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A TEST OF THE CHITTY HYPOTHESIS: INHERITANCE OF LIFE-HISTORY TRAITS IN MEADOW VOLES *MICROTUS PENNSYLVANICUS*

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Abstract.—The Chitty hypothesis proposes that the demographic changes occurring in microtine cycles are mediated by natural selection operating on the genetic composition of the population. Implicit in this hypothesis is the assumption that a suite of life-history traits is simultaneously undergoing selection and that these traits are strongly heritable. We tested this in two ways: first, by determining whether the year-to-year differences in phenotypes in fluctuating meadow vole populations in the field are maintained in samples of young animals raised in the laboratory, and second, whether the variation seen in the field has a heritable basis as determined by half-sib analysis. Parents were obtained in the springs of successive years from a fluctuating meadow vole population. These animals were bred in small field enclosures, and their progeny were raised in the laboratory. Animals raised in the laboratory differed significantly from those in the natural field population. In the field, young from the year when population size was increasing grew more rapidly than those from the peak year; in the laboratory, the opposite occurred. The ages at sexual maturity showed similar differences. Heritability analysis was performed on body weight, growth rate, and age and weight at sexual maturity. Virtually all these traits showed significant dam effects, but small or nonexistent sire effects. Thus, most of the variation was nongenetic in origin; maternal and other environmental effects were of overriding importance. We conclude that the heritabilities of these traits in nature are usually lower than necessary for natural selection to operate in the time frames characteristic of microtine cycles.

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In spite of the fundamental importance of genetic variation to evolutionary change and the key roles of body size, age at sexual maturity, and fecundity in determining the fitness of an organism, almost nothing is known in natural populations of mammals of the genetic variation of complex traits which together form a life history (Stearns, 1977). Many of these phenotypic traits display continuous variation and presumably have a polygenic basis. In the last decade, polygenic models of variation in life-history

traits have become more widely known (Dingle and Hegmann, 1982; Lande, 1982). Classical life-history questions such as the "cost of reproduction" have increasingly been reframed in terms of genetic variance and covariance, e.g., the theory of "antagonistic pleiotropy" (Rose, 1982). Application of this new approach to the study of life histories has been rapid, and examples of studies employing this approach are now found in wild taxa ranging from plants (Antlfinger et al., 1985), insects (Hegmann

and Dingle, 1982), amphibia (Berven and Gill, 1983), fish (Stearns, 1984), and birds (Findlay and Cooke, 1983; Boag and van Noordwijk, 1987).

Many, but not all, lemming and vole populations fluctuate with a periodicity of 2–5 years (Krebs and Myers, 1974; Taitt and Krebs, 1985). Population fluctuations are characterized by a relatively repeatable pattern of changes in body weight (Boonstra and Krebs, 1979), body growth, age at sexual maturity, and length of the breeding season (Krebs and Myers, 1974; Krebs, 1978). Unable to account for these changes by standard extrinsic explanations (predation, disease, or lack of food), Chitty (1967) proposed a genetic-behavioral hypothesis. During low and increasing densities, mutual interference is minimal and nonaggressive genotypes with a high reproductive effort are selected. At high population densities, mutual interference is intense, and aggressive genotypes are selected for their ability to withstand intraspecific competition. Implicit in Chitty's Hypothesis is the assumption that the many changes that occur with density have a genetic basis and that these changes are correlated with changes occurring in spacing behavior.

The results of approaches used to test the Chitty Hypothesis have been inconclusive. Electromorph-frequency changes associated with density fluctuations have been found in numerous studies and were initially regarded as evidence consistent with the hypothesis. However, in a review of the literature, Gaines (1985) concludes that these changes are likely to be an effect, not a cause, of density changes. To conclude that changes in spacing behavior and life-history characters are a result of genetic changes, it must be shown: 1) that variation in the traits involved has a genetic basis, and 2) that natural selection acts on this variation during the course of population fluctuations in the field.

There are several approaches to these questions, including explants (removal of individuals from field populations to a standardized environment), transplants, experimental crosses, artificial selection, and pedigree-based heritability studies to establish the genetic basis for phenotypic variation in a population (Falconer, 1981). The dem-

onstration of selection is difficult and ideally requires longitudinal studies of known individuals during an entire population cycle (Endler, 1986). Only Anderson (1975; see Krebs [1979] for a published summary of Anderson's work) has used a heritability approach with noncyclic populations of *Microtus townsendii* to tackle the first problem. She found no evidence of heritable variation for adult body size, winter breeding activity, or adult aggressive behavior. Juvenile growth rate, age at sexual maturity, and dispersal tendency appeared to be strongly heritable, although the full-sib experimental design made it impossible to eliminate nongenetic causes of high heritability, such as maternal effects. A longitudinal study of selection is particularly difficult for small mammals, because their rapid turnover and secretive nature makes it difficult to follow marked individuals from birth to death, relative to studies involving larger mammals such as deer (Albon et al., 1987).

In our study, we evaluate the plausibility of the Chitty Hypothesis by testing for a genetic component to variation in several microtine life-history traits. Ideally, such a study should include behavioral characters, as these are particularly important in the hypothesis. However, as we discuss later, this proved difficult, and hence we emphasize more easily measured characters such as body size and growth rate. We carried out an explant experiment and also measured the heritability of a number of traits in *Microtus pennsylvanicus* using a half-sib design which allowed us to determine the proportion of the total phenotypic variance which has an additive-genetic basis. Additive genetic variance is responsible for the genetically induced phenotypic similarity of related individuals, and the rate at which a trait evolves is directly proportional to the additive genetic variance for that trait. The Chitty Hypothesis predicts that changes in the means of certain traits are the product of selection (Table 1). For natural selection to act, the traits must be heritable. If the Chitty Hypothesis is correct, we predict significant heritabilities for traits such as body growth and size and age at sexual maturity within groups of young obtained from animals captured at different phases in the

fluctuation. We also predict that young obtained from animals taken from different phases of a population fluctuation should retain phase-specific characteristics when reared under controlled laboratory conditions. A preliminary analysis of part of our data, together with an expanded discussion of various approaches to the study of heritable variation in mammal population is presented elsewhere (Boag and Boonstra, 1987).

MATERIALS AND METHODS

Study Site.—The study site was located on the outskirts of Toronto, Canada, at the Station for Atmospheric Experiments. The site is a flat grassland composed predominantly of *Agropyron repens*, *Bromus* spp., and *Poa pratensis*. The grassland is 44.5 ha in size, and, prior to our study, the grass was cut several times during each growing season. The habitats surrounding the Station were suitable for voles, and movement in and out was unrestrained.

Source Population.—A control grid to monitor population dynamics in the field was set up in May 1982 and had 49 trap stations spaced at intervals of 7.62 m, arranged in a 7×7 pattern. This grid allowed us to monitor the density of animals in the field and to compare their phenotypes with those of animals raised in the laboratory. One or two Longworth live traps were placed at every station and baited with oats; cotton was provided for warmth. Seventy-seven traps were on the grid at all times. Traps were protected from the elements by snow shelters (Iverson and Turner, 1969). During the main breeding season (see Fig. 1), traps were set every second week, and trapping was continued until December–mid-March, when there was continuous snow cover. Traps were also set on one occasion in January 1984. *Microtus pennsylvanicus* was the most abundant small mammal caught, but *Peromyscus leucopus* and *Blarina brevicauda* were also trapped occasionally. All voles were ear-tagged, and on each capture the following were recorded: tag number, trap location, trap type, body weight, sex, and sexual condition. Animals were classified as adults (>33 g), subadults (22–32 g), or juveniles (<22 g).

Quantitative-Genetics Experiment.—In

TABLE 1. Characteristics of voles that are predicted to be favored by selection during population fluctuations according to the Chitty Hypothesis (1967).

Traits	Increasing populations	Peak and declining populations
Innate capacity for increase	High	Low
Age at sexual maturity	Early	Late
Juvenile growth rate	High	Low
Adult body size	Heavy	Light
Length of breeding season	Long	Short
Tolerance to crowding	Low	High
Degree of spacing behavior	Docile	Aggressive

late March, April, and early May of 1982, 1983, and 1984, adult animals, which had overwintered, were obtained from traps set in the same field, but at least four home-range diameters from the control grid, so as not to disturb the grid population. These animals represented a random sample of the animals living adjacent to the control grid and, therefore, should not have differed from the control population genetically. There were no other experimental trapping grids (e.g., removal grids, etc.) at the Station during the three years. The animals were held in the lab for a 1–2-week period before they were mated. Because attempts in 1981 to obtain consistent breeding of all pairs in the lab were unsuccessful (usually only a fraction of animals breed in the laboratory, and these probably represent a nonrandom, “domesticatable” subset of the individuals), we constructed 42 3-m $\times$ 3-m enclosures in the field in the spring of 1982, using galvanized sheet metal (91 cm wide, half above and half below the ground). Each enclosure had three live-traps and one snow shelter, and the grass in all enclosures was fertilized in the spring of each year. Pairs of males and females were placed in these enclosures for mating.

