

MATERNAL EFFECTS ON WEANING WEIGHT OF MICE

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ABSTRACT

The role of maternal effects on weaning weight was analysed in a population of mice under selection for this trait, with a total of 7042 records. Weaning weight was significantly affected by sex, class of litter size at birth, and generation number, together with linear and quadratic effects of dam inbreeding. Offspring inbreeding had no significant effect on weaning weight. Covariances among paternal half-sibs, full sibs, dam-offspring, and individuals with the same maternal grandsire were estimated by least squares. Covariances among relatives were taken as dependent variables, while the independent variables were the theoretical coefficients of causal components of genetic and environmental variances. Heritabilities of maternal and direct effects were obtained from the causal components, the coefficients being 0.286 and 0.275 respectively. However, the environmental component represented by littermates accounted for 41.83 percent of the phenotypic variance, constituting therefore, the main source of variation for weaning weight. The covariance between direct and maternal additive genetic effects was negative, and the respective correlation coefficient was -0.652. Total heritability of weaning weight was 0.079, i.e., less than the magnitude of heritability estimated from paternal half-sib correlations.

INTRODUCTION

Since the early times of animal breeding (especially for mammals) man has recognized the importance of maternal influence on the phenotype of offspring. The idea that events which affecting the mother during mating or gestation would affect the foetus has long persisted and may be found in Genesis (Legates, 1972).

Males and females contribute equally to the offspring genotype, but especially in domestic mammals, the male affects his offspring through the gametic cells only, while

the dam exerts both a genotypic influence and a postnatal maternal influence expressed through milk production and maternal care.

In the present paper, maternal effects are defined according to Legates (1972), as being phenotypic expressions from the mother, affecting traits of her offspring, but excluding the direct effects of maternal genes. According to Willham (1983) though maternal effects are entirely environmental in relation to the offspring, it is a result of genetic variation in some other trait of the dam. Therefore, the phenotypic expression of a trait (e.g., the weaning weight) depends on two genetic components: the genotype of the offspring (direct genetic effect) and the genotype of the dam (maternal genetic effect).

The objectives of this study were to estimate direct and maternal genetic variances, the covariances between them and environmental variances on weaning weight in mice selected for this trait.

MATERIAL AND METHODS

The data were obtained from 15 generations of mice selected individually for weaning weight, during an experiment conducted by one of us (R.G.S.) for other purposes, including a total of 7042 individuals (3594 males and 3448 females) (unpublished data).

The base population was constituted of individuals from four different origins, mated at random. The animals were bred in wooden cages 31.5 cm long, 20.0 cm wide and 15.0 cm high, covered with wire mesh. These cages were maintained in a room with a constant environment at 22°C and 63 - 70% relative humidity.

Feeding and water were given *ad libitum*, and light variation was the natural daylight cycle.

Selection proceeded on the basis of individual weaning weight (at 21 days of age), the heaviest 50 males and 100 females being mated at random to produce the next generation. All generations were discrete. Also, each female had only one parturition.

Four models were considered in the analysis. Adjustment factors for the effects of sex, litter size at birth, and regression on dam inbreeding were obtained from the first model, by the least squares method, after Harvey (1960). In this model only statistically significant effects (as previously tested) were considered. Thus, offspring inbreeding was not in the model, as it was not a significant effect. The first model was used to estimate the observational phenotypic variance component (σ_p).

The second model was:

$$Y_{ijkl} = \mu + G_i + S_{ij} + D_{ijk} + \epsilon_{ijkl}, \quad (1)$$

where Y_{ijkl} was individual weaning weight, adjusted for the fixed effects (except generation) and corresponding to the l th generation, which was an offspring from the mating of the j th sire and the k th dam; μ was the overall mean; G_i was the fixed effect on the i th generation ($i = 1, \dots, 15$); S_{ij} was the effect of the j th sire within the i th generation; D_{ijk} was the effect of the k th dam within the j th sire within the i th generation; ϵ_{ijkl} was the random error and μ the overall mean. The effects and G_i and μ were considered as fixed, while the other effects were random. This model was used to estimate covariance components between full-sibs (σ_F^2) and paternal half-sibs (σ_S^2).

The third model was:

$$Y_{ijkl} = \mu + G_i + S_{ij} + b(X_{ijk} - \bar{X}) + \epsilon_{ijkl}, \quad (2)$$

where b was the regression coefficient of offspring weaning weight on dam weaning weight; X_{ijk} was the weaning weight of the k th dam within the j th sire within the i th generation; $\bar{X}$ was the average weaning weight of the dams. This model was used to estimate dam-offspring covariance ($\text{cov}\{O, D\}$).

