Chapter 4

Physical Correlates of Human Intelligence

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INTRODUCTION

One of the oldest commonly held beliefs is that there is an association between mental and physical traits in humans. This belief is much older than the history of psychology as an empirical discipline. Indeed, the notion of correlations between physical and mental characteristics can be traced back at least as far as the ancient Greeks. Aristotle is credited with formalizing the theory of physiognomy, though it was not until psychology became established as an independent discipline and began trying to imitate the natural sciences that the relationship between physical and mental traits became the subject of empirical investigation and objective measurement.

It may seem rather surprising, in retrospect, that so many studies were conducted along this line of inquiry during the early history of psychology. Yet

specialists in a young science often show particular zeal and pride in applying their methods to the debunking of long-held popular beliefs, and certainly most of the early studies in this realm reported negative results. Negative findings were evidently so easy to come by, and so readily, even eagerly, approved by psychologists, that computing correlation coefficients between mental and physical measurements became a popular area of psychological research in the first quarter of this century, and, for a time, it seems that findings of significant correlations between physical and mental characteristics were generally less prized than the demonstration of nonsignificant correlations.

The many names associated with this early research have been largely forgotten, probably because, in the history of science generally, little lasting credit or honor is granted to investigators whose only contribution has been to demonstrate, however correctly or convincingly, that some particular null hypothesis could not be rejected. This is not to say, however, that the generalized debunking attitude with respect to correlations between physical and mental traits has had no lasting effect. Its legacy endures in the prevailing conviction among present-day psychologists that, with the exception of certain pathological conditions in which mental defect and physical anomalies clearly occur together, the null hypothesis best summarizes the association between physical and mental traits. Clearly, the real problem is not so much whether this conclusion, as a generality, will ultimately be proven right or wrong, but whether the attitude that has been sustained by such a sweeping negative generality has hindered behavioral scientists' inquiring spirit to winnow the chaff from the grain in this broad question.

One important landmark in the history of this subject is Donald G. Paterson's *Physique and Intellect* (1930). In this work, Paterson assembled and critically reviewed virtually all the studies concerned with the association between physical and mental measurements completed prior to 1930. About two-thirds of Paterson's 300-page review examined various physical correlates of intellectual abilities; the remaining one-third of his monograph concerned physical correlates of temperament, or personality traits. Among the main physical correlates of mental ability (usually general intelligence, as indexed by IQ) critically examined in Paterson's review were stature (height and weight), cranial measurements, morphological indices (e.g., height-weight ratio), anatomical and physiological age (as indicated by skeletal development, eruption of permanent teeth, and onset of puberty), and physical health and fitness. In his time, Paterson was respected as a highly competent and sophisticated methodologist in psychometrics and differential psychology. These strengths, combined with his scholarly thoroughness, his meticulous accuracy in reporting, and his consistently objective critical acumen, make it practically unnecessary to reexamine the numerous studies reviewed in *Physique and Intellect*.

The one justifiable criticism that might be leveled at Paterson's effort is that he too, like so many of his contemporaries, was somewhat overly imbued with a

zeal for debunking popular myths, thereby incurring the risk of overworking the then-favored null hypothesis. Despite Paterson's evident caution in appraising the results of each study, one gets the impression that if he had to risk drawing the wrong conclusion, because of obvious weaknesses or uncertainties in a particular study, he much preferred to risk making what statisticians term a Type II error (i.e., accepting the null hypothesis when it is false) than to risk making a Type I error (i.e., rejecting the null hypothesis when it is true). The recently developed methodology of meta-analysis (Glass, McGaw, & Smith, 1981) and its various applications to substantive questions in psychology have already demonstrated that the properly *aggregated* statistical results of a large number of studies may yield a quite different conclusion from that arrived at by examining each study separately and then summarizing their results in terms of a "box score," reporting the frequencies of significant and nonsignificant statistical tests. For example, a dozen independent studies may all show a nonsignificant correlation between variables x and y, and we might conclude that the null hypothesis ($\rho_{xy} = 0$) is true, or more correctly, that we cannot reject it. But if all of these independent and separately nonsignificant correlations are consistently positive (or consistently negative), the probability that the true correlation is zero is at most $(1/2)^{12}$ or .0002. This simple example of a meta-analysis shows our previous conclusion to be a Type II error. But scarcely anything resembling meta-analysis was in the air in Paterson's day.

Although Paterson's *Physique and Intellect* remains a valuable reference, its net influence in psychology has been generally to discredit the idea that there are significant physical correlates of intellect and to dampen interest in research on this topic. Such was probably not Paterson's aim, yet his frequent reference to correlations which, even when statistically significant, were "too low to be of practical value," served to warn psychologists and the general public that physical appearance and anthropometric measurements could not be substituted for psychometric techniques, in the assessment of intelligence and temperament. During Paterson's time, this was probably a needed and beneficial point to emphasize, but such emphasis unfortunately detracted interest from the possible theoretical significance of discovering particular physical correlates of mental abilities. In general, it can be said that Paterson seemed constantly to imagine a kind of straw-man hypothesis of supposed *strong* correlations between physical and mental measurements, and he could always show, rightly, that his review of the existing evidence completely discredited any such hypothesis. For many readers, and some authors of psychology textbooks, Paterson's largely negative conclusions were generalized beyond the qualifications of his actual statements to include virtually all hypotheses about physical and intellectual traits. Much the same kind of fate befell Sir Francis Galton's early conjecture that certain simple laboratory tasks involving discrimination and reaction time would reflect individual differences in intelligence. The notion remained in its popularly discredited

status among psychologists for more than half a century before modern investigators finally discovered that Galton was right, or at least mostly right, after all (Vernon, 1987).

It can be said in Paterson's defense, however, that the quality of a great many of the studies he reviewed, in terms of their poor methodology, would hardly encourage rejection of the null hypothesis, even when their results were statistically significant! From a methodologist standpoint, most of the studies prior to 1930 form a depressing picture. The research standards of psychology and psychometrics during that era were shockingly weak. This was the general condition, with only rare exceptions. Study after study yields no truly interpretable results, so lacking in essential information are the published reports. The effects of restriction of variance on the correlation coefficient seemed wholly unknown to most early investigators, and attenuation of correlations by errors of measurement was hardly better known. Because of inadequate reporting of results and weak or inappropriate statistical analyses of the data, much investigative effort yielded little dependable knowledge. Too many, if not most, of these early studies now appear disgracefully inept by present-day standards of behavioral research.

Theoretical Significance of Physical and Mental Measurements

The theoretical significance of correlations between physical and mental traits is, of course, an issue that can be properly addressed only after detailed investigation of the empirical facts. Yet certain a priori considerations would seem to afford an incentive for such investigation.

From a purely theoretical standpoint, we need not take much heed of the caveat, so frequently reiterated in the literature, that the correlation between some physical and some mental measurement is too low to be of any "practical significance." Certainly, no one expects to substitute the observation of physical characteristics for the administration of psychometric tests. On a purely theoretical basis, what is more, only quite small correlations between most single physical traits and a complexly determined behavioral trait, such as intelligence, are expected to be the rule, trait variation due to multiple factors being unlikely to show a large correlation with any single causal factor. Hence, even rather small correlations, provided they are statistically significant and, more importantly, are consistently replicable, are of theoretical interest, especially if the intercorrelations among a number of physical and psychometric variables show a consistent pattern. Theoretical interpretation depends upon a network of intercorrelations. Thus, any single correlation, however reliable, can hardly be expected to do more than to spark interest in further inquiry as to its causes and links within a larger correlational network of organismic variables.

To discover a reliable correlation between a psychometric variable, say, intelligence, and some physical characteristic is to point up the intriguing fact

that a test devised strictly for measuring a particular behavioral trait is also actually measuring something more, and something apparently very different from the trait originally targeted for measurement. Such correlations pose an important theoretical challenge in that they seem to suggest that there is some greater, more substantial significance and causal underpinning to the behavioral trait expressly measured by the test. Test scores, in other words, apparently measure something more than the elicited behavior that meets the eye or that can be fathomed merely in terms of the item contents of the test. An adequate theory of intelligence should be expected to explain such correlations, if not in precise detail in every case, at least in principle.

Reliable correlations between physical and psychometric variables may also provide clues to the causes of individual variation in mental abilities. All correlations, however, are not of equal value for this purpose, regardless of their magnitude. Some correlations may be the result of relatively short-term environmental influences, such as differences in nutrition, which may simultaneously affect two or more distinct characteristics. Other correlations may be the result of cross-assortative mating for two genetically independent traits, both of which happen to be valued by mating partners in a particular culture. Still other correlations may be the result of a long evolutionary process involving the natural selection of genetically conditioned coadapted traits, or genetic characteristics that have more often than not simultaneously met the same fate in the sieve of natural selection during the evolution of different human populations. The existence of such a mechanism as a possible cause of correlations between physical traits and intelligence, of course, depends upon the validity of the biological view of intelligence, that is, that intelligence has arisen through the same processes that gave rise to other biological properties. Hence, it is subject to the same evolutionary pressures as other biological traits and may play the same evolutionary role as is played by other biological traits. One must reasonably question the extent to which "biological intelligence," here conceived as a product of evolution, is the same intelligence as that which is measured by psychometric tests of mental ability, in which individual differences are expressed on some norm-referenced scale, such as the IQ scale. Analysis of various biological correlates of IQ affords one more avenue of approach to this question, in addition to the methods of quantitative genetical analysis. The many applications of genetic analysis to psychometric variables, particularly IQ, now leave virtually no doubt that a substantial part of the total variance in psychometric intelligence is attributable to genetic factors and their covariation with environmental influences. Genetic analysis by itself, however, cannot elucidate the chain of causality between genes and behavior or explain why individual differences in some superficially dissimilar phenotypic characteristics are correlated with one another. To tackle such questions, research must advance on a broader front than is possible by means of genetic methodology alone. Genetic analysis nevertheless must play an essential role in the theoretical interpretation of

intercorrelations among physical and behavioral variables, as becomes immediately obvious when we examine the various possible causes of correlation between phenotypic characteristics.

Causes of Correlation

The most amazing feature of the research literature on correlations between mental and physical traits is that, until very recently, virtually no attention was paid to the various *causes* of correlations or to the fact that different types of correlations have quite different theoretical significance. We are not here referring to the different statistical techniques for expressing the degree of association, but to fundamentally different types of correlation with respect to their cause. Although the early literature frequently voices the familiar dictum that correlation does not necessarily imply causation, the causes of correlations are never considered at all in this early work. Part of the reason for this neglect seems to be that all organismic variables, physical and mental alike, have been treated merely as statistical predictors; research has been oriented toward the comparative validity of physical and mental measurements for predicting socially significant behavior. If correlations are viewed as a means of pragmatic prediction, there need be no concern as to their causal underpinnings. Moreover, because the prevailing attitude of psychological researchers on this topic has been to favor the null hypothesis and to discredit the age-old belief in physical characteristics as dependable clues to mental traits, there has been no need to enquire as to the causes of such correlations, which were usually found to be either statistically nonsignificant or too small to be of practical predictive value in any case.

This unfortunate failure to recognize different types of causes of correlation has resulted in a dearth of the kinds of evidence in studies of physical-mental associations that are crucially needed to permit scientifically interesting inferences from the correlations that are actually found to exist. The theoretically important questions about the observed correlations are hardly ever asked. In order to bring such questions to bear on the research evidence surveyed here, it is necessary first to define the various types of correlation and assign them a consistent terminology.

Environmental correlation. An environmental correlation is defined as a correlation between two (or more) distinctly measurable phenotypic characteristics for which the cause of the correlation exists entirely in the environment and for which there is no genotypic counterpart of the observed or phenotypic correlation. Environmental correlation can exist between characteristics of varying degrees of heritability or between heritable and nonheritable traits. The higher the broad heritability (i.e., the proportion of variance in phenotypes attributable to variance in genotypes) of both traits, however, the greater, in

general, is the likelihood that the phenotypic correlation also reflects some degree of genetic correlation. But this is not always or necessarily the case. Consider, for example, a hypothetical population in which, because of some peculiar custom, brunettes are nutritionally favored during the growth period, and blondes are nutritionally deprived, so that brunettes grow up to be of generally greater stature than blondes, thereby creating a purely environmental correlation between hair color and stature, despite the fact that the heritability of both traits remains very high. In such a case, the correlation between hair color and stature could be reduced to zero in a single generation by merely equalizing the nutrition of blondes and brunettes.

Similarly, an environmental correlation can arise between traits because of selection of an individual on one trait as a basis for differential training on some other trait. Many gender differences in our society are clear examples of this phenomenon. Gender, for example, shows opposite correlations with knowledge of cooking and knowledge of auto mechanics. These are strictly environmental correlations due to differences in sex-role acculturation. The possession of the ability known as "absolute pitch," a highly heritable trait, is correlated with musical knowledge and skill, because children who are discovered to have absolute pitch are more apt to be singled out for music lessons and parental pressure to pursue music study seriously.

Experimentally, of course, particular environmental correlations will not hold up from one generation to the next if the environmental conditions are changed, even when there is no change in the mating system across generations. Conversely, if there is no change in the environment across generations, environmental correlations will persist despite changes in the mating system. This is one of the operational distinctions between environmental correlation and genetic correlation.

Genetic correlation. Genetic correlation between distinct characteristics or traits is a considerably more complex matter than environmental correlation, not only because the causal agents—the genes—are unobservable, but also because there are several distinctive types of genetic correlation which can be easily confused with one another in their phenotypic effects. These various types can usually be distinguished, however, by means of certain analytic techniques.

Genetic correlation is a generic term that refers to any correlation between phenotypic traits in which the correlation (or some proportion of the covariance between the traits) is the result of one (or some combination) of several distinct genetic mechanisms. The main types of mechanisms are: (a) *Simple genetic correlation* due to (i) correlated social stratification of two or more genetically conditioned traits in the population, or (ii) cross-assortative mating for two or more genetically conditioned traits ("genetically conditioned" refers to a phenotypic character with a heritability greater than zero); (b) *Pleiotropy,* or the effect of a single gene on two or more characters; (c) Genetic *linkage*, as a result of genes that affect different phenotypic characters being located on the same

chromosome, usually with their loci in close proximity to each other; and (d) *Supergenes*, a special kind of linkage.

