A COMPARISON OF GENETIC AND ENVIRONMENTAL INFLUENCES ON THE SHAPE OF THE AXIS IN MICE

DONALD W. BAILEY¹

Roscoe B. Jackson Memorial Laboratory, Bar Harbor, Maine

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BIOMETRICAL analyses of the regulation of development have necessarily dealt with the relationships between anatomical parts. Most of these attempts have remained in the realm of allometry, the principles of which were developed and elaborated upon by HUXLEY (1932) and others (see BONÉ 1955; and BERRILL 1955). However these studies have failed to establish a universality of the allometry equation since many exceptions to a strict adherence of data to the hypothesized straight-line relationships have been noted (see RICHARDS and KAVANAGH 1945; and REEVE and HUXLEY 1945). Nevertheless, allometric methods still provide a useful descriptive tool.

Genetic studies of this problem have been concerned largely with comparisons of body proportions of animals differing in one or more major genes, the number of genes being either known or hypothesized (see PECK and SAWIN 1950).

An altogether different approach to the problem of regulation of development was made by WRIGHT (1932, 1954) who relegated the relationships of sizes of parts in the rabbit and the fowl to a hierarchical grouping of general and special factors. In the same study he roughly estimated the relative contributions of genetic and environmental factors. BURT (1943) has also used the methods of factor analysis on human physical data.

Other than in the study made by WRIGHT (cited above), differences in the roles played by the environment and the genome have especially been lacking. Usually these sources of influence are compounded in analyses such as the studies of allometry—one possible answer for the deviations from the straight-line relationship. Consequentially important questions left unanswered are: How do the environmental and genetic forces differ in their control of relative growth and where do environment and heredity meet in their shaping of an organ during development? Does the environment act through or upon developmental pathways, augmenting or diminishing gene action as suggested by GOLDSCHMIDT (1938)?

Also wanting in these biometric studies are analyses dealing with single organs such that the relationships of the adjacent parts and the shape of the organ are analysed. However, a descriptive analysis along this line was made by BATEMAN (1954) in which he made use of two inherited skeletal anomalies for deciphering the shape changes of individual bones occurring during early development in the mouse. The present study is largely concerned with the latter two problems, i.e. (1) the differences and interrelationships between the environmental and genetic effects and (2) the properties of the size relationships between parts of a single bone.

With the premise that the shape of an organ may be regarded as the resultant of

¹ U. S. Public Health Service Postdoctoral Fellow.

the many forces of the genome, the environment and their interactions, the following study is an attempt to analyse some of the influences of these forces upon the shape of the axis. This has required the analysis of the forces into three portions or sources of variance—inter-strain (genetic), inter-litter (environmental) and intra-litter (environmental)—for a number of shape-defining dimensions. The relationships between these various dimensions were then analysed for each of these sources of variance. (The genetic-environmental interaction portion of variance has been assumed to be negligible in this study.)

METHODS

Skeletons of adult, 90-day old male mice were prepared by the papain-digestion technique (LUTHER 1949, quoted by BATEMAN 1954). Each of 18 strains was represented by six animals (except the N/Blk strain having but four). Intra-strain grouping of the six animals into three subgroups, two mice per subgroup, was made on the basis of (1) their being littermates or (2) their possessing a common time of birth but not coming from the same mother. It was regretted that littermates could not have been the sole basis for this subgrouping. However the only efficient means of collecting representative groups of the 18 strains in a reasonable period of time made this procedure necessary. On this account, in the analyses below the variances and correlation coefficients termed "intra-litter" will contain an unknown portion of the inter-litter variance.

The 18 strains used are shown in table 1 with their inbreeding histories. With the exception of the Large strain, all are inbred at least to the point of F being 0.90.

TABLE 1
Inbreeding histories of the strains used in the experiment

Strain	Generations B-S inbred
A/Jax	89
Ake	14
BALB/Jax	90
CBA	84
C57BL/6	39
C3H/Ks	14+
DBA/My	20+
Large (GOODALE)	3*
LP	42
ND	30
129	14+
RIII	16
Small (MACARTHUR)	11
S/Emv	19
ST	39
Swiss	19
SWR	42
N/Blk	15

* Previous closed-line mating system brings estimate to $F = .60$ (personal communication from DR. CHAI).

