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International Journal for Parasitology 35 (2005) 11–18
www.parasitology-online.com

Epizootiology of blood parasites in an Australian lizard: a mark-recapture study of a natural population

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Received 16 July 2004; received in revised form 15 September 2004; accepted 17 September 2004

Abstract

The dynamics of a naturally endemic blood parasite (*Hepatozoon hinuliae*) were studied in a lizard (*Eulamprus quoyii*) host population, using 2 years of longitudinal data. We investigated how parasite abundance in the population varied over time, examined whether certain host sub-populations were more prone to infection, and compared parasite loads in relation to host reproductive behaviour. We recorded blood parasite infections of 331 individuals, obtained in 593 captures. Prevalence (the proportion of the host population infected) of blood parasites was high; approximately 66% of the lizard population was infected. Probability of infection increased with host age and size, but did not differ between the sexes. Within individuals, parasite load (the intensity of infection within individuals) did not vary over time, and was independent of host reproductive behaviour. Parasite load was significantly higher in males compared to females.

Keywords: Haemogregarine; Wildlife disease ecology; Hepatozoon

1. Introduction

The consensus from research into the evolutionary ecology of host–parasite relationships is that parasites can be important selective agents affecting host population biology, ecology and evolution (Dawkins, 1990; Gulland, 1995; Morand and Poulin, 2000; Hudson et al., 2002). Parasitic diseases can pose major threats to endangered animal populations and have serious economic impacts on agriculture and human public health, and therefore, research into host–parasite ecology is critical (Cleaveland et al., 2002; Galvani, 2003). However, knowledge of how hosts and parasites interact in natural systems remains limited, and consequently, international bodies like the World Health Organisation have advocated an increased study of host–parasite ecology in wild host populations (Real, 1996; Schall and Pearson, 2000).

Parasite abundance can vary in response to numerous factors, both in terms of prevalence, defined as the proportion of the host population that is infected, and parasite load, which is the number of parasites in an individual host. Factors that can influence parasite abundance include host sex and age (Schall et al., 2000; Smallridge and Bull, 2000), host reproductive effort (Norris et al., 1994; Nordling et al., 1998; Veiga et al., 1998), host condition and physiology (Salvador et al., 1997; Appleby et al., 1999; Dowell, 2001), vector biology (Sol et al., 2000; Readon and Norbury, 2004), and host density (Arneberg et al., 1998). Abiotic factors, such as temperature and rainfall, can also influence parasite abundance (Bennett and Cameron, 1974; Forbes et al., 1994; Fuller, 1996; Bajer et al., 2001).

Studies of lizards naturally infected with protist blood parasites have been important in the development of our understanding of host–parasite evolutionary ecology (Keymer and Read, 1991; Schall, 1996), but have focused mainly on malaria parasites (*Plasmodium* spp.) in the Americas and West Africa (Schall, 1996). Some work has examined unspecified haemogregarines (protist blood parasites) in *Lacerta vivipara* (common European lizards; Sorci, 1995), and on *Haemolivia mariae* in *Tiliqua rugosa* (Australian sleepy lizards; Smallridge and Bull, 2000, 2001).

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doi:10.1016/j.ijpara.2004.09.005

However, sampling times of these studies were seasonally restricted: Sorci (1995) reports parasite loads 1 year after initial capture, and Smallridge and Bull (2000) only examined lizards during the main activity season.

Here, we examine the dynamics of a previously unstudied lizard–parasite system: *Eulamprus quoyii* (Australian eastern water skink), and the haemogregarine *Hepatozoon hinuliae*. We determine whether parasite abundance is affected by host age, size and sex, and use a 24-month mark-recapture study to examine temporal changes in parasite abundance at both individual and population levels. In addition, we examine whether reproductive status of hosts influences parasitaemia in the wild.

2. Materials and methods

2.1. Study system

Eulamprus quoyii is a large (snout-to-vent length (SVL) up to 133 mm, mass up to 47.5 g) heliothermic skink, which gives birth to live young (litter size = 1–8). Unlike lizard hosts studied by Schall and co-workers, *Eulamprus*' life span is long (up to 8 years, Salkeld, unpublished data). Haemogregarines (Apicomplexa: Adeleina) are parasitic blood protists closely related to coccidia and *Plasmodium* (Perkins and Keller, 2001). Members of the *Hepatozoon* genus possess a complex life cycle; vertebrate hosts become infected with *H. hinuliae* when they ingest an infected haematophagous vector (Smith, 1996; Smallridge and Bull, 1999).

