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Testosterone and competitive ability in male house mice, *Mus musculus:* laboratory and field studies

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Abstract. Testosterone enhances aggression and can facilitate dominance in mammals, but most studies have been conducted on laboratory strains of mice and in simple environments. Unlike previous studies, laboratory and field tests were conducted using wild house mice, *Mus musculus,* to determine whether males with high, but physiological, testosterone levels (High T) would achieve dominance compared with males with low testosterone levels (Low T). All males were castrated and androgen was replaced with a single injection of testosterone enanthate, a long-acting androgen. At 8 days post-treatment the mean testosterone concentration in High T males was 5·1 ng/ml, whereas the level in Low T males was 1·3 ng/ml. Contests in the laboratory revealed that High T males achieved dominance over Low T males in the presence of females. In the field, equal numbers of High and Low T males, and a corresponding number of females, were released on three highway islands at densities equivalent to the highest density reported on similar sites. Periodic retrapping produced a capture history for males in each group and differences in survival were analysed using the program SURVIV. High T males had a higher probability of survival than Low T males on one island, but on the other two islands the higher capture rates of High T males were not statistically greater than those for Low T males. Of those that survived to the first trapping episode, the High T males had greater subsequent longevity than Low T males. On all three islands High T males were caught more frequently at feeding stations that were established to enhance food competition. There were no differences among the home range sizes of High T males, Low T males and females. However, on two of the islands the ranges of High T males overlapped more of the ranges of resident females than those of Low T males. The findings from this study, using testosterone-treated subjects, suggest that natural variation in basal testosterone also may affect competitive ability in wild house mice.

Testosterone regulates spermatogenesis and mating behaviour in males. However, testosterone also mediates male-male aggression, presumably due to its aromatization to oestrogen in the brain (Naftolin et al. 1971; Dessi-Fulgheri et al. 1976; Brain 1983). Socially dominant males are often the most aggressive animals and are assumed to be capable of acquiring more resources than less aggressive individuals. Thus, one might expect individual males with higher testosterone levels to be more aggressive, to command greater control over resources, including females, and to have the highest fitness. However, a relationship between plasma testosterone, dominance and fitness has been difficult to establish. While testosterone appears to promote aggression, this behaviour does not necessarily facilitate dominance (e.g. Mazur 1976; Dixson 1980; Caldwell et al. 1984; Sachser & Prove 1984; Berkovitch & Coy 1990). Moreover, dominance does not always guarantee high relative fitness; a myriad of other factors influence reproductive success. Even where a relationship between androgens and dominance has been discovered it is often unclear whether a difference in testosterone caused the dominance pattern or resulted from it (Beach 1965). Luteinizing hormone and testosterone levels rise during a male 'challenge' (Wingfield et al. 1990) or after exposure to a receptive female (Batty 1978; Bronson & Desjardins 1982) and can fall quickly after a defeat (Bronson & Desjardins 1971; Bronson et al. 1973; Martinez et al. 1989). Testosterone levels also fluctuate greatly even without social stimulation (Bartke et al. 1973; Bartke & Dalterio 1975; Coquelin & Desjardins 1982). Fluctuations in basal testosterone levels

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and changes in testosterone levels due to social conditions make it difficult to relate variation in underlying levels of dominance and competitive ability.

Despite this complexity, several mammalian studies have demonstrated a positive relationship between plasma testosterone and aggressive dominance in the laboratory and the field (e.g. Rose et al. 1971, 1972; Bramley & Neaves 1972; Lincoln et al. 1972; Buhl et al. 1978). Laboratory studies relating dominance to plasma testosterone levels in rodents have produced mixed results. Schuurman (1980) demonstrated a significant relationship between plasma testosterone and patterns of aggressive behaviour in rats; a finding of special significance because testosterone levels were measured repeatedly in each individual to control for its pulsatile release. Similarly, Buhl et al. (1978) found that dominant male collared lemmings, *Dicrostonyx hudsonicus*, had higher plasma testosterone levels and heavier ventral prostates than subordinates. The size of the androgen-dependent flank gland in male hamsters, *Mesocricetus auratus*, is positively related to dominance status, and dominance also is related to the dose of testosterone administered (Drickamer et al. 1973). Although Selmanoff et al. (1977) found no relationship between dominance and testosterone in inbred strains of *Mus*, subordinate individuals had lower testis and seminal vesicle weights. Sachser & Prove (1984) found evidence that the absolute value of plasma testosterone was less important in explaining fighting intensity than the similarity of testosterone levels between opponents. Also arguing for some relationship between dominance, aggression and plasma testosterone is the genetic covariance of aggression and testosterone levels. House mice selected for short attack latencies had higher plasma testosterone levels than unselected controls (Van Oortmerssen et al. 1987) and inter-male aggression has been correlated with serum testosterone in several different strains of mice and their F_1 hybrids (Maxson et al. 1983).

A considerable amount of negative data has been reported as well. Maruniak et al. (1977) demonstrated that a reduction in circulating testosterone concentration was not a prerequisite for subordination in mice. Submissive behaviour and reduced urinary marking occurred in the losers of forced encounters even when their testosterone levels were artificially elevated. Hull et al. (1977) claim that in the gerbil, *Meriones unguiculatus*, there is no simple relationship between basal levels of testosterone and aggressiveness, and Barkley & Goldman (1977) discovered that body weights in mice explained victory or loss in dominance trials more than did each individual's respective testosterone level.

While the laboratory studies are informative they have their drawbacks. Testosterone titres and levels of aggression can be higher, and more variable, in wild than in captive males (e.g. Drickamer 1973; Wingfield & Moore 1987). Isolation housing can alter the neural aromatization of androgens (Di Prisco et al. 1978), a process considered necessary for the expression of inter-male aggression. In addition, the rearing and testing context (especially presence or absence of females, e.g. Sachser & Lick 1991) and the type of opponent (Martinez et al. 1989) can profoundly influence the outcome of dyadic encounters. Failure to discover a consistent relationship between testosterone, aggression and dominance may be influenced by the variety of laboratory contexts that have been used (Brain 1983). Add to these problems the difficulty of trying to characterize the mean plasma testosterone level on the basis of a single measurement and it is not surprising that the relationship between agonistic behaviour and androgen levels has been inconsistent.

For the reasons outlined above, field studies of the relationship between testosterone and dominance are of particular relevance. Natural social units, ambient climatic conditions and the natural stressors of food limitation, pathogens and predators may be important to understanding the role of androgens in competitive ability. Caldwell et al. (1984) collected blood samples from wild wood rats, *Neotoma fuscipes*, recently removed from the field and determined that plasma testosterone levels correlate with seasonal changes in aggression. However, elevated testosterone was not required for the maintenance of aggression. Gipps et al. (1981) and Krebs et al. (1977) found no population or social consequences of treating intact male voles, *Microtus townnsendi*, with additional testosterone. But, treatment with hyperphysiological levels of testosterone can be less effective than replacing testosterone in castrates (Suchowsky et al. 1969) and can also produce atypical actions (Brain 1983). While it is possible that a simple relationship between circulating androgens and dominance behaviour in natural populations does not exist, this conclusion may be premature since most studies probably occur during periods of relative social stability. This condition may not be representative of the potential for interaction between

testosterone and dominance. Several studies of testosterone levels in free-living mammals (Sapolsky 1983) and birds (Hegner & Wingfield 1987; Dufty & Wingfield, personal observations, cited in Wingfield & Marler 1988) found a relationship between testosterone and aggressive dominance during periods of instability that was absent during social equilibrium.

