Lizard Ecology

Studies of a Model Organism

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4 Lizard Malaria: Parasite-Host Ecology

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PARASITIC SPECIES COMPOSE a significant fraction of the world's fauna (Dogiel, 1966; Price, 1977). This remarkable richness of species is evident from the array of organisms parasitizing lizards. For example, Telford (1970) found an average of 11 endoparasitic species per host species in his survey of lizards from southern California. When bacteria and viruses are included, it is safe to assume that every one of the approximately 3,000 extant species of lizards is infected by several to many species of parasites. Thus, the potential number of parasite-lizard interactions must be vast, perhaps even eclipsing the number of competitive or predator-prey interactions.

A widespread and potentially very important group of lizard parasites are the saurian malarias (*Plasmodium*). Biologists generally are more familiar with malarias of birds and mammals, including the 4 species infecting humans. However, some 120 species attack nonhuman vertebrates, and about half of these are parasites of lizards (Ayala, 1977). Although new species of bird or mammal malaria are rarely discovered, surveys in previously uninvestigated regions frequently uncover new plasmodia of lizards. For example, Telford (1978) found 14 species of saurian malaria from a limited region of Venezuela, 5 of which were undescribed forms. *Plasmodium* appears to be primarily a parasite of lizards and perhaps originally evolved from a gut-dwelling lizard parasite (Manwell, 1955).

Although lizard malaria has been known to biologists for over 70 years (Aragão and Neiva, 1909; Wenyon, 1909), detailed knowledge of these organisms and their effects on hosts is scant. Recently Ayala (1978) catalogued the entire world literature on saurian malaria and found only 156 publications, mostly of a taxonomic nature or dealing with host and locality records. The literature of interest to ecologists consists of only a sparse handful of papers.

In contrast, the severe impact of human malaria on its host is well documented. These protozoans kill millions of humans worldwide each year, disable hundreds of millions of others, and have greatly influenced the evolution and history of our species (Livingstone, 1971; Harrison, 1978). Could lizard malarias have a similar effect on their hosts? Despite a lack of supporting data, the general view among malariologists has been that lizard malaria is a relatively benign parasitic disease (Russell et al., 1963; Telford, 1971). This view is based on the hypothesis that very old parasite-host relationships are likely to be benign compared to more recently established associations, because a fairly stable coevolutionary equilibrium has been reached (Burnet, 1962). Whatever the merits of this hypothesis, the data presented here provide the first reasonably complete inventory of effects of a malarial parasite on a lizard host (the first, in fact, for any lizard parasite) and demonstrate that these effects are substantial.

Since 1978 I have been studying a lizard malaria system in northern California. The host is the western fence lizard (*Sceloporus occidentalis*), and the parasite is *Plasmodium mexicanum.* When I initiated this research I asked the following questions: What are the costs (virulence) to individual lizards resulting from malarial infection? Can parasite-induced hematological pathology be related to physiological and behavioral effects and ultimately to reduction in reproductive success (fitness)? An ultimate goal of the research described here is to determine the variation in virulence among lizard malaria species and to discover the evolutionary origin of such variation.

Study System

Lizard malaria is widely distributed geographically and occurs in most lizard families (Ayala, 1977). Greatest species richness seems to occur in the neotropics where as many as 6 species of *Plasmodium* can be found in a very limited area (Telford, 1977, 1978), but this pattern may simply reflect unequal collecting efforts. Three species have been described in the United States: *P. mexicanum,* a parasite of *Sceloporus* in the western United States, *P. floridense* infecting *Anolis* and *Sceloporus* in the southeastern states, and *P. chiricahuae,* known from *Sceloporus* in Arizona.

Lizard malaria often has a locally patchy distribution; adjacent populations of lizards often differ greatly in parasite prevalance (Ayala, 1970; Jordan and Friend, 1971). For example, *P. mexicanum* has a patchy distribution in northern California. Infected lizards were found at 5 of the 13 sites I surveyed in Alameda, Sonoma, Contra Costa, Marin, Napa, Mendocino, and Sacramento counties. Even at sites where the parasite occurred, foci of infection were often very local, existing just a few hundred meters from similar but parasite-free habitat.

My primary study site was the University of California Hopland Field

Station, an agricultural research facility in southern Mendocino County, 90 miles north of the Golden Gate. The 5,300-acre Hopland Station is primarily foothill oak woodland with chaparral at higher elevations. Summers are warm and dry; precipitation falls primarily during the relatively mild winters. During the past decade approximately 10,000 trees have been felled to open the habitat for sheep grazing. These fallen logs are excellent lizard habitat, and *Sceloporus* are exceptionally abundant in such areas.

I sampled lizards during their seasonal activity period from April through September 1978–1980. Blood was drawn from a toe clip, and a smear made for staining and microscopic examination (Ayala and Spain, 1976). Approximately 2,500 lizards were sampled over the 3-year period.

Fence lizards at Hopland were infected by 4 species of blood parasites. Two were common: malaria and another protozoan, *Schellackia occidentalis.* Only sporozoites of *Schellackia* occur in the erythrocytes; other stages are in the intestinal epithelium (Bonorris and Ball, 1955). Lizards were rarely infected by a haemogregarine (only gametocytes are in blood cells) or a free-swimming trypanosome. As I wished to study the effects of malaria on lizard hosts, I have not included data for lizards infected with other blood parasites in the comparative analyses.

Plasmodium has a complex life cycle involving both a vertebrate and insect host. The invertebrate host of lizard malaria is unknown, although Ayala and Lee (1970) found sporogonic development of *P. mexicanum* in the psychodid fly, *Lutzomyia vexatrix.* This fly spends the day in burrows of ground squirrels at the Hopland Station, emerging at night to take blood meals from lizards and other ectothermal vertebrates (Ayala, 1973).

