Effects of Testosterone on Locomotor Performance and Growth in Field-Active Northern Fence Lizards, Sceloporus undulatus hyacinthinus

Author(s): Matthew Klukowski, Craig E. Nelson and Nicole M. Jenkinson

Source: *Physiological Zoology*, Vol. 71, No. 5 (September/October 1998), pp. 506-514

Published by: The University of Chicago Press. Sponsored by the Division of Comparative Physiology and Biochemistry, Society for Integrative and Comparative Biology

Stable URL: https://www.jstor.org/stable/10.1086/515949

Effects of Testosterone on Locomotor Performance and Growth in Field-Active Northern Fence Lizards, *Sceloporus undulatus hyacinthinus*

Matthew Klukowski[1,2]
Nicole M. Jenkinson[2]
Craig E. Nelson[1,2]
[1]Center for the Integrative Study of Animal Behavior, Indiana University, Bloomington, Indiana 47405;
[2]Department of Biology, Indiana University, Bloomington, Indiana 47405

Accepted 3/16/98

ABSTRACT

The role of steroids in locomotor performance and growth was examined in free-living lizards. Male northern fence lizards (*Sceloporus undulatus hyacinthinus*) with experimentally elevated plasma testosterone concentrations had greater sprint speed (+24%) and burst stamina (+17%) than sham-implanted males after 14–23 d in the field. This enhanced performance was associated with significant energetic costs, as the testosterone-implanted lizards had reduced growth rates, and, in a companion experiment, field-active testosterone-implanted lizards had smaller fat-body masses than controls after just 3–4 wk. These results suggest that, in addition to influencing a variety of behavioral and morphological traits, testosterone may play an important role in the regulation of locomotor performance. Also, natural levels of locomotor performance may be constrained, in part, by associated costs of elevated plasma testosterone concentrations.

Introduction

The locomotor performance capacity of an individual is likely to have important fitness consequences. For instance, sprint speed is known to influence prey-capturing success (Avery et al. 1982; Webb 1986), social dominance (Garland et al. 1990), and the success with which lizards escape their predators (Shine 1980; Christian and Tracy 1981; Huey and Hertz 1984). Stamina should be especially important in actively foraging species, as well as in species where success in winning territorial interactions depends on extended periods of displaying or fighting.

Physiological Zoology 71(5):506–514. 1998. 0031-935X/98/7105-97105$03.00

Given the presumed importance of locomotor performance to an individual's fitness, locomotor performance should generally be maximized. However, several workers have shown that locomotor performance varies seasonally (Bennett 1980; John-Alder 1984; Garland and Else 1987). In some species, the period of maximal locomotor performance coincides with that of enlarged testes and thus, presumably, with elevated plasma testosterone concentrations (Goldberg 1974).

Experimentally elevated testosterone has been shown to increase the stamina of *Anolis sagrei* (John-Alder 1994), *Uta stansburiana* (B. Sinervo, personal communication), and *Sceloporus undulatus* (H. B. John-Alder, personal communication). Recently, John-Alder et al. (1996) have shown in *S. undulatus* that endurance can be modulated by the social environment; the effect is dependent on the presence of intact testes and thus appears to require androgens.

Evidence that locomotor performance is sometimes submaximal suggests that a cost is associated with the enhanced performance. If such a cost exists and the benefits of enhanced performance vary seasonally, then natural selection may have linked the expression of the trait to seasonal changes in plasma steroid concentrations.

The costs associated with enhanced locomotor performance have not yet been studied, but those associated with elevated plasma concentrations of testosterone have received attention. In particular, Marler and Moore (1988, 1991) presented evidence that male mountain spiny lizards (*Sceloporus jarrovi*) with experimentally elevated testosterone suffer reduced survivorship relative to sham-implanted males. Salvador et al. (1996) report similar results for *Psammodromus algirus.* In a polymorphic population of *U. stansburiana,* the ultraterritorial male morph has both greater plasma testosterone concentrations and greater mortality than the other two male morphs (B. Sinervo, personal communication).

Potential costs of enhanced performance include an increased energy expenditure required for enhanced performance or for muscle tissue growth and, possibly, trade-offs among performance variables, most notably sprint speed and stamina. In mammals, a trade-off between sprint speed and stamina is well supported. For example, Dohm et al. (1996) report a negative genetic correlation between these two performance variables in mice. As Garland (1988) points out, there are several physiological reasons to expect a trade-off. However, reptilian locomotor physiology seems to be fundamentally different from that of mammals. Several studies on reptiles report no trade-off between these two performance variables (Bennett

1980; Garland 1984; Garland and Else 1987; Sorci et al. 1995). The apparent contrast with mammalian physiology must be interpreted with caution, however, because an absence of phenotypic correlations does not exclude the presence of genetic correlations (see Dohm et al. 1996).

The present study evaluated whether elevations of plasma testosterone concentrations enhanced locomotor performance (sprint speed, burst stamina, or both) in field-active northern fence lizards (*Sceloporus undulatus hyacinthinus*) and whether elevated testosterone was associated with increased costs as measured in terms of either growth or fat-body mass.

Lizards in the population studied, near the northern limit of the species' range (Conant and Collins 1991), are active from April through September. The most intense activity occurs during the breeding season, usually from May through June. During the latter period, males engage in aggressive territorial encounters that sometimes last over 15 min and include wrestling, biting, and vigorous displays (M. Klukowski, unpublished observations).

Material and Methods

Thirty males were captured by noosing in Monroe County, Indiana, between July 6 and 12, 1995. At capture, individuals were given unique toe clips and paint marks for future identification. Lizards were ranked by mass and assigned sequentially to treatment groups in order to have similar distributions of body sizes in the two groups.