We tested whether a difference in basal testosterone level would affect aggression in the laboratory and also affect a male's ability to survive periods of social turmoil in the field. The castrated males treated with the higher of two doses were considered to simulate either the endocrine state of males during a victorious encounter, the high end of natural variation in basal testosterone levels, or both. In addition to determining whether a difference in testosterone levels affects competitive ability in males, we compare our results to a similar study where female competitive success increased significantly after testosterone treatment (Zielinski & Vandenbergh 1991).

METHODS

Subjects

The subjects were descendants of wild house mice caught near Alberta, Canada (Perrigo & Bronson 1985) and were produced from a colony maintained in our laboratory for 3 years. At 25 days of age males were weaned to individual cages (10 x 13 x 23 cm) and female pups were housed in groups of two to four. Laboratory photoperiod was maintained on a 14:10 h 1ight:dark cycle at a temperature of about 22°C.

Preliminary Laboratory Experiments

Testosterone measurements in intact and castrated untreated males

Before we began the field experiment it was necessary to determine the endogenous levels of testosterone in intact males so we could produce experimental males for the field-release with either high or low levels of testosterone within the normal physiological range. Because all males would be castrated prior to treatment we also sought to verify that castration significantly reduced circulating testosterone.

Serum testosterone was determined for 65- to 99-day-old males that were either (1) intact and isolated since weaning (N = 6), (2) intact and housed with an adult female for 3 days prior to blood collection (N = 10), or (3) isolated since weaning and castrated at about 40 days of age (N = 8). Castrations were conducted under metafane (Methoxyflurane; Pitman-Moore, Mundelein, Illinois) anaesthesia and incisions were closed with surgical staples that were removed 10 days later. Trunk blood was collected from all males within 15 s of opening the cage. The experimental samples from individual animals were assayed in duplicate 50 µl aliquots. Testosterone concentration was measured using a commercial radioimmunoassay kit for Total Testosterone (Diagnostic Products Corporation, Los Angeles, California). The range of the standard curve was from 0·2 to 16 ng/ml. The intra-assay coefficient of variation was 1·6% and dilutions of serum calibrators provided with the kit displaced labelled testosterone in a manner parallel to the standard curve. Recovery of known quantities of testosterone averaged 95·2%. Cross-reactivity with androstenedione, corticosterone, 5 alpha-dihydrostestosterone, and oestradiol was 0·4, 0·002, 3·3 and 0·02%, respectively. Mean (± SE) testosterone concentrations for males either paired with a female, isolated since weaning or castrated were $4{\cdot}37 \pm 2{\cdot}02$ ng/ml (range = 0·1–20·0), $2{\cdot}48 \pm 2{\cdot}4$ ng/ml (range = 0·16–13·5) and $0{\cdot}19 \pm 0{\cdot}03$ ng/ml (range = 0·15–0·24), respectively.

Effect of time since treatment on plasma testosterone, preputial mass and aggression

On the basis of the radioimmunoassay results presented above and the results of a previous study involving testosterone-treated females (Zielinski & Vandenbergh 1991) we administered single injections of either 500 µg (High T) or 50 µg (Low T) testosterone enanthate in 0·05 ml of peanut oil to castrated males. Testosterone enanthate is a long-acting ester that is capable of maintaining ejaculation in castrated rats up to 9 weeks after a single injection (Beach & Sprague 1971). Testosterone enanthate has minimum crossreactivity with the testosterone antibody (0·12%; Diagnostic Products Inc.) so the assay detected only testosterone that had been cleaved from the ester. The 500 and 50 µg doses resulted in mean (± SE) serum testosterone concentrations of $12{\cdot}4 \pm 1{\cdot}8$ ng/ml and $1{\cdot}74 \pm 0{\cdot}27$ ng/ml at 2 days post-treatment, respectively. Testosterone levels in intact, adult males in two previous studies were 0·9-38·0 and 1·6–5·7 ng/ml (Grota 1971; Bartke et al. 1973). Other work with testosterone esters has indicated that these values

would decline by about 50% within a week (Kuhl et al. 1979; Marshall et al. 1983), so we expected the average values through 1 week post-injection to be sufficiently disparate and within the physiological range. We used these doses in all subsequent experiments.

Because it was not possible to monitor serum testosterone levels and to witness aggressive encounters in mice after their release in the field, we collected these data from a separate group of wild mice in the laboratory at specified intervals after treatment. At a minimum of 2 weeks after castration we weighed 69 males and injected them with either 500 or 50 μg of testosterone enanthate in 0·05 ml of peanut oil. At this time pairs of mice that were of equivalent weight were identified for subsequent dominance testing. Any slight weight disparity favoured the Low T male. We tested a separate subset of these males for dominance at either 2, 7 or 14 days post-treatment. Shortly before a trial began we handled each male, and chose one at random to receive a distinctive hairclip so that each individual could be identified during the trial. Trials were conducted in the afternoon, a few hours before lights went off, by an observer unaware of the treatment identity of the participants. One male from each treatment was placed into a neutral cage (13 x 18 x 28 cm) and the pair was observed for 15 min or until persistent attacks appeared to threaten a mouse's well-being. The dominant mouse was easily identified by its persistent attacks and assertive behaviour patterns (e.g. aggressive grooming, deliberate approaches and increased activity) while the subordinate male displayed frequent submissive behaviour patterns (e.g. immobility, rearing and grooming tolerance) and did not respond to attacks with aggressive defence (Catlett 1961; Barkley & Goldman 1977). When attacks were infrequent or when both males were equally aggressive the trial was designated a draw. Twenty-four hours after their encounter we killed the males (3, 8 and 15 days post-treatment, respectively) and collected trunk blood for testosterone assay. We also removed the androgen-sensitive preputial glands (Brown & Williams 1972) while in a semi-frozen state, and weighed them.

Effect of presence of a female on testosterone-mediated dominance

Dominance in the wild is established in the presence of females and may be enhanced by stimuli from females. Laboratory studies of dominance indicate that aggressive interactions between males are more frequent and intense when females are present (e.g. Brain et al. 1978). Therefore, we also observed the dominance relations of 14 pairs of weight-matched High and Low T males in a cage containing an adult female. We observed each trio for 20 min immediately after grouping, and again for 5 min after they had been grouped for 48 h. Dominance was assessed as previously described. After the second observation we killed both males in six of the 14 trials, then skinned and examined the underside of each pelt to determine the extent of wounding (Selmanoff et al. 1977; Brain 1983). We used the information from this sample to validate the accuracy of our behavioural assessment of dominance.

Field Procedures

Our field sites were 'highway islands' formed by the exit and entrance ramps at a highway interchange (Massey & Vandenbergh 1980; Massey 1982; Zielinski & Vandenbergh 1991). Highway islands were used because many include habitats used by wild house mice and because emigration off the islands is low (Massey 1982; Coppola 1986).

The particular interchange we used was developed and seeded 9 years prior to this study, and the vegetation is typical of an old-field community, 5-10 years after abandonment (Keever 1950). Dog fennel, *Eupatorium canadense*, goldenrod, *Solidago* sp., blackberry, *Rubus* sp., broomsedge, *Andropogon* sp., and isolated loblolly pines, *Pinus taeda* were the dominant native species. However, sericea and annual lespedezas, *Lespedeza* sp. and fescue, *Festuca* sp., that were established during construction were also widespread. Although the plant community on each island was similar we characterized the vegetative cover on each island using 10 systematically arranged 4 m^2 plots per island. Vegetation height and density reflect soil characteristics and both plant cover and soil conditions can influence the suitability of the habitat for house mice (Newsome 1970; Massey 1980). We collected vegetation samples in late August, approximately a month after the conclusion of the experiment. We recorded the mean height of the vegetation by averaging the four measurements taken at each corner of the plot. We assessed vegetative cover by determining the percentage of the plot covered by each plant species found there. This was determined by

estimating the percentage of the plot that a shadow cast by all the individuals of a particular species would occupy, and thus could exceed 100%. The following categories of per cent coverage were used; 0-1, 2-5, 5-10, 10-25, 25-50, 50-75, 75-95, 95-100. For purposes of analysis these were ranked from 1-8 and the sum of these ranks for a particular plot was referred to as the vegetative cover index.