Lizards were caught at Blackdown Tableland National Park, a large sandstone mesa in central Queensland, Australia, from December 2000 to December 2002. The main study area was at South Mimosa Creek (149°04′E, 23°47′S, altitude 760 m), a creek running through open dry sclerophyll forest. The creek was seldom flowing, but small pools of water remained throughout the study period. Long-term average annual rainfall at Blackdown Tableland is 1150 mm/year but the total annual rainfall was less than half the average in 2001 and 2002 (582 and 567 mm, respectively). Animals were caught either by hand, with hand-held nooses or by using baited sticky traps (Downes and Borges, 1998). Animals were kept in cloth bags before being released at the original sites of capture within 24 h. Snout-to-vent length and tail length were measured to the nearest millimeter using a transparent plastic ruler. Body mass was recorded to the nearest 0.5 g with a 60 g-spring balance. All lizards were marked with a toe clip combination that allowed subsequent individual identification (no more than four toes were removed from an individual). Few ectoparasites were found on *E. quoyii* during this study (two ticks, Metastigmata, in total), so they are not discussed further.

Lizard blood was sampled from the caudal vein ventral to the tail vertebrae using heparinized syringes. Blood slides were air-dried, fixed in methanol, and then stained with Giemsa, and were examined under a microscope at ×400 magnification. Presence or absence of haemogregarines was determined by searching transects of the blood smear; if no haemogregarines were found after 6 min of searching the sample was considered uninfected. Parasite load was quantified by counting the number of infected cells within a minimum of 2000 red blood cells, and is expressed as a percentage.

2.2. Seasonal changes in host reproductive condition

Eulamprus quoyii mate during October, and females give birth in December and January. The allocation of host resources to fats and proteins in yolk and testes begins in April/May, occurs throughout the winter period of inactivity (May to September) and peaks by October (Veron, 1969). To study temporal changes in parasite load which might correspond to seasonal changes in host physiology, lizards were captured at four times in the year: (1) September, when lizards began to emerge after the winter period of inactivity; (2) October, when mating was taking place; (3) December, when females are pregnant and close to parturition; and (4) March, approximately 2 months after parturition, but before animals begin yolk and testes development. More males than females were captured during September because males emerge from winter inactivity before females. No lizards were caught in October 2002 because fire prevented access to Blackdown Tableland. We compared parasite load at first and second capture of marked individuals (Sorci, 1995). In addition, we compared parasite loads of individuals captured at intervals spanning important life-history transitions (e.g. before and after parturition in females).

2.3. Determining age classes

Although SVL is often indicative of age, lizard growth is continuous and highly variable, so assigning discrete age classes based on size can be difficult (Blomberg and Shine, 2001). However, in the Blackdown Tableland population females only give birth over a short period in summer (December/January), making it easy to recognise animals born in the previous summer based on both size and appearance. Size-at-recapture data for animals originally caught at known ages (i.e. 1- or 2-year olds) were used to generate size-at-age classes of lizards for animals up to 5-years old. Animals older than this were difficult to classify and were all included in the 'Age 6' class. All pregnant animals had SVLs of 90 mm or larger, therefore animals smaller than this size were considered to be juveniles and in the first year of life.

2.4. Analyses

Change in parasite load over time was examined by comparing parasite load in the same individuals at different captures, using product-moment parametric correlations. For analyses of parasite abundance at the population level, we used only the parasite load from the blood sample obtained at first capture of any individual. Parasite loads were not normally distributed, so Kruskal–Wallis and Mann–Whitney *U* tests were used to compare the parasite load among various host sub-populations. Chi-squared contingency tests were used to compare parasite prevalence in host groups, using the raw frequency data. Logistic regression was used to describe the change in infection probability with age for this population. Statistical analyses were performed on SPSS 10.0.

3. Results

During the 2-year mark-recapture study, we made a total of 593 captures, comprising 331 individuals: 146 males and 185 females. Overall, there was a high prevalence of blood parasites in water skinks at Blackdown Tableland with 218/331 (65.9%), of individuals infected. Parasite load differed among hosts; many hosts had small parasite loads, while some hosts were very heavily infected (Fig. 1).

3.1. Temporal variations in infection—within individuals

There was a significant correlation between parasite loads of individuals at first capture and when they were captured a year later ($n=10$, $r=0.85$, $P=0.002$). In fact, when combining data from all recaptures and disregarding the time interval between first and second capture (mean average duration between captures = 138 days, minimum 7, maximum 725), parasite load at each capture was highly and significantly correlated ($n=134$, $r=0.84$, $P<0.001$; Fig. 2).

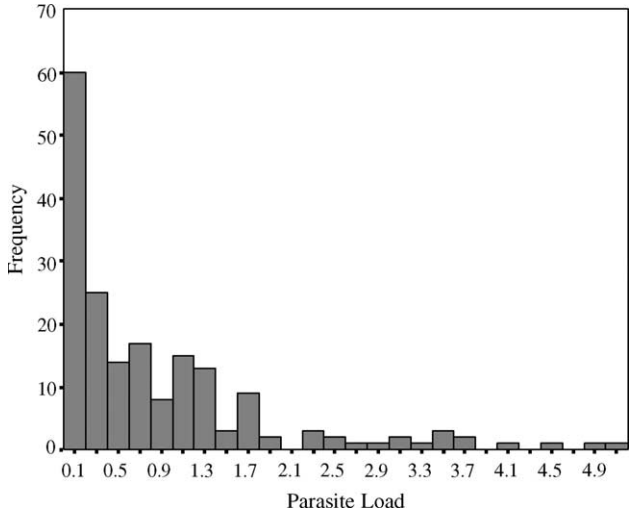

Fig. 1. Distribution of parasite loads (% infected cells) in naturally infected eastern water skinks, *E. quoyii*.

