Aggression, testosterone, and the spring decline in populations of the vole *Microtus townsendii*

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Many vole populations decline sharply in size at the start of the spring breeding season. We attempted to test the hypothesis that aggressiveness controls the magnitude of the spring decline in *Microtus townsendii* by implanting male voles with testosterone pellets in January 1975. The spring decline was very slight on the control grid and nearly absent on the experimental area, the opposite effect to what we predicted. The amount of skin wounding increased at the same rate on both experimental and control areas as voles entered breeding condition. Aggression tests in a neutral arena showed no differences in aggressive behavior scores for testosterone-implanted males compared with control males. In a more carefully controlled laboratory test we were unable to alter aggressive behavior by either of two levels of testosterone injections. We concluded that aggressiveness in *Microtus townsendii* could not be altered by testosterone treatments on intact males. Our field test of the aggression hypothesis thus failed.

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Plusieurs populations de campagnols subissent une diminution importante de leur effectif au début de la saison de reproduction du printemps. On a tenté de vérifier l'hypothèse selon laquelle l'agressivité est responsable de l'amplitude de cette diminution; des boulettes de testostérone ont donc été greffées à des mâles de *Microtus townsendii*, en janvier 1975. La diminution d'effectif a été très faible chez la population témoin et négligeable dans l'aire expérimentale; l'effet a été contraire aux prédictions. Après le début de la saison de reproduction, les blessures à la peau se sont mises à augmenter au même rythme dans les deux aires étudiées. L'analyse de l'agression en territoire neutre a révélé qu'il n'y avait pas de différences de comportement agressif entre les mâles traités à la testostérone et les mâles témoins. On a essayé, en laboratoire, de modifier ce comportement agressif des campagnols, par injection de deux doses différentes de testostérone; l'expérience n'a donné aucun résultat. Il faut donc conclure que, chez *Microtus townsendii*, le comportement agressif ne peut être modifié par administration de testostérone à des mâles intacts. L'expérience sur le terrain n'a donc pas réussi à vérifier l'hypothèse.

[Traduit par le journal]

Populations of many species of voles, mice, and lemmings may undergo sudden declines in numbers in the spring at the start of the annual breeding season (Sadleir 1965; Chitty and Phipps 1966; Pitelka 1958; Watts 1969; Flowerdew 1972). The timing of this sudden drop suggests that, at least in males, it is a result of socially induced mortality or dispersal and is caused by aggressive behavior among the breeding animals (Fairbairn 1976). In vole populations there is considerable variation in the rate at which numbers fall and in the time period of the spring decline. We hypothesize that the intensity and duration of the spring decline are direct functions of the level of aggressiveness in the individuals comprising the breeding population. Aggressiveness is widely believed to be partly under the control of testosterone in male rodents (Beeman 1947; Johnson 1972; Davidson and Levine 1972). In this experiment we implanted male voles (*Microtus townsendii*) with testosterone pellets in the hope of increasing their aggressiveness and thereby triggering a strong population decline. The experiment failed and we now examine why.

Methods

The study was carried out in grasslands at the Ladner Airbase at Boundary Bay, B.C., 16 km south of Vancouver, from January 13 to May 22, 1975. Two live-trapping areas were used. The control area, grid E, has been trapped as a control population since 1971 (cf. Krebs *et al.* 1976); it contains 100 trap sites spaced in a checkerboard 7.6 m (25 ft) apart. The experimental area, grid U, had been used by Redfield *et al.* (in preparation) for a previous experiment which ended 7 months before the present experiment began. Grid U was a square checkerboard of 100 trap points set at 7.6-m intervals.

These areas were trapped with 100 Longworth live traps for 2 days and nights every 2nd week. Longworth traps were left in position and locked open between the trapping periods.

Each individual *Microtus* was ear-tagged upon first capture and its weight, sex, wounding on rump, relative size of hip glands, and breeding condition noted. Voles were usually released immediately after processing, except for individuals that were removed for 2 days so that aggression tests could be run in the laboratory.

Population parameters were determined by direct enumeration. Trappability was very high on both trapping areas, always exceeding 83%, and hence we feel our population data are accurate (Hilborn *et al.* 1976).

