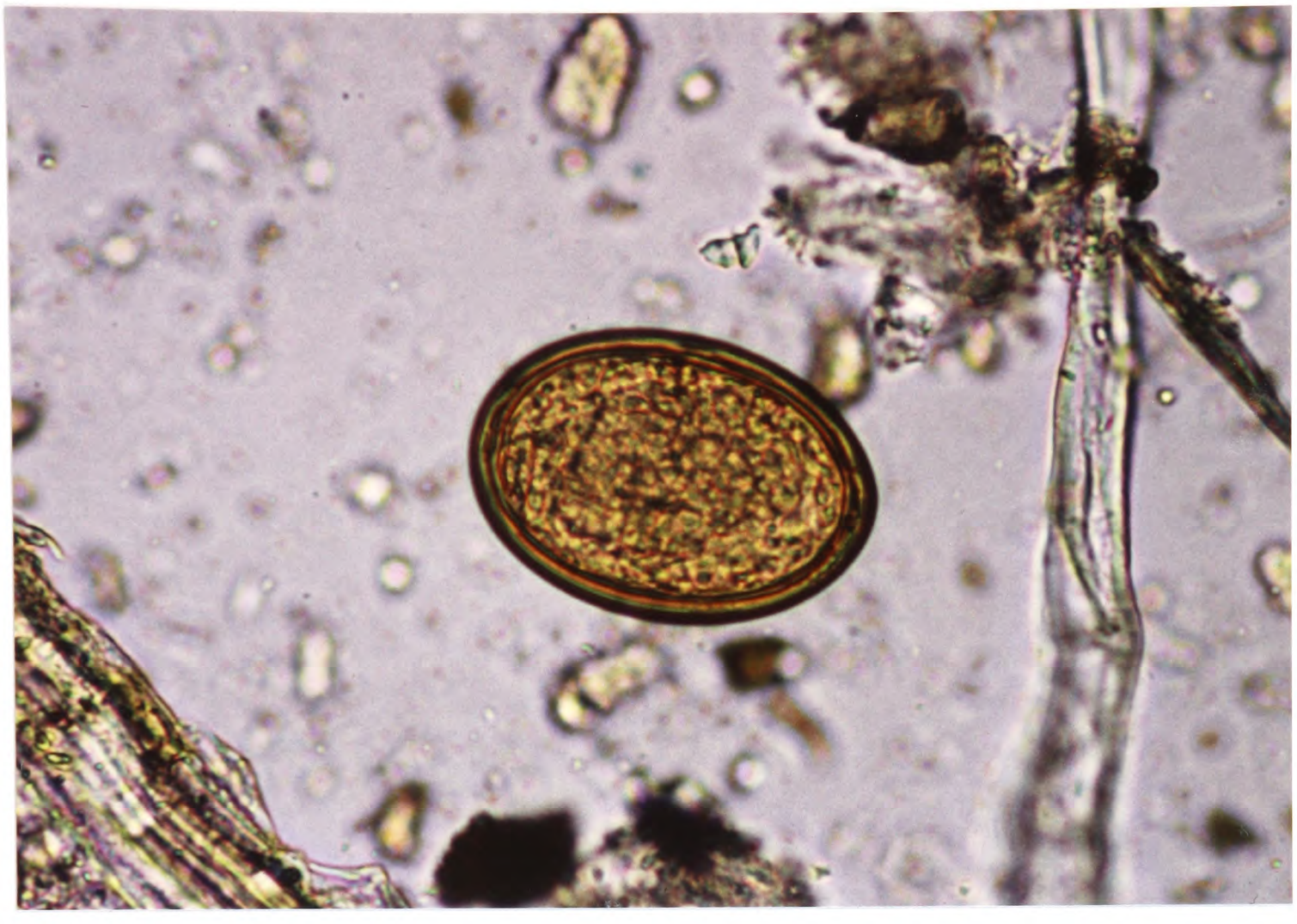


Plate 7.4. Spiruid egg.

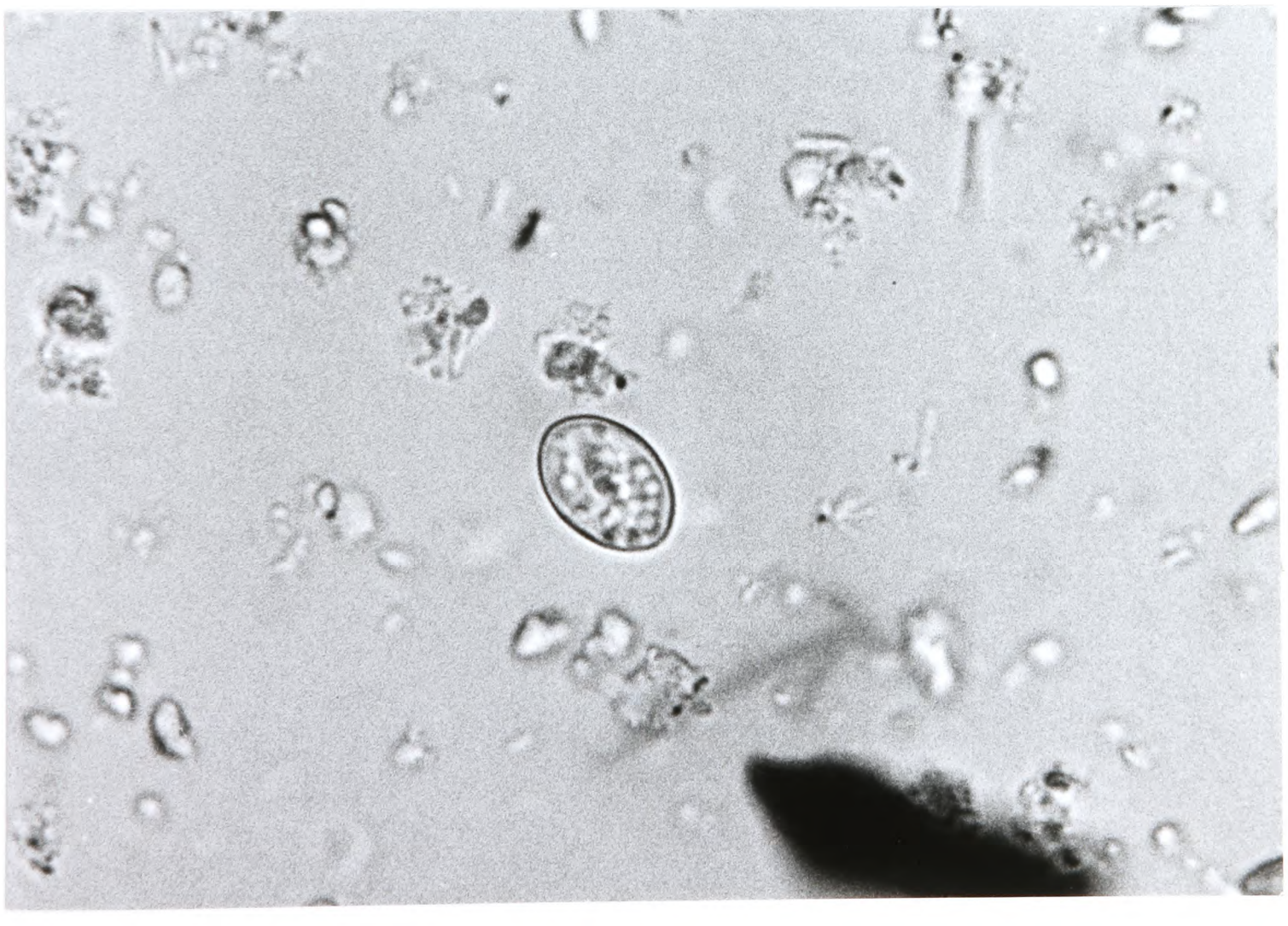


Plate 7.5. *Sarcocystis* spp. cyst.

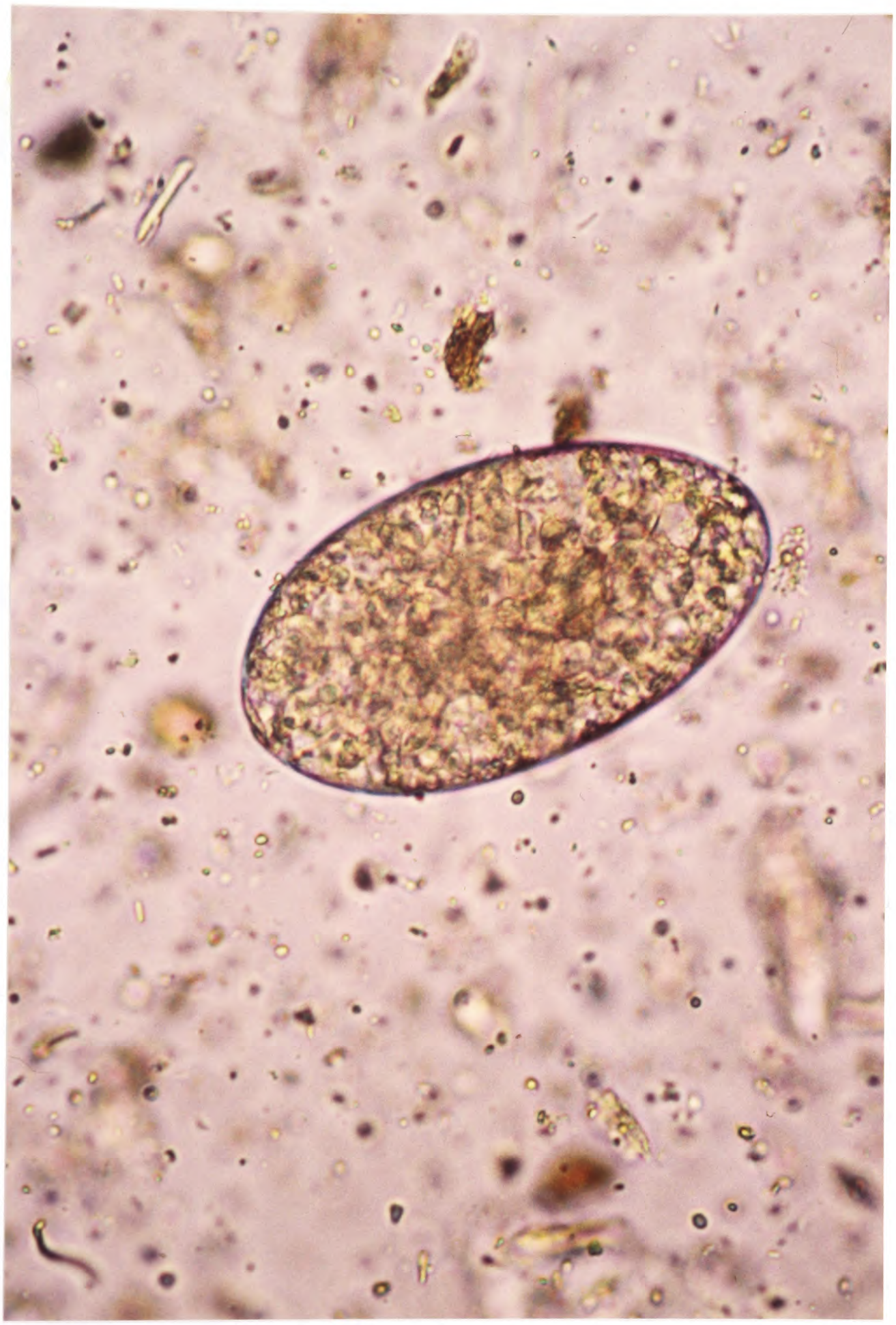


Plate 7.6. Paramphistome-like egg

Epidemiology of intestinal parasites in two populations of African lions (*Panthera leo*)

ABSTRACT

Parasite infections were studied in two populations of lions in the Serengeti and the Ngorongoro Crater in Tanzania, East Africa. The two lion populations lived in different habitats and differed genetically; lions in the Serengeti were outbred, whereas lions in the Ngorongoro Crater were inbred. Parasite infection was estimated by faecal samples collected from individually known lions between March 1991 and November 1992 and related to demographic and ecological data available for all lions. Prevalence, intensity, severity and richness were recorded. Almost all individuals were found to be parasitized. Lions were diagnosed to be infected with 11 different helminth taxa and 4 protozoan taxa. Three of the helminth parasites are believed to be spurious parasites. The distribution of all parasite taxa was aggregated. However, large differences between the two populations could not be observed; only two parasites, *Spirometra* spp. and hookworm, showed different levels of prevalence between the two populations, which could not be clearly linked to host genetics and may also be explained by, for instance, habitat differences. Under present conditions host genotype, as far as measured, appears not to be a major determinant of infection status. Apart from differences between the two populations further spatial differences were found, which may be related to environmental factors, ecology of prey or possibly group size. Only one parasite, *Spirometra* spp., showed fluctuations according to season. Temporal variation in other parasite taxa seemed not to be associated with season. Age-prevalence and age-severity profiles showed associations

between age and levels of infection. Lions acquired most parasites at an early age. However, only 2 spurious parasites exhibited a significant difference between age-classes. Sex, reproductive status and parasite-parasite interactions did not show much influence on parasite burden. The number of significant associations between parasitic infection and known factors was low and could only account for some of the observed variation. Spatial variation emerged as the strongest effect.

Plate 8.1. Lions (*Panthera leo*) in the Serengeti.

8.1 INTRODUCTION

The distribution of macroparasites among host individuals is typically overdispersed and can be influenced by several host specific and environmental factors (Crofton 1971; Anderson and Gordon 1982; Anderson and May 1991). Knowledge of these factors is important for the understanding of the ecology and evolution of host-parasite interaction and may also have practical consequences. For instance, if an individual's genotype has considerable impact on the susceptibility to disease or if certain environmental factors influence infection status, then these have to be considered in management plans for species conservation.

Few studies have investigated host-parasite systems of wild carnivores and analysed the factors influencing their parasite burden. This study examines the interactions of lions and their parasites in two populations of wild lions (*Panthera leo)* in the Serengeti and the Ngorongoro Crater in Tanzania, East Africa (Plate 8.1). Faecal samples were obtained from individual lions that were subjects of long-term demographic and ecological studies (Schaller 1972; Bertram 1978; Packer 1986; Packer *et al.* 1988; Packer *et al.* 1991; Hanby *et al.* in press). This allowed parasite infection to be related to known characteristics of individual animals. The two host populations were chosen because they differed in genetic structure and habitat. The Serengeti lions are part of a large outbred population, whereas the Ngorongoro Crater population is small, isolated and inbred (O'Brien *et al.* 1987; Yuhki and O'Brien 1990). The two populations might be expected to show different levels of parasitic infection. Higher levels of genetic uniformity might reduce resistance to parasites (May 1988; O'Brien and Evermann 1988), thus a comparison of the parasitic infections of the two lion populations might indicate whether these inbred lions suffer from an increased susceptibility to parasitic infection. On the other hand, the Ngorongoro Crater has been considered as an "ecological island" (Packer *et al.* 1991) and may harbour fewer species of parasites. In addition, levels of infection of the two populations may also differ because of the ecological differences between the habitats, including differences in prey availability (Packer *et al.* 1988; Hanby *et al.* in press). Allozyme data from individual lions in the Crater and the Serengeti were used to examine whether differences in infection are linked to genotype. Variation in parasite burden may, however, also be influenced by several other factors which were investigated. Samples were collected over five seasons, which may influence parasite population dynamics (Dogiel 1964; Cheng 1986; Montgomery and Montgomery 1988; Esch *et al.* 1990; Gulland 1991; Théron *et al.* 1992), especially as seasons are related to the movement of prey, which are the intermediate hosts for several lion parasites.

Infection can vary with host age because of different exposure, innate or acquired immunity (Bundy 1988b; Anderson and May 1991). Host sex may influence intestinal parasite burden (Bundy 1988a); furthermore, female reproductive status can alter susceptibility to infection with pregnant or lactating females likely to exhibit higher parasite egg output (Jackson *et al.* 1988; Behnke 1990). Differences in host group size may account for variations in infection depending on the type of parasite involved (Freeland 1976; Møller *et al.* 1993; Coté and Poulin 1994). It was also considered whether parasite-parasite interactions can have an influence on the distribution of parasites (Price 1980; Esch *et al.* 1990; Sousa 1994).

8.2 MATERIALS AND METHODS

Study area

The study deals with the intestinal parasites of two lion populations located in a 2000 km^2 area in the Serengeti National Park (coordinates of Seronera, the research station in the centre of the Serengeti, 34°50' E, 02°30' S) and the 250 km^2 floor of the nearby Ngorongoro Crater (35°35' E, 03°10' S), part of the Ngorongoro Conservation area, 120 km southeast of Seronera in Tanzania, East Africa (Figure 8.1.a and b).

The Serengeti ecosystem is dominated by two main vegetation types, grassy plains with scattered rocky outcrops (kopjes) and open woodlands. The Ngorongoro Crater is an extinct volcanic caldera with a primarily open grassland floor that includes a number of swamps and marshes (Schaller 1972; Sinclair 1979; Packer *et al.* 1988). The Serengeti ecosystem is characterized by the migration of the herbivores according to season, whereas in the Crater there is only minor seasonal variation in herbivore distribution and prey is consistently abundant (Kruuk 1972; Packer *et al.* 1988; Hanby *et al.* in press). Here, the resident biomass of the lions' preferred prey species is the highest in Africa (Packer *et al.* 1988). Crater lions have a higher food intake than the lions of the Serengeti plains (Hanby *et al.* in press) which are subjected to greater nutritional stress during certain times of the year. The lions in the Serengeti woodlands have better access to prey than the lions in the plains during times when migratory prey is rare, but they are nevertheless subject to seasonal variation (Scheel and Packer in press). The Crater contains a smaller, but denser lion population (Packer *et al.* 1988; Hanby *et al.* in press). The average pride size is the same in both habitats (C. Packer, personal communication).