Simple genetic correlation. This type of correlation, first described by Karl Pearson (1909, 1931), results from the common assortment of genes in a population through correlated stratification of two or more traits. In this type of correlation there is no inherent or causal connection between the traits or the genes for the traits. If most of a population were divided between two religions, for example, and if membership in the one required that members have brown eyes and tall stature, and membership in the other required members to have blue eyes and short stature, a simple genetic correlation between stature and eye color would be observed among successive generations. There would be nothing intrinsic or necessary about this correlation; the relationship might just as well have been reversed, with brown eyes associated with short stature and blue eyes with tall stature. Correlated stratification of traits in this fashion may result because of seemingly arbitrary cultural mores, or because the two traits are, in some sense, "synergistic" for economic success in a particular culture, as when certain traits so complement each other as to enhance a person's chances of attaining higher socioeconomic status. On this basis, for example, we might expect some degree of positive correlation between intelligence and physical stamina, because both characteristics give their possessor an advantage in almost any kind of competition. Note that selection acts on phenotypes, which in the case of many adaptive physical and behavioral traits will be complex and hence polygenic. Selection therefore will affect *groups* of genes, which will therefore be intercorrelated. If survival in a particular society were to depend upon success in hunting, for example, one can reasonably suppose that a genetic correlation would arise between the constituents of hunting ability—visual acuity, motor coordination, tracking, and running—since all of these are selected simultaneously. The same pattern of genetic associations would not be found in a population whose survival depended upon food gathering or agriculture.

We can represent genetic correlations of various types by simple diagrams in which distinct genetic factors are indicated by circles labeled G and phenotypic traits are indicated by squares labeled x, y, and z (for different traits). *Causal* correlation is indicated by arrows; noncausal correlation is indicated by curved lines, a solid curved line indicating a within-families correlation, a broken curved line indicating a between-families correlation. In terms of this scheme, a *simple genetic correlation* is represented in Figure 4.1. Note that this type of correlation is only a *between-families* correlation. The absence of a within-families correlation rules out all other types of genetic correlation. By *between-families* correlation, we mean simply that the correlation is entirely dependent upon the association of traits x and y among persons from different families. (The term "family" is used here in the genetic sense, as a cohort of full siblings, and does not include the parents.) As fully explicated elsewhere (Jensen, 1980b), when measures of variables x and y are obtained on sets of siblings in N numbers

of families, the between-families correlation between x and y is the correlation between the N pairs of means of the siblings within each family on variables x and y. The within-families correlation between x and y is the correlation between the sibling differences on x and the sibling differences on y. (This correlation therefore cannot in the least reflect differences between families.)

When genes for distinct traits become segregated together because of common assortment of the genes in the population, the resulting simple genetic correlation will exist only between families. In accordance with Mendel's law of independent segregation of the parental genes in the process of gametogenesis, each sibling in the family receives a random assortment of the segregating genes; this random assortment of segregating genes precludes any *within*-families correlation between traits. (Segregating genes are those that are polymorphous, that is, two or more different alleles [i.e., different forms of the gene], producing variation in the gene's phenotypic effect, can exclusively occupy the gene's locus on the chromosome. All trait variation within a species is the result of segregating genes; nonsegregating genes do not contribute to individual variation.) Hence, the finding of a *within*-families correlation necessarily indicates some type of genetic correlation other than what we have termed *simple* genetic correlation.

Simple genetic correlation may also arise from *cross-assortative mating* for two traits. If women who deviate positively from the population mean on trait x mate with men who deviate positively from the mean on trait y, the result will be that their offspring, on average, will deviate positively from the mean on *both* x and y, and these traits will then be correlated in the population—a simple genetic correlation. Again, this would be a *between*-families correlation only, since independent segregation of the parental genes for x and y prevents a correlation

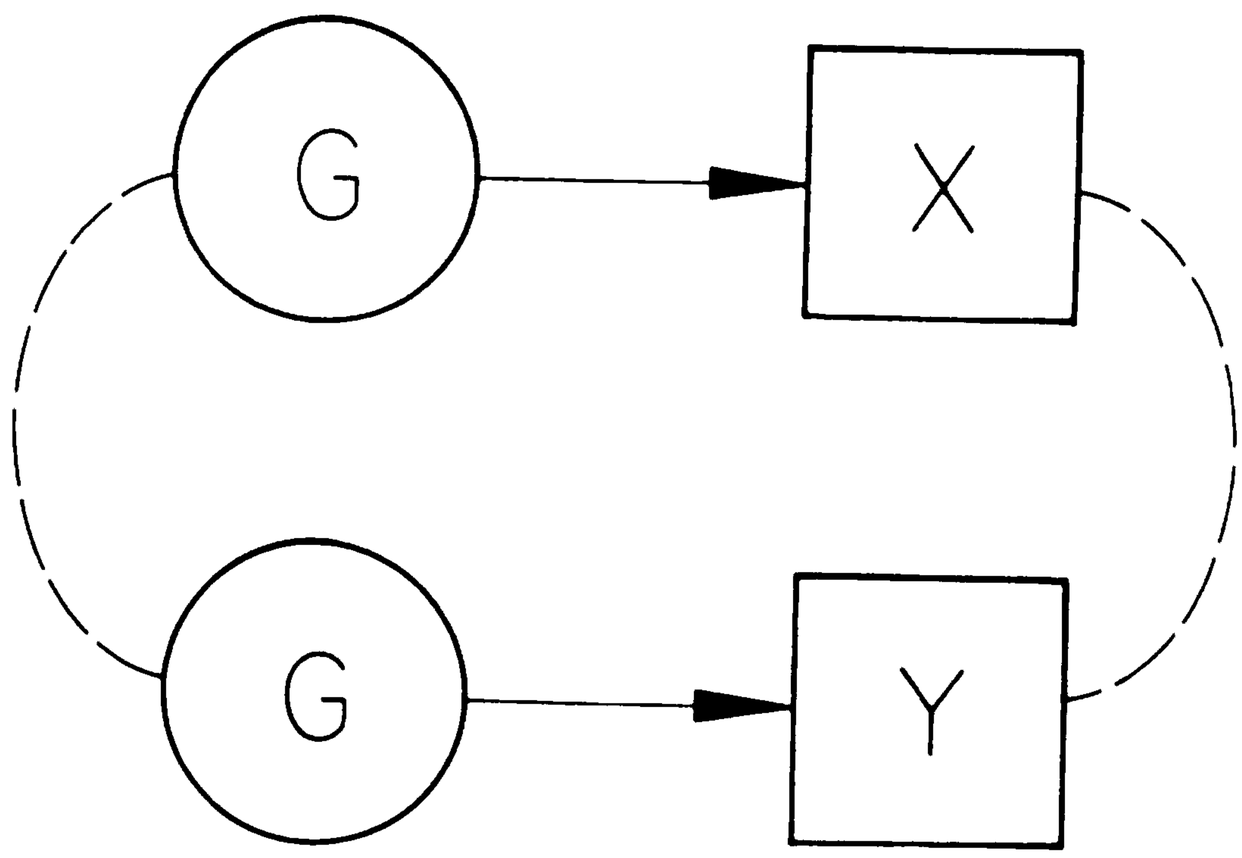


Figure 4.1. Path diagram of a simple genetic correlation between two distinct phenotypic traits, X and Y, each conditioned by a different gene (G). Arrows represent a causal connection; broken lines represent a between-families correlation.

between x and y among siblings. Indeed, the sine qua non for a simple genetic correlation between traits is the absence of a within-families correlation in the presence of a between-families correlation. In this respect, however, a simple genetic correlation may be mimicked by a purely environmental correlation, assuming the traits in question are at least moderately susceptible to environmental deviations of the kind and magnitude that prevail in the population. Social class and cultural differences in factors that can affect physical or behavioral traits will cause between-family correlations but not within-family correlations between traits; this is because such environmental factors generally represent differences between families but not within families. One can think of certain exceptions, however. Differential rearing of boys and girls, for example, could create a within-families environmental correlation between sex and those traits susceptible to the differences in rearing. This also suggests the fact that both genetic and environmental correlations for two (or more) traits may coexist, and may even be of opposite direction, thereby canceling each other, wholly or partially. Thus an observed phenotypic correlation represents the *net effect* of genetic and environmental correlations, although one or the other may predominate for any particular pair of traits.

Falconer (1960, Chapter 19) presents methods for estimating the genetic and environmental components of the phenotypic correlation between two characters. These methods have been applied successfully to the study of correlated characters in farm animals, such as milk-yield and butterfat-yield in cows and body length and backfat thickness in pigs. But such methods are exceedingly limited in their applicability for the analysis of phenotypic correlations between human traits, particularly when one or both traits are behavioral in nature. There are two main reasons for this limited applicability. First, there is the fact that these methods only permit analysis of the phenotypic correlation into two components: (a) a component due to correlated *additive* genetic deviations (i.e., "breeding values"), and (b) a component due to correlated environmental deviations *plus* correlated nonadditive genetic deviations. Phenotypic correlation between traits with considerable nonadditive genetic variance (i.e., high broad heritability but relatively low narrow heritability), therefore, cannot be analyzed in a way that is of main interest for our present purpose, that is, into a wholly genetic component and a wholly environmental (or nongenetic) component. (This consideration is of little importance to animal breeders, who are mainly interested in the breeding values [i.e., additive genetic deviations] that contribute to trait variance.) Second, there is the fact that the method of analyzing phenotypic correlations cannot practicably take account of the degree of genotype-environment correlation for each of the traits in question, and behavioral traits especially are likely to show some degree of genotype-environment correlation. For example, children who are genotypically favored for superior intelligence are more likely than less favored children to grow up in an environment that stimulates intellectual development.

The scientific interest in correlated traits that show *between*-families but not *within*-families correlation is largely of a sociological or cultural nature, regardless of whether the correlation is mainly genetic or mainly environmental. Such correlations reveal nothing about the essential nature of each of the correlated traits per se or about their causal underpinnings. The only interest is in why different values of the two traits have become simultaneously stratified in the population, thereby creating a between-families correlation. This can be an interesting question in its own right, the answer to which may reveal something about the relative social valuation of various traits and their organization within a particular culture. Exclusively between-families correlations, being largely cultural products (even if entirely due to simple genetic correlation), will be less consistent across different cultures than correlated traits showing both between-families and within-families correlation. This latter type of correlation is most probably indicative of pleiotropy.

Pleiotropic correlation. Pleiotropy is the effect of a single gene upon two (or more) distinct characters; if the gene is segregating, the two characters are affected simultaneously, and the resulting correlation between them is termed a *pleiotropic correlation.* A pleiotropic correlation between traits x and y is illustrated in Figure 4.2. The correlation r_{xy} may also be mediated by a causal chain of one or more other pleiotropic effects of a gene, acting as an intermediate causal effect between the gene and the phenotypes of interest (in this case, x and y) see for example, Figure 4.3.

A pleiotropic correlation always exists within families as well as between families, as indicated by the solid and broken curved lines in the preceding figures. If variation in each character (x and y) is attributable to only a single segregating gene and there are no other sources of variation, then, of course, the

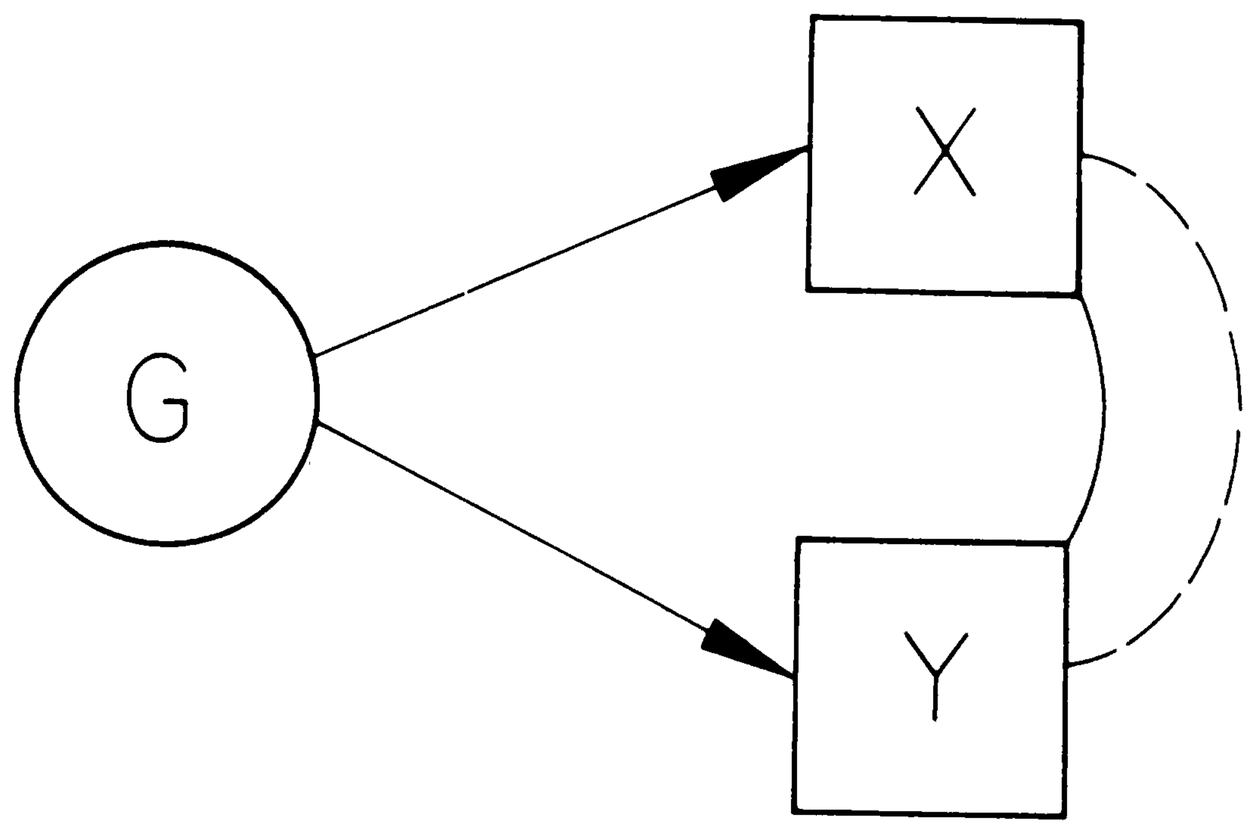


Figure 4.2. Path diagram of a pleiotropic correlation between two distinct phenotypic traits, X and Y, each influenced by one and the same gene(s) (G). Arrows represent a causal connection; curved broken line is a between-families correlation; curved solid line is a within-family correlation.

