# Hormonal Alterations and Reproductive Inhibition in Male Fence Lizards (*Sceloporus occidentalis*) Infected with the Malarial Parasite *Plasmodium mexicanum*

**Kent D. Dunlap**[1]
**Jos. J. Schall**[2,*]
[1]Department of Zoology, University of Washington, Seattle, Washington 98195;
[2]Department of Biology, University of Vermont, Burlington, Vermont 05405

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**Abstract**

*When naturally infected with the malarial parasite* Plasmodium mexicanum, *Western fence lizards,* Sceloporus occidentalis, *exhibit numerous reproductive pathologies. Infected males display fewer courtship and territorial behaviors, have altered sexually dimorphic coloration, and have smaller testes. Infected animals store less fat by late summer, which, for females, reduces clutch size in the spring. We describe hormonal alterations associated with malarial parasitism that may underlie this reproductive inhibition. We caught lizards in the field and bled them either immediately or 1 h after capture for measurement of basal and stress levels of steroid hormones. Compared to uninfected lizards, infected lizards had lower levels of basal plasma testosterone (24 vs. 38 ng/mL) and higher levels of corticosterone (18 vs. 8 ng/mL) following 1 h of capture and confinement. Infected animals also had lower levels of plasma glucose (243 vs. 270 mg/dL). When we experimentally elevated plasma corticosterone of uninfected lizards in large outdoor enclosures, the animals showed decreases in testosterone, testis size, and stored fat similar to those of infected animals in the wild. We hypothesize that the parasite induces alterations in the adrenal response to acute stress, resulting in a suppression of testosterone and the inhibition of reproductive behavior.*

## Introduction

Parasites can significantly reduce the fitness of their hosts (Price 1980; Barnard and Behnke 1990). In some cases the sequence of events that leads

* To whom correspondence should be addressed.

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to this cost to reproductive success is obvious. For example, in parasitic castration of invertebrate hosts, parasites directly disrupt reproduction by damaging the host's germinal tissue (Baudoin 1975). Pathogens can also influence reproduction in more subtle ways. Infected males may have difficulty maintaining territories and attracting females (Schall and Dearing 1987), or the appearance of parasitized males can be altered such that females may be able to detect and avoid infected potential mates (Ressel and Schall 1989).

Studies of the Western fence lizard, *Sceloporus occidentalis,* infected with the malarial parasite *Plasmodium mexicanum* have documented numerous physiological and behavioral pathologies attributable to parasitic infection in the wild. Although many species of mammals, birds, and reptiles can carry malaria, lizards are the only nonhuman vertebrate hosts for which the behavioral and reproductive consequences of natural infection are known (Schall 1990*a,* 1990*b*). Male fence lizards infected with *P. mexicanum* have smaller testes and altered sexual coloration, exhibit fewer courtship and territorial behaviors, and store less fat during the warm season than uninfected males. Infected females store less fat by late summer than uninfected females, reducing the number of eggs they produce the next spring. In this study, we examine the hormonal alterations that accompany malarial infection in males that may contribute to their reproductive inhibition.

In many vertebrates, glucocorticoid hormones from the adrenal gland coordinate physiological responses to diverse noxious stimuli, such as disease, social aggression, inclement weather, and starvation (Greenburg and Wingfield 1987). Although glucocorticoids are critical for surviving such challenges, prolonged or repeated elevation of these hormones can have numerous deleterious effects, including inhibition of the reproductive and immune systems (Munck, Guyre, and Holbrook 1984; Sapolsky 1987). In males, glucocorticoids often inhibit reproduction by suppressing the production of testosterone, a gonadal steroid that is usually necessary for both the generation of sperm and the activation of courtship and territorial behaviors (Moore and Zoeller 1985; Greenburg and Wingfield 1987; Sapolsky 1987; Moore 1988). Elevated glucocorticoid levels can also directly inhibit territorial behavior in males even without a reduction in testosterone (DeNardo and Licht 1993). These findings suggested to us that malarial lizards might secrete more glucocorticoids in response to infection and that this elevated glucocorticoid level may cause the reproductive consequences observed in infected lizards, either directly or by altering testosterone secretion.

