Behav Ecol Sociobiol (2001) 49:289–295
DOI 10.1007/s002650000298

ORIGINAL ARTICLE

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Ectoparasite loads in free-ranging northern fence lizards, *Sceloporus undulatus hyacinthinus*: effects of testosterone and sex

Received: 23 June 2000 / Revised: 19 October 2000 / Accepted: 31 October 2000 / Published online: 20 December 2000

Abstract More knowledge of the proximate factors that influence parasite loads would help us understand the selective pressures faced by hosts and host-parasite evolution. Testosterone has been associated with increased parasite loads in vertebrates. Here we asked whether experimentally elevated testosterone affected ectoparasite loads in free-ranging northern fence lizards (*Sceloporus undulatus hyacinthinus*). Males were captured, given testosterone or sham implants, and released. In 2 consecutive years, testosterone-implanted males had significantly more ectoparasites at recapture than did controls. Additionally, ectoparasite loads were positively correlated with testosterone concentrations in unmanipulated males, and males had significantly more ectoparasites than did females. The results are consistent with an effect of testosterone on parasite loads. However, rather than elevated testosterone increasing mite loads in experimental males, it appeared that high testosterone inhibited a natural seasonal decline in mite loads. Testosterone-implanted males also lost body mass whereas controls gained mass. Among controls, those retaining the most ectoparasites over the course of the experiment experienced the smallest gains in body mass, suggesting that the mites are costly.

Keywords *Sceloporus undulatus* · Testosterone · Parasites · Trombiculid mites · Condition

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Introduction

Parasite effects on host fitness include castration (Baudoin 1975), transmission of debilitating diseases (Camin 1948), fewer matings (Hamilton and Zuk 1982; Schall and Dearing 1987; Johnson and Boyce 1991; Kavaliers and Colwell 1995; Møller et al. 1999), and decreased reproductive success (Møller 1990; Lopé et al. 1998; review in Møller 1997). In red grouse, parasites have recently been implicated as causal agents in population cycles (Hudson et al. 1998). In lizards, sons of mite-parasitized female *Lacerta vivipara* have decreased survivorship (Sorci et al. 1994), and malaria-infected *Sceloporus* lizards have reduced red blood cell volumes, reduced hemoglobin, and impaired physical stamina (Schall et al. 1982) as well as reduced fat stores, lower fecundity, and smaller testes (Schall 1983a, 1983b).

Proximate factors that influence parasite loads in nature include genetic background, season, and host age, size, sex, and hormonal state. Thus genetic heterozygosity has been implicated in resistance to ectoparasitic mites in lizards (Brown et al. 1995), older or larger lizards have more parasites (Christian and Bedford 1995; Schall and Marghoob 1995), seasonal shifts in parasite loads occur in diverse taxa (Zuk 1987; Mitchell 1989; Teel et al. 1998; Theodoropoulos et al. 1998), and parasites are more prevalent or abundant in males in many animals (Pickering and Christie 1980; Schall 1983b; Alexander and Stimson 1988; Mooring et al. 1996; Poulin 1996) including salamanders (Anthony et al. 1994), western fence lizards (Schall and Marghoob 1995), red jungle fowl (Zuk 1990) and reindeer (Folstad et al. 1989). In humans too, males usually suffer greater disease mortality (but see Garenne and Lafon 1998).

Concomitant effects of season, age, and sex suggest important relationships between the endocrine system and parasite loads, and are best studied for testosterone (Folstad and Karter 1992; Hillgarth and Wingfield 1997). In laboratory studies, experimentally elevated testosterone increases parasite loads for rainbow trout (Buchmann 1997) and increases the establishment and

reproduction of intestinal parasites in rodents, while castration reduces parasite loads (Mock and Nacy 1988; Harder et al. 1992). Field studies indicate likewise for several species (Weatherhead et al. 1993; Saino and Møller 1994; Saino et al. 1995; Salvador et al. 1996). For example, male red-winged blackbirds parasitized by mites had higher plasma testosterone (Weatherhead et al. 1993) and testosterone-implanted males harbor more ectoparasites in birds (Saino et al. 1995) and lizards (Salvador et al. 1996).

