A Twin Study of Palatal Dimensions Partitioning Genetic and Environmental Contributions to Variability

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Since the time of Galton, human twins have provided a natural experimental population for elucidation of nature-nurture questions and for study of genetically complex anthropometric characteristics. Traditionally, estimates of genetic contribution to variability for human metric traits, such as maxillofacial dimensions, have depended on contrasting variability between monozygotic (MZ) twins with that between dizygotic (DZ) twins. In light of the apparent relationship between palatal dimensions and several congenital malformations,[1-3] it seemed appropriate to obtain estimates of sources of variability for these dimensions. Perhaps even more meaningful, the method used in this study serves to illustrate the utility of a technique that has been insufficiently exploited in studies of human craniofacial growth and development. Using analyses described here, those anthropometric traits susceptible to more precise genetic analysis and those for which specific environmental factors must be sought can be identified.

LITERATURE REVIEW

Palatal form and its genetic basis have long been of interest, but only a few investigators have used the twin approach in its analysis.[4-8] In general, intrapair variability of palatal dimensions was greater for DZ twin pairs than for MZ pairs. Most recently, Hunter[8] analyzed cephalometric data from 35 DZ and 37 MZ twins. Fourteen craniofacial depth dimensions and 12 craniofacial height dimensions were studied. Measurements of facial skeletal height demonstrated a significantly higher component of genetic variability than did depth dimensions.

In most studies simple heritability estimates were used:

$$H = \frac{\text{Variance DZ} - \text{Variance MZ}}{\text{Variance DZ}}$$

i.e., the difference between the average variability between DZ twin pairs and MZ twin pairs as a percentage of DZ variability.[9] Because of reservations concerning these estimates, Osborne and De George[10] chose not to calculate H values. They felt that in twin studies statistical methods employed should require the fewest possible assumptions and chose therefore to apply simple variance analyses to the various intrapair differences. Their method enabled comparison of observed intrapair twin differences with measurement error (ME) and with the equivalent of unrelated individuals, and permitted testing for sex differences. Furthermore, they felt that heritability estimates provided no information of importance not obtained by probability levels of variance ratios.[11] Accordingly, much of the methods of analysis used here were those described by Osborne and De George.[10]

METHODS

Sample.—In order to be useful, it is necessary that the number of twin pairs be relatively large, and I was fortunate that dental casts of 102 twin pairs accumulated in two earlier twin studies were made available for the present study by Dr. W. S. Hunter, Ann Arbor,

Table 1 Source and subtypes of 102 adult twin pairs

Source	MZ Male	MZ Female	DZ Male	DZ Female	Subtotal
Michigan	12	21	5	15	53
New York	15	15	8	11	49
Subtotal twin types	27	36	13	26	
TOTAL	63		39		102

Michigan, and Dr. S. L. Horowitz, New York, New York. The twin pairs are tabulated by source, zygosity and sex in Table 1. Each source possessed more twin pairs than used in the present study. Edentulous or partially edentulous cases were excluded as were several cases in which casts revealed the use of orthodontic appliances.

Zygosity diagnosis.—Methods used to establish zygosity of the twins studied have been described previously.[10,12,13]

Measurements.—Each plaster model was measured to obtain values for palatal height, width and length using a procedure previously reported.[14] A horizontal plane was established by contact with three reference areas: the lingual-cervical lines of the two maxillary first molars and the labial tip of the interdental papilla between the maxillary central incisors. Palatal width was the distance between the first molars. Palatal height was measured from the established horizontal plane to the posterior boundary of the hard palate in the midline. Palatal length was the distance between the central incisors on the labial and the junction of the hard and soft palates. At least three measurements were made of each dimension and their means recorded.

Analyses.—The variability which exists for measurements of any metric trait in a population may be considered the sum of the following sources of variability: measurement error (ME), intrafamilial genetic differences (G), intrafamilial environmental differences (E), and extrafamilial (P) (environmental and genetic) differences. However, it is not always possible for a particular trait to detect the contribution of each of these sources. Yet it is necessary, before detailed study of environmental or genetic factors involved in the development of a complex trait, to know whether the gross contribution of all environmental factors to variability can be detected. Accordingly, it was the aim of the analyses used here to determine which sources of variability could be detected for each dimension studied. Since identification of detectable sources of variability for a particular trait is a logical prelude to more detailed studies, these analyses may be thought of as a screening procedure. In addition to determining which sources of variability can be detected, the relative magnitude of these sources can be estimated for each trait.

