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Quantitative genetics and the evolution of ontogeny

I. Ontogenetic changes in quantitative genetic variance components in randombred mice

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SUMMARY

We report the results of an ontogenetic analysis of quantitative genetic variance components with two replicates drawn from the randombred ICR strain of mice. A total of 432 mice from 108 full-sib families raised in a cross-fostering design were used to estimate direct effects heritability, maternal effects, and environmental effects for weight, head length, trunk length, trunk circumference, and tail length at 17, 24, 31, 38, 45, 52, 59, and 66 days of age. There was no significant difference in heritability between the replicates. Heritabilities either stayed more or less constant with age at about 0·30 (weight, trunk length, trunk circumference) or increased slightly with age (head length, tail length). Maternal effects decreased with age from a maximum of about 0·50 at weaning to about 0·15 at age 66 when growth was nearly complete. Environmental effects increased in relative importance during ontogeny.

1. INTRODUCTION

Recent treatments of evolutionary theory have stressed the role of the life-cycle or ontogeny in evolutionary response to selection (Bonner, 1974; Gould, 1977; Alberch *et al.* 1979; Riedl, 1979; Lande, 1982). While selection may act at a particular stage of the life-cycle such as birth, weaning, age at first reproduction, and adulthood, the evolutionary response to this selection always involves changes in the life-cycle or growth curves of the traits. Unfortunately, few authors examining the evolution of ontogeny have considered genetic factors (e.g. Bonner, 1974; Gould, 1977; Alberch *et al.* 1979). Little empirical work has been done on the morphogenetic and evolutionary consequences of the patterns of genetic variation and covariation in ontogeny outside of agricultural genetics.

In this paper we examine ontogenetic changes in variance components associated with direct genetic, maternal, and non-maternal environmental effects for five live

body traits in mice. Most previous studies of the ontogeny of variance components have been restricted to single characters, such as weight. In order to determine whether the ontogenetic patterns discovered for weight can be generalized to other live body traits, it is necessary to analyze a battery of traits in the same sample. Future work in this area will include extensive genetic analyses of univariate, bivariate, and multivariate growth curves and age-related changes in covariance patterns from an evolutionary perspective.

Table 1. *Sources of variation and expected mean squares for analysis of variance*

(V_d is the dam variance, V_n is the nurse variance, V_{dn} is the dam by nurse interaction variance, and V_R is the residual variance.)

Source	Expected mean square
Dams within pairs	$V_R+2V_{dn}+4V_d$
Nurses within pairs	$V_R+2V_{dn}+4V_n$
Dams × nurses within pairs	V_R+2V_{dn}
Residual	V_R

Previous studies of ontogenetic variation in heritability, maternal effects, and environmental effects for weight in mice indicate that heritabilities either increase or exhibit no linear trend with age (Jara-Almonte & White, 1973; Young, Legates & Farthing, 1965; Herbert, Kidwell & Chase, 1979; Monteiro & Falconer, 1966; Rutledge *et al.* 1972) while maternal effects uniformly decrease with age (Young *et al.* 1965; Herbert *et al.* 1979; El Oksh, Sutherland & Williams, 1967; Rutledge *et al.* 1972). In perhaps the most comprehensive study of growth changes in variance components, Rutledge *et al.* (1972) found no significant linear relationship between age and the heritability of weight. Guinea pigs do show increased heritability for weight with increased age (Dillard *et al.* 1972) while sex-corrected estimates in rats do not (Atchley & Rutledge, 1980). Atchley & Rutledge (1980) also found no significant age-related increase in sex-corrected heritability for trunk circumference while tail length's heritability was significantly positively correlated with age. Agricultural genetic studies of weight in cattle (Mavrogenis, Dillard & Robison, 1978; Trail, Sacker & Fisher, 1971), sheep (Dzakuma, Nielsen & Doane, 1978; Mavrogenis *et al.* 1980; Martin *et al.* 1980; Chopra & Acharya, 1971) and swine (Ahlschwede & Robison, 1971; Kuhlers, Chapman & First, 1977) show similar results to those obtained with mice. However, maternal effects are only weakly negatively correlated with age in swine (Ahlschwede & Robison, 1971; Kuhlers *et al.* 1977).

Despite the wide variety of experimental and statistical designs used, it appears that in general maternal effects tend to decrease with age, especially during the period following weaning, while heritabilities either increase with age or remain at the same level. If heritabilities only increase weakly with time and maternal effects show strong, significant declines, non-maternal environmental effects must increase their proportional contribution to phenotypic variance with age. We will test the hypothesis that direct genetic and non-maternal environmental effects become relatively more important, while maternal effects decrease in importance with age.