Seasons

The dry season typically starts in June and lasts until October while the rains normally begin in November and continue until May (Schaller 1972; Sinclair 1979). However, there is considerable variation in the date of onset of the wet season. Serengeti rainfall data from 1990 until 1993 were used to classify each season with precision (Figure 8.2.). Rainfall was read regularly each month at gauges in the Serengeti woodlands near Seronera. Comparison with readings from previous years at several gauges in the Serengeti, suggests that these rain gauge readings provide a good indication of the overall rainfall patterns (C. Packer, personal observation) as plains rainfall is highly correlated with woodland rainfall (Packer *et al.* 1988). Rainfall patterns in the Ngorongoro Crater are similar to the Serengeti (Packer *et al.* 1988) and data from the Serengeti were used for the Crater as well.

Seasons in the sampling period were classified as follows: the first wet season from August to June 1990/91, the first dry season July until September 1991, the second wet October 1991 to May 1992, second dry June 1992 to September 1992 and the third wet October 1992 until April 1993 (see Figure 8.2.).

The lion population

Lions prides normally consist of 2 - 9 related females (range 1 - 18), their dependent offspring and a coalition of 2 - 6 adult males (range 1 - 9) (Packer *et al.* 1991). The resident males are either closely related or unrelated to each other (Packer and Pusey 1982; Packer *et al.* 1991). Overall pride size averages 15 members (range 1 - 37) (Schaller 1972). Prides are "fission-fusion" social units. Pride members do not remain in constant association, but form subgroups throughout the pride-range and spend some time alone (Pusey and Packer 1987; Cairns 1990; Packer *et al.* 1990). A small number of females live alone in stable ranges and some males are nomadic and live outside prides.

Lions are carnivores. They not only kill their own prey but also scavenge from other predators and eat animals that have died from disease and other causes (Schaller 1972). They kill almost any animal they can catch, but in most areas prey from a few species predominates. The Serengeti lions feed mainly on wildebeest (*Connochaetes taurinus*), zebra (*Equus burchelli*), wart-hog (*Phacochoerus aethiopicus*), buffalo (*Syncercus caffer*), Thomson's gazelle (*Gazella thomsoni*) and topi (*Damaliscus korrigum*) (Schaller 1972; Scheel 1993). Season and location, however, have a significant influence on prey choice (Schaller 1972; Scheel 1993; Scheel and Packer, in press).

Individual lions were identified by their earmarks and whisker spots (Pennycuick and Rudnai 1970; Packer 1992; Packer and Pusey 1993). During this study, 26 prides of lions were monitored, 18 prides in the Serengeti (containing 190 lions) and 8 prides in the Ngorongoro Crater (containing about 70 lions).

Although the genotype is not available for each individual in this study, the two lion populations are known to differ genetically. The Serengeti population is more outbred than in the Crater. The Crater lions were reduced from 72 to 10 - 15 individuals following an outbreak of biting flies (*Stomyxys calcitrans*) in 1962 (Packer *et al.* 1991). The present Crater population is descended from only 10 - 15 founders and most individuals can be traced back to 4 female ancestors (Packer 1992). There has been no immigration into the Crater since 1969 (Packer *et al.* 1991).

In contrast male lions disperse throughout the Serengeti ecosystem and mating partners are typically unrelated (Packer *et al.* 1991). Empirical data reveal that, in comparison to the Serengeti, lions in the Crater show a lower degree of genetic diversity in isozymes and a striking lack of diversity in restriction fragments of the major histocompatibility complex (MHC) class I gene (Yuhki and O'Brien 1990), which appears to play a role in the immune response to parasitic infections.

Genetic data were available for a subset of the sampled lions (n = 31). Genotypes at six or seven polymorphic allozyme loci were used in the analysis and an overall score for heterozygosity was calculated for each individual (see O'Brien *et al.* 1987; Packer *et al.* 1991).

Collection of faecal samples

Faecal samples from individually identified lions were collected opportunistically between March 1991 and November 1992 as described in chapter 2. More samples were collected in the Serengeti (n = 129) than in the Crater (n = 39) and more in the wet season (n = 114) than in the dry season (n = 53; date unknown n = 1). Overall, 168 samples were examined from 112 individuals (Table 8.1.). Parasite eggs were distinguished by morphology and size, and prevalence, intensity and severity of infection was recorded. Only prevalence is reported for protozoans. Not all samples were examined for all parasites.

Parasite species

Eleven different helminth species, including 9 different types of eggs and 2 types of larvae, as well as 4 protozoan taxa could be distinguished in the lion faeces as described in chapter 7. Three of the helminth eggs, a paramphistome-like egg, a trichostrongyloid egg and *Trichuris* sp. eggs are presumed to belong to parasites of the prey rather than the lions themselves. *Trichuris* and the trichostrongyloid egg have so far not been reported from lions and are therefore assumed to be prey parasites. Arguably, the paramphistome-like egg is also regarded as a prey parasite because of its measurements. The presence of a number of unidentified parasite eggs was recorded.

Statistical analysis

The definitions of prevalence, intensity, severity, richness and total helminth load are the same as used in chapter 4. The full list of parasite taxa used to calculate richness can be seen in Table 7.1 in chapter 7, but *Giardia* sp. and *Sarcocystis* spp. were excluded from the number of taxa in calculating richness because of their unreliable detection. *Physaloptera* sp. was excluded from analysis of prevalence, intensity and severity because it was only observed in one animal.

Variation in prevalence, intensity and severity of parasitic infection and richness was analysed using logistic and GLM employing the "Statistix" package (Analytical Software 1992; Siegel 1992). "Statview II" (Abacus Concepts 1987) and "Statview 4" (Abacus Concepts 1992). Intensity and severity data were log(x+1) transformed prior to analysis. The summed binomial distribution was calculated as described in chapter 4.

Independent variables consisted of season (either dry vs. wet, or as the five individual seasons), location (Serengeti vs. Crater), individual prides (pride of residence), pride size (measured as number total of pride members or number of adults or number of cubs in the pride), sex, age, whether females were pregnant or lactating, and level of overall heterozygosity (either scored across 6 - 7 loci together or as homozygous/ heterozygous for the individual alleles; C. Packer, personal communication). Analysis was repeated on the Serengeti subset of the data using the independent variable habitat (woodlands vs. plains).

The analysis controlled for the following variables that can influence the egg counts: number of samples, volume of sediment left after centrifugation and sand in each sample. The presence of sand can obscure the presence of cysts and parasite eggs. The degree of dilution of the sediment, after the last centrifugation for extraction from the tube, can influence egg detection as fewer cysts and parasite eggs may be detected if they are

less concentrated. Repeated samples from the same individual were combined, intensity was averaged before transformation and richness was controlled for sample effort (see below).

For the analysis of seasonal variation repeated samples of the same individual in the same season were pooled; only data from one season were used per individual. The other data were removed, thus ensuring that each animal appeared only once in one season in the data set and that data points were independent. Simple regression models were fitted for either dry vs wet or individual seasons in separate analyses. If either temporal effect was significant, a stepwise analysis was carried out, adding each of the other independent variables in turn and eliminating non-significant variables at $P < 0.05$. This tested whether temporal effects were robust to inclusion of other variables. If both temporal effects (for wet vs dry and for an individual season) were significant, the one which contributed more to the explained variance was included in further analyses. P-values for differences among seasons are reported from this data set.

Analysis for all other variables included all samples combined across seasons. For this analysis different samples from the same individual were pooled across seasons with each season weighted as the fraction of the overall number of samples from that individual. When temporal variation was significant the relevant seasonal variable was always entered into multiple regression. Variables were first analysed using simple regression. All variables significant in a single regression were then entered into a stepwise regression model and removed at $P < 0.05$ through backward elimination. P-values for these variables are reported from the final multiple regression model. Predominantly prevalence and severity data are reported, intensity data are only included if they contain additional information. Age-severity curves are only included if most age-classes contained more than 10 infected individuals.

To find associations between the presence and the severity of different parasite taxa logistic regression and partial correlation were used respectively, controlling for age and sex and any significant variables from the multiple regression models. Cases with ≤ 10 infected individuals were excluded from the analysis. In all the analyses the significance level is set at the 5% level.

To analyse the interactions of the parasite taxa with the host and host ecology required a large number of tests. However, a reduction of the number of variables and thus the number of tests could have lead to an omission of important information. Significant results at 5% have to be treated with caution because by chance alone 5% of the tests would be expected to be significant (Stevens 1986). Such an event is likely due to the high number of tests performed. Significant results at the 5% level are reported here because of the likelihood of a type II error. Type II errors are likely because of small sample sizes, large numbers of variables, and high variances. For prevalence, out of 12

comparisons - one for each parasite taxon - with 13 variables for each taxon, approximately 8 tests could be expected to be significant at the 5% level. For severity, out of 8 comparisons with 13 variables approximately 5 tests may be significant. However, these are considerable overestimates of the number of tests carried out as some variables are highly correlated, such as overall dry /wet seasons and individual seasons, or pride size and number of adults in a pride.

Sample effort

Sample effort is defined as the number of samples collected from the same individual. Significant positive relations were found between the sample effort and species richness in simple regression across the samples (regression n = 112, $r^2 = 0.18$, p = 0.0001) (Figure 8.3.). The more an individual was sampled, the more species of parasites were discovered. Unevenly distributed repeated sampling can bias estimates of parasite richness (Kuris and Blaustein 1977; Kuris *et al.* 1980; Kennedy and Bakke 1989; Gregory 1990; Gregory and Blackburn 1991; Gregory *et al.* 1991). Sample effort was controlled by using standardized residuals of the regression of species richness on sample effort. A linear regression was fitted (although the overall curve is presumably rectangular) because the relationship was well approximated by a straight line over the observed range.

The regression between sample effort and mean of the intensity (when repeated samples were pooled) revealed no significant association (regression n = 112, $r^2 = 0.001$, p = 0.71). Intensity of infection for repeated samples was therefore estimated from the simple mean of all repeated samples. However, to exclude any bias due to unevenly distributed sampling, the number of samples for each datapoint was always tested for significance in the stepwise regression.

8.3 RESULTS

Prevalence and intensity of infection

The overall prevalences of all lion parasites for the whole study is shown in Figure 8.4.. *Spirometra* spp. and the Taeniidae had the highest prevalence while *Physaloptera* sp., *Giardia* sp. and *Toxocara* sp. had a very low prevalence. Only 2.7 % (n = 3) individuals showed no evidence of any infection. Prevalence of *Echinococcus* sp. among the Taeniidae was 9 % (n = 3 from 33 samples infected with taeniid eggs out of 75 samples tested).

Prevalences of prey parasites are shown in Figure 8.5. Paramphistome-like eggs were the most prevalent.

Taxa richness for the whole sample set was distributed as shown in Figure 8.6. The modal host had 3 different parasite taxa while the highest number of parasite taxa found in one animal was 6. The distribution of species richness was not significantly different from that of a summed binomial (χ^2 =10.39, $p > 0.05$ df = 6) or a Poisson distribution ($\chi^2 = 6.77$, $p > 0.10$ df = 6). However, the observed variance differed from that of a summed binomial ($\chi^2 = 149.44$, $p = 0.0088$, df = 111; covariance 0.245), but it did not differ from that of a Poisson distribution ($\chi^2 = 72.56$, $p = 0.10$, df = 111).

The mean severity was by far the highest for *Spirometra* spp. (Table 8.2.). The distributions of the total helminth load and individual parasite species were overdispersed (Eliott 1983): many individuals harboured few parasites and few hosts harboured large numbers of parasites as shown in Figure 8.7 a and b.

Temporal variation

Variation in the levels of parasite infection was observed for a number of parasite taxa (Tables 8.3. and 8.4.) when compared for wet vs. dry season or for a particular season compared to all the others. A lower prevalence of *Spirometra* spp. was found in the wet seasons than the dry seasons (Coef/SE* = -2.75, df = 107, p = 0.006); however, the second wet season (October 1991 - May 1992) was a significant factor itself (Coef/SE = -2.39, df = 107, p = 0.017). Besides an overall difference between the dry and wet seasons ($F_{1, 111} = 10.56$; $p = 0.0015$), *Spirometra* spp. intensity was also significantly affected by the first dry season (July 1991- September 1991; $F_{1,111} = 8.12$; $p = 0.005$) and the following wet season (October 1991 - May 1992; $F_{1,111} = 6.2$; $p = 0.014$). The same pattern was repeated for total helminth severity, where during the first dry season severity was significantly higher ($F_{1,102} = 5.34$, $p = 0.023$), whilst in the second wet season it was significantly lower ($F_{1,102} = 4.33$, $p = 0.026$), resulting to some extent from the considerable contribution of *Spirometra* spp. intensity to the overall severity. The following parasites showed a significant effect in only one season: Coccidia had a significantly higher overall prevalence in the first dry season (June 91-September 91; Coef/SE = 2.26, df = 104, p = 0.024), *Trichuris* sp. only occurred in the dry seasons (first dry season Coef/SE = 3.06, df = 110, p = 0.0022) and a higher severity of spiruid eggs was observed in the last dry season (June 1992 - September 1992; $F_{1,21} = 7.21$, $p = 0.014$). For the other parasite species no significant association with seasons could

* Notation of statistic is related to $\chi 2$ test in analysis of deviance

be detected. Taxa richness was significantly lower during the wet seasons ($F_{1,109} = 5.02$, $p = 0.017$; $n = 112$).

Spatial variation

Prevalence and intensity/severity of parasitic infections varied for certain taxa between the Crater and the Serengeti study areas, within the two Serengeti habitats and among prides (Tables 8.3. and 8.4.). Prevalence of *Spirometra* spp. and hookworm differed significantly between the Serengeti and the Crater: *Spirometra* spp. prevalence was significantly higher in the Crater than in the Serengeti (Coef/SE = 2.65, df = 108, $p = 0.0082$), and hookworm prevalence was significantly less in the Crater than in the Serengeti (Coef/SE = -3.08, df = 110 $p = 0.002$). Two parasites of the lions' prey in the Serengeti, *Trichuris* sp. and the trichostrongyloid egg, could not be detected in the Crater lions.

Considering the differences between woodland and plains habitat in the Serengeti alone, *Sarcocystis* spp. were more prevalent in the woodlands than in the plains (Coef/SE = 2.25, df = 55, $p = 0.024$). Woodlands lions also showed a higher intensity of *Spirometra* spp. ($F_{1,81} = 7.62$, $p = 0.0071$). Severity of *Spirometra* spp. was significantly higher in prides with a large pride size ($F_{1,66} = 10.56$, $p = 0.0018$). The effects of pride size and habitat (woodlands) cannot be clearly separated as large prides live mainly in the Serengeti woodlands.

Not all parasites were detected in each pride. Prevalence of *Giardia* sp. was significantly higher in the woodland Masai Kopje pride than in other prides (Coef/SE = 2.29, df = 60, $p = 0.022$; $n = 62$); *Toxocara* spp. were more prevalent in the Crater Munge pride (Coef/SE = 3.10, df = 53, $p = 0.0019$, $n = 55$). The severity of taeniids (*Taenia* spp. and *Echinococcus* sp.) was lowest in the Crater Gorigor pride ($F_{1,63} = 5.57$, $p = 0.022$).

Age and prevalence and severity

Age was related significantly to levels of parasite infection in only two cases, both of which were spurious parasites. Severity for paramphistome-like eggs was highest in old animals ($F_{1,34} = 13.54$, $p = 0.0008$) while trichostrongyloid eggs were discovered only in the cubs, subadult and young adults (Coef/SE = -2.38, df = 93, $p = 0.017$; $n = 95$), but not in older animals.

Age-prevalence profiles describing the relationship between prevalence of individual parasite taxa and the age of the host are shown in Figures 8.8.a - k and age-severity profiles in Figures 8.9.a - d. Parasite prevalence and severity for *Spirometra* spp. was highest in the unweaned cubs and lower and approximately the same for the other age-classes. Prevalence of infection with Coccidia was higher in younger than in older animals. *Taenia* spp. and *Echinococcus* sp. eggs had the lowest prevalence and severity in cubs. Hookworm and helminth larvae prevalence was low during the juvenile phase, but hookworm did not show the typical hookworm curve for humans with a plateau reached in adolescence (Anderson 1982; Anderson and May 1991; Bundy and Medley 1992). No *Trichuris* sp. eggs were found in young animals whereas the trichostrongyloid parasite was only found in cubs, subadults and young adults. *Toxocara* sp. were found in 5 individuals, however, only 3 were of known ages, 1 subadult and 2 young adults. *Giardia* sp. was detected only in subadults, young adults and middle-aged adults, but not in unweaned or old animals. Spiruid egg, helminth larvae, paramphistome-like and *Sarcocystis* spp. did not exhibit a distinctive age-related pattern. Total helminth load was strongly influenced by the intensity and severity of *Spirometra* spp. infection. If the egg counts for *Spirometra* spp. were excluded, severity of all the other helminths across the age groups was roughly the same ($F_{4,90} = 1.184$, $p = 0.3232$).

Sex

There was no significant difference in the prevalence, intensity, severity or taxa richness of parasites between the two sexes (Table 8.3. and 8.4.).

Reproductive status

One significant association between the reproductive status of the females and parasitic infection was found: *Spirometra* spp. severity was lower in lactating females ($F_{1,64} = 10.18$, $p = 0.0022$) (Table 8.3. and 8.4.).

Genetics

The available genetic data were tested for an association with parasite infection. The overall score for heterozygosity was significantly negatively associated with prevalence of taeniids (Mann-Whitney *U*, $U = 56$, z corrected for ties -2.547, $p = 0.011$;

n = 31) (Table 8.3. and Table 8.4.). None of the other taxa showed any correlation with overall heterozygosity or with genotype at any locus.

Parasite communities

Correlations between parasite taxa were found for prevalence of *Spirometra* sp. and helminth larvae (negative; Coef/SE = -2.01, df = 87, p = 0.045) and paramphistome-like and Coccidia (positive; Coef/SE = 2.00, df = 89, p = 0.045). There was no correlation between the taxa for severity.