Estimates of measurement error variances [V(ME)] (for each measurement in each sex) were taken from analyses of variance of factorial experiments designed to test accuracy and consistency of palatal measurements.[15] Factors analyzed were the effect on measurements of the examiner, the instrument, and the state of the palate (intraoral or dental models). In the analysis of variance, V(ME) was equivalent to the residual mean square. V(ME) estimates were more conservative (that is, larger) than those obtained from mere replication of measurements, since more variables were examined. It should be noted that replicated measurements would suffice for V(ME) estimates.

Variability due to intrafamilial environmental differences was estimated by obtaining the average variability between MZ twins [V(MZ)]. Since MZ twins are assumed to possess identical genetic material, any difference between them must be attributable to environ-

mental differences. However, V(ME) is included in differences between MZ twins. Therefore, intrafamilial environmental factors were represented by the difference between V(MZ) and V (ME). If the difference between V (MZ) and V(ME) was statistically significant, then environmental factors were considered detectable. If they were detectable, search for specific environmental agents in development of the trait was indicated.

The difference in average variability between DZ twins [V(DZ)] and between MZ twins may be used as an estimate of the intrafamilial genetic portion of total variation. Again, V(MZ) is due to environment and measurement error. It is assumed that the average environmental differences for DZ twins and for MZ twins are the same. The average variation between DZ twins is, then, due to environment, plus measurement error, plus genetic differences:

$$V(DZ) = E + G + ME, \text{ and}$$

$$V(MZ) = E + ME.$$

Therefore, the differences between V (DZ) and V(MZ) are theoretically due to genetic sources. If V(DZ) was significantly greater than V(MZ), then a genetic source of variation was considered detectable. If a genetic source was detectable, then one could consider more precise genetic analyses in families. If no genetic source of variation is detectable, family studies would seem to be fruitless.

The difference between total population variability and that between sibs can serve as an estimate of extrafamilial (genetic and environmental) source of variability since

$$V(\text{sibs}) = E + G + ME, \text{ and}$$

$$V(\text{population}) = E + G + ME + P.$$

Average differences between DZ twins were used to estimate total intrafamilial variability since DZ twins are no more alike genetically than are any two sibs. Since each pair represented a different family, the variability of all pairs served as an estimate of the general population. Only DZ pairs were used for obtaining an estimate of the population since they provide a more conservative comparison with DZ intrapair variances. If the population or interpair variances [V(IP)] were significantly greater than V(DZ), then one would conclude that extrafamilial factors were detectable.

In order to learn something about sex influence on these dimensions, a comparison of intrapair variances between MZ-male (MZ-M) and MZ-female (MZ-F) twins and between DZ-M and DZ-F twins was made. The comparison of male and female MZ twins was a potential test for sex influences on extragenic differences. Similarly, a comparison of DZ twins served as a test for sex influences on genetic differences.

Appropriate F ratios were calculated for each of the above variance comparisons and probability values were obtained.[16]

Results

In Tables 2a, 3a, and 4a the following data are tabulated for palatal height, width and length, respectively: V(IP), V(DZ), V(MZ) and V(ME). The following F ratios and probability values are listed in each table: V(IP)/V(DZ), V(DZ)/V(MZ), and V(MZ)/V(ME). Males and females were analyzed and are tabulated separately.

Height.—The significant difference between V(MZ) and V(ME) in males and females indicated that measurement of palatal height was sufficiently accurate to detect extragenic influences producing differences between MZ twins. Based on V(DZ)/V(MZ) ratios, genetic contribution to variance was detectable in females but not males.

