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Testosterone has opposite effects on male growth in lizards (*Sceloporus* spp.) with opposite patterns of sexual size dimorphism

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Summary

Sexual size dimorphism (SSD) has received considerable attention from evolutionary biologists, but relatively little is known about the physiological mechanisms underlying sex differences in growth that lead to SSD. Testosterone (T) stimulates growth in many male-larger vertebrates, but inhibits growth in the female-larger lizard *Sceloporus undulatus*. Thus, opposite patterns of SSD may develop in part because of underlying differences in the hormonal regulation of male growth. In the present study, we examined the effects of T on male growth in two sympatric congeners with opposite patterns of SSD (*S. virgatus*: female-larger; *S. jarrovii*: male-larger). During the mating season, yearling males of both species have higher plasma T levels than females, but whereas yearling males of *S. virgatus* grow only half as fast as females, yearling males of *S. jarrovii* grow more quickly than females. Thus, we hypothesized that T inhibits growth in yearling *S. virgatus* males, but promotes growth in yearling *S. jarrovii* males. In support of this hypothesis, we found that castrated (CAST) males of *S. virgatus* grew faster than castrated males given T implants (TEST). In contrast, TEST males of *S. jarrovii* grew faster than CAST males. Our results provide the first direct evidence for opposite effects of T on male growth in closely related species with opposite patterns of SSD. We speculate that growth inhibition by T reflects an energetic trade-off between growth and reproductive investment, and propose that such 'costs' of male reproduction may help explain the evolution of female-larger SSD in *Sceloporus*.

Key words: body size, growth, hormone manipulation, proximate mechanism, reproductive investment, sexual size dimorphism, *Sceloporus jarrovii*, *Sceloporus virgatus*, testosterone.

Introduction

Sexual size dimorphism (SSD) is a widespread biological phenomenon in which members of one sex are characteristically larger than those of the opposite sex for a given population or species. Studies of the selective forces shaping SSD have figured prominently in the evolutionary literature since Darwin (1871). However, relatively little is known about the proximate physiological mechanisms underlying sex differences in growth (Badyaev, 2002; Cox et al., 2005; Duvall and Beaupre, 1998). Testosterone (T) is commonly regarded as an anabolic steroid that promotes skeletal and muscular growth, but most of the evidence supporting this generalization comes from studies of mammals, fishes and birds with male-larger SSD (Borski et al., 1996; Fennel and Scanes, 1992a; Ford and Klindt, 1989; Gatford et al., 1998; Holloway and Leatherland, 1998; Huggard et al., 1996; Kuwaye et al., 1993; Wehrenberg and Giustina, 1992). Interestingly, T inhibits growth in some reptiles with female-larger SSD (Abell, 1998a; Cox et al., 2005; Crews et al., 1985; Lerner and Mason, 2001). Collectively, these studies suggest that opposite patterns of SSD may develop in part because of underlying differences in the hormonal regulation of male growth. However, comparisons among these studies are complicated by the taxonomic and biological disparity of the study organisms and by significant differences in methodology. In the present study, we provide the first direct comparison of the effects of T on male growth in two closely related species with opposite patterns of SSD.

Life history ecologists have long recognized that growth may be constrained by the preferential allocation of available energy to reproduction (Fisher, 1930; Reznick, 1985; Williams, 1966). More recently, behavioral endocrinologists have begun to explore the role of hormones as proximate mediators of such trade-offs (Ketterson et al., 1992; Ricklefs and Wikelski, 2002). For example, in male lizards, T increases activity, endurance, locomotor performance, territorial aggression and home range size (Cox et al., 2005; DeNardo and Sinervo, 1994; John-Alder et al., 1996; Klukowski et al., 1998, 2004; Marler and Moore, 1988; Moore, 1988; Moore and Marler, 1987). These behavioral and physiological effects of T presumably enhance male reproductive success, but they also

incur costs in the form of decreased energy acquisition, increased energy expenditure and increased parasitism (Cox et al., 2005; Klukowski et al., 2001; Marler and Moore, 1989, 1991; Marler et al., 1995; Olsson et al., 2000; Salvador et al., 1996; Uller and Olsson, 2003). These associated costs may explain why T often inhibits growth in lizards (Abell, 1998a; Cox et al., 2005; Hews et al., 1994; Hews and Moore, 1995; Salvador and Veiga, 2000). However, the implications of such trade-offs with regard to SSD remain largely unexplored.

In a previous study (Cox et al., 2005), we showed that T inhibits growth while increasing daily activity, movement and home-range size in males of *Sceloporus undulatus*, a lizard with female-larger SSD. We interpreted these results as evidence for a T-mediated energy allocation trade-off between male growth and reproductive investment, and proposed this energetic growth constraint as an explanation for SSD in this species. In the present study, we show that T also inhibits male growth in *S. virgatus*, a closely related species in which female-larger SSD develops because yearling females grow more quickly than males during the mating season. In contrast, we show that T promotes male growth in *S. jarrovii*, a sympatric congener in which male-larger SSD develops because yearling males grow more quickly than females. Our results provide the first direct evidence for opposite effects of T on male growth in closely related species with opposite patterns of SSD. Further, our study is the first to demonstrate a stimulatory effect of T on skeletal growth in any squamate reptile. Despite this potential for growth promotion by T in *Sceloporus*, we speculate that energetic costs of elevated T may indirectly constrain male growth in *S. virgatus*. These T-mediated costs of male reproductive investment may help explain the evolution of female-larger SSD in *Sceloporus*.