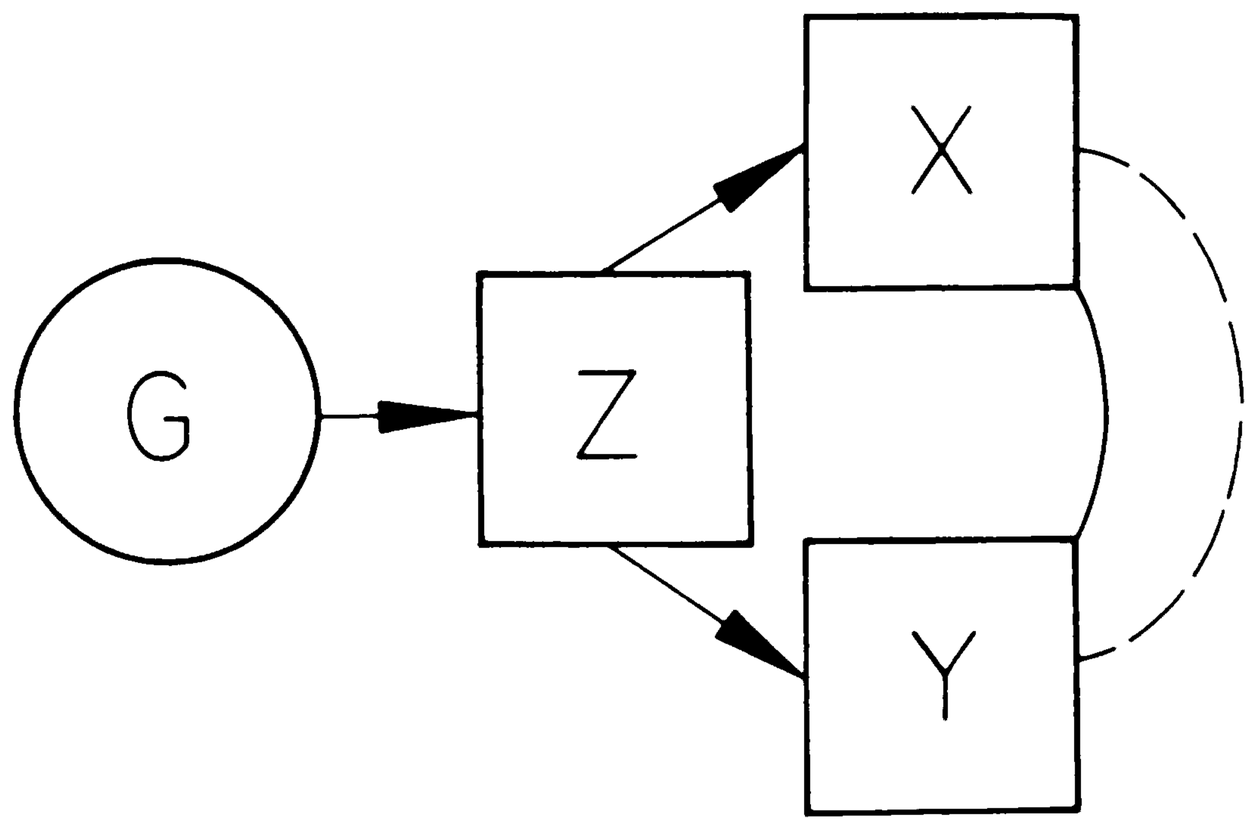


Figure 4.3. A pleiotropic correlation between phenotypes X and Y, where a single gene (G) conditions a phenotypic characteristic, Z, which in turn affects traits X and Y. Arrows represent causal connections; solid curved line is a within-family correlation; broken curved line is between-families correlation.

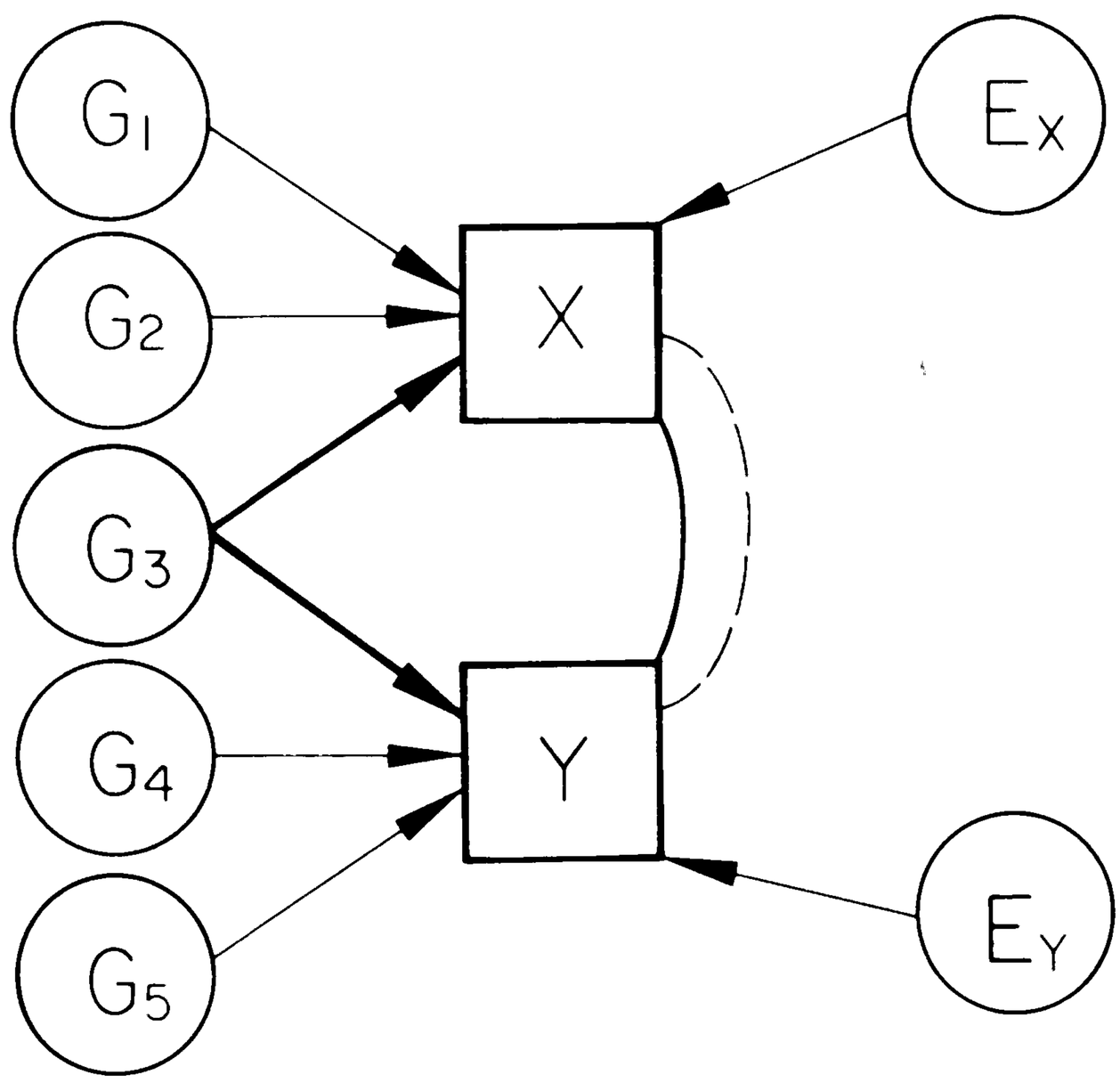


Figure 4.4. Path diagram illustrating how a pleiotropic correlation between two distinct polygenic traits, X and Y, is attenuated by sources of variance attributable to uncorrelated genes (G_1, G_2, G_4, G_5) and uncorrelated environmental effects, E_x and E_y. The pleiotropic correlation is attributable to a single gene (G_3) in the polygenic system. Arrows represent causal connection; curved solid line is a within-family correlation; curved broken line is a between-families correlation.

correlation will always be perfect ($r_{xy} = 1$). If the characters are polygenic, however, the pleiotropic correlation can take any value between 0 and ± 1, depending upon the number of other nonpleiotropic genes contributing to the total genetic variance in either one or both traits, and also depending upon the proportion of nongenetic variance in the traits. A single pleiotropic gene affecting two polygenic traits of relatively low heritability may cause only a very small pleiotropic correlation between the traits, as illustrated in Figure 4.4, in which E_x and E_y represent independent environmental effects on traits x and y.

Pleiotropic correlations can be either positive or negative, and they may be obscured, or completely obliterated, by either simple genetic correlation or environmental correlation acting in the opposite direction to the pleiotropic correlation. The pleiotropic effect on one of the traits may be detrimental to fitness, yet the effect on the correlated trait may be especially beneficial, so that the *net* effect is beneficial to fitness, or if the two traits together are *synergistically* beneficial, the pleiotropic gene will be maintained at some equilibrium frequency in the population.

A pleiotropic correlation between a highly heritable physical character and a behavioral trait, especially when the physical character has no phenotypically discernible functional relationship to the behavioral trait or is even negatively (i.e., unbeneficially) correlated with the behavioral trait, affords strong evidence that genetic factors play a role in the behavioral trait—evidence that is entirely independent of the usual quantitative-genetic methods for estimating the heritability of a trait from twin correlations or other kinship correlations.

A pleiotropic correlation is distinguished from a simple genetic correlation by the fact that a pleiotropic correlation exists within families as well as between families, whereas a simple genetic correlation exists only between families. Selection for one trait in a breeding experiment will result in a directional change in a pleiotropically correlated trait as well. Also, siblings reared apart will show the same correlation between two traits as siblings reared together if the correlation is pleiotropic, whereas an environmental correlation between traits will usually be smaller for siblings reared apart than for siblings reared together. When a highly heritable physical trait is found to be correlated (within and between families) with a behavioral trait, the most likely cause of such a correlation is pleiotropy. Evidence for pleiotropy is ipso facto evidence for a genetic component in the behavioral trait.

Linkage. When the genes for two distinct characters are carried on the same chromosome, they are said to be linked—the degree of linkage being directly related to the proximity of their loci. Mendel's law of independent or random segregation of genes does not hold in the case of linked genes, which have a greater-than chance probability of segregating together, and hence, of being transmitted together from parent to offspring, and siblings have a greater-than chance probability of being concordant for the two characters. Because of the mechanism of *crossing-over* in the process of gametogenesis, however, linkages

are broken up and genes are recombined on homologous chromosomes, the rate of recombination being directly related to the distance between the genes' loci. Hence, the linkage between any two closely linked genes gradually diminishes from one generation to the next, approaching a "linkage equilibrium." Therefore, in a stable population, that is, a population which is not outbreeding with other populations, linkage will not be a cause of correlation between traits in the population as a whole; this is true for both within-families and between-families correlations.

Linkage, however, can cause some degree of correlation between characters in the first generation after a mixture of different breeding populations, but the (within- and between-families) correlation will decrease in each subsequent generation at a rate inversely related to the closeness of the original linkage. For correlated polygenic traits, linkage would account for such a small fraction of the covariance as to be virtually undetectable, as there would be completely independent segregation for the vast majority of polygenes affecting the trait. Hence, the genetic aspect of within-families correlations between traits is most likely to be pleiotropic.

Although linkage can result in only transiently correlated characters in populations derived from crosses between divergent strains, linkages between characters can be established within separate families by analysis of family pedigrees of the two characters over two or more generations. Unfortunately, the methodology of such linkage analysis is practically limited to two gene loci and hence would not be suitable for polygenic traits in which single genes each contribute only a small fraction of the trait variance. If linkage analysis should discover a linkage between some single gene character, such as a blood antigen, and a presumably polygenic trait, such as IQ, this finding would strongly suggest a "major gene" effect on IQ: that is, most of the IQ variance would have to be attributable to a single gene, with perhaps only a small number of genes (polygenes) slightly modifying its expression. (Polygenes are structurally no different from any other genes; they are merely a number of genes, on the same or different chromosomes, each producing similar and slightly more or less equal phenotypic effects.) Such a "major gene" for general intelligence has been hypothesized (Weiss, 1978), but the evidence for it is exceedingly weak in our opinion.

Thus far we have been discussing only linkage of autosomal genes (i.e., genes located on any of the chromosomes that do not determine sex). Sex-linkage and X-linkage are a quite different matter. In these cases, we are dealing with genes carried on the sex chromosomes. But we are concerned only with the genes on the X chromosomes, which are expressed phenotypically as an X-linked character. (In humans there are very few genes on the Y chromosome besides those that determine sex.) A character that occurs with different frequencies or different average phenotypic values in males and females is often referred to as sex-

linked. A female parent can transmit an X-linked trait to her sons or daughters, but a male parent can transmit an X-linked trait only to his daughters. This creates a distinctive pattern of correlations for mother-daughter, mother-son, father-daughter, father-son, same-sex, and opposite sex siblings. All of these correlations are close to 1/2 for autosomal genes, but some will depart markedly from 1/2 in the case of X-linked genes: for example, the theoretical value of the father-son correlation is zero. If an X-linked gene is dominant, the phenotypic character it controls will appear with greater frequency in females; if the gene is recessive, the phenotype will be expressed more frequently in males. A trait that can be conclusively shown to have these distinctive characteristics of parent-offspring and sibling correlations and sex differences in frequency or central tendency is thereby proven to have a genetic basis. A behavioral trait for which X-linkage has been claimed is spatial visualization ability (Bock & Kolakowski, 1973), though the evidence for this claim is not entirely consistent (Bouchard & McGee, 1977). X-linkage has been claimed for some part of the variance in general intelligence (Lehrke, 1978), but the reported evidence for this hypothesis seems far from conclusive.

