

A Theoretical Note on Sex Linkage and Race Differences in Spatial Visualization Ability

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Evidence on the poorer spatial visualization ability in various Negro populations compared to the white populations and on the direction and magnitude of sex differences in spatial ability relative to other abilities suggests the genetic hypothesis that spatial ability is enhanced by a sex-linked recessive gene and that, since the 20–30% admixture of Caucasian genes in American Negroes came mostly from male white ancestors, relatively fewer X-linked than autosomal Caucasian genes were transmitted to the American Negro gene pool. The genetic model as explicitly formulated indicates the kinds of data which could substantiate or disprove the theory, but which do not now exist.

KEY WORDS: spatial ability; sex linkage; race.

INTRODUCTION

The genetic study of racial differences in mental abilities is encumbered by great technical difficulties, not the least of which is that genetic variation affecting most mental traits is due to segregation of genes at several (probably many) loci. If the influence of single genes on specific components of mental ability could be reliably discerned, this would open the possibility of estimating gene frequencies in different populations and of fitting mental test data to specific quantitative-genetic models. An important advance in this direction is the demonstration by Bock (1973) and Bock and Kolakowski (1973) of a sex-linked major gene influence on spatial visualization ability. The purpose of the present note is to point out some theoretical implications of this finding for the genetic investigation and interpretation of the

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reportedly greater deficit of American Negroes, relative to whites, in spatial ability than in other abilities.

SEX LINKAGE OF SPATIAL ABILITY

Spatial visualization is one of the most clearly established factors of mental ability (Smith, 1964; Vernon, 1950, pp. 64-75). As measured by tests such as Thurstone's Primary Mental Abilities and the Differential Aptitude Tests, the spatial factor can be most specifically characterized as the ability to visualize the appearance of an object after the object has been rotated in the plane or in three-dimensional space. The spatial factor is best measured by the tests specifically designed for this purpose and validated in factor analytic studies, such as the Thurstone tests, but the spatial factor also enters into performance on many other nonverbal tests, such as figure copying, formboard, matrices, block designs, and mechanical aptitude. Spatial ability also may enter into school subjects such as geometry, mechanical drawing, organic chemistry, and graphic arts.

Vernon (1950, p. 67) notes that the one point on which all researchers on spatial ability agree is that girls and women perform less well than boys and men in tests of the spatial factor. In fact, the presence of the sex difference has been used as an indicator that a test is loaded on the spatial factor. No other ability identified by factor analysis shows so consistent and marked a sex difference (as much as $\frac{2}{3}$ SD) in favor of males as do spatial tests.

A number of investigations have suggested that spatial ability is genetically sex-linked (Corah, 1965; Hartlage, 1970; Herzberg and Lepkin, 1954; O'Connor, 1943; Stafford, 1961). Most recently Bock and Kolakowski (1973) have presented evidence which, if confirmed by independent data, is of great interest. Their genetic analysis of family data, consisting of correlations between all the possible pairs comprised of mother, father, brothers, and sisters, leads them to the conclusion that an appreciable part of the variance in spatial ability is attributable to a single gene locus on the X chromosome. The gene that enhances spatial ability is recessive. (Note that it is the single enhancing gene that is recessive with respect to spatial ability, and not the characteristic itself, which could conceivably be produced by a variety of gene combinations.)

Since females inherit XX, the enhancement of the trait (spatial ability) will appear only if both X chromosomes carry the gene; otherwise, the dominant (nonenhancing) gene is manifested phenotypically. Males, however, inherit XY (i.e., X from their mother and Y from their father), and since the Y chromosome carries almost no genes except those that determine the individual's sex during embryogeny there is probably no gene in

the Y chromosome which dominates the recessive gene for spatial ability carried on the X chromosome. Therefore, only a single recessive gene in the male would be sufficient for the phenotypic manifestation of the gene for spatial ability. This means that females must inherit the recessive gene from both parents in order to manifest enhancement of the trait, but males need only inherit it and indeed can inherit it only from the mother. This is what is meant by sex-linked or X-linked inheritance.