Materials and methods

Study species

Sceloporus virgatus Smith (striped plateau lizard) and *S. jarrovii* Cope (Yarrow's spiny lizard) are sympatric in the Chiricahua Mountains of southeastern Arizona and share many pertinent ecological attributes. Both species are primarily saxicolous, employ sit-and-wait foraging tactics, share common prey and predators, attain sexual maturity as yearlings (i.e. within 12 months of birth), reproduce only once annually, and exhibit polygynous mating systems characterized by aggressive intrasexual male competition for breeding females (Ballinger, 1973, 1979; Ballinger and Ketels, 1983; Rose, 1981; Ruby, 1978; Ruby and Dunham, 1984; Smith, 1985; Smith et al., 1995; Vinegar, 1975b). However, while adult females of *S. virgatus* average 10% larger than adult males (longer in snout–vent length, *SVL*), adult males of *S. jarrovii* average 10% larger than adult females (Cox et al., 2003; Cox, 2005; R. M. Cox and H. B. John-Alder, manuscript submitted). Although these species are congeners, they belong to separate clades within *Sceloporus* (see Wiens and Reeder, 1997), and also differ in several other important regards. *Sceloporus virgatus* is small (maximum female size: 73 mm *SVL*, 12 g body mass when gravid), mates in the spring, and lays a single clutch of eggs (oviparity) in the summer. In contrast, *S. jarrovii* is relatively large (maximum male size: 103 mm, 30 g), mates in the fall, and gives birth to live young (viviparity) in late spring. Despite these differences, *S. virgatus* and *S. jarrovii* offer a unique opportunity to investigate sex-specific growth regulation in two phylogenetically and ecologically similar species with opposite patterns of SSD.

We studied both *S. virgatus* and *S. jarrovii* along a single 2 km section of streambed in Cave Creek Canyon (North Fork), located 1–3 km northwest of the American Museum of Natural History's Southwestern Research Station in the Coronado National Forest, Cochise Co., Arizona, USA (31°53–54′N, 109°13′W, elevation 1660–1760 m). We obtained collecting permits from the Arizona Game and Fish Department (SP 696192, 751920 and 553889) and land use permits from the United States Forest Service. The Rutgers University Animal Care and Facilities Committee approved our procedures (protocol 01-019). Over three consecutive years (2002–2004), we used standard mark-recapture techniques to describe the ontogeny of sex differences in growth rate and body size. These data, which are reported elsewhere (Cox, 2005; R. M. Cox and H. B. John-Alder, manuscript submitted), are summarized here because they provide a detailed natural history framework for the design and interpretation of this study.

In both *S. virgatus* and *S. jarrovii*, SSD is slight over the first few months of life, but develops rapidly thereafter, reaching a magnitude of about 10% within a year of birth. In both species, this rapid development of SSD begins after only 2–3 months of postnatal activity and growth (excluding ca. 4 months winter dormancy in *S. virgatus*), roughly coincident with the onset of their respective mating seasons. On our study plot, most males and some females of each species attain sexual maturity as yearlings (Ballinger, 1973, 1979; Ballinger and Ketels, 1983; Smith et al., 1995). However, while yearling males of *S. virgatus* grow only half as fast as females during their spring mating season, yearling males of *S. jarrovii* grow more quickly than females during their fall mating season (Cox, 2005; R. M. Cox and H. B. John-Alder, manuscript submitted). These observations raise a question of central importance with regard to the proximate causation of SSD: during their respective mating seasons, why do yearling males of *S. virgatus* grow more slowly than females, yet yearling males of *S. jarrovii* grow more quickly than females? We hypothesized that elevated T inhibits growth in yearling males of *S. virgatus*, but promotes growth in yearling males of *S. jarrovii*. Thus, we predicted that castration would promote and exogenous T would inhibit male growth in *S. virgatus*, while predicting opposite growth effects of castration and T replacement in *S. jarrovii*.

Experimental design

We collected yearling males by hand-held noose in late April (*S. virgatus*, N=71) and late August (*S. jarrovii*, N=67) of 2004, near the onset of the mating season for each species.

We measured *SVL* to the nearest 1 mm with a ruler, body mass to the nearest 0.1 g with a Pesola® spring scale (Pesola AG, Baar, Switzerland), and gave each animal a unique toe clip for permanent identification. We then assigned males to one of three size-matched treatment groups: castrated males receiving a placebo implant (CAST), castrated males receiving a T implant (TEST), and intact control males receiving a placebo implant (CON). Following surgical treatments (see below), we released animals at their location of capture and left them undisturbed until recapture in June (*S. virgatus*) or October (*S. jarrovii*). Upon recapture, we recorded *SVL* and mass for each experimental animal and calculated individual growth rates (mm day^{-1}) by assuming linear growth and dividing change in *SVL* by elapsed time.

Testosterone implants

We constructed tonic-release T implants from 5 mm lengths of Silastic® tubing (Dow Corning, Midland, MI, USA; 0.058″i.d., 0.077″o.d.). After sealing one end of each tubule with silicone adhesive gel (Dow Corning), we used a Hamilton® syringe to inject 3 μl of a solution of T (T-1500, Sigma-Aldrich Inc., St Louis, MO, USA) dissolved in dimethyl sulfoxide (DMSO; 100 μg T μl^{-1} DMSO) into the open end of each implant. We then sealed each tubule with silicone adhesive and waited several days for the DMSO to evaporate and diffuse through the tubing, leaving 300 μg of crystalline T within the lumen (ca. 1.5 mm length) of each implant. We constructed placebo implants in identical fashion, but injected them with pure DMSO, which left an empty tubule after evaporation and diffusion.

