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EFFECTS OF INBREEDING ON MENTAL-ABILITY FACTORS

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Summary— The differential effects of inbreeding on 11 subtests of the WISC, in a Japanese (Hiroshima) population, are related to the factor structure of the tests. The degree of inbreeding depression on mental abilities is most strongly correlated with the subtests' loadings on the General factor, *g*, which is common to all of the subtests. Loadings on the Verbal factor are also positively correlated with inbreeding depression. The (non-verbal) Performance factor, however, is slightly *enhanced* by inbreeding. One possible inference from these findings is that at least the General factor shows genetic dominance, which is theoretically consistent with natural selection for *g* in the course of human evolution. It is also noted that the factor structure of the WISC in the Japanese population is highly similar to that of the U.S. standardization sample.

1. INTRODUCTION

Is human mental ability a trait of biological relevance, that is, a fitness characteristic in the Darwinian sense, as suggested by the British geneticists Jinks and Eaves (1974)? This seemingly reasonable conjecture is based primarily on quantitative genetic analyses of mental test scores obtained on twins and various other kinships, which indicate some degree of dominance of the genetic factors that enhance mental ability. Genetic variation is the raw material upon which natural selection works, and according to the modern genetical theory of evolution, as developed by Fisher (1930) and others, directional selection for a trait over many generations gradually increases the genetic dominance of those alleles (i.e. alternate forms of genes) which most enhance the advantageous phenotypic expression of the trait. Advantageous, in this case, means Darwinian fitness, that is, increased chances of survival of individuals or their kinship group and the perpetuation of their genes through successive generations.

Fisher (1930) theorized that genetic dominance is itself a product of evolution. His theory of the evolution of dominance, which he regarded as one of his most important theoretical contributions to genetics, is essentially the idea that any mutant alleles which tended to suppress the expression of harmful or less advantageous mutants would be favorably selected for, and hence the property of dominance itself would evolve as a result of natural selection. The development of dominance, of course, implies the recessiveness of less advantageous alleles. Some recessive mutants may also confer a selective advantage, which gradually increases the frequency of the recessive genes in the population in subsequent generations, although the rate of increase is very much slower than in the case of non-recessive genes.

The proportion of dominant alleles affecting a polygenic trait in a population is gradually increased by the fact that positive directional selection (i.e. selection favoring advantageous alleles) decreases or 'uses up', so to speak, those alleles whose combinations have strictly additive effects on the expression of the trait. Because the influence of alleles with additive effects is always manifested to some degree in the phenotype, on which natural selection directly operates, the deleterious or non-enhancing alleles are more rapidly eliminated by directional selection than would be the case for non-additive allelic combinations, in which the expression of disadvantageous recessive alleles is suppressed to some degree by the presence of dominant alleles. Deleterious or relatively non-advantageous alleles that also happen to be recessive are thus often hidden from phenotypic expression as a result of being paired with a dominant allele (the *heterozygous* condition); phenotypic effects are manifested by a recessive allele only when the allele is paired with another recessive allele at the same locus on the chromosome (the *homozygous* condition).

No one today disputes that those forms of behavior characterized as examples of mental ability are dependent upon the brain, or that the brain is a product of biological evolution. In the course of human evolution there has been directional selection for increased brain size. The human brain has tripled in size during the 5 million years of human evolution, from Australopithecus to modern man. As there is no discernible anatomical or physiological advantage, and many such disadvantages, to increased brain size, natural selection for increased brain size must have acted directly upon the greater capacity for complex behaviors conferred by a larger cerebrum, resulting in developments such as the capacity for true language, conceptual learning, problem solving, the invention of tools and weapons and of communal arrangements that promoted survival.

Such forms of behavior are generally associated with the concept of intelligence. This naturally raises the question of whether the trait which psychologists can nowadays reliably measure with their standardized tests of intelligence shows the theoretically-expected features of a biological fitness character. In the context of the genetical theory of evolution, probably the best test of the hypothesis that the ability measured by psychometric tests of intelligence is a fitness character, which has developed through natural selection in the past history of *Homo sapiens*, would be evidence for the genetic dominance of superior intelligence.

A closely-related question concerns the degrees of generality and specificity of the abilities which may have developed in the course of evolution. Has natural selection influenced primarily specific forms of behavior, or has it influenced only broad traits? If a considerable variety of cognitively-complex activities conferred a selective advantage, and especially if their combined effects were synergistic, the evolutionary process would be expected to select genes favoring what might be termed *general* cognitive ability. The proportion of such genes with a selective advantage would have increased over the course of evolution, and, as explained previously, such genes would also have increased in dominance. From such theoretical considerations, it seems a reasonable hypothesis that the broadest abilities, such as Spearman's g (the General factor common to all complex cognitive tests), would evince dominance to a greater degree than would narrower ability factors (the so-called 'group factors') or highly task-specific sources of variance in performance. Tests of this hypothesis require techniques for detecting genetic dominance and estimating its magnitude for various abilities.

1.1. *Estimation of dominance from kinship correlations*

The precise genetic mechanisms through which the phenomenon of dominance affects the genetic variance of a trait and differentially affects various kinship correlations has been explicated elsewhere (Jensen, 1978). It is from the distinctive effects of dominance on kinship correlations that quantitative genetic analysis detects its presence. At least theoretically, the presence of dominance is revealed by comparison of parent–offspring correlation with the correlation between siblings. Because the sibling correlation reflects approximately one-fourth of the dominance variance (in addition to one-half of the additive genetic variance) and the parent–offspring correlation involves only the additive genetic variance, four times the difference between the two correlations affords an approximate estimate (i.e. neglecting epistasis, or higher-order gene interactions) of the proportion of genetic variance attributable to dominance. Also, the regression of offspring values on midparent values on the trait in question is an estimate of the narrow heritability, i.e. the proportion of phenotypic variance due to additive genetic effects. Broad heritability (the proportion of phenotypic variance due to all genetic factors) is generally estimated from correlations between monozygotic twins, who share all genetic components in common. The difference between the broad and narrow heritabilities, then, provides an estimate of the non-additive genetic variance, most of which is probably due to dominance deviation. It should be noted, however, that tests of dominance based on parent–child and sibling correlations may be seriously attenuated when applied to traits, such as intelligence, for which there is a substantial degree of assortative mating (Jensen, 1978). Assortative mating increases both sibling and parent–child correlation, thereby to some degree counteracting and obscuring the effect of dominance on the correlations. Dominance effects in parent–child and sibling correlations can also be confounded with common home-environmental effects (Li, 1977).