On the day of capture, lizards were transported to Indiana University for implantation. Lizards were locally anesthetized by injection (subcutaneous) of 0.02 mL of 0.05% lidocaine anterior to the right hind limb and then placed on ice to induce hypothermia. Silastic implants (3 mm packed length, inner diameter 1.47 mm, outer diameter 1.96 mm; Konigsberg Instruments, Pasadena, Calif., catalog no. VST058077) were inserted into the peritoneal cavity through a small ventrolateral incision. Testosterone implants were filled with crystalline testosterone (Sigma, St. Louis, Mo., catalog no. T-1500); sham implants were empty. On average, the empty sham implants weighed 0.004 g less than the filled implants. Sutures and Nexaband glue were used to secure the wound. In all cases, lizards were released the day after capture and within a few meters of where they were captured.

Ten testosterone-implanted lizards and nine sham-implanted lizards were recaptured after 14–23 d in the field. The length of time between release and recapture did not differ significantly for the two groups ($P = 0.29$). During this same period, an additional 15 male lizards were captured to serve as double controls (untreated males). Lizards were transported to the laboratory and tested, the same day they were captured, between 1300 and 1800 hours.

Locomotor Performance

Before their locomotor performance was tested, lizards were placed in a 35° ± 2°C environmental chamber for 1 h, to control for the effect of temperature on performance (Hertz et al. 1983; Huey and Dunham 1987). This temperature is close to the reported mean field-active body temperature for this species (Crowley 1985; John-Alder 1990). The experimenter conducting the locomotor tests was blind to the treatment of each lizard.

Sprint speed was measured by filming lizards as they ran along a linear (2 m × 9 cm) track. The track had opaque Plexiglas sides and a substrate of fiberglass, mineral-surfaced roll roofing material, marked in 0.25-m intervals.

Lizards were stimulated to run at apparently maximal speeds by chasing them down the track and touching them on the tail with a plastic rod if they stopped. Lizards readily ran toward a blackened hide-box that was placed at the end of the track. Each lizard was tested twice, with 1 h of rest between tests (Huey and Dunham 1987; Sinervo and Adolph 1989). Between tests, lizards were kept in the 35°C environmental chamber.

Sprint speed was calculated from the videotapes (Garland 1984). The video camera filmed at 30 frames per second; extrapolation between frames yielded an effective resolution of 1/60 s. An individual's fastest 0.25 m out of the two tests was used in the analysis as maximum sprint speed. We also report for each individual the faster of the two initial 0.5-m starts and the fastest of any 0.5-m interval.

One hour after the second sprint test, burst stamina was tested twice, with 1 h of rest between tests. To measure burst stamina, lizards were stimulated to run around a banked oval track (perimeter 3.85 m, width 11 cm). This track was also constructed with Plexiglas walls but had an artificial-turf substrate. As in the sprint tests, lizards were stimulated to run by touching them on the tail with a plastic rod.

Burst stamina was defined as the total distance run in 90 s. This duration was chosen because, in preliminary tests, males stopped running at approximately this time. Our measure of burst stamina is analogous to distance capacity and running stamina reported by earlier workers (Bennett 1980; Schall et al. 1982). An individual's longest distance out of the two trials was used in the analysis as maximum burst stamina. We also report qualitatively how mean velocity changed with each succeeding lap. We include data only through the twelfth lap, because after this lap very few lizards continued to run.

Allometry

To estimate the relationship between body size and sprint speed independent of treatment, log sprint speed was regressed on log body size using data from only the 15 untreated males. Sham-implanted males were not included because their sprint speeds differed significantly from those of the untreated males.

Similarly, log maximum burst stamina was regressed on log body size, but data from all 24 control males (nine sham-implanted and 15 untreated males) were used. In those cases where locomotor performance scaled allometrically on body size, we corrected for size differences in all groups by dividing the performance variable by the size variable scaled to the appropriate allometric exponent (b), for example, $\log_{10}$(stamina/body mass^b) (Garland et al. 1987). Analyses used the size-corrected variables.

Hematocrit and Radioimmunoassay

Two and a half to three hours after the final performance test (see below), each lizard was bled postorbitally. Corticosterone levels were probably still elevated by the stress of testing at this time (Moore et al. 1991; Gleeson et al. 1993). Blood was centrifuged (10 min at 1,000 *g*), and hematocrit was determined from the ratio of red blood cell volume to total volume of blood in heparinized microcapillary tubes. The resulting plasma was frozen (−80°C) until assayed.

In general, radioimmunoassay followed the methods of Ketterson et al. (1991). 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 dichloromethane. Testosterone and corticosterone were separated from one another through celite chromatography using an increasing concentration of ethyl acetate in isooctane. Steroid concentrations were determined through competitive binding of endogenous steroid and labeled steroid for antibody binding sites. The intra-assay coefficients of variation for testosterone and corticosterone were 19% and 14.6%, respectively. Mean recovery for testosterone was 69%, and 78% for corticosterone. Sample sizes for the steroid data were smaller than for the hematocrit data because not enough plasma was obtained from some of the lizards to run a radioimmunoassay.

Growth Rate and Fat-Body Mass

Size-specific growth rates were defined as 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). In order to replicate measures of growth and to determine whether elevated testosterone levels affected the mass of fat bodies, in early August 1996 an additional 22 male fence lizards were captured and implanted with testosterone or sham implants, as described above. Because of inclement weather, we were able to recapture only five testosterone-implanted males and four sham-implanted males after 21–30 d. Fat bodies were dissected and weighed to the nearest 0.1 mg. Recapture time was not significantly different for the treatment groups ($P = 0.17$).

Statistics

Because the ratio of the largest group sample size to the smallest exceeded 1.5 and variances were nonhomogeneous, nonparametric statistics were used in comparisons among the groups (Hays 1991), unless otherwise stated. In addition, ANCOVA could not be used to control for size differences among groups because of the significant interaction between treatment and body mass (i.e., slopes were not homogeneous). Where a significant difference was found among all groups using Kruskal-Wallis tests, we performed Mann-Whitney U-tests to determine which pair comparisons were different. For all statistics, P-values are two-tailed. Although rank-order statistics were used, for clarity we report means ± 1 SE.