Surgical treatments

We anaesthetized animals with an intramuscular injection of ketamine (Vetus Animal Health, MFA Inc., Columbia, MO, USA; 130 mg kg^{-1} body mass). We then exposed the testes with a single ventral incision and bilaterally castrated (orchiectomized) CAST and TEST males by ligating each spermatic cord with surgical silk, ablating each testis, and cauterizing each ligated spermatic cord after removal of the testes. For CON males, we performed 'sham' surgeries in which we made identical incisions to expose and manipulate the testes while leaving them intact. We then inserted either a T implant (TEST) or a placebo implant (CAST and CON) into the coelomic cavity and closed the incision with Nexaband® surgical glue (Veterinary Products Laboratories, Phoenix, AZ, USA). We performed surgeries within 2 days of capture, and released animals at their site of capture within 3 days of surgery. All animals appeared healthy and vigorous upon release, and survival from surgery to release was high for both *Sceloporus virgatus* (68 of 71, 96% survival) and *S. jarrovii* (65 of 67, 97%).

Plasma testosterone levels

To document natural seasonal and sexual variation in circulating T levels, we periodically collected blood samples from unmanipulated yearling males and females captured on or adjacent to our experimental plot. For each species, we sampled 10–15 individuals of each sex at approximately monthly intervals spanning the entire first year of life (excluding winter). Animals were permanently marked so that no individual lizard was bled more than once. To validate the efficacy of our T manipulations, we obtained blood samples from experimental males upon recapture. Hereafter, we distinguish between 'natural' *vs* 'experimental' animals when discussing plasma T levels.

We collected blood samples from the postorbital sinus within 2 min of capture using heparinized microhematocrit capillary tubes (Fisher Scientific, Pittsburgh, PA, USA). We held samples on ice until they could be centrifuged (within 6 h of collection), and stored the separated plasma at –20°C until subsequent assays. We performed radioimmunoassays (RIAs) for plasma T concentration following methods reported elsewhere (Cox et al., 2005; Smith and John-Alder, 1999). Samples were extracted twice in diethyl ether (mean 81% extraction efficiency), dried under a stream of ultra-filtered air, and reconstituted in phosphate buffered saline with gelatin (PBSG). Reconstituted samples were assayed with ^{3}H-T as a radiolabel (PerkinElmer Life Sciences Inc., Boston, MA, USA) and T antiserum (1:18000 initial dilution) developed in rabbits by A. L. Johnson (The University of Notre Dame, IN, USA). We did not separate T from other androgens prior to RIA, so our 'plasma testosterone' values should be interpreted with the caveat that they reflect any additional binding of the T antibody to 5α-dihydrotestosterone (DHT, 50% cross-reactivity). However, plasma DHT levels are typically only 2–4% of plasma T levels in these species (Abell, 1998c; Woodley and Moore, 1999), so our values primarily reflect plasma T. Within each species, we randomized samples across assays to avoid confounding sex or sampling date with any inherent interassay variation (typically 6%; Smith and John-Alder, 1999). Limits of detection were 1–5 pg T per assay tube.

Statistical analyses

We compared seasonal and sex differences in natural plasma T using analysis of variance (ANOVA) with sex and sampling month as class effects with interaction. Within each sex, we tested for seasonal patterns in natural plasma T levels using ANOVA with sampling month as the main effect, and determined *post hoc* differences among months using the Ryan–Einot–Gabriel–Welsch test (REGWQ; SAS Institute 1989). Within males sampled during the breeding season, we assessed the allometry of plasma T levels using analysis of covariance (ANCOVA) with sampling month as a class effect and *SVL* as a covariate (i.e. based on covariance with *SVL* after accounting for variance due to sampling month). We used $\log_{10}$-transformed plasma T levels and *SVL* values for these analyses and verified homogeneity of allometric slopes among months before employing ANCOVA.

For experimental males, we compared plasma T levels using ANOVA with treatment as the main effect and determined *post hoc* separation among treatments using the REGWQ test.

Growth typically decreases with size, so we compared growth rate among treatment groups using ANCOVA with treatment as the main effect and initial *SVL* as a covariate. We tested for homogeneity of slopes among treatment groups with a treatment-by-*SVL* interaction term, which we retained in our final model when significant (separate slopes model, *S. virgatus*), and omitted when non-significant (ANCOVA, *S. jarrovii*). We determined *post hoc* separation by comparing least-square mean growth rate based on these models. To facilitate direct inferences regarding the effect of exogenous T on growth in each species, we performed additional ANCOVA analyses excluding CON males. This allowed us to directly compare castrated males that differed in discrete fashion with regard to the presence (TEST) or virtual absence (CAST) of circulating T. All statistical analyses were conducted using SAS (version 8.2, SAS Institute, Inc.).