Whatever the insect host may be, *Plasmodium* sporozoites are passed onto a lizard and parasites eventually appear in the blood. There they follow a course of infection similar to that of bird or mammal malarias. In *P. mexicanum* a uninucleated small merozoite enters an erythrocyte and begins to feed. This feeding stage, or trophozoite, eventually undergoes nuclear division to form a multinucleated schizont. In mature schizonts (segmenters) of *P. mexicanum,* the 8 to 20 nuclei align themselves along the periphery of the cell mass just prior to cellular division. The red blood cell ruptures, freeing the daughter merozoites which reinfect other cells.

Rather than proceed through this schizogonous cycle, some merozoites mature into gametocytes ("females" are macrogametocytes, and "males" are microgametocytes). Gametocyte sex ratio in *P. mexicanum* is not constant but varies among infections (range: 20 to 70 percent males). Gametocytes are carried into the insect host when it takes a blood meal. Fertilization takes place in the insect's stomach and the resulting sexual cycle eventually produces new sporozoites.

Early in the spring at the Hopland Station, *Plasmodium* blood infections in lizards are of two kinds. Some consist only of trophozoites and schizonts (presumed "new" infections), and others consist of all stages, including many old gametocytes (large heavily vacuolated cells). These infections have probably persisted over the winter in the lizards. Active schizogony in blood cells occurs primarily during the early part of the season; by late August and September most infections consist mainly of gametocytes. The presumed vector, *Lutzomyia,* is not active at Hopland during April and May (personal observation of traps and communication from J. Anderson). Therefore, transmission probably takes place in late summer, and blood infections do not become patent until the next spring.

Parasite Prevalence

Parasite prevalence (percent of lizards infected) differs considerably between sexes (Table 4.1 and Fig. 4.1). A consistently greater proportion of males is infected at any time. Such higher prevalence of parasitic infection among male hosts is common in a wide variety of vertebrate parasites (Nawalinski et al., 1978). In this case, perhaps female lizards are killed by the parasite more often than are males. However, there appears to be no such differential mortality between sexes; sex ratio of adult lizards does not differ significantly from 1:1 (739 males versus 707

Table 4.1 Prevalence (percent of lizards infected) of *Plasmodium mexicanum* and *Schellackia occidentalis* in the western fence lizard at the Hopland Field Station, Mendocino County, California, 1978–1980.

SVL in mm	1978	1979	1980
		Plasmodium	
60–64	36 (87)	26 (50)	15 (26)
	13 (64)	14 (36)	5 (21)
65–70	37 (170)	27 (187)	26 (100)
	20 (143)	22 (159)	20 (46)
>70	40 (25)	33 (57)	35 (37)
	33 (63)	19 (111)	22 (64)
Total	37 (282)	28 (294)	26 (163)
	21 (270)	20 (306)	18 (131)
Grand total	29 (552)	24 (600)	23 (294)
		Schellackia	
Total	6 (552)	13 (600)	16 (294)
(Rainfall)	(126.2 cm)	(66.5 cm)	(108.1 cm)

NOTE: Data for males above those for females. Sample size in parentheses. Rainfall periods, October to May preceding each summer.

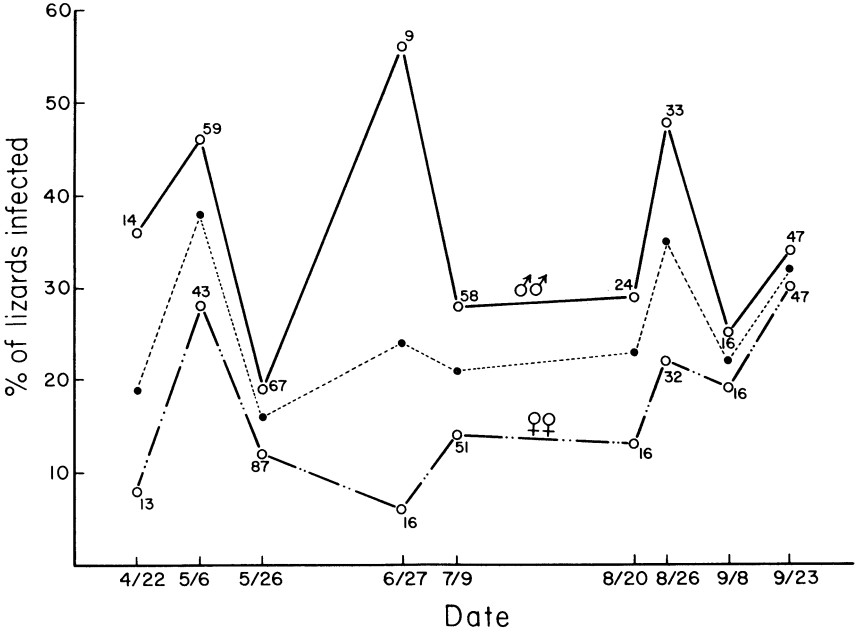

Figure 4.1 Percent of western fence lizards infected by malaria at the Hopland Field Station for 8 sampling periods (first day indicated) during 1978. Males indicated by upper line, females by lower, and overall percent by middle dashed line. Sample sizes indicated next to points.

females, $\chi^2 = 0.354$, $P > 0.05$), nor does the sex ratio of juveniles hatched from laboratory-maintained eggs (274 males versus 270 females, $\chi^2 = 0.015$, $P > 0.05$).