Table 2a Palatal height mean variances

	No. twin pairs	Variance	F ratio	p
Male				
Measurement error (ME)	13	.311		
MZ/ME			4.63	<.01
Monozygotic (MZ)	37	1.440		
DZ/MZ			1.41	<.25
Dizygotic (DZ)	13	2.032		
IP/DZ			8.87	<.01
Interpair (IP)	12	18.013		
Female				
Measurement error (ME)	14	.339		
MZ/ME			3.51	<.01
Monozygotic (MZ)	36	1.190		
DZ/MZ			2.36	.01
Dizygotic (DZ)	26	2.803		
IP/DZ			3.91	<.01
Interpair (IP)	25	10.969		

Table 2b Palatal height sex comparisons

	No.	Variance	F ratio	p
Monozygotic				
Male	27	1.440		
M:F			1.21	>.25
Female	36	1.190		
Dizygotic				
Male	13	2.032		
M:F			1.38	>.25
Female	26	2.803		

The difference between V(IP) and V(DZ) was highly significant in both sexes and indicated that extrafamilial factors, genetic and extragenetic, provided the greatest contribution to variability of height. The absence of difference between males and females suggested that sex had no influence on genic and extragenic factors (Table 2b). As noted in Table 5, the sources of variability for palatal height had the following relative magnitude: EXTRAFAMILIAL > GENETIC > ENVIRONMENTAL.

Table 3a Palatal width mean variances

	No. twin pairs	Variance	F ratio	p
Male				
Measurement error (ME)	13	.164		
MZ/ME			14.23	<.01
Monozygotic (MZ)	27	2.326		
DZ/MZ			1.33	.25
Dizygotic (DZ)	13	3.105		
IP/DZ			4.24	<.01
Interpair (IP)	12	13.154		
Female				
Measurement error (ME)	14	.140		
MZ/ME			14.20	<.01
Monozygotic (MZ)	36	1.988		
DZ/MZ			1.64	<.10
Dizygotic (DZ)	26	3.264		
IP/DZ			2.25	<.05
Interpair (IP)	25	7.329		

Table 3b Palatal width sex comparisons

	No.	Variance	F ratio	p
Monozygotic				
Male	27	2.326		
M:F			1.17	>.25
Female	36	1.988		
Dizygotic				
Male	13	3.105		
M:F			1.05	>.25
Female	26	3.264		

Width.—The ME variances were quite small in both sexes and significantly smaller than the MZ variances, thus permitting the detection of extragenic causes for differences between MZ twins. The hereditary component of variation as judged by the V(DZ)/V(MZ) ratios was not strong although in females V(DZ) was significantly greater than V(MZ). Extrafamilial fac-

Table 4a Palatal length mean variances

	No. twin pairs	Variance	F ratio	p
Male				
Measurement error (ME)	13	.519		
MZ/ME			7.82	<.01
Monozygotic (MZ)	27	4.006		
DZ/MZ			.96	>.25
Dizygotic (DZ)	13	3.833		
IP/DZ			1.09	>.25
Interpair (IP)	12	4.167		
Female				
Measurement error (ME)	14	.620		
MZ/ME			6.82	<.01
Monozygotic (MZ)	36	4.228		
DZ/MZ			1.02	>.25
Dizygotic (DZ)	26	4.320		
IP/DZ			1.35	<.25
Interpair (IP)	25	5.822		

Table 4b Palatal length sex comparisons

	No.	Variance	F ratio	p
Monozygotic				
Male	27	4.006		
M:F			1.06	>.25
Female	36	4.228		
Dizygotic				
Male	13	3.833		
M:F			1.13	>.25
Female	26	4.320		

tors made the largest contribution to variance of any source since V(IP) was significantly greater than V(DZ) in both sexes. The lack of significant difference between males and females (Table 3b) reflected the absence of sex influences on genetic factors (DZ twins) and extragenic factors (MZ twins) in the determination of this trait. In Table 5 it may be noted that extrafamilial fac-

Table 5 Relative magnitude of sources of variation for dimensions

Source of Variation	Estimate General	Height male	Height female	Width male	Width female	Length male	Length female
extrafamilial	$V_{IP} - V_{DZ}$	16	8	10	4	.3	1.5
genetic	$V_{DZ} - V_{MZ}$	1.5	1.6	.8	1.3	-.2	.1
environmental	$V_{MZ} - V_{ME}$	1.1	.9	2.1	1.8	3.5	3.6
error	V_{ME}	.3	.3	.2	.14	.5	.62

tors were the largest source of variation in palatal width in both sexes. Of these, environmental factors were apparently of greater magnitude than genetic. The relative strength of the factors contributing to palatal width variability was therefore: EXTRAFAMILIAL > ENVIRONMENTAL > GENETIC.