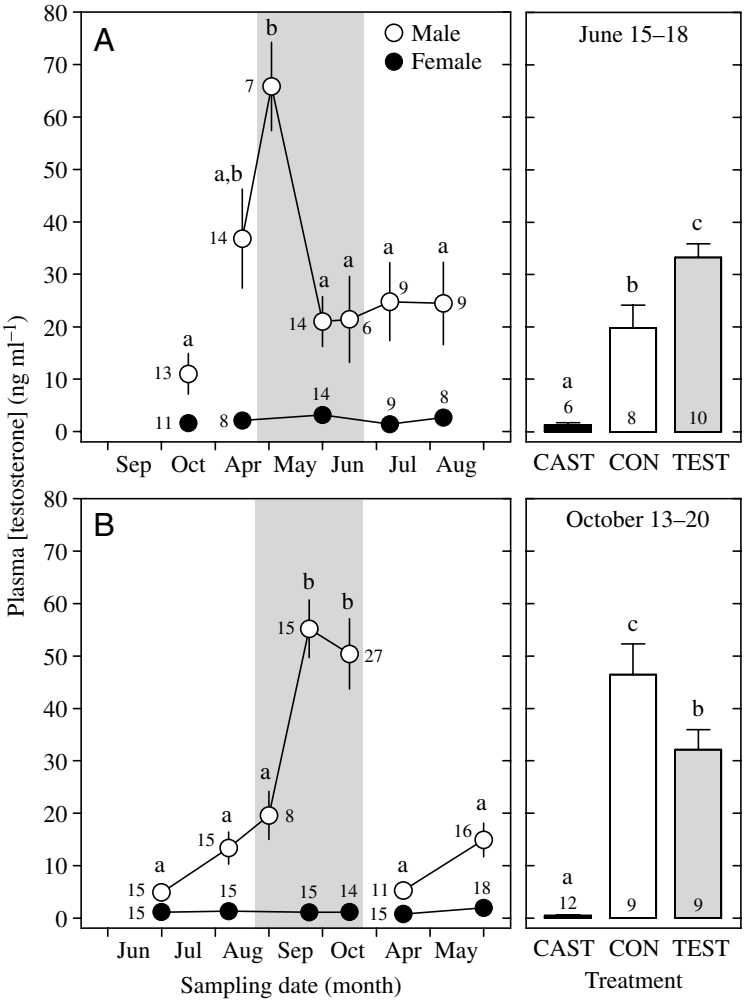

Fig. 1. (A,B, left) Plasma testosterone (T) concentration *vs* sampling date for natural yearling males and females of (A) *Sceloporus virgatus* and (B) *S. jarrovii*. Lowercase letters denote monthly differences within males (ANOVA with REGWQ *post hoc* test). Shaded areas indicate the approximate duration of T manipulation experiments. (A,B, right) Plasma T concentration for treatment groups at the conclusion of our experiments. Lowercase letters denote differences among treatments (ANOVA with REGWQ *post hoc* test). Values are means ± 1 S.E.M. (*N* values are given beside symbols and in bars). See text for statistical analyses.

Results

Recapture success

For *Sceloporus virgatus*, we recaptured 9 of 21 CAST (43%), 16 of 22 CON (73%) and 15 of 20 TEST (75%) in July (mean 42 days post-treatment). Recaptured males of *S. virgatus* were smaller in initial *SVL* (45.11±1.46 mm, mean ± 1 S.E.M.) than males that were never recaptured (46.95±1.46 mm; $F_{5,56}$=5.33; P=0.025). This size-dependence of recapture success did not differ significantly among treatment groups ($F_{5,56}$=1.67; P=0.198), but was particularly pronounced within CON males (recaptured=44.06±0.98 mm; not recaptured=49.60±1.03 mm). For *S. jarrovii*, we recaptured 12 of 20 CAST (60%), 10 of 20 CON (50%) and 13 of 21 TEST (62%) in October (mean 51 days post-treatment). Recapture success was independent of initial size in *S. jarrovii* ($F_{5,56}$=0.01; P=0.960).

Plasma testosterone levels

Across all sampling points, mean plasma T concentration was higher in natural yearling males than in females for both *S. virgatus* ($F_{12,141}$=32.91; P<0.001; Fig. 1A) and *S. jarrovii* ($F_{12,198}$=100.25; P<0.001; Fig. 1B). Female plasma T concentration was uniformly low across sampling points for both *S. virgatus* ($F_{5,60}$=1,56; P=0.186; Fig. 1A) and *S. jarrovii* ($F_{5,91}$=1.24; P=0.297; Fig. 1B). However, we observed striking seasonal differences in mean plasma T concentration within yearling males of *S. virgatus* ($F_{5,74}$=2.67; P=0.028) and *S. jarrovii* ($F_{6,100}$=17.08; P<0.001), with peak levels attained during the mating season in both species (Fig. 1). After controlling for variance attributable to sampling month within the mating season, we observed a positive relationship between plasma T concentration and *SVL* (both variables $\log_{10}$-transformed) in yearling males of both species (Fig. 2). While this allometry is robust in *S. virgatus* ($F_{1,48}$=35.87; P<0.001), it is weak and driven primarily by several small males in *S. jarrovii* ($F_{1,40}$=6.71; P=0.012). Thus, even during the mating season, many small males of *S. virgatus* exhibited low plasma T levels similar to yearling females (Fig. 2A). In contrast, only a few of the smallest males of *S. jarrovii* exhibited such low plasma T levels during the mating season (Fig. 2B).