RACIAL DIFFERENCES IN SPATIAL ABILITY

Reviewing the evidence comparing mental abilities in American Negroes and whites, Tyler (1965, pp. 318-319) notes that the tests on which Negroes perform most poorly, relative to whites and relative to Negro performance on other tests, "suggests some inadequacy in perceptual development." This is not easily explained in terms of environmental disadvantages associated with the generally lower socioeconomic status of American Negroes, since, as Bock and Kolakowski (1973) point out, among all the Primary Mental Abilities spatial ability is one of the least correlated with social class in the white population. Yet spatial ability is the one ability which most consistently shows the largest white vs. Negroes mean difference (in relation to the white standard deviation), even when socioeconomic differences are controlled. For example, the block designs subtest of the Wechsler Intelligence Scale, which is the most spatially loaded subtest in the whole battery, is the most difficult test for Negroes (Shuey, 1966, p. 71). The block designs test also shows a significant sex difference in favor in males, as should be theoretically expected for an X-linked recessive trait (Matarazzo, 1972, pp. 352-355). One study (Tenopyr, 1967) comparing Negroes and whites on spatial and other abilities controlled socioeconomic status both by selection of subjects and by partialing out a detailed index of SES from the point-biserial correlations between race and ability. The correlation between spatial ability and the index of socioeconomic status within each racial group was nonsignificant. The correlation between spatial ability and race, with the effect of socioeconomic status partialled out, was 0.21 ($p < 0.01$). Even when verbal IQ was statistically controlled (simultaneously with socioeconomic status), the race \times spatial ability correlation was 0.19 ($p < 0.01$), corresponding to a mean group difference of almost $\frac{1}{2}$ SD.

Jamaican Negroes, who, like American Negroes, are a hybrid population derived mainly from African and European stocks, show their poorest performance on the formboard test among a battery of 16 mental tests (Vernon, 1969, p. 172). The formboard test has the largest loading on spatial ability of any of the tests in the battery. In the same study, another

spatial test, Kohs block designs, also yielded very low scores. Performance on these spatial ability tests was far below the mean for all the other tests in the battery, by almost 1 SD. Vernon (1969, p. 184) gave the same battery of 16 tests to African Negroes in Uganda who had no Caucasian ancestry. Interestingly, the Kohs blocks and the formboard scores, while on the low side, are not far from the average score for the whole battery and, in fact, are higher than the scores on three nonspatial tests. Thus the test profiles of the Ugandan and Jamaican groups differ most in the two spatially loaded tests. In this respect, the Jamaican Negroes are similar to American Negroes. Whether this difference in mental ability profiles between the East African Ugandan Negroes, on the one hand, and the hybrid Jamaican and American Negroes, on the other, is due to the latter groups' admixture of Caucasian genes is not known, but it is suggestive and is consistent with the hypothesis presented in the following section. The original Negro slave populations of America and the West Indies were not derived from the East African stock represented by Uganda, but from West Africa. Unfortunately, data comparable to those reported by Vernon on Ugandans and Jamaican Negroes are not available on West African Negroes without any Caucasian ancestry. Such data would be needed in order to actually test the predictions indicated in the following section.

The sex difference in spatial ability within the racial groups is also theoretically relevant. The only appropriate data on this point are from a study by Baughman and Dahlstrom (1968, pp. 56-57), in which Thurstone's Primary Mental Abilities Tests were administered to groups of American white and Negro children, ages 7-14. The Primary Mental Abilities consist of five relatively factor-pure tests: Verbal Meaning, Perceptual Speed, Number Facility, Reasoning, and Spatial Visualization. The scores on the Spatial Visualization test, expressed on an IQ scale (with mean = 100, SD = 15), are shown for boys and girls in the white and Negro groups in Fig. 1. It can be seen that the sex difference appears much greater in the white than in the Negro group. However, the sex differences in the Negro group should be evaluated in comparison to all the other tests, on which Negro girls scored higher than Negro boys. The cause of this consistent sex difference in the Negro group (which also appears, though to a lesser degree, in the white group) is not definitely known, as the writer has noted in a detailed discussion of this phenomenon (Jensen, 1971). In view of the generally higher test scores of Negro girls, it is therefore noteworthy, and consistent with an X-linked recessive gene effect, that Negro boys perform at least as well as Negro girls on the spatial test. Baughman and Dahlstrom summarize their findings: "The results of the sex comparisons . . . differ from those found on other PMA subtests. This [spatial ability] is the only subtest on which the white boys clearly do better than the white girls and