In the course of an unrelated experiment in 1996 (Klukowski et al. 1998), we observed significantly higher ectoparasite loads on free-ranging testosterone-implanted male fence lizards than on sham-implanted controls (see below). Testosterone-treated males also had significantly slower growth rates and smaller fat bodies than did controls. For the subsequent season, we made the following predictions and gathered appropriate data to test them:

- Growth rates should again be lower for testosterone-treated males than for sham-implanted controls.
- Ectoparasitic mites should again be more abundant on males with testosterone implants than on controls.
- Mite levels should be higher on males with higher levels of testosterone.
- Mite levels should be higher on unmanipulated males than on females.
- As testosterone may depress corticosterone levels (Péczely 1979), the latter should be lower in males with testosterone implants than in controls.

Methods

Host-parasite background

The northern fence lizard, *Sceloporus undulatus hyacinthinus*, occurs widely across the eastern United States. Plasma testosterone levels in males are highest during the spring breeding season (April–June) when males are intensely territorial, are lowest (i.e., basal) during the post-breeding season, and rebound in the fall as a new spermatogenic cycle begins (McKinney and Marion 1985; Klukowski and Nelson 1998).

Fence lizards are often parasitized by ectoparasitic trombiculid mite larvae (chiggers). On our lizards these were *Eutrombicula cinnabaris* (Ewing), a species common in the eastern USA (W. Wrenn, personal communication). The chiggers usually attach in a fold of skin between the lizard's ear and shoulder, termed the neck pocket (after Arnold 1986), by penetrating the host's skin with spearlike chelicerae and secreting a cement-like salivary material; an additional salivary secretion digests the host's cells (review in Arlian and Vyszenski-Moher 1987). After feeding, the engorged larvae fall off and transform into non-parasitic soil-dwelling nymphs (Wharton 1952).

Field experiment 1996

Twenty-two male fence lizards were captured by noosing between 7 and 16 August 1996 from the rocky shore and woodlands adjacent to Lake Monroe (Monroe County, Ind.). Natural levels of testosterone at this time are near basal. Individuals were given unique toe-clips and paint marks for future identification, ranked by mass and assigned sequentially to treatment groups (in order to have similar distributions of body sizes in the groups). As these males were initially part of an experiment with an entirely different purpose, no notes were taken of initial mite loads.

On the day of capture, lizards were transported to Indiana University, locally anesthetized by subcutaneous injection of 0.02 ml of 0.05% lidocaine anterior to the right hind limb and placed on ice to induce deep hypothermia. Silastic implants (3 mm packed length, inner diameter 1.47 mm, outer diameter 1.96 mm; Konigsberg Instruments, Pasadena, Calif.) were inserted into the peritoneal cavity through a small ventrolateral incision that was then closed with one suture and Nexaband glue. Implants were either filled with crystalline testosterone (Sigma, St. Louis, Mo.) or, for controls, were empty. Lizards were released the next day within a few meters of where they had been captured.

Five testosterone-implanted lizards and four controls were recaptured after 20 to 30 days in the field. Immediately upon recapture, each lizard was bled post-orbitally and the percentage of each neck pocket covered by mites was visually estimated. The time between release and recapture did not differ significantly for the two groups (U=4.5; P=0.17). The growth rates and mass of fat bodies of these males have been reported previously (Klukowski et al. 1998). In brief, testosterone-treated males had significantly slower growth rates and smaller fat bodies than did controls.

Field Experiment 1997

Between 21 and 24 July 1997, 32 male fence lizards were captured, treated as described above, and released. As in the 1996 experiment, natural levels of testosterone at this time were low. Twelve testosterone-implanted males and 9 controls were recaptured after 30–41 days. Immediately upon recapture, each lizard was bled post-orbitally and the number of mites in the neck pocket was estimated with the aid of a magnifying glass. Within 4 h, mites were counted in the laboratory using a dissecting microscope. In each case, there was close agreement between the field estimate and the laboratory count. In this 1997 sample, we also recorded whether males had shed their skin (and the initial paint mark) between release and recapture.