2. MATERIALS AND METHODS

Two independently sampled groups of mice were obtained from the randombred ICR stock. The groups were raised in the Animal Genetics Laboratory at the University of Wisconsin-Madison about 9 months apart. Males and females were randomly mated within each foundation stock and longitudinal growth data was collected on 108 full-sib families of four pups each (432 mice). Sixty full-sib families (240 mice) were measured in replicate A by JMC and 48 families (192 mice) in replicate B by LJL. Litters born on the same day were obtained from a pair of non-full-sib dams and standardized at birth to eight pups, four males and four females. A random half of each litter was exchanged between the pair of dams. This cross-fostering design allows the separate estimation of direct genetic and maternal effects from the covariance among full-sibs and nurse litter-mates respectively (Rutledge *et al.* 1972; Atchley & Rutledge, 1980). The offspring were removed from their nurse mothers at 21 days of age and randomly assigned to single sex cages containing four mice. Only four of the original eight pups per nurse were retained for the present studies on the genetics of growth.

Seven live body traits were measured in replicate A: weight (WT), head length (HL), head breadth (HB), ear length (EL), trunk length (TRL), trunk circumference (TRC) and tail length (TL). Mice were measured weekly at 17, 24, 31, 45, 52, 59 and 66 days of age. Measurements were not taken at 38 days due to the vagaries of winter weather. Only five of these traits – weight, head length, trunk length, trunk circumference and tail length – were measured in replicate B due to the low repeatability of ear length and the lack of growth in head breadth during the period being considered (see below). For replicate B, mice were measured at 17, 24, 31, 38, 45 and 52 days, at which time they were sacrificed for inclusion in a cross-sectional analysis of skeletal growth. These data constitute longitudinal data as defined by Cock (1966) because individual animals were followed over much of their post-natal growth period. One animal out of every four was randomly chosen for repeated measurement so that repeatabilities could be estimated.

The mice were anesthetized with metaphane in order to measure the seven live body traits. The metaphane treated mice were compared to a control group composed of their full-siblings and nurse-mates for weekly weights from 14 to 70 days. Repeated administration of metaphane had a small but statistically significant effect on the growth of mice (Cheverud, unpublished data). After two administrations, metaphane-treated mice were about 0·80 g or 0·25 standard deviations smaller than the controls. The difference did not increase with age after 28 days. Since all mice included in this analysis were treated with metaphane, it should not be a significant source of phenotypic variability.

Variance components were estimated from an analysis of variance within cross-fostered pairs. The linear model used was

$$Y_{ijkln} = u + S_i + P_j + d_{k(j)} + n_{l(j)} + dn_{kl(j)} + e_{kln(j)}, \quad (1)$$

where Y_{ijkln} is the measurement on the nth pup of sex i nursed by the lth nurse born of the kth dam nested in the jth pair. Effects due to dam (d), nurse (n), dam by nurse interaction (dn) and the residual (e) were assumed to be random effects

with zero means and variances V_d, V_n, V_{dn} and V_R, respectively. Sex is included in the model to remove its effects from the analysis. Therefore the variance component estimates reported are the average variance components for the two sexes. Table 1 provides the expected mean squares for each factor identified in the model and used in genetic analysis. Sums of squares were obtained from the GLM procedure in SAS-79 (Helwig & Council, 1979) while mean squares and variance components were obtained with a BASIC program.

Table 2. *Age- and sex-specific means for seven live body traits: weight (WT), head length (HL), head breadth (HB), ear length (EL), trunk length (TRL), trunk circumference (TRC), and tail length (TL)**

		Age (days)						
Sex	Trait	17	24	31	45	52	59	66
Male	WT	8·196	13·100	22·467	29·528	31·953	33·615	35·290
Female	WT	8·266	12·302	18·890	25·099	26·497	27·665	28·515
Male	HL	2·162	2·264	2·379	2·525	2·590	2·625	2·638
Female	HL	2·156	2·241	2·347	2·466	2·527	2·575	2·592
Male	HB	1·079	1·118	1·171	1·217	1·216	1·223	1·221
Female	HB	1·080	1·101	1·142	1·188	1·197	1·197	1·187
Male	EL	1·033	1·298	1·414	1·535	1·589	1·661	1·657
Female	EL	1·005	1·305	1·393	1·504	1·571	1·640	1·655
Male	TRL	4·084	4·858	5·796	6·819	7·145	7·312	7·300
Female	TRL	4·032	4·756	5·524	6·458	6·789	6·912	6·915
Male	TRC	4·997	5·271	6·131	6·481	6·733	6·943	7·003
Female	TRC	4·988	5·197	5·749	6·169	6·352	6·483	6·500
Male	TL	4·617	6·003	7·063	8·586	8·983	9·251	9·373
Female	TL	4·703	5·924	7·000	8·342	8·725	8·971	9·082

* Weight was measured in grams while all other traits are reported as centimetres.

