vestigation to substantiate this concept. Zeidman and Buss noted in 1954 that when V2 or Brown-Pearce carcinoma cells were injected into the afferent lymphatic of the rabbit popliteal lymph node and the nodes were removed 1 to 42 days later, only 2 of 30 animals demonstrated growth of tumor in pelvic nodes. This suggested to them that the popliteals as well as other lymph nodes were an effective, albeit temporary barrier to the further spread of cancer. The failure to observe tumor growth at sites other than in the injected node is, in our opinion, not conclusive evidence that transnodal passage of tumor cells failed to occur. Consequently, we have reinvestigated this aspect of tumor spread.

The afferent and efferent lymphatics of the popliteal lymph nodes of anesthetized rabbits were identified with or without the use of Evans Blue dye previously injected into the foot pad. With minimal trauma and no manipulation of the node, these vessels were cannulated with polyethylene tubing (PE-50). The cannula in the afferent lymphatic was connected by a Y connection to a 1-ml syringe which, by means of constant perfusion with a Harvard pump, delivered a suspension of either V2, or Brown-Pearce, or Walker tumor cells. The last tumor grows only in rats. The other limb of the Y tube was connected by polyethylene tubing to a pressure transducer, strain gauge, and recorder. In 20 such preparations 0.5 to 1.0 ml of a tumor cell suspension containing from 0.5 to 20 × 10⁶ cells were delivered during a 30- to 60-minute period. Pressures were constantly monitored and were maintained below 50 mm-Hg—for the most part less than 30 mm—during infusion. In some animals the efferent lymphatic was cannulated prior to tumor cell delivery and in others the efferent catheter was inserted 2 hours after injection. Proof that afferent lymph vessels did not circumvent test nodes was obtained by dye studies with injected dyes and anatomical dissections. Lymph was obtained after tumor cell injection with and without saline infusion into the afferent vessel. It was collected in tubes containing citrate and formalin and permitted to fix for 24 hours. Samples were passed through 5-μ Millipore filters which were subsequently stained with hematoxylin and eosin, mounted on glass slides, and examined for cells. Tumor cells morphologically identical with those of the parent tumors were observed in one or more of the lymph samples collected from 15 such preparations. They were present in the efferent lymph following perfusion of as few as 500,000 cells and at pressures as low as 10 mm-Hg. All three types of tumor cells were found in efferent lymph.

To eliminate the possibility that results were an artifact of the perfusion, Brown-Pearce and V2 rabbit tumor cell suspensions (1 to 5 ×10⁶ cells in 2 ml of saline) were injected into the foot pad after the efferent lymphatic of the popliteal node had been cannulated. Lymph was collected and treated in a manner similar to that described above. Tumor cells were evident in all of ten such preparations within 60 minutes after inoculation; not infrequently tumor cells were observed in lymph collected 10 minutes after inoculation. In many, cells appeared not only singly, but in clumps.

Extensive investigation is under way to quantitate the transnodal passage of such cells, to evaluate the effect of nodal alteration, and to equate cell viability following its egress from the node. The data obtained from these experiments prompts the conclusion that while tumor cells may be sequestrated in the lymph node, they also traverse that structure.

BERNARD FISHER
EDWIN R. FISHER
*Departments of Surgery and
Pathology, University of Pittsburgh,
Pittsburgh, Pennsylvania 15213*

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11. Supported by PHS grants CA-05716, CA-06670, HE-07822, and American Cancer Society grant P-142. The technical assistance of Joyce Mitchell, E. Rodis, and R. Pellegrini is acknowledged.

