Maternal effects and additive genetic inheritance in the collared lemming *Dicrostonyx groenlandicus*

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Summary

We examined patterns of inheritance of size, growth and behavioural traits of collared lemmings (*Dicrostonyx groenlandicus*). Work was conducted on field-caught parents from the Canadian Arctic and their lab-born progeny. We partitioned inherited variance in traits into additive genetic and maternal effects components by using a half-sib breeding experiment in which each sire was mated to two dams. We found no evidence of statistically significant amounts of additive genetic variance in any of the traits measured. However, significant maternal effects were detected for several size- and growth-related traits. Three behavioural traits involving aggression, dispersal and activity showed no statistically significant inheritance of any kind. The presence of maternal effects may have consequences for population dynamics by causing 'lags' resulting in inappropriate phenotypes being produced under regimes of fluctuating selection pressure. We recommend that maternal effects should be investigated as a potential general cause of population cycles in small mammals.

Keywords: aggression; behaviour; Canadian Arctic; collared lemming; cycles; *Dicrostonyx groenlandicus*; dispersal; heritability; maternal effects; size

Introduction

Inheritance of traits can cause organisms to be mismatched with their environments by causing a lag between the phenotype of the organisms and their current environment (e.g. Chitty, 1967; Rossiter, 1991). Such lags can cause multi-annual population fluctuations (Caswell, 1972; May, 1976; Turchin, 1990; Royama, 1992), such as those occurring in microtines (lemmings and voles), snowshoe hares and some forest insects. Traditionally, the mechanism of inheritance that has been considered is additive genetic (Maynard Smith, 1989; Stearns, 1992; Boake, 1994). However, recent work has indicated that maternal inheritance (maternal effects) is a good candidate as a mechanism for introducing time lags into population dynamics (e.g. Rossiter, 1991, 1994; Ginzburg and Taneyhill, 1994). By maternal effects we mean the effects of parental phenotype on offspring phenotype that do not have a Mendelian genetic basis. Theoretical work (Kirkpatrick and Lande, 1989) has shown that maternal effects cause resemblance not just between parents and offspring, but between grandparents and grandoffspring, so maternal effects are a good candidate for causes of time lags between populations and their environments. While the presence of maternal effects in insects has been demonstrated several times (see Mousseau and Dingle, 1991, for a review) and their role in cyclic forest insect species has been investigated (Rossiter, 1994), little work has been done on other groups of animals that undergo cyclic changes in population size. In rodents, the existence of maternal effects in laboratory animals is known (e.g. Atchley and Newman, 1989).

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However, the presence of maternal effects in wild rodents, particularly for traits linked with fluctuating population dynamics, has received little attention (see Boonstra and Boag, 1987, for an exception).

Research on population dynamics of small mammals has a long history (e.g. Elton, 1924; Elton and Nicholson, 1942), with a large part of the research concentrated on microtine rodents (e.g. Krebs and Myers, 1974; Taitt and Krebs, 1985). For microtines, a wide range of potential proximate causes of cycling have been investigated, including predation (e.g. Erlinge *et al.*, 1983; Henttonen *et al.*, 1987; Hanski *et al.*, 1991), food (e.g. Krebs and Myers, 1974; Cole and Batzli, 1978; Desy and Thompson, 1983), stress (Christian, 1980), social environment (e.g. Krebs, 1985; Heske *et al.*, 1987; Lambin and Krebs, 1991), age structure (Boonstra, 1994), genetic variation at or near individual loci (e.g. Gaines, 1985) and heterozygosity (e.g. Smith *et al.*, 1975; Boonstra *et al.*, 1994). Others have advocated examining multiple factors simultaneously by testing for synergisms among factors as a cause of population cycles (e.g. Lidicker, 1988; but see Gaines *et al.*, 1991). Boonstra and Boag (1987) tested the Chitty hypothesis (Chitty, 1967) on the meadow vole (*Microtus pennsylvanicus*), a species which has shown cyclic fluctuations in some, but not all, of the populations (reviewed in Taitt and Krebs, 1985). 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. However, Boonstra and Boag (1987) found that most of the variation in a series of life-history traits were non-genetic in origin and that maternal and other environmental effects were of overriding importance. They concluded that heritabilities were too low for natural selection to operate in the time frames characteristic of microtine cycles. These data suggest that maternal effects are more likely than additive genetic inheritance to produce the cross-generation effects needed to produce population cycles. However, one component of Chitty's hypothesis remains to be tested: the presence of heritability of behaviour, either through additive genetic means or through maternal effects.