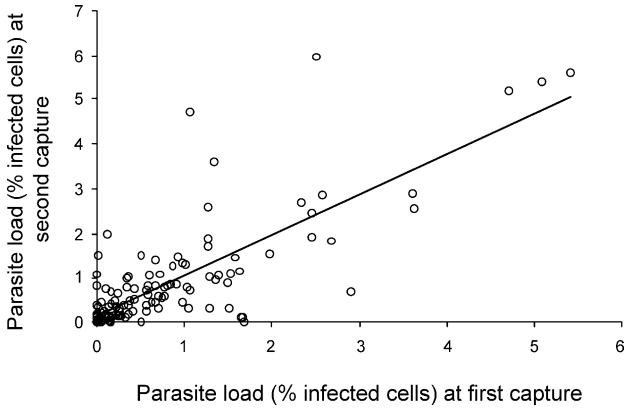

Fig. 2. Parasite load of *E. quoyii* individuals at first and second capture for all recaptured individuals (capture interval varies).

We compared male parasite loads between September/October (mating), and the following December (mating completed). Parasite loads of individual males captured in September/October and again in December were significantly correlated ($n=12$, $r=0.91$, $P<0.001$).

In females, individual parasite load was significantly correlated for animals captured in March (before vitellogenesis begins) and again in September/October (post-vitellogenesis; $n=5$, $r=0.82$, $P=0.013$). Similarly, between September/October (mating) and December (late pregnancy) loads were significantly correlated ($n=24$, $r=0.82$, $P<0.001$).

3.2. Temporal variations in infection across the population

Prevalence changed significantly in the water skink population over time ($X^2=36.4$, d.f. = 7, $P<0.001$; Fig. 3), and this was due to differences in prevalence in females ($X^2=31.0$, d.f. = 7, $P<0.001$) across time, in particular, low

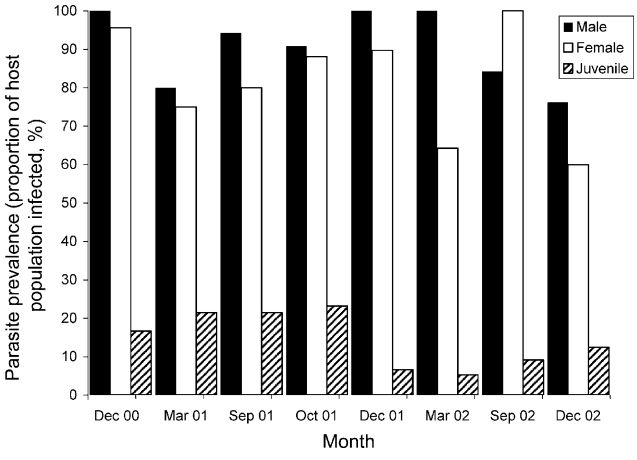

Fig. 3. Blood parasite prevalence in the Blackdown Tableland *E. quoyii* population over the two-year study period.

prevalence during March and December 2002. Prevalence did not change over time in males ($X^2=10.8$, d.f.$=7$, $P=0.15$) or juveniles ($X^2=8.32$, d.f.$=7$, $P=0.31$).

There was no significant temporal change in parasite load in the population during the 2-year study (adult infected males, Kruskal–Wallis test, $H=5.50$; d.f.$=7$; $P=0.60$; adult infected females Kruskal–Wallis test, $H=4.46$; d.f.$=7$; $P=0.73$; all adults, Kruskal–Wallis test, $H=4.99$; d.f.$=7$; $P=0.66$).

3.3. Host factors influencing parasite infection—sex

There was no significant difference in prevalence between males and females (no. of males infected$=$ 101/146$=$69.2%; no. of females infected$=$117/185$=$ 63.2%; $X^2=1.28$, $P=0.26$).

Parasite load was significantly higher in infected adult males ($n=95$) than in infected adult females ($n=105$; Mann–Whitney U test, $z=$-3.07, $P=0.002$). Mean parasite load in adult males was 1.2% infected cells and reached a maximum of 5.1%. In adult females, mean parasite load was 0.59% infected cells and reached a maximum of 3.6%. Parasite load was not significantly different in infected juvenile males and females (Mann–Whitney U test, $z=-1.41$, $P=0.16$).

3.4. Host factors influencing parasite infection—size and age

Logistic regression revealed that prevalence increased significantly with body size ($X^2=199.8$, $P<0.001$; Fig. 4). Grouping animals into age classes showed similar results, as juveniles (SVL$<$90 mm, 1-year olds) were significantly less likely to be infected than adults (SVL$>$90 mm; no. of juveniles infected$=$18/94$=$19.1%; prevalence in adults$=$ 200/237$=$84.4%; $X^2=127.41$, $P<0.001$).