Infected fence lizards appear impaired both in long-term processes (e.g., fat storage, red blood cell formation, and testicular growth) and in short-term challenges (e.g., social interactions and aerobic exercise; Schall 1983*a*, 1990*b*). Adrenal hormones are secreted in response to both chronic stress and acute challenge (Moore, Thompson, and Marler 1991). We thus chose to measure both the basal levels of adrenal hormones and the adrenal response to acute challenge. We assayed the response of the adrenal gland by comparing plasma concentrations of corticosterone, the principle glucocorticoid in lizards (Callard and Callard 1978), immediately after capture and after 1 hr of confinement. Such capture stress has been used in many studies of free-living vertebrates as a way of applying standard stimuli to all individuals equally regardless of age, sex, and physiological state (Sapolsky 1987; Astheimer, Buttermer, and Wingfield 1992; Wingfield, Vleck, and Moore 1992). We also experimentally elevated corticosterone levels of uninfected lizards in large, outdoor enclosures to determine whether exogenous corticosterone can inhibit the reproductive system of *S. occidentalis* in ways observed in previous studies of animals infected with *P. mexicanum.*

## Material and Methods

*Field Study*

We captured lizards at the University of California Hopland Field Station near Hopland, California, approximately 140 km north of San Francisco. In this oak-savanna habitat, *Sceloporus occidentalis* are active from mid-April to late September. We conducted our study in June 1990, in the second half of the fence lizard breeding season. Schall has studied lizard malaria at this site for many years and describes the site and the biology of *Sceloporus-Plasmodium* interactions elsewhere (Schall 1983*a*, 1990*a*, 1990*b*).

We caught lizards by noose and took blood (~150 μL) from the postorbital sinus either immediately after capture (within 60 s) or after a 1-h confinement in a cloth bag (10 cm × 30 cm). We consider these our basal and stress measurements, respectively. Only males of > 57 mm snout-vent length (SVL) were bled, and no individual was bled more than once. We recorded the time each blood sample was taken to determine whether hormone levels fluctuate in a diel pattern.

After collection in the field, the blood was placed immediately on ice in a portable cooler for 3–4 h and then centrifuged in the lab at ~350 *g* for 10–15 min. The plasma was stored at −20°C.

After bleeding, the lizards were marked and brought into the lab to measure their SVL (mm), mass (g), and degree of infection by blood-borne *Plasmodium mexicanum.* For all lizards, we made smears from a drop of blood taken from toe clips and stained them with Giemsa. The slides were viewed to determine if the animal was infected, and, if so, the number of parasites per 10,000 red cells (parasitemia) was counted to determine the degree of infection. We estimated the age of the infection by tabulating the proportion of erythrocytes containing parasites in the asexual and gametocyte stages of the life cycle. Lizards with only asexuals were considered recent infections, while those with large vacuolated gametocytes were considered older infections (Bromwich and Schall 1986). We returned all lizards to the field within 1–3 d.

*Experimental Study*

To test the effects of exogenous corticosterone, we set up two 3.7 × 3.7 × 2.5-m screen tents in an open, grassy field. We placed logs and rocks in the tents to simulate the natural habitat and covered the edges of the tents to prevent the lizards from escaping. We implanted males with Silastic tubes (0.8 × 7.5 mm; Dow Corning 602-235) containing either corticosterone (Sigma C-2505; experimental group, $n = 9$) or nothing (control group, $n = 8$). The tubes were sealed at both ends and punctured slightly with a needle. The lizards varied in size (8–13 g, 59–69 mm SVL), so we distributed lizards of different sizes equally between the experimental and control groups. To anesthetize the animals, we placed them in a refrigerator for 15–20 min. While the lizards were held on ice, we made small cuts in the skin (~3 mm), placed the tubes subcutaneously, and resealed the cuts with Superglue. We released the animals into the screen tents ~1 h postoperatively and ~4 h after their initial capture.