The direct additive genetic variance is estimated as twice the dam variance ($2V_d$) and heritability (h^2) is the proportion of the total phenotypic variance due to direct additive genetic effects ($h^2 = 2V_d/V_p$). The variance due to maternal effects is estimated by the between-nurse variance (V_n), m^2 representing the proportional contribution of maternal effects to the phenotypic variance ($m^2 = V_n/V_p$). The non-maternal environmental variance is defined as the sum of the dam–nurse interaction and residual variances ($V_{dn} + V_R$) and its proportional contribution to phenotypic variance symbolized by e^2.

Repeatabilities are reported as intraclass correlation coefficients using the linear model

$$Y_{ijk} = S_i + I_{j(i)} + e_{jk(i)}, \tag{2}$$

where Y_{ijk} is the kth record on the jth animal of the ith sex. These repeatabilities only measure the extent of intra-observer error. Inter-observer error should not affect the variance estimates because all variances are measured only within pairs (see above) and inter-observer error would always be between pairs in this design.

Whenever estimates for repeatabilities or any of the variance components from

replicates A and B are combined, they are given as the weighted average of the separate estimates. The weights used are the proportion of cases per replicate, 0·555 for the replicate A estimates and 0·445 for the replicate B estimates at ages 17, 24, 31, 45 and 52. Only replicate B included measurements at 38 days and only replicate A included measurements at 59 and 66 days of age.

Table 3. *Repeatabilities (intraclass correlations) for head length (HL), head breadth (HB), ear length (EL), trunk length (TRL), trunk circumference (TRC), and tail length (TL) combined over sex and repeat*

Age	HL	HB	EL	TRL	TRC	TL
17	0·795	0·372	0·613	0·925	0·879	0·988
24	0·920	0·796	0·714	0·950	0·881	0·986
31	0·875	0·702	0·708	0·938	0·837	0·986
38	0·908	—	—	0·926	0·803	0·986
45	0·894	0·763	0·480	0·893	0·818	0·971
52	0·764	0·583	0·540	0·845	0·783	0·968
59	0·770	0·912	0·661	0·824	0·785	0·956
66	0·554	0·922	0·366	0·874	0·753	0·951
Avg.	0·810	0·720	0·580	0·900	0·820	0·974

Table 4. *Heritabilities for weight (WT), head length (HL), trunk length (TRL), trunk circumference (TRC), and tail length (TL) for repeats A and B*

	WT		HL		TRL		TRC		TL	
Age	A	B	A	B	A	B	A	B	A	B
17	0·303	0·271	0·313	0·223	0·237	0·179	0·154	0·090	0·394	0·207
24	0·316	0·013	0·265	0·168	0·290	0·270	0·353	0·075	0·392	0·209
31	0·242	0·237	0·432	0·129	0·343	0·129	0·288	0·163	0·292	0·129
45	0·234	0·541	0·258	0·334	0·329	0·355	0·210	0·494	0·389	0·370
52	0·497	0·446	0·517	0·272	0·200	0·355	0·134	0·244	0·461	0·206

3. RESULTS

The male and female growth curves for replicate A for weight, head length, head breadth, ear length, trunk length, trunk circumference and tail length are given in Table 2. This data was not pooled with replicate B due to consistent significant differences in mean values between replicates. Statistically significant sex differences first appear in weight at 24 days, in head length, head breadth, trunk length, and trunk circumference at 31 days and at 45 days in tail length in replicate A. Ear length shows no significant sex dimorphism. Data from replicate B confirm this trend in sex dimorphism. Males and females are virtually the same size at 17 days and then diverge due to lower female growth rates. Statistically significant age differences occur for all traits except head breadth until the age 59–66 comparison when all traits except weight in males show a statistically insignificant increase. This indicates that growth is nearly complete at 59 days in these mice. Head breadth stops growing by age 45 days, as there is no significant change in head breadth between 45 and 66 days. Since head breadth shows little age-related variation, it was removed from the analysis of longitudinal growth. Lack of