We promoted competition and social instability among the populations by (1) introducing a total number of animals on each island that was equivalent to the highest densities recorded on similar sites (Massey & Vandenbergh 1980; Coppola & Vandenbergh 1987) and (2) by establishing a few feeding stations on each island. We assumed that competitively dominant males would defend areas around permanent supplies of food (Stueck & Barrett 1978; Ims 1987). Cracked corn and sunflower seeds were provided every 10 days under the cover of 1 x 1 m wooden boards supported by bricks.

Three weeks before we released the mice on the islands we set traps to capture and relocate the resident small mammals. These were checked twice a day for at least 3 days each week. We used the same trap distribution to remove and later recapture the experimental house mice. We placed 7 x 7 x 25-cm Sherman live-traps at 5-m intervals along two traplines, situated 5-m apart, and placed two traps at each feeding station.

The experiment included three replicates run on three different islands at the interchange. The northwest, southeast and southwest islands included l·02, 0·95 and 0·64 ha of unmowed area and were covered with 78, 73, and 44 traps (including those at feeding stations), respectively. A replicate consisted of releasing 20 High and 20 Low T males and 40 untreated females. The males were approximately 60 days old and had been castrated about 2 weeks earlier. Each male was administered its dose of testosterone 3 days before release. We released the mice at the centre of the island in the late afternoon on 14 May 1991. We recaptured the males on four occasions during the 7 weeks that followed their release. Each occasion included 3 consecutive days where traps were set in the late afternoon, checked at dawn, closed for the rest of the day and reopened again in the afternoon. The first morning of each of the four recapture occasions occurred 8, 21, 35 and 56 days after each release resulting in four between-capture intervals of 7, 11, 12, and 18 days, respectively.

To determine whether mice were leaving the islands immediately after release, and whether the emigrants differed on the basis of treatment, we also set 20 traps immediately outside the roadway that isolated each island. These traps were situated under a hedgerow along the circumference of the roadway and were set the afternoon of the release and checked each morning and afternoon for 3 days.

Analyses of Survivorship, Dominance and Space Use

We analysed differences in survival between High and Low T males, between males and females and among islands using the program SURVIV (White 1983). SURVIV was used to test differences in survival rates and differences in capture rates. These effects must be tested separately because the treatment can influence the ability of one group to be captured relative to the other, independent of treatment effects on survival. For example, the treatment may affect a group's response to traps or may decrease activity, which in turn would decrease the group's rate of encountering traps. These effects could be confounded with true differences in survival.

Using the number of marked animals recaptured at each trapping episode, SURVIV tests hypotheses using chi-squared tests of log-likelihood ratios. First, a General Model is maximized where the survival or capture probabilities for all four trapping intervals and for each treatment group are estimated from the data separately. By definition, the fit of this model is quite good. Then, additional constraints (e.g. the survival rate of High T and Low T males are equal; the capture rate of High T males is the same on all islands) are imposed to define each alternative hypothesis. The log-likelihood is maximized for each alternative and it is compared to the General Model. The ratio of log-likelihoods can be interpreted as a chi-squared test for the purpose of testing whether the alternative model, with its constraints, is equivalent to the fully parameterized General Model. For example, if we wish to compare the survival rates of High and Low T males, SURVIV will use the recapture data to generate a General Model that estimates the survival rates of High T and Low T males independently. Then, an alternative hypothesis that the survival rates are indistinguishable is constructed. A significant chi-squared indicates

that the alternative is an unacceptable model of the data and that the male survival rates differ.

The area that an individual used was also calculated for each mouse that was captured at least three times using the location analysis program McPAAL (M. Stuwe & C. E. Blohowiak, unpublished data). The mean number ($\pm$SE) of captures for males included in the analysis was 6·15 $\pm$ 0·6 (range = 3-12). McPAAL calculates minimum convex polygons for each individual; the means of these areas for High T males, Low T males and females were compared using *t*-tests. An additional measure of dominance was determined by comparing the number of High T males, Low T males, and female mice caught at feeding stations. Dominant animals were presumed to establish home ranges near feeding stations.

Terminal Serum Testosterone Concentrations and Preputial Masses

All males trapped at the final capture occasion were brought into the laboratory and killed to determine serum levels of testosterone and preputial gland masses. The longest time period between treatment and measurement of these responses in the laboratory was 15 days. Thus, the collection of field-released mice at the end of the experiment allowed us to observe the effects of treatment approximately 2 months after injection.

RESULTS

Laboratory Experiments

Effect of time since treatment on plasma testosterone, preputial weight and aggression in paired encounters

Serum testosterone decreased with the time since administration of testosterone enanthate (Fig. 1). At 3 days post-treatment, serum levels in High T males were about six times higher than those in Low T males. By 15 days post-treatment, the difference was only about four-fold. At all three time points the High T values were significantly greater than the Low T values (3 days, $F = 8{\cdot}71$, $P = 0{\cdot}0001$; 8 days, $F = 3{\cdot}92$, $P = 0{\cdot}0002$; 15 days, $F = 3.29$, $P = 0{\cdot}0001$). Serum testosterone in High T males was indistinguishable from intact males at 8 and 15 days post-treatment, but significantly greater than that in intact males at 3 days post-treatment ($F = 11{\cdot}80$, $P = 0.0025$).

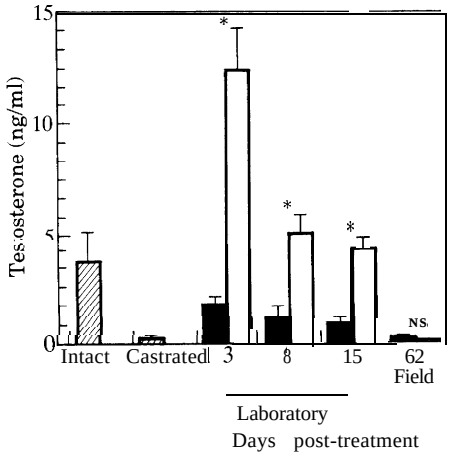

Figure 1. Serum testosterone levels in intact, untreated castrated (▨), and High T (□) and Low T (■) males. Asterisks indicate significant differences ($P < 0{\cdot}05$) between High and Low T males (laboratory and field) examined at the same number of days post-treatment.

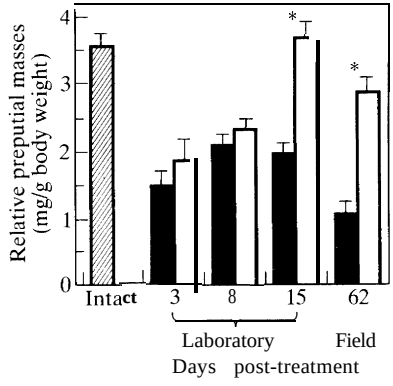

Figure 2. Relative preputial masses of intact males (▨), and castrated High T (□) and Low T (■) males. Asterisks indicate significant differences ($P < 0{\cdot}05$) between High and Low T males (laboratory and field) examined at the same number of days post-treatment.

Preputial weights did not differ between treatments until 15 days post-treatment, apparently requiring some time before they responded to the restored testosterone levels (Fig. 2). However, by 15 days the preputial weights in High T males were no different than those of intact males.