Supergenes. When particular combinations of different genes are especially favored by selection relative to other genetic combinations, they tend to become linked and to resist the process of crossing-over (which breaks up linkages) through the chromosomal mechanisms of inversion and translocation, processes which are themselves subject to genetic selection. The genetic suppression of crossing-over hence allows the formation of complexes of genes which are said to be *coadapted* to one another and which tend to be transmitted from one generation to the next as a unit, termed a *supergene* (Dobzhansky, 1970, Chapter 5). The process of chromosomal *inversion* during gametogenesis acts to resist crossing-over and recombination of linked genes so as to preserve favorable gene linkages. Different combinations of genes, coadapted for relatively optimal fitness in particular environments, form different supergenes in various strains of a species which are adapted to different habitats. The cross-breeding of such strains with different complexes of linked coadapted genes breaks up linkages through crossing-over, and results in recombinations of genes that are less favorably adapted to the habitat of either strain.

Practically all of our knowledge of supergenes maintained by inversions is derived from studies of various strains of Drosophila, other insects, and mice and rats (Dobzhansky, 1970, pp. 146–151). For technical reasons we need not explicate here, the detection of inversions is presently either difficult or impossible in many organisms, including humans. This fact, however, does not contradict the presence of supergenes in such organisms or diminish their potential theoretical significance. But it does mean that supergenes may often be practically undistinguishable from pleiotropy as a cause of correlated characters. Within some segment of the population, supergenes, like pleiotropy, would

produce both within-family and between-family correlation between characters. The importance of inversions that maintain supergenes in humans remains speculative, for, as Dobzhansky (1970) notes "it is by no means certain that inversion polymorphism is absent or even rare in human populations" (p. 150).

Cautions in Research on Physical-Mental Correlations

Several general cautions should be noted in evaluating reported findings of associations between mental and physical traits.

Unreplicated findings and Type I error. In studies involving psychometric testing, it not infrequently happens that certain physical measurements are also available on the same group of subjects, for whatever reason, even when there has been no prior research plan to investigate associations between the psychometric and physical measurements. The researcher often calculates correlation coefficients among all of the available variables, but may report only those which are large enough to be statistically significant or to look "interesting." Nonsignificant, nondescript, or inconsistent correlations are more apt to be left unreported. This tendency disposes toward Type I error (i.e., rejection of the null hypothesis when it is in fact true) with respect to reported correlations between physical and mental traits. Hence, a reported correlation, however significant, should be regarded with some suspicion if it has not been independently replicated. In this field, at least, independent replications are much more convincing evidence for the reality of a phenomenon than is statistical significance per se. One should also note whether a study has been explicitly designed to test a particular hypothesis or whether a reported finding merely represents the adventitious by-product of investigating some other phenomenon; published reports of such adventitious findings are more liable to Type I error.

Meta-analysis and the risk of Type II error. The other side of the coin is the risk of Type II error (i.e., accepting the null hypothesis when it is in fact false). Undoubtedly much more frequent than Type I error in psychological research generally, Type II error is especially common in the literature on physical correlates of mental abilities. This type of error is most likely to occur when the true correlation is relatively small and investigators employ samples that are not large enough to give the null hypothesis a fair chance of rejection. Replications of the study under similar conditions merely reinforce and perpetuate the Type II error. Reviewers of the literature who merely report "box scores" of the number of studies with statistically significant and nonsignificant results draw conclusions which further entrench the Type II error as supposedly reliable psychological knowledge. The remedy for this condition is a meta-analysis of all the reported results, that is, a statistical assessment of the entire set of results, regardless of their authors' conclusions as to their significance or nonsignificance

(Hunter, Schmidt, & Jackson, 1982). The consistency of direction of the reported correlations (or other measures of association) in addition to their sample sizes and the associated statistical *p* values (probabilities) can be used, along with information as to attenuating factors such as unreliability of measurement and restriction of variance, to determine the overall statistical significance of the composite results and to estimate the true unattenuated value of the correlation in a specified population.

Dependence of correlation on the normal-defective saddle-point. A "saddle-point" is a region in the total frequency distribution of a variable, in which there is overlap of the underlying distributions of two essentially different populations. In the distribution of IQ, the saddle-point falls in the region between about IQs 50 to 70—the region of maximum overlap between the distribution of biologically normal variation of intelligence and the distribution of IQs of the mentally deficient, whose deficiency is often attributable to some specific defect. The causes of such defects are most commonly mutant or recessive major gene effects (e.g., phenylketonuria, galactosemia, Tay-Sachs, microcephaly), chromosomal anomalies (e.g., Down's syndrome, Kleinfelder's syndrome), and brain damage due to prenatal or postnatal trauma or disease. When the bivariate frequency distribution for a mental-physical correlation coefficient spans the normal-defective saddle-point, the resulting correlation may not be generalizable to the normal population; indeed, the analogous correlation may be zero (or even opposite in sign) when calculated for the normal population only. Hence, the theoretical interpretation of a correlation will be dependent upon the degree to which it depends upon the saddle-point. Some quite substantial correlations fall to near zero when they are calculated for the part of the bivariate distribution that lies beyond the saddle-point. Failures to replicate correlations in independent studies are often the result of the presence or absence of a saddle-point in the various samples. The effect of the saddle-point is usually apparent from inspection of the correlation scatter-diagram, but this source of information is seldom obtained by investigators and is rarely provided in published reports. Without such information, the generalizability of any correlations based on samples that include mentally deficient subjects should be questioned. Both the magnitudes and causes of correlations between physical and mental variables within the biologically mentally defective population can be quite different from the biologically normal population. Generally speaking, therefore, it is methodologically unsound to combine samples of the two populations in a correlational study.

Spurious index correlations in pre-adult samples. Both mental and physical variables show developmental changes with chronological age; consequently, in a sample that ranges in age, the correlation between the variables of interest may be due to this factor alone. The relationship of both variables to age should be analyzed for linearity of their regression on age. If the regressions do not differ

significantly from linear, age (in months) should be partialed out of the correlation. When the relationship to age is nonlinear, the nonlinear components should be partialed out by means of a stepwise multiple regression analysis, in which successive powers of age (in months) are entered as the independent (predictor) variables ahead of entering the physical variable as the final independent variable, with the dependent variable being the psychometric score. Increasing powers of age (i.e., age^1, age^2, age^3, etc.) are entered stepwise into the multiple regression equation until the increment in R^2 is nonsignificant (at any desired level of confidence α), as determined by the usual F test. The age-partialed correlation between the physical and psychometric variables, in this case, is the square-root of the *increment* in R^2 resulting from the final-step entry of the physical variable in the multiple regression equation. (That is, the final increment in R^2 is the proportion of variance in the psychometric variable associated with the physical variable independent of age.) In many studies, possible nonlinear correlations between age and the variables of interest have not been ruled out, and some of the correlation between the variable of interest may be due to nonlinear age effects that remain after the simple partialing-out of the linear effect of age.

It should not be assumed that the use of age-standardized psychometric scores, such as the IQ, obviates the need for partialing age out of the physical-mental correlation. Although the age-standardization procedure necessarily causes IQ to be uncorrelated with age in the standardization sample, IQ may in fact be correlated with age in a particular study sample. Also, even if IQ, or any other age-standardized psychometric variable, has zero correlation with age, this fact does not obviate the need to partial out age from the correlation between the psychometric variable and the physical variable (physical measurements are usually not age-standardized). If an age-standardized score, x, is uncorrelated with age, a, and if a is correlated with variable y, the zero-order correlation between x and y, that is, r_{xy}, is not the same as the partial correlation between x and y with age held constant (i.e., $r_{xy \cdot a}$), as some studies have erroneously assumed. The age-partialed correlation in this case is always larger than the zero-order correlation, that is $r_{xy \cdot a} = r_{xy}/\sqrt{1 - r_{ya}^2}$.

Correlations within and between populations. A correlation found to exist between different traits within populations cannot be generalized to differences in the mean values of the traits between different populations, even if the very same size of correlation between the traits exists *within* each of the populations. In other words, knowing the regression of trait Y on trait X *within* populations does not permit any inference about the regression of the means of trait Y on the means of trait X in different populations, such as males and females and different racial groups. Whether the regression is the same or different between populations as within populations is a question that can be answered only by empirical investigation and not by logical inference. For example, the regression of body

weight on height in the male population cannot predict the mean difference in weight between men and women from information on the mean height of each sex. The regression of weight on height is different for males and females. The same caveat applies to any correlated traits within any two or more populations.

Limitations of the Present Review

We have attempted to survey virtually the entire literature on physical correlates of mental ability in humans, but have set several limitations which exclude topics that are less germane to our primary focus of interest, which is physical-mental correlations that have arisen in normal populations as a result of (a) environmental correlations (i.e., a single environmental factor affecting both physical and mental development), (b) genetic correlations between physical and mental traits due to (i) common segregation of genes through simultaneous selection on two or more traits, (ii) cross-assortative mating for two or more traits, and (iii) pleiotropy (i.e., the property of genes by which they affect two or more traits). We recognize that environmental and cultural factors may be involved in all of the above types of environmental and genetic correlation.

We have excluded consideration of relatively rare exogenous environmental factors that can affect intellectual performance, such as pathological conditions, or brain damage, or the effects of various drugs and toxic substances, or drugs that temporarily facilitate neural efficiency. These drug effects have been reviewed by Cattell (1971, pp. 198–204), who concludes that "*except for people in diseased or subnormal conditions,* no artificial drug has appeared that is capable of significantly increasing fluid general intelligence or bringing more than momentary improvements in crystallized intelligence . . ." (p. 201).

Also, we are not concerned here with trait intercorrelations that depend upon the "saddle-point" between normal variation in intelligence and mental defect, or with rare genetic or chromosomal syndromes that display both physical and psychological anomalies. These conditions and their causes have been thoroughly treated in a classic work by the British geneticists Penrose and Haldane (1969). Correlated effects due to disease and trauma are also excluded.

Our focus is on mental *abilities,* particularly general intelligence, to the exclusion of other psychological traits. The term "IQ" is here used generically for scores on any psychometric tests that experience has shown to be predominantly loaded on the *g* factor common to all complex tests of cognitive ability, including standard tests of intelligence. Special abilities are identified as such.

The purpose of this chapter is to provide an overview and guide to the literature concerning what seem to be certain more or less general and enduring correlations between physical factors and mental abilities in the population. Our

intention is to be critically comprehensive rather than exhaustively encyclopedic. When other satisfactory reviews are available, we cite them and indicate their conclusions rather than citing all the original articles reviewed therein.

GROSS ANATOMICAL CORRELATES OF INTELLIGENCE

General Body Size

Numerous studies have found correlations between body size, as measured by height and weight, and ratings on psychometric assessments of general intelligence, or IQ. The fact that the correlation between height and weight is close to .70 within homogeneous age-groups indicates a large common factor—general body size. Hence, consistent or substantial differences between the correlations of height and weight with intellect are virtually nonexistent. What small differences exist can mostly be accounted for by sampling error. Various investigators have rationalized the particular (but inconsistent) differences they observed in various ways. Penrose and Haldane (1969, p. 36), for example, explained the higher correlation of weight ($r = +.324$), than of height ($r = +.154$), with intelligence in a sample of criminals as the result of weight's being a more comprehensive measurement of body size than any linear measurement, such as stature. They cite a factor analytic study (Burt & Banks, 1947) of body measurements in adult males, which showed that weight had the highest correlation of any measurement with the general factor reflecting overall body size. On the other hand, Tanner (1969) has argued that weight is a poor measure of body size because it is so affected by fat; he claims that the true relationship underlying the correlation of IQ with the dimensions of height and weight is the correlation between IQ and general body size. In this case, the ideal correlation to be sought is the correlation of IQ with factor scores on the general factor derived from the factor analysis of a comprehensive set of body measurements, including height and weight. Unfortunately, there is no evidence in the literature that such an analysis has ever been done. Short of this, other approaches are to use partial and multiple correlations. These methods are not applicable to most of the correlations reported in the literature, however, as published reports often fail to give one or two of the three zero-order correlations (i.e., height × IQ, weight × IQ, and height × weight) required to calculate the partial or the multiple correlations. In some cases sample sizes are too small for significant statistical resolution of the various components of covariance revealed by partial and multiple correlations when two of the zero-order correlations are generally very small to begin with. When the differences between the zero-order, partial, and multiple correlations are within the margin of sampling error (say, the 95% confidence interval), of course, no validity can be claimed for comparisons of these statistics. Provided the data and sample sizes have permitted such calculations,

however, we have presented them. Also, insofar as possible, we have presented all correlations accompanied by their 95% confidence interval (i.e., $r + 1.96SE_r$); if this confidence interval does not subtend zero, the correlation is significant at $p < .05$ by a two-tailed test.

Correlation in adults. The correlation of height and weight with IQ is less complexly determined and more easily interpretable in adults than in children. Studies of adults and studies of children therefore should be dealt with separately. In adults, the correlation is not confounded with individual differences in the growth rates for physical and mental development. In young adults, moreover, stature and mental test scores are not confounded with age, so the correlation between these variables requires no statistical correction for age differences, as is required for correlations obtained in children or elder adults.

Sir Francis Galton, the first scientist to comment on the positive relationship between stature and intelligence, noted in his famous work *Hereditary Genius* (1869, p. 321) that men of genius tend to be above average in height and weight. This finding arises from the fact, now well established, that the positive relationship between body size and intelligence extends over the entire range of both variables. (Certain genetic and endocrine anomalies found at both extremes of stature account for the only exceptions.) Even among the mentally retarded, all with IQs below 70 or 75, there is a positive correlation between body size (height and/or weight) and IQ (Penrose & Haldane, 1969, p. 36; Whipple, 1914, pp. 70–72). The mentally retarded, on average, are shorter and lighter than the nonretarded population, although there is great overlap between retarded and normal groups in height and weight. At the other extreme, it has been noted that groups which are selected for superior mental ability, such as university students, are above the general average of their age peers in height and weight (Tanner, 1969).