Table 5. *Heritabilities (h^2), proportion maternal effects (m^2), proportion environmental effects (e^2), and phenotypic variances (V_p) for weight (WT), head length (HL), trunk length (TRL), trunk circumference (TRC), and tail length (TL)*

Trait	h^2	m^2	e^2	V_p*	Trait	h^2	m^2	e^2	V_p
WT17	0·290	0·582	0·128	164·928	TRC17	0·124	0·431	0·445	7·669
WT24	0·227	0·433	0·340	418·323	TRC24	0·219	0·375	0·406	8·507
WT31	0·226	0·461	0·313	694·645	TRC31	0·235	0·279	0·486	9·429
WT38	0·394	0·438	0·168	383·700	TRC38	0·353	0·176	0·471	5·350
WT45	0·360	0·348	0·292	433·496	TRC45	0·318	0·157	0·525	6·487
WT52	0·476	0·209	0·314	437·086	TRC52	0·184	0·064	0·752	5·425
WT59	0·208	0·200	0·592	516·578	TRC59	0·221	0·038	0·741	5·251
WT66	0·339	0·202	0·459	608·008	TRC66	−0·002	0·116	0·886	3·964
HL17	0·283	0·349	0·368	0·628	TL17	0·333	0·645	0·022	22·631
HL24	0·230	0·354	0·416	0·647	TL24	0·320	0·574	0·106	34·565
HL31	0·343	0·338	0·317	0·499	TL31	0·238	0·490	0·272	29·887
HL38	0·157	0·294	0·549	0·266	TL38	0·210	0·411	0·379	17·780
HL45	0·283	0·123	0·594	0·302	TL45	0·383	0·356	0·261	16·740
HL52	0·433	0·178	0·389	0·298	TL52	0·357	0·280	0·363	12·498
HL59	0·535	0·122	0·344	0·332	TL59	0·396	0·266	0·338	13·956
HL66	0·479	0·092	0·428	0·332	TL66	0·521	0·187	0·293	12·314
TRL17	0·215	0·467	0·318	8·875					
TRL24	0·282	0·513	0·205	10·224					
TRL31	0·265	0·411	0·324	8·053					
TRL38	0·219	0·417	0·364	5·180					
TRL45	0·340	0·181	0·479	4·429					
TRL52	0·268	0·153	0·579	3·435					
TRL59	0·135	0·166	0·699	3·234					
TRL66	0·325	0·213	0·462	4·170					

* All trait scores were multiplied by ten before variances were estimated.

age-related variation is to be expected for this trait in the age interval analysed here because brain growth, which is closely correlated with head breadth, is completed early in the postnatal growth period.

The repeatabilities, combined for males and females and replicates A and B, are presented in Table 3. The low repeatability of ear length is especially notable and resulted in its removal from further analysis. Repeatabilities were not taken for

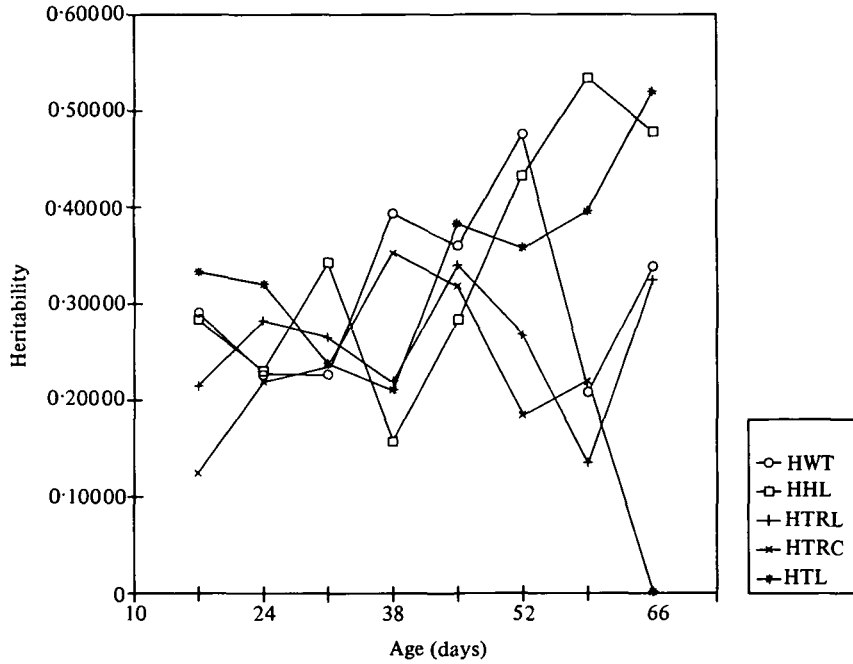

Fig. 1. Ontogenetic change in heritability for weight, head length, trunk length, trunk circumference, and tail length.