As expected, we observed a dramatic difference in plasma T concentration among experimental male treatment groups in both *S. virgatus* ($F_{2,23}$=23.74; P<0.001; mean 48 days post-treatment) and *S. jarrovii* ($F_{2,29}$=44.37; P<0.001; mean 51 days post-treatment). In both species, castration reduced plasma T to levels typical of natural yearling females, while T implants restored plasma T of castrated males to levels typical

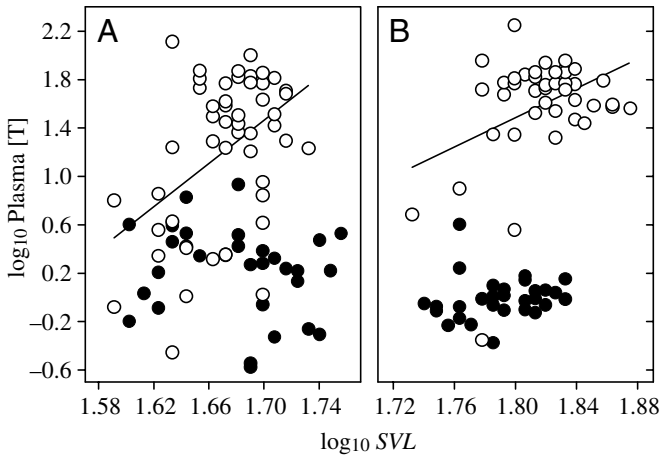

Fig. 2. Log_{10}-transformed plasma testosterone (T) concentration *vs* snout–vent length (*SVL*) for natural yearling males (open symbols) and females (filled symbols) of (A) *Sceloporus virgatus* and (B) *S. jarrovii*, during the mating season. Least-squares regression lines are shown for males of each species: (A) y=8.92x–13.69; (B) y=6.07x–9.44. See text for statistical analyses.

of natural yearling males (Fig. 1). Three TEST of *S. jarrovii* had low plasma T concentrations similar to CAST, presumably indicating that their implants had exhausted. Excluding these individuals, our implants produced remarkably similar mean plasma T levels of 32 and 33 ng ml^{-1} in TEST of *S. virgatus* and *S. jarrovii*, respectively (Fig. 1). These values are nearly identical to mean plasma T levels previously induced in *S. undulatus* (34 ng ml^{-1}; Cox et al., 2005). In both species, we observed a close agreement in mean plasma T between intact CON males and natural yearling males sampled at the end of the experimental period (Fig. 1). However, CON of *S. virgatus* had lower plasma T levels than TEST (Fig. 1A), while CON of *S. jarrovii* had higher plasma T levels than TEST (Fig. 1B). These discrepancies reflect two things. First, experimental *S. virgatus* males were sampled after plasma T had declined from peak mating season values, while experimental *S. jarrovii* males were sampled when plasma T was still at peak levels (Fig. 1). Second, as in natural yearling males, small CON of *S. virgatus* had relatively low plasma T concentrations, such that we observed a positive correlation between plasma T concentration and *SVL* in this group (r^2=0.630; P=0.019). In contrast, plasma T concentration was uniformly high and unrelated to body size in CON of *S. jarrovii* (r^2=0.001; P=0.925). We found no relationship between plasma T concentration and initial *SVL* within the CAST and TEST groups of either species.

Growth rate

Growth rate (mm day^{-1}) decreased with body size across experimental *S. virgatus* treatment groups, but the slope of this relationship was particularly steep for CON (Fig. 3A), yielding a significant *SVL*-by-treatment interaction ($F_{2,39}$=8.72; P<0.001). Our separate slopes regression model retaining this interaction term revealed significant effects of both treatment

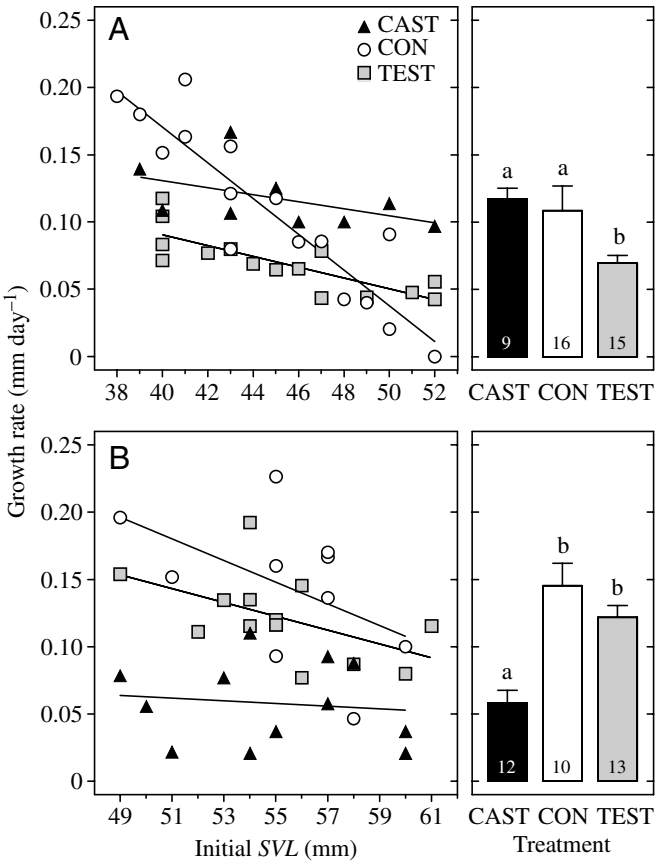

Fig. 3. (A,B, left) Growth rate *vs* initial snout–vent length (*SVL*) for individual males of (A) *Sceloporus virgatus* and (B) *S. jarrovii,* by treatment group. (A,B, right) Growth rate by treatment group, from data in left panels. Lowercase letters denote differences among treatments based on *post hoc* comparison of growth rates from (A) separate slopes or (B) ANCOVA models with initial *SVL* as the covariate. Values are means ± 1 S.E.M. (*N* values are given in bars). Regression equations: (A) CAST, y=–0.002x+0.235; CON, y=–0.013x+0.702; TEST, y=–0.004x+0.251; (B) CAST, y=–0.001x+0.113; CON, y=–0.008x+0.591; TEST, y=–0.005x+0.406. See text for statistical analyses.