The literature on the genetics of intelligence shows considerable agreement among investigators that dominance effects are present, but I can find no very satisfactory estimates of the dominance variance. In this respect, the picture seems much less clear now than it appeared prior to the discrediting of Burt's data (Jensen, 1974). Most studies of genetic dominance in intelligence test scores have estimated dominance variance as constituting about 10–25% of the total phenotypic variance. Using a methodologically-sound procedure for estimating dominance, Cavalli-Sforza and Bodmer (1971, pp. 548–550) attributed 23.4% of the total IQ variance to dominance, but this estimate unfortunately is based merely on a re-analysis of the questionable kinship correlations presented by Burt and Howard (1957), in which, by other methods, Burt had estimated dominance at 21.9%. The estimate of 15.7% dominance variance by Jinks and Fulker (1970, p. 342), is based on the difference between broad and narrow heritability for IQ data from twins and other kinships, but again, unfortunately, a large part of their evidence is derived from Burt's publications. Loehlin *et al.* (1975, p. 83), using virtually all the relevant kinship correlations for IQ reported in the literature (of which Burt's correlations constitute only a small fraction), estimated dominance variance at 11% of the phenotypic variance and 15% of the total genetic variance (the remainder being attributable to additive genes and assortative mating). Probably the most reliable estimate of dominance variance is that provided by Fulker and Eysenck (1979, pp. 125–127). It excludes Burt's data completely and is based on the best-available correlations for MZ twins, siblings, parent–child and assortative mating, which are the essential data for estimating dominance. The dominance variance is estimated at 17–22% of the total genetic variance (69% of the phenotypic variance); and the dominance variance is about one-half as large as the strictly additive variance (excluding variance due to assortative mating).

For whatever these estimates may be worth, none of them seems to indicate a very large amount of dominance variance—perhaps only one-third to one-half as much as of the additive genetic variance. But the amount of dominance variance really should be considered in relation to the theoretical maximum dominance variance that could occur depending upon the proportions of dominant and recessive alleles in the population. When there is *complete* dominance of *all* the alleles that enhance the trait, and the dominant and recessive alleles are in the proportions of $p = q = 0.5$, then the ratio additive variance/dominance variance is 2:1 (assuming there is no inbreeding). The proportion of dominance variance increases as a monotonic function of $p - q$, where p is the relative frequency of the dominant alleles. Even with complete dominance at all loci, the additive genetic variance will not be less than the dominance variance until the frequency of the dominant alleles is more than twice the frequency of the recessive alleles. As Falconer (1960) points out,

> "[t]he concept of additive variance does not carry with it the assumption of additive gene action; and the existence of additive variance is not an indication that any of the genes act additively (i.e. show neither dominance nor epistasis)" (p. 138).

The additive genetic variance will approach zero as a result of natural selection only if the trait is perfectly correlated with fitness in the Darwinian sense and if there is zero mutation rate. The additive variance will attain some value greater than zero for traits which are imperfectly correlated with fitness. We would not expect extremly high correlations of human intelligence with fitness. It is even likely that selection for intelligence has been somewhat relaxed with the advance of civilization. In human cultures the fitness of persons of quite low intelligence may be enhanced by the contributions of relatively few individuals at the high end of the ability scale, e.g. through inventions, advances in agriculture, hygiene and so forth, which benefit all persons in the society regardless of their individual levels of intelligence.

1.2. *Inbreeding depression*

Another, less indirect, indicator of positive dominance is the phenomenon known as *inbreeding depression*. The effect of inbreeding on the mean of a quantitative trait is directly related to the coefficient of inbreeding and the amount of directional dominance deviation involved in the particular trait. The coefficient of inbreeding, f, is the average probability over all gene loci that the same allele on both homologous chromosomes comes from the same ancestor (Crow

and Kimura, 1970, pp. 64–65). Thus if there is dominance to the alleles which enhance the phenotypic expression of the trait, inbreeding will lower the mean of the trait relative to the mean in a non-inbred population—the phenomenon known as inbreeding depression. Conversely, if the alleles that enhance the phenotype are recessive, inbreeding will raise the mean, because inbreeding directly increases the average homozygosity, permitting greater phenotypic expression of recessive alleles. Inbreeding also increases the variance of a trait among the inbred progeny. The precise genetic mechanisms through which inbreeding produces these effects on the mean and variance have been explicated in detail elsewhere (Jensen, 1978, pp. 78–90).

Inbreeding depression has been found in many quantitative human characteristics: birth weight, height, head circumference, chest girth, muscular strength, fetal and infant viability, resistance to infectious disease and dental caries, rate of physical maturation and age of walking, to name a few that are well documented in the literature on human genetics. Mental ability is also subject to the effects of inbreeding.

1.2a. *Offspring of cousins.* It has long been recognized that there is a much higher incidence of mental retardation among the offspring of cousin marriages, as well as of incestuous matings (parent–offspring and full siblings), than among the offspring of unrelated parents (Penrose, 1969). Böök (1957), in a study of children of first cousins, reported a significantly higher incidence of mental retardation, and fewer gifted children, than in a control group of non-inbred children, when ratings of ability were based on teachers' judgments of intellectual level from scholastic performance. In fact, the incidence of retardation was over three times higher for the inbred children, despite the author's claim that social backgrounds were the same in the inbred and control groups. Reed and Reed (1965) reported a 4-fold increase in the incidence of mental retardation in the children of first cousins. Of course, relatively small shifts in the mean of the total inbred groups' IQs, when these have an approximately normal —or Gaussian— distribution, will have a large effect on the proportion of the group that falls below the very low level of IQ (below 70 or so) which is unambiguously recognized as mental retardation.

Other studies have shown lower IQs, on average, of the offspring of cousin matings as compared with non-inbred controls (Bashi, 1977; Cohen *et al.*, 1963; Neel *et al.*, 1970; Schull and Neel, 1965, 1972; Slatis and Hoene, 1961). Although the mean effect of inbreeding on IQ falls short of statistical significance in some of the studies based on smaller samples, it is noteworthy that all of the studies agree in showing similar-sized effects of inbreeding, a depression of between 2.5–3.5 IQ points for the offspring of first cousins. With the theoretically-expected increase in the variance also caused by inbreeding (and clearly evinced in some studies), and the small average effect on the mean for offspring of first cousins (whose coefficient of inbreeding is only 1/16), quite large samples are required to detect the small amount of inbreeding depression at a statistically-significant level. The effects of inbreeding on a number of highly-hereditary physical traits is no greater, in standard deviation units, than for IQ, and are usually even slightly smaller (e.g. Schull and Neel, 1965).