We caught and removed three, three, and 18 weasels (*Mustela erminea*) during 1982, 1983, and 1984, respectively, from the control grid, breeding enclosures, and immediately adjacent areas. The weasels probably had a significant impact on the control population in 1984. They probably also caused the disappearance of most of our breeding males and females from the breeding enclo-

tures in early summer 1984, with the result that few of our males were able to sire more than one litter. As a result, the quantitative-genetics study in 1984 was largely a failure, and the data from this year will not be presented here.

Each male was sequentially mated to at least two females during the spring and summer to produce litters of half-sibs. This design was successful for most of the 1982 matings, but in 1983, the smaller number of available females forced us to remate some of them with different males. This deviation from random mating increased the similarity of sireships and hence decreased the 1983 heritability estimates by a small but unknown amount. Pregnant females were brought into the laboratory, where they gave birth. Young were weaned when they were 20 or 21 days old, separated into same-sex groups of three; usually littermates were put together. At weekly intervals for up to 13 weeks, the young were weighed and reproductive maturity was noted (testes scrotal or abdominal in males; vagina perforate or nonperforate in females). In the laboratory, all animals were maintained on an 18L:6D light cycle at 20°C. Water and food (Purina Lab Chow and Purina Rabbit Chow) were provided *ad libitum*, and lettuce was provided twice weekly. All animals were housed in 48 × 27 × 16-cm polypropylene cages on a bedding of hardwood chips and given cotton nesting material. All animals were tagged with fingerling ear-tags. The 1982 data set consisted of 488 offspring produced by 25 sires and 57 dams. The 1983 data set consisted of 426 offspring produced by 23 sires and 58 dams.

Statistical Analysis of Field Data.—The Jolly trappability estimate was calculated (see Krebs and Boonstra, 1984) and the Jolly-Seber method was used to estimate density and survival (Seber, 1982). Since the age of the voles was unknown, we used weight as an index of age. The weight at sexual maturity was calculated by the technique of Leslie et al. (1945). Maturity was judged in males by the presence of scrotal testes and in females by the presence of a perforate vagina, lactation, or pregnancy.

Statistical Analysis of Laboratory Data.—The weekly measurement data for the litters were processed to produce several summary

characters for each individual. We discuss a subset of these here: 1) maximum weight attained, 2) a linear regression producing growth rate in grams per day between 20 and 65 days of age (K), 3) estimated weight at 50 days, 4) weight at sexual maturity, and 5) age at sexual maturity. Other, secondary variables were measured and used to summarize factors common to a litter: 1) maternal nonpregnant weight, 2) paternal weight, 3) litter size at birth, 4) litter size at weaning, 5) an index of sex ratio in a litter (measured by assigning young males scores of 1, females 2, and averaging), and 6) birth date. We also recorded the maximum age an animal was allowed to reach and the age when its maximum weight was recorded. These litter variables or secondary individual variables were examined for correlations with the primary variables, and, as discussed later, were used as covariates to remove variation due to differences in birth date, litter size, time span covered by the regression, and so on.

The heritability analysis was carried out with PROC NESTED (SAS Institute, 1982). Raw data or residuals from the covariance analyses described above were cast in the form of two-level nested ANOVAs with random effects; progenies were nested in dams, which were nested in sires. PROC NESTED uses classic Model II ANOVA to produce variance components for dams, sires, and progenies, which were then used to estimate heritabilities (see Table 2). We estimated a "maternal component" as the difference between sire and dam components; this quantity includes maternal and other common-litter environmental effects, as well as one-fourth of the dominance effects (see Table 2). The significance of dam components was tested using the ratio of dam to progeny mean squares, while sire components were tested using the ratio of sire to dam mean squares; Satterthwaite's approximation was invoked where appropriate (Sokal and Rohlf, 1981). Approximate standard errors of heritabilities were calculated according to Turner and Young (1969).

RESULTS

Demography of Field Population

It is crucial to the explant experiment that

the source population from which the animals were taken show a multi-annual fluctuation in numbers. The mean trappability of the control population over the three years was 67%, and thus our estimates of density and survival should be reasonably accurate. Though we do not have population estimates of early spring 1982 densities, the population density on the control grid at that time was probably not dramatically different from the estimates obtained in June. To obtain the necessary breeding animals for the quantitative-genetics study, trapping took us three times as long in 1982 as in 1983. Thus the population in 1982 appeared to be already low and not in the process of a major spring decline. Figure 1 shows that the control population did show a multi-annual fluctuation, with 1982 being a year of rapid increase from low numbers in the late spring to peak numbers by December, with 1983 being a peak year with no sustained growth, and with 1984 showing an initial increase during the late spring, but then a decline in August. The rate of population increase (r) was 0.08 per week during 1982, 0.003 during 1983, and -0.005 during 1984. In their review of the population dynamics of *Microtus* in North America, Taitt and Krebs (1985) report that the *M. pennsylvanicus* populations going through cyclic fluctuations ($N = 10$) had mean densities between low and peak years of 22.5/ha and 155.5/ha (ratio 1:6.8), whereas populations showing annual fluctuations ($N = 16$) had mean densities varying between 57.2/ha and 171.6/ha (ratio 1:3.3). Our population fluctuation, though not extreme, had a range of densities closer to that of the cycling populations (population size on 24 June 1982 = 37.8 ± 16.6 /ha; population size on 24 June 1983 = 226.0 ± 26.2 ; ratio 1:5.9).

We calculated survival estimates for adults and subadults pooled for four periods of the year: late spring-summer (May-August), fall (September-October), winter (November-early March), and early spring (mid-March-April). Survival was generally lower in males than females and much lower in the summers of 1982 and 1984 than in 1983 (Fig. 1). At all other times, survival was generally high. We determined the survival of juvenile animals in each of the years

TABLE 2. Design of half-sib heritability analysis.

$V_P = V_A + V_D + V_I + V_{Ec} + V_{Ew}$,	
where	
V_P	= Total phenotypic variance
V_A	= Additive genetic variance
V_D	= Nonadditive (dominance) genetic variance
V_I	= Nonadditive (epistasis) genetic variance
V_{Ec}	= Environmental variance between litters
V_{Ew}	= Environmental variance within litters.
Variance components:	
Between sires	$s^2_S = 1/4 V_A$
Between dams	$s^2_D = 1/4 V_A + 1/4 V_D + V_{Ec}$
Within litters	$s^2_W = 1/2 V_A + 3/4 V_D + V_{Ew}$
Total	$s^2_T = s^2_S + s^2_D + s^2_W = V_P$
Heritability (h^2) estimates:	
From sire component:	$h^2 = \frac{4s^2_S}{s^2_T}$
From dam component:	$h^2 = \frac{4s^2_D}{s^2_T}$
From full sibs:	$h^2 = \frac{2(s^2_S + s^2_D)}{s^2_T}$
Thus, assuming $V_D = 0$,	
V_P	= s^2_T
V_A	= $4s^2_S$
V_{Ec}	= $s^2_D - s^2_S$
V_{Ew}	= $s^2_W - 2s^2_S$.

from the number of new young (<30 g) trapped per lactating female. In 1982, 1983, and 1984, there were 1.39 ($N = 186$ juveniles), 0.83 ($N = 142$), and 0.89 ($N = 117$) young caught per lactating female. Thus, survival of young to recruitment was highest in the year when population size was increasing (henceforth the "increase year") and lowest in the peak and post-peak years. The breeding season was also extended by about one month in 1982 to mid-October, in comparison with 1983, when it ended in mid-September; a prolonged breeding season is typical of an increase year (Krebs and Myers, 1974). In 1983, over 50% of the females were lactating by 29 April, whereas in 1984, this level of breeding was delayed until 18 May; delayed onset of the breeding season often occurs during decline years (Krebs and Myers, 1974).

The age at sexual maturity also affects reproductive rate and tends to be lower in increasing populations and higher in peak and declining populations (Krebs and Myers, 1974). Males showed relatively little vari-

FIG. 1. Population and survival changes in *Microtus pennsylvanicus* on a 0.37-ha trapping grid calculated by Jolly-Seber methods. Mean survival estimates ($\pm$ SE) are given for males (upper value) and females (lower value) for summer, fall, winter, and spring. Hatched areas indicate the nonbreeding season.

ation in median weight at sexual maturity, but females during the increase year were 5–10 g lighter during the late spring and summer than during the peak and post-peak years (Table 3).