Female parasite load was not related to age (Kruskal–Wallis test, $H=8.78$, d.f.$=5$, $P=0.12$), but there was a significant difference among males of different ages (Kruskal–Wallis test, $H=11.73$, d.f.$=5$, $P=0.039$) because 3-year-old males ($n=17$) had significantly higher parasite loads than 2-year olds ($n=17$; Mann–Whitney, $Z=-2.57$, $P=0.01$). Three-year-old males also had higher parasite loads than 4-year olds ($n=22$), although this difference only approached significance (Mann–Whitney, $Z=-1.78$, $P=0.074$).

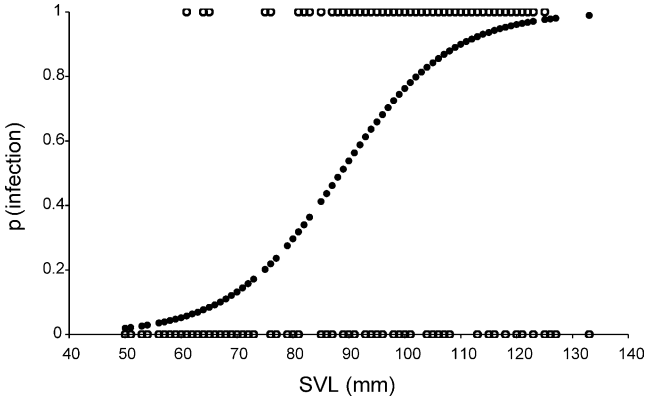

Fig. 4. The relationship between body size (snout–vent length; mm) and probability of blood parasite infection (prevalence) in *E. quoyii*. Open circles are actual data and filled circles are the probabilities of infection, determined by a fitted logistic regression to the data.

3.5. Evidence of self-curing

Out of 120 infected individuals that were caught at least twice, six individuals (all female) showed signs of clearing parasite infections, as parasite loads dropped to zero. There was a mean of 223 days (SD$=$152) between the previous positive measure of parasitic infection and the negative one. Mean parasite load, at the sample previous to the negative load, was 0.13% (SD$=$0.20). Two lizards, recaptured a third time, i.e. once more after being regarded as uninfected, displayed parasitaemia, although with parasite loads of 0.01% or lower. Recrudescence of infection in individuals with low parasite loads may indicate that detecting very low parasite loads was difficult, and subject to error, or it may indicate that these females were re-infected with the disease after having recovered from an infection.

4. Discussion

The number of *E. quoyii* infected with *H. hinuliae* was high, with over 65% of animals infected at Blackdown Tableland. Other lizard–blood parasite systems are typically much lower: malarial parasite prevalence in American lizards approximates 25%, but can reach 40% at tropical sites (Staats and Schall, 1996; Eisen, 2000); and only 11.5% of the Australian lizard *T. rugosa* are infected by the haemogregarine *H. mariae* (Smallridge and Bull, 2000). Many populations of *E. quoyii* in eastern Australian exhibit a prevalence of *H. hinuliae* comparable to that at Blackdown Tableland (Salkeld, unpublished data), suggesting that the parasite prevalence described here is not exceptional for this host–parasite system.

In our study, parasite loads remained constant in individuals, and showed no changes attributable to host reproductive behaviour. Admittedly, sample sizes of some before-and-after groups were small, but analysis of all recaptured lizards ($n=134$) showed that an individual's blood parasite loads were consistent, regardless of the sampling interval.

Persistent parasite loads may be a result of re-infection. However, *Hepatozoon* infections remained in four *E. quoyii* that were housed in captivity for 18 months (Salkeld, unpublished data). Because no blood-feeding invertebrates were present on the lizards or in the laboratory, and because captive lizards were fed on commercial cat food and captive-bred crickets, there was no possibility that these

long-lasting infections were due to new infection. Thus, it appears that *Hepatozoon* infections can be long-lived.

Blood parasite infections in other lizard species also remain reasonably constant after a peak following the initial infection (Bromwich and Schall, 1986; Sorci, 1995; Eisen, 2000; Smallridge and Bull, 2001), and similar stability occurs in blood parasite infections of birds (Dufva, 1996; Appleby et al., 1999). Some authors have suggested that stable parasite loads may be the result of parasites successfully evading a low-grade or inadequate reptilian immune system (Bromwich and Schall, 1986; Sorci, 1995). However, other studies have demonstrated that lizard hosts do have active immune systems (Saad and El Ridi, 1988; Saad et al., 1990), and possess the potential to control haemogregarine infections (Wozniak et al., 1996, and references therein). Furthermore, lizards and haemogregarines apparently can co-evolve, as lizard populations inhibit growth rates of local strains of blood parasites more than they inhibit non-local strains (Oppliger et al., 1999). Moreover, in experimental infections, mean parasitaemia of haemogreagarine blood parasites was significantly lower in lizards previously exposed to the parasite than in naïve hosts (Smallridge and Bull, 2001), suggesting that hosts may have built up some degree of immunity. Therefore, it seems that lizards are capable of mounting immune responses to haemogregarine infections. The presence of a competent immune system, combined with stable and persistent parasite loads within individuals, suggests that blood parasite load is a function of the individual host–parasite relationship, and this relationship is probably mediated by both genotypic and phenotypic variation.