weight but they are believed to be about 0·90. There is a strong, statistically significant ($r_s = -0{\cdot}80$), tendency for repeatability to decrease with age. This decrease is probably due to the generally lower phenotypic variances at later ages (see Table 5) combined with a relatively constant absolute magnitude of error.

Heritabilities for the two replicates first will be compared and then later combined for further analysis. The heritabilities for replicates A and B for all five remaining traits (weight, head length, trunk length, trunk circumference, tail length) at ages 17, 24, 31, 45 and 52 days are presented in Table 4. Standard errors for replicate A are approximately 0·13 while those for replicate B are about 0·16. None of the heritability estimates are significantly different between replicates at the 0·05 level. However, replicate A's heritabilities are greater than replicate B's in 76% of the cases. Since there are no significant differences between replicates, they are combined for further analysis.

The combined heritabilities (replicate A and B) are presented in Table 5, along with m^2, e^2 and V_p. The average heritability of the entire data set is 0·293 with a standard deviation of 0·11. The direct additive genetic, maternal, and environmental variances can be obtained by multiplying each component by the

phenotypic variance given in Table 5. The standard errors for the heritabilities are about 0·10 at 17, 24, 31, 45, and 52 days, 0·13 at 59 and 66 days, and about 0·16 at 38 days. Only six heritabilities (HL38, TRL38, TRL59, TRC17, TRC66, TL38) are not significantly different from zero at the 0·05 level.

The relationships of heritability, maternal effects, and environmental effects with age are demonstrated in Fig. 1–3. There is no significant trend for increased heritability with age for weight, trunk length, or trunk circumference (Fig. 1).

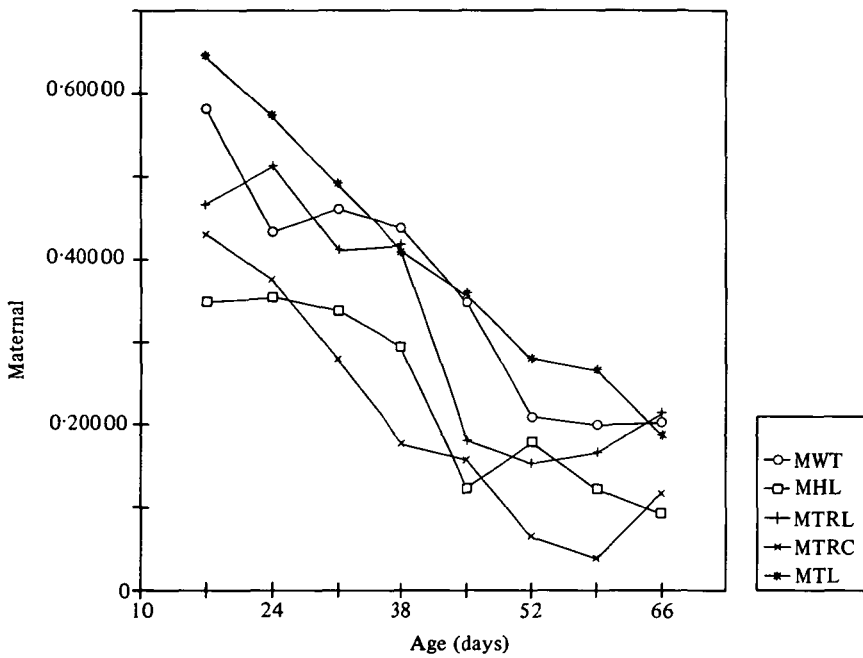

Fig. 2. Ontogenetic change in the proportion of phenotypic variance due to maternal effects for weight, head length, trunk length, trunk circumference and tail length.

However, heritabilities for head length ($r_s = 0{\cdot}67$) and tail length ($r_s = 0{\cdot}74$) do significantly increase with age. However, the ranges of heritability values for head length and tail length are small and the highest heritability is not significantly greater than the lowest heritability for either trait. Therefore, one should not over-emphasize the importance of these age-related trends in the estimates.