Although evidence is weak, the sexual trend in parasite prevalence seems to occur even in subadults (Fig. 4.2). Small juveniles (< 50 mm snout-vent length, SVL) are very rarely infected; however, larger (50–59 mm) subadult (prereproductive) males are more often infected than are females of this size. Sample size for the 50–59 mm group is small (only 89) and the difference between males and females is not statistically significant ($P = 0.26$). Other hypotheses explaining the sexual difference in parasite prevalence might be proposed, but any hypothesis should confront the possible difference in prevalence even among small, prereproductive animals. If the sexual difference in prevalence were apparent only in adult lizards then any one of many physiological, behavioral, or ecological differences between sexes could be implicated as potential causative factors. However, if the difference in parasite prevalence exists for juvenile lizards, there must be some important difference between sexes even at this early age.

The higher parasite prevalence in larger lizards suggests that an in-

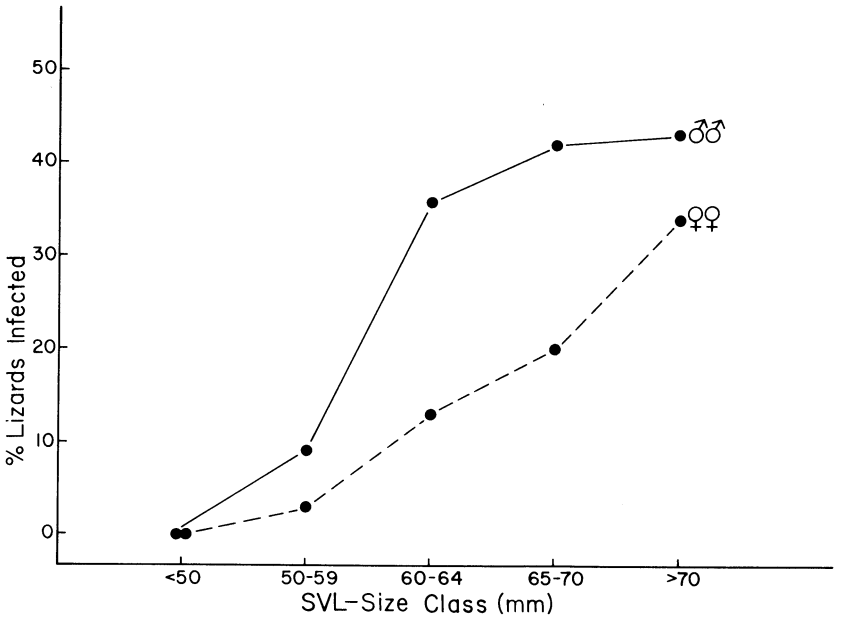

Figure 4.2 Percent of male and female western fence lizards infected with malaria, plotted against snout to vent length (SVL). Data are from 1978 sample.

fected lizard may maintain at least a low-level infection for years and perhaps for the duration of its life. Malarious animals kept in large laboratory pens never lost their infection, but parasitemias often dropped to very low levels. In Panama *Anolis limifrons,* a small, short-lived animal exhibits a similar pattern (S. Guerrero, personal communication).

Malaria prevalence did not vary significantly among years ($\chi^2 = 5.88$, $P > 0.05$) despite changes in environmental conditions resulting from variability in winter precipitation (Table 4.1). The winter prior to the 1978 season was wet and followed a long and severe drought. Consequently, vegetation growth in spring 1978 was exceptionally luxuriant compared to that of the next two seasons. This short-term constancy in parasite prevalence in the face of environmental perturbation suggests that lizard malaria may be a stable system. Ten years prior to my study, Ayala (1970) recorded a similar level of lizard malaria prevalence at Hopland. However, in Georgia *P. floridense* in *Sceloporus undulatus* remained at a fairly constant low prevalence over a 13-year period but exhibited long-term but dramatic changes (50 percent to 10 percent prevalence) in another host, *Anolis carolinensis* (Jordan and Friend, 1971). In comparison, *Schellackia* at my study site significantly increased in prevalence from 1978 to 1979 and 1980 (Table 4.1, $\chi^2 = 23.16$, $P < 0.001$).

The incidence of multiple infections of *Plasmodium* and *Schellackia* presents an equivocal pattern. In 1978 the two parasites occurred together in random frequency (8 multiple infections observed, 9.6 expected; χ^2 test, $P > 0.05$), but in 1979 and 1980 multiple infections were rare, suggesting some sort of interaction between parasite species (8 observed, 18.7 expected in 1979; 2 observed, 10.8 expected in 1980; χ^2 tests, P's < 0.05). The nature of such a possible interaction is intriguing, especially as *Schellackia* does not reproduce in blood cells and *Plasmodium* does not infect intestinal tissues.

Physiological Consequences of Infection

Larger lizards generally have a greater probability of being infected (Table 4.1). To eliminate bias I carefully matched body sizes of experimental groups in the following comparisons. Also, only animals with natural infections were used.

Lizards usually respond to malarial infection by producing copious numbers of polychromatic erythrocytes (Ayala, 1970; Scorza, 1971; Telford, 1972; Ayala and Spain, 1976; Guerrero, Rodriguez, and Ayala, 1977). These immature red blood cells (iRBC) are easily distinguished, as their cytoplasm has an affinity for the basic or blue portion of Giemsa stain, probably because they have less hemoglobin and more RNA in their cytoplasm (Diggs, Sturm, and Bell, 1978).