Length.—ME variances were greater for palatal measurements of length than for the other dimensions. Nevertheless, they were sufficiently small (and/or environmental factors were sufficiently great) to permit detection of extragenic causes of differences between MZ twins. The virtual equality between V(DZ) and V(MZ) in both sexes is indicative of an exceedingly small relative contribution of heredity to the total variance for palatal length. Furthermore, the IP variances were very small and the resultant low V(IP)/V(DZ) F ratios indicated that there was no difference between variance of DZ twins and variances of all twin pairs. Accordingly, a contribution to variability of palatal length by extrafamilial factors appeared to be slight. As with height and width, no apparent sex influences were revealed. The relative magnitude of sources of variability (Table 5) was ENVIRONMENTAL > EXTRAFAMILIAL > GENETIC.

In Table 6, probability values for those measurements which provided statistically measurable variability are indicated: those which measured extrafamilial variability (genetic and environmental), those which measured genetic variability, and those which measured environmental influences. The probabilities with which these sources of variability are detectable are given in the body of the table.

Discussion

Despite its inclusion as a basic tool in human genetics for nearly one hundred years, criticism of the twin approach has been the subject of numerous reviews.[10, 17-22] Included among areas of potential bias inherent in twin studies are accuracy of zygosity diagnoses,[10,20] the possibility of a third type of twinning,[19,23] the assumption that the magnitude of environmental differences between MZ and DZ twins are the same, i.e., that MZ and DZ twins are equivalent except for heredity,[19,22] and the possibility of constitutional inferiority of MZ twins.[24] Moreover, the validity of statistical assumptions used in obtaining heritability estimates has been questioned.[21] Vandenberg demonstrated a lack of stability in variance estimates and F ratios.[25] He compared results obtained from several similar studies and found discrepancies in conclusions concerning the heritability of various anthropometric traits. The instability of these estimates may be due

Table 6 Statistically measureable variability*

Source	Palatal Dimensions					
	height		width		length	
	male	female	male	female	male	female
EXTRAFAMILIAL (IP/DZ)	<.01	<.01	<.01	<.05	---	<.25
GENETIC (DZ/MZ)	<.25	.01	---	<.10	---	---
ENVIRONMENTAL (MZ/ME)	<.01	<.01	<.01	<.01	<.01	<.01
SEX INFLUENCES	---	---	---	---	---	---

*probability values in body of table from tables of F distributions (Dixon and Massey, 1959) for appropriate F ratios and degrees of freedom.

---in table indicates that p = or >.25

to sample sizes and/or to real differences in different populations. Because of these and other difficulties, Lenz[21] concluded that "even upon application of the most perfected statistical procedures and upon consideration of all known contributing factors, all attempts at a quantitative solution of the problem of heredity are doomed to failure." Allen[22] was less pessimistic when he admitted that "sampling biases must always be assumed in twin studies," but although "Only qualitative conclusions are likely to have general validity . . . quantitative conclusions about twins themselves may be useful in constructing or excluding general hypotheses."

In addition to biases, a variety of limitations of twin studies which mitigate their usefulness has been recognized.[19] Neither information about genotypes nor identification of specific environmental factors can be obtained and only rarely can genetic hypotheses be tested. Furthermore, inferences from twin results must usually be restricted to the sample studied. The aforementioned biases and limitations, in addition to reported discrepancies between twin data and other genetic techniques, led Neel and Schull[18] to state that: "In its present context, the twin method has not vindicated the time spent in the collection of such data." Nevertheless, the recently acquired concept of Downs' syndrome,[26] a condition previously commonly used to demonstrate the shortcomings of heritability estimates based on twin data, has provided support for the use of twins. Neel and Schull[18] compiled estimates from various sources and calculated a heritability estimate for Downs' syndrome of 0.881. This exceedingly high value was clearly contradictory to the minor role of heredity indicated by demonstrably important environmental factors such as maternal

age and health that had been convincingly related to the condition. However, "twin data published by Orel, together with the nature of the disorder, apparently prompted Waardenberg's early suggestion of a chromosomal etiology,"[22] which was confirmed by Lejeune and others twenty-seven years later.