8.4 DISCUSSION

Comparison with zoo lions

This is the first study to describe prevalence and intensity of intestinal parasites in wild lions. Previously, only parasite prevalence and intensity in zoo lions have been reported (Barutzki *et al.* 1985; Movsessian *et al.* 1987; Ghoshal *et al.* 1988; Tang *et al.* 1988). The prevalence of parasite species observed in the faeces of the Serengeti and Ngorongoro Crater lions differed considerably from that of zoo lions. *Toxascaris* was not found at all in wild lions, but appears to be common in zoo lions (Tscherner 1974; Gaur *et al.* 1979; Ippen and Henne 1982; Barutzki *et al.* 1985; Ghoshal *et al.* 1988). *Toxocara* was found less frequently in wild lions than in zoo lions (Ippen and Henne 1982; Barutzki *et al.* 1985; Ghoshal *et al.* 1988), whereas taeniids were more common in wild lions, probably due to higher infection rates in wild prey. *Spirometra* was the most common parasite in wild lions, but has not yet been recorded in zoo lions. In general these differences may be explained by the fact that zoo lions feed on the meat of domestic animals and live in close proximity to a wide variety of species from all over the world.

Overall distribution

Almost all lions were infected. Faecal samples from only 3 (2.7%) lions showed no evidence of infection, and even this might be due to too few eggs to be detected. Across the lion populations, both the distribution of any one parasite species and the overall mean intensity of infection were overdispersed, a characteristic pattern of most macroparasite infections, indicating differences in exposure, innate and/or acquired

immunity (Anderson 1978; Anderson and Gordon 1982; Anderson and May 1991). These may be related to host factors such as age, sex, reproductive status and genetics or to environmental factors or seasonal changes. The number of significant associations with the measured variables was low. However, not all lions had the same parasites; a patchy distribution of parasites across the study populations could be observed. Most differences were found in relation to habitat or pride. Some variance in infection may be explained through sampling effects, measurement errors and unmeasured host factors such as immunity, nutrition and behaviour.

Spatial Variation

Crater vs. Serengeti

Spatial variation in infection may be caused by differences in the genetics of host and parasite populations, differences in environmental factors between habitats, or different host group sizes. Crater lions are more inbred than Serengeti lions with a lower degree of genetic diversity in the isozymes tested (Yuhki and O'Brien 1990), and may therefore be more susceptible to disease. However, the effect of inbreeding may be confounded by several other factors; the Crater lions are better fed and form a denser population, but have on average prides the same size as the lions in the Serengeti. Furthermore, the two habitats vary in their ecology, and the Crater lion population is ecologically isolated. Although differences in parasite burden between Crater and Serengeti lions may therefore be expected, there was no evidence of greater lion parasite richness nor overall higher parasite burden in the Crater or the Serengeti. *Spirometra,* with significantly higher prevalence and intensity in the Crater, was the only parasite which showed the predicted difference. *Spirometra* is the most common parasite of lions and therefore this difference may indicate differences in susceptibility to infection between the two populations and could influence the fitness of both populations. Higher susceptibility to *Spirometra* in Crater lions may have a genetic basis. On the other hand, no significant relationship between allelic heterozygosity and *Spirometra* prevalence was found. It is possible that differences in *Spirometra* infection between the two lion populations are more influenced by environmental factors, such as the higher occurrence of swampy areas in the Crater, which provide transmission opportunities (as the first intermediate host is a copepod), or the high year-round availability of prey (Kruuk 1972; Packer *et al.* 1988; Hanby *et al.* in press), which may transmit infection.

The other difference between the Serengeti and the Crater in the contrary direction was the lower hookworm burden in the Crater. The reasons for the differences in

hookworm infection are not obvious. Perhaps the better nutritional status of the Crater lions has an impact on their susceptibility. In the laboratory, better nourished mice have been shown to mount a more effective immune response against hookworm under conditions of repeated infections and high levels of exposure (Keymer and Slater 1990). Differences in the type of soil may also influence hookworm distribution (Augustine and Smillie 1926).

No differences in levels of infection between the Crater and the Serengeti were observed that also correlated significantly with the genetic data of the lions. The only association between genetics and parasitic infection, the negative correlation between heterozygosity and taeniid prevalence, may indicate that greater homozygosity can increase susceptibility to taeniids. However, the result has to be treated with caution because of its low significance level. It was also not supported by significant differences in taeniid prevalence between the Serengeti and the Crater.

Several reasons may explain why genetic variability had little noticeable effect on the observed parasite levels. The effects of inbreeding might only be revealed once the lions find themselves in a nutritionally stressful situation (Frankel 1981) or under the impact of new diseases, when a more heterozygous immune system may provide a better chance of an effective immune response. Additionally, animals which survived the bottleneck might have been the healthiest or most resistant individuals and possessed a MHC well equipped to deal with the common parasitic diseases. The effects of a more homozygous MHC are subtle and possibly much larger sample sizes over a greater span of time would have been required to observe any significant effects, because there may be only small differences in parasite burden between inbred and outbred individuals masked by confounding factors such as short-term environmental factors or other host specific factors such as nutrition. In this study, only a limited number of isozymes were tested for homo- and heterozygosity for some lions and thus might not reflect the overall interactions between host immunity and parasites for each parasite species. Moreover, other genes outside the MHC may be important for response to infection (Kennedy 1990).

The Crater has been suggested to be an "ecological island" with fewer or different parasites because of the isolation of the lion population (Packer *et al.* 1991). In contrast to the Serengeti, lions may migrate out of the Crater but no immigration has been recorded since 1969 (Packer *et al.* 1991). This situation is not reflected in the prevalence of lion parasites. The same parasite species were found in both areas with the exception of the prey parasites: *Trichuris* and the trichostrongyloid parasite, which were not found in faeces from lions in the Crater.

Other spatial differences

Spatial differences were not only found between the Serengeti and the Crater, but also among prides and within the Serengeti. Differences between prides may be related to habitat or group size. If they were related to group size, higher levels of infection may be expected in larger groups as none of the parasites studied was transmitted via a biting-vector, where bigger groups may be associated with a reduction of levels of infection (Anderson and May 1991; Coté and Poulin 1994). Only two parasites, *Spirometra* and the helminth larvae, showed positive associations. *Spirometra* showed higher intensity in the woodlands and higher severity in the larger prides. However, large prides predominantly live in the woodlands; hence these factors cannot be easily separated. The positive correlation between numbers of adults in a pride and prevalence of helminth larvae and the negative correlation between number of adults in a pride and prevalence of Coccidia may be more closely related to differences in infection between cubs and adult lions than to group size, as total pride size did not indicate any significant differences. The effects of group size cannot be clearly demonstrated here. Possibly pride size is not important for differences in infection between prides, due to the low influence of group size on such indirectly transmitted parasites as cestodes. Furthermore, the range in pride sizes may have been too narrow and the sample size for each group too small to detect subtle differences.

Spirometra, the Taeniidae and *Sarcocystis* are acquired from prey, so that differences in prey between prides or differential levels of prey's infection in different habitats may be reflected in differences in parasite burden among lion prides. Furthermore, differences in distribution of other intermediate hosts and differences in transmission opportunities for the parasite may influence the parasite burden among prides. For instance, lions in the woodlands were found more frequently with buffalo (*Syncercus caffer*) and warthog (*Phacochoerus aethiopicus*) carcasses; topi (*Damaliscus korrigum*) and hartebeest (*Alcelaphus buselaphus*) were also more common in the woodlands than on the plains (Scheel and Packer, in press). Because the woodland prides share the same type of habitat and prey, in contrast to the plains lions, these differences may be mirrored in certain parasitic infections. Woodland prides showed higher prevalence of *Sarcocystis* infection and higher intensity of *Spirometra* infection. The higher levels of infection may be related to higher prey density in the woodlands, type of prey and/or higher infection of the intermediate hosts. The main water source in the woodlands for lions and their prey are stagnant water holes. In contrast, on the plains animals drink from water holes fed by springs. Possibly the water holes in the woodlands may provide good transmission opportunities for several parasites including *Spirometra*, which has a copepod as an intermediate host. Of the direct life cycle parasites, infection

with *Giardia*, transmitted via water, was found to have the highest prevalence in one of the prides (Masai Kopje) resident in the woodlands. *Toxocara* was most prevalent in one pride in the Crater. Since prides share habitats, predation behaviour and prey, it is not surprising that differences among prides and between prides of certain habitat types emerge most strongly from these analyses.

Temporal variation

Parasitic infection showed seasonal variation only for *Spirometra*. However, the lower prevalence of *Spirometra* in the wet season is strongly influenced by the second dry season. A possible hypothesis to explain the seasonal pattern for *Spirometra* is that the higher parasite egg output in the dry season may facilitate transmission. *Spirometra* may discharge more eggs in the dry season when lions and a high density of intermediate hosts are more likely to visit particular water holes thereby increasing the chances of transmission. Seasonal egg release in *Spirometra* may also be a result of its phylogenetic status, intermediate between cestode species still showing seasonal transmission and other cestodes species that do not (Kennedy 1983). For the other taxa temporal differences were found for one season and these differences were mainly significant at the 5% level. Only *Trichuris* showed considerable fluctuations.

Even though few associations between parasites and seasons were detected, season may still be important in determining parasite population dynamics. Seasonal categories based on rainfall data may not be sensitive enough to detect a pattern of variation as parasite prepatent periods can be long (Soulsby 1982); this could obscure association of season with parasite egg output. In addition, prey movements or the mode of parasite transmission, which may be connected with season but do not show variation according to rainfall, may also confound seasonal patterns. The longevity of certain parasites could further mask seasonal effects. On the other hand, some temporal variation is probably not correlated with season but other events, such as changes in the population dynamics of the intermediate hosts.

Age

Lion parasite prevalence and severity were not significantly correlated with age. Only two spurious parasites, paramphistome-like and trichostrongyloid eggs showed a significant association with age. Paramphistome-like egg severity was highest in old lions. Eggs of these parasites are found in the gut and intestines of the prey. Hence, lion

feeding behaviour may influence their acquisition. The part of the prey animal a lion eats varies with lion age; adult lions have much better access to the gut and intestines (Schaller 1972; C. Packer, personal communication). *Trichuris* eggs were not found in the very young animals, but were not significantly correlated with age. If exposure by diet explains the correlation between age and parasite burden, it is puzzling why on the other hand the eggs of the other spurious parasite showed an opposite trend. Trichostrongyloid eggs were not prevalent in middle - aged adults and old animals.

Although the other parasites were not significantly correlated with age, age-prevalence/severity curves provided evidence for associations between age and infection. Age-prevalence curves for *Spirometra* and Coccidia showed a rapid acquisition of parasites in cubs. All other parasites peaked later. Rapidly rising prevalences imply high transmission rates. A higher prevalence of Coccidia in young animals has been observed for other host species as well (Hammond and Long 1973; Pellerdy 1974). Although *Taenia*, *Echinococcus* and *Spirometra* are all transmitted through prey, they did not show the same age related patterns. Only *Spirometra* had a high prevalence in unweaned cubs. Unweaned cubs first eat meat at the age of about 4 - 6 weeks (Schaller 1972; C. Packer personal communication). Peak parasite burden in cubs may be caused by lower adult exposure, lower adult innate susceptibility or acquired immunity. Cubs may have lower immunity against *Spirometra* as compared to the taeniids, or *Spirometra* may be transmitted by different intermediate hosts than *Taenia* and *Echinococcus*. *Giardia* is transmitted by water, and unweaned cubs presumably have less contact with water. Many of the older animals without *Giardia* infection may have acquired immunity against the parasite (Schmidt and Roberts 1989). Hookworm and helminth larvae in the lions were quickly acquired during the juvenile phase and declined afterwards. Possibly the decrease in hookworm prevalence in older lions results from an acquired immunity, a change in behaviour or physical properties, such as thicker skin.

Sex and reproductive status

With one exception neither sex nor reproductive status seem to play a role in lion parasite distribution. Although male lions have higher testosterone levels (see also Festa-Bianchet 1990; Zuk 1990; Folstad and Karter 1992), a higher white blood cell count (C. Packer, personal communication) and have higher chances of parasitic contact due to greater food intake rates, males did not show higher levels of parasite infection than females. However, the larger body size of male lions may dilute the higher intake of infective stages. Hormones may not exert a large influence on the distribution of lion parasites. This may also be reflected by the low association between reproductive status

and parasitic infection. Lactating females showed lower severity of infection than pregnant and other females only for *Spirometra*, which contrasts with other studies which reported higher levels of infection in pregnant and lactating females (Connan 1976; Michel 1976; Behnke 1990; Brabin and Brabin 1992). However, the number of females that were pregnant (n = 6) or lactating (n = 13) may have been too small to reveal significant effects for the less frequent parasites.

Parasite-parasite interactions

An association among parasite species may indicate competition for host resources, density constraints, benefits from the presence of another parasite, predisposition of the host for certain parasite species or correlation because of joint presences or absences of parasite species, possibly due to related routes of transmission (Lotz and Font 1994). Only 2 marginally significant interactions were detected between any parasite species, which might have been the result of the number of tests performed. On the other hand, the low prevalence of some taxa in this survey and the estimate of parasitic infection by faecal egg counts could obscure actual relationships. However, the analysis suggests that parasite-parasite interaction might not play an important role in the lion-parasite system. Moreover, this was supported by the result that the observed distribution of parasite richness did not differ significantly from a summed binomial distribution, indicating no interaction between parasite species, although the variance in the observed richness was larger than expected. The observed distribution does not necessarily imply that the distribution of parasite species among hosts is a random process, but that patterns of infection in one particular parasite species are not correlated with patterns of infection in another parasite species.

8.5 CONCLUSION

The distribution of parasites in individual lions was overdispersed, but the large differences expected between the Ngorongoro Crater and Serengeti populations were not observed. Much of the observed variation in parasite prevalence and intensity/severity was unaccounted for by the known factors relating to host characteristics and environmental variables. The factor that showed the greatest impact on levels of infection was spatial variation between populations and between groups within populations. Although genetic variability may be important in determining levels of parasite infection, differences in infection between the Crater and the Serengeti could not be separated from other factors or

shown to be linked to genetic differences. Here, measured host genetics does not appear to be a major factor for determining parasite distribution. Spatial variability in infection may be related to ecology of the prey, environmental factors or possibly group size. Seasonal variation in infection was only important for *Spirometra*; however, fluctuations occurred in levels of infection that were not related to season. Age-prevalence and age-severity curves indicated possible relationships between behavioural and immunological processes and parasites. Parasite-parasite interactions or host sex and reproductive status did not account for much of the variance in parasite burden.

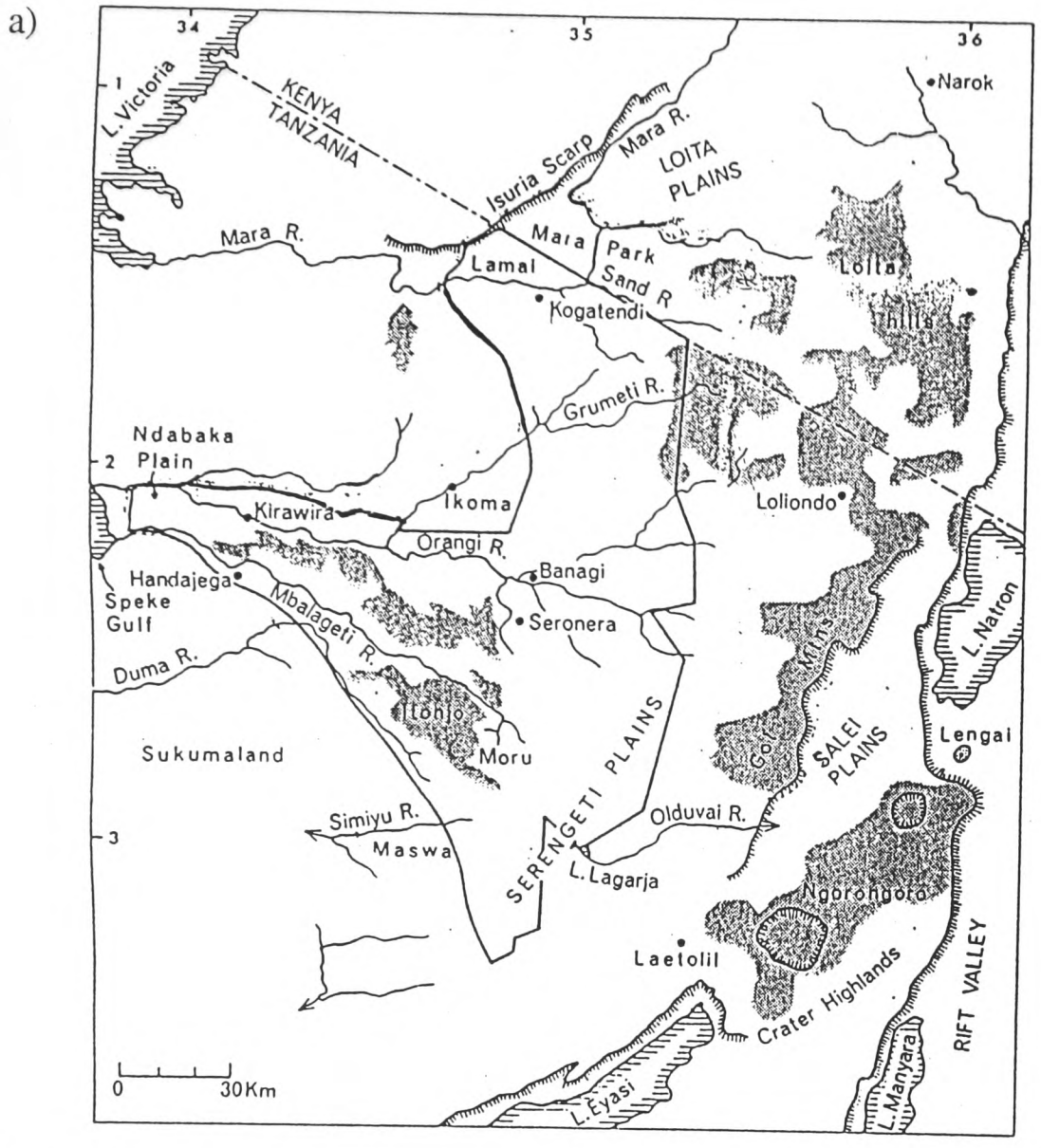


Figure 8.1. a) The Serengeti ecosystem and Ngorongoro Crater. Map from Sinclair, (1979)
b) Study areas in the Serengeti National Park and Ngorongoro Conservation Area. Approximate pride ranges are drawn in the study areas. The woodlands prides are those whose ranges are primarily in the woodlands habitat at the northern edge of the study area. Map from Packer (1988)