Infected *Sceloporus* have significantly more iRBC than do noninfected animals (U-test, $P \ll 0.001$). Typically, up to 2 percent of erythrocytes are immature in noninfected animals whereas the range is much greater (1 to 30 percent) for infected lizards (Fig. 4.3). Time lags may be responsible for the scatter; that is, infected lizards may have a high proportion of iRBC for some time after parasitemia has reached a "crisis" stage and declined. Increase in iRBC is probably a result of the lizard's hemopoietic response, which is mobilized to replace destroyed red blood cells (RBC). As parasitemia rises, the lizard may be forced to place immature erythrocytes into circulation to maintain RBC abundance. However, iRBC levels sometimes rise early in an infection, when parasitemia is low. *Plasmodium mexicanum* is primarily a parasite of mature erythrocytes and when *P. mexicanum* does infect iRBC, the parasites appear stunted. Thus, production of iRBC may function as an antiparasite tactic, weakly analogous to sickle cell anemia or the Duffy negative antigen defense (Friedman and Trager, 1981). This possibility deserves careful further investigation.

If iRBC contain less hemoglobin per cell than do mature erythrocytes, there should be measurable physiological consequences to infected lizards. The details of measurement of these effects are reported elsewhere (Schall, Bennett, and Putnam, 1982); here I will summarize the results. Compared to the noninfected lizards, blood of infected animals contains significantly less hemoglobin (5.5 versus 7.3 g/ 100 ml, U-test,

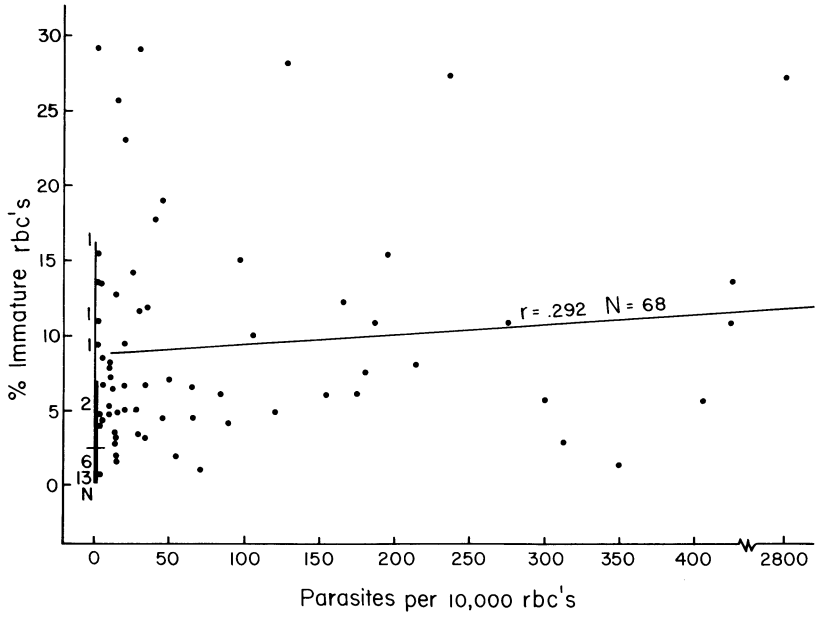

Figure 4.3 Malarial parasitemia plotted against percent of immature red blood cells (iRBC) for 68 infected *Sceloporus occidentalis;* also plotted is distribution for 24 noninfected lizards ($\bar{x}$ = horizontal line, S.D. = dark vertical bar, range = light vertical bar). Three lizards in "noninfected" class with high percent of iRBC (> 5) may be a result of false negatives.

$P < 0.001$). This does not appear to be a result of a decrease in numbers of RBC as neither hematocrits nor direct RBC counts differ significantly (U-test, $P > 0.05$). However, there is a negative correlation between percent iRBC and hemoglobin concentration ($r = -0.508$, $P < 0.01$, N = 49), confirming that decrease in hemoglobin concentration is due to the increased number of iRBC in circulation.

Except for lizards with overwhelming infections, malarious lizards in the laboratory typically appear healthy and behave "normally." This has led some observers to conclude that lizard malaria is a benign parasite. However, a reduction of blood hemoglobin concentration by about 25 percent should reduce the lizard's ability to deliver oxygen to body tissues and certainly should have important behavioral and ecological consequences.

Oxygen usage by resting lizards is very low, just a fraction of their maximal consumption (Bennett, 1978). As might be expected, resting O_2 consumption does not differ between infected and noninfected lizards. However, oxygen consumption during maximal activity is significantly lower for infected lizards (1.3 cc/g • h for infected versus 1.53 cc/g • h for noninfected, U-test, $P < 0.05$). The increment in O_2 consumption from

resting to maximal activity, an indication of ability to support activity aerobically (aerobic scope), also differs between the two groups (0.71 cc/g•h for infected versus 1.00 cc/g•h for noninfected, U-test, $P < 0.01$). Both increments in oxygen consumption and maximal oxygen consumption are positively correlated with blood hemoglobin concentration ($r = 0.72$, $P < 0.01$, N = 28, and $r = 0.68$, $P < 0.01$, N = 28, respectively). This suggests that reduction in aerobic capacities of infected lizards is a result primarily of the deficit in hemoglobin levels, rather than of other pathological effects of infection.

A likely important consequence of reduced oxygen consumption during exertion would be a decrease in aerobically sustained locomotory ability. Very short bursts, or sprints, of activity are supported in lizards by anaerobic mechanisms (Bennett, 1978). Burst running speed, measured electronically in a 2-m track, does not differ between infected and noninfected lizards ($\bar{X}$ = 1.28 versus 1.44 m/s, U-test, $P \approx 0.10$). Running stamina, though, is supported in large part by aerobic means and is significantly reduced for infected lizards. For example, infected lizards, when forced to run continuously for 30 s, covered an average of about 17 m, whereas noninfected animals ran about 21.3 m (U-test, $P < 0.01$). A similar reduction in stamina was observed for lizards running a full 2 min (27 versus 32 m; U-test, $P < 0.05$).