Experimental and control lizards were distributed equally between the two tents. Each day, we fed them insects swept from the surrounding grass. Eight days after implantation, we collected blood immediately after capture (1200–1400 h) and froze the plasma and carcasses for later assays and dissection. The testes, liver, and fat bodies were removed and weighed to the nearest 1 mg. To compensate for variation among individual lizards in both body size and gut contents, we dissected the digestive tract from the animal, weighed the remaining carcass, and calculated organ size as a fraction of carcass weight.

*Measurement of Hormone and Glucose Concentrations*

We separated and measured the steroid hormones using column chromatography followed by radioimmunoassay (Wingfield and Farner 1975; Wingfield et al. 1992). Steroids were extracted from 20 μL of plasma with dichloromethane, suspended in 10% ethyl acetate in isooctane, and added to columns of diatomaceous earth (Sigma D-5384) containing propylene and ethylene glycol. Testosterone and corticosterone were eluted from the columns with increasing concentrations of ethyl acetate (20%–50%) to separate the hormones on the basis of their polarity. After resuspending the steroids in buffer, we incubated the samples with their respective tritiated steroid and antibody overnight at 4°C. The unbound hormone was removed by adding dextran-coated charcoal and centrifuging. The samples were run in two assays, and the intra- and interassay variations were 5.1% and 7.2%, respectively.

Blood glucose was measured with the glucose oxidase method in duplicate samples on a Beckman glucose analyzer.

*Statistics*

We tested for statistical differences between infected and uninfected and between experimental and control animals with ANOVA followed by Fisher's least significant difference test. When variables were correlated with body mass (testis, liver, and fat body mass), the data were log transformed and analyzed with ANCOVA with log body mass as a covariate. For these statistical tests, significance was defined at $P < 0.05$. For other correlations, we adjusted for experiment-wise error (Sokal and Rohlf 1981) by dividing this alpha level by 10, the number of comparisons. Therefore, for correlations, significance was defined at $P < 0.005$.

## Results

*Field Study*

Compared to uninfected lizards, malarial lizards had significantly lower levels of basal testosterone (37% lower, $F = 6.4$, $P < 0.05$; fig. 1; table 1) but no significant difference in stress levels of testosterone. Infected and uninfected animals had similar basal corticosterone, but infected lizards had higher stress levels (75% higher, $F = 7.1$, $P < 0.005$; fig. 1). These hormone levels are similar to those reported in other studies of *Sceloporus* lizards