The groups did not differ in initial mite loads (see below) or in mean length of time between release and recapture [testosterone-implanted (T)-males=33.7±0.88 days (n=12), controls=34.0±1.07 days (n=9), T=–0.24, P=0.81]. Nonetheless, to correct for individual differences in the duration of treatment, we also report size-specific growth rates, k, where $k=(\ln s_2-\ln s_1)/(t_2-t_1)$, and s_1 and s_2 are sizes at times t_1 and t_2, respectively (Andrews 1982).

Sex difference 1997

As the lizards were being recaptured in 1997, unmanipulated sexually mature males (n=14) and females (n=19) were captured to test for a sex difference in ectoparasite loads. Mites were counted in the field with the aid of a magnifying glass and males were bled as described above.

Hematocrit and radioimmunoassay

Blood was centrifuged (10 min at 1,000 g) in heparinized microcapillary tubes. The hematocrit was determined from the ratio of red blood cell volume to total blood volume. The resulting plasma was frozen (–80°C) until assayed. The radioimmunoassay procedure has been described previously (Klukowski and Nelson 1998). In brief, 40-µl plasma samples were equilibrated with 41 Bq (2,000 cpm) of each label overnight, and then extracted once with 5 ml of ether. In 1997, steroids were separated from one another through celite chromatography using an increasing concentration of ethyl acetate in isooctane, and testosterone and corticosterone concentrations were determined through competitive binding of endogenous and labeled steroid for antibody-binding sites. In 1996, direct assays of total androgens (i.e., not involving chroma-

tography) were performed. Thus in 1996 we could not distinguish between testosterone and dihydrotestosterone. However, since levels of dihydrotestosterone in lizards are typically only 10% of testosterone levels (Crews et al. 1978; Grassman and Hess 1992), the 1996 androgens were probably largely testosterone. For the 1997 samples, intraassay coefficients of variation were 10.1% and 8.5% for testosterone and corticosterone, respectively. For the 1996 samples, the intraassay coefficient of variation for total androgens was 9%.

Statistics

Group means were compared with independent-sample *t*-tests if the parametric assumptions were met, otherwise the Mann-Whitney *U*-test was used. Similarly, either the Pearson correlation (*B*=regression coefficient) or the non-parametric Spearman rank correlation (R_s) test was used. Statistical tests used SYSTAT. Although rank-order statistics were sometimes used, for clarity we report means±1 SE. Unless otherwise stated, reported *P*-values are for two-tailed tests. Since we had no strong basis to predict effects of testosterone on mite loads in 1996, two-tailed tests were used. However, one-tailed tests were used in 1997 since we had the data from the previous year on which to base various predictions (see Introduction).

Results

Field experiment 1996

The implants elevated total plasma androgens (means: T-males=120.9±5.9, controls=4.6±4.0 ng/ml; *U*=0, *P*=0.014). Testosterone-implanted males had significantly more neck pocket area infested with mites than did controls [means: T-males=23.2% (*n*=5), controls=1.9% (*n*=4); *U*=0, *P*=0.014].

Each testosterone-implanted male was clearly parasitized, whereas two control males had no observable mites and the other two were very lightly parasitized.

Field experiment 1997

The testosterone-implants elevated plasma levels of testosterone (means: T-males=129.1±8.3, controls=7.7±2.8 ng/ml; *U*=0, *P*=0.0002). Average levels of corticosterone in testosterone-implanted males were half those in controls (means: T-males=8.1±1.2, controls=16.3±4.0 ng/ml; *U*=70, *P*=0.089). Neither testosterone nor corticosterone were significantly correlated with time of day, time to capture, time to bleed, total time to capture and bleed, final mite loads, or the change in mite loads within either group. Hematocrit levels were similar in the testosterone-implanted and control males (means: T-males=0.437±0.014, controls=0.45±0.029, *U*=41, *P*=0.93) and were also not correlated with final mite loads or the change in mite loads within either group (*P*>0.50).

At recapture, testosterone-implanted males had significantly more mites than did controls [means: T-males=31.5±6.80 mites (*n*=12), controls=13.4±6.98 mites (*n*=9); one-tailed *T*=1.82, *P*=0.04; Table 1]. The lizards that were recaptured had not differed in initial mite loads (means: T-males=36.6±12.33, controls=41.2±9.18; *T*=–0.30, *P*=0.77) nor initial body mass (means: T-males=10.84±0.737 g, controls=11.51±0.886 g; *T*=–0.58, *P*=0.57). The change in mite loads was not significantly different between the two groups (means: T-males=–5.1±13.24, controls=–27.8±9.37; two-tailed *T*=1.4, *P*=0.18).