Costs to Reproductive Success

Reproductive success, or Darwinian fitness, is notoriously difficult to measure, especially in males. Ideally, we should count lifetime number of offspring produced by an individual and determine survival of those offspring. In practice I used a number of measures, which when taken together provide a reasonable index of reproductive success. Results for males are equivocal but data for females clearly demonstrate that malaria infection reduces female fitness.

Testis Size. During 1978 I collected 8 samples of males (N = 266) and weighed the testes of each animal (Fig. 4.4). Testis mass declined during the reproductive season and began to increase in late August after reproduction ceased. The pattern for infected and noninfected males was very similar until late summer when noninfected males had significantly larger testes (U-test, $P < 0.001$). These results are confirmed by samples collected exactly 1 year later; again, infected animals had smaller testes (N = 22 infected, 41 noninfected, U-test, $P < 0.002$). In both years the average reduction in testis size was about 37 percent. I know of no evidence that male lizards with larger testes experience higher reproductive success, but larger testes may produce more gametes and hormones, and both are obviously important in male reproduction.

Stored Fat. Over 8 sampling periods in 1978, I weighed paired inguinal fat bodies of 530 *Sceloporus* (Fig. 4.5). Fat bodies remained small during the reproductive season and increased after reproduction ceased, a pat-

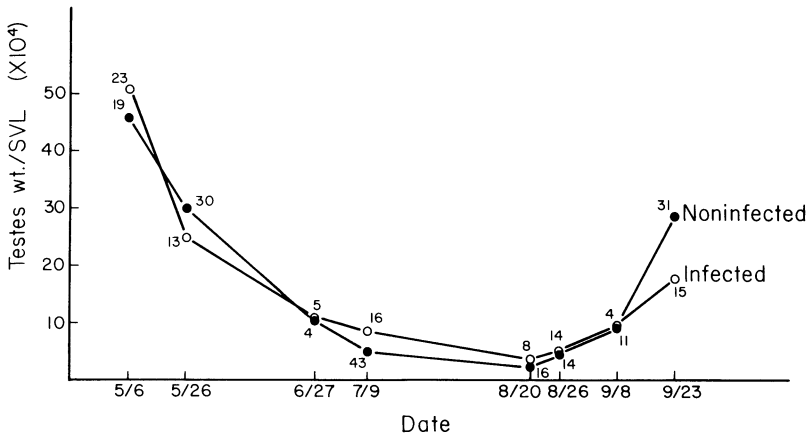

Figure 4.4 Mean testis size of western fence lizards that were infected (open points) or noninfected (closed points) by malaria for 8 sampling periods at the Hopland Field Station during 1978. Because body mass varies, depending on contents of the digestive tract, mass of paired testes is divided by snout-vent length for each animal to correct for body size. Sample sizes are indicated next to points.

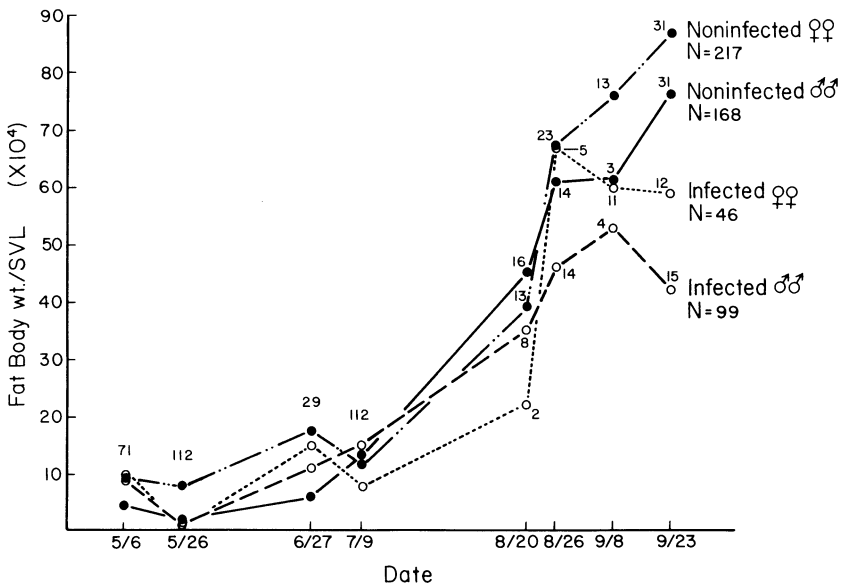

Figure 4.5 Mean mass of both fat bodies corrected for body size for western fence lizards for 8 sampling periods during 1978. Sample sizes indicated next to points.

tern typical of temperate zone lizards (Schall, 1978). By summer's end, females store more fat than males, and infected animals store less fat than do noninfected animals of the same sex. One year later I collected another sample. Once again, for both sexes, infected lizards store less fat (Table 4.2).

A comparison between years produces an unexpected result (Table 4.2). Both male and female noninfected lizards stored very similar amounts of fat by season's end in both years. However, in 1979, infected lizards of both sexes stored more fat than did infected animals the previous year. This suggests that resources may have been more abundant in 1979. Although I have no quantitative measures, insect density (especially grasshoppers) appeared much greater in 1979, a result perhaps of the lush vegetative growth the previous year. There may be an optimal amount of fat to be stored by these lizards, and they simply stop adding fat tissue once this level is reached. Therefore, in a very productive year, fat stored by noninfected lizards is about the same as during an average year. Infected lizards, though, have greater difficulty in storing fat, so the amount of fat they store varies to a greater degree on resource availability.