In this study, we tested for the presence of maternal effects in a captive population of collared lemmings (*Dicrostonyx groenlandicus*) in which the parents came from the wild and the young were born in the lab. Lemmings are the historical archetypical cyclic species, regularly reaching peak numbers every 3–4 years and then declining dramatically (Shelford, 1943; Krebs, 1964; Stenseth and Ims, 1993; Chitty, 1996). Our experimental design allowed us to test simultaneously for additive genetic and maternal effects and to compare the magnitudes of each form of inheritance. There were two main objectives to the study. First, we tested for the inheritance of behaviour. Second, we tested whether the high maternal effects in key life-history characteristics were present in a microtine species other than *M. pennsylvanicus*. This was the first study to investigate the maternal inheritance of behaviour together with key life-history traits in a wild mammal. The magnitudes of maternal effects are contrasted with narrow-sense heritability of these same traits, measured from the same half-sib breeding experiments. We show little additive genetic basis for variation in the traits that we measured. Behaviour was largely not under the influence of maternal effects, but up to 80% of variation in some growth and size traits was the result of maternal effects. These results suggest that maternal inheritance is a better candidate than additive genetic inheritance as a cause of population cycles. Further, it appears that if inheritance of traits causes fluctuations in population size, then life-history traits, and not behaviours, are the important traits in the process. Non-Mendelian inheritance should be considered in future studies investigating the causes of population cycles in microtines.

Methods

The lemmings used in this study came from Pearce Point, on the coast of the Beaufort Sea in the Northwest Territories, Canada (Krebs *et al.*, 1995). Lemmings were captured in the summers of 1987 and 1988 with Longworth live-traps; active burrows were identified in 1988 using the powdered slide technique of Boonstra *et al.* (1995). Each year we attempted to capture 100 lemmings that were either overwintered or young-of-the-year to transport them back to the Scarborough Campus, University of Toronto rearing facility. Animals were captured over a 20 km² area in a range of tundra habitats to minimize the possibility of sampling-related lemmings. The demography of this population was monitored by intensive live-trapping from June to August, 1987–89. The populations appeared to be in a phase of continuous increase, with reproduction occurring over winter and in spring before snowmelt, resulting in numbers often being highest in spring (0.7–1.5 ha⁻¹) and declining over summer despite continuous breeding. Predation from a variety of avian and mammalian predators was intense during the summer (Boonstra *et al.*, 1990; Krebs *et al.*, 1995; Reid *et al.*, 1995), accounting for much of this decline. Hence, the lemmings obtained for this study were not obtained from populations showing cyclic characteristics over the time period we were present. Nevertheless, the examination of heritability of lemmings from this area allowed us to determine whether there was sufficient additive genetic variance for selection to act and produce the characteristics associated with cyclic populations or whether maternal effects played a major role in accounting for variance.

All behavioural and breeding experiments were conducted in the laboratory. Lemmings were kept at between 10 and 16°C, on a light:dark cycle of 22:2 h. Water and food (Purina Mills Lab Chow and Purina Mills Guinea Pig chow) were provided *ad libitum*, and apples were provided weekly. All mated pairs were housed in 51 × 41 × 22 cm polycarbonate cages and the young were housed individually in 48 × 27 × 16 cm polypropylene cages on a bedding of hardwood chips and given cotton nesting material. We mated each male with two females and, to minimize inbreeding, chose females from areas other than those from which males had been trapped. Pairs were separated 10 days after mating. Males (both wild-caught and their progeny) were tested in aggressive encounters with each other immediately after they were separated from the female; second matings followed 7–14 days after the behavioural test. Male opponents were approximately equal in body weight (± 7 g). Females' behaviour in dyadic encounters were tested when they were still lactating and their young were 10–15 days old; again both wild-caught females and their progeny were tested at this time. Female opponents were not lactating and weighed up to 20 g less than the lactating females. Young were removed from their mother's cage when 25 days old. Activity tests (static and exploratory behaviour) were conducted 7 days after removal of young (wild-caught females), 7 days after separation from the female (wild-caught males), or 7 days after separation from the mother (offspring).