An important problem in all inbreeding studies involves the selection of an adequate control (non-inbred) group. General population norms cannot properly serve as a control, because systematic differences, particularly in socio-economic status (SES), are generally found for inbred samples. Therefore, an attempt is usually made to control for extraneous background factors that are also correlated with IQ, either by direct matching of the inbred and outbred parent groups on the relevant background variables, or by means of statistically regressing out these variables. Naturally, such procedures, which are absolutely demanded by any use of non-experimental data, can always leave some room for doubt as to how completely all possibly relevant background variables have been controlled. In his comprehensive but ultra-skeptical review of inbreeding studies, Kamin (1980) capitalizes on this doubt, often straining it to the utmost, along with his stance of intransigent statistical–methodological perfectionism, to reject virtually all of the studies claiming inbreeding depression of IQ. But this is made possible only by conjecturing (rather than demonstrating) that certain uncontrolled factors *might* have affected the results of any given study, and by ignoring the considerable overall consistency of results among all of the studies, despite the fact that they were conducted in a wide variety of contrasting populations and cultures.

More important is these studies' overall consistent accord with theoretical predictions. Practically without exception, the higher the degree of inbreeding [going from: second cousins ($f = 1/64$); first cousins once-removed ($f = 1/32$); first cousins ($f = 1/16$); double first cousins ($f = 1/8$); and incestuous matings ($f = 1/4$)], the greater is the degree of inbreeding depression on IQ—a pattern not tenably explained by SES or other background variables. In the largest published study of cousin marriages, Bashi (1977) found that children of double first cousins show significantly greater inbreeding depression of scores on three mental tests, including Raven's matrices (a highly *g*-loaded non-verbal test of intelligence) and four tests of scholastic achievement, than children of first cousins, who scored lower than children of unrelated parents. Also, in accord with theoretical expectation, there was larger variance among the children of double first cousins than among the children of first cousins. Bashi argues,

> "The inbreeding depression found in this study is consistent and cannot be explained by the effects of socioeconomic status" (p. 440).

In this Arab sample, the SES of unrelated parents is lower than in the cousin marriages, so the effect of SES would, if anything, tend to counteract the appearance of inbreeding depression. In this Arab society, marriage among relatives is encouraged and hence is relatively common—about 34% of all marriages are between cousins of varying degree, with about 4% between double first cousins.

1.2b. *Offspring of incestuous matings.* It has been argued that the severe inbreeding effects on the offspring of incestuous matings were probably the original disposing cause of the strong universal taboo against incest found in all known societies, even though this reason is not generally recognized in primitive societies (Lindzey, 1967). The late British geneticist, Darlington (1978, pp. 70–71), however, has argued that the deleterious effects of inbreeding are largely the *consequence* rather than the cause of the incest taboo. He points out that the tendency toward outbreeding, and the avoidance of inbreeding, is an evolutionary characteristic found in nearly all sexually-reproducing plants and animals, producing the selective advantage to the species of greater genetic variation and hence potentially greater adaptability to a changing environment. Thus, the avoidance of close inbreeding, as between parents and offspring and between siblings, is itself genetically conditioned. As a consequence of the greater heterozygosity produced by outbreeding, a larger proportion of deleterious recessive genes due to mutation remains hidden, and cumulates from generation to generation, whereas under inbreeding, with its increase in homozygosity, a much larger proportion of the undesirable recessive mutants would be expressed phenotypically and hence would be more quickly eliminated through natural selection.

The three published studies of the offspring of incestuous matings illustrate the nature of inbreeding effects. The offspring of 7 brother–sister and 6 father–daughter unions were investigated at 4–6 years of age by Carter (1967). Only 5 of the 13 were in the normal range of IQ; 1 child was too severely retarded to be tested, and 4 were retarded, with IQs between 59 and 76; 3 had died of recessive genetic diseases that are extremely rare in the general population.

A similar study of 18 incestuous matings (12 brother–sister and 6 father–daughter) included a non-inbred control group of children in which there was an attempt to match each control mother with the mother of an inbred child for age, height, race, SES and intelligence, although IQs were not available for all of the mothers (Adams and Neel, 1967; Adams *et al.*, 1967). The authenticity of consanguinity was checked by blood-group testing. Three of the offspring of the incestuous matings died shortly after birth and two were so severely retarded as to be untestable. Four were mildly retarded, with IQs between 60 and 70. The mean IQ of the 13 inbred offspring who could be tested was 94 as compared with 102 for the 18 controls. Some of the incestuous parents were from middle-class families and included college graduates, and several of the inbred progeny had fairly-high IQs. The variance of IQs in the inbred group was more than twice the variance in the control group—a significant ($F = 2.42$, $df = 12/17$, $p < 0.05$) difference despite the small sample. These findings are consistent with the theoretical genetic expectation that when there is some dominance in the gene action contributing to variation in

a given trait, the effects of inbreeding are to lower the mean and increase the variance, because inbreeding brings out previously-hidden recessive factors which contribute to the phenotypic variance (Crow and Kimura, 1970, pp. 99–100; Jensen, 1978).

The largest study of children of incestuous matings involved 161 inbred births. In this study the controls were the inbred children's half-siblings, i.e. they were born to the same mothers when impregnated by men not genetically related to the mother (Seemanova, 1971). Among the inbred offspring there were 40 cases of moderate and severe mental retardation; there were no retardates in the control group. As in previous studies, the inbred children also showed much higher rates of mortality and physical malformations.

The results of all these studies of the effects of inbreeding on mental ability, viewed together, can hardly be explained without recourse to certain well-established principles of genetics, in terms of which the present findings are not only explainable but were entirely predictable. The distinctive pattern of results could be accounted for in non-genetic terms only by *ad hoc* conjectures of various possible, even if improbable, environmental factors or methodological artifacts more or less peculiar to each particular study. It seems at least more heuristic to explore further the hypothesis that directional dominance, in addition to other genetic factors, is involved in individual differences in human mental abilities.

2. PURPOSE

The aim of the present study is, first, to inquire whether the effects of inbreeding on human cognitive abilities differ systematically for different types of ability, and, second, to determine whether the effect of inbreeding is most pronounced on the General factor of mental ability, or Spearman's *g*, which is common to a wide variety of cognitive tests. That inbreeding should predominantly affect *g* is hypothesized from the theory that general mental ability is a biological fitness trait, having evolved through genetic selection, and that this trait is reflected to a high degree in performance on psychometric tests of intelligence.

Answers to these questions may be found by analyzing the relationship between the latent factor structure of a variety of standard psychometric tests and the average effect of inbreeding on each of these factors.