An estimate of the maximum average genetic variance remaining within all 18 strains, is three percent, a very small amount. Any genetic variance or covariance that might remain within strains would be included in the inter- and intra-litter portions. This contamination would tend to dilute any differences found between the genetic and environmental kinds of variances and correlation coefficients.

In this study only the axis and the innominate bone were used. The innominate length rather than body weight was selected to serve as a standard of general skeletal size, for body weight is more subject to everyday environmental influences and it involves all kinds of tissues. Measurements were made on camera-lucida outlines of the posterior views of the axis. The innominate was measured directly by a vernier caliper.

The several measurements are shown in figure 1 as:

AH—arch height, distance from mid-point of dorsal surface of centrum to mid-point of ventral surface of arch.

AW—arch width, distance between medial surfaces of arch at dorsal edges of articulating surfaces.

L—lamina length, distance from mid-point of ventral surface of arch to medial surface of arch at dorsal edge of articulating surface.

P—pedicle length, distance from line measuring transverse process length perpendicularly to medial surface of arch at dorsal edge of articulating surface.

T—transverse process length, distance from dorsal corner of transverse process tip to mid-point of line drawn from dorsal to ventral centrum-base junctions.

B—base length, distance from point where pedicle meets line of transverse proc-

FIGURE 1.—Drawing of posterior view of the axis showing dimensions used in the analysis.

ess measurement to mid-point of line drawn from dorsal to ventral centrum-base junctions.

CH—centrum height, distance from mid-dorsal to mid-ventral surfaces of centrum.

CW—centrum width, distance between mid-points of a line drawn from dorsal to ventral centrum-base junctions.

I—innominate length, distance of longest dimension measured from crest of ilium to caudal edge of the pubis.

Right and left measurements of bilateral dimensions (except the innominate) were combined for more accurate measurement. The mean values and subsequent calculations of these bilateral measurements are based upon these "doubled" values except where otherwise noted. Each unit of measure is equal to 0.0513 mm for the axis and to 0.1 mm for the innominate.

RESULTS

Variance

The components of variance for each of the measurements and for each of the sources of variance (inter-strain, inter- and intra-litter) are shown in table 2. Those variances which were of statistical significance when tested in an analysis of variance are indicated. All of the inter-strain variances are shown to be of high statistical significance. This is not surprising, for strains of such diverse origin and maintained under such close inbreeding as the majority of the ones used here would be expected to have quite diverse mean measurements if heredity were at all important.

The inter-litter variances, on the other hand, are of statistical significance for only a few of the dimensions. It is seen that the dimensions which are of significance deal with laterad and associated measurements.

The differences in variability are more easily comparable in table 3 where the coefficients of variation are presented for each dimension and for each source of

TABLE 2
Estimates of the components of variance

Dimension	Source			Heritability estimate
	Inter-strain	Inter-litter	Intra-litter	
AH	2.84**	0.31	2.73	.318 ± .098
AW	4.72**	0.65*	1.89	.482 ± .089
L	10.24**	0.00†	6.64	.435 ± .121
P	11.11**	5.29**	5.26	.344 ± .072
T	14.48**	1.85*	5.06	.512 ± .088
B	11.44**	3.53*	6.89	.354 ± .081
CH	1.62**	0.10	0.59	.539 ± .095
CW	4.01**	0.91*	1.83	.425 ± .084
I	115.90**	9.29*	10.26	.748 ± .052

* Significant at the 5% level.

** Significant at the 1% level.

† Intra-litter mean square greater than inter-litter mean square.