During the increase, animals also grow more rapidly than during the peak and decline (Krebs and Myers, 1974). We calculated the instantaneous relative growth rates

for males and females during the May–August breeding season and performed an analysis of covariance comparing growth in the three years. There were significant differences in slope for both males ($F = 10.9$, $d.f. = 2, 203$, $P < 0.0005$) and females ($F = 5.2$, $d.f. = 2, 301$, $P < 0.01$), with animals showing the following rankings in both sexes: 1982 > 1983 = 1984 (Fig. 2).

TABLE 3. Median body weights (g) at sexual maturity for *Microtus pennsylvanicus* on the control grid, with 95% confidence limits in parentheses.

	Males	Females
1982: June–July	29.3 (26.3–32.7)	20.5 (15.9–26.3)
August	29.2 (26.5–32.5)	23.6 (15.0–37.0)
September	32.2 (26.7–38.9)	27.2 (18.2–42.4)
1983: May–July	27.7 (25.4–30.2)	26.1 (23.5–29.0)
August	29.4 (26.6–32.4)	32.8 (29.8–35.0)
September	34.8 (32.2–37.5)	37.6 (34.2–41.3)
1984: May–July	28.2 (26.0–30.4)	29.8 (27.2–32.5)
August	25.3 (8.6–74.7)	25.0 (18.2–32.6)

FIG. 2. Instantaneous relative growth rates for *M. pennsylvanicus* during May–August. Regression lines for 1982, 1983, and 1984 are based, respectively, on 31, 122, and 56 observations for males and on 21, 172, and 114 observations for females. Slopes of the three regression lines are significantly different ($P < 0.01$).

We conclude that our control population did show a multi-annual fluctuation with many of the changes in birth rates, death rates, and growth rates that characterize cycling microtine populations. Controlled breeding of animals from the source population should provide a test of the Chitty Hypothesis.

Results from the Laboratory Populations

1982 and 1983 Animals Compared.—The mothers used for laboratory breeding were significantly larger in 1983, but this was not true of the fathers (Table 4). The growth curves and statistics for the young were quite different, despite identical rearing conditions. Although 1983 mothers were only 8% larger than their 1982 counterparts, their young were about 18% heavier at weaning and at 50 days, and had 32–74% higher growth rates.

The 1983 young also weighed more at sexual maturity (Table 4). However, the relationship between growth rate and age at sexual maturity in the two years differed

between the two sexes. Females reared in the laboratory in 1983 were only 0.2% older but 20% heavier at sexual maturity compared with 1982 females, while 1983 males were 16% younger but 5% heavier. This suggests that sexual maturity in females is primarily a function of age, while in males sexual maturity is a function of body weight.

Correlations and Covariates.—Phenotypic correlations among the variables measured in lab-reared young are shown in Table 5, pooled across 1982 and 1983. Note the time-of-breeding effects in the birth-date correlations; later litters tend to have larger parents resulting from the parents' continued growth. Maximum weight is highly correlated with growth rate (K), estimated weight at 50 days, age at sexual maturity, and weight at sexual maturity, and may reflect a common underlying factor associated with rapid growth and large size. Note also the regularly significant correlations with maternal weight, a hint that maternal effects pervade the data.

The main implication of these correla-

TABLE 4. Comparison of *Microtus* parents and their progeny bred in the laboratory in two years. See Materials and Methods for explanations of variables.

Variable	1982		1983		<i>F</i> (Overall ANOVA)
	$\bar{x} \pm \text{SE}$	<i>N</i>	$\bar{x} \pm \text{SE}$	<i>N</i>	
Maternal weight ¹ (g)	38.38 $\pm$ 0.44	90	41.57 $\pm$ 0.55	79	29.7***
Paternal weight (g)	50.32 $\pm$ 0.59	79	49.37 $\pm$ 0.73	79	0.0
Litter size	5.76 $\pm$ 0.16	93	5.99 $\pm$ 0.21	79	0.6
Sex ratio in litters	1.51 $\pm$ 0.02	93	1.51 $\pm$ 0.02	79	0.1
Female progeny:					
Maximum weight (g)	28.84 $\pm$ 0.42	228	34.69 $\pm$ 0.51	205	38.4***
Growth rate (<i>K</i>) (g/day)	0.19 $\pm$ 0.01	264	0.33 $\pm$ 0.01	233	33.6***
Estimated weight at 50 days (g)	26.43 $\pm$ 0.36	228	32.08 $\pm$ 0.43	228	46.1***
Age at sexual maturity (days)	38.10 $\pm$ 1.29	158	38.20 $\pm$ 0.98	148	0.1
Weight at sexual maturity (g)	24.34 $\pm$ 0.42	158	29.22 $\pm$ 0.53	148	42.3***
Male progeny:					
Maximum weight (g)	37.11 $\pm$ 0.53	224	41.09 $\pm$ 0.61	193	13.4***
Growth rate (<i>K</i>) (g/day)	0.37 $\pm$ 0.01	224	0.49 $\pm$ 0.01	193	20.1***
Estimated weight at 50 days (g)	32.59 $\pm$ 0.43	224	37.53 $\pm$ 0.52	193	19.5***
Age at sexual maturity (days)	49.72 $\pm$ 0.78	204	41.86 $\pm$ 0.58	182	14.7***
Weight at sexual maturity (g)	32.98 $\pm$ 0.34	204	34.62 $\pm$ 0.32	182	0.2

¹ Average of nonpregnant values closest to parturition date of each litter in analysis.*** *P* < 0.001.

tions for the quantitative-genetic analyses that follow is that an individual's score for variables such as maximum weight is affected by date of birth, the maximum age to which it was measured, and so on. We used multiple-regression techniques to establish which covariates had significant effects on a primary variable, and then removed those effects in a covariance analysis (ANCOVA). The residuals were used for the analyses described next. Tables 6 and 7 indicate which covariates were significant for each analysis; we present the uncorrected results as well, for comparison.

Quantitative-Genetic Results.—Only female progeny consistently showed nonzero among-sire variance components (see Tables 6 and 7). Four times this component gives an estimate of heritability; heritability in 1982 females ranged from 0 for maximum weight and estimated weight at 50 days to 0.27 for *K*. Dam components were usually considerably greater than sire components. Assuming that nonadditive genetic variance is negligible, this argues for substantial between-litter common environment (e.g., maternal) effects (Falconer, 1981 pp. 157–158). For convenience, in the rest of this paper, we refer to the difference between sire and dam components as a “maternal component,” although this difference does contain common environmental

effects as well as part of the dominance variance. In fact, the explant experiment suggests that much of the common-litter effect is a product of variation in maternal condition (see Discussion).

The 1982 males had a nonzero sire component for *K* only, giving a heritability estimate of 0.33, with other characters showing no sign of a heritable component. The average dam component was twice what it was for the 1982 females; that is, maternal effects were especially significant for male offspring.

Unlike the 1982 results, the design used to gather the 1983 data (Table 7) partially violates the assumptions of the quantitative-genetic model, so that the between-sire differences are reduced since progeny of different sires are actually more than half-sibs; they also include a fraction of V_D . Because some of the same females were remated to different males in the 1983 experiment, we should be underestimating h^2 to some extent. Once again, the female offspring show the higher heritabilities, ranging up to 0.62 for weight at sexual maturity. However, for the 1983 females, the dam components are not much larger than the sire components. The 1983 males showed little heritable variation; the dam components for male offspring in 1983 were again much larger than those for female offspring.

The 1983 data were reanalyzed excluding all dams mated to more than one sire. The small sample size remaining (14 sires, 29 dams, 182 young) makes it difficult to estimate heritabilities accurately. Estimates for the various characters were ranked similarly to those shown in Table 7, with average heritability estimates ($\pm$ SE) for female progeny of 0.52 ± 0.45 and average estimates of 0.27 ± 0.52 for male progeny. None of these estimates is significantly different from zero, but they are larger than those for the entire data set including remated females, as expected.