($F_{2,39}$=9.36; P<0.002) and initial *SVL* ($F_{1,39=}$30.59; P<0.001) on growth rate. Overall, least-square mean growth rate was significantly lower in TEST than in CAST and CON. However, the heterogeneity of slopes among treatment groups renders this comparison strongly size-dependent. Small yearling CON tended to grow at high rates comparable to CAST, while large yearling CON tended to grow at low rates characteristic of TEST (Fig. 3A). These growth patterns are intriguing in light of our plasma T data: CAST and small CON had low plasma T concentrations and high growth rates, while TEST and large CON had high plasma T concentrations and low growth rates. Although our comparisons involving all three groups were confounded by allometry in plasma T levels and growth rates within CON, our direct ANCOVA comparison of CAST *vs* TEST unequivocally demonstrated that exogenous T inhibits growth in castrated males of *S. virgatus* ($F_{1,23}$=27.67; P<0.001).

In *S. jarrovii*, growth rate of experimental males decreased with *SVL* ($F_{1,34}$=5.14; P=0.031), and the slope of this relationship was similar across treatment groups ($F_{2,34}$=1.24; P=0.304). ANCOVA revealed a strong effect of treatment on growth rate ($F_{2,34}$=18.08; P<0.001), such that least-square mean growth rate was lower in CAST than in CON or TEST (Fig. 3B). Our ANCOVA comparison of CAST *vs* TEST reinforced the conclusion that exogenous T promotes growth in castrated males of *S. jarrovii* ($F_{1,24}$=27.47; P<0.001), directly opposite our findings for *S. virgatus*. Treatment effects on mass gain (data not shown) were nearly identical to those that we observed for growth rate in *SVL*.

Discussion

Our plasma T data for natural yearlings of *Sceloporus virgatus* and *S. jarrovii* reveal similar overall patterns with respect to sex and season. In both species, mean plasma T levels were higher in males than females throughout the first year of life, and males exhibited a seasonal peak in plasma T levels during the mating season (Fig. 1). In adult males of *S. virgatus*, Abell (1998c) reported a peak in plasma T levels during the mating season (mean 76.78 ng ml^{-1} in April and May), with somewhat lower levels observed after the mating season (mean 41.38 ng ml^{-1} in August). Our values for yearling males (Fig. 1A) are slightly lower in both May (mean 65.86 ng ml^{-1}) and August (mean 25.46 ng ml^{-1}), probably because our samples included some immature yearlings with low plasma T levels (see below). Despite these minor differences, our respective descriptions of plasma T levels in *S. virgatus* reveal similar overall patterns with respect to sex and season. In adult males of *S. jarrovii*, Moore (1986) reported dramatically higher plasma T levels during the fall mating season (mean about 55 ng ml^{-1} in October) than during the summer (mean about 4 ng ml^{-1} in June). These values are nearly identical to our data for yearling males in early July (mean 4.87 ng ml^{-1}) and late September (mean 55.21 ng ml^{-1}).

During the mating season, many small yearling males of *S. virgatus* had low plasma T levels similar to females, but only a few of the smallest males of *S. jarrovii* had such low plasma T levels (Fig. 2). This may reflect size-dependent variation in the attainment of physiological maturity among yearling males of *S. virgatus* (Ballinger and Ketels, 1983). Despite this difference, yearling males of both species exhibited a seasonal peak in plasma T during the mating season, and sex differences in plasma T levels were maximal at this time. These similarities between species stand in contrast to the observation that yearling males of *S. jarrovii* grow more quickly than females during the mating season, whereas yearling males of *S. virgatus* grow more slowly than females during the mating season (Cox, 2005; R. M. Cox and H. B. John-Alder, manuscript submitted). Our experimental data are consistent with the interpretation that these opposite growth patterns between species reflect underlying differences in the effect of T on male growth (Fig. 3).

Our results provide the first direct evidence for opposite effects of T on male growth in two closely related species with opposite patterns of SSD. Even within entire vertebrate classes, opposite effects of androgens on growth are virtually unknown, regardless of patterns in SSD. In the only other comparable study, Fennell and Scanes (1992a,b) found that androgens inhibit growth in male-larger domesticated chickens but promote growth in male-larger domesticated turkeys. Among mammals, androgens stimulate the somatotrophic axis and promote growth in numerous male-larger primates, ruminants and rodents (Borski et al., 1996; Ford and Klindt, 1989; Gatford et al., 1998; Wehrenberg and Giustina, 1992). Similar studies of female-larger mammals are generally lacking, although castration promotes male growth in the female-larger golden hamster (Swanson, 1967). Androgens stimulate the somatotrophic axis and promote growth in several male-larger fishes (Holloway and Leatherland, 1998; Huggard et al., 1996; Kuwaye et al., 1993; Larsen et al., 2004), but we are not aware of any analogous studies of female-larger species. Castration has no effect on male growth in the only amphibian studied to date, the bullfrog *Rana catesbeiana* (Hayes and Licht, 1992). However, the direction of SSD varies in this species and is probably the result of sex differences in survival to large size, rather than sex differences in age-specific growth rate (Howard, 1981). Unfortunately, the ecological and evolutionary relevance of many of the above studies is uncertain, since most were conducted in artificial laboratory or feedlot environments on laboratory strains or domesticated varieties produced by artificial selection (often for desired growth and body composition phenotypes).