TABLE 3

Means for each measurement with coefficients of variation arranged according to source of control (inter-strain, inter-litter and intra-litter). The figures in parentheses are rank orders of the variabilities from low to high and of the means from high to low within each source

Dimension	Source				Mean**
	Inter-strain	Inter-litter	Intra-litter	Total	
AH	4.33 (2)	1.43 (2)	4.25 (5)	6.23 (3)	38.906 (2)
AW	4.27 (1)	1.58 (3)	2.70 (2)	5.30 (1)	50.868 (5)
L	4.76 (3)	0.00* (1)	3.84 (4)	6.12 (2)	67.198 (3)
P	8.40 (8)	5.80 (9)	5.78 (7)	11.74 (7)	39.689 (6)
T	6.25 (5)	2.23 (6)	3.69 (3)	7.59 (5)	60.915 (4)
B	6.47 (6)	4.90 (8)	6.85 (9)	12.21 (8)	38.330 (7)
CH	8.17 (7)	2.04 (5)	4.92 (6)	9.76 (6)	15.576 (8)
CW	9.43 (9)	4.48 (7)	6.36 (8)	12.23 (9)	21.245 (9)
I	5.98 (4)	1.69 (4)	1.78 (1)	6.46 (4)	179.915 (1)

* Intra-litter mean square greater than inter-litter mean square.

** Bilateral measurements (see text) have been reconverted to "single" values in computing rank orders of means.

variation. There is a considerable diversity among the dimensions in their variabilities. All three sources of variance show similar rankings of the dimensions in regard to the magnitudes of the variabilities.

Heritabilities

Estimates of the heritabilities of the dimensions were made on the assumption of the strains having been developed as unselected lines from a common panmictic population, and the present strains having been inbred an infinite number of generations. On an additive scheme, heritability will be equal to:

$$\frac{\sigma_s^2}{\sigma_s^2 + 2\sigma_L^2 + 2\sigma_I^2}$$

where σ_s^2 is the inter-strain variance, σ_L^2 is the inter-litter variance and σ_I^2 is the intra-litter or individual variance. The estimates are shown in table 2.

Heritability usually refers to the proportion of the variability in a random bred population that is genetic; here it is the genetic component in an array of closely inbred lines. Only if all gene action were additive would the two be equivalent.

Correlations

The interrelationships of the dimensions are shown in tables 4, 5 and 6 as correlation coefficients. Those coefficients which individually show a statistically significant difference from zero are indicated. The correlation coefficients were computed from their respective mean squares and products for the intra- and inter-litter sources and from the variance and covariance components for the inter-strain source. Since the inter-litter mean square for L (lamina length) was less than the intra-litter mean square, it was necessary to use the mean squares and product to compute the inter-

TABLE 4

Inter-strain correlation coefficients between the various measurements of the axis and the innominate

Measurement	AH	AW	L	P	T	B	CH	CW
AW	.05							
L	.58*	.73*						
P	.01	-.30	-.41					
T	.20	.27	.16	-.07				
B	-.06	.54*	.33	-.68*	.39			
CH	.07	-.37	-.22	.59*	.12	-.84*		
CW	.09	.20	.17	.05	.04	-.41	.63*	
I	.36	-.22	.00	.22	.46	-.28	.73*	.53*

* Significantly different from zero at the 5% level.

TABLE 5

Inter-litter correlation coefficients between the various measurements of the axis and the innominate

Measurement	AH	AW	L	P	T	B	CH	CW
AW	-.11							
L	.30	.55*						
P	.31	.20	-.13					
T	.35*	.09	.20	.06				
B	.28	-.03	-.14	-.25	.50*			
CH	.04	.26	.26	.24	.02	-.39*		
CW	-.19	.41*	.32	.19	-.41*	-.63*	.39*	
I	-.21	.28	.42*	.00	-.07	-.50*	.44*	.53*

* Significantly different from zero at the 5% level.