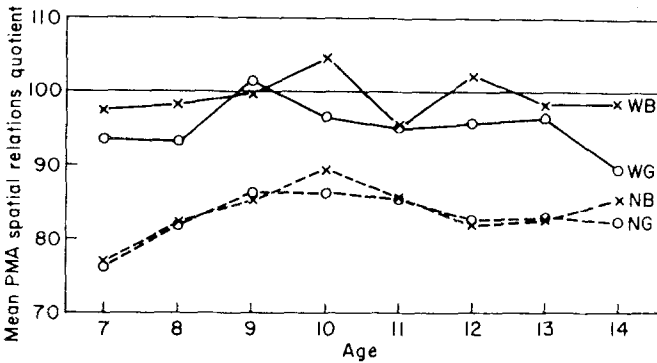


Fig. 1. Mean Primary Mental Ability Spatial Visualization scores for both sexes of both races at eight age levels. W, White; N, Negro; B, boys; G, girls. From Baughman and Dahlstrom (1968, p. 56).

on which the Negro girls do not surpass the Negro boys” (p. 56). Even when general ability level (as assessed by all PMA tests) is controlled, the sex difference is smaller in the Negro than in the white group. This point is relevant to predictions from a genetic model based on the X linkage of spatial ability.

All the foregoing evidence should be regarded as only suggestive in terms of the following discussion, since none of these studies was designed explicitly to test particular theoretical genetic predictions, and the data as reported do not permit statistical tests of goodness-of-fit to a theoretical model. Yet it is interesting that all the theoretically relevant differences turn out in the direction of expectation for an X-linked recessive in the particular racial populations under consideration. Undoubtedly an *ad hoc* explanation of such findings wholly in terms of environmental factors could be devised, but the significant point is that the evidence, under certain likely conditions, would seem to fit a genetic hypothesis which is not *ad hoc* but follows from preexisting established genetic principles. Spelling out the genetic model in some detail serves to highlight the gaps in our knowledge and to indicate precisely the kinds of evidence that would be needed to put the theoretical predictions to a proper test.

THEORETICAL FORMULATION

When there are equal numbers of males and females, two-thirds of the X-linked genes in a population are carried by females and one-third by males. If the recessive allele which enhances spatial ability is designated as *a* and the dominant, nonenhancing alleles as *A*, and if the proportions of the two types of alleles in the population are *q* and *p*, respectively, then the

genotype proportions for males and females will be as shown in Table I. If we arbitrarily assign the genic values of $A = 0$ (i.e., nonenhancement) and $a = +1$ (i.e., enhancement), and if there is complete dominance of A , then the genotype values will be as shown in the last column of Table I. It will be seen that for an X-linked recessive allele which enhances the trait (provided that its proportion is less than unity) males will have a higher mean genotype value than females, regardless of the proportions p and q . The male and female means are $q + k$ and $q^2 + k$, respectively, where q is the proportion of the trait-enhancing recessive gene in the population, and $q + (1 - q) = 1$, and k is the genotypic value of the autosomal component of spatial ability, which is the same for both sexes. The addition of k is, of course, necessary, since according to the present hypothesis the major gene effect of the X-linked recessive only *enhances* spatial ability, which is not assumed to be totally lacking in the heterozygote or the dominant hemizygote. The absolute sex difference therefore is $q - q^2$. As the value of q departs from $\frac{1}{2}$, either more or less, the *absolute* sex difference must decrease, since $q - q^2 = q(1 - q) = qp$, which is at a maximum when $p = q = \frac{1}{2}$. Of course, in statements about absolute differences we must assume that the genes act additively on the measurement and act similarly at different parts of the scale.