Voles to be implanted with testosterone pellets were brought into the laboratory for this operation. Implantation was accomplished by placing the animals under light ether anaesthesia, making a small incision in the dorsal skin above the shoulders, and loosening the skin with the blunt tip of a pair of forceps. One 20- to 25-mg pellet of testosterone proprionate was then placed under this fold of skin and the incision was closed with a wound clip. Control animals were also implanted in the same manner with a 20- to 25-mg pellet of cholesterol. Two out of three males on the experimental grid were implanted with testosterone and the same fraction of males on the control grid were implanted with cholesterol. One in three males was left intact on both areas so we had a set of internal controls for each grid.

Aggressive behavior tests were carried out in much the same way that Krebs (1970) and Turner and Iverson (1973) did. Two voles of about equal size were matched in a neutral arena for 10 min, and the following behavioral data recorded: (1) time to first contact; (2) number of approaches (as defined by Krebs (1970)), (3) boxing (two voles stand on their rear legs and appear to box with their forelegs); (4) wrestling and chasing (strong aggressive interactions involving biting and wounding); (5) pounces (a mixed category of weak aggressive moves in which a vole springs toward his opponent and may or may not make contact); (6) vocalizations (the number of squeals); (7) uprights (a vole stands on his hind legs when the other vole approaches); (8) avoidance (as defined by Krebs (1970)); (9) submission (as defined by Krebs (1970)); (10) groom other (a weakly developed behavior pattern in this species which involves sniffing and pawing the fur on the back of the opponent); (11) groom self (the vole wipes his head with his forepaws and licks at his fur); (12) activity counts (the number of 10 × 10 cm squares that a vole moves through during its 10-min bout. The unit of observation was an *approach* and these behaviors were tallied on this basis as much as possible. When continuous events such as vocalizing occurred, we adopted the 10-s rule and tallied one new event every 10 s.

All of us did some behavior tests. We worked together for the first 4 weeks to reduce variation in scoring procedures.

We attempted to do aggressiveness tests on every male in the control and experimental populations, and to test as many as possible twice during the experiment. Because of the amount of work this entailed we were unable to test females for aggressiveness during the experiment.

Results

Density Changes

Changes in population density for males and females are shown in Fig. 1. Two points should be noted. First, the density of males on the testosterone grid U and control grid E were nearly equal, but females on the testosterone grid were only two-thirds the density of females on the control grid. Second, the spring decline was very slight on the control area and virtually absent on the testosterone area, exactly the opposite effect to the one we had hoped to produce. Males declined slightly (10%) on the control grid from March 10 to April 8 and declined very slightly (4%) on the testosterone grid from March 25 to April 8. Females declined moderately (16%) on the control grid from March 10 to April 23 and did not decline at all on the testosterone grid.

Breeding Season

A few voles were breeding on the control area

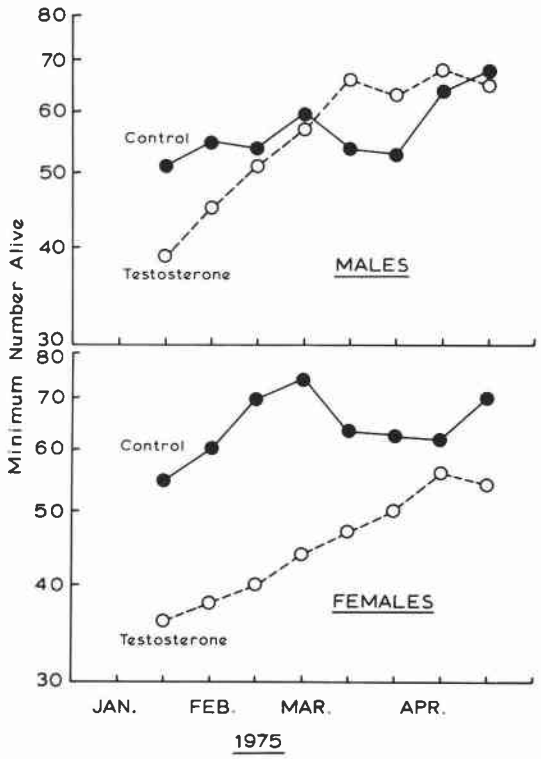

FIG. 1. Population density changes for *Microtus townsendii* on the control area and on the experimental area in which males were implanted with testosterone pellets.