Data on growth rate and maturation were collected on a weekly basis for all lab-born offspring. These data can be divided into two classes: size and rate of development. The size data gathered were: mass at weaning, mass at sexual maturity, mass at 50 days of age, maximum mass and growth rate. The data describing rate of development were: age at weaning, age at sexual maturity and date of maximum mass. Reproductive maturity in males was defined as presence of scrotal testes and in females by a perforate vagina. Growth was defined as the rate in grams per day put on by the lemming between weaning and day 50 of life. For those young not actually weighed on day 50, weight at day 50 was the linear interpolation based on the known weight before and after that age. Classification of variables into development rate and mass-related groups is not arbitrary: the mass variables are all highly intercorrelated, as are the rate-of-development variables. However, correlations between individual rate-of-development and mass variables were smaller than corre-

lations within each of these groups. The only exception was growth rate, which tended to be poorly correlated with other variables. To analyse the data from all offspring together, we had to eliminate the effects of a number of potential confounding variables. The potential confounding variables were: sex of offspring, number of offspring born into a litter, number weaned from a litter, julian date of birth (both linear and quadratic effects), year of mother's capture and maximum age. Maximum age was a confounding variable and not a dependent variable in our analysis because all lab-born lemmings were sacrificed at the end of June of the year following birth; correlations of other variables with maximum age are an artifact of this truncation. Number born and number weaned were almost perfectly correlated ($r > 0.98$), so of these two, only a single confounding variable, number weaned, was examined. Potential confounding variables were screened simultaneously, along with all second- and third-order interactions among variables. Each of our mass and development-rate variables was affected by at least one of the potential confounding variables, and in some cases second- and third-order interactions were present. We corrected for all statistically significant confounding variables by taking the residuals from general linear models incorporating only the significant confounding variables. The residuals became our standardized measures of mass and development rate. In addition, effects on offspring of mother's mass were noted but not corrected for, as mother's mass might be responsible for maternal effects.

We also gathered data on litter size and sex ratio within litters. We did not calculate additive genetic variance and heritability of litter size and sex ratio, because we had at most two data points (i.e. litters) for each parent. These small sample sizes rendered our approach to calculating heritabilities (see below) impossible. However, we were able to calculate an overestimate of the broad-sense heritability, by calculating repeatabilities of litter size and sex ratio in litters of individual sires.

Aggression was measured in dyadic encounters between lemmings. All pairs of lemmings were of the same sex. Before each encounter, both lemmings were weighed and their reproductive condition was assessed. Animals were then placed in a 30.5×61 cm wooden box fronted by a one-way-view mirror and with a sliding wooden partition which divided the arena in half. One lemming was termed the resident and bedding from its cage was placed on the floor of the arena on both sides of the divider. Animals were allowed to habituate to the arena for 5 min before the partition was raised. The trial was allowed to continue for 10 min unless one lemming appeared in danger of receiving mortal injury, in which case the trial was stopped and the time recorded. The arena was cleaned between every trial. Behaviours recorded (based on Allin and Banks, 1968) were: approach, retreat, chase, attack, tumble, boxing, offensive, defensive, dig, mount, groom and vocalize. The number of separate bouts of each behaviour was recorded. When bouts were terminated early, counts of behaviours were extrapolated to their total numbers in a 10 min period by assuming that proportions would remain unchanged for the entire period if interactions had been allowed to continue.

We summarized our observations using an index of aggressive behaviour based on principal components analysis (PCA). The PCA was based on a correlation matrix of the initial variables. *A priori* we decided that the behaviours most directly related to aggression were approach, retreat, offensive and defensive. Thus any principal component describing aggression should show the pair's approach and offensive behaviour to increase together, and also show retreat and defensive behaviour to increase together; approach and offensive behaviour should be negatively correlated with the other two key variables. Non-rotated principal component axes failed to meet these criteria, but the first principal component following varimax rotation matched our criteria. This component explained 17.1% of total variance in the observed behaviours.

Levels of open-field, or non-dispersive, activity were measured using an activity monitor (Stoelting Co., Chicago, IL). These activity monitors emit a resonating radio frequency which is disrupted when the lemming moves and each disruption is tallied. Four monitors were used si-

multaneously and their sensitivities were standardized by means of a metronome (though there were monitor-dependent differences which had to be removed statistically; see below). A lemming was placed inside a $48 \times 27 \times 16$ cm polypropylene cage which was set on the activity monitor. Trials were conducted in an isolated room, and extraneous visual or auditory stimuli were avoided during trials. Animals were allowed to acclimatize for 5 min before the recorder was turned on. Trials lasted for 15 min, and we attempted to test each lemming three times. Repeated trials of the same lemming were conducted on different days.