Alternatively, the long life span of reptilian erythrocytes (600–800 days) compared to mammals and birds (Campbell, 1996) may mean that 2-year studies, or even 2-year life-spans, are not long enough to observe animals recovering from infection. In other words, if the actual infected red blood cells are not destroyed by the host's immune system, perhaps malaria parasites or *Hepatozoon* can persist in blood cells for longer than the lizards are studied or survive p.i. Previous studies have suggested that there is no evidence of self-curing of malaria parasites in lizards (Bromwich and Schall, 1986). There was evidence in this study of self-curing, although infections reappeared in two of six individuals, so apparent cures may actually be due to error in diagnosis at very low infection levels, or to reinfection. Continued monitoring of marked individuals within the Blackdown Tableland population would reveal the dynamics of long-lived infections in long-lived hosts.

The prevalence of blood parasite infections is often relatively stable for long periods (Bennett and Cameron, 1974; Eisen, 2000; Schall et al., 2000; Smallridge and Bull, 2000), and independent of seasonal climatic changes in temperature (Schall, 1986). In this Blackdown Tableland population, prevalence of blood parasites in males and juveniles did not change significantly over the course of our 2-year study, although prevalence in females was more variable. It is not clear what causes the prevalence in females to vary, since there was no consistent pattern in the variance (e.g. related to seasonal change or reproductive activity). Additional years of sampling would reveal whether this pattern of high female variance was consistent. Parasite prevalence appeared independent of patterns of rainfall; low annual rainfall in 2001 and 2002 did not significantly alter parasite prevalence compared to that in 2000.

Stable prevalence of blood parasites may be typical of long-lived hosts. Host–parasite systems that experience large fluctuations in prevalence may do so because of large variations in vector abundance, or host immunity, and such fluctuations may be more obvious in host populations with high turnover rates (Bennett and Cameron, 1974; Schall and Marghoob, 1995; McKenzie et al., 2001). *Eulamprus* lizards are long lived and, as parasite infections are also persistent, prevalence in adults will remain fairly stable once infection has occurred.

Previous studies have suggested that males may be more prone to parasitic infection than females due to the immunosuppressive action of testosterone (Poulin, 1996; Salvador et al., 1996, 1997; Schalk and Forbes, 1997; Veiga et al., 1998). For example, there is a male bias in blood parasite prevalence in mammals (Schalk and Forbes, 1997). However, there is no overall sex bias in blood parasite prevalence in birds, and in cases where a bias exists (e.g. *Haemoproteus* in breeding birds) females are more prone to infections than males (McCurdy et al., 1998).

In our study, prevalence of parasites was not significantly different between males and females of any age class. However, parasite load was significantly higher in adult males than in adult females. Higher parasite loads in males suggest that the immune response to *Hepatozoon* may be gender dependent. Testosterone may be responsible for this observed difference, although a study of malaria parasites (*Plasmodium mexicanum*) in western fence lizards (*Sceloporus occidentalis*) found little evidence to suggest that testosterone affects the parasite's life-history traits (Eisen and DeNardo, 2000). Interestingly, parasite loads did not differ between the sexes in juveniles, suggesting that hormonal differences in the adult sexes may indeed account for the higher parasite loads in males. Differences in behaviour of the sexes may also account for higher parasite loads in adult males. For example, males may consume more vectors than females, or have closer contact with conspecifics, which might raise the chance of encountering infected invertebrate vectors. The higher parasite loads in males may also be responsible for maintaining the parasite presence within the host population (Ferrari et al., 2004).

A review of the literature on reptile blood parasites shows that 3/7 studies demonstrate a higher prevalence of blood parasites in male lizards compared to females, but sex-dependent differences in parasite load have rarely been reported (Table 1). This phenomenon merits further attention.

Table 1
Literature review of sex-bias in blood parasite infection in lizard hosts

Host	Parasite	Effect	Reference
Aruban whiptail, *Cnemidophorus arubensis*	*Hepatozoon* sp.	Male bias in prevalence	Schall (1986)
Agama agama	*Plasmodium giganteum* and *P. agamae*	No bias	Schall and Bromwich (1994)
Western fence lizard, *S. occidentalis*	*P. mexicanum*	Male bias in prevalence, no bias in prevalence	Schall and Marghoob (1995) and Eisen (2000)
Anolis sabanus	*Plasmodium azurophilum*	No bias	Staats and Schall (1996)
Anolis gundlachi	*Plasmodium azurophilum* and *P. floridense*	Male bias in prevalence	Schall et al. (2000)
Australian sleepy lizard, *T. rugosa*	*H. mariae*	No bias in prevalence, parasite load higher in females	Smallridge and Bull (2000)
Eastern water skink, *E. quoyii*	*H. hinuliae*	No bias in prevalence, parasite load higher in males	This study