The validity of twin data was thus clearly demonstrated in the case of Downs' syndrome. However, this unique example is not sufficient in itself to justify disregarding the biases and limitations noted. Probably a more meaningful justification for the use of twins in human genetics is the virtual absence of any other genetic method for answering questions about the relative role of genetic and environmental factors that contribute to the development of complex traits such as those examined in the present study. Furthermore, "Despite the many difficulties of twin research . . . no material but twins can provide such convincing evidence for environmental etiologic factors prior to demonstrations of the factors individually."[22]

In the majority of studies in which, in spite of its shortcomings, twin material has been used, a "heritability" constant, usually defined as that proportion of the total variance in a trait due to genotypic variance, has been calculated. The closest approach to heritability in twin studies is the proportion of variance between DZ pairs that is lost when the genotype is held constant.[9] This formula has been subject to much criticism, e.g., between-family environmental variance is omitted and some portions of environmental variance are held constant in MZ twins along with the genotype.[22,23] Besides, its standard error is usually not given and is difficult to compute, so that heritability is not very useful even if it was genetically meaningful. Since the only actual use of these estimates is the detection of measurable hereditary variability, the variance ratio[10] which provides the same information may be used. This approach, although still subject to environmental effects, is less misleading than heritability and, more important, lends itself to tests of significance. Furthermore, information about the accuracy of measurements [V(MZ)/V-(ME) ratio] and familial factors [V-(IP)/ V(DZ) ratio] can also be considered.

As stated by Kempthorne and Osborne:[23] "The initial problem for the analysis of twin data is to determine whether genetic and environmental components of variability can be measured by the technique employed and whether the components estimable from various types of data are consistent with a genetic model." Accordingly, it seemed that the analyses used by Osborne and De George[10] were most appropriate for examining palatal height, width and length, traits which are obviously the culmination of many genetic and environmental factors.

The highly significant V(MZ)/ V(ME) ratios suggested that the measurements were sufficiently accurate to permit detection of environmentally caused differences between MZ twin pairs. Since V(ME) were absolutely quite small, it is probable that had the environmental factors been less potent, they still would have been detectable.

The MZ and DZ comparisons were essentially conventional heritability estimates, significant V(DZ)/V(MZ) ratios suggesting a relatively large genetic component of variability in a population for a trait. However, the ratio used here avoided the inferences implicit in "heritability" and provided, rather, information as to whether genetic factors could be detected. In reality, similar conclusions are drawn but with F ratios probability statements can be made.

Based on V(DZ)/V(MZ) ratios, it is probable that in the sample studied the genetic contribution to variability of palatal height and width can be detected, although this conclusion can be stated with confidence only for palatal height in females. It is of interest that the probability that MZ twins were less variable than DZ twins was greater in females for height and width. The larger female sample size was probably responsible for this finding. Palatal length findings indicated that either genetic influence on variability is exceedingly small or environmental factors are quite significant in producing variability, since no difference between MZ and DZ was detected in either sex.

V(IP) were chosen to represent variances for the dimensions in the population from which the twin samples were chosen and DZ twin pairs were more or less representative of intrafamilial relationships. It is intuitively apparent that the major source of variability for a trait in a population would be derived from extrafamilial factors, i.e., whether genetic or environmental factors predominate, it is expected that the variance for a trait among unrelated individuals would be greater than that within a family. Therefore, where V(IP) was significantly greater than V(DZ) one would conclude, as expected, that both genetic and environmental factors in different families accounted for the variation among the families to a much greater extent than comparable factors within families. However, significant findings for these ratios permitted no further conclusions since factors accounting for variability among families cannot, at present, be resolved. Palatal height and width results were as generally expected: variances among unrelated individuals were significantly greater than between DZ pairs. However, V(IP) for palatal length in males was apparently no greater than V(DZ) ($p = >.25$) and in females the greater V(IP) was not significant ($p = <.25$).

Table 7 Within-pair variability (s) and heritability (H) estimates for palatal dimensions in twins*

(from Lundstrom, 1948: pp. 121, 127)

	DZ		MZ		
Dimension	N	s	N	s	H
Width P_1	40	2.6	58	1.1	.82
Width M_1	44	2.6	60	1.8	.52
Length	44	3.0	60	1.3	.81
Height	44	1.9	60	1.0	.72

* $H = \frac{(s_{DZ})^2 - (s_{MZ})^2}{(s_{DZ})^2}$

Several explanations for the small length V(IP) and insignificant V(IP)/V(DZ) F ratios exist. Since length variances (male and female) in normal adults[14] were significantly greater than V(IP), also an estimate of the population, and because twin sample sizes were fairly small, it is probable that small V(IP) were due to sampling.