Surprisingly, there were no differences in the number of times a High T or a Low T male was considered a winner in laboratory dominance trials

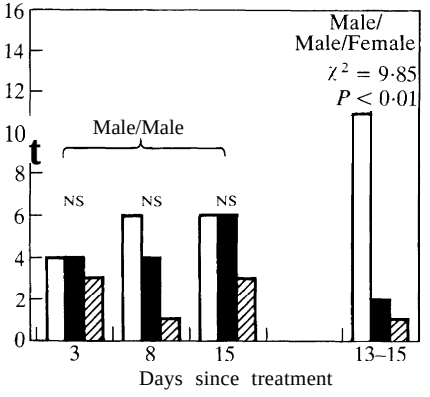

Figure 3. Results of laboratory dominance trials conducted at different times since treatment and for different social conditions. High T win (□), High T loss (■) and Draw (▨).

when there was no female present (Fig. 3). Equivalent numbers of High and Low T males were winners at 3, 8 and 15 days post-treatment.

Effect of presence of a female on testosterone-mediated dominance

The results of the laboratory dominance trials changed significantly when High and Low T males were tested in the presence of a female, and observed 48 h after the trios were placed in a common cage (Fig. 3). In 11 of 14 trials (78·6%) the High T male achieved dominance. One trial was a draw, and the Low T male won in two trials. In the cases where the Low T male won, he weighed 3·9 g more than his opponent in one case and 0·8 g less than him in the other. The dominance pattern was developing in the 20 min immediately after grouping (five High T male wins, three Low T male wins and six draws), but it was much easier to identify the dominant male, and in a shorter period of time, 48 h later. In the first six trials we verified our behavioural observations by assessing wounds on the internal surface of skins. In all six cases the male that was designated the loser on the basis of behaviour had conspicuous superficial hemorrhages, and in four cases numerous puncture wounds, in the area of the hindquarters and the tail.

Body size effects on dominance

Males in laboratory dominance trials were assigned treatments on the basis of weight and the slight difference always favoured the Low T males

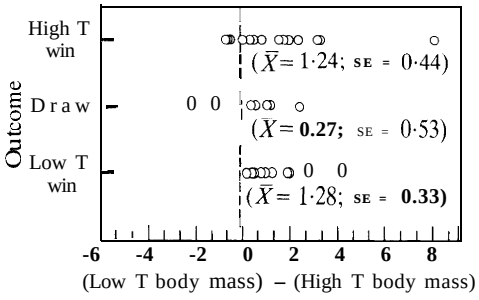

Figure 4. Body mass differences between High and Low T males in laboratory dominance trials related to the outcome of the trial. Trials that included a female are not included ($\bar{X} \pm$ SE).

($\bar{X} \pm$ SE: High T = 21·7 ± 0·4; Low T = 22·7 ± 0·5). Although these weights were recorded at the time of injection, males were tested at various times later. On the day of testing we reweighed both males and in some cases considerable disparity had developed. On five of 51 occasions the High T male surpassed the weight of the Low T male. Because body weight can influence the outcome of dominance trials in rodents (e.g. Drickamer et al. 1973; Barkley & Goldman 1977) we calculated the net weight difference (Low-High) and assessed whether the outcome of the trial was affected by this factor. In no case, whether males were paired alone ($F = 1{\cdot}22$, $P = 0{\cdot}30$ or in the presence of females ($F = 0{\cdot}11$ $P = 0{\cdot}75$), did weight difference influence outcome (Fig. 4). Pooling the outcomes for males tested without females at 3, 8 and 15 days post-injection, the High T males won 14 of the 31 trials in which the Low T males outweighed them. Of the remainder, Low T males won 12 and five were a draw. In those five cases where the High T male was heavier, he won three, and two were a draw. Interestingly, considering all trials, in three cases the High T male won despite the fact that his opponent outweighed him by 3·5 g (16·7%), 6·9 g (25·6%) and 8·0 g (33·5%).

Field Experiment

Survival rates on islands

The initial analysis was conducted to determine whether the response to treatment differed among islands, and if so for which groups. The hypothesis that survival rate of High T males was the same for all islands was rejected ($\chi^2 = 11{\cdot}50$, $P = 0{\cdot}025$; Table I) as was the hypotheses that survival rates were homogeneous across islands for Low T males

Table I. Chi-squared tests for equality of survival and capture rates among islands for High T and Low T males and female mice (*df*= 4 for each test)

	Null hypothesis	Chi-squared	*P*
High T males	Equal capture rates on all islands	10·02	< 0·05
	Equal survival rates on all islands	11·50	< 0·025
Low T males	Equal capture rates	4·12	<0·50 NS
	Equal survival rates	10·74	<0·05
Females	Equal capture rates	2·08	< 0·90 NS
	Equal suurvival rates	30·80	<0·005

Table II. Chi-squared tests for testosterone treatment effects and for sex differences for each island (*df* = 1 for all tests)

Island	Hypothesis	Chi-squared	*P*
Northwest	High T = Low T		
	Survival	10·18	0·0014*
	Capture	3·98	0·046†
	Male = Female		
	Survival	0·98	0·325
	Capture	0·81	0·5215
Southwest	High T = Low T		
	Survival	0·75	0·385
	Capture	2·16	0·141
	Male = Female		
	Survival	0·06	0·800
	Capture	1·00	0·317
Southeast	High T = Low T		
	Survival	0·04	0·844
	Capture	0·95	0·331
	Male = Female		
	Survival	1·18	0·2757
	Capture	0·58	0·4439

*High > Low.
†Low > High.

(χ^2 = 10·74, P< 0·05) and females (χ^2 = 30·8, P< 0·005). The assumption of equality of capture rates across islands was violated for High T males only (χ^2 = 10·02, P< 0·05). Due to this heterogeneity, all subsequent analyses on treatment effects are presented separately for each island. Treatment differences were greatest on the northwest island where the survival rate of High T males was considerably greater than that for Low T males (χ^2 = 10·18, P=0·0014) (Table II; Fig. 5). After the first 3 weeks on the island, when serum testosterone levels were at their most disparate and aggressive interactions were expected to be the greatest, 70·0% of the High T males on the northwest island were known to be alive, whereas only 20·0% of the Low T males were. The number known alive is the sum of those captured at a particular occasion and those individuals not captured then, but captured at a subsequent occasion. The higher survival rate of High T males on the northwest island is even more remarkable considering that Low T males, despite their poor survival rate were more likely to be captured than High T males (χ^2 = 3·98, P=0·046; Table II). Program SURVIV could not discern differences in survival on the southwest (χ^2 = 0·75, P= 0·38) or southeast (χ^2 = 0·04, P = 0·84) islands,

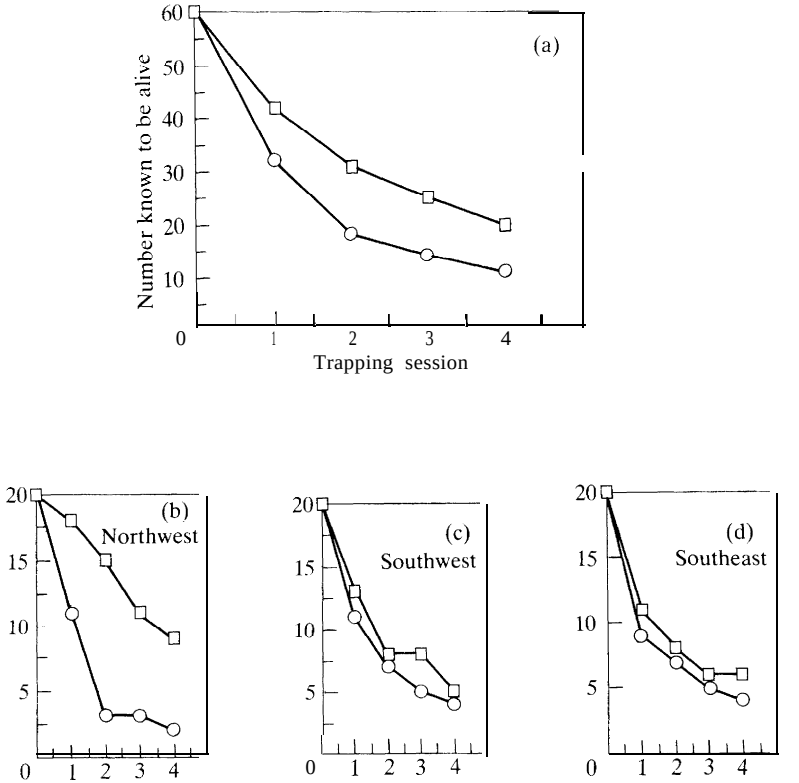

Figure 5. Number of High T (□) and Low T (O) males known to be alive at each of four recapture occasions. (a) All males of each treatment pooled, (b-d) data for each island separately.