Table 2
Literature review of age-bias in blood parasite infection in lizard hosts

Host species	Parasite	Effect	Reference
Western fence lizard, *S. occidentalis*	*P. mexicanum*	Maximal parasite load higher in juveniles	Bromwich and Schall (1986)
		Adult bias in prevalence	Schall and Marghoob (1995)
European common lizard, *L. vivipara*	Haemogregarine	Adult bias in prevalence	Oppliger and Clobert (1997)
Australian sleepy lizard, *T. rugosa*	*H. mariae*	Adult bias in prevalence	Smallridge and Bull (2000)
Eastern water skink, *E. quoyii*	*H. hinuliae*	Adult bias in prevalence	This study

This study, similar to other studies on lizards (Table 2), demonstrated higher parasite prevalence in adults, that is, adult water skinks were more likely to harbour infections of *H. hinuliae* than juveniles. An increased probability of infection in adults may be due either to behavioural changes, changes in diet, the advent of reproductive maturity (immunosuppressive hormones or increased social contact), or simply an increased chance of exposure over time. A study of ontogenetic shifts in habitat of *E. quoyii* suggested that juveniles occupy more marginal habitats than adults, due to intraspecific aggression (Law, 1991). If there are ontogenetic habitat changes, then transmission rates may change, either due to closer association with infected larger animals which raise the possibility of encountering and eating infected vectors, because of changes in vector abundance due to habitat, or because of changes in diet (Eisen and Wright, 2001).

Infected adults and juveniles did not differ significantly in parasite load, but 3-year-old male lizards had higher parasite loads than other age classes. Lower mean parasite loads in older age groups could suggest that highly parasitised animals suffer parasite-induced mortality, and the lower mean parasite loads of older animals are representative of survivors with smaller parasite loads (Anderson and Gordon, 1982). Alternatively, concomitant increase in prevalence in 2- and 3-year olds suggests that more of these animals may be exhibiting the peak parasite loads typical of newly infected hosts (Smallridge and Bull, 2001). The equivalent parasite loads in adults and juveniles suggest that it is variation in the individual host–parasite relationship that determines persistent parasite load, rather than the accumulation of multiple infections over time. The results from recaptured animals further support this.

In conclusion, parasite load of *Hepatozoon* is influenced by host factors such as sex and age. Individual variation in parasite load may be due to host behaviour, immune system or genotype. Alternatively, variations in parasite strain or infection stage may be responsible for the observed heterogeneities in parasite load. Prevalence appears reasonably stable in the water skinks of Blackdown Tableland, and suggests that *H. hinuliae* is a persistent and endemic parasite of this lizard population.

Acknowledgements

We thank Mandar Trivedi, John Farquhar, Kristin Sherwood, Ameer Abdulla and Stewart Lee for help with fieldwork, and the Queensland Parks and Wildlife Service rangers at Blackdown Tableland National Park for their help throughout the two years. DJS was funded by a Leverhulme Study Abroad Scholarship. Research funding was provided by James Cook University IRA and MRG grants to DJS and LS, respectively, and an ARC small grant to LS.

References

Anderson, R.M., Gordon, D.M., 1982. Processes influencing the distribution of parasite numbers within host populations with special emphasis on parasite-induced host mortalities. Parasitology 85, 373–398.

Appleby, B.M., Anwar, M.A., Petty, S.J., 1999. Short-term and long-term effects of food supply on parasite burdens in tawny owls, *Strix aluco*. Funct. Ecol. 13, 315–321.

Arneberg, P., Skorping, A., Grenfell, B.T., Read, A.F., 1998. Host densities as determinants of abundance in parasite communities. Proc. R. Soc. Lond. B 265, 1283–1289.

Bajer, A., Pawelczyk, A., Behnke, J.M., Gilbert, F.S., Sinski, E., 2001. Factors affecting the component community structure of haemoparasites in bank voles (*Clethrionomys glareolus*) from the Mazury Lake District region of Poland. Parasitology 122, 43–54.

Bennett, G.F., Cameron, M., 1974. Seasonal prevalence of avian hematozoa in passeriform birds of Atlantic Canada. Can. J. Zool. 52, 1259–1264.

Blomberg, S.P., Shine, R., 2001. Modelling life history strategies with capture-recapture data: evolutionary demography of the water skink *Eulamprus tympanum*. Aust. Ecol. 26, 349–359.

Bromwich, C.R., Schall, J.J., 1986. Infection dynamics of *Plasmodium mexicanum*, a malarial parasite of lizards. Ecology 67, 1227–1235.

Campbell, T.W., 1996. Clinical pathology. In: Mader, D.R. (Ed.), Reptile Medicine and Surgery. W.B. Saunders Company, Philadelphia, PA, pp. 248–257.