Over the 30–41 days, testosterone-implanted males on average lost body mass, whereas controls gained mass (means: T-males=–0.843±0.230 g, controls=1.070+0.596 g; one-tailed *U*=88, *P*=0.008, Table 1). Correcting for differences in the duration of treatment by examining size-specific growth rates yielded similar results (means: T-males=–0.00213±0.00055, controls=0.00306±0.00152, *U*=92; *P*=0.007). Only among testosterone-implanted males was there a significant correlation between the plasma concentration of testosterone and the change in body mass (*B*=–30.85, Pearson R^2=0.736, *P*=0.00036; Fig. 1).

Controls with the greatest decreases in mite load gained the most in body mass (*B*=–11.29, Pearson

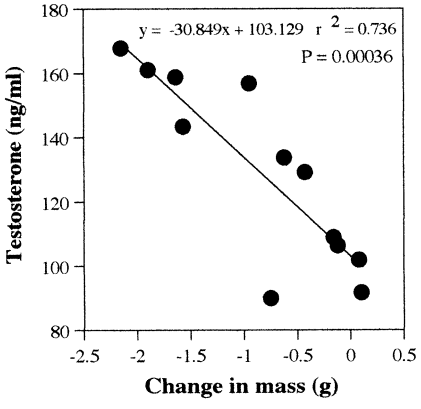

Fig. 1 Relationship between plasma testosterone concentration and change in body mass in the testosterone-implanted male fence lizards

Table 1 Mean (±SE) mite load and body mass (*BM*) of testosterone-implanted (T-males) and control-implanted (C-males) males in 1997 before and 30–41 days after hormonal manipulation. *P*-values are one-tailed

Group	Initial mites	Final mites	Change in mites	Initial BM	Final BM	Change in BM	*n*
T-males	36.6 (±12.33)	31.5 (±6.80)	–5.1 (±13.24)	10.84 (±0.737)	10.00 (±0.575)	–0.843 (±0.230)	12
C-males	41.2 (±9.18)	13.4 (±6.98)	–27.8 (±9.37)	11.51 (±0.886)	12.58 (±0.549)	1.07 (±0.596)	9
Statistic	*T*=–0.3	*T*=1.82	*T*=1.4	*T*=–0.58	*T*=–3.24	*U*=88	
P-value	0.38	0.04	0.089	0.29	0.002	0.008	

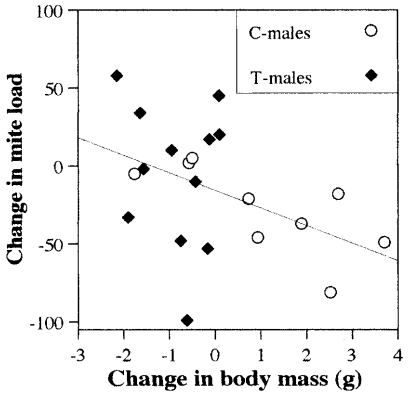

Fig. 2 Relationship between change in mite loads and change in body mass in testosterone-implanted (*T-males*) and control male (*C-males*) fence lizards. Only the regression line for control males is graphed (y=-11.29x-15.69; r^2=0.517, P=0.029)

R^2=0.517, P=0.029; Fig. 2). These variables were not associated in testosterone-implanted males (Spearman R_s=–0.04, P>0.50).

Skin was shed by significantly fewer testosterone-implanted males than controls (42 vs 89%; Fisher's exact test, P=0.037). Within the testosterone-implanted males, those that shed had similar changes in mite loads to those not shedding (mean change for shed: +9.4 mites, non-shed: –15.4; U=22, P=0.46). Within the testosterone-treated group, males that shed lost significantly more body mass than males that did not shed (means: shed: –1.54±0.160 g, non-shed: –0.35+0.295; U=3, P= 0.019).