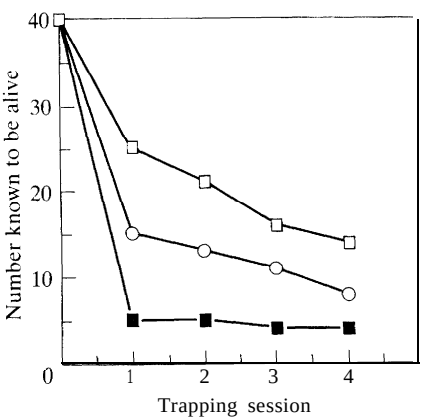

Figure 6. Number of females known to be alive at each of four recapture occasions for each island; northwest (□), southwest (O) and southeast (■).

though in both cases the absolute number of High T males known alive at each occasion exceeded that of the Low T males.

Interestingly, there was also significant heterogeneity of response among females depending on island ($\chi^2 = 48{\cdot}8$, $P < 0{\cdot}00001$; Fig. 6). The survival rate for females, like that of males, was highest on the northwest island. Seven days after release, 60·0% of the males and 47·5% of the females were known to be alive on the northwest island, whereas the numbers were 475% and 25·0% for males, and 35·0% and 10·0% for females on the southwest and southeast islands, respectively. It was clear that the southeast island was the Least hospitable for mice of both sexes.

We also analysed the recapture data on the basis of subsequent longevity of those mice that survived to at least the first recapture episode. Longevity was defined as the amount of time from release to the date of last capture. There were no significant differences among islands ($F = 0{\cdot}23$; $P = 0{\cdot}79$) and when data from all three islands were pooled the High T males and Low T males had mean ($\pm$ SE) longevities of $25{\cdot}9 \pm 2{\cdot}3$ and $16{\cdot}7 \pm 3{\cdot}0$ days, respectively, a statistically significant difference (Fisher's

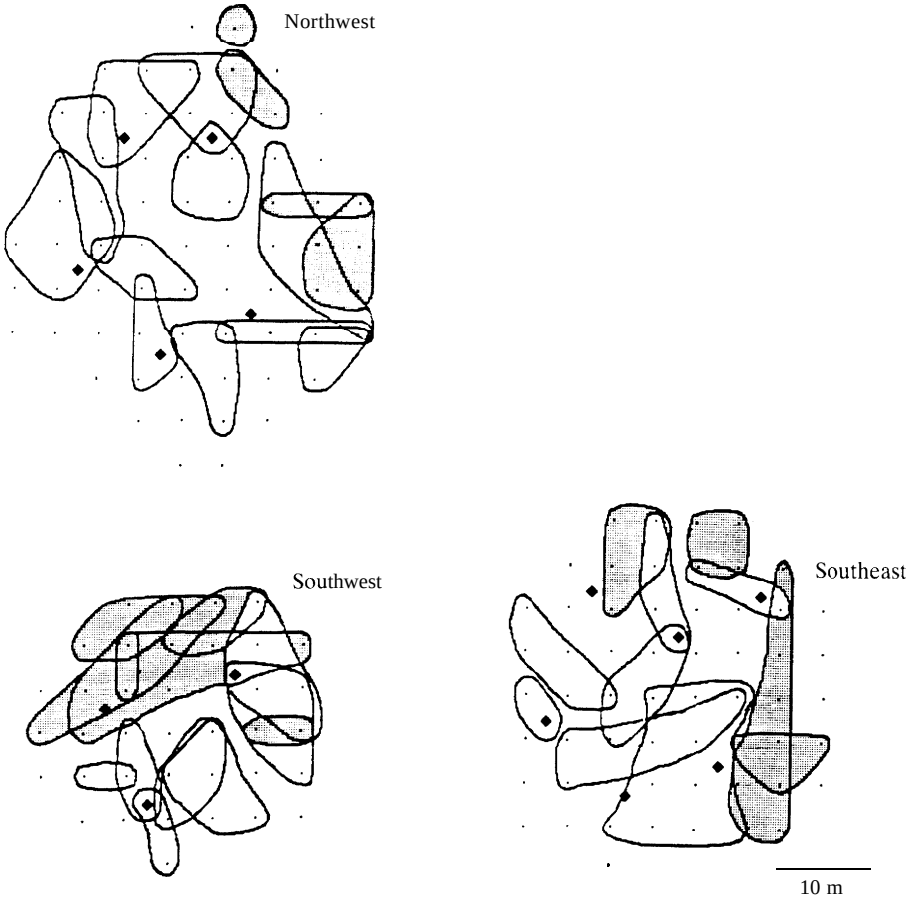

Figure 7. Home ranges of High T (□) and Low T (▨) males on the three islands. Dots are trap sites and diamonds indicate locations of feeding stations.

protected least significant difference, $D = 9{\cdot}22$, $P = 0{\cdot}03$). Interestingly, female mice that survived to at least the first recapture period had mean longevities of $43{\cdot}9 \pm 2{\cdot}7$, significantly greater than the High T males ($D = 18{\cdot}0$, $P = 0{\cdot}0001$).

Body weight at release had little effect on survival because it did not influence whether or not a male was caught on any island (northwest: $F = 0{\cdot}5$, $P = 0{\cdot}63$; southwest: $F = 0{\cdot}7$, $P = 0{\cdot}49$; southeast: $F = 0{\cdot}5$, $P = 0{\cdot}63$). Similarly, the body weights at the time of release were no different for High and Low T males on the northwest and southwest islands; however, High T males were significantly heavier on the southeast island ($P = 0{\cdot}041$). Because the survival rates on the southeast island were most equivalent, body weight differences at release were unlikely to have influenced survival.