Dam components were often significantly different from zero, while none of the sire components in the adjusted data was significant. The heritabilities based on the sire components show correspondingly large standard errors. In most of the covariance analyses for the variables examined here, date- and litter-size-related covariates (birth date, maximum age, and litter size at birth) were of prime importance; litter size is in turn date-related, insofar as mothers grew over the season, and as they grew their litter sizes increased. We suspect that the significant among-sire components in the unadjusted 1982 females were largely due to correlated birth dates of females mated to the same male. Thus, when effects of birth date (or variables correlated with birth date) are removed, the sire effects are diminished. We tested for such an effect by carrying out ANOVAs on birth date. Although no statistically significant sire effects were found, the analysis suggested that there may be a biologically important sire component (about 17%; $F = 1.76$, $d.f. = 23, 33$, $P < 0.10$) for 1982 females; in all other cases there was no correlation between the parturition dates of females sharing the same sire.

Repeatability of Litter Size.—Although it is not shown in Tables 6 and 7, we also examined the repeatability of litter size each year; it was low but significantly repeatable for females in both years (1982: $r = 0.26$, $F = 1.91$, $d.f. = 35, 55$, $P < 0.01$; 1983: $r = 0.44$, $F = 3.08$, $d.f. = 26, 44$, $P < 0.001$). As expected, the multiple litters sired by each male were not significantly repeatable in either year. Interestingly enough, however, when we scored offspring as either 1

TABLE 5. Pooled within-group phenotypic correlations among variables for 1982 and 1983 female progeny (below diagonal) and male progeny (above). Correlations not significant at $P < 0.05$ are omitted. Sample sizes for females is 223, except for correlations with age or weight at sexual maturity where $N = 173$; corresponding samples for males were 277 and 255, respectively. See Materials and Methods for explanations of variables.

	Maximum weight	Growth rate (K)	Estimated weight at 50 days	Age at sexual maturity	Weight at sexual maturity	Birth date	Litter size at birth	Maximum age	Maternal weight	Paternal weight	Sex ratio in litters
Maximum weight	1	0.74	0.92	-0.56	0.78	—	—	—	0.26	—	0.15
Growth rate (K)	0.77	1	0.81	-0.42	0.60	—	0.14	—	—	—	—
Estimated weight at 50 days	0.95	0.82	1	-0.62	0.82	—	—	-0.14	0.25	0.13	0.18
Age at sexual maturity	-0.17	—	-0.26	1	-0.30	—	—	—	—	—	-0.13
Weight at sexual maturity	0.50	0.38	0.49	0.53	1	—	—	-0.16	0.24	—	—
Birth date	—	—	—	—	—	1	0.15	-0.32	—	0.29	—
Litter size	—	0.19	—	—	—	—	1	—	0.28	—	0.42
Maximum age	—	-0.18	—	0.32	—	-0.21	—	1	—	—	—
Maternal weight	0.16	—	0.14	—	0.27	0.30	0.20	—	—	—	0.21
Paternal weight	0.18	—	0.14	—	—	0.24	—	0.15	1	—	—
Sex ratio in litters	0.15	0.16	0.16	-0.18	—	—	—	-0.34	—	0.17	1

TABLE 6. Variance components (%), heritabilities ($h^2 \pm \text{SE}$), and total phenotypic variances for 1982 data. Upper data are adjusted for covariates, lower data are not adjusted (see text).

Character	Female progeny ^a						Male progeny ^b						Significant covar- iated ^d	
	Sire	Dam	Litter	Mat- ernal ^c	Sire h ²	Total variance	Significant covar- iated ^d	Sire	Dam	Litter	Mat- ernal ^c	Sire h ²		Total variance
Adjusted:														
Maximum weight	0.0	38.6***	61.4	38.6	0.00 ± 0.34	22.91	1, 2, 4, 5, 6	0.0	46.6***	53.4	46.6	0.00 ± 0.32	50.19	1, 2
Growth rate (K)	6.8	11.1	82.1	4.3	0.27 ± 0.30	0.0109	1, 2, 5, 6	8.3	12.8	78.9	4.5	0.33 ± 0.38	0.0215	3, 5
Estimated weight at 50 days	0.0	35.3***	64.7	35.3	0.00 ± 0.34	19.13	1, 2, 4, 5, 6	0.0	47.9***	52.1	47.9	0.00 ± 0.33	35.17	3
Age at sexual maturity	1.4	0.0	98.6	0.0	0.06 ± 0.27	158.7	2, 6	0.0	55.6***	44.4	55.6	0.00 ± 0.38	105.6	1, 2, 5
Weight at sexual maturity	3.4	11.0	85.7	7.6	0.13 ± 0.38	13.73	1, 3	0.0	39.6**	60.4	39.6	0.00 ± 0.33	24.53	1, 3
Unadjusted:														
Maximum weight	13.1	33.4***	53.5	20.3	0.52 ± 0.46	29.48	—	0.0	51.7***	48.3	51.7	0.00 ± 0.30	60.95	—
Growth rate (K)	24.1*	6.0	69.8	0.0	0.96 ± 0.45	0.0152	—	8.8	13.8	77.4	5.0	0.35 ± 0.22	0.0216	—
Estimated weight at 50 days	18.6*	24.2**	57.2	5.6	0.74 ± 0.47	22.79	—	0.0	46.5***	53.4	46.5	0.00 ± 0.34	34.67	—
Age at sexual maturity	6.7	7.9	85.4	1.2	0.27 ± 0.39	186.8	—	0.0	56.1***	43.9	56.1	0.00 ± 0.45	106.7	—
Weight at sexual maturity	2.9	28.2*	68.9	25.3	0.11 ± 0.46	16.64	—	0.0	40.0***	60.0	40.0	0.00 ± 0.39	24.99	—

^a 22 sires, 52 dams, 178 offspring.^b 24 sires, 54 dams, 160 offspring.^c Maternal component calculated as the difference between sire and dam components and, hence, reflects the effects of maternal and other common-litter environmental effects, as well as one-fourth of the dominance effects (see Table 2).^d Covariates: 1) birth date, 2) age when maximum weight recorded, 3) maximum age an animal was allowed to reach, 4) litter size at birth, 5) litter size at weaning, 6) sex ratio in a litter.* $P < 0.5$.** $P < 0.01$.*** $P < 0.001$.

TABLE 7. Variance components (%), heritabilities ($h^2 \pm SE$), and total phenotypic variances for 1983 data. Upper data are adjusted for covariates, lower data are not adjusted (see text).

Character	Female progeny ^a						Male progeny ^b						Significant covar- iates ^d	
	Sire	Dam	Litter	Maternal ^c	Sire h ²	Total variance	Significant covar- iates ^d	Sire	Dam	Litter	Maternal ^c	Sire h ²		Total variance
Adjusted:														
Maximum weight	9.2	7.6	83.2	0.0	0.37 ± 0.31	36.28	1, 2, 3, 4, 6	0.9	40.3***	58.8	39.4	0.04 ± 0.39	57.17	1, 2, 6 2, 3
Growth rate (K)	2.6	0.0	97.4	0.0	0.11 ± 0.16	0.0191	1, 2, 3, 5, 6	13.6	11.0	75.4	0.0	0.54 ± 0.38	0.0287	
Estimated weight at 50 days	6.8	13.7	79.5	6.9	0.27 ± 0.32	27.58	1, 2, 3, 4, 6	2.0	43.5***	54.5	41.5	0.08 ± 0.41	41.78	
Age at sexual maturity	15.0	7.6	77.4	0.0	0.60 ± 0.47	132.8	3	0.0	23.2*	76.8	23.2	0.00 ± 0.30	55.34	1
Weight at sexual maturity	15.5	0.0	84.5	0.0	0.62 ± 0.40	26.62	1, 6	0.0	43.8***	56.2	43.8	0.00 ± 0.26	21.38	1, 2
Unadjusted:														
Maximum weight	5.3	27.9**	66.8	22.6	0.21 ± 0.37		—	0.3	47.5***	52.2	47.2	0.01 ± 0.42	71.62	—
Growth rate (K)	0.0	14.1	85.9	14.1	0.00 ± 0.25	47.73	—	13.4	15.1	71.4	1.7	0.54 ± 0.39	0.0315	—
Estimated weight at 50 days	2.0	35.8***	62.2	33.8	0.08 ± 0.38	0.0224	—	4.8	45.9***	49.3	41.1	0.00 ± 0.43	50.75	—
Age at sexual maturity	12.3	12.6	75.0	0.3	0.49 ± 0.46	35.97	—	1.2	24.7*	74.1	23.5	0.05 ± 0.36	56.33	—
Weight at sexual maturity	9.5	11.2	79.3	1.7	0.38 ± 0.42	136.9	—	0.0	43.8***	56.2	43.8	0.00 ± 0.27	22.24	—

a 23 sires, 54 dams, 172 offspring.
b 23 sires, 54 dams, 161 offspring.
c Maternal component is calculated as the difference between sire and dam components and, hence, reflects the effects of maternal and other common-litter environmental effects, as well as one-fourth of the dominance effects (see Table 2).
d Covariates: 1) birth date, 2) age when maximum weight recorded, 3) maximum age an animal was allowed to reach, 4) litter size at birth, 5) litter size at weaning, 6) sex ratio in a litter.
* $P < 0.5$.
** $P < 0.01$.
*** $P < 0.001$.

or 2 (depending on their sex) and looked at the between-sire component for "sex score," the 1982 females showed a between-sire component of 12%. The same analysis in 1983 gave a smaller between-sire component (6%). In neither year was there a dam effect on sex ratio.