Exploratory or dispersive activity was measured by recording activity of lemmings in a long narrow horizontal dispersal maze. The apparatus consisted of a small habituation chamber ($13 \times 12.5 \times 10$ cm) enclosed completely in black plexiglass with a removable lid and a maze ($182 \times 13 \times 10$ cm) with black plexiglass walls and a clear, removable plexiglass roof, above which hung a fluorescent light. The habituation cage was separated from the maze by a door that could be opened remotely. At every 30 cm along the length of the maze were alternating baffles (5×12 cm), which prevented the lemmings from seeing to the end of the maze. At the entrance of the maze and at the five baffles were placed photoelectric sensors which were tripped when a lemming passed that point. Sensors were connected to a data logger (Apple IIe) which recorded the times of first and last triggering of each sensor and how many times each sensor was triggered. The floor of the maze was covered by a sheet of Kraft paper which was waxed on one side. After each trial, this paper was replaced and the habituation chamber was washed with soapy water. For each trial, an animal was weighed and reproductive condition assessed. The lemming was then placed in the cage at the end of the passage and allowed to acclimatize for 5 min. The door was then opened and the trial was allowed to proceed for 15 min. No extraneous visual or auditory stimuli were allowed during the trials. We attempted to test each lemming twice, with trials on separate days. Trials were conducted between 08.00 and 17.00 h, at an ambient temperature of 15.5°C .

Data collected from each trial were times of first and last triggering of each photocell, and the total number of times that any light beam was interrupted. These data were summarized in a simple index of exploratory behaviour with PCA. The PCA was conducted on a correlation matrix of the data and axes were not rotated. Multiple observations for each individual were represented as separate data points in the PCA. *A priori* we expected that a highly active animal would be quick to cross each photocell in the passage, would remain active throughout the entire trial (have a late last time of crossing of each photocell), and have a high total number of interruptions of photocells. The principal component chosen as our index of exploratory activity explained 31% of all variance in the data set.

We examined whether our three indices of behaviour (aggression, non-dispersive and exploratory) varied with sex and reproductive condition of the lemmings. Additionally, we tested whether aggression was affected by differences in mass and reproductive condition of the paired animals, and whether static and exploratory behaviour changed systematically between trials for each individual. We tested whether our measure of static activity was affected by differences between platforms on the activity monitor. For animals of known age (i.e. only captive-born lemmings), we also tested whether age affected behaviour. Aggression was found to vary between sexes, and was affected by differences in mass and reproductive condition of opponents. Static activity was affected by reproductive condition, sex, trial number and the sensor platform used to conduct measurements. Exploratory activity varied between sexes, but was not affected by reproductive condition. Where behaviours were found to vary with tested factors, the effects of these factors were statistically removed by either subtracting categorical means from individual data points or taking residuals from linear regression (for difference in mass, the only continuous variable examined). Little interaction between factors was found, so effects were removed singly, in the following order: reproductive condition (or difference therein between opponents), sex, difference in mass and trial

number. For static and exploratory behaviour we were able to calculate repeatability of behaviour within individuals (e.g. Lessells and Boag, 1987; Sokal and Rohlf, 1981, p. 216), because each animal was measured more than once, whenever possible. Repeatability was 57.7% for non-dispersive behaviour and 18.8% for exploratory behaviour.

Phenotypic variance for the above factors was partitioned into components resulting from sires, dams and progeny and from these heritabilities were calculated using the methods outlined in Falconer (1989) and summarized in Boonstra and Boag (1987). Both narrow-sense heritabilities and maternal effects could be calculated using our half-sib experimental design. Briefly, we used a two-level nested Model II ANOVA from the PROC NESTED routine (SAS Institute Inc., 1988) to calculate components of variance that were due to fathers and mothers. Heritability was calculated as four times the proportion of variance attributed to fathers (Falconer, 1989, p. 170). Maternal effects were calculated as the difference between maternal and paternal mean squares. This value includes one-quarter of the additive genetic variance, one-quarter of the non-additive dominance variance, and the environmental variance due to between-litter environment effects. In cases in which the additive genetic variance is essentially equal to zero (an estimate of which can be obtained from the males), and assuming that the dominance variance is equal to zero (Falconer, 1989, pp. 170–172), then all we are left with is the between-litter (i.e. between-dam or maternal) effects. Significance of heritabilities was calculated as the ratio of paternal mean squares to maternal mean squares calculated in the nested ANOVA. Because of unequal numbers of offspring for each male, Satterthwaite's correction (Sokal and Rohlf, 1981, pp. 296–301) was employed in testing statistical significance of heritabilities. Statistical significance of maternal effects was calculated as the ratio between maternal and error (within-litter) mean squares. Standard errors were approximated following Turner and Young (1969). Other statistical analyses were conducted using either SAS (SAS Institute Inc., 1988) or SYSTAT (Wilkinson, 1988).