Sex difference 1997

Unmanipulated males had significantly more mites than did females [means: males=11.1±2.7 mites (n=14), females=3.7±1.42 (n=19], T=2.42, P=0.025). Unmanipulated males had mean plasma testosterone levels of 4.1±1.2 ng/ml and mean corticosterone levels of 7.9±2.2 ng/ml. Mite loads for unmanipulated males were significantly positively correlated with plasma levels of testosterone (Spearman R_s=0.47, P<0.05, one-tailed) but not of corticosterone.

Discussion

Several lines of evidence suggest that testosterone affected mite loads in these fence lizards. First, in each of 2 consecutive years, testosterone-implanted males had significantly greater mite loads at recapture than did controls. This was not a pharmacological effect, as the implants raised plasma testosterone only to levels found in free-ranging males (Klukowski and Nelson 1998), albeit at an unusual time. Second, there was a significant positive correlation between ectoparasite loads and plasma testosterone levels in freshly captured, unmanipulated males. Third, these unmanipulated males had significantly greater mite loads than did females, although this by itself could also be explained by androgen-independent sex differences in behavior or microhabitat use (see below).

Experimental elevation of testosterone levels did not increase ectoparasite loads after 30–41 days of treatment (Table 1). Instead, elevated testosterone apparently inhibited a natural seasonal decline in mite loads as seen in controls but not in testosterone-treated males (Table 1). Additional data on seasonal variation in mite loads and effects of testosterone are clearly needed.

Although fewer testosterone-implanted males than controls shed their skin, there was no evidence that ecdysis rid lizards of their ectoparasites. Within the testosterone-implanted males, those that shed gained on average 9.4 mites, while those not shedding tended to lose mites.

An effect of testosterone on ectoparasite loads may occur more generally in lizards than previously appreciated. This is the second field study of lizards indicating such an effect. Testosterone-implanted male *Psammodromus algirus* had significantly greater increases in *Ixodes* tick loads than did controls (Salvador et al. 1996). The similarity in results is striking given that the two species are only distantly related (*Sceloporus*: Phrynosomatidae; *Psammodromus*: Lacertidae) and that in both studies, effects were observed in a relatively short time frame (30–41 days and about 48 days). Given the relative ease of counting ectoparasites, additional studies should examine the generality of the effect of testosterone on ectoparasites in lizards.

In other vertebrates, many field studies have also indicated an effect of testosterone on ectoparasite loads (citations in Introduction). In addition, laboratory studies indicate an effect of testosterone on internal parasite loads in, for example, mice (Swanson et al. 1984; review in Folstad and Karter 1992; Barnard et al. 1996). However, the only lizard study that examined internal parasite loads failed to find an effect of testosterone (Veiga et al. 1998).

There are several hypotheses for how testosterone treatment may increase parasites. One of the most cited is testosterone-mediated immunosuppression. Several laboratory studies have shown that elevated levels of testosterone can suppress immune function (fish: Slater and Schreck 1993, 1997; Slater et al. 1995; reptiles: Saad and Shoukrey 1988; Saad et al. 1992; chicken: Arnold and Holt 1995; mammals: Swanson et al. 1984; Grossman 1985; Alexander and Stimson 1988). However, these laboratory studies have examined the effects of levels of testosterone far greater than those seen in nature. Furthermore, there are no published field studies that convincingly link testosterone and immunosuppression, though there is some correlative evidence for it (Zuk et al. 1995; Veiga et al. 1998). Most recently, a robust laboratory test of immunosuppression in black-headed gulls failed to find any evidence of immunosuppression; in fact, the humoral immune response was actually enhanced in some cases by testosterone (Ros et al. 1997).

Thus, while an effect of testosterone on parasite loads has been established in the field for several species, there is still no strong evidence causally linking testosterone-mediated immunosuppression to increased parasitism. This has led some researchers to suggest that testosterone treatment may not cause immunosuppression per se, but rather immunoredistribution or resource reallocation (Wedekind 1992; Braude et al. 1999). Alternatively, there may be no direct link between testosterone and immunosuppression. Instead, poor body condition itself, indicative of energetic balance, may dictate immunosuppression (Møller and Saino 1994; Sheldon and Verhulst 1996). Numerous factors, in addition to testosterone, can lead to poor body condition. Finally, in some cases, the immunosuppression, if it occurs, may not result directly from testosterone but another hormone, such as a corticosteroid, that itself is influenced by testosterone. Corticosterone is known to reduce immune function in lizards (Saad et al. 1987; Saad and El Ridi 1990) and other reptiles (Morici et al. 1997).