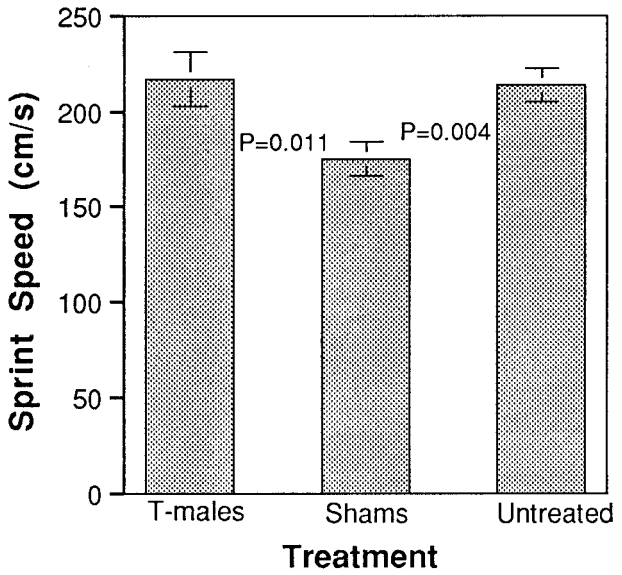

Figure 1. Maximum 0.25-m sprint speed of testosterone-implanted (T-males), sham-implanted, and untreated male *Sceloporus undulatus*. Sprint speed was greater in testosterone-implanted ($n = 10$) and untreated males ($n = 15$) than in sham-implanted males ($n = 9$). See "Results" for additional data on maximum 0.5-m and initial 0.5-m sprint speed. Values presented are means ± 1 SE.

Results

Sprint Speed

Sprint speeds in the two trials were significantly correlated ($P = 0.0001$; $r = 0.61$; $b = 0.57$; $n = 34$), indicating that differences among individuals were repeatable across trials. Absolute values of maximum sprint speed in the two trials were similar ($P = 0.30$, paired t-test; $n = 34$). Because speed was not significantly related to either body mass ($P = 0.79$; $r = 0.075$; $b = 0.087$; $n = 15$) or snout-vent length ($P = 0.70$; $r = 0.109$; $b = -0.39$; $n = 15$) in the untreated males, untransformed data are presented (Fig. 1). The three groups differed significantly in maximum sprint speed ($P = 0.0087$; testosterone-implanted = 217.1 ± 14.2 cm/s; sham-implanted = 175.2 ± 9.2; untreated = 214.0 ± 8.7), with sham-implanted lizards

being significantly slower than both the testosterone-implanted ($P = 0.011$) and the untreated males ($P = 0.0044$). The latter groups did not differ ($P = 0.95$).

The three groups almost differed significantly in their fastest 0.5-m speed ($P = 0.056$; testosterone-implanted = 99.0 ± 6.8 cm/s; sham-implanted = 80.8 ± 5.4; untreated = 96.7 ± 5.1), but not in mean velocity over the initial 0.5 m ($P = 0.28$; testosterone-implanted = 81.6 ± 4.9 cm/s; sham-implanted = 70.6 ± 4.9; untreated = 82.3 ± 5.6). In both cases, however, the sham-implanted animals were noticeably the slowest.

Burst Stamina

The two burst stamina trials were significantly correlated ($P = 0.00003$; $r = 0.65$; $b = 0.52$; $n = 34$), again indicating repeatability. Absolute values of burst stamina, however, were significantly less in the second trial ($P = 0.026$, paired t-test; $n = 34$), indicating that lizards were slightly fatigued. Maximal burst stamina varied significantly with body mass ($P = 0.021$; $r = 0.47$; $b = -0.42$; $n = 24$). Hence, we divided an individual's burst stamina by (body mass)$^{0.42}$ and used this size-corrected variable in analyses.

The three groups differed significantly in size-corrected maximum burst stamina measured as the total distance run in 90 s ($P = 0.020$; mean $\log_{10}$ [meters run/body mass$^{0.42}$]: testosterone-implanted = 1.972 ± 0.011; sham-implanted = 1.932 ± 0.01; untreated = 1.915 ± 0.015). Testosterone-implanted lizards had significantly greater burst stamina than both sham-implanted lizards ($P = 0.018$) and untreated males ($P = 0.013$), but the latter groups did not differ significantly ($P = 0.70$). When the trials were examined separately, the three groups still differed significantly in burst stamina (trial 1, $P = 0.001$; trial 2, $P = 0.00008$), with means in the same pattern as above. The three groups also differed significantly in uncorrected maximum burst stamina ($P = 0.0005$; testosterone-implanted = 38.5 ± 0.56 m; sham-implanted = 32.99 ± 0.9; untreated = 31.96 ± 1.21; Fig. 2).

Body mass was significantly correlated with mean velocity per lap for the 24 controls in only one of the 12 laps per trial (lap number 8 in the first trial and lap number 2 in the second trial). In both trials, testosterone-implanted males had greater stamina than both sham-implanted and untreated males, as measured by their greater mean velocity per lap for each of the 12 laps (Fig. 3). The same was true of the size-corrected data (not shown).

Hematocrit and Plasma Hormone Levels

The three groups did not differ significantly in hematocrit ($P = 0.34$; mean: 0.360 ± 0.008, 0.386 ± 0.016, and 0.389 ± 0.015, for the testosterone-implanted, sham-implanted, and untreated males, respectively).

The three groups differed significantly in plasma testosterone ($P = 0.0015$; testosterone-implanted = 122.0 ± 21.6 ng/mL; sham-implanted = 2.57 ± 0.32; untreated = 4.92 ± 1.41; Fig. 4*A*). Testosterone-implanted lizards had significantly greater plasma testosterone concentrations than both sham-implanted ($P = 0.0027$) and untreated males ($P = 0.0011$), but the latter two groups did not differ significantly ($P = 0.56$). The three groups also differed significantly in plasma corticosterone ($P = 0.0011$; testosterone-implanted = 2.98 ± 1.50 ng/mL; sham-implanted = 31.8 ± 7.68; untreated = 27.31 ± 6.03; Fig. 4*B*). Testosterone-implanted lizards had significantly lower levels of plasma corticosterone than both the sham-implanted lizards ($P = 0.0015$) and the untreated males ($P = 0.0012$), but the latter two groups did not differ significantly ($P = 0.63$).