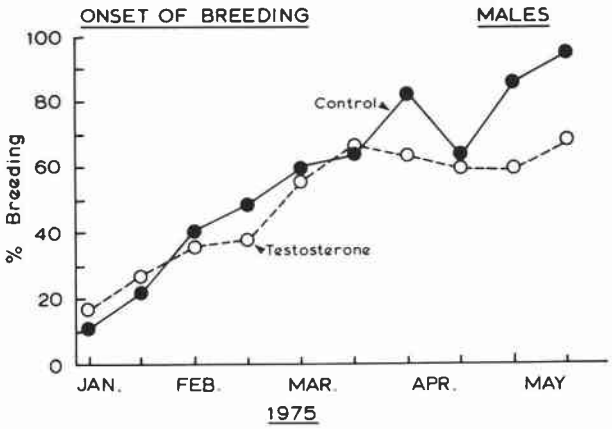

FIG. 2. Onset of breeding for male voles in the testosterone and control populations.

throughout the winter of 1974–1975, and hence the onset of summer breeding was not as dramatic as it could have been. Figure 2 shows the onset of breeding in males. By the end of February more than half of the males on both areas were in breeding condition and by mid-March one in three adult females was lactating (21-day gestation period). Testosterone pellets were implanted in experimental males beginning in January, and we could detect no difference in the onset of breeding between the experimental and control populations. During April and May the percentage of males breeding was reduced on the testosterone grid, possibly because exogenous testosterone caused gonadal recrudescence in the implanted males. This reduction in male fertility did not affect the lactation rate in April and May on the testosterone grid.

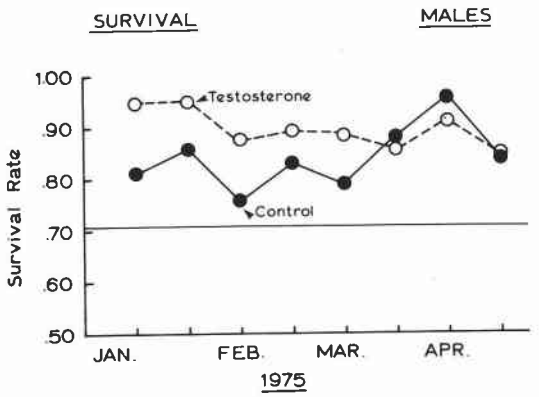

FIG. 3. Minimum survival rates for male voles on the control and experimental areas. The horizontal line at a survival rate of 0.71 divides the graph into zones of 'good' survival and 'poor' survival. Survival was good throughout this experiment in both populations.

Survival and Recruitment

Survival rates of males were better on the experimental area than on the control until April (Fig. 3). But at no point during this experiment was survival poor, and the differences shown in Fig. 3 are between good survival and excellent survival. Survival rates of females were excellent on both grids throughout this experiment. The average survival rate per 2 weeks of females on the control area was 88%, on the experimental area 89%. Corresponding averages for males were as follows: control, 84%; experimental, 89%.

Recruitment rates were similar on the two areas. From mid-February until the end of May, 145 voles were newly tagged on the control grid and 130 voles on the testosterone grid. More juvenile voles (<30 g) were recruited to the control population than to the experimental but most of this recruitment occurred near the start of this study and was presumably a result of winter breeding.

Hip Glands and Wounding

Microtus townsendii has highly developed hip glands (Quay 1968), and the size of these glands is testosterone dependent in males (MacIsaac 1975). We scored the size of these glands in field animals from 1 to 4, and Fig. 4 shows the changes in these glands during the experiment. The response of males on the testosterone grid to implants of testosterone was particularly striking. By early February hip glands on the testosterone grid became larger than those on the control and