TABLE 6

Intra-litter correlation coefficients between the various measurements of the axis and the innominate

Measurement	AH	AW	L	P	T	B	CH	CW
AW	.36*							
L	.74*	.54*						
P	.27	-.07	-.13					
T	.22	.04	.13	-.06				
B	.24	.34*	.00	-.13	.32*			
CH	.11	.05	.19	.15	-.04	-.05		
CW	-.02	.37*	.22	-.19	-.29*	-.36*	.23	
I	.04	.15	.14	-.02	.23	-.02	.13	.04

* Significantly different from zero at the 5% level.

litter coefficient of the lamina. This procedure was carried out for the other dimensions as well, in order to be consistent. Actually the use of the components of variance and covariance gave but little change in the values derived from the mean squares and products. (It has been assumed here that the inter-litter correlations do not differ

TABLE 7
Tests of homogeneity of correlation coefficients

Source	d.f.	χ^2	<i>P</i>
Within sets:			
Inter-strain	35	91.07	< .01
Inter-litter	35	106.42	< .01
Intra-litter	35	120.64	< .01
Between sets:			
Inter-strain			
Inter-litter	36	40.23	.30 to .35
Inter-strain			
Intra-litter	36	46.28	.10 to .15
Inter-litter			
Intra-litter	36	14.73	.95 to 1.00

between strains nor that the intra-litter correlations differ between litters other than by chance.)

Tests for homogeneity (see RAO 1952, p. 233) of the three sets of correlation coefficients are summarized in table 7. There is strong evidence that the coefficients are heterogeneous within the sets ($P < .01$). On the other hand, when the coefficients are tested between sets, there is no evidence that corresponding coefficients are different from one another. This latter finding made desirable further inquiry into the possible existence of more subtle differences between these sets of coefficients than was demonstrable by the tests of homogeneity.

Correlated responses to "selection"

The present data were not of a high enough level of reliability to warrant the use of such techniques as factor analysis or WRIGHT's method of hierarchical grouping. Therefore, the following procedure was devised in order to uncover what consistent trends in differences between the three sets of correlation coefficients the array of coefficients may offer.

Each dimension in its turn was arbitrarily increased by 30 percent of its mean measurement and the theoretical correlated responses for all the other dimensions were calculated. This procedure is an elaboration of an equation concerning correlated genetic responses to artificial selection presented by LERNER (1950), p. 236. Thus, each dimension increased provides the resulting theoretical change in all other dimensions and thus in shape of the whole vertebra as shown diagrammatically in figure 2. The same procedure has been carried out for the environmental correlation coefficients as well as the genetic, a procedure admittedly lacking in much biological meaning and practical use other than serving for comparison purposes in the present study.

The procedure has the advantages of condensing the information from several dimensions into a single diagram indicating shape change and of supplementing the information derived from the correlation coefficients, with the computed variances.

FIG. 2a.

FIGURE 2.—Diagrams of change in shape of the axis due to increase by 30 percent of the indicated dimension. Only halves of vertebrae are shown. A line along side the vertebral half represents the innominate length. The standard (average) vertebral shape and innominate length are indicated by ●—●, whereas the altered vertebral shape and innominate length are indicated by ○—○.

However, the procedure provides no means of statistically evaluating the evidence obtained; only tendencies may be described.

The diagrams in figure 2 represent the theoretical changes in shape of the vertebra due to the effects of the increase in the specified dimension upon all the other dimen-

INCREASED P

INCREASED T

INCREASED B

FIG. 2c.

sions. Only half of a vertebra is depicted. All distances are reconstructed in reference to the "unselected" average vertebra. The mid-dorsal point of the centrum is the common reference point for each diagram. A line, with its reference point below, is also drawn along side the vertebra half to show the relative changes in the innominate length.

DISCUSSION

Variance

It is reasonable to expect certain anatomical parts to be more or less variable than others. Developmental systems may involve pathways more or less affected by either the genetic background or the environment. The degree of variability brought about by outside agents may be due to adaptive physiological arrangements built up by natural selection for a more efficient system of development, or it may be due to the intrinsic degree of stability of the physico-chemical systems involved. In this sense, an increase in the variability of a tissue may be due to an adaptive change of the tissue under observation (e.g. muscular hypertrophy in response to exercise) or it may be due to an instability of the tissue which lacks means to adjust to the particular environmental stresses present (e.g. the non-regenerating mammalian limb). On the other hand, a decrease in the variability may be due to adaptive changes in the regulatory processes of the substance under observation (e.g. the homeostatic regulation of the blood sugar or blood pH level) or it may be due to an intrinsic stability of the observed tissue toward the particular environmental stresses present. It would be most difficult to distinguish between adaptive and intrinsic causes of variability or to determine the degrees to which both kinds are present.