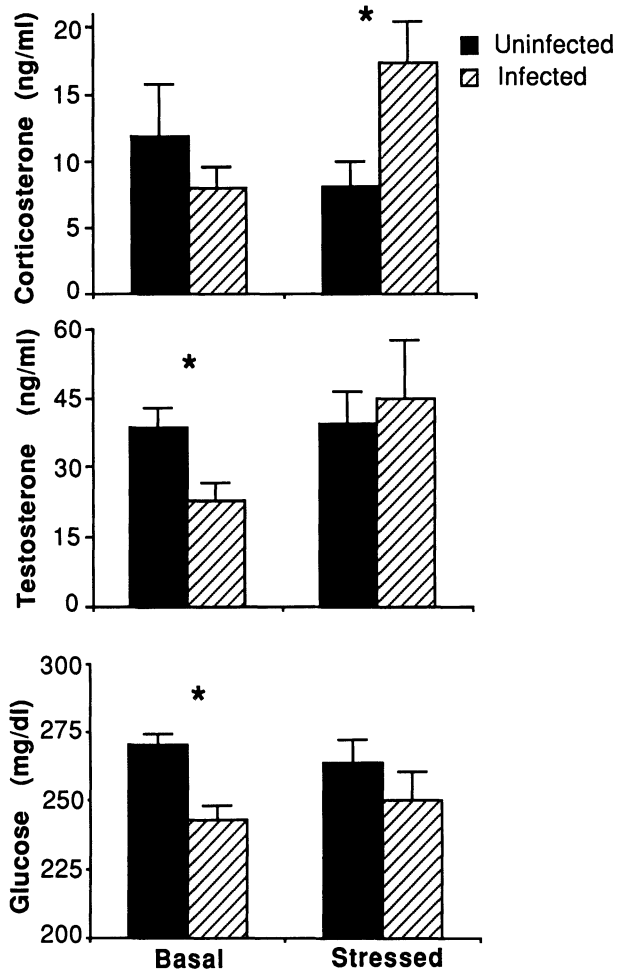


*Fig. 1. Plasma concentrations of hormones and glucose (mean ± SE) in uninfected (solid bars) and infected (striped bars) lizards. Basal measurements were taken immediately after capture, stressed measurements 1 h after capture. Asterisks indicate statistically significant differences between infected and uninfected animals.*

(McKinney and Marion 1985; Moore 1987*a*). In no case did steroid concentration vary with age of infection (recent vs. old), degree of infection (parasitemia), time of day, or SVL ($P > 0.05$).

Malarial lizards had significantly lower basal plasma glucose levels (10% lower, $F = 10.2$, $P < 0.005$; fig. 1) than uninfected lizards but did not differ significantly in stress levels of glucose. Among infected lizards, levels of basal glucose correlated negatively with parasitemia ($r^2 = .22$, $P < 0.005$).

TABLE 1

*Plasma hormones, glucose, and organ weights of lizards with experimentally elevated corticosterone and lizards naturally infected with malaria*

| | Experiment | | | Field | | |
|---|---|---|---|---|---|---|
| | Control ($n$ = 8) | Corticosterone ($n$ = 9) | $P$ Value | Uninfected ($n$ = 28) | Infected ($n$ = 24) | $P$ Value |
| Corticosterone (ng/mL) | 8.1 ± 1.6 | 47.5 ± 10.9 | <.005 | 11.5 ± 2.6 | 7.8 ± 1.4 | >.05 |
| Testosterone (ng/mL) | 39.6 ± 3.9 | 21.1 ± 6.1 | <.05 | 37.5 ± 3.9 | 23.5 ± 3.5 | <.01 |
| Glucose (mg/dL) | 265 ± 5 | 312 ± 11 | <.005 | 270 ± 4 | 243 ± 5 | <.005 |
| Testis mass (mg) | 164 ± 21 | 109 ± 10 | <.05 | 180[a] | 123[a] | |
| % Body mass | 1.9 ± .1 | 1.2 ± .1 | <.05 | 1.8[a] | 1.3[a] | |
| Fat body mass (mg) | 139 ± 18 | 94 ± 24 | .07 | 155[a] | 90[a] | |
| % Body mass | 1.5 ± .2 | 1.0 ± .2 | .06 | 1.5[a] | 1.0[a] | |
| Liver mass (mg) | 341 ± 49 | 365 ± 29 | >.05 | | | |
| % Body mass | 3.8 ± .4 | 4.0 ± .2 | >.05 | | | |
| Body mass[b] (g) | 8.7 ± .5 | 9.1 ± .4 | >.05 | 10.0[a] | 9.2[a] | |

Note. Values are mean ± SE. All field measurements are basal values (see fig. 1).

[a] Estimates based on data collected by Schall (1983*b*).

[b] Mass of carcass after removal of viscera.

*Experimental Study*

Exogenous hormone implanted into male lizards increased basal corticosterone concentration five- to sixfold, lowered testosterone levels (47% lower, $F = 5.7$, $P < 0.03$), reduced testis mass (34% lower, $F = 6.4$, $P < 0.025$), increased plasma glucose levels (17% higher, $F = 12.8$, $P < 0.005$), and lowered hematocrit (14% lower, $F = 6.1$, $P < 0.02$) compared to control-implanted animals (table 1). Fat body mass decreased with corticosterone administration, but this decrease just approached statistical significance ($P = 0.06$). Liver mass was unaffected by corticosterone treatment.