23 February 1966 ■

Maternal Effect in Dental Traits of the House Mouse

Abstract. *The size of the second and third mandibular molar teeth of inbred mice is altered if the mice are fostered on a different strain. The dental change is opposite in direction to that of body weight. The prenatal and postnatal components of variance are correlated with the developmental histories of the two tooth types.*

The maternal effect in mammals is generally regarded as the nongenetic influence of a dam on her progeny. It includes a prenatal element associated with the egg cytoplasm or the uterine environment or both; it may also include a postnatal element associated primarily with the lactational performance of the dam. Its presence has been demonstrated in several mammalian species, most particularly with respect to measures and correlates of overall body size (*1*). Maternal effects acting on a specific anatomical system independently of overall body growth or on a specific morphogenetic event have only rarely been demonstrated in mammals (*2*).

With respect to quantitative traits in an isogenic strain, a maternal effect will result in a correlation of sibs and, perhaps, in a regression of offspring on dam. The differences between sibships in an isogenic strain in a controlled macro-environment may be considered as due principally to differences in the micro-environment or "noise" acting via the maternal physiology. In genetically variable populations, the maternal effect may include, in addition to the noise component, the effect of differences in maternal physiology consequent to the variation in the maternal genotypes. This effect is, of course, nongenetic with respect to the traits of the offspring.

Only recently has the relative importance of the maternal effect on dental dimensions of a genetically variable mammalian population been determined. In a randomly bred strain of house mice approximately 16, 17, and

Table 1. A comparison of the means of the molar widths (lower jaw) and body weight of the two sublitter types within each strain. The first letter denotes the parental strain, the second letter denotes the strain on which the offspring was nursed. The probability value is based on a paired comparison t-test.

Type	Width		Body weight (g)
	M_2 (μ)	M_3 (μ)	
AA	920.51	583.24	18.80
AB	928.46	587.99	18.11
AB—AA	+7.95	+4.75	—0.69
P value	< .001	0.05-.02	< .005
BB	954.99	668.46	18.39
BA	946.18	661.28	19.06
BA—BB	—8.81	—7.18	+0.67
P value	< .001	< .001	< .01

27 percent of the total variance in mandibular molar width was due to the maternal effect in the first (M_1), second (M_2), and third (M_3) mandibular molars, respectively (3). This report is of a cross-fostering experiment between inbred strains of the house mouse in which the relative importance of the prenatal and postnatal maternal environments to molar size and variation is analyzed.

The inbred strains used were A/J (strain A) and C57BL/10J (strain B) both of which had over 80 generations of brother-sister mating when obtained from the Jackson Laboratory, Bar Harbor, Maine. Single-pair, intra-strain matings were made at approximately 8 weeks of age. Within 12 to 24 hours after parturition the litters were counted, sexed, and one-half of each litter (a sublitter) was interchanged reciprocally with a litter of the same size of the opposite strain. Litter size varied from four to ten with a mean of approximately 6.7 in each strain. The sex ratio was kept as near to unity as possible within each sublitter, although differences attributable to sex are nearly absent in the dental traits studied (4). At 5 weeks of age the offspring were killed and weighed, and the skulls were prepared by the papain digestion technique (5). The traits studied were the widths of the M_2 and the M_3 measured essentially at right angles to the anterior-posterior axis of the tooth. The teeth of both sides of one male and one female (when both sexes were present) within each of 90 sublitters within each strain were measured. Measurements were taken with a Gaertner traveling microscope to the nearest 0.001 mm at ×39; the measurements were unaffected by wear on the teeth.

The M_2 shows differentiation on about day 16 of development in utero; it begins dentinogenesis, which is followed by amelogenesis between the 2nd and 4th days after birth of the mouse, and starts to erupt about the 18th day. The relatively small, semivestigial M_3 does not initiate differentiation until about 5 days after the birth of the mouse and remains 9 days behind the M_2 in stage of development (6).