Results

Growth and sexual maturity

There was little difference between years in the average values of size- and growth-related traits (Table 1); hence, we combined data from the 2 years in our analyses. Our measures of growth and maturity were not statistically independent of each other (Table 2), as noted in the Methods section. Instead, all measures of mass (mass at weaning, mass at maturity, day 50 mass and maximum mass) were significantly correlated with each other. In all cases, a higher mass at one age was related to a higher mass at another age. Also, variables related to maturation (weaning age, age at maturity and age at maximum mass) tended to be intercorrelated. Lemmings that were weaned at an early age tended to reach their maximum mass at an earlier age. Growth rate correlated with some variables in both 'size' and 'growth' categories. Hence, significant heritabilities or maternal effects on any one of the 'growth' or 'size' variables will suggest a similar effect for all related variables.

None of the variables measuring growth and maturity displayed a statistically significant heritability (Table 3). Further, the majority of heritabilities for either sex were estimated to be zero. From these results it appears that the additive genetic basis for size and rate of maturity was relatively small in these lemmings.

In contrast, there was considerable maternal determination of size and rate of maturity (Table 3). Of the four mass-related traits, only maximum mass exhibited no statistically significant maternal effect. However, maternal effects were relatively large, and given the intercorrelation of all mass traits (Table 2), maximum mass is also likely to be influenced by maternal effects. Two of the

Table 1. Variation in life-history parameters between years

Trait	1987			1988			P
	Mean $\pm$ S.D.	n		Mean $\pm$ S.D.	n		
Dam							
Litter size	3.5 $\pm$ 1.2	25		3.8 $\pm$ 1.4	47		0.43
Sex ratio	1.5 $\pm$ 0.3	31		1.5 $\pm$ 0.3	45		0.56
Dam's mass	47.0 $\pm$ 6.4	29		46.3 $\pm$ 8.3	45		0.68
Female offspring							
Mass at weaning	30.9 $\pm$ 5.9	56		29.1 $\pm$ 5.4	94		—
Mass at maturity	35.2 $\pm$ 14.6	56		30.2 $\pm$ 16.3	94		—
Maximum mass	59.4 $\pm$ 13.5	56		60.1 $\pm$ 14.8	94		—
Day 50 mass	40.5 $\pm$ 7.9	56		37.1 $\pm$ 8.9	94		—
Growth rate	0.39 $\pm$ 0.2	56		0.36 $\pm$ 0.2	94		—
Age at weaning	25.3 $\pm$ 0.5	56		25.1 $\pm$ 2.9	94		—
Age at maturity	58.9 $\pm$ 50.2	51		51.8 $\pm$ 41.3	79		—
Age of max. mass	159.5 $\pm$ 42.4	51		156.1 $\pm$ 61.8	85		—
Male offspring							
Mass at weaning	31.6 $\pm$ 5.4	50		28.8 $\pm$ 4.3	92		—
Mass at maturity	44.9 $\pm$ 11.4	50		34.3 $\pm$ 23.2	92		—
Maximum mass	59.1 $\pm$ 11.2	50		55.7 $\pm$ 10.5	92		—
Day 50 mass	41.3 $\pm$ 7.3	50		37.9 $\pm$ 7.4	92		—
Growth rate	0.4 $\pm$ 0.2	50		0.4 $\pm$ 0.2	92		—
Age at weaning	25.4 $\pm$ 0.5	50		25.0 $\pm$ 0.9	92		—
Age at maturity	96.0 $\pm$ 35.4	48		116.3 $\pm$ 45.3	66		—
Age of max. mass	140.62 $\pm$ 44.9	34		115.7 $\pm$ 56.3	68		—

P-values are from one-way ANOVAs. Statistical tests were not performed on data from offspring because data are the values calculated from all individual lemmings at data points, but offspring within a litter are not statistically independent of each other.

four age-related traits, age of weaning and sexual maturity, showed statistically significant maternal effects. In contrast, growth rate and age of maximum mass had calculated values for maternal effects that were extremely small in both male and female offspring, and none approached statistical significance.

The analyses used to detect maternal effects (Table 3) do not tell us whether mothers and offspring resembled each other (positive maternal effects) or were the opposite of each other (negative maternal effects). Negative maternal effects are commonly found (e.g. Cothran *et al.*, 1983; Hansson, 1978; Janssen *et al.*, 1988), including larger than average mothers tending to produce smaller than average offspring in rodents (e.g. Falconer, 1965; Millar, 1983). The only trait for which data were gathered from mothers was maximum (non-pregnant) mass. In a plot of offspring mass against mother's mass, we can assume that any resemblance is due to maternal effects, given that no additive genetic variance was detected for this trait. Mothers and offspring tended to resemble each other, indicating positive maternal inheritance of mass (Fig. 1).