The possibility that behavioral differences are the real cause of the association between testosterone and parasite burdens is one of the reasons testing immunosuppression in field studies has been so difficult. Testosterone-treated male lizards may have greater ectoparasite loads because their behavior puts them at greater risk of (1) encountering mites (encounter hypothesis), or (2) attracting mites (mite attraction hypothesis). These two hypotheses need not be mutually exclusive.

There is inferential evidence to support the encounter hypothesis in lizards. For example, a chigger species that commonly parasitizes reptiles is principally active from early morning to evening (Wharton 1952). Testosterone increases the daily activity period of lizards in the field (Marler and Moore 1989; DeNardo and Sinervo 1994) probably making them more likely to encounter a chigger and become parasitized. Similarly, testosterone treatment increases home range size in lizards (Fox 1983; DeNardo and Sinervo 1994), again making them more likely to encounter mites. Finally, chigger numbers vary substantially among microhabitats (Wharton 1952; Clopton and Gold 1993). Thus, testosterone-implanted lizards may encounter more chiggers if they spend more time in microhabitats that harbor greater numbers of chiggers; this remains to be tested.

Alternatively, testosterone may alter a lizard's physiology or behavior in such a way as to make a male more attractive to mites. For example, chiggers are activated and attracted by carbon dioxide, which plays an important role in host location (Sasa 1961; Arlian and Vyszenski-Moher 1987). As mentioned above, testosterone treatment has been shown to increase activity period and, in addition, it increases the field metabolic rates of lizards (Marler et al. 1995) making this a viable hypothesis. Similarly, testosterone has been shown to increase display frequency in lizards (Adkins and Schlesinger 1979; Moore and Marler 1987; M. Klukowski, C.E. Nelson, unpublished data). This increase in physical activity may attract mites, some species of which have been shown to be attracted to moving objects (Wharton 1952). Finally, host odor is important in finding a host, as shown for snake mites (Camin 1953) among others (Egan et al. 1975). Testosterone-treated male fence lizards might exude a more powerful or more attractive odor.

While these behavioral hypotheses are difficult to test, they nonetheless should be investigated along with the immune hypotheses as the three hypotheses are not mutually exclusive, though one may be more important biologically. Indeed, for fence lizards at least, both immune and behavior hypotheses are viable. Earlier work with this species has shown numerous behavioral effects of testosterone (Klukowski et al. 1998; unpublished data), and mites, including the species studied here that occurs on these fence lizards (Wright et al. 1988), are known to elicit immune responses in vertebrates (Wikel 1982; Goldberg and Holshuh 1992).

Are these ectoparasites costly? Most testosterone-implanted males lost mass whereas most control males gained mass, yielding a significant difference in weight change. This agrees with earlier studies of testosterone-treated lizards (Marler and Moore 1989; Klukowski et al. 1998). The extent to which this effect on growth is a direct effect of testosterone versus an effect of increased parasite loads remains to be determined. However, the significant negative correlation found between change in mite loads and change in body mass in control males suggests that these ectoparasites are inflicting at least some cost. Finally, several studies have shown that ectoparasites can be quite costly in reptiles (Camin 1948; Dunlap and Mathies 1993; Sorci and Clobert 1995; Salvador et al. 1996) as well as in other vertebrates (O'Kelly and Seifert 1970; Rechar et al. 1980; Møller 1990; Aeschlimann 1991; Lehmann 1993; Lopé et al. 1993).

Acknowledgements We thank William Wrenn for identifying the mites, Ellen Ketterson for the use of her laboratory to run the radioimmunoassays, Lori Klukowski for help catching lizards, and three anonymous reviewers for helpful comments. This research was supported by Sigma Xi and a Gaige Grant from the American Society of Ichthyologists and Herpetologists. Lizards were collected with the permission of the Indiana Department of Natural Resources. Animal care and use conformed to Indiana University guidelines.

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