Does stored fat contribute directly to reproductive success? Fat bodies of males are assumed to be a source of energy during winter brumation (Gaffney and Fitzpatrick, 1973) and for establishing territories in the spring. Fat bodies of females are also used to produce the first clutch of eggs in spring (Hahn and Tinkle, 1964). Caloric content of fat bodies and of *S. occidentalis* eggs are known. Assuming that almost all calories in fat bodies are used in egg production, the deficit in fat stored by infected animals has been used to calculate a predicted decrease in clutch size (Schall, 1982). The decrement in fat stored by infected compared to noninfected lizards is equal to the calories in 1.42 eggs for 1978 and 1.00

Table 4.2 Mass of both inguinal fat bodies corrected for body size for western fence lizards infected and not infected by malaria, late September sampling periods.

	1978		1979	
Males				
Non-infected	0.0076 (0.0030, 31)	$P < 0.001$	0.0076 (0.0027, 42)	$P < 0.05$
Infected	0.0042 (0.0026, 15)		0.0059 (0.0038, 22)	
Females				
Non-infected	0.0087 (0.0031, 31)	$P < 0.01$	0.0092 (0.0029, 44)	$P < 0.05$
Infected	0.0059 (0.0032, 12)		0.0076 (0.0036, 21)	

NOTE: Mean fat-body mass in g/SVL; SD and sample size in parentheses. *P* for U-test given also.

eggs for 1979. Therefore, an infected lizard on the average should produce clutches with 1 to 2 fewer eggs than noninfected females.

Clutch Size. The reproductive season for *S. occidentalis* at Hopland extends from emergence in mid-April, when most adult females have enlarged yolked ovarian follicles, to about mid-July when at least some large adults produce their second clutch for the season (Schall, unpublished observations). The proportion of infected and noninfected females that were gravid at any time did not differ (χ^2 test, $P > 0.05$). Because fat bodies are utilized in only the first clutch, the following analysis is restricted to clutches from females collected in May and June.

Results for 1978 and 1979 are presented in Figures 4.6 and 4.7 respectively. Body size and clutch size are positively correlated so I compared samples by an analysis of covariance. For both years residual variance and regression slopes did not differ between infected and noninfected animals ($P > 0.05$), so I was able to compare regression elevations. Infected lizards produced significantly smaller clutches both years ($P < 0.01$). The difference was approximately 1 to 2 eggs as predicted above from the analysis of stored fat.

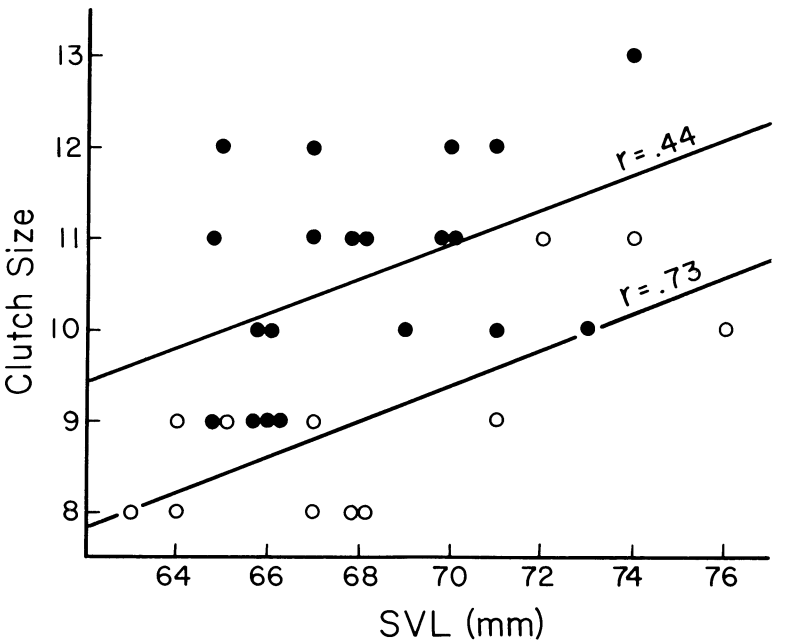

Figure 4.6 Body size plotted against clutch size of 12 *Sceloporus occidentalis* infected with malaria (open points, lower regression) and 20 noninfected (closed points, upper regression), collected at Hopland Field Station during spring 1978. Clutch size determined from counts of large yolked ovarian follicles, oviducal shelled eggs, or eggs laid in laboratory.

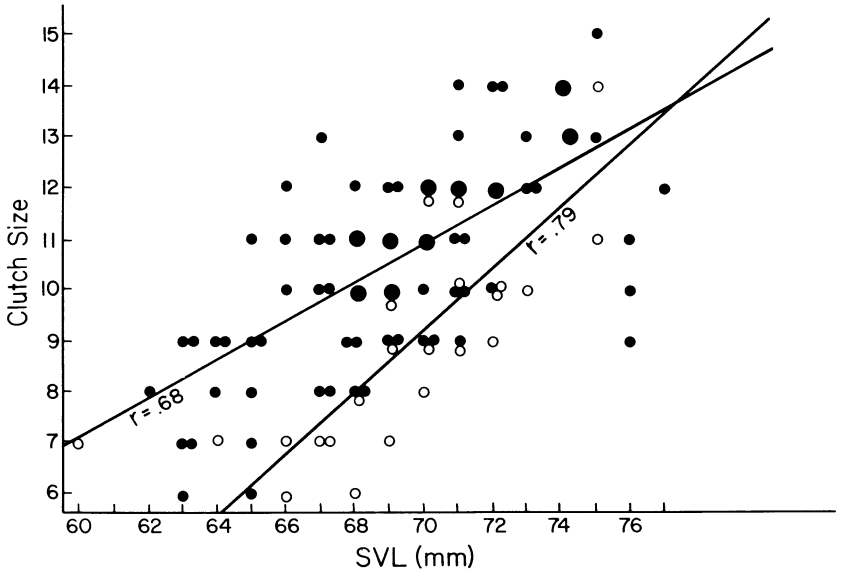

Figure 4.7 Data similar to those in Figure 4.6 except gathered during spring 1979. Large points indicate two or more overlapping data. Sample size was 23 infected and 99 noninfected lizards.