The fourth model was:

$$Y_{imno} = \mu + G_i + H_{im} + D_{imn} + \epsilon_{imno} \quad (3)$$

where H_{im} was the effect of the m th maternal grandsire within the i th generation; D_{imn} the effect of the n th dam within the m th maternal grandsire and the i th generation. Model (3) was used to estimate covariance component between individuals from the same maternal grandsire (σ_{II}^2).

Maternal effects were evaluated according to the method of Willham (1963, 1972), under the assumption of no dominance or epistatic effects. The components of variance and covariance among relatives were estimated according to Koch (1972) from the analysis, following models 1 to 4. A series of simultaneous equations were then solved to provide estimates of the causal components. These equations considered the covariances among relatives as dependent variables, while the theoretical coefficients of the causal components of genetic and environmental variance were the independent variables. Table I shown these equations, for which the following causal components were considered:

- $\text{var}\{A^o\}, \text{var}\{A^m\}$ = additive genetic variance components for direct and maternal effects, respectively;
- $\text{cov}\{A^o, A^m\}$ = covariance between direct and maternal effects;
- $\text{var}\{E^o\}, \text{var}\{E^m\}$ = environmental variance components for direct and maternal effects, respectively;
- $\text{var}\{P\}$ = phenotypic variance component.

Table I - Simultaneous equations used to estimate causal components of variance.

Component	Causal component				
	var {A ^o }	var {A ^m }	cov {A ^o , A ^m }	var {E ^o }	var {E ^m }
σ_s^2	1/4	0	0	0	0
σ_{II}^2	1/16	1/4	1/4	0	0
σ_p^2	1/2	1	1	1	0
cov {O,D}	1/2	1/2	5/4	0	0
σ_p^2	1	1	1	1	1

The component var {E^m} estimates the environmental effects common to the individuals of the same litter, (since each dam had only one parturition), thus excluding permanent environmental effects.

From the causal variance and covariance components, the following coefficients were estimated:

$$h_t^2 = (\text{var } \{A^o\} + 1.5 \text{ cov } \{A^o, A^m\} + 0.5 \text{ var } \{A^m\}) \text{ var}^{-1} P \quad (4)$$

$$h_o^2 = \text{var } \{A^o\} \text{ var}^{-1} \{P\} \quad (5)$$

$$h_m^2 = \text{var } \{A^m\} \text{ var}^{-1} \{P\} \quad (6)$$

$$r_{om} = \text{cov } \{A^o, A^m\} (\text{var } \{A^o\} \text{ var } \{A^m\})^{-0.5} \quad (7)$$

Where h_t^2 = total heritability; h_o^2 = heritability of direct effects; h_m^2 = heritability of maternal effects and r_{om} = genetic correlation for direct and maternal affects.

RESULTS AND DISCUSSION

Table II shows the estimate observational components of variance and covariance. The sum of these variance components does not result in the phenotypic variance, which was obtained from different analyses.

The causal variance components, presented in Table II, are evidence of the role of direct and genetic maternal effects on the weaning weight of offspring. The ratio of maternal additive genetic variance (var {A^m}) to total phenotypic variance (var {P}) of

weaning weight is an estimate of h_m^2 and presented a value of 0.276, close to that of direct additive genetic variance ($\text{var } \{A^0\}$), which was $h_o^2 = 0.286$. These heritability estimates indicate that both weaning weight (direct effect) and maternal ability may responde to selection.

Table II - Estimated observational components.

Component	Estimate
σ_s^2	.3240
σ_{m}^2	.1332
σ_p^2	2.7503
cov {O,D}	-.0286
σ_p^2	4.5284

Eisen *et al.* (1970), Robinson *et al.* (1974) and Hanrahan and Eisen (1974) obtained heritability estimates from 0.222 to 0.516 for direct effects, and from 0.061 to 0.646 for the maternal effect of weight at 12 days of age.

The common environmental variance was $\text{var } \{E^m\} = 1.894$, which constituted about 41.83% of phenotypic variance, and was the main source of variation of weaning weight. Dam contribution ($\text{var } \{A^m\} + \text{var } \{E^m\}$) to total variation reached 69.4%.

Despite the fact that h_o^2 is an indicator of responsiveness of weaning weight to applied selection, it is expected that mass selection for this trait will be inefficient, due to the large influence of maternal environment, as was also observed by Eisen *et al.* (1970) for weight at 12 days of age.