Paterson's (1930) comprehensive review of the research literature before 1930 gives a frequency-weighted mean of all the correlations (r) for which there were no apparent shortcomings in the data or its statistical treatment, as mean $r = +.12$ for height and IQ, and mean $r = +.15$ for weight and IQ. Samples of university men, who are considerably more homogeneous as a group than men in general with respect to both IQ and social class of origin, show an average correlation of $+.10$ between height and IQ.

Large-scale studies of adult samples since Paterson's 1930 review are scarce, but results of the few such studies available are fairly consistent. These studies, as reported in Tanner's (1969, p. 188) review, in addition to more recent studies by Susanne (1979) and Passingham (1979), are summarized in Table 4.1. The unit-weighed mean $r = +.218$. The N-weighted mean $r = +.229 \pm .04$. (The procedure for computing the 95% confidence interval for the N-weighted mean of a number of correlation coefficients based on independent samples is given in Table 4.2, footnote b.) The correlations in these more recent studies are significantly higher than the average of those reported for older studies by Paterson (1930). This is of interest, because in the populations represented by

Table 4.1. Correlation (r) Between Height and Intelligence Test Scores in Adult Samples

Study	*Sample*	N	r[a]
Husén (1951)	Swedish conscripts	2250	+.22 ± .04
Schreider (1956)	French conscripts	566	+.29 ± .08
Scott, Illesley, & Thomson (1956)	Aberdeen women	270	+.24 ± .02
Udjas (1964)	Norwegian conscripts, age 20	—	+.16
Susanne (1979)	Belgian conscripts, ages 17–25	2071	+.179 ± .04
Passingham (1979)	English men, ages 18–75	212	+.12 ± .13[b]
Passingham (1979)	English women, ages 18–75	203	+.14 ± .14[c]

[a]Pearson *r* given with 95% confidence interval.
[b]With age partialed out, $r = +.13$.
[c]With age partialed out, $r = +.15$.

these studies, there has been a general improvement and trend toward social-class equalization of the health and nutrition factors affecting physical and mental development—factors often proposed to explain observed correlations between body size and intelligence. The cogency of such explanations is, of

Table 4.2. Correlation[a] (with 95% Confidence Interval) between Height and IQ in British Children

Study	*Age Group*	N	r
Scottish survey	11.0–11.9 years	6490	+.25 ± .02
London survey	10 and 11 years	4000	+.23 ± .03
National survey	8 years	2864	+.14 ± .04
National survey	11 years	2864	+.14 ± .04
National survey	15 years	2864	+.12 ± .04
N-Weighted Mean			+.193 ± .10[b]

[a]Age (in months) partialed out of correlations.
[b]The 95% confidence interval for the combined samples is computed as follows:

$$s_r^2 = \frac{\Sigma[N_i(r_i - \bar{r})^2]}{\Sigma N_i},$$

where s_r^2 is the *N*-weighted variance of *r* across samples.
N_i is the sample size for a single sample.
r_i is the correlation obtained in a single sample.
$\bar{r}$ is the *N*-weighted mean of all sample values of r_i.
Then the estimated standard error of $\bar{r}$ is

$$SE_{\bar{r}} = \sqrt{s_r^2 - \frac{(1 - \bar{r}^2)^2 K}{\Sigma N_i}},$$

where K = number of samples.
The 95% confidence interval, then, is $r \pm 1.96SE_r$

course, weakened by these findings. If the stature × IQ correlation were primarily the result of social inequalities in health care and nutrition, one should predict a decrease in the correlation obtained in studies of contemporary adult populations as compared with correlations obtained prior to 1930. The observed increase in correlation, however, is consistent with the hypothesis that there has been an increase in assortative mating for both height and intelligence, a topic to be discussed in later sections on socioeconomic status and the between-family and within-family correlations of body size with IQ. An opposite trend, however, has been reported for data obtained in Denmark, in the largest study ever made of the height × intelligence correlation (Teasdale, Sørensen, & Owen, 1989). A virtually random sample of 43,979 Danish males, all 18 years of age, were obtained from draft board records, which contained measurements of height and scores on tests of intelligence and scholastic achievement. The data were divided into five cohorts according to the year of birth, between the years 1939 and 1967. The overall mean correlation between height and IQ is .231 ± .012 (99% confidence interval). (For height × educational achievement, mean r = .253; the multiple R of both IQ and achievement with height is .265.) The r = .231 is remarkably close to the N-weighted mean r = .229 of the earlier studies summarized in Table 4.1. But the more important feature of the Danish study is the quite regular and highly significant secular decrease in the height × IQ correlation, from .269 in the 1939–43 cohort to .195 in the 1964–67 cohort. There is a linear decrease in the r of .03 per decade (the correlation between year of birth and the height × IQ correlation is −.90). A similar secular trend is seen for the height × achievement correlation. For Danish males born during the period 1939 to 1967 there was a highly significant increase in height (measured at age 18 years) of 4.3 cm, or approximately 1.1 cm per decade. The study's authors interpret these findings as follows:

> The decline in height differences between groups varying in intelligence and educational level is probably to be attributed to changing social factors, perhaps specifically a greater homogeneity of nutritional conditions across different social classes. It is notable, however, that the decline in group differences, and in the corresponding correlations, appears to have been more pronounced among those generations who were in their infancy during the 1940's. The decline thereafter has been less pronounced. It remains, therefore, to be seen whether such differences will disappear, particularly as the secular increases in height appear to be ending—Danish draft board records show the average height of males to have remained virtually stable at about 180 cms. for almost the last ten years. (p. 1293)

Given the indicated trend, we would predict that the height × IQ correlation would remain stable at about r = .20, being maintained in a nutritionally homogeneous population by a simple genetic correlation due to both assortative and cross-assortative mating for height and intelligence.

Nonlinearity of the height × ability correlation. Assuming normal distributions of height and general mental ability, or *g*, a linear correlation (Pearson *r*) of + .20 between these variables implies that the tallest 2 percent and the shortest 2 percent would deviate, on average, one half of a standard deviation above or below the mean of the total distribution of *g*. A significant discrepancy from this prediction would indicate nonlinearity of the regression of *g* on height, and would imply that the Pearsonian correlations most commonly reported in the literature to some degree underestimate the true relationship between height and *g*. The difficulty in examining this possibility is that a very large and representative population sample would be required to demonstrate with confidence a relatively small departure from linearity.

A recent study (Teasdale, Owen, & Sørensen, 1991) addressed this question with analyses based on a very large (N = 71,528) and highly representative sample of men in Denmark, aged 18 to 26 (mean 19.7) years. On a composite measure of general ability, the mean of the shortest 2 percent of this huge sample fell approximately two-thirds of a *SD* below the overall mean, but the tallest 2% were only slightly more than one-half of a *SD* above the mean. A plot of intellectual ability as a function of height shows a very slight but highly significant departure from linearity, such that when a quadratic term (i.e., height^2) was included in the regression equation, the increment over linear correlation is highly significant ($p < 3 \times 10^{-73}$!). Although the authors analyzed their data in a rather complex way, using canonical correlations, here we can express their essential results in terms that more easily permit direct comparison with all the previous studies we have reviewed based on simple correlations. With geographical region of origin and year of birth statistically controlled, the simple linear correlation (Pearson *r*) between height and mental ability is + .2207. The multiple correlation, adding a quadratic component, with both height and height^2 as the independent variables, is .2215. Although the slight difference between these correlation coefficients is undoubtedly reliable, given the enormous sample size, its theoretical meaning may seem hard to imagine. The authors suggest that some proportion of very short individuals may have been subjected to factors that are detrimental to both physical growth and mental development, a hypothesis which accords with a review (Skuse, 1987) of studies of short-stature persons that claims evidence of some "minimal impairment" in this group.

Correlation in children. It was proposed by the noted anthropologist Franz Boas (1895) that the correlation between intelligence and body size in children arose from individual differences in the rate of growth and development; growth rate was assumed to affect both body size and intelligence. This *co-advancement* theory, as it was known, also assumed that as the rate of development decreased as children grew older, the correlation between body size and IQ should decrease to zero by adulthood. Boas supposed there was no relationship between stature and mental status after maturity. Boas's co-advancement theory now has at least four strikes against it: (a) the size = IQ correlation is found to be at least as high,

or even slightly higher, in adults than in children; (b) a longitudinal study (Bayley, 1956) of children from ages 7 to 21 years showed high positive correlations (between .30 and .50) between height and intelligence scores at every age but found a nonsignificant *negative* relationship between mental and physical advancement *rates;* (c) although there are *within*-family differences (i.e., differences between siblings) in rates of physical and mental development, the evidence indicates that there exists no *within*-family correlation between IQ and height or weight; and (d) the correlations of height and weight with IQ (IQ measured at age 4 years) are virtually constant between the ages of 4 months and 4 years and between 8 years and 15 years of age (see Tables 4.2 and 4.3) no such constancy over these age ranges would be expected, because physical and mental growth rates are very negatively accelerated from infancy to maturity, and the sizes of the IQ × height (or weight) correlations do not appear to change systematically with the changing average differences in physical and mental growth rates throughout the course of development.

Three of the largest studies of the correlation between height and IQ in children, conducted in England and Scotland and described by Tanner (1969), are summarized in Table 4.2. The best estimate of the mean and the 95% confidence interval for the overall population value of the correlation is + .193 ± .10. A similar significant correlation was found between height and Raven Matrices scores in 98 third-grade Mexican children in Guadalajara (Pardo, Diaz, Hernandez-Vargas, & Hernandez-Vargas, 1971).

The largest American study with data relevant to this question is the Collaborative Perinatal Project of the National Institute of Neurological Disease and Stroke (an agency of the National Institutes of Health), which conducted a longitudinal investigation of 26,760 children from birth to 4 years of age (Broman, Nichols, & Kennedy, 1975). One hundred and seventy prenatal, perinatal, and early developmental variables, including Stanford-Binet IQ at age 4, were assessed in this study. Reported correlations between IQ at age 4 and height and weight measured at birth, 4 months, 8 months, 1 year, and 4 years are shown in Table 4.3. The correlations fluctuate very little with age. The correlations of height and weight with intelligence do not differ significantly at any age. The partial correlations indicate that weight (independent of height) has a slightly larger correlation with IQ than does height (independent of weight). Although higher levels of SES are underrepresented in this sample, the correlations shown in Table 4.3 probably do not appreciably underestimate the correlations between IQ and height or weight in the total population, because there is no restriction of range in IQ as compared with Stanford-Binet norms. The Collaborative Sample's IQ standard deviation is about 16.5 for whites and 13.9 for blacks.

The Pearson *r*, however, somewhat underestimates the true correlation of IQ with height and weight, because of nonlinear regression of IQ on the physical variables. The 4-year data, shown in Figure 4.5, are fairly typical. The higher Pearson *r*s (in Table 4.3) for blacks, as compared with whites, is clearly due to

Table 4.3. Zero-Order, Partial, and Multiple Correlations between Stanford-Binet IQ (I) at Age 4 Years and Height (H) and Weight (W) Measured at Various Ages

	Sample Size[a]				*Correlation*[b]											
	Height		*Weight*		*White*						*Black*					
Age	*White*	*Black*	*White*	*Black*	r^c_{HW}	r^d_{IH}	r^d_{IW}	$r_{IH \cdot W}$	$r_{IW \cdot H}$	$R_{I \cdot HW}$	r^c_{HW}	r^d_{IH}	r^d_{IW}	$r_{IH \cdot W}$	$R_{IW \cdot H}$	$R_{I \cdot HW}$
Birth	11,937	14,292	12,199	14,536	71	07	08	02	04	082	74	12	12	05	05	129
4 months	11,278	13,446	11,235	13,476	62	09	11	03	07	113	68	14	16	04	09	165
8 months	4,427	5,368	4,436	5,381	66	10	10	05	05	110	66	16	14	09	05	166
1 year	10,826	13,161	10,826	13,155	64	11	11	05	05	121	61	13	14	06	08	151
4 years	9,966	12,227	9,953	12,287	68	11	12	04	06	126	71	14	16	04	09	164
Mean[e]	9,686.8	11,708.8	9,729.8	11,767.0	664	095	104	036	054	109	686	135	144	051	074	153

[a]From Broman, Nichols, & Kennedy (1975), Appendix 3, Table 1.
[b]Decimals omitted. The zero-order correlations of IQ with height or weight are corrected for attenuation, based on IQ reliability of .83 (see Broman et al., p. 37). The partial and multiple correlations are based on the disattenuated zero-order correlations. All the zero-order correlations (r_{HW}, r_{IH}, r_{IW}) are significant at $p < .001$. The 95% confidence interval for all zero-order, partial, and multiple correlations (except those at age 8 months) is $r \pm .02$; for those at 8 months, it is $r \pm .03$.
[c]From Broman et al., Table 9.2, p. 126.
[d]From Broman et al., Appendix 4, Table 1.
[e]*N*-weighted mean, using Fisher's *z* transformation of *r*.

the greater linearity of the regression of IQ on height and weight in blacks than in whites. The shortest and lightest children contribute disproportionately to the correlation, which therefore would be markedly reduced if the smallest children within each age group were excluded. However, a significant relationship of IQ to height and weight has been found at every level of IQ.