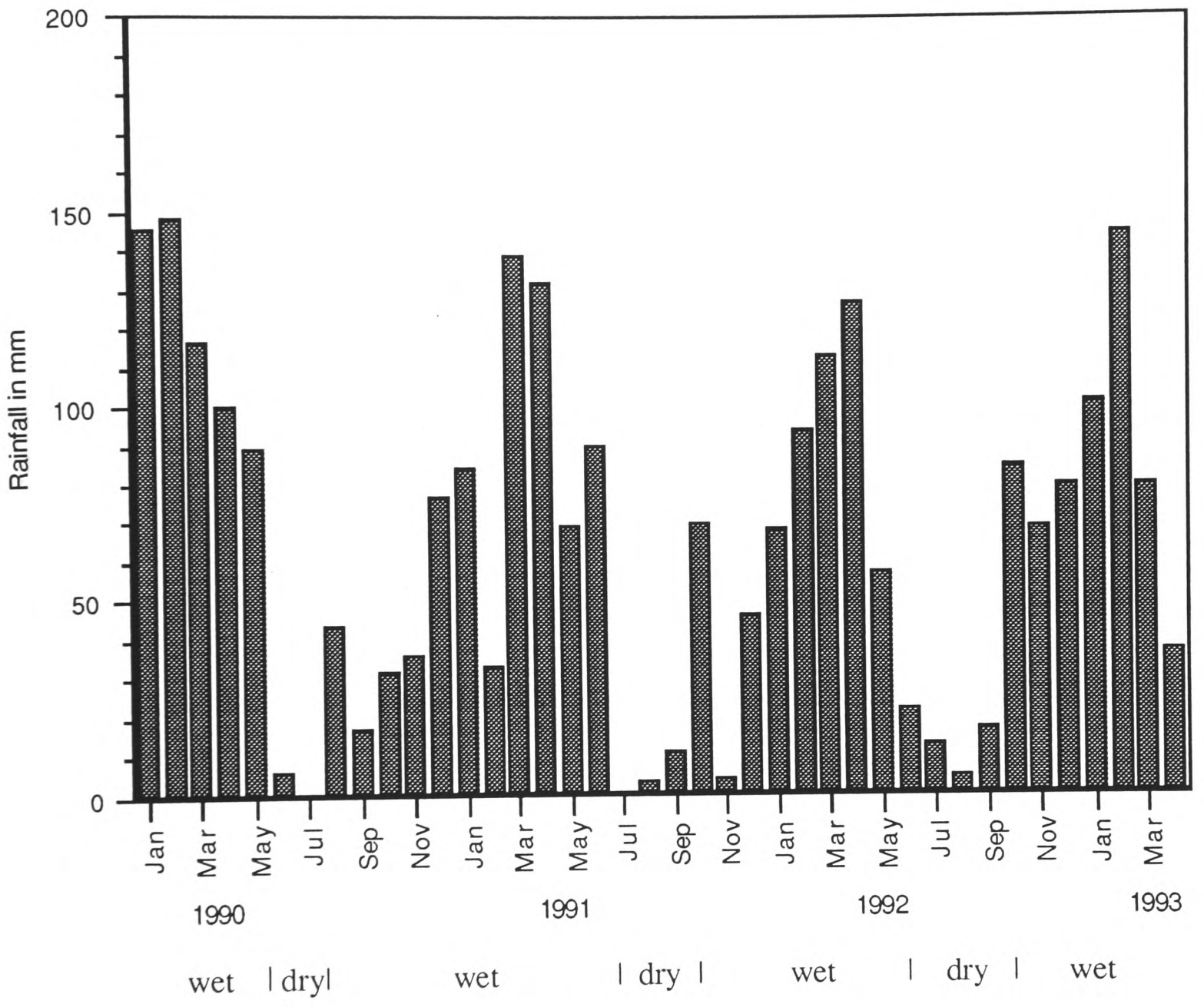


Figure 8.2. Rain was measured in mm at rain gauges in the Serengeti woodlands near Seronera. Seasons are indicated.

Table 8.1. Distribution of samples across the study area

Pride	no of individuals sampled	pride size at time of sampling	Serengeti/ Crater	Woodlands/ Plains
Barafu	1	11	S	P
Campsite	20	25 - 28	S	W
Gorigor	4	10 - 12	C	
K2	2	4	S	P
Kibumbu	4	4	S	P
Lakettes	3	6 - 8	C	
Lakes	2	13	C	
Loliondo	6	21 - 26	S	W
Munge	5	15	C	
Masai Kopje	15	14 - 20	S	W
Masai	3	12 - 13	S	W
Sangere NW	5	8 - 15	S	W
Plains	5	16 - 15	S	P
Seneto Mounds	7	10 - 21	C	
Tokitok	4	10 - 16	C	
Transsect	16	18 - 19	S	W
Simba	1	14	S	P
Simba West	1	6	S	P
Simba Numbers	3	15 - 16	S	P
Sametu	1	4	S	P

Four animals for whom pride of residence was unknown or who were solitary were also sampled. Pride sizes changed over the study period.

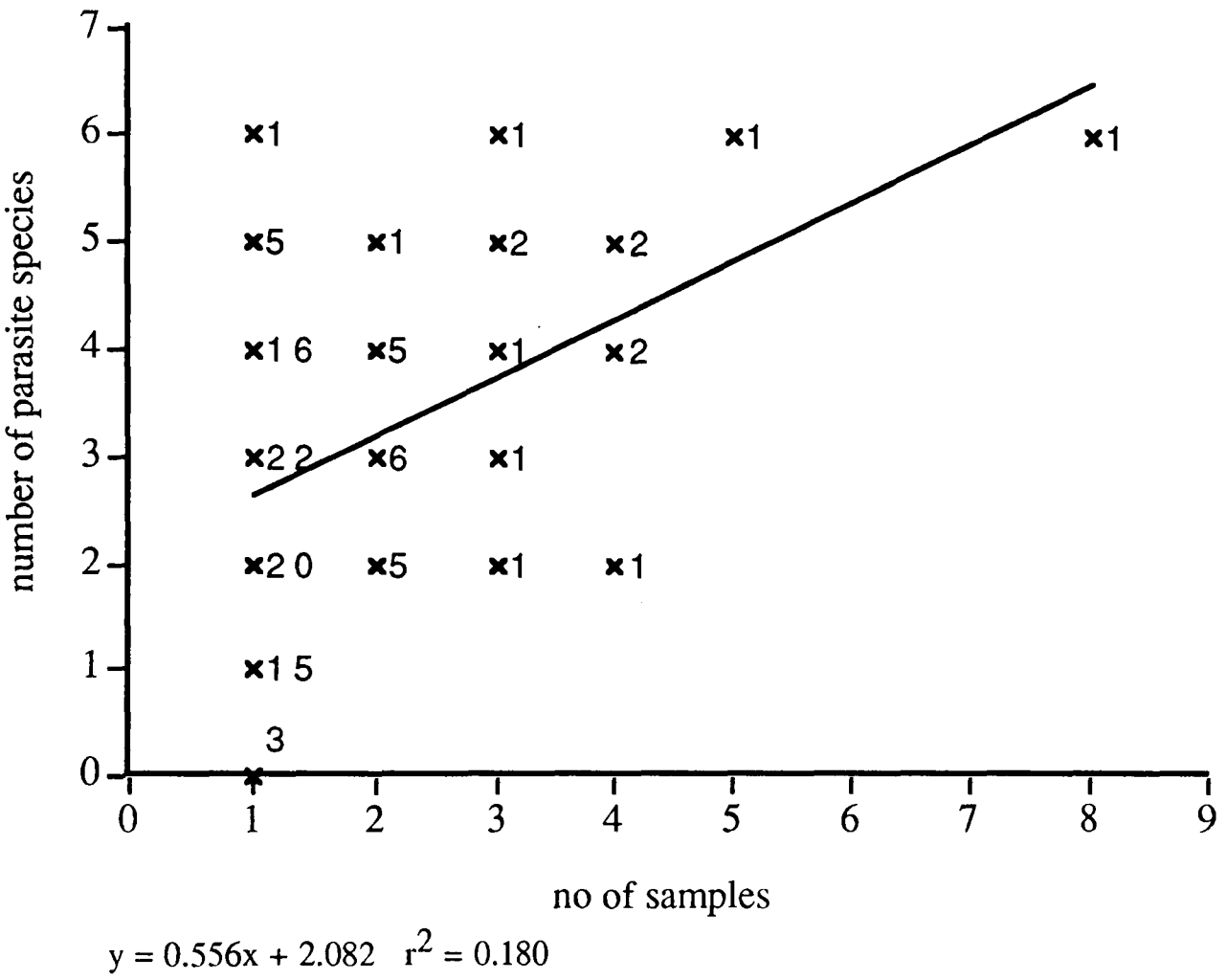


Figure 8.3. Sample effort.
Regression of number of identified parasite taxa against number of samples collected. Numbers indicated the number of data points per point on graph (n = 12).

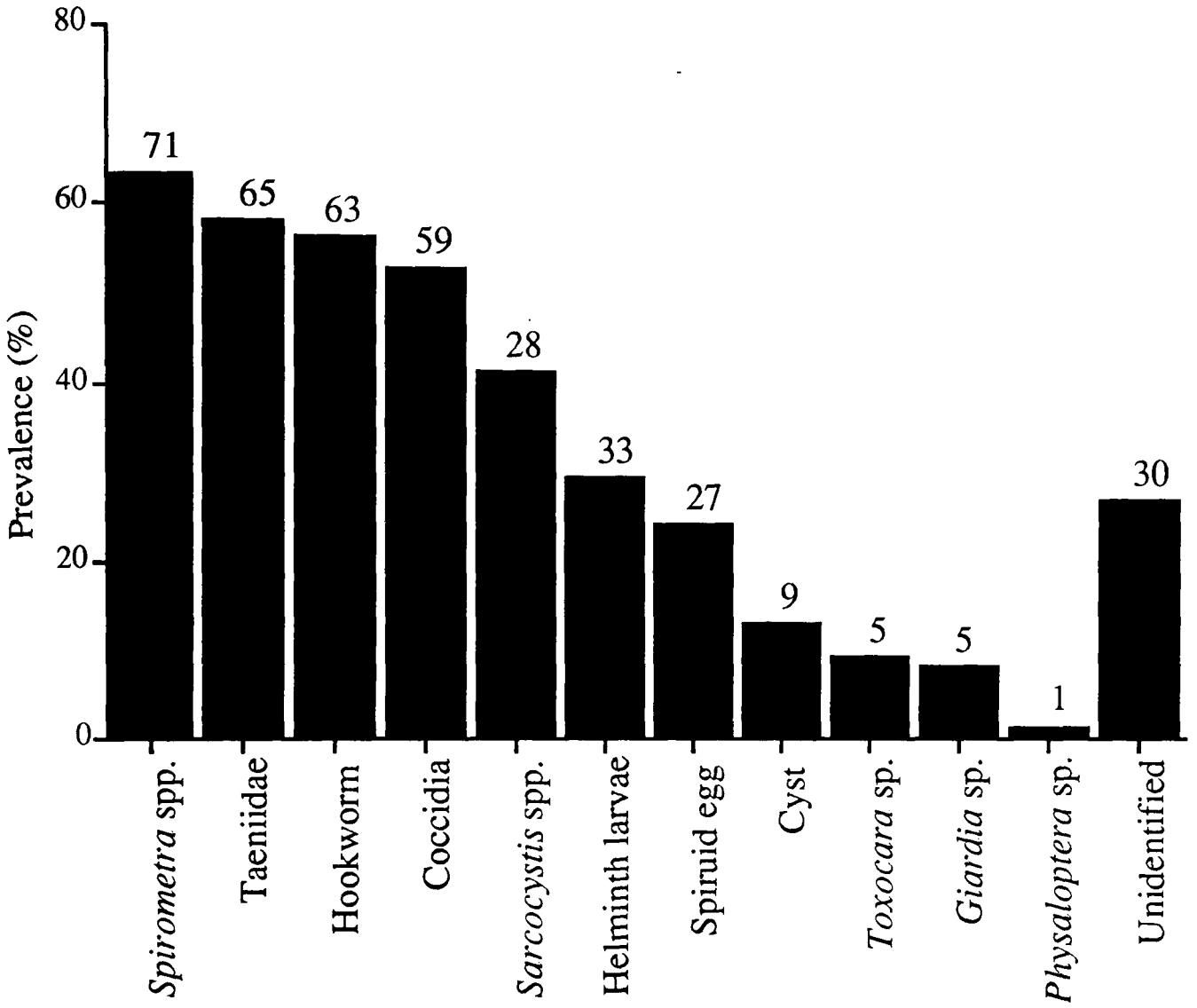


Figure 8.4. Prevalence of lion parasites.
Numbers of individuals found infected with each parasite are indicated on top of each bar.
N = 112 except for: *Toxocara* sp. n = 55, helminth larvae n = 108, Coccidia n = 108, *Sarcocystis* spp. n = 58, *Giardia* sp.n = 62 and cyst n = 70.

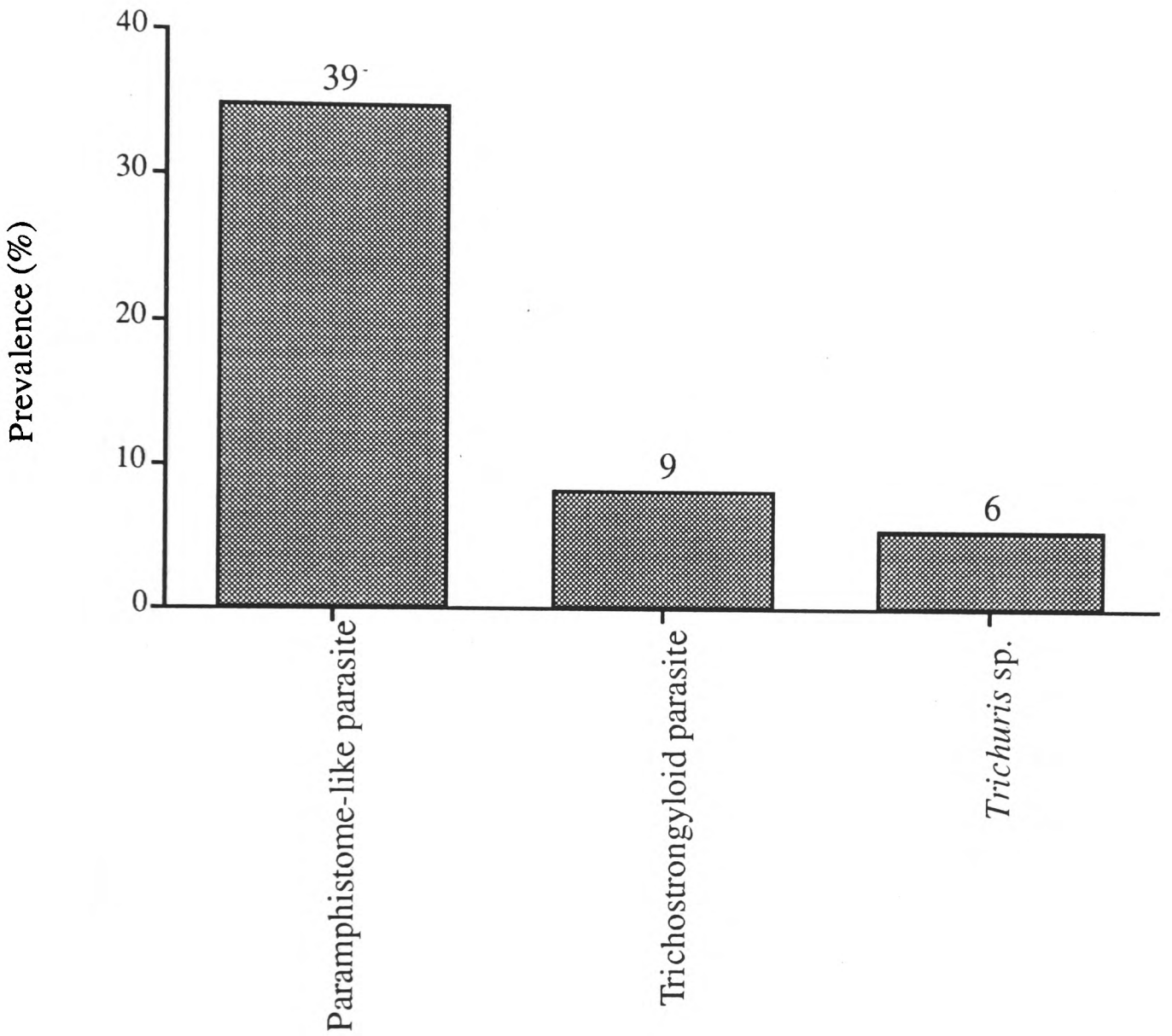


Figure 8.5. Prevalence of spurious parasites.
Numbers of individuals in whose faeces each parasite was detected are indicated on top of each bar (n = 112; except for the trichostrongyloid parasite n = 95).