Terminal serum testosterone levels and preputial masses in the field

Serum testosterone levels in High T and Low T males that were returned to the laboratory after the conclusion of the field study were no different ($F = 3{\cdot}31$, $P = 0{\cdot}09$) and were each similar to the level in castrates ($F = 4{\cdot}76$, $P = 0{\cdot}054$ and $F = 0{\cdot}2$, $P = 0{\cdot}66$, respectively; Fig. 1). There is a suggestion that mean levels of testosterone were higher in castrates than in High T males, but this has little biological importance given the low values. High T males had significantly heavier preputial glands than did the Low T males (means ($\pm$ SE) = $67{\cdot}14 \pm 5{\cdot}7$ and $24{\cdot}26 \pm 4{\cdot}9$, respectively; $F = 32{\cdot}42$, $P = 0{\cdot}0001$; Fig. 2). Thus, although there was no difference in testosterone level 2 months after treatment, the effect of differences earlier in the experiment were reflected

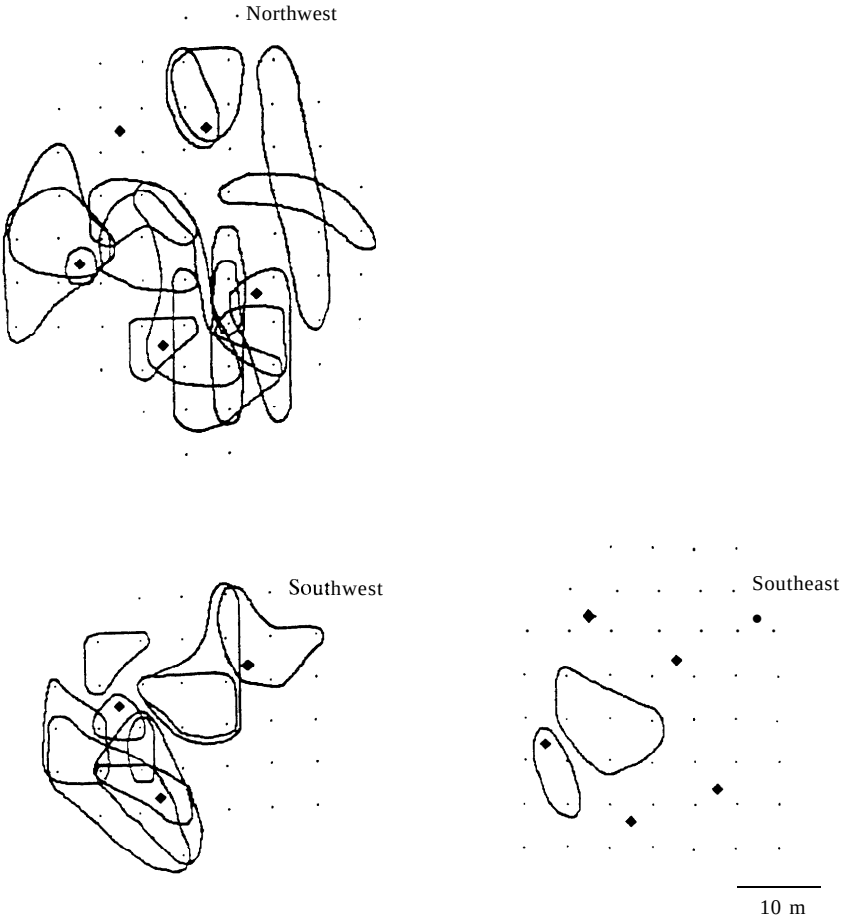

Figure 8. Home ranges of females on the three islands. Dots are trap sites and diamonds indicate locations of feeding stations.

in a significant disparity in the mass of androgen-sensitive preputial tissue.

Off-island captures

We captured 10 marked males (seven Low T and three High T) and three marked females during 180 off-island trap-nights. Only one of the males, a High T, and two of the females were recaptured more than once during the 3-day period. One High T male was released on the southwest island and by the first retrapping occasion was caught on the northwest island where he established a home range and persisted until the end of the experiment.

Feeding station use

The High T males were clearly over-represented in the feeding station capture pool. High T males were captured at feeding stations on 23 of 25 (90·5%) occasions, and of the 16 different individual males captured 14 were High T. The higher incidence of feeding station use by High T males was more equivalent across islands than the survival data presented above. The number of feeding station captures of High T males on the northwest, southwest and southeast islands were seven, eight and eight, whereas the number of Low T males was one, zero and one, respectively. When standardized by the total number of captures, High T males on the northwest island were caught at feeding stations seven times more often than Low T males, whereas on the rest of the island they were caught only 1·8 times as often. Pooling the data for all islands, High T males were caught at feeding stations 11·5 times more often than Low T males but only 1·6 times more often in non-feeding station traps.

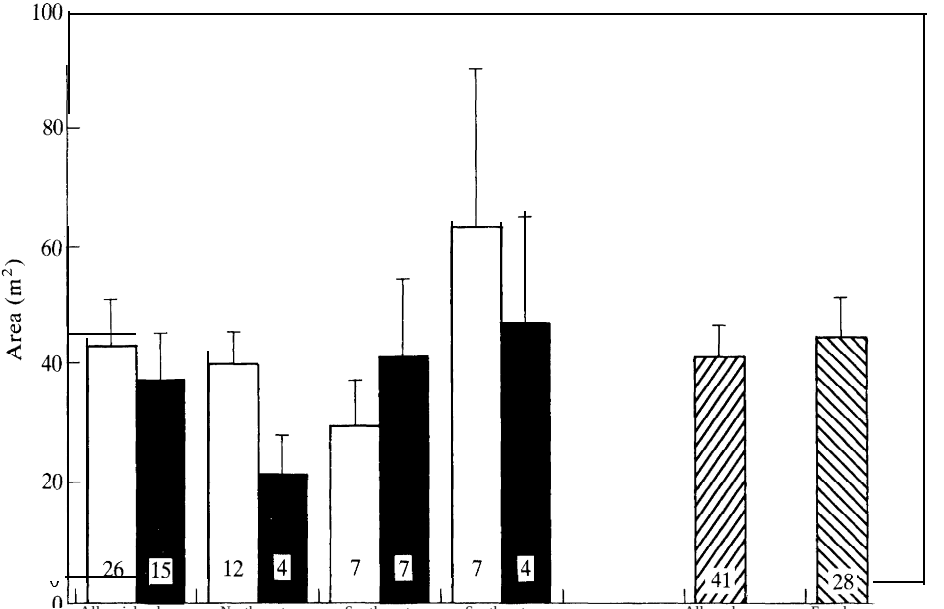

Figure 9. Mean (+ SE) sizes of areas used by High T males (□) and Low T males (■) for all islands and, for each island separately; and for all males (▨) and females (▧) for all islands pooled. No differences were statistically significant.

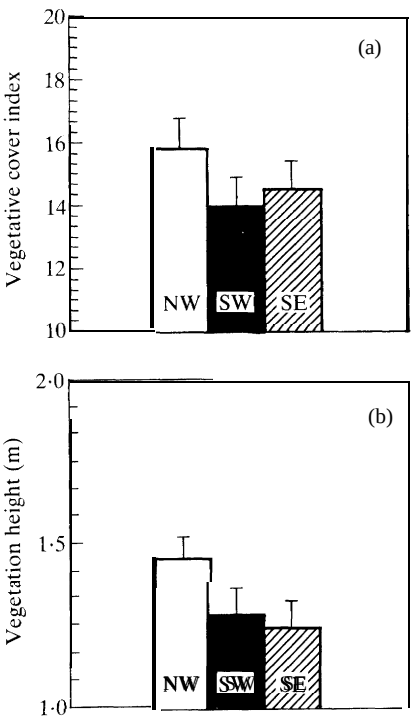

Figure 10. Mean (+ SE) cover index and height of vegetation for the three islands. There were no significant differences between islands.

During the course of the experiment, females were captured 44 of 186 (23·7%) times in traps at feeding stations. Thus, females had a considerably higher capture rate at feeding stations than did males, which were caught only 25 (8·6%) of 290 times at feeding stations.

Space use

On the northwest island the Low T males occupied predominantly the northeast corner of the island while the High T home ranges were fairly evenly distributed (Fig. 7). As a result, the High T males also overlapped more female home ranges. However, we caution that the temporal juxtaposition of the animals was unknown; we have summarized all the captures during the experiment without reference to whether particular males and females co-occurred. On the southwest island male home ranges appeared to occupy all available space and there was a curious segregation of Low T males on the northern half and High T males on the southern half of the island (Fig. 7). Female home ranges on this island occurred predominantly in the southwest and northeast quarters and therefore did not appear to overlap High T more than Low T male ranges (Fig. 8). High T male home ranges occurred over a larger portion of the southeast

island even though their survival on this island was previously shown to be no greater than that of Low T males. The ranges of High T males completely overlapped those of the two females captured frequently on this island (Figs 7, 8).