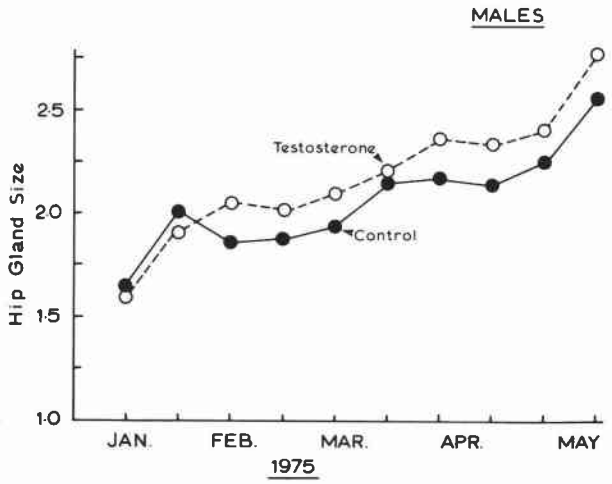

FIG. 4. Average size of hip glands in male voles from the control and experimental populations. Males were implanted with testosterone in late January.

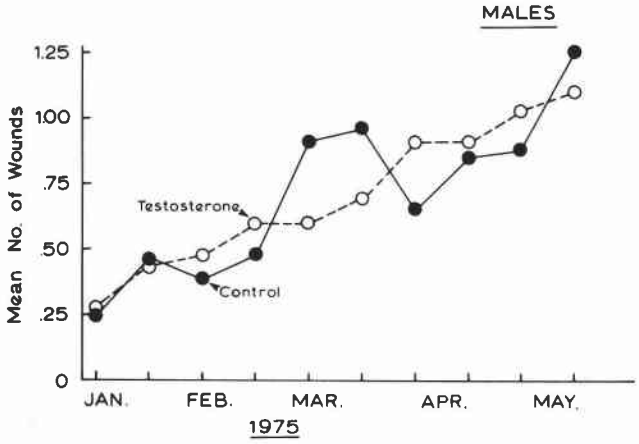

FIG. 5. Average number of wounds on the rumps of male voles of the control and experimental populations.

this difference was maintained until the end of the experiment. We interpret these differences to be due to testosterone treatment. By contrast, females on the experimental grid consistently had smaller hip glands than females on the control area until the end of April when the two areas became similar.

Teeth marks on the rump can be readily seen after voles fight vigorously, and we counted the number of wounds of this type on all field animals. Females always have few wounds compared with males. During this entire experiment 40% of males handled had wounds while only 5% of females were wounded. Figure 5 shows the mean number of wounds in males from the two populations. There was no indication that wounding was more severe on the testosterone grid; and, except for an episode of more intensive wounding on the control grid during March, there is a smooth trend toward more wounding in both populations as the fraction of breeding animals increases (cf. Fig. 2). During the experiment 40% of the males on the control area had wounds and 39% of the males on the experimental had wounds. For females the corresponding percentages were 6% for the control and 5% for the experimental.

Thus we have field evidence of higher testos-

TABLE 1. Means and standard deviations (SD) for transformed behavior variables for male *Microtus townsendii* before and after the start of breeding. All variables were transformed by log $(x+1)$. Grid U is the testosterone grid. Standard deviation is the weighted mean standard deviation for the three groups

	Before February 15				After February 15			
		Grid U				Grid U		
Variable	Grid E controls	Normal	Implanted	SD	Grid E controls	Normal	Implanted	SD
Time to first contact	1.27	1.36	1.24	0.71	1.40	1.47	1.53	0.70
No. of approaches	0.69	0.65	0.65	0.37	0.61	0.55	0.59	0.40
Boxing	0.19	0.05	0.15	0.25	0.21	0.17	0.16	0.28
Wrestling and chasing	0.08	0.06	0.06	0.18	0.09	0.07	0.11	0.23
Pounces	0.49	0.50	0.54	0.39	0.46	0.54	0.47	0.40
Vocalizations	0.66	0.70	0.61	0.47	0.66	0.60	0.55	0.50
Uprights	0.36	0.24	0.34	0.35	0.46	0.33	0.36	0.40
Avoidance	0.02	0.04	0.02	0.11	0.10	0.05	0.06	0.19
Submission	0.04	0.04	0.01	0.10	0.02	0.02	0.00	0.08
Groom other	0.06	0.09	0.07	0.20	0.02	0.03	0.02	0.11
Groom self	0.84	0.81	0.78	0.35	0.72	0.74	0.75	0.42
Activity count	1.51	1.51	1.51	0.53	1.45	1.35	1.38	0.57
Sample size	89	34	60		107	68	128	

terone levels in our experimental males but this effect was not translated into higher amounts of wounding in either males or females.