The present analysis takes a closer look at what, for this purpose, are probably the best-available data on inbreeding depression, those of Schull and Neel (1965), to determine the effects of inbreeding on the General (*g*), Verbal (*V*) and Performance (*P*) factors of the Wechsler Intelligence Scale for Children (WISC). If the genes involved in *g*, the General factor common to all complex mental tests, have been subjected to directional selection in the course of human evolution, we should expect to find a positive correlation between the amount of inbreeding depression of the means on the various subtests of the WISC and the magnitudes of the *g*-factor loadings of the subtests. Less-pronounced correlations might be expected between inbreeding effects and the loadings on the narrower, more specialized group factors that are uncorrelated with *g*, although there exists at present no broad theoretical basis for a strong hypothesis on this point.

3. METHOD

3.1. *Source of inbreeding data*

The essential data for the present analyses were obtained by geneticists W. J. Schull and J. V. Neel (1965) for a large-scale study of the physical and mental effects of inbreeding in Japan, where some 5% of all marriages are between first cousins, first cousins once-removed or second cousins. The extensive data were obtained in Nagasaki and Hiroshima between 1958 and 1960, and no children were included whose parents had been exposed to any significant radiation as a result of the atomic bomb dropped on Hiroshima in 1945 (Schull and Neel, 1965, p. 5).

A Japanese version of the WISC (Kodoma and Shinegawa, 1953) was administered to 1854 children in Hiroshima; 52% were males. The mean age of the total sample was 8 yr and 7 months. The sample sizes and degree of inbreeding for the various consanguinity groups are

Table 1. Consanguinity groups, sample sizes (N) and inbreeding coefficients (f)

Parents' consanguinity	N	%	f
1st Cousins	486	26.2	1/16
1st Cousins once-removed	191	10.3	1/32
2nd Cousins	188	10.1	1/64
Unrelated	989	53.3	0[a]

[a] The coefficient of inbreeding, f, for the offspring of unrelated parents is conventionally set at zero, although it actually has some very small value, varying from one population to another. The mean f for offspring of nominally unrelated parents estimated from samples of the populations in three cities in Japan is approx. 1/760.

shown in Table 1. The average degree of inbreeding for the inbred groups is approx. 1/22, which is equivalent to having parents who share 1/11th of their genes through common ancestors. (First cousins share 1/8th of their genes through common ancestors.)

3.2. *Measurement of inbreeding depression*

To measure the effect of inbreeding on test scores, the scaled scores (with sex and age regressed out) on each of the 11 subtests of the WISC (the Digit-Span subtest was omitted) were regressed on the coefficient of inbreeding for all consanguinity groups. The slope of the regression of test scores on degree of inbreeding serves as a measure of the effect of inbreeding on the test performance. All of the WISC subtests displayed some degree of inbreeding depression, i.e. a lowering of the mean test score of the inbred groups as compared with the 'control' group (i.e. the offspring of unrelated parents). Schull and Neel expressed the degree of inbreeding depression in terms of the percentage reduction in test scores (using the control-group scores as baseline) per 10% increase in inbreeding. This percentage reduction amounted to between 4 and 12% for the various subtests of the WISC, with an average of about 7%. The overall effect, for the degree of inbreeding in the inbred groups of this study, was a depression of the Full-Scale IQ of 3.7 points. This degree of inbreeding depression amounts to about 5 IQ points for the offspring of first cousins, as compared with the offspring of unrelated parents. Schull and Neel describe these effects as "modest" but "statistically significant and undoubtedly real" (p. 298). A similar study by these authors, in Hirado, Japan, found similar inbreeding depression on a Japanese version of the Binet IQ, but with an inbred group of only about one-third the size of their Hiroshima groups, the modest effect was not statistically significant (Neel *et al.*, 1970; Schull and Neel, 1972).

In the calculation of all of the inbreeding effects just mentioned, a number of IQ-related variables had to be statistically controlled by means of multivariate regression analysis, because these variables were also correlated to some slight extent with degree of inbreeding, and, unless statistically controlled, their effects could spuriously inflate the measured effects of inbreeding on the WISC scores.

3.3. *Control of parental variables*

In addition to controlling child's age, sex, birth rank and month of examination by multivariate regression, a number of parental variables were similarly taken into account, in order to equate statistically the inbred and control groups on socio-economic background. This equating is especially important from the standpoint of the present study, because parental consanguinity is slightly correlated with socio-economic status (SES), and on theoretical grounds it would be expected that SES, like the effect of parental consanguinity, would be most strongly related to the g factor of cognitive abilities. Much evidence indicates that educational and occupational selection, which in modern societies are the main determinants of social mobility and SES, involve selection for g to a far greater extent than for any other single measurable

human characteristic (Jensen, 1981). Hence SES differences theoretically would be expected to mimic the theoretically-expected effects of inbreeding with respect to a variety of cognitive tests differing in their *g* saturations.

Schull and Neel were fully cognizant of the importance of controlling SES in their study, and for this purpose they obtained exceptionally fine-grained measures of a number of variables which in many sociological studies have proven to be the best indicators of SES: (1) father's education; (2) mother's education; (3) father's occupation; (4) mother's occupation; (5) number of persons in household; (6) home area (floor space) per person; and (7) monthly food expenditures per person. Also, the number of family pets was considered. The thoroughness of these listed variables as a control for SES by means of multiple regression is suggested by the common experience of researchers that any combination of two or three such SES indicators generally accounts for by far the most of the IQ variance attributable to SES when each indicator is entered into a stepwise regression, and any additional indicators contribute practically negligible increments to the variance accounted for. It is highly doubtful that Schull and Neel could have included any other variables which would have regressed out a significant or appreciable increment from the originally slight (but significant) correlation between SES and degree of parental consanguinity, over and above the seven SES indicators they selected. Even the addition of parental IQs, however desirable this would have been from a theoretical standpoint, would probably not have altered the result appreciably, as parental education and occupation are quite good 'stand-ins' for parental IQ. (On the basis of other studies of the correlations between IQ, education and occupation, the multiple correlation between midparent education and occupation, on the one hand, and midparent IQ, on the other, would be estimated between 0.70 and 0.80.)

It should be noted that the multiple correlation, *R*, between the coefficient of inbreeding, *f*, and the various SES indicators for all consanguinity groups in the Hiroshima study is only 0.105 (a negative correlation) which amounts to about 1% of the total variance in SES accounted for by *f* (Schull and Neel, 1965, Table 4.16, p. 83). When the inbred and control groups are compared on any of the physical or mental measurements in this study, any difference between them that could reasonably be attributable to SES, *after* the SES variables have been statistically held constant, must indeed be vanishingly small.