Male home range sizes did not differ among islands ($F = 1{\cdot}33$, $P = 0{\cdot}28$), nor did they vary on the basis of treatment ($F = 0{\cdot}38$, $P = 0{\cdot}54$, Fig. 9). Because there were relatively fewer Low T males caught on each island, and few of these were caught frequently enough to calculate a reasonable home range, it is possible that more representative sampling would have produced different results. But, on the basis of the data collected we have no reason to believe that High T males had larger home ranges. We also failed to demonstrate that males (both treatments pooled) had larger home ranges than did females ($F = 0{\cdot}07$, $P = 0{\cdot}79$, Fig. 9).

Vegetative cover

The vegetative cover index ($F = 1{\cdot}02$, $P = 0{\cdot}37$, Fig. 10a) and mean height of vegetation did not differ among islands ($F = 2{\cdot}56$, $P = 0{\cdot}09$, Fig. 10b). Our casual observations of vegetation status during the experiment, however, and the low *P*-value in the test of mean height suggest that the northwest island had considerably thicker vegetation. Unfortunately, the sampling occurred late in the season after, we believe, the pronounced differences noticed earlier had disappeared.

DISCUSSION

The findings reported here indicate that variation in male serum testosterone concentration can affect dominance in the laboratory and persistence in populations established in the wild. High T males dominated Low T males in laboratory trials with females present, and outcompeted Low T males on the northwest island. Pooling data from all three islands, High T males that survived to the first trapping period had greater subsequent longevities that Low T males. In addition, feeding station use supports the conclusion that High T males on all three islands occurred in the highest quality microhabitats. Other incidental data support the conclusion that High T males had a competitive advantage as well. First, the home ranges of High T males collectively occupied more of the northwest and southeast island areas than Low T males and thus, overlapped more of the home ranges of resident females. In fact, only High T male ranges overlapped female ranges on the southeast island. Second, although the sample is small, more than twice as many Low T as High T males were caught leaving the islands; a result that may indicate that Low T males received more harassment from High T males than vice versa. Taken together, the laboratory trials, the survival data, especially on the northwest island, the feeding station capture records on all islands, the home range overlap data and the emigration data all suggest that testosterone level can affect dominance and competitive ability in wild house mice and in natural environments.

The field results require some interpretation since treatment effects, using SURVIV, were significant on only the northwest island. However, we believe that mice on these islands probably did not survive long enough (i.e. were not recaptured in sufficient numbers) to determine whether a treatment effect occurred. For example, the southwest island was only 60% of the size of the other islands and in a previous experiment conducted at this interchange (Zielinski et al. 1992) mouse survival was also poorer on the southwest than the northwest island. Eighty mice were released on each island, largely to assure that competition among them would be keen, but also to maximize the potential for recapturing adequate numbers of males to identify a significant treatment effect if it were to occur. The southwest island may have been too small to support the population of mice long enough to test the hypothesis adequately. In addition, the density of vegetative cover on the southwest and southeast island appeared lower than on the northwest island. While we were not able statistically to confirm this conclusion by vegetation cover sampling, the lack of a significant difference was undoubtedly because the sampling occurred in late August when the cover on the southeast and southwest islands had 'caught up' with that on the northwest island. The southeast island was more similar to the northwest island in terms of vegetative cover and size than the southwest, but mouse survival, especially of females, was lower on the southeast island than on the other two. The reason for this difference is not apparent but on two occasions a feral cat was observed on this island. We believe that ideal habitat conditions, relatively large island size and perhaps fortuitous freedom from predation on the northwest island all may have contributed to the greater success of the

mouse population released there. This success allowed us to statistically verify the treatment effect.

About 90% of all male captures at feeding stations were of High T males. This greatly exceeded the number expected if males were captured on the basis of their relative availability. These sites provide shelter as well as food, and mouse burrow systems were frequently associated with feeding stations. We cannot assess the limitation of food and cover on the island, but we suspect that animals raised in the laboratory would be attracted to, and perhaps compete for space near food depots and cover. Southwick (1955) found that dominant male house mice can prevent subordinates from feeding freely. However, many more individuals appeared to include a feeding station in their home range than were caught at feeding station traps. Therefore, it is impossible to know whether these animals were excluded from the food and shelter or were merely not attracted to baited traps at a site where food was so readily available. Interestingly, females were caught more at feeding stations than either type of male. We expect that with their greater need for a reliable food source during pregnancy and lactation females may have valued the feeding sites more than males. In summary, we do not know why feeding station use differed between treatments and between sexes; we can only conclude that trapping data at feeding stations supports the hypothesis that elevated testosterone enhances competitive ability.

There was no difference in the home range sizes of Low T and High T males. Initially, we suspected that the High T males would have larger ranges based on the observation that most polygynous male rodents use larger areas than females (DeLong 1967; Harestad & Bunnell 1979; Gaulin & Fitzgerald 1988) and that this difference may be due to differences in circulating levels of androgens. In studies involving birds, testosterone treatment significantly increased the size of home range (Wingfield 1984). And, in an experiment with female mice, those individuals that occupied intrauterine positions between two males, and were presumably exposed to the highest androgen levels, had larger home ranges than those that occupied a position between two females (Zielinski et al. 1992). Lacking information on spatial variation in habitat quality it is difficult to determine whether the High T males may have been occupying higher quality, albeit similar sized, home ranges. Several additional reasons could account for the lack of relationship between testosterone level and home range size in the present study. First, a relationship between home range size and basal testosterone levels in males may not exist. Second, it is possible that ranging behaviour, like other sexually dimorphic behaviour patterns, may be 'organized' by perinatal exposure to androgens (Phoenix et al. 1959; Williams et al. 1990) and circulating levels of testosterone in adulthood may have little influence. Third, in a previous study conducted on the same islands (Zielinski et al. 1992) there was a significant effect of season on male, but not female, home range size. Male home ranges were twice as large in autumn as they were in spring. Because the present study was conducted in the spring, perhaps the potential for testosterone to influence space use was less than it would be in the autumn. The fact that male and female home ranges were no different supports this conclusion. Finally, with only five or six locations, on average, used to construct home ranges and with a small sample of Low T males available for comparison it is possible that there were insufficient data to detect a difference. Telemetry studies may be necessary to answer the question with certainty.

Our field results suggest the following scenario. Immediately after release, the interactions among male and female mice were frequent and intense. Intra-sexual tolerance was probably greater for females and thus fewer females probably left the islands than males. This is supported by the fact that only one-third of the emigrants captured off the islands were females. Presumably, High T males, especially on the northwest island, were better able to defend their areas than Low T males and the latter began to die in situ or emigrated from the island. Previous work in enclosures has determined that aggressive, territory-holding males will drive non-territorial males from the area (Lidicker 1976; Berry 1981). High T males may have discovered feeding stations first, perhaps as a result of greater activity levels (Lloyd 1973), and may have defended territories that included these sites. If this scenario occurred, the advantages gained by High T males in our experiment may also accrue to intact males with basal testosterone levels in the higher physiological range. Thus, we can imagine that they could achieve higher fitness than males with lower basal levels.

The results of paired encounters in the laboratory, at first, were difficult to reconcile with results from

the field and with previous studies. When males were paired in the absence of females, there was no difference in dominance status on the basis of treatment. Realizing that males in the field were exposed to females and their odours but those tested in the laboratory were not, we redesigned the laboratory test to include a female and found that their presence was necessary to demonstrate a significant domination of High T males over Low T males. This is not surprising given that previous research on aggression in rodents established that the presence of females (Flannelly & Lore 1977; Sachser & Lick 1991) and food limitation (Zook & Adams 1975) both increase the level of inter-male aggression. In fact, these are the selection pressures that probably favoured the evolution of heightened aggression in males.