## Discussion

Male fence lizards, when infected with the malarial parasite *Plasmodium mexicanum,* spend less time in social interaction, are less able to maintain territories, have smaller testes, and have altered ventral colors when compared with noninfected males (Schall 1983*a,* 1983*b,* 1990*b;* Schall and Dearing 1987; Ressel and Schall 1989). Both males and females store less fat during the warm season; for all lizards this may reduce overwinter survival, and for females the reduced fat results in smaller clutches of eggs in the spring (Schall 1983*b*). Here we have shown that malaria-infected male lizards have elevated corticosterone levels when exposed to short-term capture stress and that basal plasma testosterone levels are reduced in infected males. Also, we produced pathologies that resembled malarial infection by experimentally elevating the corticosterone of uninfected lizards. These results suggest that the complex behavioral and reproductive changes observed in malarial lizards may result from parasite-induced alterations to those hormone systems that coordinate the generalized response to stress and regulate male reproductive behaviors and physiology.

*Infection, Steroids, and Reproduction*

Fence lizards infected with malaria suffered a reduction in levels of basal testosterone. In most vertebrates, full expression of male sexual and aggressive behavior during the breeding season requires elevated basal levels of gonadal steroids. For example, in the closely related lizard *Sceloporus jarrovi,* castrated males display fewer courtship behaviors

toward females and behave less aggressively toward intruding males than intact males (Moore 1987*b*). Both courtship behavior and aggression are fully restored by implanting castrated animals with exogenous testosterone (Moore 1988). Castrated *S. jarrovi* and infected *Sceloporus occidentalis* show a qualitatively similar decrease in social displays (e.g., head bobs and push-ups) in undisturbed observations and in staged encounters with males and females. Although castrated *S. jarrovi* have substantially less testosterone than most infected *S. occidentalis,* the qualitative similarity in their behavior suggests that the suppression of testosterone in infected lizards contributes to reduction in their reproductive behaviors.

In contrast to testosterone levels, basal levels of corticosterone were similar in infected and uninfected animals, but corticosterone levels 1 h after capture were greater in infected animals. It is interesting that *S. occidentalis* faced with drought conditions in the Mojave Desert show a similar pattern of adrenal secretion: an unchanged basal level of corticosterone secretion but a greater adrenal response to acute stress (Dunlap 1993). Future studies of hormonal responses to parasites and environmental stressors thus need to examine not only the basal hormone condition but also the hormonal responses to acute stress. The apparent absence of an adrenal response to parasitic infection in fish and birds reported by other researchers (Davison, Chapman, and Harvey 1985; Laidley, Woo, and Leatherland 1988; Rand and Cone 1990) may be due to the fact that they measured only basal corticosterone concentrations. Assessing the host's acute adrenal response is important because periodic surges of corticosterone, as well as chronically elevated corticosterone, may have detrimental effects on reproductive and immune functions.

In this study, uninfected lizards showed no rise in corticosterone after 60 min of confinement. This apparent absence of response during the breeding season has been found repeatedly in several populations of *S. occidentalis* (Dunlap and Wingfield 1993). However, in more detailed studies examining the time course of glucocorticoid responses in fence lizards, corticosterone rises consistently after longer periods of confinement during the breeding season and after the same period of confinement (60 min) in other seasons (Dunlap 1993; Dunlap and Wingfield 1995). In particular, uninfected lizards at the Hopland site showed significant elevation of corticosterone after 60 min of capture in August and after 240 min of capture in June. Consequently, the low levels of stress corticosterone in uninfected lizards in this study probably indicate that, during the breeding season, corticosterone rises more slowly than in other seasons rather than completely lacks a response to capture and confinement.

*Experimental Study*

The experimentally elevated plasma corticosterone in uninfected lizards resulted in a variety of changes that mimicked pathologies observed in malaria-infected animals: basal testosterone levels, testis mass, and fat body mass all decreased (table 1). We did not measure the behavioral response to exogenous corticosterone, but several studies in other lizards show that corticosterone dramatically affects male reproductive behavior. Corticosterone treatment reduced aggressive behavior, plasma testosterone, and testis size in brown anoles, *Anolis sagrei* (Tokarz 1987). Similarly, elevated endogenous corticosterone and enlarged adrenal glands are associated with subordinate behavior in green anoles, *Anolis carolinensis* (Greenberg, Chen, and Crews 1984), and six-lined race runners, *Cnemidophorus sexlineatus* (Bracklin 1978), respectively. Elevated corticosterone levels may also reduce male territorial displays more directly. Male *Uta stansburiana* with experimentally elevated corticosterone levels reduced social interactions even when testosterone was experimentally forced to normal levels (DeNardo and Licht 1993).