DISCUSSION

Our data provide two different tests of the Chitty Hypothesis. First, we can ask the question of whether year-to-year differences in the phenotypes of animals in the field are maintained in samples of those animals bred in the field and reared in the lab. This is a type of explant experiment reminiscent of those traditionally carried out by botanists seeking to understand the origins of ecotypic variation in plants (e.g., Clausen et al., 1948). Second, we can use the heritability data to ask the question of whether there is heritable variation among animals alive in one season such that selection might produce a different mean phenotype in the next season.

The 1982 laboratory-raised young had lower growth rates than their 1983 counterparts (Table 4), yet the 1982 field animals had higher growth rates than did the 1983 field animals (Fig. 2). This suggests that the 1983 field animals should have grown much faster than they did. The most obvious difference between the two years was that 1982 began with low densities, while 1983 began with high densities (Fig. 1). Other studies have also found poorer growth rates in young at higher population densities (Boonstra, 1978; Beacham, 1980; Sauer and Slade, 1986; c.f. Gaines and Rose, 1976), but these studies were not able to separate the effect of higher density from the effect of lower nutrition, which probably accompanies higher density. Taitt and Krebs (1981) found that the addition of food can improve growth in spite of high density. Newson and Chitty (1962) and Krebs (1966) removed voles of different species from declining populations and found that some grew well in the laboratory, while none grew well in the field. Thus, in our study, poor growth in the field was probably caused by some factor (such as density, nutrition, and/or behavior [Krebs, 1985]) that restrains growth, rather than the selective removal of animals with

the potential for rapid growth. The 1983 laboratory young were born to larger mothers than their 1982 counterparts (Table 4). Other studies on rodents have found that offspring growth and weight are directly related to maternal condition and size (Monteiro and Falconer, 1966; El Oksh et al., 1967; Moore et al., 1970; Myers and Masters, 1983; Roth and Klein, 1986). Thus, the differences in maternal weight between the two years may be sufficient to account for the differences in offspring performance in the laboratory.

Comparison of the age at sexual maturity of field and laboratory young is complicated, because we do not know the age of young in the field. Nevertheless, we can deduce whether animals are younger at one time than another by examining relative growth; if animals are growing faster in one year than another, but both groups have the same weight at sexual maturity, the former must be younger (Krebs and Myers, 1974). Though the median weights at sexual maturity in the field were similar in 1982 and 1983 (Table 3), males were growing approximately twice as fast in 1982 as in 1983 (Fig. 2; 0.51 g/day versus 0.25 g/day, for a 30-g male). Thus, field males were younger at sexual maturity in 1982 than in 1983. In contrast, 1982 laboratory males were older at sexual maturity than 1983 laboratory males (Table 4). Median weights at sexual maturity were approximately 6 g lower for field females in 1982 than in 1983 (Table 3), and 1982 females were growing approximately three times as fast as in 1983 (Fig. 2; 0.64 g/day versus 0.20 g/day for a 30-g female). Thus, field females were younger at sexual maturity in 1982 than in 1983. In contrast, 1982 and 1983 laboratory females were similar in age at sexual maturity (Table 4). Thus, in both sexes, the 1983 field young could potentially have matured much earlier than they did and they were not intrinsically late-maturing animals. Other field studies have found that maturation is inversely related to density, especially adult female density (Bujalska, 1973; Boonstra, 1978; Saitoh, 1981). Our work also indicates that maternal effects, which give rise to faster growing laboratory young in 1983 than 1982 and the earlier maturation in 1983 laboratory males, may be overridden by

postweanling effects determined by the local environment in the field.

The quantitative-genetics results suggest that half-sib heritabilities in this study average about 0.24 in female offspring and 0.10 in male offspring, or 0.28 in females and 0.17 in males if the 1983 data excluding remated females are used. They range from 0 to 0.62, depending on which group one examines; our maximum values are similar in magnitude to those found by Anderson (1975) for *Microtus townsendii*. However, Anderson's results were based on full-sib estimates for the most part, and the consistent maternal effects demonstrated here and in other quantitative-genetic studies of domestic rodents (Eisen and Durrant, 1980; Eisen et al., 1980; Falconer, 1981) make it almost certain that her estimates were high. For our data, the full-sib heritability is twice the sum of the sire and dam components. This brings the 1982 average heritability up to 0.43 for females and 0.84 for males. The 1983 full-sib estimates were similar: 0.31 for females and 0.71 for males. Even excluding remated females from the 1983 data produces little change in these estimates: 0.35 for females and 0.65 for males. Averaging across sexes, these full-sib results are closer to those reported by Anderson (1975); given the discrepancy between sire and dam estimates we found, (particularly in male progeny), such averages are clearly inflated by a maternal effect.

Heritabilities can be low either because there is little additive-genetic variance, or because there are large environmental contributions to phenotypic variance. One possible reason for reduced V_A in microtines is inbreeding caused by small effective population size during population lows (e.g., June 1982 in Fig. 1). Though we lack data to dismiss this possibility, it seems improbable. The study area adjoins an abundance of suitable vole habitat, and *M. pennsylvanicus* is an excellent disperser (Lomolino, 1984). Furthermore, vole populations are usually low for a relatively short time, after which there is rapid increase. Nei et al. (1975) suggest that under such conditions, loss of genetic variation due to bottlenecks may be minimal. Finally, the electrophoretic data available for microtines suggests that they usually possess high levels of het-

erozygosity despite population fluctuations, unless a population is very isolated, as on an oceanic island (Kilpatrick and Crowell, 1985). There is some evidence that bottlenecks may encourage changes in gene frequencies as a result of genetic drift (Gaines, 1985; Gyllenstein, 1985), but this does not seem to be associated with major reductions in overall variation.

The most likely reason why our heritabilities are low is because the phenotypic variance is relatively high, and because maternal effects channel much of this variation between dams. The coefficient of variation for estimated weight at 50 days in our 1982 female progeny was 18%, which is higher than the CV for spring weight in the 1982 breeding females (13%). Other laboratory studies of *M. pennsylvanicus* have recorded average CV's for body weight in females of 25% (Morrison et al., 1977), and 16% (Innes and Millar, 1979). Thus, although body weight in our young voles is more variable than among our adults, our rearing regime has not accentuated phenotypic variation compared with other lab studies. It would be desirable to have additional data on the means and variances of our characters for young of known age that were born and reared in the wild, but such data are scarce. We cannot readily compare the variances of lab and wild voles for characters such as K or weight at sexual maturity, because the lack of voles of known age required these measures to be calculated indirectly from the field data. It is likely that older adults that have overwintered, such as the spring 1982 breeding females, have undergone additional selection since they were 50 days old; such selection probably reduces phenotypic variation in most traits, particularly by culling individuals that are environmentally disadvantaged early in life. Although heritabilities increased from 1982 to 1983, on average the total variance among progeny increased, as did the proportion of variance within litters (Tables 6 and 7). Thus the increased sire-based heritabilities seen in 1983 were primarily due to diminished maternal effects. The most likely explanation for the large maternal effects in 1982 was the smaller size and the probably poorer condition of the 1982 mothers, which we discuss later in more detail. For passerine

birds, there is evidence that when broods are enlarged, or in springs where the food supply for nestlings is poor, the heritability of body size is reduced, because of added environmental contributions to phenotypic variance; selection may later preferentially eliminate the small "phenodeviants," hence reducing V_p and increasing h^2 (van Noordwijk, 1984; van Noordwijk et al., unpubl.).

Why should male offspring show less between-sire variance than females (Table 6 and 7)? One reason is the higher growth rates of males. This results in greater measurement error for most male characters, which tends to increase the total phenotypic variance for males (Table 6 and 7). Much of the increased variance occurs between dams as a common-environment effect. This is because groups of sibs were housed together after weaning and, hence, weighed together. If the offspring of a sire's first dam were weighed in the morning and those of his second dam were weighed six hours later, the young of the second dam would, on average, weigh 10% more (for their age) than those of the first. Since males were growing twice as fast as females, the effect is especially pronounced in male variance components. Such effects are unlikely to increase sire components, because there was no timing bias in the measurement of offspring within sires; however, these effects reduce sire heritabilities because these are ratios of sire variance components to total variance, and the latter is increased. This is also why growth rate itself (K) was the only variable to show consistent signs of nonzero heritability in males.