Another explanation for differences in variability is that some developmental systems may involve a greater or lesser number of pathways—along with their various interactions—and thereby may allow a greater or lesser chance for outside agents to upset the normal course of events. This would be most evident when several functionally and developmentally distinct tissues are in conjunction.

In table 3 all three sources of variance show the higher variabilities for the centrum and nearby measurements (CW, CH, P and B), whereas the neural arch measurements (AW, AH and L), essentially neural tube dimensions, tend to be the least variable. It may be that a greater stability of the latter tissue is essential to the organism. The more variable measurements are those which could no doubt be influenced by developmental systems of different tissues, viz. neural, muscular and skeletal. These kinds of tissues would also be expected to possess more adaptive flexibility to cope with outside influences. These general areas of differences in variabilities may be seen below to be associated with the changes in shape of the vertebra.

In the present data there is an indication of a negative relationship existing between the variability and the size of the means. This is especially evident after the means of the bilateral measurements have been halved. This relationship is noticeable in the ranking presented in table 3. This relationship, however, may be a spurious one and due to the fact that the larger dimensions are concerned with arch measurements while the smaller dimensions are concerned with the centrum and adjacent measurements. Thereby the differences in variability may be intrinsic to the tissues involved (above) and not necessarily related to the size of the dimension.

The heritability estimates are comparable to heritabilities found for body weights in mice (FALCONER 1953). One exception in the present case is the unusually high value for the innominate measurement. The innominate measurement is exceptional in that it probably is highly correlated with body weight, for the Large strain showed an overly long innominate (deviation $> 2\sigma$). This one value in the calculation of the between-strains variance might well have unduly increased the genetic variance

estimate for the innominate in comparison to the other values. The Small strain however did not show an exceptionally small innominate.

The genetic variances used to compute the estimates of heritability were derived from the between-strain variance which would possess non-additive as well as additive gene effects. Therefore the computed heritabilities would be larger than those based on correlation between relatives in randomly mating populations.

There is no particular pattern of areas of the axis possessing high heritabilities as was observed for the variabilities above. Also the heritabilities show no consistent trend in their relationships to the magnitudes of the total variabilities (coefficients of variation) of the dimensions. This means that according to these data there is just as much genetic control over the total variability of a dimension which is greatly variable as over that which is but little variable. This is one outcome which concurs with the finding above that the magnitudes of the variabilities of the dimensions are generally correlated between the three sources of variance.

Correlations

The simultaneous change in one dimension of an organ with that of another dimension may be due, other than to chance, to any of several causes, genetic or environmental. Genetic correlations may be due to pleiotropy (common genes active in the development of both dimensions) or may be due to linkage (not likely in the present case). Pleiotropy may be effective by biochemical similarities (e.g. developmentally through like competences of the two tissue masses) or by mechanical associations, (e.g. one tissue part passively altered by the active growth of an adjacent part).

In relative growth studies, positive relationships seem to be the usual finding. This is due to the fact that the data usually involve actively growing parts. Growth, of course, tends to make strong positive correlations or regressions. Also the measurements used in most of the studies on relative growth and those measurements used by WRIGHT are of body parts remotely connected, i.e. body length, humerus length, femur length, etc. and sometimes of adjacent bones, i.e. ulna, radius, etc. but usually these are compared to general body size and not to each other. Measurements on the same organ seem to have been neglected. The present study involves several measurements on the same bone which may well account for the discrepancy found here with other studies in regard to the sign of the coefficients.