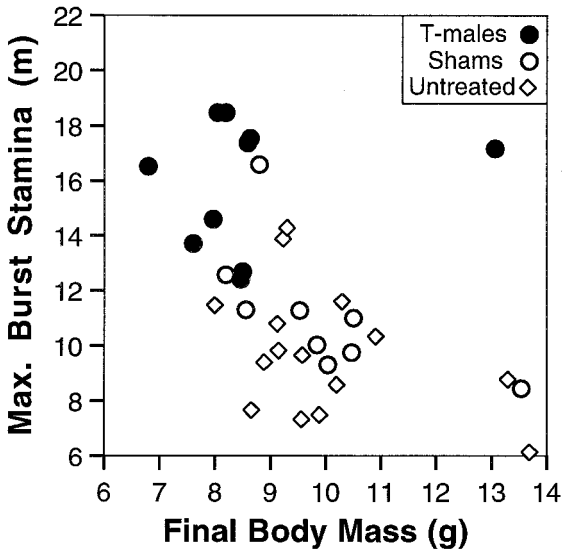

Figure 2. Maximum burst stamina (distance run in 90 s) versus final body mass of testosterone-implanted (T-males), sham-implanted, and untreated male *Sceloporus undulatus*. Size-corrected burst stamina was greater in testosterone-implanted ($n = 10$) than in either sham-implanted ($n = 9$) or untreated males ($n = 15$). Burst stamina was size-corrected as: $\log_{10}$(burst stamina/body mass$^{0.42}$) in the analyses. Uncorrected, original values of distance run are graphed.

Growth Rate and Fat-Body Mass

Testosterone-implanted and sham-implanted males that were later recaptured had not differed significantly at the time of surgery in body mass ($P = 0.22$; mean: 8.98 ± 0.72 g with range: 7.1–15 g [$n = 10$] and mean: 9.35 ± 0.49 g with range: 7.9–12.8 g [$n = 9$], respectively) or in snout-vent length ($P = 0.28$; mean: 64.7 ± 1.20 mm with range: 62–74 mm and mean: 65.40 ± 1.00 with range: 62–72 mm, respectively). By the time of testing, however, the testosterone-implanted males had lost mass, the sham-implanted males had gained mass,

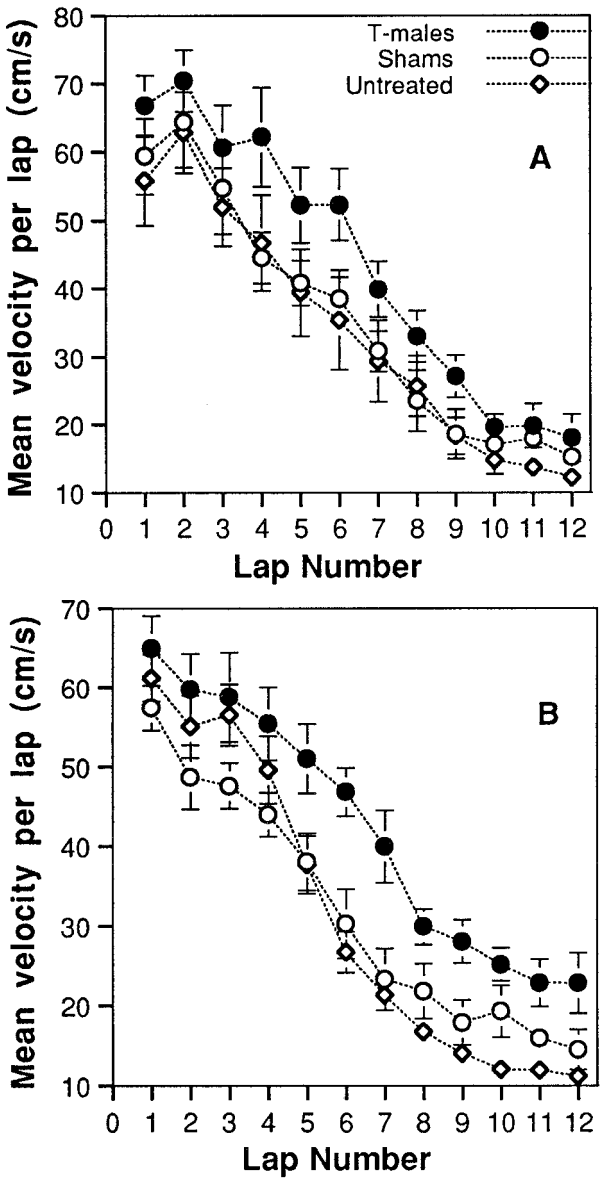

Figure 3. Mean velocity per lap (±1 SE) for each lap in trial 1 (*A*) and trial 2 (*B*). For each of the 12 laps in both trials, testosterone-implanted males (T-males) were faster than both sham-implanted males and untreated males.

and the differences among the three groups were statistically significant ($P = 0.005$; mean: 8.59 ± 0.53 g [$n = 10$], 9.94 ± 0.53 [$n = 9$], and 9.99 ± 0.41 [$n = 15$] for the testosterone-implanted, sham-implanted, and untreated males, respectively). The testosterone-implanted males were significantly lighter than both the sham-implanted and the untreated males ($P = 0.013$ and $P = 0.0019$, respectively), but the latter two groups did not differ in mass ($P = 0.98$). The masses were not biased by differential tail loss, as no males lost their tails. The three groups did not differ significantly in final snout-vent length ($P = 0.24$; mean: 65.5 ± 1.04 mm, 67.7 ± 1.07, and 66.8 ± 0.82, for the testosterone-implanted, sham-implanted, and untreated males, respectively).