A sample of 594 of Terman's gifted children, selected for Stanford-Binet IQs of 140 or above, showed the following correlations of Stanford-Binet mental age with height and weight, holding chronological age constant (Terman, 1926, p. 168):

	Boys (N = 312)	Girls (N = 282)
Height:	.219 ± .11	.211 ± .12
Weight:	.051 ± .11	.035 ± .12

The Harvard Growth Study (Dearborn, Rothney, & Shuttleworth, 1938) is a large longitudinal study of the correlation between physical and mental measurements obtained on the same group of children at yearly intervals between the ages of 7 and 18 years. The correlations are based on about 500 boys and 700 girls; a small percentage of the children was not measured every year, however, so the sample sizes for the obtained correlations vary slightly from year to year. Because the groups were very homogeneous in age at each measurement period, the correlations of height and intelligence with age are so small that partialing age out of the correlation between height and intelligence would not make an appreciable difference. The reliability of the intelligence measures varies slightly but unsystematically from year to year, averaging about .80. (The height-intelligence correlations could be corrected for attenuation by dividing them by the square root of the reliability, i.e., about .90, which increases the correlation by about .02 to .04.) Table 4.4 shows the zero-order uncorrected correlations between height and intelligence at every year of age between 7 and 18 years, for girls and boys. The most striking feature seen in Table 4.4 is the rather consistently lower correlation between height and intelligence in boys than in girls. (Boys' mean r = .227, SD = .036; girls' mean r = .287, SD = .054.) We can think of no plausible explanation for this correlation difference between the sexes. Another developmental study (Brucefors et al., 1974), based on 202 children, found a positive correlation between physical and mental growth *rates* over a much shorter age range for boys (4 weeks to 2 years) than for girls (2 to 8 years), resulting in a slightly higher correlation for girls, although the correlation for boys remains significant throughout the entire period of the study, from 4 weeks to 8 years.

The fact that the correlations for both sexes in the Harvard Growth Study are generally larger than those found in the other studies is probably attributable to the greater heterogeneity of the sample, which was obtained in Boston in the 1930s. The authors present evidence that the large proportions of children of

Northern and Southern European heritage in this population contributes to the height-intelligence correlation, as those of Northern European extraction measure taller and obtain higher intelligence test scores, on average, than those of Southern European extraction. In this population, for both girls and boys, the size of the correlation between height and intelligence shows a slight curvilinear (inverted U) relationship to age (see the correlations in the principal diagonal in Table 4.4).

The data of the Harvard Growth Study have been recently subjected to a number of sophisticated statistical analyses by Humphreys, Davey, and Park (1985) in an effort to understand better the nature of the height × IQ correlation. They confirm the overall correlation of about + .2 and the slight, but apparently real, sex difference in the correlation, for which they offer no explanation. However, a cross-lagged correlation analysis, which analyzes the changes in the correlation between height and intelligence when each variable is measured at *different* ages, showed that, for girls, individual differences in height predict individual differences in IQ measured several years later, which suggests a common causal factor in girls' development that affects individual differences in height earlier than in intelligence. The same effect shows up to a much slighter degree in boys, for whom the cross-lagged height × IQ correlations remain relatively more constant across all age intervals. In both sexes, but especially in girls, early height predicts later intelligence better than early intelligence predicts later height. Humphreys et al. state:

> Changes in biological functioning, whatever the causes may be, are not expected to have an immediate effect on intelligence behaviors. The intelligence measured by a standard test is a behavioral repertoire that is acquired over time. A biological deficit of less than traumatic proportions could affect future acquisitions but not the current repertoire. Thus intelligence would lag behind growth. (p. 1477)

Another interesting finding of this analysis by Humphreys et al. (1985) results from the different correlations obtained between IQ and *sitting* height as compared with *standing* height. The difference between the two height measurements, of course, reflects leg length. It turns out that virtually all of the height × IQ correlation is attributable to leg length. The correlation between sitting height and IQ, when standing height is partialed out, is either zero (for boys) or negative (for girls); but partialing sitting height out of the correlation between standing height and IQ has scarcely any effect for either sex. For girls, sitting height is negatively correlated with IQ; the girls' highly positive correlation between standing height and IQ is entirely attributable to individual differences in leg length! We can think of two speculative interpretations of this phenomenon: (a) Nutritional differences affect both mental and physical development, but the growth of the long bones of the legs is much more affected by nutritional factors than is the growth of other somatic features. (b) There is cross-assortative mating

Table 4.4. Correlations (Decimals Omitted) between Height and Intelligence Measured at Yearly Intervals from Age 8 to Age 17 for Girls and Boys (in Parentheses). (From Dearborn et al., 1938.)

	Intelligence at Age (Yrs.)									
Height at Age	***8 →***	***9 →***	***10 →***	***11 →***	***12 →***	***13 →***	***14 →***	***15 →***	***16 →***	***17 →***
8	31 (21)	31 (17)	35 (28)	38 (24)	41 (25)	33 (19)	32 (19)	34 (21)	32 (20)	25 (14)
9	30 (20)	32 (21)	34 (29)	38 (26)	40 (27)	35 (24)	33 (23)	35 (24)	32 (24)	25 (16)
10	29 (20)	31 (20)	33 (27)	37 (24)	39 (27)	33 (24)	32 (22)	33 (22)	32 (23)	25 (15)
11	27 (19)	29 (20)	31 (27)	35 (25)	38 (28)	32 (25)	30 (23)	32 (24)	32 (24)	25 (28)
12	28 (19)	29 (20)	29 (26)	33 (25)	37 (28)	32 (25)	30 (22)	32 (25)	31 (24)	25 (17)
13	27 (19)	29 (20)	28 (26)	31 (24)	34 (29)	31 (24)	29 (21)	30 (25)	30 (22)	23 (17)
14	25 (20)	27 (20)	26 (26)	30 (24)	33 (28)	29 (25)	28 (21)	28 (24)	27 (22)	24 (18)
15	22 (18)	25 (20)	22 (26)	29 (25)	31 (28)	27 (25)	26 (22)	26 (25)	24 (23)	23 (18)
16	20 (16)	24 (19)	21 (27)	27 (25)	30 (28)	25 (26)	24 (23)	23 (25)	22 (24)	22 (18)
17	17 (13)	21 (16)	16 (25)	23 (24)	25 (28)	21 (26)	19 (22)	18 (23)	18 (22)	21 (18)

between IQ (of men) and leg length (of women), resulting in a simple (between-families) genetic correlation between IQ and leg length in the offspring generation. A longstanding Western cultural stereotype of the attractive female (from the male viewpoint) is the long-legged female exemplified in the Petty-girl "calendar art." Beauty contestants—Miss America, Miss Universe—are notably tall and relatively long-legged compared to the average woman. Of course, these two hypotheses are not mutually exclusive; both could contribute to the observed phenomenon.

IQ, body size, and socioeconomic status (SES). The correlation between IQ and SES, as indexed by education, occupation, and income, is so well established and well known as to scarcely call for documentation. In school-age children the correlation averages about + .40, ranging from about .25 to .55 in various samples; in adults, the correlation is considerably higher, averaging about + .65 and ranging between about .55 and .75 (Eysenck, 1979; Jensen, 1973, 1980a; Tyler, 1965).

There is also a low but reliable positive correlation between body size (height or weight) and SES (Dearborn et al., 1938; Schreider, 1967; Tanner, 1966, 1969; Whipple, 1914, p. 70). Also, Schreider (1967) found that there is a negative correlation between the average height in different occupations and the standard deviation of IQs within those occupations; in other words, taller occupational groups (which also have higher average IQs) show smaller variability in IQ than shorter occupational groups.

The correlations between height and SES (correlations are not given between weight and SES) found in the Collaborative Study by Broman et al. (1975) are typical of those found in other studies of children, yet are much more reliable because of the large sample sizes. These are most easily summarized in terms of Figure 4.6. SES and IQ are correlated with height to about the same degree; partialing out each variable, of course, lowers the correlation, but not by very much. (Significant differences between zero-order and partial correlations are indicated by arrows.) The partial *r*s are all significant, indicating that both IQ and height are independently correlated with SES, which in this study is a composite index based on the head of household's education, occupation, and income. That IQ is correlated with body size independently of SES is further shown by the fact that a correlation of nearly the same magnitude also exists within broad SES categories, as shown in the study by Broman et al. (1975) and summarized in Table 4.5. These comparisons are especially important from a theoretical standpoint, because they make it implausible that environmental factors often associated with SES, such as nutrition and health care, could be major causal factors in the association between IQ and body size. The correlations *within* SES categories are barely smaller than the correlations in all of the SES categories combined. (Compare the within-SES correlations in Table 4.5 with the correlations for the combined SES groups at age 4 years in Table 4.3.)

Tanner (1969, pp. 194–198) has pointed out that height, considered indepen-

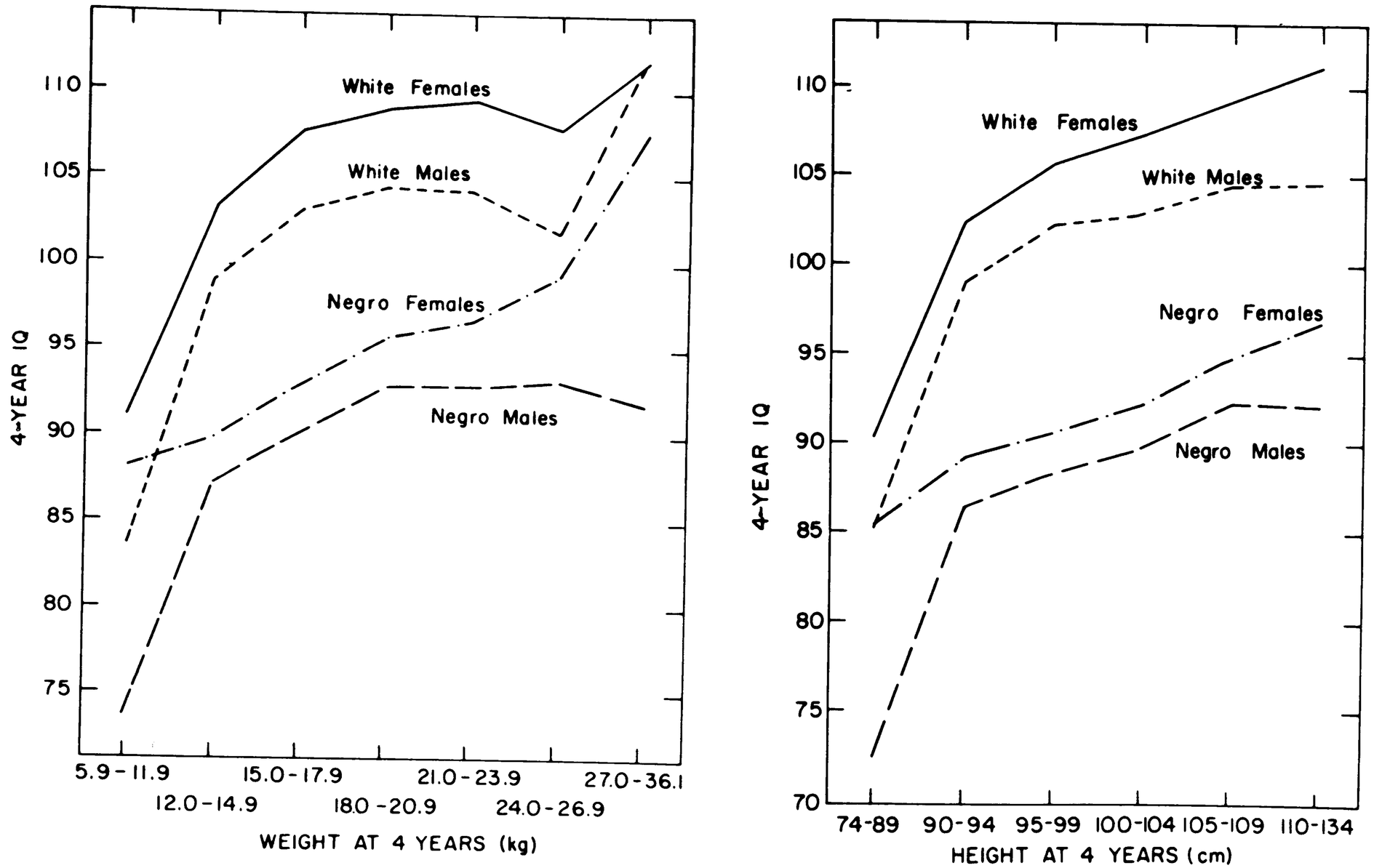


Figure 4.5. Four-year IQ by height and weight at 4 years. (From Broman et al. 1975, pp. 184, 188, reprinted by permission.)