In the present study, several negative correlations were observed. The most likely explanations for the negative relationships are that active growth is not compounded with the other sources of variance present and that adjacent parts are involved in the measurements, one part passively submitting to active growth of the other part during development, e.g. the lamina-pedicle relationship. Even if two parts are not adjacent, themselves, a measurement strongly correlated with a distant measurement which is in turn adjacent to a third measurement, may be negatively correlated with the third measurement, e.g. base length-centrum height relationship, where the centrum height is positively correlated with the centrum width, which is adjacent to the base length. These negative correlations, however, do not preclude the general size factors of CASTLE, WRIGHT and others, so often hypothesized.

The significant negative genetic correlations are found in those relationships of base length. It is reasonable to suppose this dimension is passively controlled. It

can be considered a function of centrum width, arch width and slope of transverse process (see fig. 1).

Environmental correlations may be due to a common reaction of different genetic systems to the same environmental change (common environment) or common genetic pathways of two or more dimensions effected by environmental stimuli of all sorts. The latter kind would tend to simulate genetic relationships for the traits involved, as perhaps they do in the present study.

The homogeneity between sets of correlation coefficients may be due to residual genetic variance, to the environment acting generally upon the genetic pathways involved, to an insensitive test, i.e. there are differences but they are not shown to be significant. None of these are mutually exclusive.

Although the amount of genetic variance remaining within strains due to using some low-inbred strains is estimated to be not greater than three percent, a greater genetic variance than expected may remain within strains due to natural selection of the heterozygotes in opposition to the pressures of inbreeding. Residual genetic variance can never be fully ruled out as the cause of the homogeneity between sets, however it is doubtful much genetic variance remains within the strains since such strong genetic variance between strains is indicated.

On the other hand, the finding of possible homogeneity between environmental and genetic sets of correlations may be interpreted to mean that the environment is acting upon the developmental pathways at time of and in a manner similar to that of gene action. This is as might be expected; the genetic pathways may well be proven to be the framework upon which the environment has its main effects. This interpretation is further strengthened by the observed relationship between the rankings of the magnitudes of the variabilities of the dimensions between the three sources (table 3). These similarities indicate common origins of the variabilities among the different sources.

The fact that the environment can simulate genetic traits has long been recognized in experiments producing phenocopies (GOLDSCHMIDT 1936, and others). Developmental defects in mammals which resemble genetic diseases have also been produced by stresses invoked upon the pregnant mother (see RUNNER 1954). Just how closely these cases tie in with the present study is merely speculative at present. However, the anomalies which are induced by stress situations on the pregnant mother, as well as the expressed phenocopy, were shown to be dependent upon the genotype. This dependency was further shown by WADDINGTON (1952) in his selection experiments in *Drosophila*. Thus, the genetic pathways must be present for the environmental effect to be brought to the forefront, just as in the present case the environment appears to bring forth the existing genetic background expressible in axis shape.

Correlated responses to "selection"

In the diagrams of figure 2 these consistent trends may be seen:

Individual (intra-litter)

- (1) Arch dimensions do not change independently of one another.
- (2) All other dimensions do change independently of one another.

Litter (inter-litter)

- (1) Arch dimensions do change independently of one another.
- (2) Centrum dimensions do not change independently of one another.
- (3) A close relationship exists between the innominate length, centrum dimensions and the bending of the articulating surfaces and the transverse processes.

Strain (inter-strain)

- (1) Arch dimensions do change independently of one another.
- (2) Centrum dimensions do not change independently of one another.
- (3) A relationship exists between the innominate length and the centrum and the innominate length and the length of the transverse process.

No doubt other tendencies may be found, however the consistency of the above noted trends and their non-random nature leads to the conclusion that differences as well as similarities may exist in the ways by which these three sources of control effect the shape of the vertebra.

The most striking difference in the influences on shape of the three sources of control, is the strong effect which the inter-litter environment has upon the innominate length, ventral tipping of the articulating surfaces and the transverse processes (fig. 2). It is difficult to conceive of a strong direct embryonic relationship between the two regions of the innominate and the cervical vertebrae. The powerful inducing effect which the innominate seems to have on the transverse processes of the sacral vertebrae would hardly be considered in operation at this distance on the transverse processes of the cervical vertebrae, for the sacral inducing effect has a relatively sharp threshold anteriorly of the sacrum.