Sham-implanted males had significantly greater size-specific body mass growth rates than testosterone-implanted males ($P = 0.014$; mean: 0.0037 ± 0.00081 and −0.0024 ± 0.0016, respectively). The differences in growth in snout-vent length followed the same pattern and were almost significant (*P*

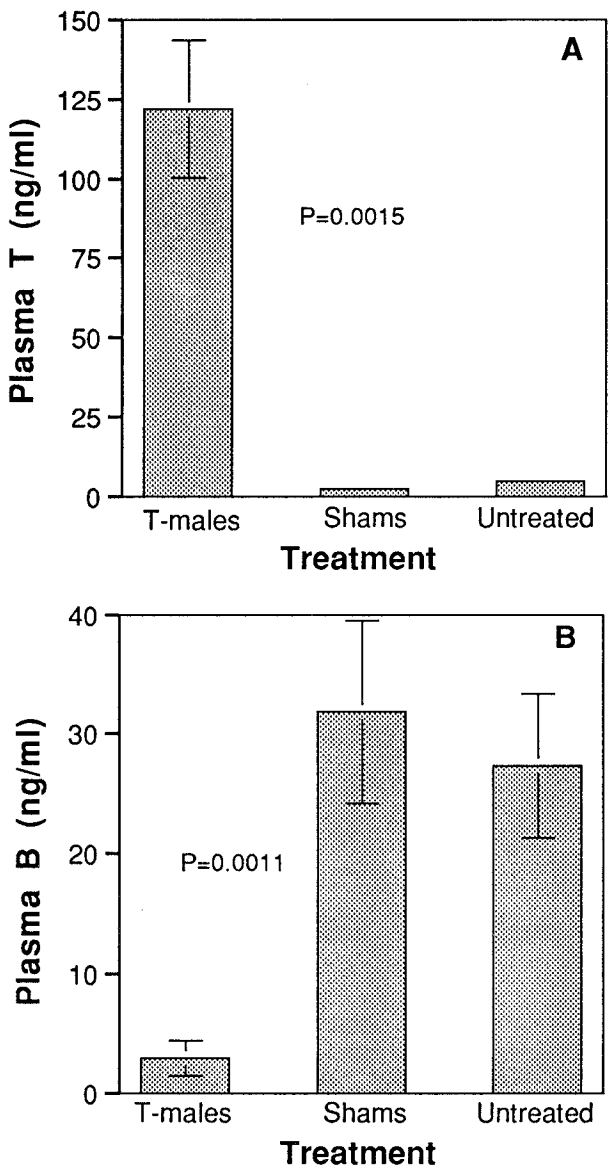

Figure 4. Plasma testosterone (*A*) and corticosterone (*B*) concentrations in testosterone-implanted (T-males), sham-implanted, and untreated male *Sceloporus undulatus*. Testosterone-implanted males ($n = 6$ in *A*; $n = 7$ in *B*) had significantly greater testosterone and lower corticosterone concentrations than either sham-implanted males ($n = 7$) or untreated males ($n = 10$). The standard errors for the testosterone concentrations for sham-implanted and untreated males were 0.32 and 1.41 ng/mL, respectively.

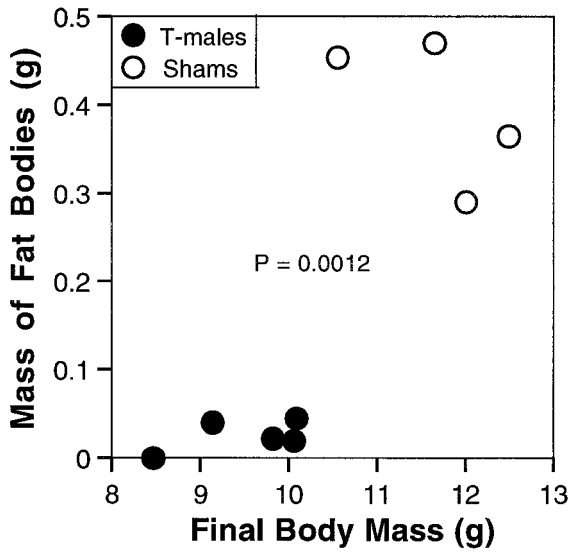

Figure 5. Mass of fat bodies versus final body mass of testosterone-implanted (T-males) and sham-implanted free-living male *Sceloporus undulatus* in the 1996 experiment. Testosterone-implanted males ($n = 5$) had significantly smaller fat bodies than the sham-implanted males ($n = 4$).

= 0.060; mean: 0.00196 ± 0.00047 and 0.00053 ± 0.00045, for the sham-implanted and testosterone-implanted males, respectively).

Similarly, in the 1996 sample, initial body mass did not differ between groups ($P = 1.0$; mean initial body masses, testosterone-implanted = 9.93 ± 0.41 g; sham-implanted = 10.50 ± 1.03). Again, however, testosterone-implanted males lost body mass and sham-implanted males gained body mass. Testosterone-implanted males were significantly lighter than sham-implanted males upon recapture ($P = 0.014$; mean final body masses, testosterone-implanted = 9.52 ± 0.31 g; sham-implanted = 11.68 ± 0.41). Again, this result is not biased by differential tail loss, as no males lost their tails. Neither initial nor final snout-vent lengths differed ($P = 0.71$ and $P = 0.45$ for testosterone-implanted and sham-implanted animals, respectively).

Fat bodies were significantly smaller in testosterone-implanted males than in sham-implanted males ($P = 0.0012$; ANCOVA with final body mass as covariate; means: testosterone-implanted = 0.0252 ± 0.0080 g; sham-implanted = 0.3946 ± 0.0419; Fig. 5).

Discussion

In free-living male northern fence lizards, experimentally elevated testosterone increased both sprint speed and burst stamina relative to sham-implanted lizards. This enhanced performance was associated with significantly reduced growth rates and with reduced fat reserves. Thus, plasma levels of testosterone appear to mediate an important trade-off between locomotor performance and growth. While previous workers have reported effects of testosterone on stamina in lizards, our study is the first to report that both stamina and sprint speed are enhanced in field-active fence lizards and that this enhanced performance has associated energetic costs.