These difficulties reflect one of the shortcomings of our experimental design; we did not cross-foster litters before or after weaning. The prevalence of common environmental effects in mammals encourages one to randomize neonates among mothers, as well as to standardize litter sizes and sex ratios at birth, where this is possible (e.g., with laboratory mice or rats; see Atchley, 1984). Since such manipulations do not occur in nature, one could argue that they should not be carried out in the lab if one wants to estimate the effective heritability of a trait in the wild. This is a difficult issue to resolve; however, it would seem reasonable to use controls to minimize artifacts

introduced by the experimental design itself. Partial cross-fostering also allows one to estimate maternal effects, direct genetic effects, and direct maternal-genetic covariance terms independently (Cheverud, 1984). Unfortunately, it is almost impossible to achieve the 90% successful, predictably synchronous pregnancies necessary for cross-fostering, when one is dealing with anything other than a highly domesticated mammal in a laboratory setting. The birth-date effects we found in our data also make it difficult to ensure that all of a male's matings are randomized over the mating season. Unfortunately, the a posteriori statistical controls we used here are a weak substitute for a priori experimental control.

The Chitty Hypothesis predicts that phenotypic variation in traits changing during microtine cycles should be strongly heritable. We have interpreted our results as showing low heritabilities and, hence, failing to support the hypothesis. This conclusion rests on two assumptions. First, we assume that our experimental design is powerful enough to detect sire effects at some minimum desired level. Second, implicit in our test is a notion of what constitutes a "low heritability." Heritabilities of 0.20–0.30 are sometimes significant and considered relatively "high," especially in measures closely linked to fitness (e.g., fecundity) (Falconer, 1981).

Several methods measure the power of an experiment to refute a null hypothesis (Sokal and Rohlf, 1981; Hays, 1981). There is little consensus on how to evaluate unbalanced, nested ANOVAs with random effects. Donner and Wells (1986) compared several methods for placing confidence intervals around intraclass correlations and concluded that the traditional method ($\pm t_{n-1}$ SE), based on the approximations available for SE (e.g., Turner and Young, 1969), was satisfactory. Klein (1974) used normal approximations to estimate the power of balanced versions of half-sib ANOVAs.

We used such techniques to approximate the minimum heritabilities we could have detected. Given that our overall analysis consists of many semi-independent tests (males, females, five characters, two years, etc.), we adopted $\alpha = \beta = 0.10$ for these

calculations, as combinations of two or more tests provide additional evidence that average heritabilities exceed zero (Sokal and Rohlf, 1981 p. 780). The minimum detectable heritability was determined as $E(h^2) = (Z_\alpha - Z_{(1-\beta)})SE$, where $Z_{(1-\beta)}$ is the Z score associated with the desired probability of avoiding a Type II error, Z_α is the Z score associated with the α level, $E(h^2)$ is the minimum significant heritability, and SE is the standard error.

Averaged over traits and sexes, such calculations indicate that in 1982 we can be 90% certain that a sire heritability of 0.39 is greater than zero at $P < 0.1$. Since the average SE is the same (0.34), the limit in 1983 is also 0.39 for the total data set. The minimum detectable heritability for the 1983 data, excluding multiply mated females, would be about 0.54. These are considerably higher than the average adjusted heritabilities actually observed: 0.08 (1982), 0.26 (1983, remates included), and 0.40 (1983, remates excluded), respectively.

The other assumption is that heritabilities on the order of 0.08 to 0.40 are "low" and may be sufficient to falsify the Chitty Hypothesis. One way to predict the heritabilities required by the hypothesis is to work backwards from observed phenotypic responses, using the relationship $R = ih^2 SD$ (Falconer, 1981), where response to selection (R) is a function of selection intensity (i), heritability (h^2), and the phenotypic standard deviation (SD) of a trait. The main difficulty with such an approach is the lack of estimates for key parameters from natural populations.

There are virtually no data on selection intensities in microtines. Endler (1986) summarized the i values available for natural populations across all taxa, noting that some studies finding weak selection are probably not published. Most (61%) published examples of directional selection on metric traits are not significantly different from zero, with about two-thirds showing $i \leq 0.3$, about the lowest intensity detectable in field studies (Endler, 1986). Among i values that differed from zero, the geometric mean was 0.59, close to the mean of studies involving artificial selection (0.71) and 95% of the significant natural examples were under 0.5. Compared with plants and

invertebrates, there are few studies of wild vertebrates in which i has been found to exceed 0.4–0.7. For instance, i was in this range for selection on Darwin's finches suffering 85% mortality in one of the most intense episodes of selection known for avian morphology (Boag and Grant, 1981). Higher i values are realized during artificial selection on rodents, but this involves truncation selection in controlled environments.

Over the short term, selection on successive generations is additive (Falconer, 1981); hence, we assume that the response per generation is constant. Little is known concerning microtine demography during population increases or declines, i.e., when selection acts or over how many generations it acts. We know that declines typically occur faster than increases and suspect that a large proportion of the animals produced during an increase results from multiple litters of older, established adult females. The data available suggest that increases involve 3–4 generations of animals at most, while declines involve 2–3 generations (Krebs and Myers, 1974).

Using such assumptions, we solved for h^2 in the response equation using two traits (female adult body size and growth rate). Peak populations typically contain individuals 20–30% larger than adults in low or declining populations (Boonstra and Krebs, 1979). (Adults in our study did not span such a range, although some progeny measures did, such as estimated weight at 50 days in 1982 versus 1983 females.) We can nevertheless ask whether selection could increase the weight of our 1982 adult females by 25%. The average weight of females in spring 1982 was 37.41 g ($SD = 4.762$, $N = 61$), so a 25% increase would lead to females weighing 46.76 g in early 1983, i.e., $R = 9.35$. Solving for h^2 under these conditions gives the heritabilities in Table 8. These suggest that such a change would be impossible over even six generations of "typical" selection intensities and would require a heritability of 0.82 over four generations of selection that is as strong as has ever been observed in wild vertebrates to date. Only under selection pressures comparable to artificial selection on lab mice could change occur with heritabilities at the upper limits predicted for our study, and even then the

TABLE 8. Estimated heritabilities required to effect phenotypic changes predicted by the Chitty Hypothesis in two, four, and six generations with different selection intensities (i). Heritabilities greater than one are impossible values. See text for details.

Character	i	Heritabilities required		
		2 gener- ations	4 gener- ations	6 gener- ations
Adult body size	0.3	3.28	1.64	1.09
	0.6	1.64	0.82	0.55
	1.2	0.82	0.41	0.27
Growth rate	0.3	1.87	0.93	0.62
	0.6	0.93	0.46	0.31
	1.2	0.46	0.23	0.16

change would require four generations or more.

For the growth-rate change between our 1982 and 1983 female progeny (Table 4), R is 0.138 (SD = 0.123). Table 8 suggests that such a change would require very strong selection over at least four generations, given any heritability short of our most conservative upper-limit estimate for the 1983 data excluding females mated to more than one sire. Our mean observed heritabilities could only lead to such a change over 4–6 generations of selection at an intensity seldom seen in wild vertebrate populations. Obviously selection could be responsible for some of the smaller changes seen in our study, but our calculations suggest that many published examples of phenotypic changes seen during microtine cycles would require high heritabilities and very strong selection over several generations.