Sex linkage has implications for interracial crosses when the two parent populations contribute greatly unequal proportions of the fathers and mothers of the racial hybrids. This was clearly the case for American Negroes. Almost all American Negroes today are racial hybrids from African and European stock. American Negroes, on the average, have inherited some 16–20% of their autosomal genes from Caucasian ancestors (Reed, 1969). According to Reed, most of the Caucasian genes in the present American Negro gene pool were introduced during the period of slavery. As Stern (1973, p. 828) has noted, the Caucasian genes came primarily through the male. There was a strong taboo against miscegenation between Negro males and white females, but the reverse form of interracial mating was not uncommon.

Table I. Theoretical Population Proportions and Genotype Values for an X-Linked Recessive Gene

Sex	Genotype	Proportion	Genotype value
Male	A	p	0
	a	q	+1 (enhancement)
Female	AA	p^2	0
	Aa	$2pq$	0
	aa	q^2	+1 (enhancement)

As a consequence of this sexually asymmetrical form of interracial mating, the introgression of Caucasian genes into the Negro population would be proportionally only about two-thirds as much for X-linked genes as for autosomal genes. In a Caucasian \times Negro cross, where the Caucasian is male, the gene flow for autosomes is $\frac{1}{2}$; i.e., each race contributes equally to the hybrid offspring in the ratio 1:1, so the Caucasian contribution is $\frac{1}{2}$ for autosomal genes. For the X chromosome, however, the ratio of Caucasian (male) to Negro (female) contribution is $\frac{1}{2}:1$, so the Caucasian contribution to the hybrid offspring is $\frac{1/2}{3/2} = \frac{1}{3}$, which is two-thirds as much as the Caucasian contribution of autosomal genes; i.e., the ratio of Caucasian X-linked genes to autosomal genes is $\frac{1/3}{1/2} = \frac{2}{3}$. In other words, in the additive genetic effects of the racial admixture, the weighting of the Caucasian contribution would be about one-third less for X-linked traits than for traits conditioned by genes on all the other (autosomal) chromosomes. If the polygenic determinants of mental ability had different distributions in the original Negro and Caucasian parent populations, the hybrid offspring of interracial mating should show less effect of the racial admixture in those abilities that involve an X-linked component. If, say, three-fourths of the ancestry of American Negroes is African and one-fourth Caucasian, then the proportions of African and Caucasian genetic ancestry for X-linked characters would be only $\frac{2}{3}$ of $\frac{1}{4} = \frac{1}{6}$. In other words, the frequencies of Caucasian and African genes in the racial hybrids would be in the ratio of 1:3 for autosomal genes and of only 1:5 for X-linked genes. (This, of course, would not be true if the interracial matings were completely reciprocal with respect to all the possible race \times sex mating combinations.)

The above can be formulated in general terms as follows. If the introgression of Caucasian genes into the Negro population is through Caucasian male ancestors, the following gene frequencies would obtain in the hybrid American Negro population:

Autosomal:

$$p_r^h = M (p_r^C) + (1 - M) p_r^N$$

Sex linked:

$$q_k^h = (\frac{2}{3}) M (q_k^C) + [1 - (\frac{2}{3}M)] q_k^N$$

where p_r^h is the gene frequency of the r th allele at an autosomal locus for the hybrid American Negro population, p_r^C is the gene frequency of the r th allele for the Caucasian population, p_r^N is the gene frequency of the r th allele for the original Negro population, q_k^h is the gene frequency of the k th allele at a sex-linked locus for the hybrid American Negro population,

etc., and M is the present proportion of autosomal genes at the locus in question which are derived from the Caucasian population.

From the foregoing, we can show the differences in relative frequencies of the trait-enhancing genes for a given autosomal or a given sex-linked locus in the original Caucasian and Negro parent populations and in the hybrid Negro offspring. These differences are given in Table II. It can be seen that if $p_C - p_N = q_C - q_N$ the difference between the Caucasian and hybrid Negro gene frequencies will be greater for the X-linked than for the autosomal character. Regardless of the values of p and q , the rate of change toward greater genetic similarity of the two racial groups due to the introgression of Caucasian genes into the Negro gene pool during the period of greatest gene flow, which was during the time of slavery, would have been more rapid for autosomal than for X-linked characters. Just the opposite would have been true for a Y-linked gene, of which the only one known (aside from the genes involved in the development of maleness during embryogeny) is the gene for hairy pinna (i.e., rim of the ear). On the basis of this characteristic, of which the frequencies have been estimated in Caucasian and African populations, Slatis (1964) estimated that some 15% of the American Negro X chromosomes and 40% of their Y chromosomes are of Caucasian origin.