Cleaveland, S., Hess, G.R., Dobson, A.P., Laurenson, K.M., McCallum, H.I., Roberts, M.G., Woodroffe, R., 2002. The role of pathogens in biological conservation. In: Hudson, P.J., Rizzoli, A., Grenfell, B.T., Heesterbeek, H., Dobson, A.P. et al. (Eds.), The Ecology of Wildlife Diseases. Oxford University Press, Oxford, pp. 139–150.

Dawkins, R., 1990. Parasites, desiderata lists and the paradox of the organism. Parasitology 100, S63–S73.

Dowell, S.F., 2001. Seasonal variation in host susceptibility and cycles of certain infectious diseases. Emerging Infect. Dis. [Serial Online], 7. Available from http://www.cdc.gov/ncidod/eid/vol7no3/dowell.htm

Downes, S., Borges, P., 1998. Sticky traps: an effective way to capture small terrestrial lizards. Herpetol. Rev. 29, 94.

Dufva, R., 1996. Blood parasites, health, reproductive success, and egg volume in female great tits *Parus major*. J. Avian Biol. 27, 83–87.

Eisen, R.J., 2000. Variation in life-history traits of *Plasmodium mexicanum*, a malaria parasite infecting western fence lizards: a longitudinal study. Can. J. Zool. 78, 1230–1237.

Eisen, R.J., DeNardo, D.F., 2000. Life history of a malaria parasite (*Plasmodium mexicanum*) in its host, the western fence lizard (*Sceloporus occidentalis*): host testosterone as a source of seasonal and among-host variation? J. Parasitol. 86, 1041–1045.

Eisen, R.J., Wright, N.M., 2001. Landscape features associated with infection by a malaria parasite (*Plasmodium mexicanum*) and the importance of multiple scale studies. Parasitology 122, 507–513.

Ferrari, N., Cattadori, I.M., Nespereira, J., Rizzoli, A., Hudson, P.J., 2004. The role of host sex in parasite dynamics: field experiments on the yellow-necked mouse *Apodemus flavicollis*. Ecol. Lett. 7, 88–94.

Forbes, M., Weatherhead, P.J., Bennett, G.F., 1994. Blood parasites of blue grouse: variation in prevalence and patterns of interspecific association. Oecologia 97, 520–525.

Fuller, C.A., 1996. Population dynamics of two species of *Eimeria* (Apicomlexa: Eimeriidae) in deer mice (*Peromyscus maniculatus*): biotic and abiotic factors. J. Parasitol. 82, 220–225.

Galvani, A.P., 2003. Epidemiology meets evolutionary ecology. Trends Ecol. Evol. 18, 132–139.

Gulland, F.M.D., 1995. Impact of infectious diseases on wild animal populations, a review. In: Grenfell, B.T., Dobson, A.P. (Eds.), Ecology of Infectious Diseases in Natural Populations. Cambridge University Press, Cambridge, MA, pp. 20–51.

Hudson, P.J., Rizzoli, A., Grenfell, B.T., Heesterbeek, J.A.P., Dobson, A.P., 2002. Ecology of Wildlife Diseases. Oxford University Press, Oxford.

Keymer, A.E., Read, A.F., 1991. Behavioural ecology: the impact of parasitism. In: Bolis, L. (Ed.), Parasitism: Coexistence or Conflict? Oxford University Press, Oxford, pp. 37–61.

Law, B.S., 1991. Ontogenetic habitat shift in the eastern Australian water skink (*Eulamprus quoyii*)? Copeia 1991;, 1117–1120.

McCurdy, D.G., Shutler, D., Mullie, A., Forbes, M.R., 1998. Sex-biased parasitism of avian hosts: relations to blood parasite taxon and mating system. Oikos 82, 303–312.

McKenzie, F.E., Killeen, G.F., Beier, J.C., Bossert, W.H., 2001. Seasonality, parasite diversity, and local extinctions in *Plasmodium falciparum* malaria. Ecology 82, 2673–2681.

Morand, S., Poulin, R., 2000. Nematode parasite species richness and the evolution of spleen size in birds. Can. J. Zool. 78, 1356–1360.

Nordling, D., Andersson, M., Zohari, S., Gustafsson, L., 1998. Reproductive effort reduces specific immune response and parasite resistance. Proc. R. Soc. Lond. B 265, 1291–1298.

Norris, K., Anwar, M., Read, A.F., 1994. Reproductive effort influences the prevalence of haematozoan parasites in great tits. J. Anim. Ecol. 63, 601–610.

Oppliger, A., Clobert, J., 1997. Reduced tail regeneration in the common lizard, *Lacerta vivipara*, parasitized by blood parasites. Funct. Ecol. 11, 652–655.

Oppliger, A., Vernet, R., Baez, M., 1999. Parasite local maladaptation in the Canarian lizard *Gallotia galloti* (Reptilia: Lacertidae) parasitized by haemogregarian blood parasite. J. Evol. Biol. 12, 951–955.