Interestingly, body weight was not as important a determinant of dominance as has been demonstrated for other species (Barkley & Goldman 1977). High T males easily overcame weight disadvantages, especially in the presence of females, to dominate the Low T males. In some cases the weight disparity amounted to one-quarter to one-third of the High T male's own body weight. Testosterone differences would be expected to influence the threshold for aggressive behaviour and to affect motivation. However, body weight should not influence motivation but only affect the outcome of aggression in progress. Thus, our data support the view that motivational differences explain the outcome of dyadic encounters more than somatic differences.

A question addressed by our work, and one that is fundamental to research on androgens and dominance is: do inherent differences in basal testosterone levels dictate social outcomes or do social interactions alter testosterone levels, or do both occur? Most studies conducted on this topic indicate that endocrine profiles change as a result of inter-male interactions and that differences in circulating androgens prior to the interactions are not as important (Mazur 1976; Dixson 1980; Bernstein et al. 1983). Running averages of androgen levels for males prior to and after realistic social contests are necessary to adequately test the hypothesis. Also, the pre-contest experiences with the same and opposite sex must be equivalent for test animals. Few studies have been conducted in a manner necessary to address these concerns. Bernstein et al. (1983) suggest that for rhesus macaques, *Macaca mulatta,* recent social experiences influence androgen levels more than pre-interaction androgen levels predict social dominance. However, they acknowledge that hormone manipulations independent of an individual's social experience will influence the tendency to engage in aggressive interactions. In olive baboons, *Papio anobis,* testosterone levels were not related to status except during a period of social chaos (Sapolsky 1983). During equilibrium periods, dominant animals were distinguished by physiological mechanisms that prevented a stress-induced depression of circulating testosterone but had no higher basal testosterone levels than subordinates. Shuurman (1980) found that while baseline testosterone levels in rats predict the level of aggression in paired encounters, testosterone levels also change as a result of aggressive encounters; losers exhibit a significant decrease, whereas victors show no change. And finally, in hamsters, the size of the androgen-dependent flank gland is positively related to the position in a linear dominance hierarchy a male ultimately attains (Drickamer et al. 1973). The data are too few to conclude that aggression and dominance are mediated less by hormones in the more socially complex primates than in rodents, but this possibility has been suggested (Dixson 1980).

The alternative to monitoring the fluctuating basal androgen levels in intact males is the kind of experiment we have presented here. By administering two different doses of a long-acting form of testosterone to castrates we avoid the difficulty of characterizing mean levels from a small sample of intact, free-living males whose testosterone profiles are changing spontaneously. We assume that the pulsatile nature of testosterone release and its circadian fluctuations are secondary in importance to the mean circulating level. Because they are castrated, we presume that these animals cannot respond to social stimuli with an increase or decrease in gonadal testosterone, and that androgens from the adrenal have minimal effect on aggression (Burge & Edwards 1971). So we can exclude the possibility that dominance is occurring due to socially mediated changes in testosterone levels. Lacking the ability to exhibit a gonadal response we can assume that High T males simulate either, (1) the condition in dominant males after a victorious encounter, or (2) the high end of natural variation in basal serum testosterone levels. The fact that we have shown a significant effect of tonic testosterone on dominance in the laboratory and a strong indication of this in the

field suggests that differences in mean testosterone levels can influence the outcome of aggressive encounters independent of the changes that can occur as a result of agonistic encounters.

This conclusion supports observations about the relationship between hormones, behaviour and mating systems made recently by Wingfield et al. (1990). They suggest that testosterone levels may be less responsive to the immediate social environment and more genetically determined in males of polygynous than monogamous species. Their hypothesis is supported by comparative data from birds, but was presented as applicable to all vertebrate classes. Thus, if house mice are polygynous or promiscuous and breed throughout most of the year, as several studies suggest (DeFries & McClearn 1970; Wolff 1985; Potts et al. 1991) Wingfield's hypothesis indicates that testosterone in males may be largely genetically determined and that there has been selection for maximum testosterone levels. Testosterone in these males would be much less responsive to immediate social conditions than in males of more monogamous mammals. If this is the case, males with the highest testosterone levels should have advantages in competition for space, food and females compared with males having lower testosterone levels; a conclusion consistent with the results of the present study. Recently, Dewsbury (1990) suggested that the propensity to 'dominate' in dyadic encounters has a genetic component. If this propensity is manifest through selection on testosterone levels, or receptor sensitivity, it supports the theory that the basal testosterone level, as much as the surge in response to particular conflicts (Batty 1978; Bronson & Desjardins 1982) can influence competitive ability. It is likely that basal testosterone levels, like any quantitative genetic trait will vary among individuals. This variation should be subject to selection. Our experiment, then, can be broadly interpreted as supporting the conclusion that males with higher basal levels of testosterone, up to a point, will win aggressive encounters against males that have lower testosterone levels. Whether this translates to increased fitness cannot be addressed in hormone-replacement experiments such as ours. However, it suggests that future work should focus on the problem of carefully measuring pre-interaction hormone levels and relating these to the outcome of aggressive interactions in laboratory and field studies.

The work presented here on males can be compared with a similar experiment on the same highway islands where we tested the ability of testosterone treatment to alter competitive ability in females (Zielinski & Vandenbergh 1991). Plasma levels of testosterone in females are only about 510% of those of males. However, the neural system modulating hormone-dependent aggression, at least in female rats, has about the same sensitivity to serum testosterone as that of males (van de Poll et al. 1981; Albert et al. 1989). And, the ability of testosterone to masculinize female behaviour is not limited to the perinatal period, but can affect aggression even when administered in adulthood (Whitsett et al. 1972; Svare et al. 1974; Zielinski & Vandenbergh 1991). In our previous study, females treated with a single 500 μg dose of testosterone enanthate won significantly more dominance trials in the laboratory and survived high-density field conditions better than oil-treated control females. An oil-treated female never won a laboratory dominance trial. Moreover, the treatment in the female experiment had a greater effect on differences in field survival than the treatment in the male experiment, with T-treated females exhibiting significantly greater survival on all three islands. Perhaps, by producing male levels of basal testosterone in females, an obvious pharmacological treatment, we caused stronger effects in females than did the physiological doses administered to males. Testosterone is aromatized to oestradiol in males and females and oestradiol can increase aggression in females, but it is unlikely that differences in aromatization could account for difference in response to testosterone treatment since oestradiol is less effective in inducing aggression than testosterone in females (Edwards & Herndon 1970; Gray et al. 1978; Simon & Gandleman 1978).

The effect of androgens on fitness in males has been frequently discussed. Sexual selection, especially in polygynous species of rodents, has apparently favoured the evolution of androgen-dependent characteristics such as large body size, heightened aggression and large home range size. However, females also respond to testosterone in a manner similar to males. This may be an artefact; either as an indirect consequence of selection on males or resulting from the similar responsiveness of common tissues that influence secondary sexual characteristics in both sexes. On the other hand, basal testosterone levels in females, despite being lower in concentration compared with males, may

be the result of direct selection, based on their value in intra- and inter-sexual competition. It is possible that low testosterone levels in females could be effective in regulating aggression if neural substrates in females are more sensitive to androgens than in males, as has been suggested in other species (Frank et al. 1985). The results of our work on the hormonal basis of competitive ability in males and females cannot address these issues. However, it does indicate that testosterone, acting alone, via its metabolites or via aromatization to oestradiol, can influence dominance behaviour in free-living populations of male and female house mice.

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