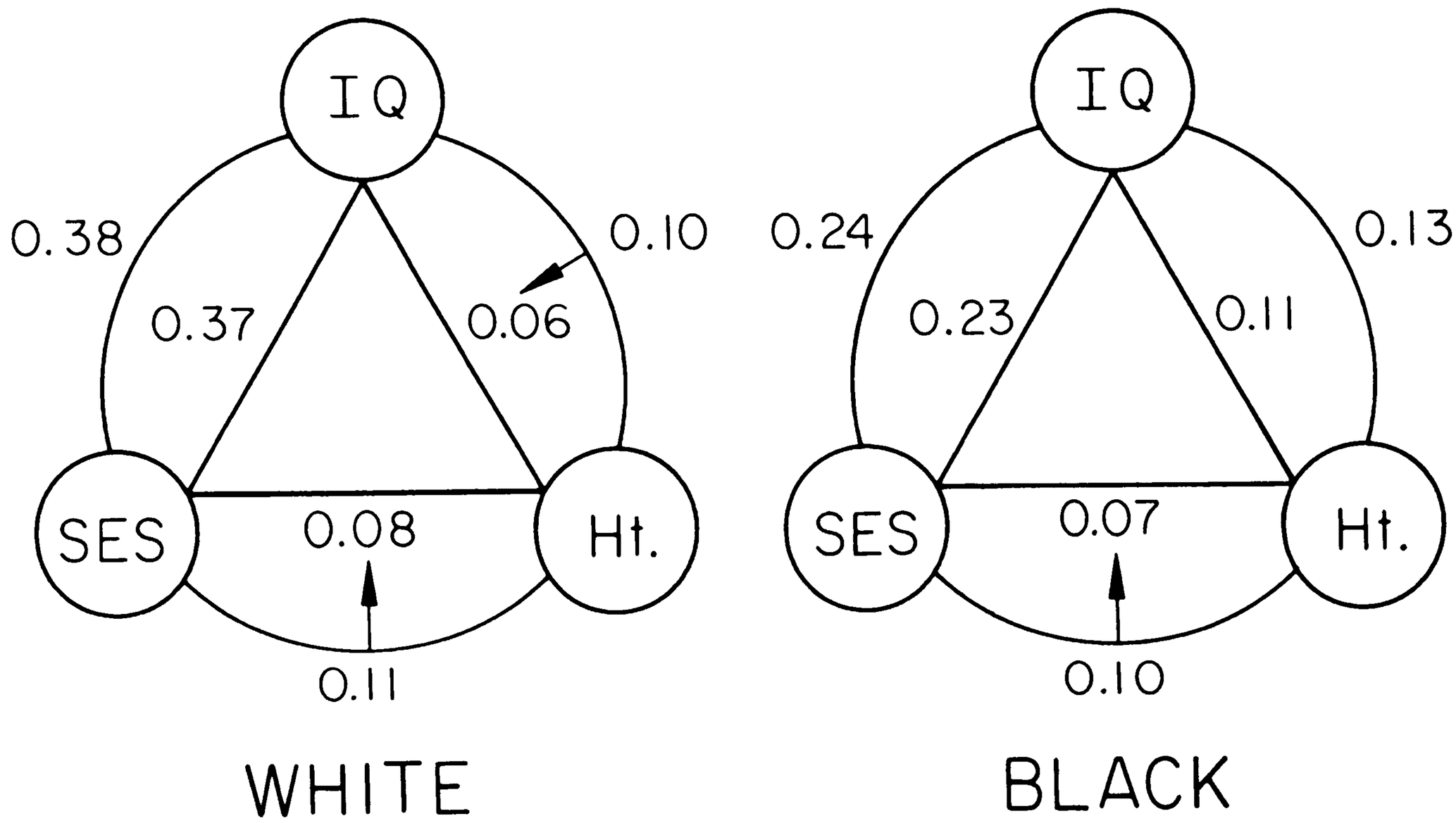


Figure 4.6. Zero-order correlations (curved lines) and partial correlations, with the third variable partialled out (straight lines) between age-4 Stanford-Binet IQ, age-4 height, and socioeconomic status. The 95% confidence interval for all correlations is $r + .02$. Arrows indicate the partial correlations which are significantly ($p < .05$, one-tailed test) smaller than the corresponding zero-order correlation. Whites $N = 9{,}790$; blacks $N = 12{,}064$. (Zero-order correlations from Broman et al. [1975].)

dently of SES and family origin, is positively related to social mobility. An analysis of this phenomenon by Schreider (1964) shows that among all women born into any given social stratum (as indexed by father's occupation), the taller women, on average, move upwards in their own occupational status and marry men of higher status, whereas shorter women, on average, move in the opposite direction. Examples of this relationship between height and social mobility may be seen most clearly in Figures 4.7 and 4.8 from Tanner (1969, pp. 196–197). (Note: The obvious drafting error in Tanner's figure [p. 197], viz., the reverse ordering of husband's occupation, has here been corrected in Figure 4.8.) The trends shown in Figures 4.7 and 4.8 are most likely mediated by the association between height and intelligence, for intelligence is even more markedly related to social mobility than height per se. However, the significant partial correlation between SES and height, with IQ held constant, suggests that height (or general body size) makes some slight contribution to social mobility independently of the association between height and IQ. If the data represented in Figure 4.7 are at all typical of other studies, husband's occupational status is highly correlated (about .65) with husband's IQ; hence, there is here a strong implication of positive cross-assortative mating for wife's stature and husband's intelligence. The genetic effect of cross-assortative mating for two heritable traits is to bring about a genetic correlation between the traits in the offspring generation, due to the

Table 4.5. Mean and Standard Deviation of 4-Year Stanford-Binet IQ and the Correlations[a] of IQ (I) with Height (H) and Weight (W) Measured at Age 4 Years in White and Black Samples[b]

	4-Year IQ				*4-Year Height*		*4-Year Weight*	
	White[c]		*Black*[c]		*White*	*Black*	*White*	*Black*
SES	*Mean*	SD	*Mean*	SD	r_{IH}	r_{IH}	r_{IW}	r_{IW}
Upper 25%	110.9	16.5	97.7	14.3	.08 ± .03	.12 ± .05	.10 ± .03	.10 ± .05
Middle 50%	101.3	15.1	92.0	13.5	.06 ± .03	.10 ± .02	.07 ± .03	.13 ± .02
Lower 25%	95.6	15.0	88.0	13.2	.12 ± .06	.14 ± .03	.11 ± .06	.15 ± .03

[a]Correlations given with 95% confidence intervals.
[b]Data from Broman et al. (1975), Tables 10.24 (p. 185) and 10.26 (p. 189).
[c]Total sample size: Whites = 9,790; Blacks = 12,064.

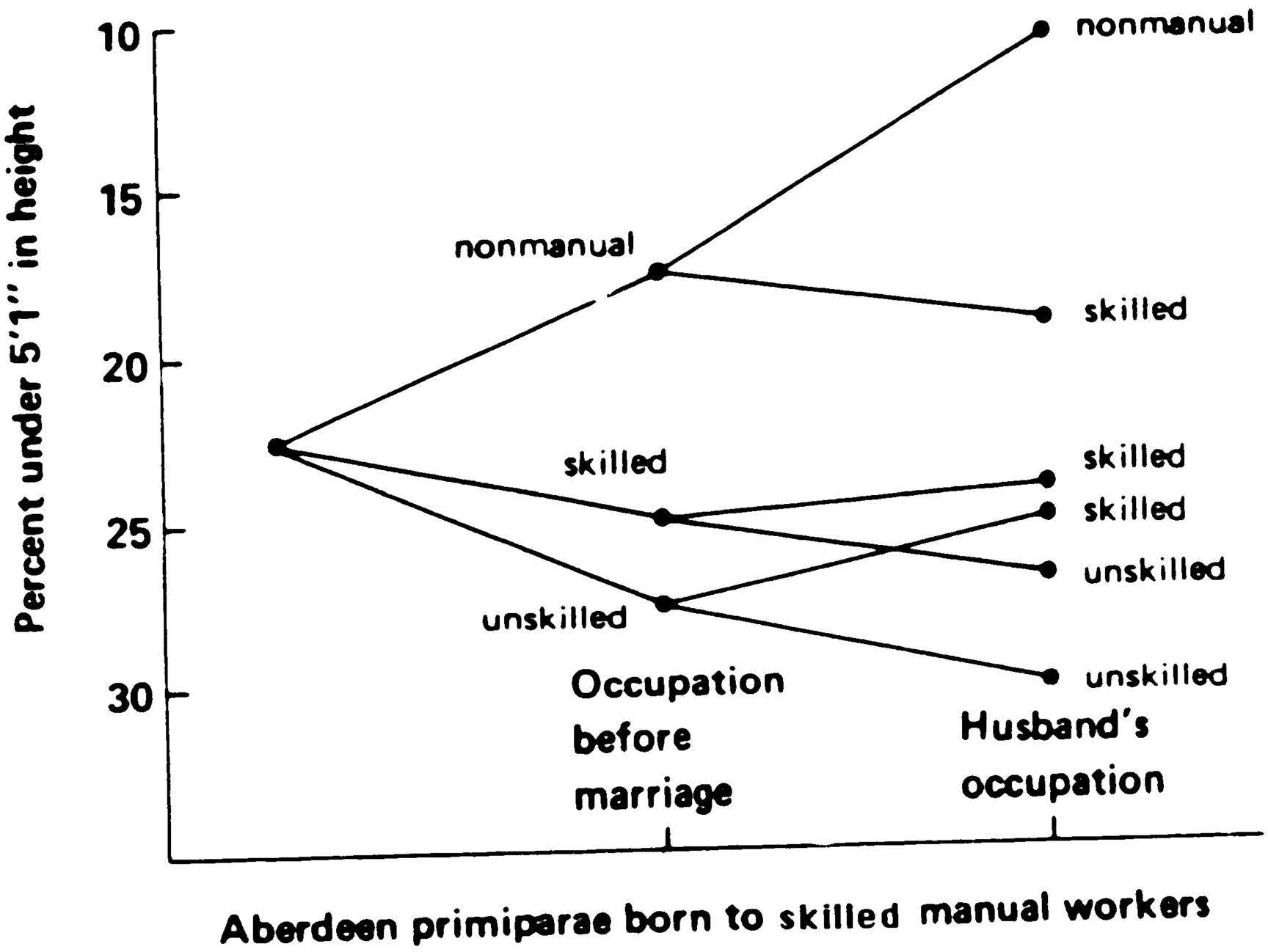


Figure 4.7. Percentage of daughters (of skilled manual workers) under 5'1" tall taking nonmanual, skilled, and unskilled manual jobs and marrying men in nonmanual, skilled-manual, and unskilled manual occupations. (From Tanner, 1969, p. 196.)

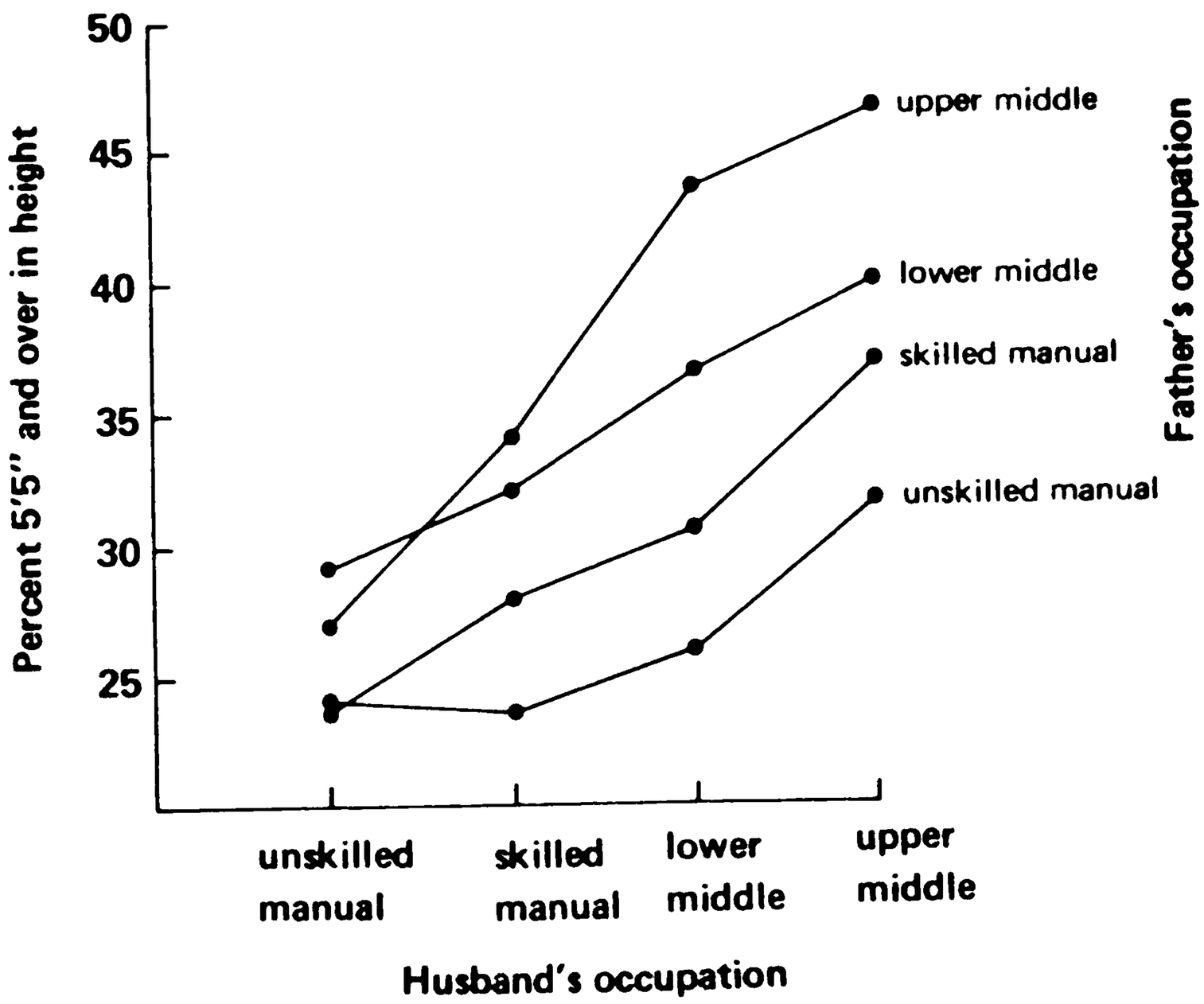


Figure 4.8. Percentage of British women 5'5" and over in height according to occupations of father and husband. (From Tanner, 1969, p. 197.)

common assortment of the independently segregating genes that affect each trait, a correlation that is manifested in the population as a correlation *between* families, but not *within* families. This interpretation of the correlation between intelligence and body size would be substantiated by studies which analyze the population correlation into its *between*-families and *within*-families components.

Between- and within-family correlations of body size and intelligence. There are studies reported in the literature which directly address the question of whether the body size × intelligence correlation observed in the population exists *within* as well as *between* families. Noting Terman's (1926) finding that gifted children, IQs of 140 and above, are taller and heavier and have generally better physiques than their agemates of average IQ, Laycock and Caylor (1964) decided to investigate this association in terms of between- and within-family correlation, by comparing "gifted" children with their "nongifted" siblings on a number of physical dimensions. They defined "gifted" to include IQs over 120 on the Stanford-Binet or over 130 on the California Test of Mental Maturity. In a large sample of gifted children of school age, they found that one in six had an older or younger sibling whose IQ was lower by 20 points or more. By these criteria, 81 pairs of gifted children and their nongifted siblings were selected for study. The gifted group had a mean IQ of 141.1, $SD = 13.5$; their nongifted siblings' mean IQ was 108.8, $SD = 10.8$. Thus the gifted and nongifted groups differ in IQ by 2.64 standard deviation units. The correlations between siblings in height ($r = .52 \pm .22$) and in weight ($r = .46 \pm .22$) are very typical of other studies, while the sibling correlation for IQ ($r = .68 \pm .22$) is higher (though not significantly so) than the r of .49, which is the average of all sibling correlations for IQ reported in the literature (Paul, 1980). Measurements of height, weight, shoulder width, and leg circumference, standardized for age and sex, were obtained on all subjects. The gifted children and their nongifted siblings showed no significant differences on any of the physical measurements. Such differences as were observed are all very small and nonsignificant, but are all in the direction that favors the gifted. The difference between gifted and nongifted siblings in height, for example, amounted to .059 *SD*, and for weight, .073 *SD*. This small disparity in these highly intercorrelated physical measurements is most likely due to sampling error and a possible slight error introduced by the study's dependence on age-standardized measurements. A more ideal, but scarcely practicable, method would have been to measure each pair of siblings at exactly the same age. (The use of same-sex fraternal, i.e., dizygotic, twins for this type of study would completely obviate the problem of age differences between full siblings, as dizygotic twins are full siblings, genetically speaking, born at the same time.)