Hatching Success. Malarial infection could lower a female lizard's reproductive success by indirectly reducing survival of her offspring. For example, infected females may be unable sufficiently to provision eggs with a vital nutrient. I therefore measured a variety of indicators of hatching success and hatchling health. Hundreds of adult females were brought into the laboratory, and 74 clutches were gathered and incubated (Table 4.3).

Table 4.3 Various measures related to hatching success of western fence lizards.

	Infected	Noninfected	Test
Mean egg mass	0.346 g (0.063,14)	0.365 g (0.071,64)	U-test, $P > 0.05$
Clutch mass/female body mass	0.269 (0.052,15)	0.271 (0.040,62)	U-test, $P > 0.05$
Days to hatch	71.1 (3.76,8)	71.7 (4.84,50)	U-test, $P > 0.05$
Percent of clutches hatching	80 (10)	80 (64)	χ^2, $P > 0.05$
Percent of eggs hatching	93 (67)	85 (482)	χ^2, $P > 0.05$

NOTE: SD, when appropriate, and sample sizes in parentheses.

Hatching success was measured three ways: time to hatch when maintained at room temperature (21 to 28 °C), percent of clutches producing hatchlings, and percent of eggs hatching from clutches that produced some hatchlings. Clutches produced by infected or noninfected lizards did not differ in any of these measures.

Mean egg mass and mean hatchling size in *S. occidentalis* are correlated ($r = 0.535$, $P < 0.01$, N = 51 clutches) suggesting that egg mass is a reasonable indicator of potential hatchling survival. To obtain a sufficient sample I weighed only oviducal eggs extracted from dissected lizards. Mean egg mass for eggs from infected and noninfected lizards did not differ and neither did overall investment by females in eggs (clutch mass/ female body mass).

These results demonstrate that, although infection with malaria results in significantly smaller clutches of eggs, eggs from malarious females are indistinguishable from those produced by noninfected lizards.

Growth Rate. Growth rate can affect a lizard's lifetime production of offspring in several ways. For example, clutch size and body size are positively correlated. Also, larger males may occupy larger or higher quality territories. I used a mark-recapture technique to study growth rate (Fig. 4.8). Growth rate declines as lizards mature, so each point in Figure 4.8 actually represents an average growth rate over the time between measurements (150 days). Also, some lizards scored on Figure 4.8 as "in-

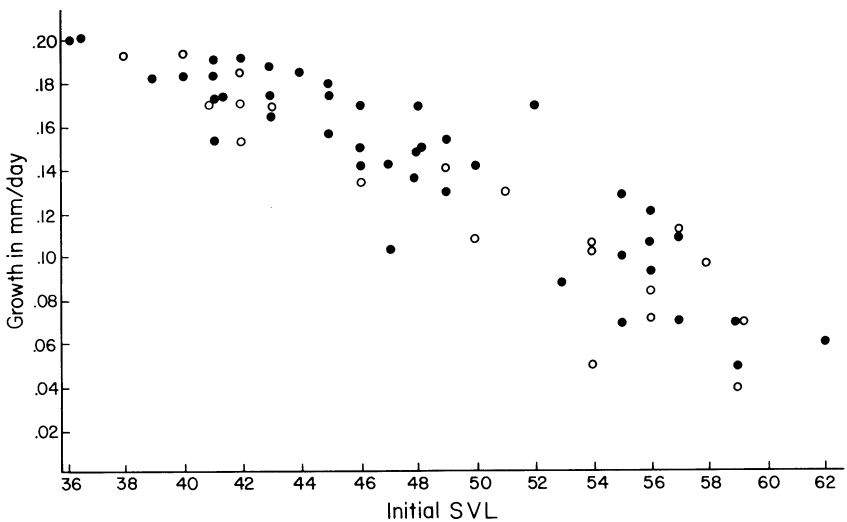

Figure 4.8 Growth rate of western fence lizards infected (open points) or noninfected (closed points) with malaria. Growth rate determined by marking lizards in spring 1979 (≈ 700 animals marked) and recapturing them about 150 days later (≈ 60 recaptured).

fected" were infected only at time of second capture (including most of lizards < 50-mm SVL at initial capture).

Infected lizards experienced only very slightly reduced growth rates, about 4 percent (analysis of covariance; residual variances and slopes, $P > 0.05$; elevations, $0.01 < P < 0.05$). Why should somatic investment (growth rate) be maintained at nearly normal levels by infected lizards when production of offspring is reduced? After all, offspring are the currency of natural selection. *Sceloporus occidentalis* may live as long as seven years (Ruth, 1977), so infected animals surviving peak parasitemia could maintain low-level infection for years. Perhaps infected lizards can maximize lifetime reproductive output by maintaining fairly rapid growth and reaching large size quickly rather than by maintaining a larger clutch size.