Behavioural variation

Our indices of behaviour do not measure entirely independent aspects of lemmings' activity. The largest correlation between any two indices was highly significant, even after correction for mul-

Table 2. Correlations among measures of mass and rate of maturity

	Weaning mass	Mass at maturity	Maximum mass	Day 50 mass	Growth rate	Age at weaning	Age at maturity	Date of max. mass
Weaning mass	—	0.27 (0.001)	0.45 (< 0.001)	0.64 (< 0.001)	0.04 (0.60)	0.04 (0.62)	-0.07 (0.38)	-0.16 (0.057)
Mass at maturity	0.36 (< 0.001)	—	0.34 (< 0.001)	0.41 (< 0.001)	0.26 (0.002)	0.18 (0.024)	0.69 (< 0.001)	0.02 (0.78)
Maximum mass	0.50 (< 0.001)	0.58 (< 0.001)	—	0.56 (< 0.001)	0.32 (< 0.001)	-0.01 (0.90)	0.08 (0.32)	0.26 (0.001)
Day 50 mass	0.60 (< 0.001)	0.40 (< 0.001)	0.57 (< 0.001)	—	0.67 (< 0.001)	0.19 (0.02)	0.06 (0.48)	-0.10 (0.21)
Growth rate	-0.07 (0.40)	0.17 (0.04)	0.29 (0.001)	0.69 (< 0.001)	—	0.36 (< 0.001)	0.10 (0.24)	-0.28 (0.001)
Age at weaning	0.17 (0.04)	0.10 (0.22)	0.16 (0.065)	0.14 (0.095)	0.09 (0.27)	—	0.09 (0.29)	-0.59 (< 0.001)
Age at maturity	0.04 (0.64)	0.80 (< 0.001)	0.25 (0.003)	0.07 (0.43)	-0.01 (0.95)	-0.07 (0.40)	—	0.09 (0.27)
Date of max. mass	-0.06 (0.50)	0.19 (0.026)	0.24 (0.004)	-0.09 (0.29)	-0.17 (0.043)	-0.01 (0.92)	0.31 (< 0.001)	—

Values are Pearson correlation coefficients, with probabilities given in parentheses. Correlations among females are presented above the diagonal, and correlations among males are given below the diagonal. For all correlations among females the sample size is 150 lemmings, and for males the sample size is 142 animals.

Table 3. Heritability (h^2) and maternal influence on growth parameters of offspring

Trait	Offspring's sex	Offspring number	% Total variance			$h^2 \pm SE_{h^2}$ Sire	P_{sire}	Maternal h^2	P_{dam}
			Sire	Dam	Litter				
Mass at weaning	Male	111	0.0	63.6	36.4	0.0 $\pm$ 0.14	0.48	0.63	< 0.001
	Female	125	0.0	81.7	18.3	0.0 $\pm$ 0.13	0.91	0.82	< 0.001
Mass at maturity	Male	78	0.0	44.1	55.9	0.0 $\pm$ 0.32	0.67	0.44	0.045
	Female	117	5.9	18.4	75.7	0.24 $\pm$ 0.33	0.39	0.13	0.14
Maximum mass	Male	78	0.4	34.1	65.5	0.0 $\pm$ 0.32	0.52	0.34	0.10
	Female	117	0.0	26.6	73.4	0.0 $\pm$ 0.31	0.65	0.27	0.07
Day 50 mass	Male	78	0.0	35.4	64.6	0.0 $\pm$ 0.32	0.63	0.35	0.097
	Female	117	0.0	48.6	51.4	0.0 $\pm$ 0.31	0.64	0.49	0.002
Growth rate	Male	111	0.0	18.0	82.0	0.0 $\pm$ 0.15	0.57	0.18	0.17
	Female	125	0.0	0.0	100.0	0.0 $\pm$ 0.14	0.58	0.0	0.63
Age at weaning	Male	78	12.8	51.4	35.8	0.5 $\pm$ 0.37	0.33	0.39	0.006
	Female	117	0.0	79.4	20.6	0.0 $\pm$ 0.31	0.99	0.79	< 0.001
Age at maturity	Male	78	0.0	57.9	42.1	0.0 $\pm$ 0.40	0.96	0.58	0.007
	Female	117	0.7	50.0	49.3	0.03 $\pm$ 0.37	0.45	0.49	0.001
Age of max. mass	Male	78	0.0	0.0	100.0	0.0 $\pm$ 0.46	0.63	0.0	0.76
	Female	117	5.5	0.0	94.5	0.22 $\pm$ 0.35	0.30	0.0	0.87

Data presented separately by sex. Thirty-five sires were represented for data from male offspring and 36 sires for data from female offspring, all except for the traits mass at weaning and growth rate, for which data were available from 37 sires (both sexes of offspring). Fifty-two dams (male offspring) or 62 dams (female offspring) were represented for all traits other than mass at weaning and growth rate; data from 64 dams were available for these traits.