3.4. *Final index of inbreeding depression*

Statistically, holding age and the SES variables constant, Schull and Neel determined the degree of inbreeding depression on each of the 11 WISC subtest scaled scores separately for males and females. The degree of inbreeding depression on subscale scores was expressed as the percentage decrement in the score per 10% increase in degree of inbreeding (*f*). (These percentage inbreeding decrements for males and females are given in Table 12.19, p. 295, in Schull and Neel's (1965) monograph.) Because this measure of inbreeding depression (henceforth labeled I.D.) is correlated (over the 11 subtests) 0.99 between the sexes, it is quite reasonable to combine the I.D. measures on males and females, and use the mean I.D. for each subtest in all of the subsequent analyses. (These mean values of I.D. are shown in the first column of Table 2.)

3.5. *Factor analysis of the WISC subtests*

Schull and Neel (1965, Tables 12.13 and 12.14, p. 291) also present the inter-correlations of the WISC subtests in their Hiroshima sample (all consanguinity groups combined), separately by sex for 7-yr-old (78–89 months) and 10-yr-old (114–125 months) children.

Each of these four correlation matrices was subjected to three mathematically-distinct types of factor analysis. In all cases, only those factors with eigenvalues greater than 1 were extracted.

Different types of factor analysis were used to meet the possible criticisms that (1) the observed results are an artifact of the particular method of factor analysis, and (2) the General factor, *g*, is not optimally represented by one or the other method of factor analysis. (One method, Varimax rotation of the principal components, cannot, by its nature, yield a general factor.)

3.5a. *Hierarchical factor analysis.* This procedure extracts a number of oblique (i.e. correlated) primary, or first-order, factors, the correlations among which are in turn factor analyzed to yield a second-order factor or factors. (This analysis begins with a common factor analysis, with communalities in the principal diagonal.) The Schmid–Leiman (1957) transformation was used,* which removes the variance shared in common among the first-order factors and adds it to the variance of the next higher-order factor(s), thereby making the first-order factors orthogonal (i.e. uncorrelated) to one another and orthogonal to the higher-order factor(s). In the present analyses, *g* emerges as the only second-order factor.

3.5b. *Principal axes or common factor analysis.* This type of analysis, with communalities in the principal diagonal, yields a number of orthogonal factors, with each successive factor accounting for a smaller proportion of the total variance in all of the variables. The first principal axis always accounts for the largest proportion of variance and represents the General factor of the matrix.

3.5c. *Varimax rotation of the principal components.* A principal components analysis (with 1's in the principal diagonal) yielded two components with eigenvalues greater than 1. The components were (orthogonally) rotated to approximate simple structure according to Kaiser's (1958) Varimax criterion, which attempts to maximize the variance of the squared factor loadings on each of the rotated factors. This type of analysis highlights only the primary, or group, factors latent in the correlation matrix; the variance of the general factor is completely submerged among the rotated primary factors.

3.6. *Relationship between factor loadings and inbreeding depression*

The aim is to quantify the degree of similarity between the profile of factor loadings of the 11 WISC subtests and the profile of the measure of inbreeding depression (I.D.) on the subtests. This is shown by the Pearson correlation coefficient, r, and by the Spearman rank-order correlation, ρ. As Spearman's ρ is calculated from merely the rank orders of the values on the two variates, it is not at all affected by their scale properties. This is desirable in view of the fact that inbreeding depression could be expressed on a number of different scales other than the one adopted by Schull and Neel, and, if the scales were not simply linear transformations of one another, the Pearson r would vary slightly according to the choice of scale. As all proper scales of inbreeding depression are at least monotonically related to one another, however, Spearman's ρ would remain exactly the same regardless of the choice of scale.

3.6a. *Control of subtest reliability.* Both the factor loadings and the index of inbreeding depression (I.D.) on each subtest are inevitably attenuated by measurement error. The internal consistency reliability coefficients of the eleven subtests of the WISC range from about 0.65 to 0.85. Because the reliability of a given subtest affects both the magnitude of its loading on any factor as well as the degree to which it is capable of reflecting the effect of inbreeding, it could be argued that the observed correlation between subtest factor loadings and I.D.s results merely from the fact that both variables are correlated with a third variable, viz. subtest reliability. It would be possible to correct the factor loadings and the I.D.s for attenuation, by dividing each one by the square root of the subtest's reliability. This method of taking account of subtest differences in reliability, however, could be deemed suspect, because by dividing each of the correlated variables by the same value, a spurious 'index correlation' could be created between the variables (Guilford, 1956, p. 328). A more defensible method in the present case is to control subtest reliability by means of partial correlation. Hence subtest reliabilities are partialled out of the correlations between factor loadings and I.D.s. This has been done only with the Pearson r, as partial correlation is not permissible with Spearman's ρ. (Partial correlation is, however, permissible with Kendall's τ, another measure of rank-order correlation; but τ is scaled very differently from r and therefore is not directly comparable, as is Spearman's ρ.) Partial correlation would be illegitimate only if there were a theoretically intrinsic relationship between each of the covariates and the controlled variable (in this case, subtest reliability). But a test's reliability, which is partly a function of test length, is not at all connected theoretically with either its factor composition or its susceptibility to inbreeding effects.

* I am grateful to Professor John Schmid for performing this analysis on these data.

Schull and Neel did not determine the WISC subtest reliabilities in their Japanese samples, and so the reliabilities in the American standardization samples of the same ages were used in the present analysis (Wechsler, 1949). In view of the high degree of similarity of the matrices of subtest inter-correlations and the high congruence of their factor loadings in the American and Hiroshima samples, the reliabilities of the subtests are most probably also much the same for Americans and Japanese.

3.6b. *Correction of the profile of subtest factor loadings for attenuation due to sampling error.* The factor loadings of tests, like any other statistics, fluctuate somewhat from one sample of the population to another. Consequently, the profile of factor loadings of the 11 WISC subtests does not have perfect reliability. But if the profiles based on different samples are positively correlated with one another, then a *composite* profile, obtained by averaging the profiles from a number of samples, would have higher reliability than the reliability of a profile for any one sample. The reliability of the composite profile can be obtained by an analysis of variance or (with virtually identical results) by means of the Spearman–Brown formula. Hence, the composite profile reliability is $r_c = (n\bar{r})/[1 + (n - 1)\bar{r}]$, where n is the number of profiles averaged into the composite and $\bar{r}$ is the average of all of the correlations among the profiles. This estimate of r_c is then used to correct for sampling attenuation the observed correlation between the profile of factor loadings and the profile of I.D.s. This correction is important if we wish to compare the sizes of the correlations of I.D. with different factors, because various factors can differ considerably in profile reliability. For example, if all of the subtests are cognitively quite complex, they will all have almost uniformly high g loadings, and hence the profile of g loadings will be very sensitive to sampling variation and consequently would not be highly reliable; whereas, in contrast, Verbal and Performance factors would be relatively insensitive to sampling variation, because for these factors, in every sample, there would be quite high loadings on some of the tests (e.g. the verbal tests) and very low loadings on the others (e.g. the performance tests), thereby making the profile of loadings highly reliable.