Testosterone-implanted lizards also had greater burst stamina than untreated males, but did not have greater sprint speed. The only apparent explanation for why sham-implanted and untreated lizards differed in sprint speed, given that they were so similar in body size and had such similar plasma steroid concentrations, is that surgery itself caused a decrement in sprint speed. Experimental elevation of plasma testosterone concentrations nevertheless compensated for the effects of the surgery.

In a 1993 field study of this population, maximal plasma testosterone concentrations averaged 84.0 ng/mL, and some males had concentrations exceeding 120 ng/mL (M. Klukowski, unpublished data). Thus, our manipulation raised testosterone concentrations to physiologically relevant levels, albeit at an unusual time.

Testosterone-treated males had lower corticosterone levels than controls and untreated males (Fig. 4*B*). Reciprocal interactions between the gonadal and adrenal axes of lizards have been reported for other lizards (Greenberg et al. 1984; Moore et al. 1991). Marler and Moore (1991) also found that testosterone-treated male *Sceloporus jarrovi* have lower corticosterone levels than sham-implanted males, but only in some years.

The corticosterone levels measured in testosterone-implanted males were very similar to those of free-ranging males at this time of year (M. Klukowski, unpublished data). But corticosterone levels in controls and untreated males were roughly 10-fold greater than those of free-ranging males, thus strongly suggesting that only these two control groups were exhibiting a stress response to testing. The magnitude of the difference is very similar to that reported for acutely stressed *Urosaurus ornatus* (Moore et al. 1991). Chronically elevated testosterone may, therefore, have prevented the stress response in the testosterone-implanted males. Whether males with naturally elevated testosterone also have a less sensitive stress response remains to be tested.

In general, our stamina results on field-active *Sceloporus undulatus* are in agreement with laboratory results for this species (John-Alder et al. 1996). However, our study is different in several important respects. First, the above authors tested directly for an effect of social environment on performance, whereas we tested for a direct effect of elevated plasma testosterone. Second, we observed an effect in a much shorter time period (14–23 d vs. 56 d). Enhanced stamina following experimental elevation of testosterone occurs in just 10–15 d in the field and 12–18 d in the lab in *Uta stansburiana* (B. Sinervo, personal communication). Third, John-Alder et al. (1996) tested stamina on a treadmill operated at 0.5 km/h (13.9 cm/

s), whereas with our testing protocol, lizards began running at workloads over four times as great (approximately 60 cm/s). Fourth, our effect was associated with much higher androgen concentrations than reported by John-Alder et al. (1996). Our testosterone-implanted males and sham-implanted males differed by up to 100 ng/mL, approximately reflecting seasonal differences found in nature, whereas the males in their laboratory treatments differed by about 2 ng/mL. Taken together, these differences suggest that the mechanism of the androgen-dependent enhancement of locomotor performance may be different in the two studies.

The mechanism by which elevated testosterone increases locomotor performance is unknown. The effect could be mediated by effects on circulation. Testosterone-treated *Anolis sagrei* have greater stamina and heart muscle mass than controls; the latter is expected to increase oxygen delivery to muscles (John-Alder 1994). Variation in heart mass also explains a significant proportion of the variation in stamina in *S. undulatus* (John-Alder et al. 1996) and *Ctenosaura similis* (Garland 1984).

Alternatively, the effect of testosterone on locomotor performance might be mediated through changes in the limb musculature itself. While we observed effects on performance in a relatively short time (14–23 d), this is long enough for measurable changes in the musculature (Sassoon et al. 1987; Tingus and Carlsen 1993; Van Breda et al. 1993; Dorloechter et al. 1994). Testosterone has been reported to alter the relative abundance of different fiber types (Kelly et al. 1985; Sassoon et al. 1987), the size and abundance of muscle fibers (Marin et al. 1990), the total mass of muscle (Griggs et al. 1989), and the abundance of key muscle enzymes (Saborido et al. 1991). In the lizard *Dipsosaurus dorsalis,* muscle fiber area is the most important predictor of sprint speed (Gleeson and Harrison 1988). Similarly, the distribution of different fiber types within muscles probably influences lizard locomotor performance. Unfortunately, little is known about the factors that influence fiber type or fiber area in reptiles. Given the abundance of effects of testosterone on muscle physiology in other craniates, it would be reasonable to examine whether testosterone has similar effects on reptilian muscles.

Nonmuscular mechanisms are also possible. Changes in hematocrit or hemoglobin concentration might improve stamina. We did not, however, find a significant effect of elevated testosterone on hematocrit.

We cannot exclude motivational explanations for our findings. The testosterone-implanted males may have been more motivated to run away from the aversive stimuli than the controls, although the biological relevance of such an effect is unclear.

An important question is whether elevated testosterone enhances locomotor performance directly through changes in muscle physiology or heart efficiency, or indirectly through changes in body size. For several reasons, our sprint speed results are unlikely to be simply a result of the testosterone-treated lizards being smaller. First, sprint speed was not significantly correlated with either body mass or snout-vent length in the 15 untreated males (nor in the other two groups). Second, theoretical arguments suggest sprint speed should scale positively with mass or be independent (see references in Dohm et al. [1996]). Third, many empirical studies of reptiles report either a positive relationship between sprint speed and body size or no relationship (Huey and Hertz 1982, 1984; Hertz et al. 1983; Garland 1984, 1985, 1988; Garland and Else 1987; Sinervo and Adolph 1989; Garland et al. 1990). This suggests that the differences we observed between testosterone-implanted and sham-implanted males underestimated the true effect of elevated testosterone on sprint speed.

Similarly, our effects on burst stamina are unlikely to result simply from smaller lizards having inherently greater stamina. First, we corrected burst stamina for body mass differences (see Material and Methods). Second, several empirical studies report either a positive relationship or no relationship between stamina and body size (Garland 1984; Garland and Else 1987; Gerwien and John-Alder 1992; John-Alder 1994). In particular, John-Alder et al. (1996) report that endurance does not vary significantly with body mass in *S. undulatus,* and the largest morph in a polymorphic population of *U. stansburiana* also has the greatest stamina (B. Sinervo, personal communication).