Figure 1. Relationship between mother's mass (g) and mass of female offspring (g). Data from mothers and offspring have been standardized to a mean of zero. The line indicates predicted linear regression, demonstrating a positive maternal inheritance of mass.

multiple comparisons (Table 4). However, even this correlation indicated that under 7% of variation in female static activity was related to variation in exploratory behaviour. We interpret these results to indicate that we can treat analyses of the three behavioural indices as being relatively independent of each other.

We found no significant heritabilities (Table 5). Three of the six calculated heritabilities were zero, and in none of the traits did both male and female progeny simultaneously have a calculated heritability of greater than zero. Hence, if there is an additive genetic basis for any of the behavioural traits that we measured, the heritability would be extremely small. In contrast, there was one significant maternal effect (Table 5): the static activity of male offspring varied statistically among mothers. However, as with heritabilities, a trait that showed a positive maternal effect for

Table 4. Correlations between behavioural indices

	Aggression	Non-dispersive	Exploratory
Aggression	—	0.13 (0.10) 175	0.05 (0.54) 170
Non-dispersive	0.12 (0.12) 165	—	0.26 (0.0002) 203
Exploratory	0.10 (0.20) 162	0.11 (0.14) 171	—

Correlations calculated separately for males and females. Correlations above the diagonal are for females, and below the diagonal for males. Data are presented as r (P) n .

Table 5. Heritability (h^2) and maternal influence on lemming behaviour

		%Total variance			$h^2 \pm SE_{h^2}$	n_{sire}	P_{sire}	Maternal		
		Sire	Dam	Litter				h^2	n_{dam}	P_{dam}
Aggression	Male	2.8	0.0	97.2	0.11 ± 0.34	36	0.41	0.00	61	0.80
	Female	0.0	15.5	84.5	0.00 ± 0.38	34	0.54	0.16	58	0.23
Non-dispersive	Male	0.0	32.5	67.5	0.00 ± 0.30	36	0.48	0.33	61	0.02
	Female	7.9	0.0	92.1	0.32 ± 0.34	33	0.31	0.00	58	0.60
Exploratory	Male	2.4	0.0	97.6	0.10 ± 0.32	36	0.44	0.00	61	0.66
	Female	0.0	7.4	92.6	0.00 ± 0.32	33	0.75	0.07	58	0.36

Data are presented separately for each sex.

one sex of offspring possessed an effect of zero for the other sex. Thus although there is some evidence for a non-genetic maternal influence on behaviour, the evidence is slight.

Litter size and sex ratio

We could not directly estimate additive genetic inheritance of litter size and sex ratio of offspring; however, we did calculate whether parents produced litters of consistent size or sex ratio. This was done by calculating the repeatability of litter size within individual parents (Lessells and Boag, 1987; Sokal and Rohlf, 1981, p. 216). Sex ratio within a litter was indexed by assigning values of 1 to each male offspring and 2 to each female. Summing these values within a litter and averaging produced an index of sex ratio. We found no clear evidence for an inherited basis for either trait: litter size had a repeatability of 5.3% for sires and 17.6% for dams, and the repeatability of sex ratio was 0% for both sires and dams.

Discussion

We found maternal effects on size and maturation traits (Table 3) but not on most behavioural traits (only non-dispersive behaviour in males was significant, Table 5); none of the variables measuring growth and maturity displayed a statistically significant amount of additive genetic variance. Both the size (e.g. Millar, 1983; Boonstra and Boag, 1987; Atchley and Newman, 1989) and behaviour (e.g. Chevalet and le Pape, 1989) of small rodents can be affected by maternal effects. Lack of maternal influence on behaviour may have resulted from our experimental design: mothers' environments were identical during pregnancy and weaning of offspring. It is well known that maternal experience during pregnancy and lactation influence progeny development and performance in mammals (e.g. in laboratory mammals: Pollard, 1986; Takahashi *et al.*, 1990; Henry *et al.*, 1994; in wild mammals: Albon *et al.*, 1987; McRoberts *et al.*, 1995). In insects, maternal effects have been clearly demonstrated when mothers' environments were altered during the time that eggs or offspring were developing (e.g. Rossiter, 1991), and a recent review (Mousseau and Dingle, 1991) suggested that most maternal effects in insects are due to influences on mothers late in their offspring's development.