Thus far the discussion has treated the hybrid Negro population as if it were the F_1 generation of the racial cross, and under this condition, as pointed out, proportionally two-thirds as many Caucasian X-linked genes as autosomal genes would have introgressed into the Negro gene pool. But as Caucasian genes continued to flow into the already mixed Negro gene pool in subsequent generations, the rate of increase of Caucasian genes on the X chromosome would become gradually greater than two-thirds the rate

Table II. Differences in Relative Gene Frequencies Between Original Negro and Caucasian Stocks and Negro Hybrids for Autosomal and X-Linked Loci

Populations compared ^a	Differences in relative gene frequencies ^b	
	Autosomal	X-linked
$C-N$	$p_C - p_N$	$q_C - q_N$
$C-H$	$(1 - M)(p_C - p_N)$	$(1 - \frac{2}{3}M)(q_C - q_N)$
$H-N$	$M(p_C - p_N)$	$\frac{2}{3}M(q_C - q_N)$

^a C , Caucasian; N , original Negro; H , hybrid Negro.

^b p , Relative frequency (i.e., proportion) of trait-enhancing autosomal gene at a given locus; q , relative frequency of trait-enhancing X-linked gene at a given locus; M , proportion of Caucasian ancestors of hybrid Negro population.

of autosomal gene flow, because in more and more cases a Caucasian gene would replace another Caucasian gene already present in the hybrid population, consequently making no change in the frequency of Caucasian genes in the hybrid gene pool. As the Caucasian gene frequencies built up in the hybrid population, there would be a relatively greater deceleration of the rate of introgression of Caucasian genes for autosomal than for X-linked loci. But up to a total Caucasian admixture of 20–25% for autosomes (which is the proportion of Caucasian genes in American Negroes determined by Reed, 1969), this deceleration of the rates of introgression of Caucasian genes would make for very little difference between the F_1 generation and subsequent generations in the *relative* frequencies of X-linked and autosomal genes. It can be shown, by integration of the increments of change, that if the ratio of X-linked to autosomal genes derived from Caucasian ancestors is 2:3 for the F_1 generation, it will be at most 3:4 for the generation in which the total amount of Caucasian admixture has reached 25%.

As the model applies to the mean white–Negro difference in spatial ability, it involves a key assumption which must be made explicit. The theory is based on evidence, such as the work of Bock and Kolakowski (1973), that a substantial part of the variance in spatial ability among whites is attributable to segregation at probably one sex-linked locus. The application of the present model to the known considerable white–Negro mean difference in spatial ability assumes that the Negro deficit in this ability is also attributable to a dominant gene at this locus. It is, of course, possible that the Negro deficit is not attributable to the same locus that controls spatial ability in Whites, or even to sex-linked genes at all. But as a working hypothesis this would seem to be a reasonable and heuristic assumption.

As can be seen in Table II, the comparison of autosomal and sex-linked traits *per se* is not informative regarding the genetic hypothesis without knowing the frequencies (i.e., p and q in Table II) of the particular autosomal and sex-linked genes in the original populations. There is no *a priori* reason to assume that $p = q$, i.e., that the frequencies or the genotype values of autosomal abilities and X-linked spatial ability are equal in the original Negro stock. All we can be reasonably sure of is that the rates of change of autosomal and sex-linked genes have differed during the process of hybridization. If it is assumed that spatial ability is approximately equal to other (autosomal) abilities in the unmixed Negro stock, the known deficit in spatial ability relative to other abilities in present-day mixed American and West Indian Negroes is consistent with the theoretical expectations for a sex-linked trait, given the particular form of mating that was chiefly responsible for the hybridization of Negroes in

America. A similar expectation would hold for red-green color blindness.² The investigation of frequencies of color blindness in men and women in West African and in American Negro populations, especially where M (the proportion of Caucasian admixture) has been independently determined, would serve as a test of the model for a trait which is much less susceptible to measurement problems and environmental influences than is spatial ability. If the theoretical expectations were not upheld for color blindness, it seems most unlikely that they would be substantiated for spatial ability.