The molar widths and body weight (means) of the two types of sublitters within each strain were compared (Table 1). In strain A, both the M_2 and M_3 are larger in the mice nursed by B dams (AB) as compared to those nursed by the A dams (AA). In strain B, mice nursed by strain B dams (BB) also have larger teeth than those nursed by females of strain A (BA). One of the four differences is significant (P < .05), and the other three are highly significant (P < .001) by the paired-comparison t-test on the 45 pairs of sublitter means. Since both the M_2 and M_3 are larger in strain B, the direction of the difference attributable to the postnatal effect is toward the phenotype of the strain of the postnatal dam. The mean change in the M_2 due to fostering represents 24 percent of the difference in tooth size between the strains. Similar calculations for the M_3 give 7 percent. The correlation between the absolute amount of tooth change and the

Table 2. General form of analysis of variance. In the degrees of freedom column, a is the number of litters, b is the number of sublitters per litter, c is the number of individuals per sublitter, and d is the number of sides per individual. L, prenatal litter; G, postnatal genotype; I, individual; S, side; d.f., degrees of freedom.

Source of variation	d.f.	Composition of mean square
Between sublitters		
Prenatal litter	$a-1$	$\sigma^2_S + d\sigma^2_I + cd\sigma^2_{LG} + bcd\sigma^2_L$
Postnatal genotype	$b-1$	$\sigma^2_S + d\sigma^2_I + cd\sigma^2_{LG} + acd\sigma^2_G$
Interaction	$(a-1)(b-1)$	$\sigma^2_S + d\sigma^2_I + cd\sigma^2_{LG}$
Within sublitters		
Individual	$ab(c-1)$	$\sigma^2_S + d\sigma^2_I$
Side	$abc(d-1)$	σ^2_S

Table 3. The analysis of variance for width of the M_2 and M_3 within inbred strains A/J (strain A) and C57BL10/J (strain B). Negative estimates of variance components are assumed to be zero.

Source of variation	Strain A				Strain B			
	d.f.	Mean square	Variance component	%	d.f.	Mean square	Variance component	%
			Analysis of M_2					
Between sublitters								
Prenatal litter	44	1259.7	117.5	29.1	44	916.8	70.4	21.3
Postnatal genotype	1	5528.4	29.6	7.3	1	6987.2	36.9	11.1
Interaction	44	345.9	—19.8	0.0	44	352.8	0.6	0.2
Within Sublitters								
Individual	85	422.9	165.5	40.9	90	350.6	127.4	38.5
Side	175	91.9	91.9	22.7	180	95.9	95.9	28.9
Total	349	367.3	384.7	100.0	359	311.0	331.2	100.0
			Analysis of M_3					
Between sublitters								
Prenatal litter	44	682.0	27.6	8.7	44	249.8	—11.5	0.0
Postnatal genotype	1	1971.9	8.6	2.7	1	4634.4	23.9	12.5
Interaction	44	467.2	9.6	3.0	44	341.9	48.4	25.3
Within sublitters								
Individual	85	429.7	156.4	49.0	90	149.0	30.0	15.7
Side	175	116.9	116.9	36.6	179	89.1	89.1	46.5
Total	349	313.8	319.1	100.0	358	167.7	179.9	100.0

tooth means of the nonfostered mice for the two teeth in the two strains was high ($r = +0.90$, $p < .05$), indicating that the change is nearly proportional to tooth size. The differences in means between the nonfostered animals of the two strains are assumed to be primarily hereditary in origin and are not unexpected in view of the relatively high heritability of these traits, as previously determined in a randomly bred strain (3). Dental regressions of offspring on sire are all nonsignificant ($P > .05$), an indication of no detectable genetic variation within a strain. Dental regressions of nonfostered (and fostered) offspring on dam are similarly nonsignificant, indicating isogenicity and also no detectable intrastrain association of maternal and progeny phenotypes due to the maternal effect. Correlations of tooth size and litter size are generally low, negative, and insignificant.