In no earlier studies of palatal dimensions in twins were the same landmarks used nor were comparable analyses carried out as in the present study. Nevertheless, Lundstrom's data is germane.[6] His findings revealed significantly larger mean intrapair variances in DZ than MZ twins for palatal height and width and *arch* length. For comparative purposes I calculated heritability estimates (H) from his data (Table 7) and from the data reported here (Table 8). In both studies H for height and width were greater than .25, but in Lundstrom's study, estimates were much larger. Length findings in the two studies were even more disparate than height and width: H (Lundstrom) = .81 and H (present study) = .00. These differences can be attributed to real differences since "heritability is always valid only for the population in which it was determined,"[21] but more specific factors may be involved. The landmarks from which the various measurements were taken were some-

Table 8 Heritability estimates (H)* for palatal dimensions

	Males	Females
Height	.29	.58
Width	.25	.39
Length	-.05	.02

$$*H = \frac{V_{DZ} - V_{MZ}}{V_{DZ}}$$

what different in the two studies, but these differences seem too slight, in the case of height and width, to account for large differences in heritability estimates. Probably more meaningful was the age difference in the two series. In Lundstrom's material ". . . the comparison (was) . . . based on the . . . age group constituted by the age 12-15 years. This is a disadvantage and it should, of course, be of more interest to examine older twins for whom the existing differences could be considered more definite . . . however . . . the number of older fraternal twins is too small."[9] In the present study, on the other hand, the youngest individuals were 16 years of age and the majority were greater than 21 years. It was not unexpected that Lundstrom arrived at greater heritability estimates in light of those twin studies of growing children "in which practically all measurements give significant differences between the mean intrapair difference of monozygotic and those of dizygotic twins, due, presumably, to the facts that most body proportions are undergoing growth changes, and growth rates and patterns of development are largely genetic."[10]

Although the factors already mentioned might also apply to palatal length, Lundstrom's measurement (labial of central incisors to a line between the central fossas of the first molars) was so different from that used here that comparison between the two is probably not valid. Nevertheless, both heritability estimates may be approximately correct as far as the genetic contribution to variability of palatal length is concerned. If they are, it would not be unreasonable to infer that the genetic source of variability of that portion of the palate anterior to the first molars is greater than that for the remaining posterior part.

The greater contribution of genetic factors to variance of palatal height in comparison with length found in the present study supports Hunter's conclusion[8] that genetic contribution to variability is greater for height than for depth dimensions.

Innumerable genetic and environmental factors are involved in craniofacial growth and development. The confluence of these factors in the production of a wide range of normal variability makes human craniofacial growth and development particularly resistant to analysis. In the foregoing study and discussion I have attempted to illustrate the use that might be made of twin material in analyses of complex traits. By partitioning sources of variation, through the use of twins, information can be obtained which may suggest which craniofacial dimensions are suitable for subsequent genetic study. Similarly, those dimensions may be distinguished for which environmental variability is most potent, and for which search and analysis of environmental variables are required.

Summary

Although biases and limitations inherent in twin analyses are recognized, the complexity of most craniofacial traits precludes application of more precise genetic techniques. Through the

use of twins and employing analyses suggested by Osborne and De George,[10] it was possible to obtain probability levels concerning the detectability of genetic, environmental, and/or familial contributions to variability of palatal height, width and length.

Detectable environmental contribution to variability of each of the dimensions was demonstrated by the significant mean monozygotic intrapair variance [V(MZ)]/ mean measurement error variance [V(ME)] F ratios. The lack of significance of most mean dizygotic intrapair variance [V(DZ)/ V(MZ)] F ratios revealed that genetic contribution to variability of these dimensions was not so readily identifiable. Mean interpair variances [V(IP)] were significantly greater than DZ variances for height and width, thus confirming the relatively large source of variation due to familial (environmental and genetic) factors. However, V(IP)/V(DZ) F ratios were not significant for palatal length, probably due to the very small IP variances.

Heritability estimates for each dimension were calculated from MZ and DZ variances for each dimension and were smaller than those found in previous studies.

No evidence of a sex effect on either genetic or environmental sources of variation was noted.

Analyses of twin material such as those described here provide a method of partitioning sources of variability for complex traits. These techniques are preliminary but prerequisite to more precise study of complex craniofacial characteristics.

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