The absolute sex difference, $q - q^2$, where q is the relative frequency of the recessive trait-enhancing allele, is a maximum when $q = 0.5$. If we assume that the relative frequency q of the recessive gene that enhances spatial ability is not much greater than 0.5 in the Caucasian population, as estimated by Bock and Kolakowski (1973), the sex difference for the hybrid population should fall somewhere between the sex differences found in the white and unmixed Negro populations, and in various hybrid groups the size of the sex difference should be a monotonic function of M , the proportion of Caucasian admixture. For example, we should expect a smaller sex difference in spatial ability among Negroes in, say, Georgia, than in California, since in these two localities the proportions of Caucasian admixture are about 0.10 and 0.20, respectively (Reed, 1969). There is no theoretical genetic basis for a parallel sex difference in abilities conditioned by autosomal loci. It would be consistent with theoretical expectation if spatial ability showed a change in the magnitude of sex differences as a function of M while other abilities did not. A completely environmental theory to explain such a finding would most likely have to be *ad hoc* and probably quite farfetched as well.

Another genetic prediction concerns the correlations among various relatives on tests of spatial ability in the Negro population. Bock and Kolakowski (1973) estimated the frequency of the recessive gene that enhances spatial ability as 0.5 in the white population. If the recessive gene frequency is appreciably lower in the Negro population, as might be hypothesized from their lower performance on spatial tests, then one should expect lower correlations between relatives (i.e., all paired combinations of mothers, fathers, brothers, and sisters) than those found by Bock and Kolakowski in their white samples, and the correlations involving females should be depressed the most. Corresponding decreases in the

² The median rate of red-green color blindness in males in 33 Caucasian samples from Europe, Great Britain, and the United States is 0.08; only four samples fall below 0.07 and only six exceed 0.089. Samples from five populations of African Negroes ranged from rates of 0.017 to 0.030, with an average of 0.024. Five samples of American Negroes ranged from 0.028 to 0.042, with an average of 0.036 (Post, 1962). Unfortunately, Post does not give the corresponding rates of color blindness for females or independent estimates of the degree of Caucasian admixture in the American Negro samples.

variance of spatial ability in the Negro population, particularly for females, would also be expected. If these various genetic expectations were borne out by the evidence, one would be hard put to find a plausible environmental explanation. At present, however, no data have been reported in the literature that are suitable for testing the genetic predictions (see the Appendix).

SUMMARY

Evidence on spatial visualization ability in American, West Indian, and East African Negroes and on the direction and relative magnitudes of sex differences in spatial ability as compared to other abilities in Negro and white groups suggests a genetic hypothesis that could explain the observed phenomena.

The hypothesis is based on recent evidence for a major recessive sex-linked gene effect which enhances spatial ability, along with the fact that the 20–30% admixture of Caucasian genes in American Negroes came largely from male white ancestors, thereby resulting in the introgression of proportionally about one-third fewer X-linked than autosomal Caucasian genes into the American Negro gene pool.

Specific predictions from the hypothesis have been indicated. These concern sex differences in spatial ability in various Negro populations with different estimated proportions of Caucasian admixture, sex differences in variance, and the relative magnitudes of correlations between relatives.

Features of the existing evidence that are relevant to the hypothesis appear to be consistent with it, as far as this can be determined, but too many key parameters remain undetermined for the present evidence to be viewed as more than merely suggestive. Specific kinds of evidence are indicated as needed for substantiation or denial of the hypothesis. The genetic model has been explicitly formulated here as a guide to obtaining more suitable data for testing theoretical explanations of the already established phenotypic racial difference in spatial visualization ability.