3.6c. *Generalizability of factor profiles.* The theoretical effects of inbreeding are a universal biological phenomenon. But the measurable effects of inbreeding on WISC subtests in the Hiroshima samples would be of less general scientific interest if the factor composition of these tests were highly specific to the Japanese population. If the WISC subtests measure quite different mental-ability factors in different populations, the finding of a correlation between factor loadings and inbreeding depression in the Japanese population would not be generalizable to ethnically or culturally more remote populations. Such factorial generalizability seems important at least for the hypothesis that g reflects a biological fitness character in human evolution, and that g is essentially the same factor in different populations.

To gain some idea of the generalizability of the WISC's factor structure in the Hiroshima samples, the same factor analyses were performed on the American standardization data for the WISC, in the age groups $7\frac{1}{2}$ and $10\frac{1}{2}$, most closely corresponding to the ages of the Hiroshima samples (Wechsler, 1949). The degree of factor similarity between the Japanese and American samples is indicated by the most generally preferred index of factor matching, the coefficient of congruence, which, like the Pearson r, takes values from 0 to ± 1, but has distinct mathematical advantages over r as a measure of factor matching (Cattell, 1978, pp. 251–255). The coefficient of congruence is $r_c = \Sigma a_i b_i / (\Sigma a_i^2 \Sigma b_i^2)^{1/2}$, where a_i is the factor loading of test i in group a, and b_i is the factor loading of test i in group b.

4. RESULTS AND DISCUSSION

4.1. *Hierarchical factor analysis with Schmid–Leiman orthogonalization*

Table 2 shows the results of this factor analysis (described in Section 3.5a) for Hiroshima boys and girls of ages 7 and 10 years. (All of the tabled factor loadings and correlations are expressed in 0.01 units.) Besides the General factor, g, only two significant group factors emerge, Verbal and Performance. Often, when the Digit Span subtest is included in the battery, a small short-term memory factor also appears in factor analyses of the WISC, but this factor cannot emerge in this analysis because Digit Span, the marker for this factor, was not used by Schull and Neel. In each of the four groups, the second-order g-factor accounts for

Table 2. Correlations of inbreeding depression with hierarchical factor loadings on WISC subtests

Subtest	I.D.[a]	Reliability[b]		Loadings of WISC subtests on the General (*g*), Verbal (*V*), and Performance (*P*) factors (decimals omitted)											
				Age 7 yr						Age 10 yr					
				Girls			Boys			Girls			Boys		
		$7\frac{1}{2}$	$10\frac{1}{2}$	*g*	*V*	*P*	*g*	*V*	*P*	*g*	*V*	*P*	*g*	*V*	*P*
Information	8.30	0.66	0.80	64	46	−02	72	36	08	61	45	−07	65	50	−02
Comprehension	6.05	0.59	0.73	44	37	−07	51	47	−16	57	40	−05	46	33	01
Arithmetic	5.05	0.63	0.84	54	31	07	57	01	34	58	34	02	45	28	05
Similarities	9.95	0.66	0.81	54	37	−01	62	22	16	58	33	02	53	41	−02
Vocabulary	11.45	0.77	0.91	58	50	−10	63	25	13	58	44	−08	67	55	−06
Picture Completion	5.90	0.59	0.66	52	15	20	54	15	17	50	06	25	46	10	24
Picture Arrangement	9.40	0.72	0.71	58	16	24	63	02	36	57	27	08	44	09	23
Block Design	5.35	0.84	0.87	46	−11	42	54	11	21	58	06	30	54	01	39
Object Assembly	6.05	0.63	0.63	64	−09	53	56	12	22	62	−14	53	44	−10	42
Coding	4.45	0.65	0.65	38	14	12	53	12	20	43	23	04	48	17	18
Mazes	5.35	0.79	0.81	55	06	32	45	−16	43	45	13	15	34	−13	38
Per cent of variance due to factor				29.0	8.5	6.3	33.3	5.0	6.1	30.9	8.7	4.3	25.5	9.8	5.7
Correlation of factor loadings with I.D.				51	60*	−52*	70**	32	−14	45	51	−42	61*	62**	−47
Partial correlations[c] with I.D.				52	70**	−62*	73**	45	−26	39	40	−30	33	54	−37
Rank-order correlation with I.D.[d]				54*	65**	−43	65**	54*	−42	42	44	−39	36	51	−52*

[a] Inbreeding depression as percentage of outbred mean; average of both sexes (from Schull and Neel, 1965, p. 295).
[b] Reliability (internal consistency) based on Wechsler normative age groups $7\frac{1}{2}$and $10\frac{1}{2}$.
[c] Subtest reliabilities are partialled out, leaving the correlation between inbreeding and factor loadings independent of subtest reliability.
[d] Spearman's ρ, corrected for tied ranks.
* $p < 0.05$, one-tail test; ** $p < 0.025$, one-tail test.

approximately twice as much variance as the Verbal (V) and Performance (P) factors combined. In two of the four groups, inbreeding depression, I.D., shows significant positive zero-order correlations with g-factor loadings. With one exception (age 10 boys), the partial correlations (with subtest reliabilities partialled out) and rank-order correlations remain about the same as the zero-order correlations. It should be noted that these correlations are based on only 9 degrees-of-freedom (or 8 *df* for the partial correlations), and therefore must be quite large to attain statistical significance.

Factor V also shows substantial positive correlations with I.D., suggesting that it, too, involves dominance of the genes that enhance verbal ability.

Factor P shows consistently negative correlations with I.D., which would suggest that the genes that enhance this trait are recessive. There is inbreeding enhancement, rather than depression, of this ability factor. The P factor has its highest average loadings on the Object Assembly and Block Designs subtests, suggesting that it is essentially a spatial-visualization factor. The fact that it is somewhat enhanced by inbreeding, suggesting some recessiveness of the genes involved in this ability, accords with research by Bock and Kolakowski (1973), which suggests that spatial ability involves an X-linked recessive gene. Considerable doubt, however, has recently been cast on the Bock and Kolakowski hypothesis of a recessive X-linked gene involved in spatial ability (Bouchard and McGee, 1977). But it is also possible that some ability-enhancing genes could be autosomal recessives, and these, of course, would result in some degree of inbreeding enhancement of the test scores for the particular abilities in which the recessive genes are involved. At present the question remains unresolved and awaits further research on the genetics of spatial ability.