Lizards with experimentally elevated testosterone lost body mass during a 14–23-d period in the field, during which sham-implanted lizards gained body mass. This finding agrees with that reported for testosterone-implanted *S. jarrovi* (Marler and Moore 1988, 1989, 1991), although males in those studies were in the field for longer periods (52–69 d). Testosterone-implanted male *S. jarrovi* also suffer reduced survivorship relative to controls, and this survivorship is linked to decreased energy intake and increased energy expenditure (Marler and Moore 1988, 1991; Marler et al. 1995). It is possible that a similar phenomenon is occurring in the fence lizard.

Besides a significant decline in growth rates, we also found in the 1996 companion experiment that testosterone-implanted males had significantly reduced fat-body masses relative to sham-implanted males. This was not unexpected, as earlier workers have reported a negative correlation between seasonal fat-body mass and plasma androgen levels in *S. undulatus* (McKinney and Marion 1985). Similarly, Marler and Moore (1991) report that male *S. jarrovi* with experimentally elevated testosterone have significantly lighter fat bodies than controls. Decreased fat-body masses might significantly increase mortality over the winter or decrease reproductive success in the spring, because males with lower plasma concentrations of testosterone would presumably emerge with larger fat bodies and, thus, have an initial energetic advantage.

Thus, substantial costs appear to be associated with elevated testosterone during the nonbreeding season. Whether these costs are greater than any potential benefits, such as increased chances of escape from predators, enhanced prey-capturing

success, or social dominance (Garland et al. 1990), is unknown. However, one might expect that testosterone-treated males are actually more susceptible to predation given their increased activity (Marler and Moore 1988), and enhanced prey-capture success seems unlikely given Marler and Moore's (1989) findings.

Our results suggest that locomotor performance may vary seasonally with plasma testosterone concentrations, such that maximum performance occurs during the breeding season. Males from this population are at least three times more active during the breeding than the nonbreeding season, as measured by movement and push-up display frequencies of focal individuals (M. Klukowski, unpublished observations). Territorial interactions in this population are intense, with durations of up to 15 min (M. Klukowski, unpublished observations). We suggest that the cost-to-benefit ratio of maintaining heightened locomotor performance may only be favorable during the breeding season. Seasonal (and socially mediated) changes in testosterone may play an important role in the phenotypic plasticity of locomotor performance.

Acknowledgments

We wish to thank the National Science Foundation for sponsoring the Exploration of Careers in Science Program, which enabled N.M.J. to participate in the study; Denise Briner, Lori Christensen, H. B. John-Alder, Dale Sengelaub, John Phillips, and two anonymous reviewers for their comments on earlier versions of this article; and Janette Fischer and John Phillips for the use of equipment. Ellen Ketterson and Michael Moore provided invaluable guidance in collecting blood samples and performing the radioimmunoassay. This work was partially supported by an Indiana Academy of Sciences Grant to M.K.

Literature Cited

Andrews R.M. 1982. Patterns of growth in reptiles. Pp. 273–320 in C. Gans and F.H. Pough, eds. Biology of the Reptilia. Vol. 13. Academic Press, New York.

Avery R.A., J.D. Bedford, and C.P. Newcombe. 1982. The role of thermoregulation in lizard biology: predatory efficiency in a temperate diurnal basker. Behav. Ecol. Sociobiol. 11:261–267.

Bennett A.F. 1980. The thermal dependence of lizard behaviour. Anim. Behav. 28:752–762.

Christian K.A. and C.R. Tracy. 1981. The effect of the thermal environment on the ability of hatchling Galapagos land iguanas to avoid predation during dispersal. Oecologia 49:218–223.

Conant R. and J.T. Collins. 1991. A Field Guide to Reptiles and Amphibians. Houghton Mifflin, Boston.

Crowley S.R. 1985. Thermal sensitivity of sprint-running in the lizard *Sceloporus undulatus:* support for a conservative view of thermal physiology. Oecologia 66:219–225.

Dohm M.R., J.P. Hayes, and T. Garland, Jr. 1996. Quantitative genetics of sprint running speed and swimming endurance in laboratory house mice (*Mus domesticus*). Evolution 50:1688–1701.

Dorloechter M., S.H. Astrow, and A.A. Herrera. 1994. Effects of testosterone on a sexually dimorphic frog muscle: repeated in vivo observations and androgen receptor distribution. J. Neurobiol. 25:897–916.

Garland T., Jr. 1984. Physiological correlates of locomotory performance in a lizard: an allometric approach. Am. J. Physiol. 247:R806–R815.

———. 1985. Ontogenetic and individual variation in size, shape and speed in the Australian agamid lizard *Amphibolurus nuchalis.* J. Zool. (Lond.) Ser. A 207:425–439.

———. 1988. Genetic basis of activity metabolism. I. Inheritance of speed, stamina, and antipredator displays in the garter snake *Thamnophis sirtalis.* Evolution 42:335–350.

Garland T., Jr. and P.L. Else. 1987. Seasonal, sexual, and individual variation in endurance and activity metabolism in lizards. Am. J. Physiol. 252:R439–R449.

Garland T., Jr., P.L. Else, A.J. Hulbert, and P. Tap. 1987. Effects of endurance training and captivity on activity metabolism of lizards. Am. J. Physiol. 252:R450–R456.

Garland T., Jr., E. Hankins, and R.B. Huey. 1990. Locomotor capacity and social dominance in male lizards. Funct. Ecol. 4:243–250.

Gerwien R.W. and H.B. John-Alder. 1992. Growth and behavior of thyroid-deficient lizards (*Sceloporus undulatus*). Gen. Comp. Endocrinol. 87:312–324.

Gleeson T.T., P.M. Dalessio, J.A. Carr, S.J. Wickler, and R.S. Mazzeo. 1993. Plasma catecholamine and corticosterone and their in vitro effects on lizard skeletal muscle lactate metabolism. Am. J. Physiol. 265:R632–R639.

Gleeson T.T. and J.M. Harrison. 1988. Muscle composition and its relation to sprint running in the lizard *Dipsosaurus dorsalis.* Am. J. Physiol. 255:R470–R477.