Aggressive Behavior

We tested the aggressive behavior of all voles before they were subjected to any experimental treatment. Table 1 gives the mean scores of the 12 behavior variables for the 'before-treatment' groups (before February 15). We subjected these data to multiple discriminant analysis (BMD: 07M) to test the null hypothesis that the mean of all the behavior variables was the same in all three groups. We accepted this null hypothesis and concluded that the behavior of the males before experimental treatment was the same in all three groups.

If testosterone implants increase aggressive behavior, we expected this to show up after February 15 in the implanted males. We tested for this effect with multiple discriminant analysis and found no significant difference between groups after experimental treatment. Table 1 gives the mean values for the aggressive variables. We also analyzed these data one variable at a time by analysis of variance. Again we could find no significant differences between implanted males and controls.

We concluded from this analysis that under the conditions of our experiments, aggressive behavior as measured in neutral-arena interactions is not affected by testosterone implants in adult *Microtus townsendii*.

Laboratory Experiment on Aggression and Testosterone Levels

We recognized the weakness of our field experiment and attempted to check our conclusions with a more carefully controlled laboratory experiment. We obtained 36 male *Microtus townsendii* from the field in January 1976 and held them for 4 weeks in the laboratory. The animals were housed in separate cages and kept on a 12 h light (L): 12 h dark (D) light cycle. We tested their aggressive behavior in the same way described above with the only exception that we split the *pounce* category into two types of pounces: an approach pounce and a defense pounce. We divided the males into three groups at random and tested them before treatment on February 9 and 10, 1976. We then injected them intraperitoneally as follows every other day: group I, 0.2 cm^3 of sesame seed oil; group II, 0.5 mg testosterone proprionate in 0.2 cm^3 of sesame seed oil; group III, 1.0 mg testosterone proprionate in 0.2 cm^3 of sesame seed oil. We tested them again after seven injections on February 23 and 24.

Table 2 gives the means for the different treatment groups. We tested group differences by means of multivariate analysis of variance techniques, and found no significant differences

TABLE 2. Means and standard deviations (SD) of transformed behavior variables for male *Microtus townsendii* used in the laboratory experiment. Some behavior variables had to be deleted because of zero frequency. All variables were transformed by log $(x+1)$. Standard deviation is the weighted mean standard deviation for the three treatment groups

	Before treatment				After treatment			
		Testosterone dose				Testosterone dose		
Variable	Controls	Low	High	SD	Controls	Low	High	SD
Time to first contact	1.39	1.78	1.62	0.71	1.30	1.62	1.45	0.64
No. of approaches	0.65	0.47	0.65	0.36	0.56	0.50	0.69	0.36
Boxing	0.15	0.01	0.06	0.19	0.10	0.06	0.04	0.14
Pounces								
Defensive	0.36	0.19	0.34	0.34	0.27	0.15	0.30	0.31
Offensive	0.23	0.04	0.11	0.24	0.07	0.05	0.16	0.24
Vocalizations	0.50	0.28	0.49	0.39	0.35	0.29	0.57	0.36
Uprights	0.26	0.16	0.26	0.31	0.23	0.17	0.27	0.27
Avoidance	0.10	0.12	0.16	0.24	0.14	0.08	0.13	0.19
Groom self	0.93	0.91	0.94	0.36	1.05	0.96	0.90	0.34
Activity count	1.52	1.21	1.64	0.59	1.48	1.32	1.55	0.60
Sample size	24	24	24		24	24	24	

among the three groups before or after treatment. In particular we analyzed the after-treatment data to look for increases in the behavioral variables indicating aggressiveness. No single variable was significantly different among groups except vocalizations, which were increased in the high-dosage testosterone group and reduced in the low-dosage testosterone group. Neither boxing nor pounces were increased in testosterone-treated males. We concluded that under our laboratory conditions the aggressive behavior of intact male voles could not be changed by testosterone injections.