Probably a more acceptable explanation for this inter-litter effect is suggested by the strong correlation of both the innominate and axis shape with the degree of general development. BATEMAN (1954) has shown how the shape of the axis changes during its early development. The axis which he has drawn (p. 223) shows a considerable lowering of the articulating and transverse processes as the vertebra develops. The length of the innominate shows a substantial increase during this same period. Thus if the inter-litter environment is essentially augmenting the progress of development we would observe the picture somewhat as that shown in figure 2 where the innominate has been arbitrarily increased by 30 percent. Interference with developmental progress would show the opposite effect.

The explanation which is tentatively most able to fit the picture of environmental action as coming from inter-litter sources, then, is that the environment acts rather closely with developmentally-early gene action, enhancing or depressing the genetically controlled processes, thus providing very similar correlation coefficients to those of the genetic source. In addition to the effect on the early processes, this enhancing and depressing effect also influences the degree of general development of the organism as a whole, thereby determining to what extent development will progress, providing the subtle effects seen in figure 2.

The lesser alterations of axis shape in figure 2 under the influences of the intra-litter environment are perhaps due to the lesser severity of and different kind of environ-

mental effects on individuals within the litter. Perhaps a large proportion of the effects seen in the intra-litter portion is also due to errors of the human kind in making the original measurements.

Effects on Selection

If the tentative conclusion reached from these data is true, i.e., that the environmental effects simulate gene action in morphogenesis, then the process which WADDINGTON (1952) has described as "selection of the genetic basis for an acquired character" may be a more common type of selection than previously supposed. If the environment aids the population to explore other genetically possible forms than represented in the population's array of genotypes evolutionary progress would be much enhanced. This would be tantamount to increasing the genotypic range or the unmasking of extreme forms which would ultimately be genetically attainable through selection. This possibility also suggests that a multiplicity of environments would provide the animal breeder in his selection program with a broader basis of evaluating the possible-attainable genotypes in his present animal population.

Such a possible effect is admittedly contrary to the concept of environmental hindrance in evolutionary and animal breeding processes. On the other hand, it is difficult to find an investigation where the environment has been directly examined in regard to its effect on genotypic range. The question of the effects of selection under varied environments has been frequently discussed (see FALCONER 1952). The present suggestion is concerned however, with a multiplicity of environmental conditions rather than an optimal one. A strong environment-genotype interaction would, of course, interfere with such a process and certainly limit its effectiveness. However, the present data suggest the existence of very little of this type of interaction for such an interaction would be expected to decrease the homogeneity between the sets of correlation coefficients.

SUMMARY

The axis and innominate bones were collected from mice of 18 inbred strains. Measurements of several dimensions of the axis as well as the innominate length were made to furnish the data required in the analysis of the genetic and environmental influences on the shape of the axis.

The analyses of variance and covariance provided information for estimating variabilities, heritabilities and intercorrelations of the dimensions separately for each source of control, i.e. inter-strain, inter-litter and intra-litter.

The inter-strain variances (genetic) were highly significant for all dimensions.

The inter-litter variances (environmental) that were significant were localized to laterad and associated measurements.

Rankings of the magnitudes of the variances of the dimensions were similar for the genetic and environmental sources.

The centrum and adjacent measurements had the higher variabilities, the neural arch measurements had the lower variabilities.

Homogeneity tests of the correlation coefficients indicated that coefficients within a set (source of control) were heterogenous but that corresponding coefficients be-

tween sets showed no evidence of being heterogeneous. This indicated a strong similarity between environmental and genetic correlation coefficients.

Theoretical responses of the axis shape to "selection" (arbitrary increase of a dimension) were diagrammed and consistent trends were indicated.

The possible relationships between environmental effects and gene action with their consequences for selection, natural and artificial, were discussed.

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