4.1a. *Congruence of factors in Japanese and American samples.* As described in Section 3.6c, the Schmid–Leiman factor analysis in Table 2 was also performed on two American samples, ages $7\frac{1}{2}$ and $10\frac{1}{2}$, from the American standardization sample. ($N = 100$ in each group, composed of equal numbers of boys and girls.) We can assess the similarity of the factors in the Japanese and American groups by computing the coefficients of congruence, r_c, among the four Hiroshima groups, and then comparing the average of these coefficients, $\bar{r}_c$, with the average of the congruence coefficients between each of the four Hiroshima samples and the two American samples. The values of $\bar{r}_c$ *among* the Hiroshima samples and *between* the Hiroshima and American samples for each of the Schmid–Leiman factors are as follows:

	g	V	P
$\bar{r}_c$ *among* Hiroshima groups	0.990	0.873	0.822
$\bar{r}_c$ *between* Hiroshima groups and American groups	0.990	0.928	0.881

The same congruence analysis was also performed on the other types of factor analysis used in this study, with highly similar results. The factors and profiles of factor loadings are highly congruent in the Japanese and American samples; the slight variation between the factors in the Japanese and American samples is even somewhat less than the factor variation among the Japanese samples. As would be expected under these conditions, the inbreeding effects measured in Hiroshima show about the same correlations with subtest factor loadings based on the American standardization data, which average 0.53 for g, 0.57 for V, and -0.38 for P, as compared with the corresponding Hiroshima averages of 0.57, 0.51, and -0.39, respectively.

4.2. *First unrotated principal factor*

This is another method for extracting the General factor (see Section 3.5b). The first principal factor, as shown in Table 3, usually accounts for about 20% more of the variance than the hierarchical g, but the profiles of factor loadings are highly congruent for the two methods. The coefficient of congruence between the first principal factor and the hierarchical g for the present groups, ranges between 0.98 and 1.00. Not surprisingly, the first principal factor loadings also show substantial correlations with I.D., as seen in the last three rows of Table 3.

Table 3. Correlations of inbreeding depression with first unrotated principal factor loadings on WISC subtests

Subtest	Factor loadings Age 7 yr		Age 10 yr	
	Girls	Boys	Girls	Boys
Information	74	78	73	78
Comprehension	52	53	67	55
Arithmetic	61	63	66	52
Similarities	62	67	66	64
Vocabulary	68	68	69	80
Picture Completion	57	58	51	50
Picture Arrangement	64	70	63	48
Block Design	47	69	59	57
Object Assembly	67	62	59	44
Coding	42	57	48	54
Mazes	59	51	48	33
Per cent of variance due to factor	36.1	39.5	37.7	32.9
Correlation of factor loadings with I.D.	64**	59*	62**	64**
Partial correlations with I.D.[a]	66**	58*	54	56*
Rank-order correlation with I.D.[b]	73**	65**	59*	44

[a] Subtest reliability partialled out, leaving the correlation between inbreeding and factor loadings independent of subtest reliability.
[b] Spearman's ρ, corrected for tied ranks.
* $p < 0.05$, one-tail test; ** $p < 0.025$, one-tail test.

4.3. *Varimax-rotated principal components*

The g variance is removed from the V and P factors in the hierarchical factor analysis, but in a Varimax rotation (see Section 3.5c) of the two principal components having eigenvalues greater than 1, the g variance remains submerged in the two primary factors, V and P, making them much larger, in terms of variances accounted for, than the 'left-over' V and P factors of the hierarchical analysis after g is removed. Yet the overall patterns of factor loadings on V and P remain highly similar, as do the correlations of these factors with I.D., shown in Table 4.

Here, as in Table 2, V loadings are positively correlated with I.D., and P loadings are negatively correlated, indicating inbreeding enhancement of the P factor. Assuming that this is a replicable phenomenon, the overall degree of inbreeding depression on various tests of IQ could differ depending on the proportion of the spatial-ability factor that enters into the total variance of test scores. Raven's matrices test, for example, often show some spatial loading in addition to its predominant g loading. The net degree of inbreeding depression reflected in Raven scores thus would represent an algebraic summation of inbreeding depression on g and inbreeding enhancement on the spatial factor. The present analysis suggests that this is also what happens to some extent in the WISC.

The average I.D. on the six performance subtests is only 6.1, as compared with 8.1 on the five verbal subtests. (It should be noted that every one of the subtests is more highly loaded on g than on either V or P.)

4.4. *Correlation of composite factor loadings with inbreeding depression*

As explained in Section 3.6b, the subtests' loadings on each factor averaged over the four groups provides a more reliable estimate of the true population values of the factor loadings. The reliability of the composite was determined from an analysis of variance of the factor loadings of the four groups, the sources of variance being groups, subtests, and the interaction of Groups × Subtests (which is the error term in this ANOVA). The reliability, r_{xx}, of the

Table 4. Loadings of WISC subtests on the Verbal (*V*) and Performance (*P*) Factors (decimals omitted), and correlations of inbreeding depression with rotated (Varimax) factor loadings on WISC subtests

	Factor loadings							
	Age 7 yr				Age 10 yr			
	Girls		Boys		Girls		Boys	
Subtest	*V*	*P*	*V*	*P*	*V*	*P*	*V*	*P*
Information	77	28	75	35	78	20	82	20
Comprehension	71	05	84	−08	75	17	64	15
Arithmetic	59	34	29	67	66	29	61	16
Similarities	69	21	61	40	66	29	76	11
Vocabulary	81	15	65	35	78	15	84	18
Picture Completion	39	51	50	40	19	69	33	52
Picture Arrangement	41	57	36	68	55	39	31	52
Block Design	−01	79	43	47	28	69	29	66
Object Assembly	17	82	45	49	13	83	09	71
Coding	33	35	43	46	53	20	45	41
Mazes	26	67	−03	82	30	50	−06	73
Per cent of variance due to factor	28.2	24.7	30.3	25.7	31.4	21.3	29.5	21.0
Correlation of factor loadings with I.D.	61**	−41	41	−13	52*	−37	55*	−44
Partial correlation with I.D.[a]	75**	−54	55*	−25	42	−28	47	−36
Rank-order correlation with I.D.[b]	62**	−41	58*	42	48	−29	50	−34

[a] Subtest reliability partialled out, leaving the correlation between inbreeding and factor loadings independent of subtest reliability.
[b] Spearman's ρ, corrected for tied ranks.
* $p < 0.05$, one-tail test; ** $p < 0.025$, one-tail test.