The prevalence of maternal or common-environment effects in our data is not an artifact of statistical design. The relatively short lifespan of these animals (perhaps 8–13 weeks at best [Krebs and Myers, 1974]) means that most individuals display “indeterminate” growth. In the laboratory, Morrison et al. (1977) found that *M. pennsylvanicus* were still gaining weight at 68 weeks of age. This, combined with the presence of overlapping generations, means that most of the body-size variation seen in a field population at any given time is likely to be largely nongenetic. The heritabilities in nature probably are low, and selection could not change the genetic makeup of the population with respect to the kinds of char-

acters examined here, within the 2–4 generations required for transitions from low to high density in microtine population fluctuations (Krebs and Myers, 1974; Krebs, 1979; Taitt and Krebs, 1985). The dramatically different young produced by relatively similar parents in 1982 and 1983 (Table 4) are probably due in large part to some physiological “condition” factor that causes females from a peak population to produce large and rapidly growing young, i.e., a maternal effect. Given the similar litter sizes in 1982 and 1983, this might be expressed as the quality or quantity of milk produced prior to weaning. Other workers (e.g., Smolten and Keller, 1979) have sought evidence that “lactational deficiency” is an important maternal effect involved in the phenotypic changes seen during microtine population cycles, but the results are not conclusive. Recently, both Hansson (1984) and Boonstra (1985) have revived Chitty’s (1952) proposal that some aspect of maternal condition is reflected in the progeny. The major enigma in population cycles is the cause of the decline, and it is not clear whether the maternal effects we have seen may also affect long-term viability. How long maternal effects last in microtines is unknown. Laboratory evidence on other species (Monteiro and Falconer, 1966; Atchley, 1984; Roth and Klein, 1986) indicates that the maternal effects attenuate with time and that the genotypic effect becomes increasingly important. However, this evidence is obtained from the benign world of the laboratory. Field conditions, such as increasing densities in a peak population followed by winter, may reinforce any maternal effects. In cases where it has been possible to follow larger mammals throughout their natural lives in the wild and to estimate lifetime reproductive success, there are indications that maternal condition has permanent repercussions on the total fitness of offspring, overriding genetic contributions to individual differences (Albon et al., 1987). We suggest that these maternal effects together with seasonality could introduce the inherent lag times characteristic and perhaps necessary for most microtine population fluctuations (Krebs and Myers, 1974; Taitt and Krebs, 1985).

Ideally, we would have liked to test

whether aggressive behavior changed over a population fluctuation and whether it was heritable, as required by the Chitty Hypothesis. Krebs (1979) suggested a direct correlation between the amplitude of population changes and the heritability of spacing behavior, with strongly cyclic populations exhibiting a high heritability of aggressive behavior. Attempts to build simulation models of population cycles incorporating genetically based aggressive behavior have met with varied success. Stenseth (1981) was unable to obtain cycling in a system with two behavioral morphs, a docile phenotype and an aggressive phenotype, which were under Mendelian control. Anderson (1975 appendix) created a more realistic model in which she had a continuum of 10 or more phenotypes with a gradation in aggression and reproductive fitness. In her overlapping-generations model, she obtained population cycles with approximately the right periodicity. However, these cycles disappeared when the heritability of aggressive behavior dropped below 0.8. Though high heritabilities for aggressive behavior have been found in wild house mice (Singleton and Hay, 1982), Anderson (1975) found a full-sib heritability of zero for voles. She did, however, find significant correlations between the mother's agonistic behavior and that of the offspring, again suggesting a maternal effect.

In 1982, we attempted to measure heritability of aggression by using standard dyadic encounters (same sex and age) under controlled conditions (see Krebs [1985] for a general description of the method) and compared the behavior of the adults with that of their progeny. The latter were tested at the end of their stay in the laboratory and when virtually all males and most females were in breeding condition. However, because we found little variation in the behavior of the progeny between litters, we abandoned this attempt. The relevant behaviors that should be investigated are probably those dealing with mate competition in males and nest-site defense in females (Boonstra, 1977, 1978; Redfield et al., 1978; Boonstra and Rodd, 1983; Ostfeld, 1985). Both of these may depend on learning, reproductive state (Svare, 1981), and conditions experienced in the field during

growth and development (for the type of factors which affect behavior in the house mouse [*Mus*] and which may operate in microtines as well, see Mackintosh [1981]), and we could not in any way replicate "natural" field conditions. We see this as the greatest obstacle to determining the heritability of biologically relevant aggression.

Together, the results of our explant experiment and half-sib heritability analysis suggest that maternal effects are major sources of phenotypic variation in the life-history characteristics examined. One of the potential complications with explant experiments is genotype-environment interaction. It would be desirable to obtain more information on the reaction norms for the characters we examined so as to eliminate the possibility that such interactions contributed to the differences observed under field and lab conditions, which we attributed entirely to environmental effects. Our results do not allow us to say that the absolute amount of additive genetic variance in these traits is low, merely that it is low relative to other causes of phenotypic variance. They suggest that some of the characteristics of microtine fluctuations, such as changes in body size, growth, and age at sexual maturity, may be effects rather than causes of these fluctuations. They do not provide strong support for the idea that individual differences in demographic characteristics are primarily genetic or that selection would be able to change the frequencies of low- versus high-density morphs sufficiently quickly to account for the times typical of microtine population changes. We recommend that similar studies be carried out on other microtines, especially lemmings (Krebs, 1979) and that particular attention be paid to the role of maternal effects in affecting viability of offspring.

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LITERATURE CITED

- ALBON, S. D., T. H. CLUTTON-BROCK, AND F. E. GUINNESS. 1987. Early development and population dynamics in red deer. II. Density-independent effects and cohort variation. *J. Anim. Ecol.* 56: 69–81.
- ANDERSON, J. L. 1975. Phenotypic correlations among relatives and variability in reproductive performance in populations of the vole *Microtus townsendii*. Ph.D. Diss. Univ. British Columbia, Vancouver, Canada.
- ANTLFINGER, A. E., W. F. CURTIS, AND O. T. SOLBRIG. 1985. Environmental and genetic determinants of plant size in *Viola sororia*. *Evolution* 39:1053–1064.
- ATCHLEY, W. R. 1984. Ontogeny, timing of development, and genetic variance-covariance structure. *Amer. Natur.* 123:519–540.
- BEACHAM, T. D. 1980. Growth rates of the vole *Microtus townsendii* during a population cycle. *Oikos* 35:99–106.
- BERVEN, K. A., AND D. E. GILL. 1983. Interpreting geographic variation in life-history traits. *Amer. Zool.* 23:85–97.
- BOAG, P. T., AND R. BOONSTRA. 1987. Quantitative genetics of life history traits in meadow voles (*Microtus pennsylvanicus*). In M. Boyce (ed.), *Population Biology and Life History Evolution of Mammals*. Yale Univ. Press, New Haven, CT. *In press*.
- BOAG, P. T., AND P. R. GRANT. 1981. Intense natural selection in a population of Darwin's finches (Geospisinae) in the Galápagos. *Science* 214:82–85.
- BOAG, P. T., AND A. J. VAN NOORDWIJK. 1987. Quantitative genetics, pp. 45–77. In F. Cooke and P. A. Buckley (eds.), *Avian Genetics—Its Application to Ecological and Evolutionary Theory*. Academic Press, London, U.K.
- BOONSTRA, R. 1977. Effects of conspecifics on survival during population declines in *Microtus townsendii*. *J. Anim. Ecol.* 46:835–851.
- . 1978. Effect of adult Townsend voles (*Microtus townsendii*) on survival of young. *Ecology* 59:242–248.
- . 1985. Demography of *Microtus pennsylvanicus* in Southern Ontario: Enumeration versus Jolly-Seber estimation compared. *Can. J. Zool.* 63: 1174–1180.
- BOONSTRA, R., AND C. J. KREBS. 1979. Viability of large- and small-sized adults in fluctuating vole populations. *Ecology* 60:567–573.
- BOONSTRA, R., AND F. H. RODD. 1983. Regulation of breeding density in *Microtus pennsylvanicus*. *J. Anim. Ecol.* 52:757–780.
- BUJALSKA, G. 1973. The role of spacing behavior among females in the regulation of reproduction in the bank vole. *J. Reprod. Fert. Suppl.* 19:465–474.
- CHEVERUD, J. M. 1984. Evolution by kin selection: A quantitative genetic model illustrated by maternal performance in mice. *Evolution* 38:766–777.
- CHITTY, D. 1952. Mortality among voles (*Microtus agrestis*) at Lake Vyrnwy, Montgomeryshire in 1936–9. *Phil. Trans. Roy. Soc. Lond. B* 236:505–552.
- . 1967. The natural selection of self-regulatory behaviour in animal populations. *Proc. Ecol. Soc. Aust.* 2:51–78.
- CLAUSEN, J., D. D. KECK, AND W. M. HIESEY. 1948. Experimental studies on the nature of species, III: Environmental responses of climatic races of *Achillea*. *Carnegie Inst. Wash. Publ.* 581:1–129.
- DINGLE, H., AND J. P. HEGMANN (eds.). 1982. *Evolution and Genetics of Life Histories*. Springer-Verlag, N.Y.
- DONNER, A., AND G. WELLS. 1986. A comparison of confidence interval methods for the intraclass correlation coefficient. *Biometrics* 42:401–412.
- EISEN, E. J., AND B. S. DURRANT. 1980. Genetic and maternal environmental factors influencing litter size and reproductive efficiency in mice. *J. Anim. Sci.* 50:428–441.
- EISEN, E. J., J. NAGAI, H. BAKKER, AND J. F. HAYES. 1980. Effect of litter size at birth on lactation in mice. *J. Anim. Sci.* 50:680–688.
- EL OKSH, H. A., T. M. SUTHERLAND, AND J. S. WILLIAMS. 1967. Prenatal and postnatal maternal influence on growth in mice. *Genetics* 57:79–94.
- ENDLER, J. A. 1986. *Natural Selection in the Wild*. Princeton Univ. Press, Princeton, NJ.
- FALCONER, D. S. 1981. *Introduction to Quantitative Genetics*. Longman, London, U.K.
- FINDLAY, C. S., AND F. COOKE. 1983. Genetic and environmental components of clutch size variance in a wild population of Lesser Snow Geese (*Anser caerulescens*). *Evolution* 37:724–734.
- GAINES, M. S. 1985. Genetics, pp. 845–883. In R. H. Tamarin (ed.), *Biology of New World Microtus*. Amer. Soc. Mammal., Stillwater, OK.
- GAINES, M. S., AND R. K. ROSE. 1976. Population dynamics of *Microtus ochrogaster* in eastern Kansas. *Ecology* 57:1145–1161.
- GYLLENSTEN, U. 1985. Temporal allozyme frequency changes in density fluctuating populations of willow grouse (*Lagopus lagopus* L.). *Evolution* 39:115–121.
- HANSSON, L. 1984. Composition of cyclic and non-cyclic vole populations: On the causes of variation in individuality among *Clethrionomys glareolus* in Sweden. *Oecologia* 63:199–206.
- HAYS, W. L. 1981. *Statistics*. Holt, Rinehart & Winston, N.Y.
- HEGMANN, J. P., AND H. DINGLE. 1982. Phenotypic and genetic covariance structure in milkweed bug life history traits, pp. 177–185. In H. Dingle and J. P. Hegmann (eds.), *Evolution and Genetics of Life Histories*. Springer-Verlag, N.Y.
- INNES, D. G. L., AND J. S. MILLAR. 1979. Growth of *Clethrionomys gapperi* and *Microtus pennsylvanicus*. *Growth* 43:208–217.
- IVERSON, S. L., AND B. N. TURNER. 1969. Under-snow shelter for small mammal trapping. *J. Wildl. Manag.* 38:722–723.
- KILPATRICK, W. W., AND K. L. CROWELL. 1985. Genetic variation of the rock vole, *Microtus chrotorrhinus*. *J. Mammal.* 66:94–101.
- KLEIN, T. W. 1974. Heritability and genetic correlation: Statistical power, population comparisons, and sample size. *Behav. Genet.* 4:171–189.