Based on the cross fostering technique, several authors demonstrated the importance of maternal effects on weights from birth to weaning (Cox *et al.*, 1959; Young *et al.*, 1965; Rutledge *et al.*, 1972, and Brandsch and Kadry, 1977, for mice; Blunn, 1969; and Rutledge and Chapman, 1975, for rats; Ahlschwede and Robison, 1971, for swine).

El-Oksh *et al.* (1967) observed that the post-natal variance component (common environment effect included) was responsible for 52% of the variation in weaning weight of mice.

Hanrahan and Eisen (1974) detected a contribution of 32.3% for the common environmental variance component, using the method of the resemblance between relatives. However, Itulya *et al.* (1983) observed a contribution of only 4,1%, for weaning weight of rats.

Table III - Causal variance and covariance components, estimated as percentage of phenotypic variance.

Component	Estimate	Percent of σ_P^2
var $\{A^o\}$	1.296	28.6
var $\{A^m\}$	1.249	27.6
cov $\{A^o, A^m\}$	-1.041	-23.0
var $\{E^o\}$	1.130	25.0
var $\{E^m\}$	1.894	41.8
var $\{P\}$	4.528	100.0

A very important component to be considered is the covariance between direct and maternal additive genetic affects (cov $\{A^o, A^m\}$). Our results showed a negative coefficient for cov $\{A^o, A^m\}$, corresponding to 23% of the total phenotypic variance. This may be an indication of antagonism between maternal environment and genes that effect weaning weight. The genetic correlation between these effects, and the regression of additive direct genetic effects on additive maternal genetic effect were also negative, with values of -0.652 and -0.837, respectively.

Hanrahan and Eisen (1974), Robison *et al.* (1974) and Itulya *et al.* (1983) also observed negative values for the covariance between direct and maternal effects.

According to these results, continuous selection for weaning weight would cause a decrease of maternal ability. According to Cundiff (1972), a negative covariance between direct and maternal effects may be considered as an evolutionary mechanism to maintain the size of individuals in a population within determined limits.

Total heritability (h_t^2), after Dickerson (1947) and Willham (1963), was 0.079, close to that estimated by Hanrahan and Eisen (1974) for weight of mice at 12 days.

On the other hand, heritability estimated by correlation of paternal half-sibs was 0.319. The value of h_t^2 estimates the amount of selection differential which will be transmitted to the next generation. Because of the magnitude of genetic covariance between direct and maternal effects, h_t^2 does not reach 30% of the estimate obtained from the correlation of paternal half-sibs. This result shows that any selection for weaning weight will be inefficient, if maternal effects are not considered in the estimation of heritability.

Results of this study are consistent with those in the literature, for both livestock and laboratory animals.

Animal growth traits, especially weights before weaning, may be selected through the use of a selection index, giving consideration to maternal effects (Van Vleck,

1970). However, the methodologies available at the present time are not free from problems: (a) they do not estimate all components in the same analysis; (b) they do not give the precision of the estimates; (c) the same individual may be in more than one relationship, resulting in a correlation between the estimates; (d) in some cases, certain components need to be zeroed, in order to permit a solution. These problems were also commented by Eisen (1967), Foulley and Lefort (1978) and Willham (1980).

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RESUMO

A importância do efeito maternal sobre o peso à desmama foi analisada em uma população de camundongos selecionada para esta característica, utilizando-se um total de 7042 dados. O peso individual à desmama foi significativamente influenciado por sexo, classe de tamanho da ninhada ao nascer, número de gerações de seleção bem como pela endogamia da mãe, efeitos linear e quadrático. A endogamia do indivíduo não foi significativa para o peso à desmama. Foram estimadas as covariâncias entre meio irmãos paternos, irmãos germanos, mãe-descendente e indivíduos com o mesmo avô materno. As covariâncias entre parentes estimadas, foram consideradas como variáveis dependentes e os coeficientes teóricos dos componentes causais de variância genética e de ambiente, como variáveis independentes em uma série de equações. A partir dos componentes causais foram estimados os coeficientes de herdabilidade dos efeitos maternal e direto, cujos valores foram 0,286 e 0,275 respectivamente. O componente de variância de ambiente comum a membros da mesma ninhada, foi responsável por 41,83% da variância fenotípica, constituindo-se na principal fonte de variação para o peso à desmama. A covariância genética entre os efeitos maternal e direto foi negativa, sendo o coeficiente de correlação igual a -0,652. A herdabilidade total do peso à desmama foi de 0,079, não chegando a 30% da magnitude do coeficiente de herdabilidade estimado por correlação entre meio-irmãos paternos.

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