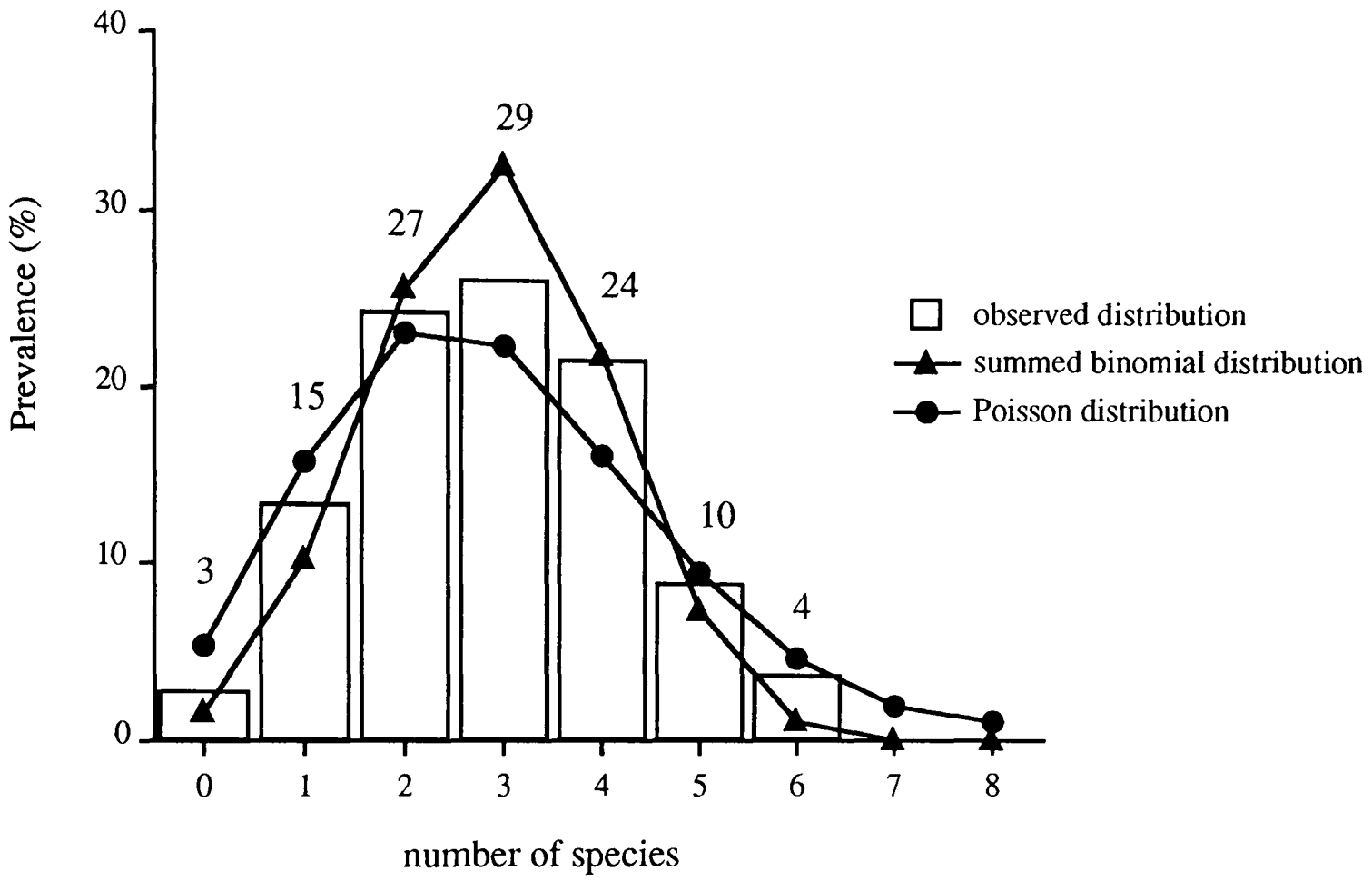


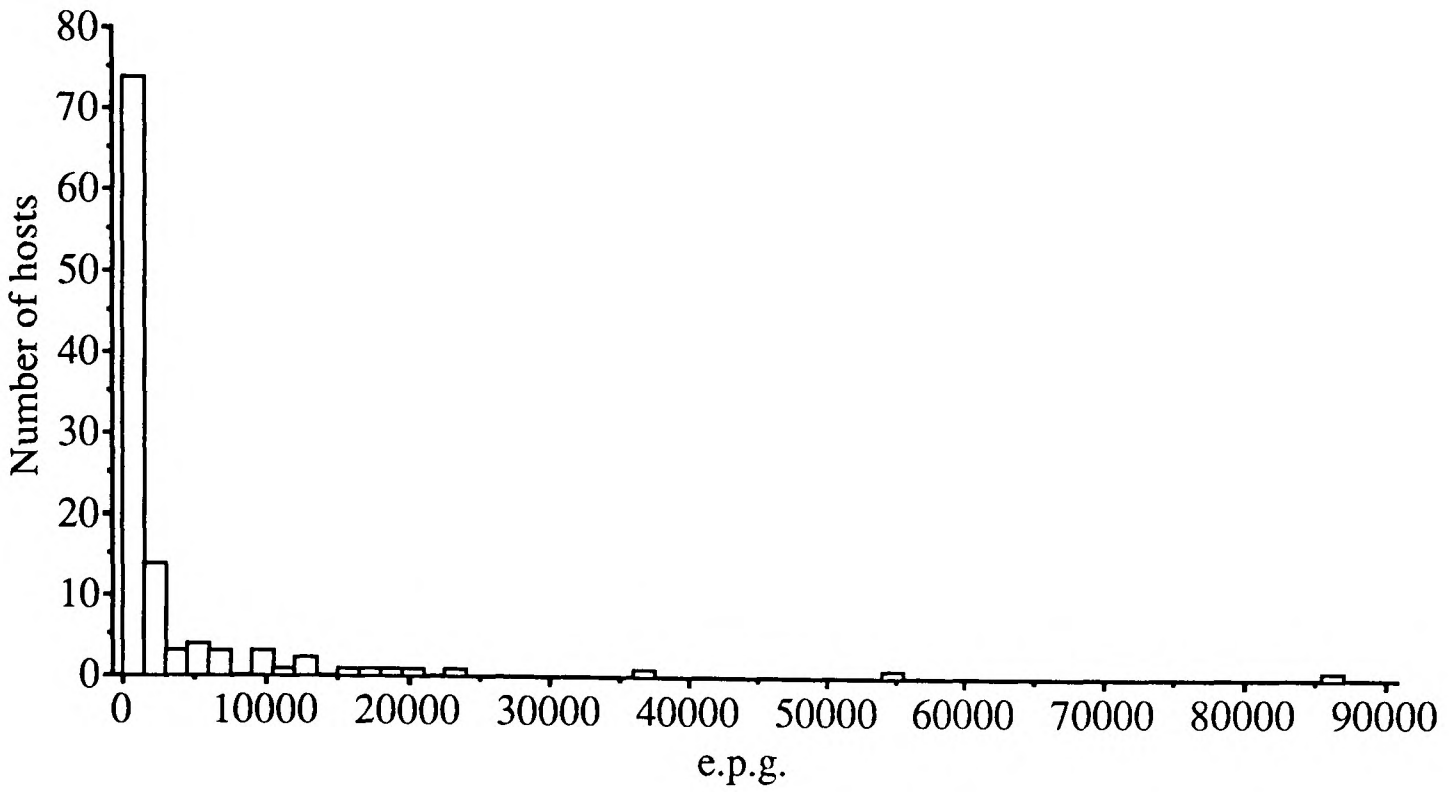
Figure 8.6. Observed frequency distribution of lion parasite richness (excluding *Sarcocystis* spp. and *Giardia* sp.; including results from multiple samples) in comparison with a calculated summed binomial and Poisson distribution. Numbers on top of each bar indicate the number of individuals found in each category (n = 112).

Table 8.2. Median severity and maximum parasite e.p.g.

Parasite taxa	median severity in e.p.g.	maximum e.p.g.
Spirometra spp.	975	83350
Taeniidae	39.6	6850
Hookworm	50.0	625
Helminth Larvae	2.3	5700
Paramphistome-like egg	25.0	3700
Spiruid egg	16.7	650
Trichostrongyloid egg	16.7	550
Toxocara sp.	8.3	483.3
Trichuris sp.	34.4	200

Physaloptera sp. was excluded as it was only detected in one animal. For numbers of infected animals see Figures 8.4. and 8.5.
e.p.g. = eggs per gram of faeces

a) Total helminth load



b) *Spirometra* spp.

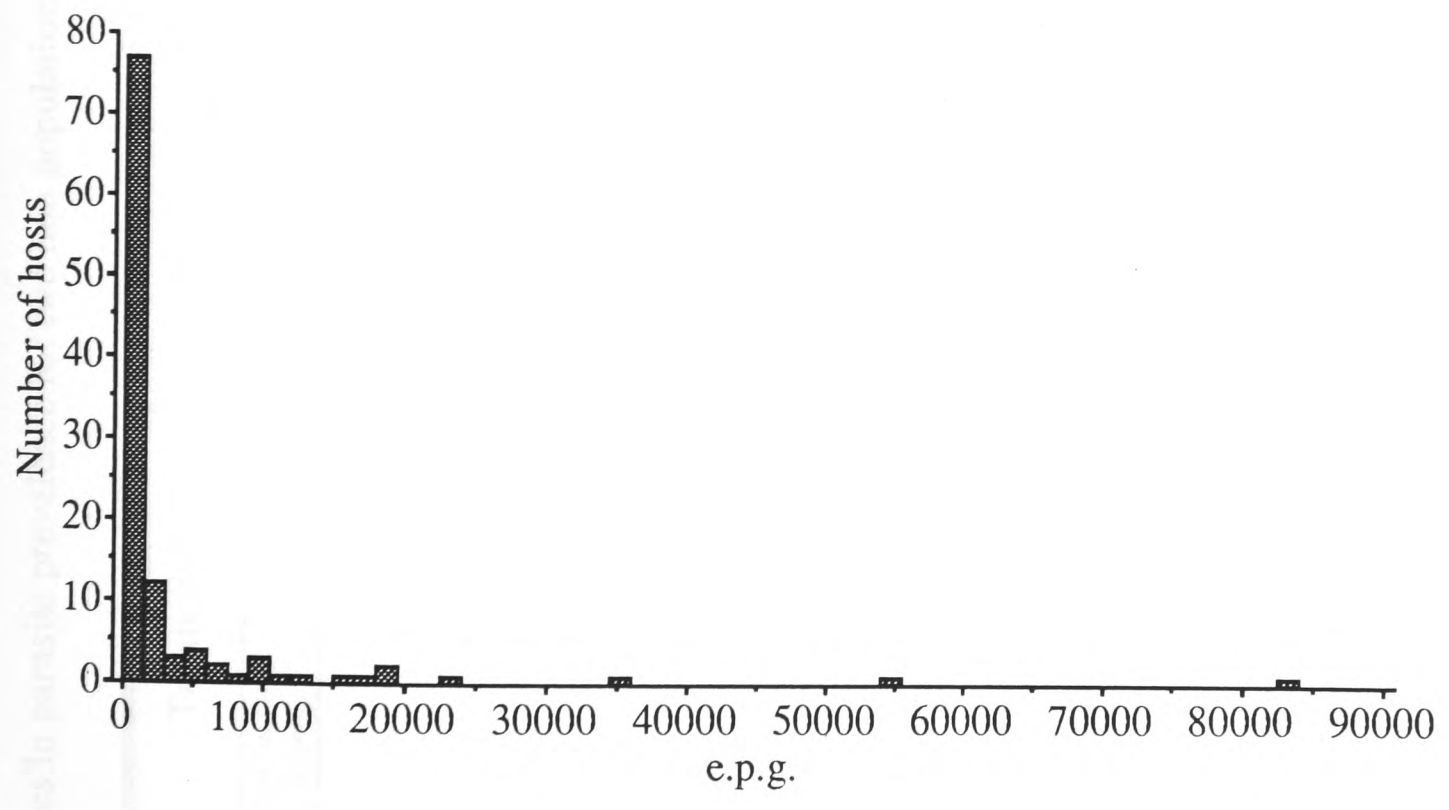


Figure 8.7. Frequency distribution of egg output (e.p.g. = eggs per gram of faeces) of a) total helminth load (arithmetic mean = 3957 e.p.g., SD = 10772) and b) *Spirometra* spp. (arithmetic mean = 3632 e.p.g., SD = 10548). Zero counts are included in the first bar. Bar intervals describe 1500 e.p.g.

Table 8.3. Differences in parasite prevalence for two lion populations in the Serengeti and Ngorongoro Crater.

	Temporal		Spatial				Hosts			
Parasite	Wet vs.Dry Season	Individual Seasons	Serengeti vs. Crater	Woodlands vs. Plains	Prides	No of Adults in Pride	Age	Sex	Reproductive Status	Heterozy-gosity
Hookworm	-	-	**(+)	-	-	-	-	-	-	-
Toxocara sp.	-	-	-	-	**	-	-	-	-	-
Helminth larvae	-	-	-	-	-	* (+)	-	-	-	-
Spiruid egg	-	-	-	-	-	-	-	-	-	-
Taeniidae	-	-	-	-	-	-	-	-	-	*(-)
Spirometra spp.	**(-)	**	** (-)	-	-	-	-	-	-	-
Giardia sp.	-	-	-	-	*	-	-	-	-	-
Coccidia	-	*	-	-	-	**(-)	-	-	-	-
Sarcocystis spp.	-	-	-	*(+)	-	-	-	-	-	-
Trichuris sp.	-	**	-	-	-	-	-	-	-	-
Paramphistome-like egg	-	-	-	-	-	-	-	-	-	-
Trichostrongyloid egg	-	-	-	-	-	-	*(-)	-	-	-

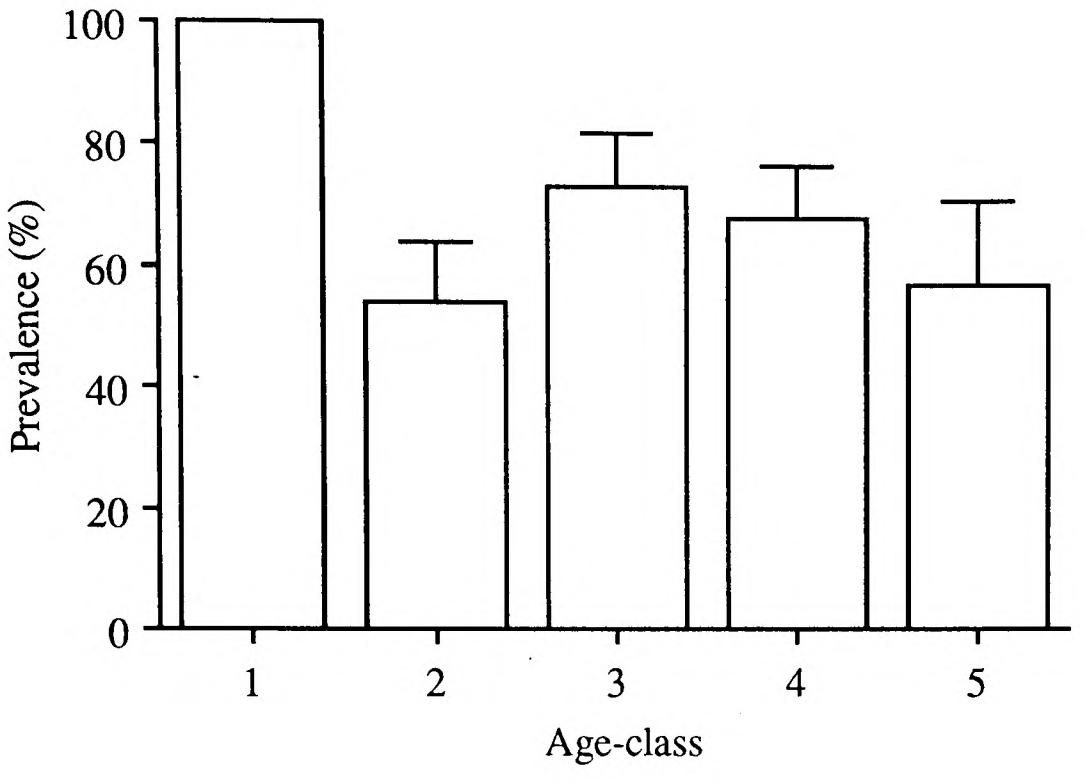
P-values are taken from logistic regression.
- = $p > 0.05$, * = $p < 0.05$, ** = $p < 0.01$, *** = $p < 0.001$; (+) or (-) indicates the direction of the coefficient or whether the first of the two categories compared has higher or lower prevalence.
If not stated otherwise n = 112 (*Toxocara* sp. n = 55, helminth larvae n = 108, Coccidia n = 108, *Giardia* sp. n = 62, *Sarcocystis* spp. n = 58, Trichostrongyloid egg n = 95).

Table 8.4. Differences in parasite severity for two lion populations in the Serengeti and Ngorongoro Crater.