Maternal effects range from 0·038 to 0·645. Standard errors of the m^2 estimates are approximately 0·05. Only two m^2 values (TRC52, TRC59) are not significantly different from zero at the 0·05 level. Maternal effects significantly decreased with age (Fig. 2), all of the rank correlations being less than $-0{\cdot}90$. The strongest maternal effect is significantly greater than the weakest maternal effect for all traits. Maternal effects are the major source of phenotypic variation at weaning accounting for about 50 % of the variance in most traits and account for a much smaller but still significant proportion of the variance at 66 days. Therefore, maternal effects can play a significant role in the evolution of traits, even in the adult (Cheverud, unpublished data). Environmental effects significantly increase with age for all traits except head length (Fig. 3).

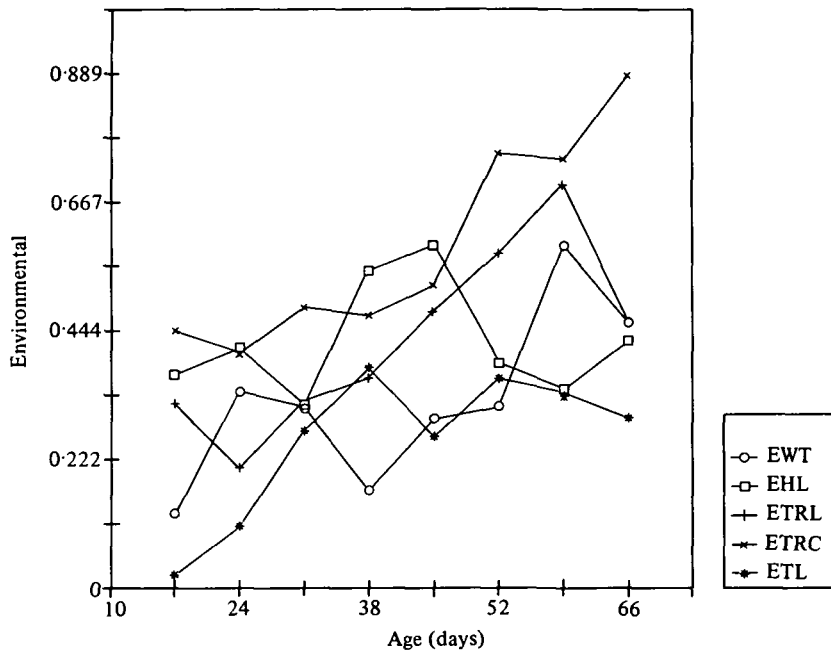

Fig. 3. Ontogenetic change in the proportion of phenotypic variance due to environmental effects for weight, head length, trunk length, trunk circumference and tail length.

4. DISCUSSION

There was no significant increase in heritability with increased age for weight. This result is consistent with the most comprehensive growth studies previously reported on mice, swine and rats (Rutledge *et al.* 1972; Ahlschwede & Robison, 1971; Atchley & Rutledge, 1980). These general observations on the ontogeny of weight's heritability are extended to include trunk length and trunk circumference which also show no significant increase in heritability with age. Head length and tail length did increase in heritability with age but the distinction between the high and low heritabilities in the age series was small.

It is possible that live body traits representing linear skeletal dimensions without much soft tissue contribution, such as head length and tail length, will typically have heritabilities which increase with age while live body traits which include a large soft tissue or fat contribution, such as weight and trunk circumference, will show no consistent linear trend of heritability increase with age. This possibility is consistent with the results reported here and with those reported by Atchley & Rutledge (1980) and Herbert *et al.* (1979) for tail length in rats and mice respectively. However, two few multivariate studies of longitudinal growth exist to allow generalization at the present time.

Maternal effects were found to decrease from about 50 % to about 15 % with age for all five traits analysed. This is consistent with previous results on maternal effects in mice (Young *et al.* 1965; El Oksh *et al.* 1967; Rutledge *et al.* 1972; Herbert *et al.* 1979) and rats (Atchley & Rutledge, 1980). One would expect maternal effects

to decrease with age after weaning because the mothers have no further opportunity for directly affecting growth. However, a substantial proportion of phenotypic variance, 10–20%, can still be attributed to maternal effects when growth is complete.

The proportion of variance due to environmental effects increased significantly through time for all traits except head length. This is a necessary result of the more or less age-constant heritabilities and the age-regressive maternal effects. However, this combination of increased environmental effects and decreased maternal effects will have important consequences for the ontogenetic dynamics of phenotypic covariance and correlation patterns.

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