- KREBS, C. J. 1966. Demographic changes in fluctuating populations of *Microtus californicus*. Ecol. Monogr. 36:239-273.
- . 1978. A review of the Chitty Hypothesis of population regulation. Can. J. Zool. 56:2463-2480.
- . 1979. Dispersal, spacing behaviour, and genetics in relation to population fluctuations in the vole *Microtus townsendii*. Fortschr. Zool. 25:61-77.
- . 1985. Do changes in spacing behavior drive population cycles in small mammals?, pp. 295-312. In R. M. Sibly and R. H. Smith (eds.), Behavioral Ecology—Ecological Consequences of Adaptive Behaviour. Blackwell, Oxford, U.K.
- KREBS, C. J., AND R. BOONSTRA. 1984. Trappability estimates for mark-recapture data. Can. J. Zool. 62: 2440-2444.
- KREBS, C. J., AND J. H. MYERS. 1974. Population cycles in small mammals. Adv. Ecol. Res. 8:267-399.
- LANDE, R. 1982. A quantitative genetic theory of life history evolution. Ecology 63:607-615.
- LESLIE, P. H., J. S. PERRY, AND J. S. WATSON. 1945. The determination of the median body-weight at which female rats reach maturity. Proc. Zool. Soc. Lond. 115:473-488.
- LOMOLINO, M. V. 1984. Immigrant selection, predation, and the distributions of *Microtus pennsylvanicus* and *Blarina brevicauda* on islands. Amer. Natur. 123:468-483.
- MACKINTOSH, J. H. 1981. Behaviour of the house mouse, pp. 337-365. In R. J. Berry (ed.), Biology of the House Mouse. Academic Press, London, U.K.
- MONTEIRO, L. S., AND D. S. FALCONER. 1966. Compensatory growth and sexual maturity in mice. Anim. Prod. 8:179-192.
- MOORE, R. W., E. J. EISEN, AND L. C. ULBERG. 1970. Prenatal and postnatal maternal influences on growth in mice selected for body weight. Genetics 64:59-68.
- MORRISON, P., R. DIETERICH, AND D. PRESTON. 1977. Body growth in sixteen rodent species and subspecies maintained in laboratory colonies. Physiol. Zool. 50:294-310.
- MYERS, P., AND L. L. MASTERS. 1983. Reproduction by *Peromyscus maniculatus*: Size and compromise. J. Mammal. 64:1-18.
- NEI, M., T. MARAYMA, AND R. CHAKRABORTY. 1975. The bottleneck effect and genetic variability in populations. Evolution 29:1-10.
- NEWSON, J., AND D. CHITTY. 1962. Haemoglobin levels, growth and survival in two *Microtus* populations. Ecology 43:163-171.
- OSTFELD, R. S. 1985. Limiting resources and territoriality in microtine rodents. Amer. Natur. 126: 1-15.
- REDFIELD, J. A., M. J. TAITT, AND C. J. KREBS. 1978. Experimental alteration of sex ratios in populations of *Microtus townsendii*, a field vole. Can. J. Zool. 56:17-76.
- ROSE, M. R. 1982. Antagonistic pleiotropy, dominance, and genetic variation. Heredity 48:63-78.
- ROTH, V. L., AND M. S. KLEIN. 1986. Maternal effects on body size of large insular *Peromyscus maniculatus*: Evidence from embryo transfer experiments. J. Mammal. 67:37-45.
- SAITOH, T. 1981. Control of female maturation in high density populations of the red-backed vole, *Clethrionomys rufocanus* Bedfordiae. J. Anim. Ecol. 50:79-87.
- SAS INSTITUTE. 1982. SAS User's Guide: Statistics, 1982 Ed. SAS Inst., Inc., Cary, NC.
- SAUER, J. R., AND N. A. SLADE. 1986. Field-determined growth rates of prairie voles (*Microtus ochrogaster*): Observed patterns and environmental influences. J. Mammal. 67:61-68.
- SEBER, G. A. F. 1982. The estimation of animal abundance and related parameters, 2nd Ed. Griffin, London, U.K.
- SINGLETON, G. R., AND D. A. HAY. 1982. A genetic study of male social aggression in wild and laboratory mice. Behav. Genet. 12:435-448.
- SMOLEN, M. J., AND B. L. KELLER. 1979. Survival, growth, and reproduction of progeny isolated from high and low density populations of *Microtus montanus*. J. Mammal. 60:265-279.
- SOKAL, R. R., AND F. J. ROHLF. 1981. Biometry. Freeman, San Francisco, CA.
- STEARNS, S. C. 1977. The evolution of life history traits: A critique of the theory and a review of the data. Ann. Rev. Ecol. Syst. 8:145-171.
- . 1984. Heritability estimates for age and length at maturity in two populations of mosquitofish that shared ancestors in 1905. Evolution 38:368-375.
- STENSETH, N. C. 1981. On Chitty's theory for fluctuating populations. The importance of genetic polymorphism in the generation of regular density cycles. J. Theoret. Biol. 90:9-36.
- SVARE, B. B. 1981. Maternal aggression in mammals, pp. 179-210. In D. J. Gubernick and P. H. Klopfer (eds.), Parental Care in Mammals. Plenum, N.Y.
- TAITT, M. J., AND C. J. KREBS. 1981. The effect of extra food on small rodent populations: II. Voles (*Microtus townsendii*). J. Anim. Ecol. 50:125-137.
- . 1985. Population dynamics and cycles, pp. 567-620. In R. M. Tamarin (ed.), Biology of New World *Microtus*. Amer. Soc. Mammal., Stillwater, OK.
- TURNER, H. N., AND S. Y. YOUNG. 1969. Quantitative Genetics in Sheep Breeding. Macmillans, Melbourne, Australia.
- VAN NOORDWIJK, A. J. 1984. Quantitative genetics in natural populations of birds illustrated with examples from the great tit (*Parus major*), pp. 67-79. In K. Wöhrmann and V. Loeschcke (eds.), Population Biology and Evolution. Springer-Verlag, Berlin, W. Ger.

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