APPENDIX

It is possible to estimate the size of samples that would be needed to test the various hypotheses suggested by this theoretical formulation. This, of course, requires that one make certain quantitative assumptions about the parameters involved in any particular theoretical prediction. Since the hypothesis of sex linkage of spatial ability predicts different kinship correlations than would be predicted for autosomal traits, it is a good place to begin our hypothesis testing. The whole structure depends upon confirmation of

the peculiar pattern of kinship correlations predicted for an X-linked character. The testing of this hypothesis can be used as an example of how one would go about estimating the required sample sizes in terms of the particular model to be tested.³

Assume that (a) the genetic variance is entirely attributable to an X chromosome pair of dominant-recessive alleles, (b) the frequency of the recessive allele is 0.25 in the Negro population (i.e., about half the value Bock and Kolakowski estimated for the Caucasian population), (c) the (narrow) heritability of actual scores in males is 0.5 (so for females h^2 would be 0.0952 under this model), and (d) the nongenetic variance is the same in males and females. Tables III gives the variances expressed as fractions of the total variance in males.

Given these assumptions, it is possible to calculate the theoretical values for the critical kinship correlations as shown in Table IV. The last two columns give the expected correlations if there were no X linkage but all the variance were autosomal, in the first case if all genetic variance were additive (as in the male X-linked case) and in the second case if the genetic variance were due to an autosomal dominant-recessive pair (or pairs) of alleles with $q = 0.25$ and variances as in the females for the X-linked case.

If comparisons between certain kinship correlations are to be made such that there is a 50% chance of finding a difference of twice the standard error between the two correlations being compared (i.e., a difference significant beyond the 5% level), the following sample sizes (number of pairs) would be needed: father \times son vs. father \times daughter = 335, mother \times son vs. mother \times daughter = 710, brother \times brother vs. brother \times sister = 271, sister \times sister vs. brother \times sister = 1865. For these N s we have assumed that the different combinations involved in a comparison are independent, which is not likely to be the case in actual practice, which would require somewhat smaller N s for confidence at the 5% level, since, if there were common ele-

³ I am indebted to Professor Everett R. Dempster for the following calculations.

Table III. Proportions of Variance Expected Under Model^a

Variance	Males	Females
Genetic		
Additive	0.5	0.06250
Dominance	0	0.09375
Nongenetic	0.5	0.5

^a See text for description of model parameters.

Table IV. Theoretically Expected Kinship Correlations Under Models of X-Linked Recessive, Autosomal Additive, and Autosomal Dominant-Recessive

Relationship	X-linked		Autosomal	
	<i>r</i>	<i>Z</i> ^a	Additive <i>r</i>	Dominance <i>r</i>
Father × son	0	0	0.25	0.0476
Father × daughter	0.154	0.155	0.25	0.0476
Mother × daughter	0.0476	0.0476	0.25	0.0476
Mother × son	0.154	0.155	0.25	0.0476
Brother × brother	0.250	0.255	0.25	0.0833
Brother × sister	0.0772	0.0774	0.25	0.0833
Sister × sister	0.1428	0.1435	0.25	0.0833

^a Fisher's *Z* transformation of *r*, used in testing significance of differences.

ments in the samples giving rise to the two correlations being compared, the *p* value for a given *N* would fall somewhere between the *p* value based on the assumption of complete independence of the samples (i.e., the *p* obtained from Fisher's *Z* test) and the *p* value based on the assumption of complete identity of the samples for one of the correlated variables, e.g., father × son vs. father × daughter, where all the fathers are the same individuals in both correlations [i.e., the *p* obtained from Hotelling's *t* test of the difference between nonindependent correlations (see Walker and Lev, 1953, pp. 256–257)]. Also, the higher the *h*², the smaller the required sample size. For example, if the nongenetic variance (for the X-linked case) were 0, the expected father × son correlation would still be 0 and the father × daughter correlation would be 0.447 and the required sample size for testing this comparison would be only 43. If the nongenetic variance were 0.25 (instead of 0.5), the required *N* would be 159. The *N*s required for cross-racial comparisons of the kinship correlations (or differences between different pairs of kinship correlations) can be estimated in a similar fashion, using Fisher's *Z* test, since the correlations would be based on independent samples. Sample sizes of about 300 families in each racial group should be adequate for testing most of the critical hypotheses.

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