	Temporal		Spatial				Hosts			
Parasite	Wet vs. Dry Season	Individual Seasons	Serengeti vs. Crater	Woodlands vs. Plains	Prides	Pridesize	Age	Sex	Reproductive Status	Heterozygosity
Total helminth load	*(-)	*	-	*(+)	-	-	-	-	-	-
Hookworm	-	-	-	-	-	-	-	-	-	-
Helminth larvae	-	-	-	-	-	-	-	-	-	-
Spiruid egg	-	*	-	-	-	-	-	-	-	-
Taeniidae	-	-	-	-	**	-	-	-	-	-
***Spirometra* spp.**	-	-	-	-	-	**(+)	-	-	**	-
***Trichuris* sp.**	-	-	-	-	-	-	-	-	-	-
Paramphistome-like egg	-	-	-	-	-	-	***(+)	-	-	-
Trichostrongyloid egg	-	-	-	-	-	-	-	-	-	-

P-values are reported from multiple regression.
- = $p > 0.05$, * = $p < 0.05$, ** = $p < 0.01$, *** = $p < 0.001$; (+) or (-) indicates the direction of the coefficient or whether the first of the two categories compared has higher or lower severity.
The number of infected animals varied for different taxa (see Figs. 8.4., 8.5.). *Toxocara* sp. was excluded because of low prevalence.

a) *Spirometra* spp.



b) Coccidia

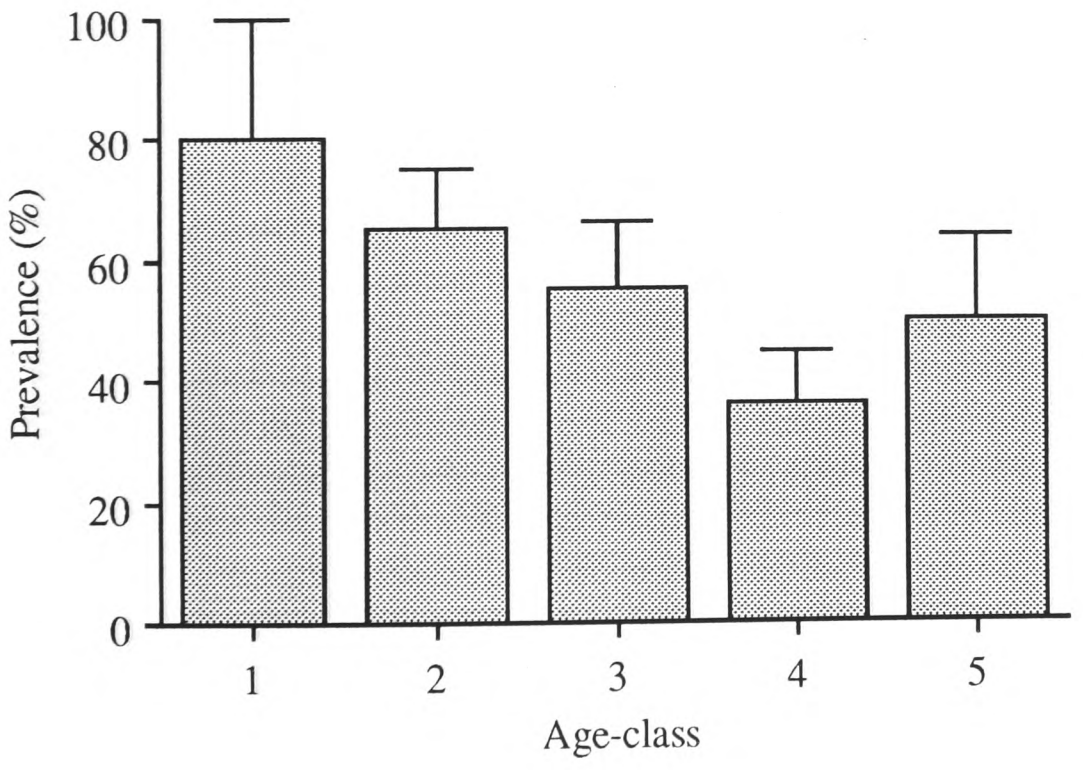
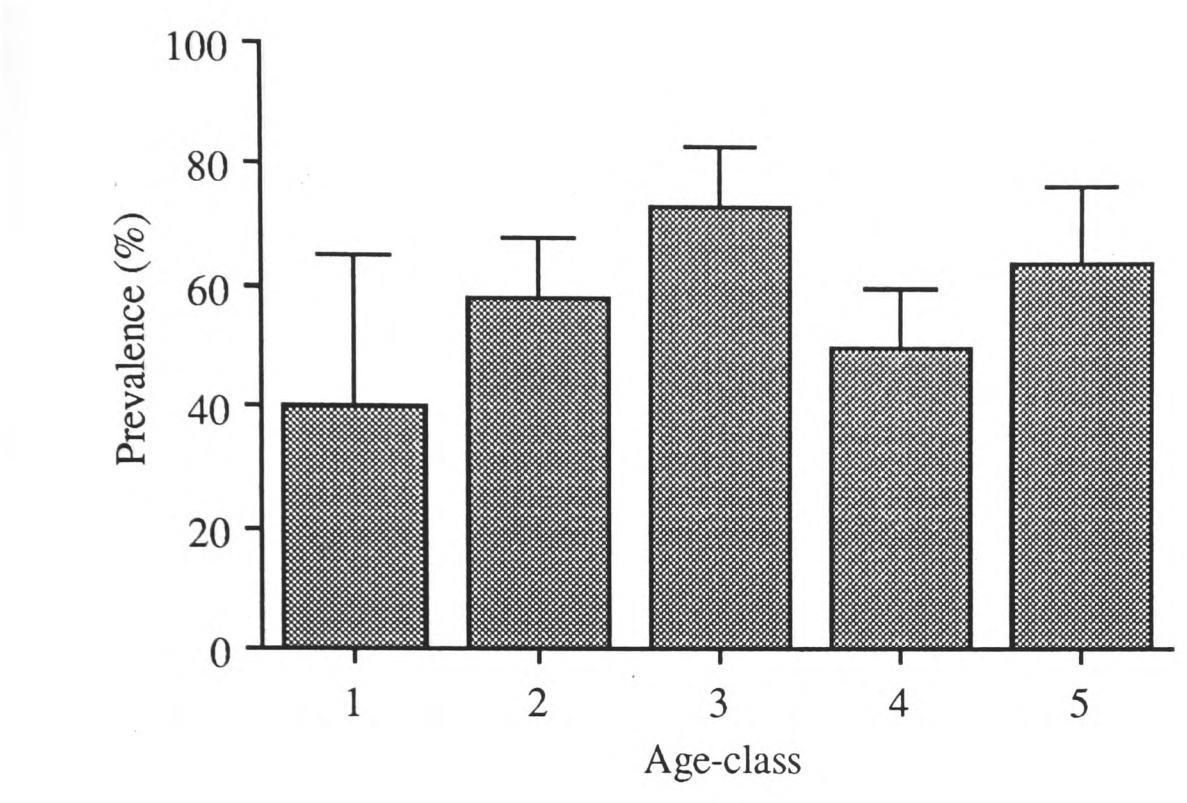


Figure 8.8. Age-prevalence profiles.
Lions were divided into five age categories: 1 = unweaned (birth to 8 months, n = 5), 2 = subadults (9 months to 2.5 years, n = 26), 3 = young adults (2.6 years to 5.5 years, n = 22), 4 = middle aged adults (5.6 years to 10.5 years, n = 28) and 5 = old individuals (> 10.5 years, n = 14); the low number of unweaned cubs, three of which were from the same pride, may influence the results. Data were taken from all sampled lions - both Serengeti and Crater - across the study period. Bars represent standard errors of the mean.

c) Taeniidae



d) Hookworm

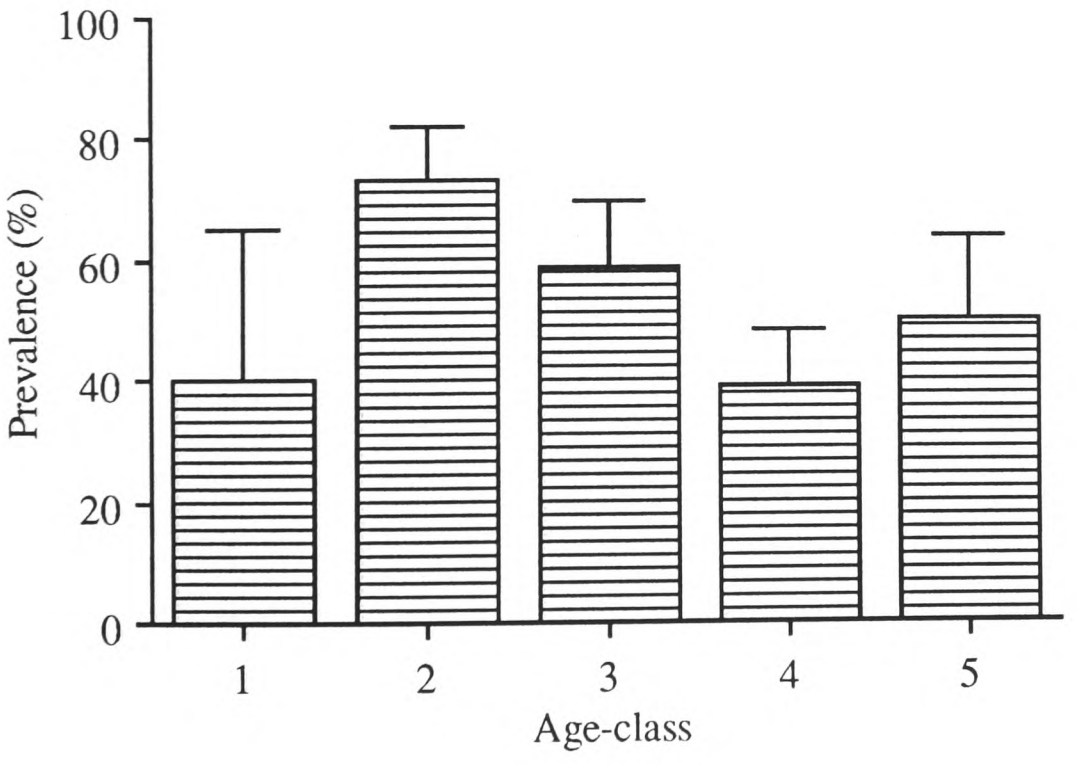


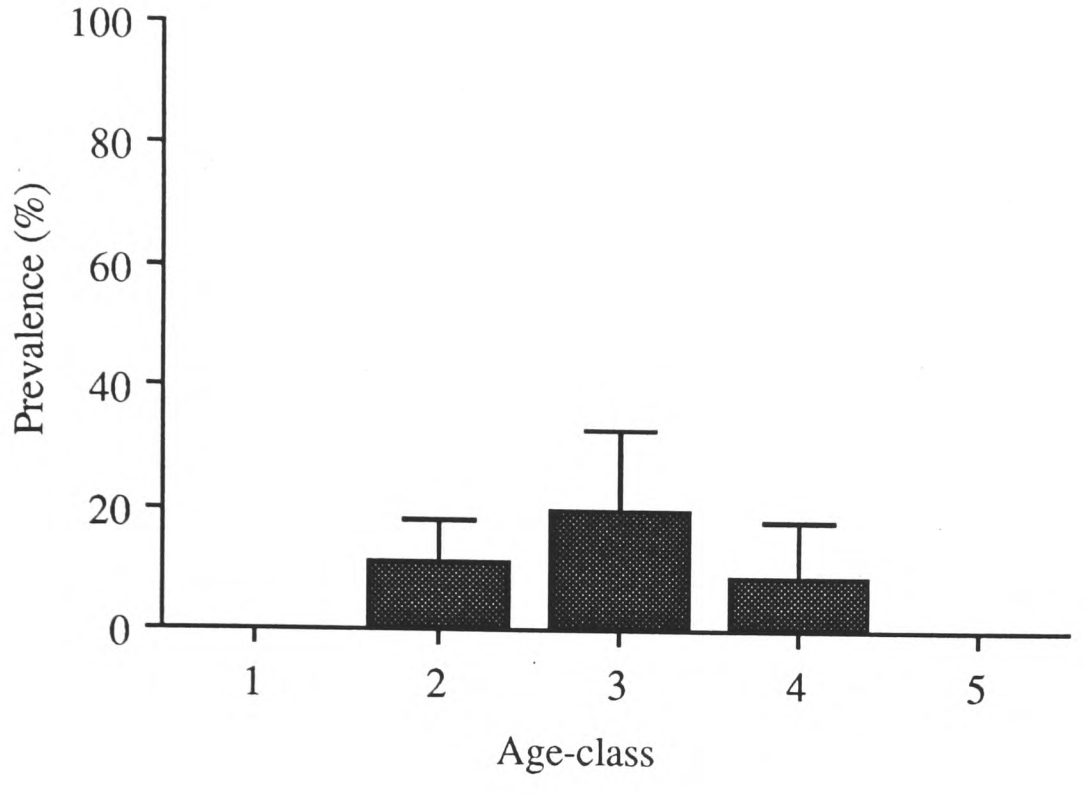
Figure 8.8. Age-prevalence profiles.

e) Helminth larvae

e) Spiruid egg

Figure 8.8. Age-prevalence profiles.

g) *Giardia* sp.



h) *Sarcocystis* spp.

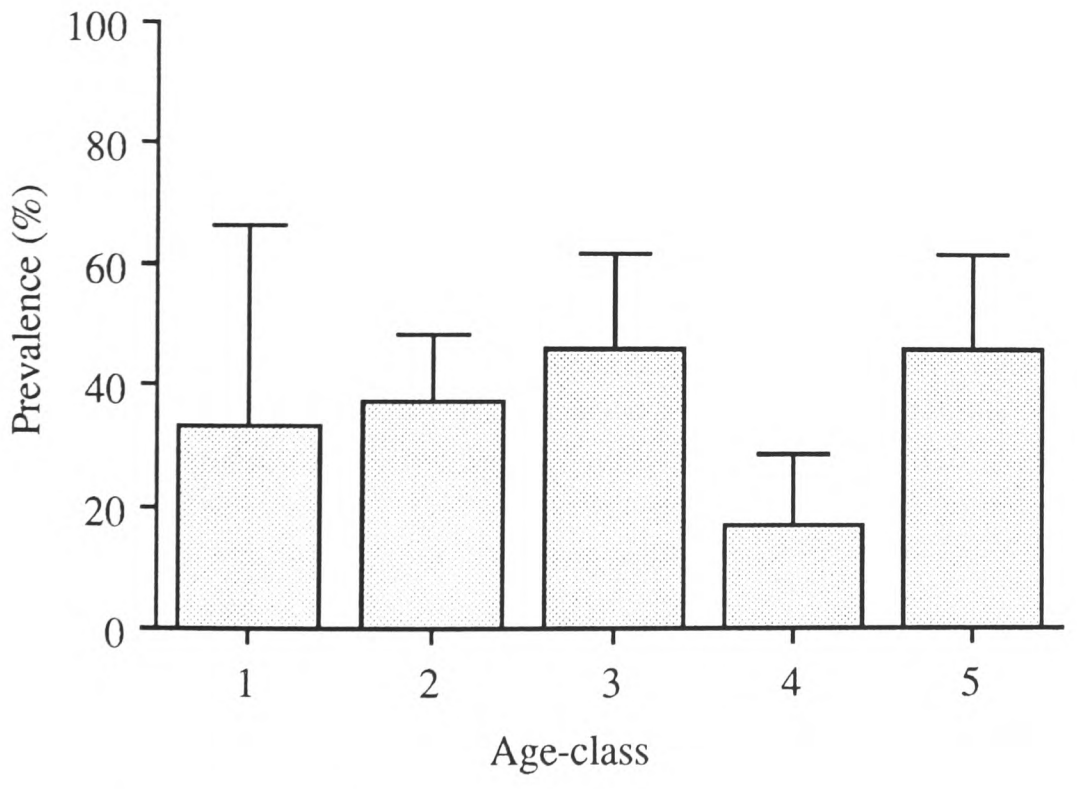
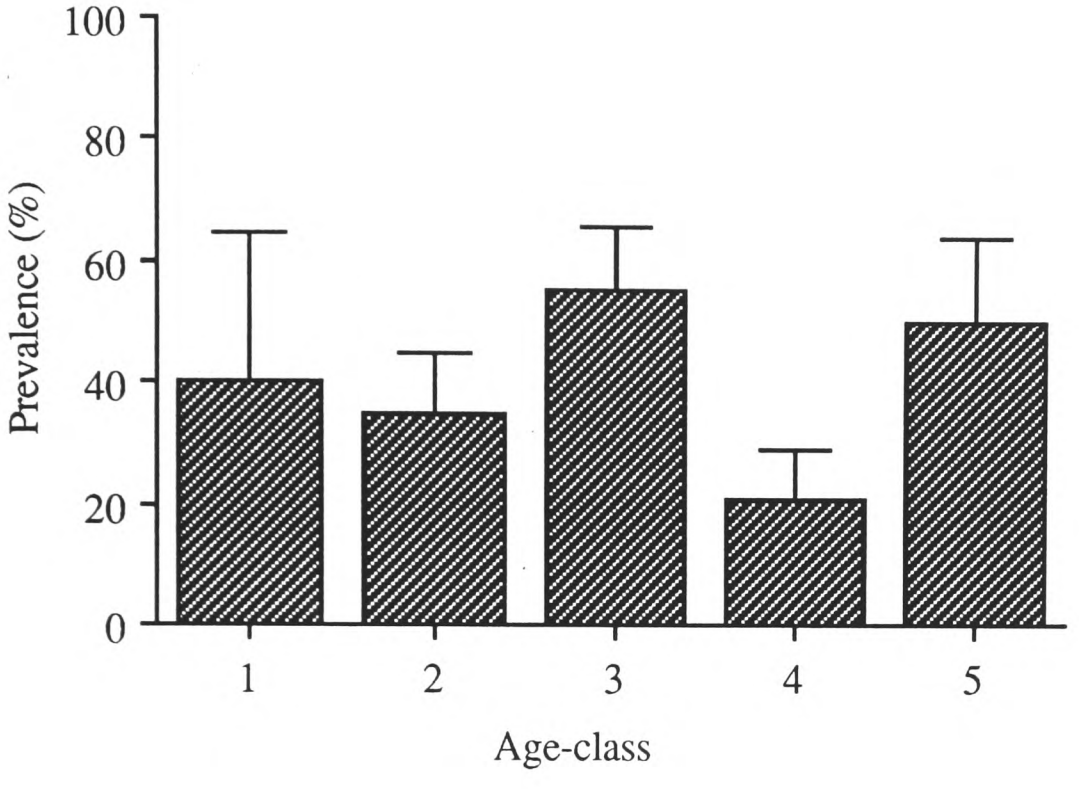


Figure 8.8. Age-prevalence profiles.
g) Numbers of individuals analysed : 1(n = 3), 2 (n = 19), 3 (n = 10), 4 (n = 11), 5 (n = 8)
h) Numbers of individuals analysed : 1(n = 3), 2 (n = 19), 3 (n = 11), 4 (n = 12), 5 (n = 11)

i) Paramphistome-like parasite



j) *Trichuris* sp.