Goldberg S.R. 1974. Reproduction in mountain and lowland populations of the lizard *Sceloporus occidentalis.* Copeia 1974:176–182.

Greenberg N., T. Chen, and D. Crews. 1984. Social status, gonadal state, and the adrenal stress response in the lizard, *Anolis carolinensis.* Horm. Behav. 18:1–11.

Griggs R.C., W. Kingston, R.F. Jozefowicz, B.E. Herr, G. Forbes, and D. Halliday. 1989. Effect of testosterone on muscle mass and muscle protein synthesis. J. Applied Physiol. 66:498–503.

Hays W.L. 1991. Statistics. Harcourt Brace, New York.

Hertz P.E., R.B. Huey, and E. Nevo. 1983. Homage to Santa Anita: thermal sensitivity of sprint speed in agamid lizards. Evolution 37:1075–1084.

Huey R.B. and A.E. Dunham. 1987. Repeatability of locomotor

performance in natural populations of the lizard *Sceloporus merriami*. Evolution 41:1116–1120.

Huey R.B. and P.E. Hertz. 1982. Effects of body size and slope on sprint speed of a lizard (*Stellio* (*Agama*) *stellio*). J. Exp. Biol. 97:401–409.

———. 1984. Effects of body size and slope on acceleration of a lizard (*Stellio stellio*). J. Exp. Biol. 110:113–123.

John-Alder H.B. 1984. Seasonal variations in activity, aerobic energetic capacities, and plasma thyroid hormones (T3 and T4) in an iguanid lizard. J. Comp. Physiol. B 154:409–419.

———. 1990. Effects of thyroxine on standard metabolic rate and selected intermediary metabolic enzymes in field-active lizards *Sceloporus undulatus*. Physiol. Zool. 63:600–614.

———. 1994. Testosterone improves exercise endurance in a lizard (*Anolis sagrei*). Am. Zool. 34:120A. (Abstr.)

John-Alder H.B., S. McMann, L.S. Katz, A. Gross, and D.S. Barton. 1996. Social modulation of exercise endurance in a lizard (*Sceloporus undulatus*). Physiol. Zool. 69:547–567.

Kelly A., G. Lyons, B. Gambki, and N. Rubinstein. 1985. Influences of testosterone on contractile proteins of the guinea pig temporalis muscle. Adv. Exp. Med. Biol. 182:155–168.

Ketterson E.D., V. Nolan, Jr., L. Wolf, C. Ziegenfus, A.M. Dufty, Jr., G.F. Ball, and T.S. Johnsen. 1991. Testosterone and avian life histories: the effect of experimentally elevated testosterone on corticosterone and body mass in dark-eyed juncos. Horm. Behav. 25:489–503.

Marin M.L., M.L. Tobias, and D.B. Kelley. 1990. Hormone-sensitive stages in the sexual differentiation of laryngeal muscle fiber number in *Xenopus laevis*. Development 110:703–712.

Marler C.A. and M.C. Moore. 1988. Evolutionary costs of aggression revealed by testosterone manipulations in free-living male lizards. Behav. Ecol. Sociobiol. 23:21–26.

———. 1989. Time and energy costs of aggression in testosterone-implanted free-living male mountain spiny lizards (*Sceloporus jarrovi*). Physiol. Zool. 62:1334–1350.

———. 1991. Supplementary feeding compensates for testosterone-induced costs of aggression in male mountain spiny lizards, *Sceloporus jarrovi*. Anim. Behav. 42:209–219.

Marler C.A., G. Walsberg, M.L. White, and M. Moore. 1995. Increased energy expenditure due to increased territorial defense in male lizards after phenotypic manipulation. Behav. Ecol. Sociobiol. 37:225–231.

McKinney R.B. and K.R. Marion. 1985. Plasma androgens and their association with the reproductive cycle of the male fence lizard, *Sceloporus undulatus*. Comp. Biochem. Physiol. 82A:515–519.

Moore M.C., C.W. Thompson, and C.A. Marler. 1991. Reciprocal changes in corticosterone and testosterone levels following acute and chronic handling stress in the tree lizard, *Urosaurus ornatus*. Gen. Comp. Endocrinol. 81:217–226.

Saborido A., J. Vila, F. Molano, and A. Megias. 1991. Effect of anabolic steroids on mitochondria and sarcotubular system skeletal muscle. J. Appl. Physiol. 70:1038–1043.

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

Sassoon D.A., G.E. Gray, and D.B. Kelley. 1987. Androgen regulation of muscle fiber type in the sexually dimorphic larynx of *Xenopus laevis*. J. Neurosci. 7:3198–3206.

Schall J.J., A.F. Bennett, and R.W. Putnam. 1982. Lizards infected with malaria: physiological and behavioral consequences. Science 217:1057–1059.

Shine R. 1980. "Costs" of reproduction in reptiles. Oecologia 46:92–100.

Sinervo B. and S.C. Adolph. 1989. Thermal sensitivity of growth rate in hatchling *Sceloporus* lizards: environmental, behavioral and genetic aspects. Oecologia 78:411–419.

Sorci G., J.G. Swallow, T. Garland, Jr., and J. Clobert. 1995. Quantitative genetics of locomotor speed and endurance in the lizard *Lacerta vivipara*. Physiol. Zool. 68:698–720.

Tingus S.J. and R.C. Carlsen. 1993. Effect of continuous infusion of an anabolic steroid on murine skeletal muscle. Med. Sci. Sports Exercise 25:485–494.

Van Breda E., H.A. Keizer, P. Geurten, G. Van Kranenburg, P.P.C.A. Menheere, H. Kuipers, and J.F.C. Glatz. 1993. Modulation of glycogen metabolism of rat skeletal muscle by endurance training and testosterone treatment. Pflueg. Arch. Eur. J. Physiol. 424:294–300.

Webb P.W. 1986. Effect of body form and resonance threshold on the vulnerability of four species of teleost prey attacked by largemouth bass (*Micropterus salmoides*). Can. J. Fish. Aquat. Sci. 43:763–771.