profile of composite loadings is derived from the F ratio for the subtests' mean square: $r_{xx} = (F - 1)/F$. This reliability coefficient can then be used to correct the correlations between the composite subtest factor loadings and I.D. The same procedure is also applicable to the ranked data used in computing Spearman's ρ. (The factor loadings were ranked separately in each group.) The method was applied to each type of factor analysis, with the results shown in Table 5. The partial correlation of subtest loadings with I.D. (with the average subtest reliabilities partialled out) is based on the correlations corrected for attenuation. These partial correlations (fourth row in Table 5) provide the most accurate picture of the relative magnitudes of the inbreeding effects on the various factors, because the correction for attenuation due to sampling error, as just described, in effect equates all of the factors for the reliability of the profile of loadings. It is evident that g is the most highly (positively) correlated

Table 5. Correlations of inbreeding depression with mean factor loadings of four groups

	Hierarchical factors				Rotated Varimax factors	
Descriptive statistic	*g*	*V*	*P*	Unrotated first principal factor	*V*	*P*
Per cent of variance due to factor	29.4	7.0	4.8	36.5	28.1	21.9
Correlation of factor loadings with I.D.	0.71**	0.59*	−0.46	0.75**	0.58*	−0.39
Correlation corrected for attenuation[a]	0.79**	0.61*	−0.48	0.82**	0.61*	−0.41
Partial correlation with I.D.[b]	0.78**	0.63*	−0.50	0.81**	0.64*	−0.46
Rank-order correlation with I.D.[c]	0.68**	0.49	−0.44	0.77**	0.49	−0.40
Rank-order correlation corrected for attenuation[a]	0.79**	0.50	−0.46	0.84**	0.51	−0.41

[a] Corrected for unreliability of factor loadings due to sampling error.
[b] Subtest reliabilities are partialled out, leaving the correlation between inbreeding and factor loadings independent of subtest reliability.
[c] Spearman's ρ, corrected for tied ranks.
* $p < 0.05$, one-tail test; ** $p < 0.025$, one-tail test.

with I.D., but that *V* also shows a substantial positive correlation, whereas *P* shows a relatively smaller and non-significant negative correlation with I.D. The rank-order correlations based on the composite ranked data, similarly corrected for attenuation, yield a similar result.

4.5. *Interpretations of the results*

The results summarized in Table 5 indicate significant and substantial positive correlations between the varying degrees of inbreeding depression on the 11 subtests of the WISC and the varying *g*-factor loadings of those subtests. This finding is consistent with the hypothesis that *g* is a fitness character and has accordingly acquired genetic dominance over the course of many generations in the past. The loadings on the Verbal factor, *V*, also show a significant, although slightly less pronounced, correlation with inbreeding depression, suggesting that verbal ability has also acquired some degree of dominance. The loadings on the Performance factor, *P*, in contrast to *g* and *V*, are negatively correlated with the index of inbreeding depression, which suggests that this factor, identifiable as mainly spatial ability, is enhanced by recessive genes. The evidence reviewed here is at least fully consistent with this genetical interpretation, which is not at all *ad hoc* but stems from long-established genetic theory.

The acceptability of this interpretation, however, clearly depends upon the cogency of alternative, non-genetic explanations for the observed outcomes. There seems to be no alternative *genetic* explanation to the account given here in terms of the accrual of genetic dominance as a result of long-term selection pressure on the particular trait. Mere differential heritability (in the narrow sense) of the WISC subtests would not be predicted to reflect the effects of inbreeding. Theoretically, only dominance and epistatic genetic effects could predictably produce the observed results, assuming that we are limited to a genetic explanation, and also assuming that the parental groups of the inbred and non-inbred offspring are, on average, genetically and environmentally equivalent with respect to all those factors which condition the development of mental ability.

The inherent logical impossibility, in any non-experimental study, of absolute certainty about the equivalence of population samples on all conceivably relevant variables except the single factor of theoretical interest, on which the samples have been expressly selected to differ, can always be summoned to cast doubt on the interpretation of any such study, however well executed it may be and however many of the variables deemed relevant have been statistically controlled.

In the present study, the only possible line of explanation which seems to merit consideration, besides the genetic hypothesis of primary theoretical interest which this study attempts to examine, involves the effect of the slight socio-economic difference between the parental consanguinity groups. Differences between the consanguinity groups accounted for only 1% of the total SES variance, as measured by an unusually fine-grained and thorough set of SES indices, including the educational and occupational levels of both parents. As noted in Section 3.3, Schull and Neel statistically removed this SES variance, by means of multivariate regression, from the measured differences between the inbred and non-inbred groups on all of the physical and mental measurements employed in their study. It would be difficult to imagine how one could do any better than this, short of direct matching of all sets of consanguinous and unrelated parents on IQ in addition to SES. As was noted previously, however, the parents' education and occupation, which were controlled, are in all likelihood so highly correlated with their IQs, that regressing out IQ in addition to the other control variables would probably have only a negligible effect. Ideally, of course, one would desire a true experiment, which would require completely-independent random assignment of mates to the various consanguinity groups—of course, a wholly unfeasible condition in genetical research with human beings. Those who would maintain a counsel of perfection should at least feel obliged to suggest any other indices of SES that would be at all likely to add any appreciably significant increment to the control of SES over and above that afforded by the seven SES indices which were regressed out by Schull and Neel.

It should be noted that SES has been regressed out of the difference between the inbred and outbred groups, separately for each of the WISC subtests. Because SES is hypothesized to be correlated with the *g* factor (for either genetic or environmental reasons) in the same way as

the effects of inbreeding, it would be theoretically incorrect to partial out the *profile* of SES effects on the subtests from the correlation between the profile of *g* loadings and the profile of I.D. on the subtests. Logically, the SES effect must be statistically removed from the outbred–inbred difference separately on each of the subtests, and this is what Schull and Neel have done. However, they have also presented the measures of inbreeding depression on each of the subtests when the effect of SES is ignored, i.e. not regressed out (Schull and Neel, Table 12.18, p. 295). When SES is not controlled in the I.D. index, how much does it raise the attenuation-corrected correlations (3rd row in Table 5) between I.D. and the subtest factor loadings, as compared with the corresponding correlations when SES has been regressed out of the I.D. index? The attenuation-corrected correlations between subtest factor loadings and I.D., with and without statistical control of SES, are as follows:

Factor	SES controlled	SES ignored
g	0.79	0.80
V	0.61	0.72
P	−0.48	−0.61

The control of SES has the least effect on the correlation between subtests' *g* loadings and inbreeding depression of the subtest scores, although the effects of controlling SES are also only slight for *V* and *P*.

The genetical interpretation of the effects of inbreeding on the factors of the WISC, in terms of the development of genetic dominance and recessiveness for fitness characters in the course of natural selection, seems more likely than the notion that the addition of any other SES variables to those already statistically controlled by Schull and Neel would either completely eliminate the inbreeding effect or drastically alter its relative influences on the factors of the WISC, especially its predominant positive correlation with *g*.

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