Within each strain there is a positive correlation of tooth size and body weight, the mean r for the M_2 and body weight being $+0.36$ ($P < .001$) and for the M_3 and body weight being $+0.18$ ($P < .01$). That the difference in tooth size related to the postnatal performance of the two types of females is not due simply to an attendant change in body weight, that is, a general growth phenomenon, can be seen in Table 1. The postnatal effect of strain A mothers was to increase body weight in both strains, whereas tooth size was decreased. Strain B mothers have the opposite effect. Thus, there may be a difference between genotypes with respect to milk and its specific effect on dentition. Alternatively, a given difference in the milk of the two strains may both inhibit body growth and promote dental development and vice versa. A possible cause of the effect may lie in the amino acids which can differ in the milk of different inbred strains of mice (7).

The magnitude of the prenatal and postnatal effects relative to one another and to the total intrastrain variance can be estimated from an analysis of variance. The general form of the analysis combines both the cross-classification and hierarchal models (Table 2). The total maternal effect is estimated by the "between-sublitter" component which includes the differences between litters born to different dams within a strain (prenatal litter factor), the difference between sublitters nursed by dams of the two strains (postnatal

genotype factor) and the interaction between the two factors, that is, the differential response of the litters to the two postnatal experiences. The interaction also includes any effect of transferring the pups to the foster mother. However, the relative variance contribution of the maternal effect as well as the total variance are very similar in the two sublitter types within each strain, an indication of little, if any, effect of the transference *per se*. The "within-sublitter" component includes differences between individuals and differences between sides within the individual.

The results of the analyses for the two teeth within each strain are presented in Table 3. For the M_2 the relative contribution of each source of variation is quite similar for the two strains. The prenatal effect is relatively large and significant, and the interaction component is essentially absent in this molar. There is a somewhat greater difference between the results for the two strains in the M_3, but a general pattern is discernible. The prenatal factor is present, but insignificant, in strain A and absent in strain B. The interaction component is present in both strains but significant only in strain B. The total relative maternal effect is very similar in three of the analyses, being somewhat lower in the M_3 of strain A.

It appears, then, that the M_2 is influenced by a nongenetic prenatal factor in addition to its strain-specific genetic determination. Evidently development in this molar has proceeded sufficiently far at birth to render it insensitive to the interaction between prenatal-litter factors and postnatal genotype factors, that is, the different prenatal litters tend to respond postnatally in a relatively uniform manner to the genotypic difference between the nursing dams. However, the tooth is clearly not so far along in development that it is unaffected by the postnatal environment, as shown by the presence of a significant postnatal genotypic component. Presumably because it initiates development at a later time, the M_3 is apparently little affected by intrastrain differences in the prenatal environment although it is, of course, strongly influenced by its strain-specific genetic determination. It is, however, more responsive to its postnatal environment than the M_2 since it manifests, at least in strain B, a significant interaction component in addition to the postnatal genotypic effect.

For traits such as general body growth, there may be a postweaning compensation and adjustment to the early influence of the maternal effect (8), and a progressive diminution of the relative importance of this effect to the total variance as the animal matures (9). In teeth of the type studied here, however, development of the crown has essentially ceased by weaning, so that the effects of the maternal environment are incorporated as a permanent part of the structure.

PAUL TENCZAR
ROBERT S. BADER
*Department of Zoology,
University of Illinois, Urbana 61801*

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10. Supported by NSF grant G-19197 and by the Research Board, University of Illinois. We are indebted to Dr. R. W. Touchberry, University of Illinois, for statistical advice.

18 March 1966

Availability of a Cationic Herbicide Adsorbed on Clay Minerals to Cucumber Seedlings

Abstract. Montmorillonitic and kaolinitic clays are effective in decreasing the toxicity of paraquat, an organic cation, to cucumber plants. The cation was adsorbed on the surface of the kaolinite clay particles and slowly became available to the plants. When it was adsorbed in the interlayer spacings of the montmorillonite clay, however, it was not available to the plants.

Investigation of the adsorption of several herbicides by various adsorbents have indicated that two organic cationic herbicides, diquat [6,7-dihydrodipyrido-(1,2-a:2′,1′-c)-pyrazidiinium dibromide] and paraquat (1,1′-dimethyl-4,4-bipyridinium dichloride), are strongly adsorbed on montmorillonite