Parasite-induced Mortality. I have no estimates of mortality caused by the parasite in free-ranging lizards. However, infected animals maintained in laboratory cages were more likely to die than were noninfected lizards. Fence lizards were maintained for one week to several months in laboratory cages. There was no bias between infected and noninfected lizards in period of time animals were kept in captivity. Of 308 males, 15.1 percent of infected and 2.5 percent of noninfected animals died. Of 300 females, 28.6 percent of infected and 11.1 percent of noninfected animals died. For both sexes, χ^2 tests were significant (P's < 0.01). These results would suggest that, at least under stressful conditions, malarious lizards in the wild could suffer increased mortality.

Discussion

Despite their abundance, both in numbers of individuals and richness of species, parasites have elicited comparatively little interest among ecologists. Most general ecology texts devote only a few lines to several pages to the subject. A notable exception is a text used in the Soviet Union (Naumov, 1972) where parasite ecology is a very active discipline. A perusal of the literature on lizard ecology (including this volume) demonstrates that the parasite-lizard interaction is very rarely investigated. For example, although there are tantalizing hints that parasites can critically affect host populations (Warner, 1968; Barbehenn, 1969; Cornell, 1974; Anderson and May, 1979; Price, 1980), the effects of parasites on lizard populations has never been evaluated. An obvious starting point is to assess the impact of a parasite infection on individual lizards. Gross pathology of some lizard parasites is known (Telford, 1971), but overall impact on host individuals of any lizard parasite has not been reported prior to this study.

The data presented here demonstrate that lizard malaria can have substantial effects on its host. Malarial infection initiates a tractable sequence of hematological, physiological, behavioral, and reproductive

consequences (summarized in Table 4.4). Infection results in release of elevated numbers of immature erythrocytes into peripheral circulation. These immature erythrocytes contain less hemoglobin than mature RBC, so blood hemoglobin concentration is reduced. This results in a decrease in oxygen transport capacity of the blood, measured as oxygen consumption during maximal activity. An important consequence of these physiological costs is a decline in aerobically supported locomotory ability (running stamina). Recovery from strenuous activity has not yet been compared for infected and noninfected lizards, but the trend described above suggests that infected lizards would require more time to recover from even relatively short bursts of activity. If so, malarious lizards would be less efficient in gathering food resources and defending territories.

Data on fat-body size support this possibility. Infected lizards store considerably less fat during late summer than do noninfected animals. The caloric content of the deficit in stored fat is approximately equal to the caloric content of 1 to 2 lizard eggs, which corresponds to the observed reduction in clutch size. Thus, reduction in reproductive success of infected female *Sceloporus* can be traced ultimately to the hematological effects of malarial infection.

These effects on individual lizards suggest that there might be populational-level consequences resulting from presence of the parasite. For example, could the malarial parasite limit the population size of *Scelo-*

Table 4.4 Summary of some costs to the vertebrate host (*Sceloporus occidentalis*) induced by a malarial parasite (*Plasmodium mexicanum*).

	Infected versus noninfected (percent decrease)
Hemoglobin concentration	24
Burst running speed	11
Active $\dot{V}O_2$	15
Increment $\dot{V}O_2$	29
Running stamina (30 s)	20
Running stamina (2 min)	17
Fat stored (females, 1978 season)	32
Fat stored (females, 1979 season)	17
Fat stored (males, 1978 season)	45
Fat stored (males, 1979 season)	22
Clutch size (1978 season)	16
Clutch size (1979 season)	13
Testis size (1978 season)	38
Testis size (1979 season)	37
Growth rate	4
Mortality	12–17 (increase)

porus at the study area? This might be possible as malarial infection increases mortality and decreases reproductive output of infected lizards. However, the density of fence lizards at Hopland is the greatest I have seen at any site. The effect of malaria on population size of lizards is probably very minor; other factors, such as availability of home sites, more likely play a more important role.

Nonetheless, ecologists comparing demographic or physiological characteristics of lizard populations should be aware that the patchy distribution of malaria (and perhaps other parasites) can add an unknown but important source of variation to their data. For example, clutch size might be observed to vary between two lizard populations, one infected with *Plasmodium,* and the other not. Without knowledge of the parasite, any explanation proposed to account for this life-history difference might be clever but spurious. Similarly, a physiologist might detect differences in blood hemoglobin concentration and oxygen consumption in *S. occidentalis* populations from high- and low-elevation habitats. These differences may appear to be a result of adaptation by the lizards to differing oxygen partial pressures or environmental temperatures. However, because lizard malaria does not seem to occur at higher elevations (presumably because of absence of the insect vector), observed physiological differences in the lizards may be simply a result of presence or absence of the parasite.

One last important populational effect of *P. mexicanum* on fence lizards must be considered: the coevolution of parasite and host. As demonstrated here, malarious fence lizards experience reduced fitness. Why has *S. occidentalis* not evolved appropriate mechanisms to reduce the impact of malarial infection? That is, what determines parasite virulence? A knowledge of variability in virulence of lizard malarias would cast light on this important problem. Preliminary evidence suggests virulence of lizard malaria does vary considerably, depending on which *Plasmodium* and host species are involved (Ayala, 1977). For example, although *P. mexicanum* has a substantial effect on its host, *P. tropiduri* and *P. balli,* which infect *Anolis limifrons* in Panama do not seem to affect the host's survival, weight, growth rate, or assimilation efficiencies (S. Guerrero, personal communication). Lizard plasmodia also vary greatly in number of merozoites produced by mature segmenters (Ayala, 1977). In some species, mother cells yield only 4 progeny, whereas in others more than 90 merozoites are produced. This hints that some species of lizard malaria may reproduce very rapidly, perhaps racing ahead of the host's immune response. These species might be exceptionally virulent, compared to more slowly reproducing forms. Lizard malaria appears to be an ideal model for studies on the evolution of parasite virulence.

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