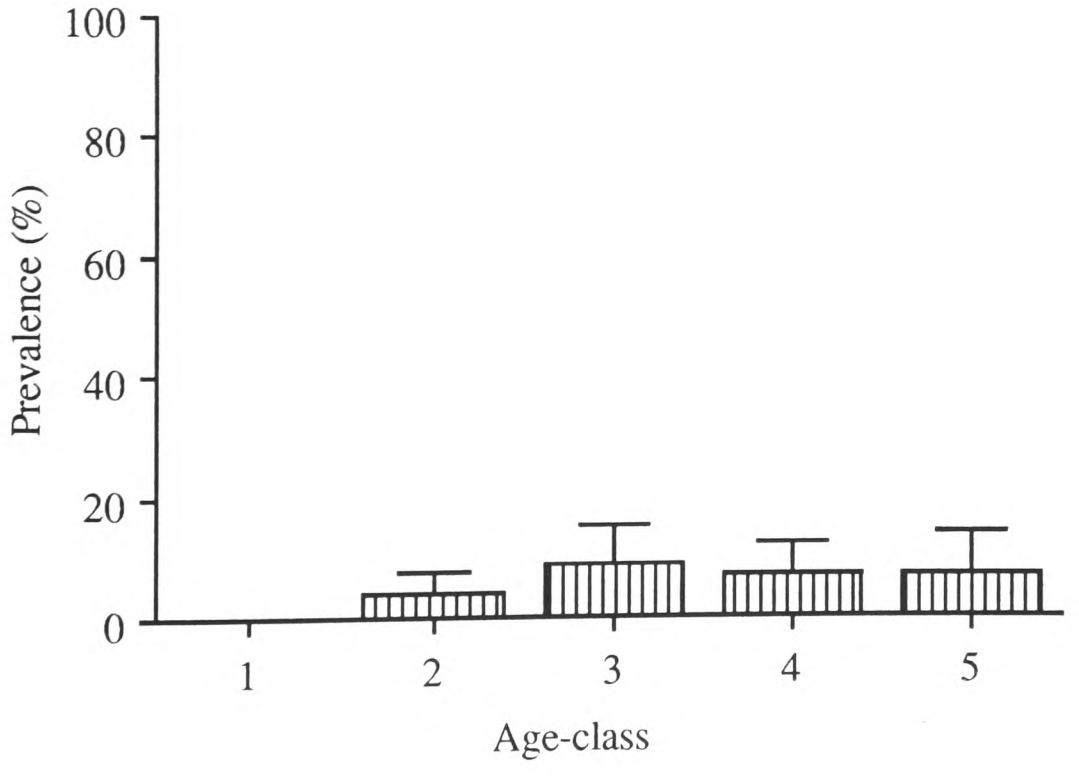


Figure 8.8. Age-prevalence profiles.

k) Trichostrongyloid parasite

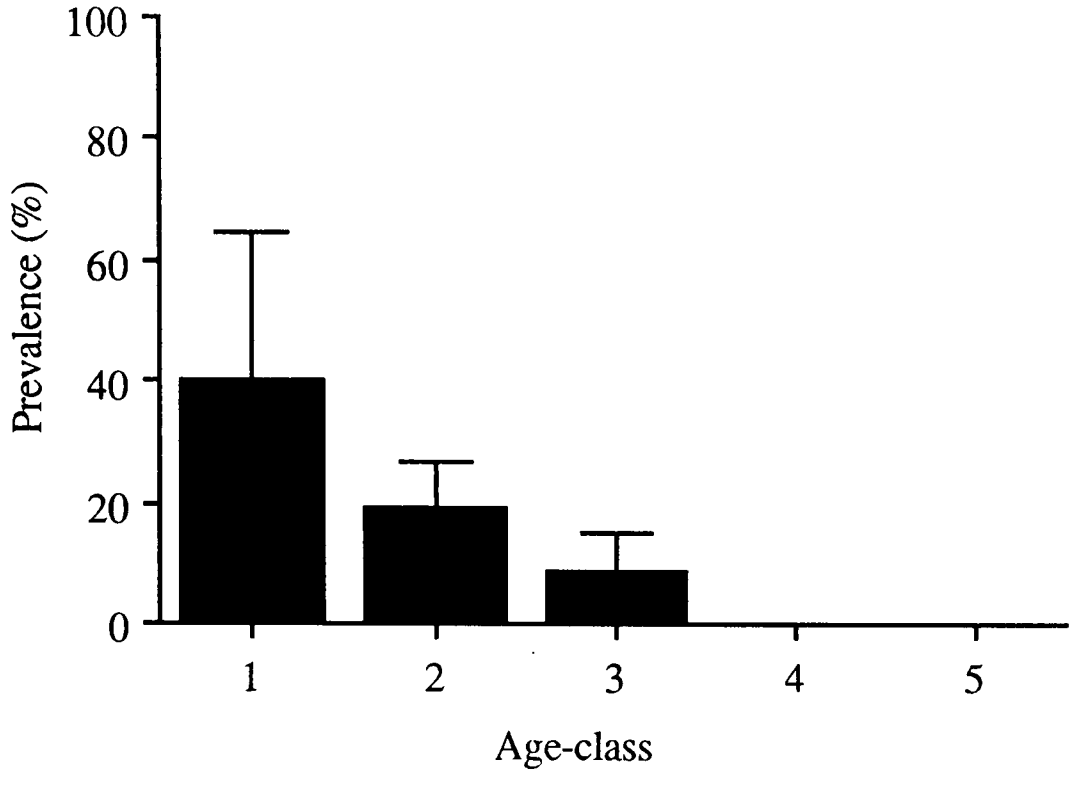
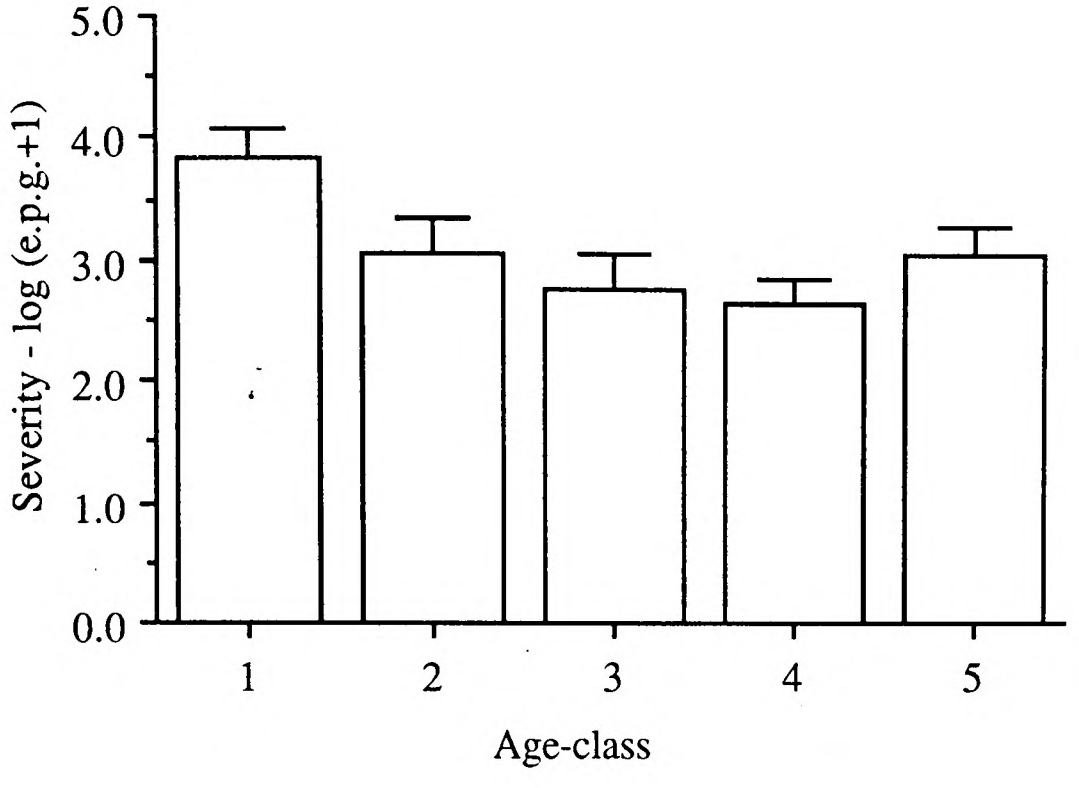


Figure 8.8. Age-prevalence profile.

a) *Spirometra* spp.



b) Taeniidae

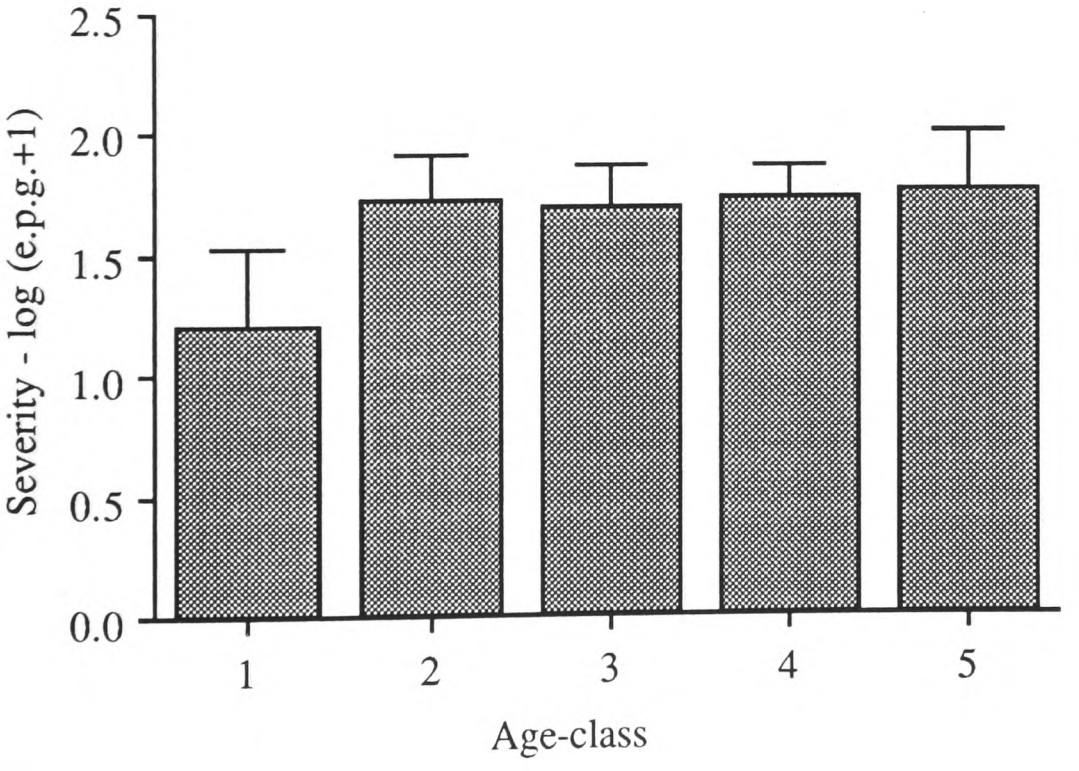
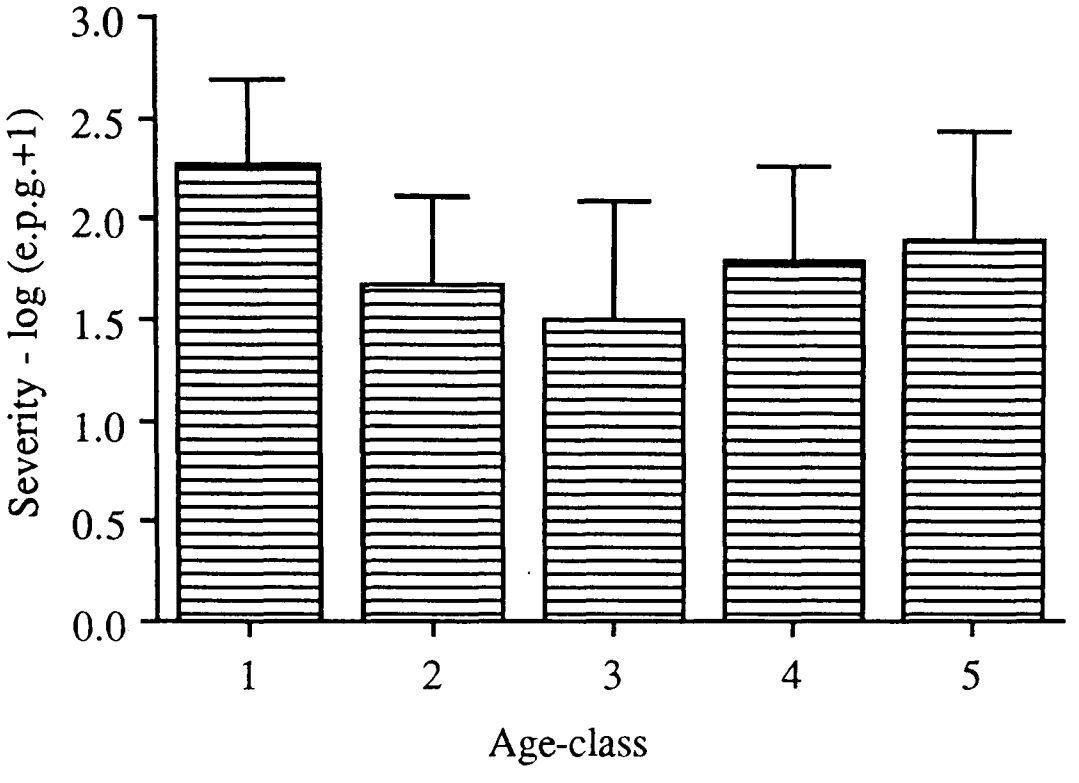


Figure 8.9. Age-severity profiles.
Age-classes were categorized as for age-prevalence curves. Bars represent standard errors of the mean.
a) Number of individuals found infected were: 1 (n = 5), 2 (n = 14), 3 (n = 16), 4 (n = 19), 5 (n = 8).
b) Number of individuals found infected were: 1 (n = 2), 2 (n = 15), 3 (n = 16), 4 (n = 14), 5 (n = 9)

c) Hookworm



d) Total Helminth Load

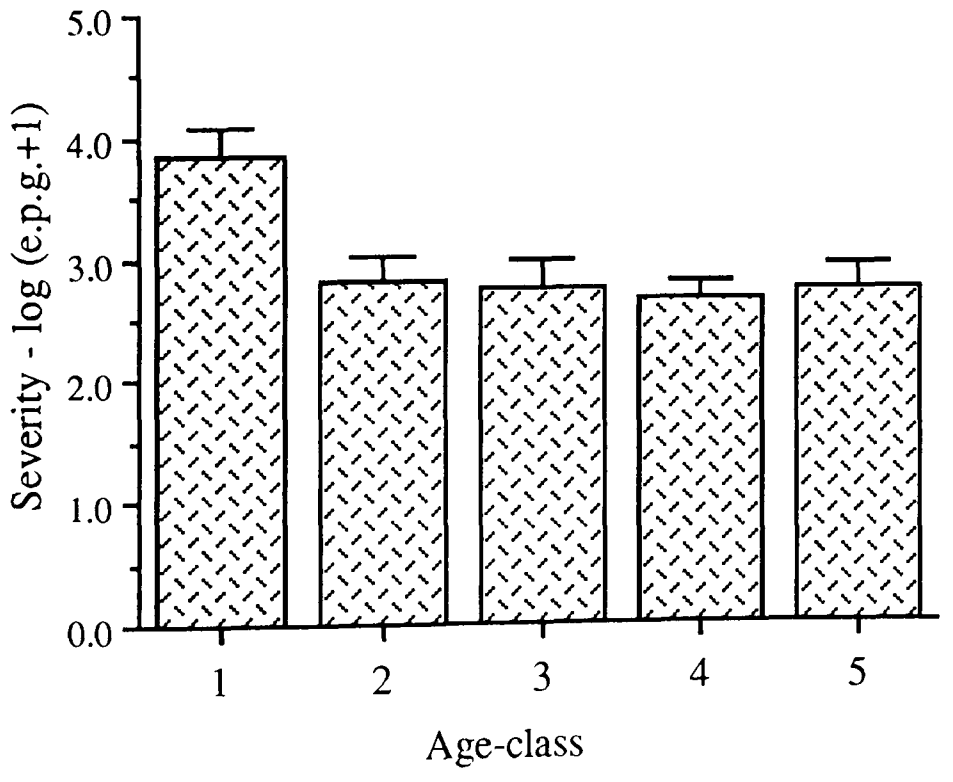


Figure 8.9. Age-severity profiles.
c) Number of individuals found infected were: 1 (n = 2), 2 (n = 19), 3 (n = 13), 4 (n = 11), 5 (n = 7)
d) Number of individuals found infected were: 1 (n = 5), 2 (n = 24), 3 (n = 21), 4 (n = 26), 5 (n = 14)

CHAPTER 9

General Discussion

This study investigated the epidemiology of intestinal parasites in well known natural populations of two species of social animals. Information about the parasite burden of lions and baboons was collected and the factors contributing to the heterogeneity of infection were studied. The discussion will consider the relative importance of these factors, the implications of this research for conservation and suggest further research.