Our results for *S. jarrovii* provide the first unambiguous experimental evidence for promotion of skeletal growth (i.e. elongation) by T in any squamate reptile. Although prenatal exposure to T stimulates postnatal mass gain in the lizard *Lacerta vivipara* (Uller and Olsson, 2003), every other study involving lizards and snakes has found either no effect or an inhibitory effect of T on skeletal growth or mass gain (Abell, 1998a; Cox et al., 2005; Crews et al., 1985; Hews et al., 1994; Hews and Moore, 1995; Klukowski et al., 1998; Lerner and Mason, 2001; Marler and Moore, 1989, 1991; Salvador and Veiga, 2000). While these studies collectively suggest that growth inhibition by T may be prevalent in this group, several caveats beg mention. First, several of the above studies reported severe mass loss and/or reduced survival among T-implanted animals (Abell, 1998a; Hews et al., 1994; Hews and Moore, 1995; Lerner and Mason, 2001), prompting some authors to acknowledge concerns over pharmacological T levels (Hews et al., 1994, pp. 110–112; Hews and Moore, 1995, p. 99; Lerner and Mason, 2001, p.223). Our experiments did not have this problem, since our induced plasma T levels were well within normal physiological limits (Fig. 1) and our measures of recapture success (an index of survival) were similar for CON and TEST groups. Second, many of these previous studies were conducted in captivity (Abell, 1998a; Crews et al., 1985; Hews et al., 1994; Hews and Moore, 1995; Lerner and Mason, 2001). In our own experience, laboratory conditions can ameliorate sexual growth differences in both *S.*

undulatus (Haenel and John-Alder, 2002) and *S. jarrovii*, and we have repeatedly failed to detect any difference in growth among CAST, CON, and TEST males of *S. jarrovii* in captivity (R. M. Cox, M. M. Barrett, K. L. Facente, V. Zilberman, and H. B. John-Alder, unpublished observations). Thus, while T may stimulate growth under natural conditions, other factors (e.g., *ad libitum* food) are presumably sufficient to promote rapid growth in the absence of T. Finally, treatment with exogenous T alone may be misleading in the absence of a complementary manipulation to remove the source of endogenous hormone. For example, neither our study nor a previous experiment (Marler and Moore, 1989) found a difference in skeletal growth between T-implanted and control males of *S. jarrovii*. However, our inclusion of a CAST treatment enabled us to demonstrate that T promotes growth in yearling males of this species. This discrepancy may also reflect the fact that Marler and Moore (1989) manipulated T levels in older males, in which growth is slow and sex differences in growth are minor.

Our results clearly show that T inhibits growth in *S. virgatus* and promotes growth in *S. jarrovii*, but we cannot definitively say how or why T elicits these opposite growth responses. One possibility is that both growth inhibition and growth promotion by T represent proximate mechanistic targets of selection for adaptive male body size. For example, intrasexual selection should favor large male body size in *S. jarrovii* because larger males win agonistic encounters and have greater reproductive success than small males (Ruby, 1978, 1981; Ruby and Baird, 1993). On a proximate level, this may result in the evolutionary coupling of a male-specific mediator such as T to some existing physiological mechanism(s) for growth promotion (Badyaev, 2002). For example, in many mammals and fishes with male-larger SSD, androgens promote growth by stimulating the transcription, synthesis and secretion of mitogens such as growth hormone (GH) and insulin-like growth factor-I (IGF-I; e.g. Borski et al., 1996; Holloway and Leatherland, 1998; Larsen et al., 2004; Riley et al., 2002; Wehrenberg and Giustina, 1992). By analogy, the evolution of growth inhibition by T may reflect intrasexual selection for small male size, resulting in inhibitory effects of T on these components of the endocrine growth axis. However, males of *S. virgatus* also exhibit intense intrasexual aggression in competition for breeding females (Smith, 1985; Vinegar, 1975a), and mating success is greater in large than small males (Abell, 1997, 1998b). This mating advantage of large size is particularly strong within yearling males (Abell, 1997, 1998b), for which we have shown that T inhibits growth. Thus, it seems unlikely that intrasexual selection for small male size has driven the evolution of either female-larger SSD or growth inhibition by T in *S. virgatus*.