Perkins, S.L., Keller, A.K., 2001. Phylogeny of nuclear small subunit rRNA genes of hemogregarines amplified with specific primers. J. Parasitol. 87, 870–876.

Poulin, R., 1996. Sexual inequalities in helminth infections: a cost of being a male? Am. Nat. 147, 287–295.

Readon, J.T., Norbury, G., 2004. Ectoparasite and hemoparasite infections in a diversetemperate lizard assemblage at Macraes Flat, South Island, New Zealand. J. Parasitol. 2004; in press.

Real, L.A., 1996. Sustainability and the ecology of infectious disease. Bioscience 46, 88–97.

Saad, A.H., El Ridi, R., 1988. Endogenous corticosteroids mediate seasonal cyclic changes in immunity of lizards. Immunobiology 177, 390–403.

Saad, A.H., Khalek, N.A., El Ridi, R., 1990. Blood testosterone level: a season-dependent factor regulating immune reactivity in lizards. Immunobiology 180, 184–194.

Salvador, A., Veiga, J.P., Martin, J., Lopez, P., Abelenda, M., Puerta, M., 1996. The cost of producing a sexual signal: testosterone increases the susceptibility of male lizards to ectoparasitic infestation. Behav. Ecol. 7, 145–150.

Salvador, A., Veiga, J.P., Martin, J., Lopez, P., 1997. Testosterone supplementation in subordinate, small male lizards: consequences for aggressiveness, color development, and parasite load. Behav. Ecol. 8, 135–139.

Schalk, G., Forbes, M.R., 1997. Male biases in parasitism of mammals: effects of study type, host age, and parasite taxon. Oikos 78, 67–74.

Schall, J.J., 1986. Prevalence and virulence of a haemogregarine parasite of the Aruban whiptail lizard, *Cnemidophorus arubensis*. J. Herpetol. 20, 318–324.

Schall, J.J., Bromwich, C.R. (1994). Interspecific interactions tested: two species of malarial porasite in a West African Lizard. Oecologia, 97, 326–332.

Schall, J.J., 1996. Malarial parasites of lizards: diversity and ecology. Adv. Parasitol. 37, 255–333.

Schall, J.J., Marghoob, A.B., 1995. Prevalence of a malarial parasite over time and space: *Plasmodium mexicanum* in its vertebrate host, the western fence lizard *Sceloporus occidentalis*. J. Anim. Ecol. 64, 177–185.

Schall, J.J., Pearson, A.R., 2000. Body condition of a Puerto Rican anole, *Anolis gundlachi*: effect of a malaria parasite and weather variation. J. Herpetol. 34, 489–491.

Schall, J.J., Pearson, A.R., Perkins, S.L., 2000. Prevalence of malaria parasites (*Plasmodium floridense* and *Plasmodium azurophilum*) infecting a Puerto Rican lizard (*Anolis gundlachi*): a nine-year study. J. Parasitol. 86, 511–515.

Smallridge, C.J., Bull, C.M., 1999. Transmission of the blood parasite *Haemolvia mariae* between its lizard and tick hosts. Parasitol. Res. 85, 858–863.

Smallridge, C.J., Bull, C.M., 2000. Prevalence and intensity of the blood parasite *Haemolivia mariae* in a field population of the skink, *Tiliqua rugosa*. Parasitol. Res. 86, 563–569.

Smallridge, C.J., Bull, C.M., 2001. Infection dynamics of *Haemolivia mariae* in the sleepy lizard *Tiliqua rugosa*. Parasitol. Res. 87, 657–661.

Smith, T.G., 1996. The genus *Hepatozoon* (Apicomplexa: Adeleina). J. Parasitol. 82, 565–585.

Sol, D., Jovani, R., Torres, J., 2000. Geographical variation in blood parasites in feral pigeons: the role of vectors. Ecography 23, 307–314.

Sorci, G., 1995. Repeated measurements of blood parasite levels reveal limited ability for host recovery in the common lizard (*Lacerta vivipara*). J. Parasitol. 81, 825–827.

Staats, C.M., Schall, J.J., 1996. Distribution and abundance of two malarial parasites of the endemic Anolis lizard of Saba Island, Netherlands Antilles. J. Parasitol. 82, 409–413.

Veiga, J.P., Salvador, A., Merino, S., Puerta, M., 1998. Reproductive effort affects immune response and parasite infection in a lizard: a phenotypic manipulation using testosterone. Oikos 82, 313–318.

Veron, J.E.N., 1969. The reproductive cycle of the water skink, *Sphenomorphus quoyii*. J. Herpetol. 3, 55–63.

Wozniak, E.J., Kanitz, C., Homer, B., Kreisle, R., Telford, S.R.J., McLaughlin, G., 1996. Demonstration of common and stage-specific anti-*Hepatozoon mocassini* antibodies in experimentally infected unnatural lizard hosts. Int. J. Parasitol. 26, 131–133.