So far, little is known about host-parasite interactions and the effects of parasites on their hosts in these two species. Only very general information exists about lion-parasite interactions mainly from surveys of captive lions in zoos or anecdotal reports of parasite infection of individual animals (Dittrich 1958; Tscherner 1974; Hasslinger *et al.* 1982). The situation is slightly better for baboons, where several surveys of wild baboons have been undertaken (see review in chapter 4). Information about the effects of parasites on baboons originates mainly from research of captive baboons (Vagtborg 1965; Hunt *et al.* 1970; Orihel 1971; Göltenboth and Klös 1977; Owen 1992). Although studies on captive animals cannot be readily compared to field studies, they do indicate a detrimental effect of parasites on their hosts and thus support the idea that parasites also have an impact on natural host populations. It is important to understand the extent to which parasites harm their hosts in nature in order to evaluate how significant a role parasites may play in host evolution. The effects of parasites on the fitness of host species could not be considered in this study because data on the pathogenicity of wild lion and baboon parasites are lacking. However, all of the baboons and almost all the lions were found to be infected in this study providing some indication that parasites may be an influential part of the ecology of these species.

9.1 RELATIVE IMPORTANCE OF FACTORS AFFECTING PARASITE BURDENS IN WILD BABOONS AND LIONS

In accordance with the majority of other field and experimental studies, the frequency distribution of parasite species in both populations was overdispersed, presumably reflecting differences in individual exposure, innate susceptibility to infection and/or acquired immunity. This study attempted to identify factors which contributed to the overdispersed distribution of parasites. Spatial differences were the predominant component of the variation in parasite burdens found among the populations studied. For both host species, parasite distribution was noticeably patchy across prides and troops (as shown in chapters 5 and 8). Almost all parasites exhibited some form of spatial variation in prevalence and/or intensity of infection. The spatial differences could be due to habitat

differences, social and age structure of groups, behaviour or genetics. Because other factors, such as genetics or group size, did not show a considerable independent effect, it seems that environmental factors/exposure may be the most important influence on parasite burdens, at least in the short-term. Other factors may be more influential in stable systems over longer time spans. Environmental factors include, for instance, different densities of infective stages in the environment together with exposure to prey and the effect of habitat on transmission opportunities for parasites.

The results here also showed that patchiness in distribution occurs on a comparatively small scale. This is especially demonstrated by the observations of the levels of infection among the different baboon troops whose home range midpoints are approximately 1 km apart. Recent studies have disclosed similar findings for other host species (Forrester *et al.* 1988; Anderson *et al.* 1993; Jaenike 1994).

Habitat can be confounded by genetics, because individuals who are related often live in close proximity. It has been difficult to disentangle these factors in humans studies (Forrester *et al.* 1988; Forrester *et al.* 1990; Anderson *et al.* 1993; Chan *et al.* 1994). It has been suggested that susceptibility to parasite infection may be related to the degree of genetic uniformity, so that more homozygous individuals may have higher parasite prevalences or intensities (May 1988; O'Brien and Evermann 1988). The two lion populations differed markedly in their levels of heterozygosity, so that if genetics was influential, one might expect a bigger difference between Serengeti and Crater lions than within the populations. This prediction could not be substantiated in this study. The differences in infection between the inbred lions in the Ngorongoro-Crater and the outbred Serengeti lions did not show up as any more important than differences among prides within the Serengeti and across both areas. The one parasite, *Spirometra* sp., which was found to differ in prevalence between Crater and Serengeti might do so because of different habitat and/or transmission opportunities rather than of genetics. Also, correlations between heterozygosity and parasite burdens were only significant for one parasite taxon, Taeniidae, and the result has to be treated with caution. This suggests further that genetics may not be of marked importance in determining levels of infection in the two lion populations. There was no evidence that more closely related baboon troops shared a more similar parasite burden.

Age structure and group size were analysed for their association with variation among groups, but their impact appeared to be limited. Age structure was controlled when analysing for spatial differences and did not, in any case, differ markedly among baboon troops. Group size was not associated with severity and intensity of infection in the baboons and only marginally significant for prevalence of one parasite taxon (chapter 5). Pride size of lions was correlated with severity of one parasite (chapter 8). However, pride size may be confounded with habitat since the largest prides lived in the same habitat.

Behaviour may influence spatial differences in parasite burden. Male baboons, for instance, migrate between troops and may be carrying parasites with them. On the other hand, behaviour does not vary markedly across groups, making behaviour an unlikely explanation for differences among baboon troops.

Spatial factors were not only important in influencing parasitic infection on its own in this study, but also interacted with other factors such as age and season in the baboons (chapter 5), so that the effect of these factors can vary according to area. These interactions may explain the often conflicting results found in other studies of one host species in different habitats, as in the comparison of baboon parasite intensities from different areas (chapter 6) during the wet or dry season. Interaction between habitat and levels of infection may also explain varying shapes of age prevalence profile from different areas (e.g. Sen-hai and Wei-xia 1990).

Temporal variation in parasite infection was present in both host species. Clearly, levels of infection fluctuate over time. For some of the baboon parasites, there is an indication that this is connected with seasonal changes (chapter 8). For the lion parasites, evidence for similar seasonal dynamics is less clear. Only *Spirometra* spp. showed a season-related pattern, although this was dominated by levels of infection in one particular season. Further monitoring over extensive periods of time will reveal the validity of the seasonal changes observed here. For both host groups, some parasites (i.e. *Sarcocystis* spp. and spiruid eggs in lions, *Trichuris* sp. in baboons and lions and "amoebae" in baboons) showed temporal, but not necessarily seasonal variation, perhaps indicating that the system is not at equilibrium. Increases in prevalence of infection in the baboons over the course of the study period, provided that they are not an artefact of sampling, could also imply that conditions (for instance environmental situation and/or population density) for the baboons are changing and they are acquiring higher levels of infection, which was further supported by the long-term data.

Associations between age and prevalence and intensity of infection do contribute to the overall heterogeneity, even though few significant differences between age groups could be found. Both host species showed rapid acquisition of most common parasites in the young age-classes, implying high transmission rates. This is probably due to the fact that they are exposed to these parasites from birth onwards, and would not have acquired immunity at this stage in their lives. The baboons generally showed age-infection profiles with apparent age-related patterns which looked similar to curves for the same parasites in humans. With some exceptions, for instance the age-prevalence curve for Coccidia, many of the lion parasites did not show distinct age-related patterns. The differences in associations between age and infections in lions and baboons may be explained in two ways: differences in immune response and behaviour. The kind of immune response

which is evoked by an infection is dependent on the particular parasite and host species. For example the cestodes, dominant among the lion's parasites, may not elicit noticeable immune responses (Rickard 1983; Cox 1993), and therefore age patterns are less obvious in the lions than in the baboons.

Apart from acquisition of immunity, feeding behaviour may also exert an influence on parasite burden. A large percentage of the lion parasites were acquired via prey. Little differences in the type of prey eaten could be observed between most lion age-classes with the exception of the very young. Only the amount consumed differed. The observation that very young age-classes (although represented by small sample sizes) showed the most distinctive difference in infection compared with the other age-classes suggests that behaviour connected with exposure is important in determining the changes in parasite burden with age. The negligible differences in feeding patterns in the other age groups may partly explain the lack of distinct age-related patterns in the lions.

Overall, sex differences do not seem to be influential in causing heterogeneity of infection on a short-term basis. Convincing sex differences in levels of infection were found for one baboon parasite only, *Schistosoma mansoni*. Apart from this one example, females did not appear to be less infected, as might be expected from general trends in sex differences of infection in other host species, for instance humans (Bundy 1988a). Reproductive status only influenced one parasite in the lions and one in the baboons. Other effects may have not been observed due to differences so subtle that larger sample sizes are needed to detect them. Only the variation of *Trichuris* sp. infection in female baboons during different reproductive states gave any support to the hypothesis that the immune system can be suppressed during pregnancy and lactation (Behnke 1990; Brabin and Brabin 1992), but higher parasite egg output was measured during lactation, not during pregnancy.

The impact of social rank on parasite infection was studied to investigate associations of behaviour and parasite burdens. In baboons, the evidence for correlations between social status and parasite infection was equivocal. In lions, which form prides with no hierarchy, rank is not important.

Interactions between parasites species within a host may have an impact on the overall structure of a parasite assemblage and influence parasite distribution in the host population (Esch *et al.* 1990). Parasite infracommunities can range from isolationist to interactive, and most mammalian parasite infracommunities examined so far have been found to be isolationist, with few interactions (Sousa 1994). The results in this study, in which not many parasite-parasite interactions could be detected, were in accordance with these findings. However, the low number of interactions may also be due to weaknesses inherent in a field study, where confounding variables, such as time and date of sampling and hormonal status of animal and relatively low sample sizes, may obscure interactions.

Additionally, determination of interactions by egg counts alone is not the best method, as egg counts are imperfect indices of adult parasite burden. Furthermore, interactions which resulted only in reduced growth and displacement of one parasite species at one site in the host by another parasite species, but not in a reduction in parasite egg output, would not have been detected.

Even when all the above factors are considered they do not explain most of the heterogeneity found in the infections. Some of this unaccounted variability is due to measurement errors and the relatively crude method of determining infection. As discussed in chapter 2, egg counts, although meaningful, can be imperfect and not always reliable correlates of parasite burden. In addition, the study could not consider factors like nutrition and individual immune systems. If heterogeneity is related to exposure even on very small scales, then within-group differences in exposure, which are almost impossible to measure, could effect parasite distribution.

9.2 CONSERVATION AND DISEASE

Disease is a significant factor affecting populations of wild animals and hence is an important consideration for conservation (Jones 1982; Henderson 1985; Dobson and May 1986; May 1988). With increasing partitioning of the natural environment, and the consequently higher densities of host populations, the impact of disease has become even more prominent. Usually, this has come to notice when an endangered population has been further decimated by the occurrence of an epidemic, for example, the Black-footed ferret was extirpated in the wild by canine distemper (Thorne and Williams 1988). The importance of disease also has been recognized when large numbers of animals have died as a result of an epidemic. Rinderpest, introduced to East Africa in 1889, which killed a high number of African buffalos (an estimated 90% died; Dobson and May 1986) and the biting-fly epidemic in 1962 which decimated the Ngorongoro lion population (Packer *et al.* 1991) are examples of such epidemics. Most of the diseases cited in this context are microparasitic diseases. However, macroparasites, which include the majority of the parasites examined here, can cause problems as well. An example is the monogenean *Nitzschia sturionis* that decimated the indigenous sturgeon (*Acipenser sturionis*) population after introduction of its host *Acipenser stellatus* in a new freshwater habitat in Russia in the 1930's (Dogiel 1964). Both host species studied here are not endangered and no unusual outbreaks of any particular disease were observed during the study period. However, the unexpected incidence of canine distemper in the Serengeti this year (1994) which is known to have killed approximately 85 of the 250 monitored lions (C.Packer, personal communication; Miller 1994; Morell 1994a; Morell 1994b), shows unmistakably how

sudden the occurrence of disease can change host population structure and also how disease must be considered in any conservation effort.

So how do studies such as the one undertaken here relate to conservation? To evaluate problems caused by disease, baseline data are first needed on the normal infection levels of populations, including age-prevalence and age-intensity profiles (Scott 1988). This study attempts to provide this information for lions and baboons. Changes in infection levels or the acquisition of new parasites, which are not immediately expressed in epidemics, may help predict problems developing in a natural system. Although group size did not have a strong effect on parasite infection in this study, the similar group sizes in the populations observed may have prevented disclosure of any such effect. If animals are forced to live in artificially high levels of density rates of transmission may increase. This situation could arise once human settlements completely surround Gombe Stream National Park and the baboons are forced to stay within the park boundaries. At the moment, infection levels do not appear to threaten the Gombe baboons. However, this study shows an increase in parasite burdens in comparison with previous studies (chapter 4; McGrew *et al.* 1988; Murray 1991). The role of disease must also be considered when hosts are introduced into new environments, as they will both introduce their parasites to the novel environment, and be exposed to novel parasites.

The two host species in this study illustrate two conservation related problems. The negative effects of inbreeding due, for example, to small isolated natural parks; and problems due to contact with humans. The results of the lion study imply that a certain degree of inbreeding may be of no practical short-term importance in a field situation, provided that environmental conditions are favourable. However, the suggested importance of the environment in determining levels of disease implies that a change in environmental conditions can lead to an increase in parasitic infections. Arguably, the lions in the Ngorongoro Crater were only equally healthy to the Serengeti lions because they were living in a benevolent environment.

Primates, because of their phylogenetic relatedness, can be endangered by human diseases as illustrated by the polio epidemic among the chimpanzees in Gombe Stream National Park (Goodall 1986). However, in this study there was little indication, with the exception of schistosomes, that baboons suffered from infections as a direct result of living close to humans. However, no comparisons with baboon populations with little or no human contact were made. Because the baboons share several parasites with humans, even without human contact, they may have already been infected with them and therefore no noticeable effect may have been generated by interspecific contact. As the life cycle of some parasites, such as *Trichuris trichiura, Necator americanus* and *Schistosoma mansoni* may be maintained in both humans and baboons, an overall higher host density and possibly increased transmission rates for both species may result from close contact.

Future research is needed to clarify the effects of human contact on parasite infection of primates.

9.3 DIRECTIONS FOR FURTHER RESEARCH

To investigate the important questions in the field of host and parasite biology, collaborative efforts from different biological disciplines are necessary. 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 *et al.* 1985; Hudson 1986; Hudson and Dobson 1991; Dobson and Hudson 1992). Long-term field studies could yield more information if individually known individuals were monitored, making it possible to relate parasite burden to reproductive success, for instance, and to shed additional light on the question of the parasite's role in controlling host abundance. Continuing to monitor parasite infection in the lion and baboon populations studied here could give information about fluctuations of parasites in relation to changes in host populations. It would also be conceivable to conduct more field experiments attempting to keep certain animals free of parasites for a length of time to see whether there is a noticeable difference in fitness in comparison to the naturally infected animals, as has been tried for a period of 100 days in the study on sheep and their parasites of St. Kilda (Gulland 1992) or on red grouse in Scotland (Hudson *et al.* 1992). However, it is not easy to administer antihelminthic drugs to lions and certain constraints in the code of Gombe Stream National Park make it difficult to experiment with baboons there, even though it is practicable. Such research could be carried out within the frame-work of a similar baboon study or with different mammals. Continued monitoring of the lion population would also yield more information on the long-term effects of inbreeding by measuring whether the Crater and Serengeti lions continue to show no significant differences in infection. Because the Crater lions went through a bottleneck fairly recently, any effects of inbreeding may just be beginning or have not yet emerged. The influence of inbreeding may be most apparent when novel diseases are encountered. The recent outbreak of canine distemper in the lions of the Serengeti was such a novel disease. It has not yet reached the inbred Ngorongoro Crater lions, so no comparisons between the two populations could be made about the effect of inbreeding on susceptibility to this disease.

The canine distemper epidemic in the Serengeti could provide information on predisposition to infection and interaction between parasites and pathogens. Data on parasites of the lions in the Serengeti need to be reanalysed with a view to the recent deaths to see whether any associations between mortality and previous parasite infection emerge. The epidemic was accompanied by an increase of *Theileria* infections (Miller 1994). As

this may be due to the stressed and subsequently weakened immune systems of the lions, similar increases in intestinal parasite infection should also be tested.

To gain a better understanding of host-parasite interactions, more studies are needed that incorporate genetics. For the lions, a more detailed analysis of the MHC is now technically possible that could allow a more accurate genetic description of each host individual. In general, little is known of parasite genetics (Nadler 1987). So far, a problem in determining helminth genotypes within or among host populations is the acquisition of sufficient adult worms from several hosts. Recent developments in molecular biology such as polymerase chain reaction (PCR) may soon make it possible to distinguish between different genotypes of parasites by analysis of their eggs (Gasser *et al.* 1991). This technique could be employed to show whether the genotype of a parasite species varies within one area or between host populations. For instance, genetical analysis could show whether the parasites shared by baboons and humans are the same strain. It has also been suggested that genetic variability in the parasite may cause spatial heterogeneity in prevalence, intensity and severity even on smaller distances. One example where this appears to be the case is the patchy distribution of *Schistosoma mansoni* in rats living in different discontinuous patches within approximately 20 km in Guadeloupe (Théron *et al.* 1992). The occurrence of different parasite strains is an unlikely explanation for the spatial heterogeneity of infection found in the Gombe baboons, because of their contact with neighbouring troops. However, it still remains to be tested whether troops are infected with the same parasite strains. Genetical analysis could also be included in long-term field studies. This could make it possible to monitor genetic changes in the parasite and host population and thus shed more light on the importance of parasites in maintaining host genetic diversity.

A further line of research should involve comparative studies within the same host species and between different host species; both require more data. To further understand the effect of exposure and habitat on parasite profiles, detailed studies of the infections of the one host species in different habitats are necessary. It has been suggested that better provisioned baboons might be less parasitized (Eley *et al.* 1989). However, this conclusion has been based on only two troops, and therefore needs confirmation. Likewise, it has been stated that baboons which live in higher altitudes show higher intensities of infection but less species richness (Appleton *et al.* 1991). Again, this has only been supported by a comparison of two troops and references from previous surveys that were not conclusive. It has been proposed that directly transmitted microparasites may afflict more communities of animals which live in dense proximity whereas solitary animals may be more infected with macroparasites (Anderson and May 1991). Parasite data on social predators such as lions could be compared with solitary feline predators with

the inclusion of microparasites, such as blood protozoans, into the list of parasites assayed or, likewise, social and solitary primate species could be compared.

Clearly, more field data are needed to evaluate the influence of parasites on the ecology and evolution of populations of large mammals in nature. Field studies can pose considerable logistic problems and data can be difficult to interpret because of the many confounding variables. However, with the right planning and statistical methods such studies can yield meaningful results. Moreover, they are of pivotal importance in assessing the applicability of theoretical and experimental results in the real world.

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