One alternative explanation is that growth inhibition by T reflects an energetic trade-off resulting from increased activity or territorial defense. Although we did not measure daily activity period, movement, or home range area of experimental *S. virgatus* males, we have previously shown that exogenous T increases each in males of a closely related species, *S. undulatus* (Cox et al., 2005). Our manipulations in *S. virgatus* may have shifted energy allocation towards either growth (CAST) or these components of male reproductive investment (TEST). Natural peaks in plasma T levels during the breeding season (Fig. 1) may mediate similar energetic trade-offs, leading to reduced male growth and the development of female-larger SSD (Cox, 2005; R. M. Cox and H. B. John-Alder, manuscript submitted). Patterns of size-dependent variation within yearling males of *S. virgatus* are consistent with this T-mediated energetic trade-off hypothesis; growth rate decreases with body size (Fig. 3), while plasma T levels (Fig. 2), reproductive maturity (Ballinger and Ketels, 1983) and mating success (Abell, 1997, 1998b) are positively related to size.

If energetic costs of T inhibit growth in *S. virgatus*, why do they not also inhibit growth in *S. jarrovii*? Previous studies have shown that T increases activity and territorial aggression in adult males of *S. jarrovii*, resulting in increased metabolic expenditure and decreased energy acquisition (Klukowski et al., 2004, 2001; Marler and Moore, 1988, 1989, 1991; Marler et al., 1995; Moore, 1988; Moore and Marler, 1987). However, although yearling males of *S. jarrovii* have elevated plasma T levels during the mating season (Fig. 1), they devote a considerably smaller fraction of their annual energy budget to reproduction than do older males (Congdon, 1977). Additionally, the environmental potential for an energetic trade-off with growth may be greater for *S. virgatus* in the spring than for *S. jarrovii* in the fall. *Sceloporus virgatus* breeds during the driest months of the year (April–June), when arthropod (prey) densities are relatively low, while *S. jarrovii* can presumably take advantage of seasonal peaks in arthropod abundance during the monsoon rains that precede the fall mating season (Smith, 1996; Smith and Ballinger, 1994). Further, *S. virgatus* breeds upon emergence from hibernation, before depleted energy reserves can be replenished (Smith, 1996), while *S. jarrovii* yearlings have several months of activity to store energy before breeding. These mechanisms are only conjectural, but it is clear that, if the energetic cost of male reproductive investment is to provide a parsimonious explanation for T-mediated growth inhibition in *S. virgatus*, then *S. jarrovii* must somehow differ such that these costs are reduced.

Given the nature of our experiments, we have focused our discussion on growth and body size of males, but any complete explanation for SSD must also consider female body size. Thus, opposite patterns of SSD in *S. virgatus* and *S. jarrovii* may be related primarily to differences in factors acting on female size. For example, Abell (1998b) argues that fecundity selection for large female size may be stronger in *S. virgatus* than *S. jarrovii* because the slope of the regression line relating clutch or litter size to female *SVL* is steeper in *S. virgatus*. Alternatively, energetic costs of reproductive investment may differentially constrain female growth in *S. virgatus vs S. jarrovii*. For example, we have found that ovariectomized yearlings of *S. jarrovii* grow faster than pregnant yearling females (Cox, 2005), providing strong experimental evidence

for an energetic trade-off between female growth and reproduction in this species. However, comparisons of reproductive *vs* naturally non-reproductive females suggest that reproduction constrains growth in *S. virgatus* as well as *S. jarrovii* (R. M. Cox and H. B. John-Alder, submitted manuscript; Vinegar, 1975b; but see Smith, 1997). Ultimately, while factors influencing female size may be important with regard to SSD, the fact remains that we observed opposite effects of T on male growth in *S. virgatus* and *S. jarrovii*.

Given the inherent limitations of two-species comparative studies (Garland and Adolph, 1994), we cannot definitively conclude that the opposite growth responses to T that we observed are directly related to differences in SSD between *S. virgatus* and *S. jarrovii*. However, our results clearly raise interesting questions about the role of T in mediating sex differences in growth and body size. The diversity of SSD in *Sceloporus* makes this group an ideal comparative system for further study. We hypothesize that growth promotion by T represents a proximate physiological target of sexual selection for large male body size, and predict that T promotes (or castration inhibits) male growth in other male-larger *Sceloporus* species. A test of this prediction would greatly strengthen our understanding of growth and SSD in *Sceloporus*, as would future studies of the effects of T on components of the endocrine growth axis (e.g. GH, IGF-I, IGF binding proteins). Such studies would be particularly informative in a comparative context involving both male- and female-larger species. For example, we propose that sexual selection favors large male size in both male- and female-larger *Sceloporus* species, but that energetic costs of elevated T may indirectly constrain growth in some species (e.g. *S. undulatus* and *S. virgatus*). Thus, at a proximate level, one might predict that T stimulates the endocrine growth axis in all *Sceloporus* species, despite growth inhibition by T at the organismal level in female-larger species. Alternatively, the opposite growth responses that we observed in *S. virgatus vs S. jarrovii* may reflect fundamental differences in the effect of T on the endocrine growth axis. This possibility is particularly interesting in light of other apparent exceptions (Fennel and Scanes, 1992b; Swanson, 1967) to the established role of androgens as growth promoters in vertebrates (e.g. Borski et al., 1996; Holloway and Leatherland, 1998; Larsen et al., 2004; Riley et al., 2002; Wehrenberg and Giustina, 1992).

Abbreviations

CAST	castrated treatment group
CON	control treatment group
DHT	5α-dihydrotestosterone
DMSO	dimethyl sulfoxide
GH	growth hormone
IGF-I	insulin-like growth factor-I
PBSG	phosphate-buffered saline with gelatin
RIA	radioimmunoassay
SSD	sexual size dimorphism
SVL	snout–vent length
T	testosterone
TEST	testosterone treatment group

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