The pattern of corticosterone exposure differed between experimentally implanted and free-ranging animals, both parasitized and noninfected. Implanted animals experienced high basal levels for a short period (8 d), while infected animals presumably experienced short, amplified waves of corticosterone as they encountered periodic acute challenges during their 3-mo breeding season. We have no direct evidence that malarial lizards in fact secrete excess corticosterone during naturally occurring acute challenges. However, several studies in which natural stressors have been simulated suggest that encounters with predators or conspecifics can elicit rises in corticosterone. Uninfected, field-active fence lizards in the same population used in this study, when captured repeatedly (2–3 times) and held very briefly (15 s) to simulate predator attacks, showed heightened surges of corticosterone and reduced levels of basal testosterone (Dunlap 1993). Also, satellite male tree lizards (*Urosaurus*) have elevated corticosterone 24 h after an aggressive encounter with a conspecific in the field (R. Knapp and M. Moore, personal communication). The similar consequences of chronic and intermittent exposure to corticosterone in the present study suggest that tissues in the reproductive system can be similarly sensitive to long-term stressors and repeated, short-term stressors. This pattern of response has been found in the immune and body-weight response to corticosterone in mammals (Akana et al. 1985).

*Hypothesis and Alternative Explanations*

On the basis of our observations and the results of related studies, we propose the following hypothesis. Malarial infection induces alterations in the response of the adrenal axis to acute challenges such as predation attempts and social conflict. The excess corticosterone over time causes a decrease in both testis size and testosterone secretion and, consequently, the inhibition of reproductive behavior. Our field and experimental results both support this hypothesis. However, several alternative explanations need further examination.

First, lizards naturally infected with malaria showed altered patterns of steroid secretion, but we do not know that infection necessarily caused the observed hormonal differences between infected and uninfected lizards. The lizards showing the hormonal alterations in our study may simply be those that were originally more susceptible to infection with the parasite rather than those that were subsequently affected by the infection. Glucocorticoids suppress the immune system in many vertebrates (Munck et al. 1984; Bateman et al. 1989), including reptiles. Saad et al. (1987) showed that, in the scincid lizard *Chalcides ocellatus,* short and repeated exposure to exogenous corticosterone reduces immune function, and a seasonal rise in endogenous glucocorticoids coincides with their annual period of natural immunosuppression (Saad and El Ridi 1990). It is thus possible that, in our group of infected lizards, changes in adrenal hormones preceded infection, making these lizards more susceptible to subsequent infection. Both infection and the associated inhibition of testosterone may be consequences of an adrenal response to other, preexisting environmental stressors.

Second, the reproductive inhibition of infected lizards may be a direct effect of the malarial parasite on testicular function. Telford (1989) found encysted schizonts of *Plasmodium sasai* in the testicular connective tissue of the Japanese lizard *Takydromous tachydromoides,* but no data exist to show whether *P. mexicanum* infects the testes of *Sceloporus,* and, if so, whether it alters their function. In addition, reproductive inhibition may be an indirect consequence of malaria-induced malnutrition. Although glucocorticoids typically also rise during malnutrition in many vertebrates, the malnutrition experienced by infected lizards may exert inhibitory effects on the reproductive system through mechanisms independent of glucocorticoids. If the parasite acts directly on the testes or indirectly through malnutrition, alteration in adrenocortical secretion would be epiphenomenal and not causally related to reproductive inhibition.

To test these alternative explanations, future experiments will need to manipulate systematically the hormonal, nutritional, and infection status of

uninfected lizards and record the consequent effects on reproductive physiology and behavior. The current study provides a basis upon which such studies can develop.

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