Our study may have underestimated the size of maternal effects occurring in nature if important maternal effects are produced during development and weaning of offspring. Hence failure to detect maternal effects on behaviour only indicates that lack of variation in mothers' environments in the lab prior to conception may result in no effect on offspring behaviour. This potential bias may also

have resulted in an underestimate of the influence of maternal effects on growth and size which could occur in the field. While the common laboratory environment offspring experienced may have prevented us from detecting some maternal effects, our experimental design provides us with confidence that the variation observed among families was not merely the result of direct environmental effects on offspring, but rather that the variation was related to differences among mothers owing to experiences carried with them from the field. Rearing conditions in the lab were kept as constant as possible among cages. Admittedly, offspring of a dam did share a common environment while with their mother, but at this time much of their nutrition was provided by their mothers and so subtle variation in rearing conditions among dams are likely to be transmitted to offspring through their mothers (i.e. these are maternal effects) if the effects we detected are nutritionally based. Another potential source of non-maternal variation among families was litter size, which was correlated with most offspring traits (unpublished analyses); however, effects of variation in litter size were removed statistically before heritabilities and maternal effects were calculated. Finally, the fact that we did find maternal effects under conditions of 'low environmental input' (Rossiter, 1994) suggests that these effects would provide even more of a lag between phenotype and environment than would be found for maternal effects that are simply responses to environmental conditions just before or after offspring's birth.

There was little evidence of narrow-sense heritability of growth, size, behaviour and litter size, and sex ratio, in our lemmings (Tables 3 and 5). This is not to say that there was no additive genetic variance for these traits, but only that it was small relative to large environmental contributions to the phenotypic variance. Size, growth (e.g. Atchley and Newman, 1989) and behavioural (e.g. Rasmuson *et al.*, 1977; Walker and Byers, 1991; but see Waser and Jones, 1989) traits have all been shown to have some genetic basis in small mammals. However, lack of statistically detectable heritability merely indicates that the additive genetic component was small, and probably incapable of driving population cycles. The additive genetic component may be small for two reasons. First, our wild-born lemmings came from Pearce Point, a coastal site where lemming populations appeared to be in a phase of continuous increase, possibly because of high predation pressure and extremely patchy vegetation (Krebs *et al.*, 1995; Reid *et al.*, 1995). These conditions may have resulted in animals with low additive genetic variance for traits normally associated with cycles. We think this unlikely, as our coastal site was within 10 km (inland) of good-quality habitat where cycles were much more probable (C.J. Krebs, personal communication). Given the dispersive capabilities of lemmings, it is unlikely that our populations were isolated genetically from these adjacent populations. Second, additive genetic variance may actually be low in all lemmings and our findings are indicative of cyclic populations as well. Given that the only other study on wild microtines that simultaneously measured maternal effects and additive genetic inheritance (on a cyclic meadow vole population: Boonstra and Boag, 1987) also found negligible additive genetic variance for growth and size traits, we suggest that the second explanation may be correct and that our results reinforce the conclusions of that study.

While maternal inheritance as a cause of population cycling in insects is receiving growing attention (e.g. Rossiter, 1991; Ginzberg and Taneyhill, 1994), there has been little recent consideration of the role of maternal effects in mammalian cycles (but see Chitty, 1952; Hausson, 1984; Boonstra, 1985; Mihok and Boonstra, 1992; Boonstra, 1994). Clearly, maternal effects are not the only forces which could potentially introduce lags needed for cycling to occur (e.g. delayed density-dependent predation is an obvious alternative; Hanski *et al.*, 1991). Future research on microtines should determine if maternal effects are even more prominent when variation is present in mothers' environments during ontogeny of offspring. The presence and size of maternal effects that we detected in this study suggest that maternally inherited traits, especially those dealing with life-

history traits rather than those dealing with behaviour, are much more viable than additive genetic traits as potential explanations of complex population dynamics.

Although we were able to measure the sizes of maternal effects, we could not determine their direction in most cases. Aside from offspring mass (Fig. 1), we do not know whether the offspring resembled their mothers (positive maternal effects) or tended to be the opposite of their mothers (negative effects). This information can influence the way that maternal effects affect population dynamics. Positive maternal effects will act similarly to additive genetic effects, whereas negative effects may produce offspring maladapted for the environmental conditions for which their mothers were selected. The prevalence and consequences of negative maternal effects in small mammals deserve further study.

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