Discussion

The hypothesis that population density in rodents can be determined by agonistic behavior has been suggested for almost 30 years (Calhoun 1949). Since 1950 a large number of studies have described demographic events in field populations so that at the present time we have a reasonably good picture of the way in which density changes in natural populations (French *et al.* 1975; Krebs and Myers 1974). Unfortunately there has not been a parallel development of work on social behavior in field populations of rodents. Most of the work on social behavior in rodents has been involved with laboratory populations and with the physiological machinery associated with the pituitary–adrenal axis (Lloyd 1975; Christian 1975). An unknown amount of this laboratory work may be applied to field populations, and we need a new generation of field experiments to clarify the role of social behavior in population processes.

One way to investigate the role of social behavior is to alter the behavioral characteristics of individuals with hormones or other chemical agents. The present experiment was a small attempt in this direction with *Microtus*. Almost no one has carried out hormonal manipulation experiments on field populations. Trobec and Oring (1972) reported an unsuccessful attempt to alter territorial arrangements in sharp-tailed grouse by testosterone implants. Watson (1970) reported that red grouse implanted with testosterone increased their territory size, became more aggressive, and survived longer than control birds.

The negative results obtained in this experiment may have been partly due to certain aspects of our experimental design. Most studies on the effects of testosterone on aggression have used castrated males and have reported a reduction in aggressive behavior after castration, with a restoration to preoperative normal levels after replacement therapy with testosterone. Suchowsky *et al.* (1969) found that while testosterone injections did not increase aggression in intact (non-castrated) laboratory mice, they did do so in castrated animals. Since the primary object of

our experiment was to find a method for increasing aggression in wild populations of mice, castration was not considered feasible and all our studies were conducted with intact animals. Thus, it is possible that the presence of the testes in our experimental animals may have prevented the exogenous testosterone from exerting any marked or additional effect on normal levels of aggressive behaviors.

The length of the photoperiod is another important variable in hormone–behavior studies (Hinde and Steel 1975). In the case of testosterone, several studies have demonstrated the critical nature of photoperiod length on behavioral effects. Studies on nest-building in fish (Hoar 1962) and in ring-doves (McDonald, personal communication) have shown that testosterone is more effective in animals kept under a long photoperiod (16 L: 8 D) than in animals kept under a shorter day length (8 L: 16 D). Likewise, Morin *et al.* (1976) have found that testosterone does not increase copulatory behaviors in castrated golden hamsters kept under short photoperiods. Interestingly, it appears that this effect of short photoperiods is restricted to behavioral responses and does not affect the ability of testosterone to stimulate accessory gland development. In our laboratory experiments, the animals were kept on a 12 L: 12 D light cycle and it is possible that in *M. townsendii* a longer photoperiod is necessary before testosterone can exert its maximum effect on aggressive behavior.

It should also be noted that little is known about the causes or kinds of aggression in wild species of rodents and it is likely that there are differences between laboratory rodents and wild species. Thus, while testosterone has been shown to increase aggression during dyadic encounters in a variety of laboratory rodents, there is, as yet, no direct evidence that it has the same effect on wild species of voles. In particular there is no evidence that aggression is proportional to testosterone levels in intact animals of any rodent species. We will have to search elsewhere to find ways of increasing aggression in *Microtus* in field populations.

A large variety of chemical compounds of diverse structure have been shown to reduce aggression in laboratory mice (Kletzkin 1969). One profitable line of future experiments would be to reduce aggression in a field population and observe its demographic consequences. Vessey (1967) did this experiment with a tranquilizer (chlorpromazine) on laboratory populations of house mice, and produced density increases associated with the reduced aggression. This kind of experiment ought to be conducted on a field population of voles.

Spring declines in *Microtus townsendii* vary from very slight declines, such as the one observed in this study, to extremely severe drops in which two-thirds of the population may disappear in a month or two. What variables are needed to predict the severity of the spring decline? If social behavior is a necessary component, we should try a variety of experiments manipulating individuals. The present experiment shows that aggressive behavior is not sufficient to produce a severe spring decline, and some additional factors must be necessary. Population density at the time of the anticipated spring decline was moderate, yet social strife, as indicated by increases in wounding, was not sufficient to produce dispersal or mortality among either sex.

Acknowledgments

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