These results are highly consistent with the hypothesis that the correlation between body size and IQ exists only between families. Laycock and Caylor (1964) attribute the positive correlation between physical and mental measurements to superior home care of gifted children and their siblings; in other words,

they hypothesize an environmental correlation. While this possibility cannot be ruled out, the very high broad heritability of stature (close to .95 in industrialized societies) and the substantial broad heritability of IQ (close to .70) would make it seem more likely that the association between IQ and stature is mainly a genetic correlation due to both assortative and cross-assortative mating for intelligence and stature, for which the average coefficients of assortative mating (i.e., correlation between mates) are about .45 and .30, respectively (Jensen, 1978).

The second study (Husén, 1959) compared the between-family (BF) and within-family (WF) correlations between height and IQ in samples of MZ and DZ male twins. Since both traits show substantial heritability, one should expect a difference in the ratio of BF/WF correlations for MZ and DZ twins if there is a WF genetic correlation between height and IQ. In that case the DZ WF height × IQ correlation should be larger than the MZ WF correlation, because in MZ twins all of the WF correlation would have to be environmental, while in DZ twins both genetic and environmental factors would contribute to the WF height × IQ correlation. Husén found no significant difference (in fact, it was slightly opposite to the theoretical prediction) between the WF correlations (relative to the BF correlations) obtained in the two types of twins. This finding is consistent with the absence of a within-family genetic correlation, and hence the absence of pleiotropy, between height and intelligence.

The third study (Jensen, 1980b) of within-family correlations is based on pairs of siblings from 1,495 white families and 901 black families in grades 2 to 6 (ages of about 7 to 12 years) in California schools. In all cases, the pair of siblings in each family nearest in age and enrolled in grades 2 to 6 was selected for study. In addition to measurements of height and weight, test scores were obtained on Verbal, Nonverbal, and Pictorial IQ (Lorge-Thorndike), Vocabulary, Reading Comprehension, and Short-Term Memory. All test scores as well as all height and weight measurements were age-standardized; score standardization was based on data for an entire school district with approximately 8,000 pupils. Correlations of height and weight with the seven mental tests were calculated *between* families and *within* families. The results clearly show that the correlation between the physical and mental measurements exists only *between* families, for which the average correlation is $+ .10 \pm .04$ (significant at $p < .01$), whereas the average *within*-family correlation is a nonsignificant $+ .02 \pm .04$.

The fourth study (Nagoshi & Johnson, 1987) examined BF and WF correlations between height and general intelligence (scores derived from the first unrotated principal component of 15 diverse mental tests, here referred to as *g*) in full siblings in the Hawaii Family Study of cognition. (Other analyses from the same Hawaii study, yielding highly similar results, are given by Baker, 1983.) The height × *g* correlations were computed separately between and within brother pairs, sister pairs, and brother-sister pairs. For Americans of European ancestry (AEA) ($N = 467$), the mean BF correlation between height and *g* is +

.17 ± .09; the mean WF correlation is + .07 ± .09. For Americans of Japanese ancestry (AJA) (N = 144), the mean BF correlation is + .10 ± .16; the mean WF correlation is − .02 ± .16. (The 95% confidence interval is given with each r.) These results are consistent with the hypothesis that the height × intelligence correlation is only a between-families phenomenon. In the AEA parents there was a significant cross-correlation between height and intelligence, but there was no significant correlation in the AJA parents, which might account in small part for the lower height × g correlation in the AJA than in the AEA offspring. The overall height × g correlation (averaged for males and females) in the AEA parents (N = 1959) is +. 14 ± 4.04; for their offspring (N = 768), + .10 ± .07. The corresponding correlations in the AJA parents (N = 766) and offspring (N = 321) are + .13 ± .07 and + .05 ± .11, respectively. The consistently higher height × g correlation in the parent than in the offspring generation suggests some environmental factor, perhaps nutrition, is largely responsible for the height × g correlation, assuming that the offspring generation has grown up under more homogeneous environmental conditions than their parents did, which seems a reasonable assumption.

Family size and birth order. Height and weight, like intelligence and SES, are negatively related to family size (see Tanner, 1969, for a good review of this evidence). All of these correlations are mediated by social class. Family size per se has virtually no causal effect on height, weight, or intelligence. A study of over 20,000 high school graduates in the United States showed that family size (i.e., number of siblings) accounted for four percent of variance in IQ; but when SES and race were controlled, family size and birth order together accounted for less than half of one percent of the IQ variance (Page & Grandon, 1979). The negative relationship of family size to IQ is clearly due to differences *between* families in other factors besides family size. Evidence from other studies supports the same conclusion. (For a comprehensive review, see Ernst & Angst, 1983.) The largest study of the relationship between height, intelligence, and family size, by Belmont, Stein, and Susser (1975), is based on a total sample of 234,837 Dutch conscripts tested at 19 years of age. Height measurements and scores on Raven's Standard Progressive Matrices were both transformed to a common scale (z scores) and plotted as a function of family size (total number of children), with the result shown in Figure 4.9. Birth order is also negatively related to both height and intelligence, as shown in the left-side panel of Figure 4.10. But when height and intelligence are plotted as a function of birth order separately for each size of family, so as to unconfound the correlated variables of family size and birth order, we see (as shown in the right-side panel of Figure 4.10) that intelligence is still related to birth order, whereas height shows no consistent relationship to birth order. The right-side panel of Figure 4.10 indicates the expected result if the negative relationship between birth order and intelligence is a *within*-family (as well as between-families) correlation, in contrast to height, which is unrelated to birth order *within* families. This finding

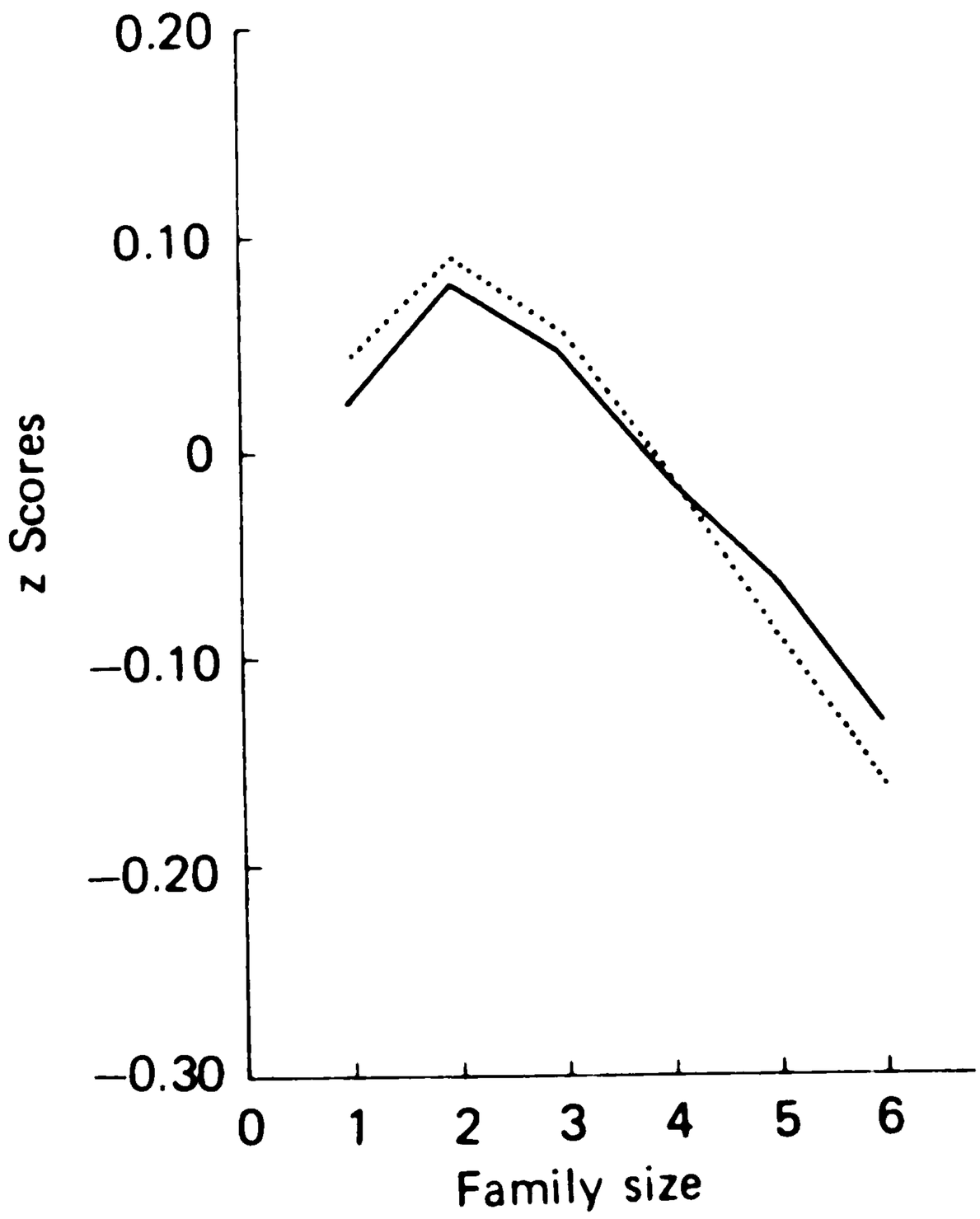
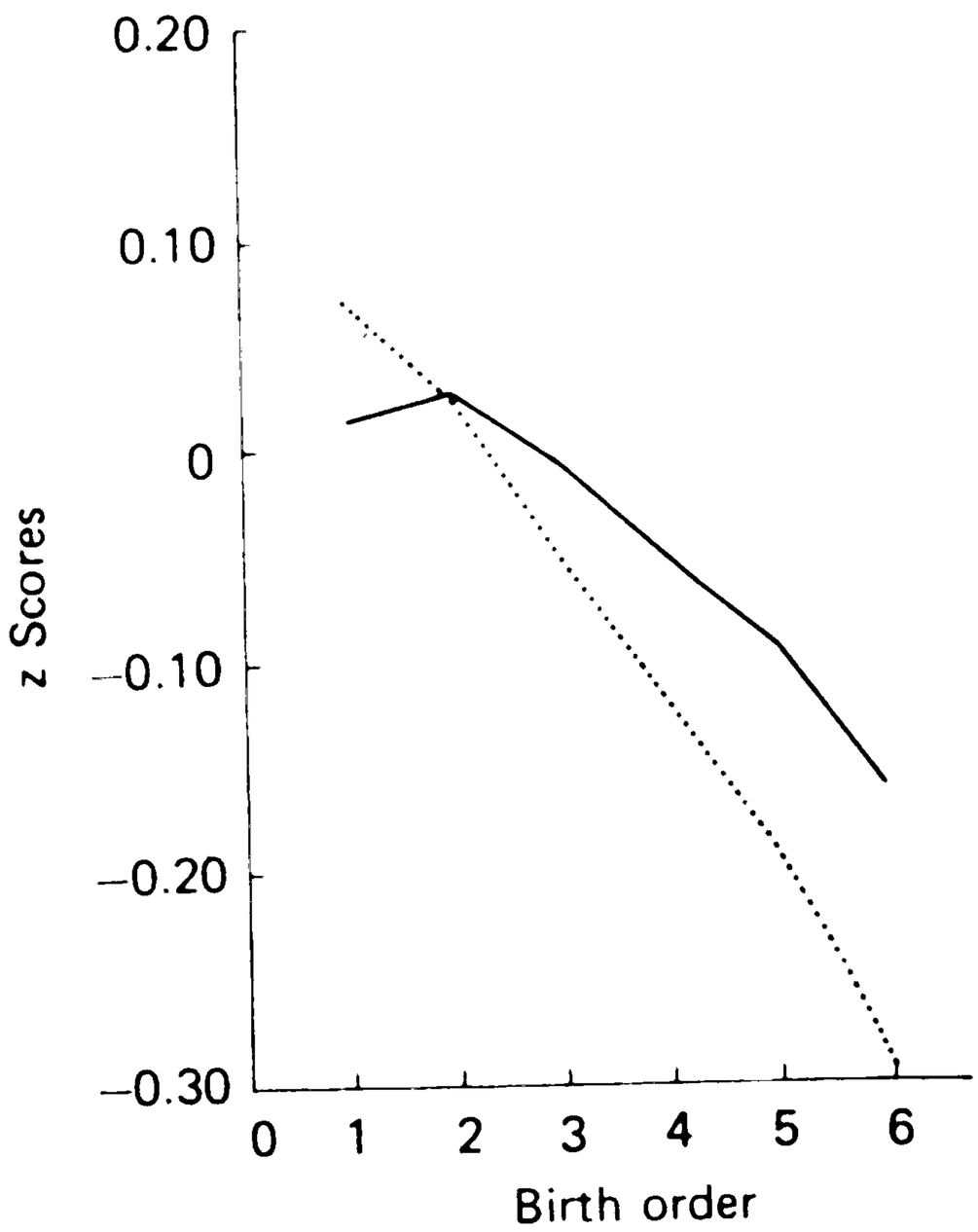


Figure 4.9. Height (solid line) and intelligence (broken line) by family size. (From Belmont, Stein, & Susser, 1975.)



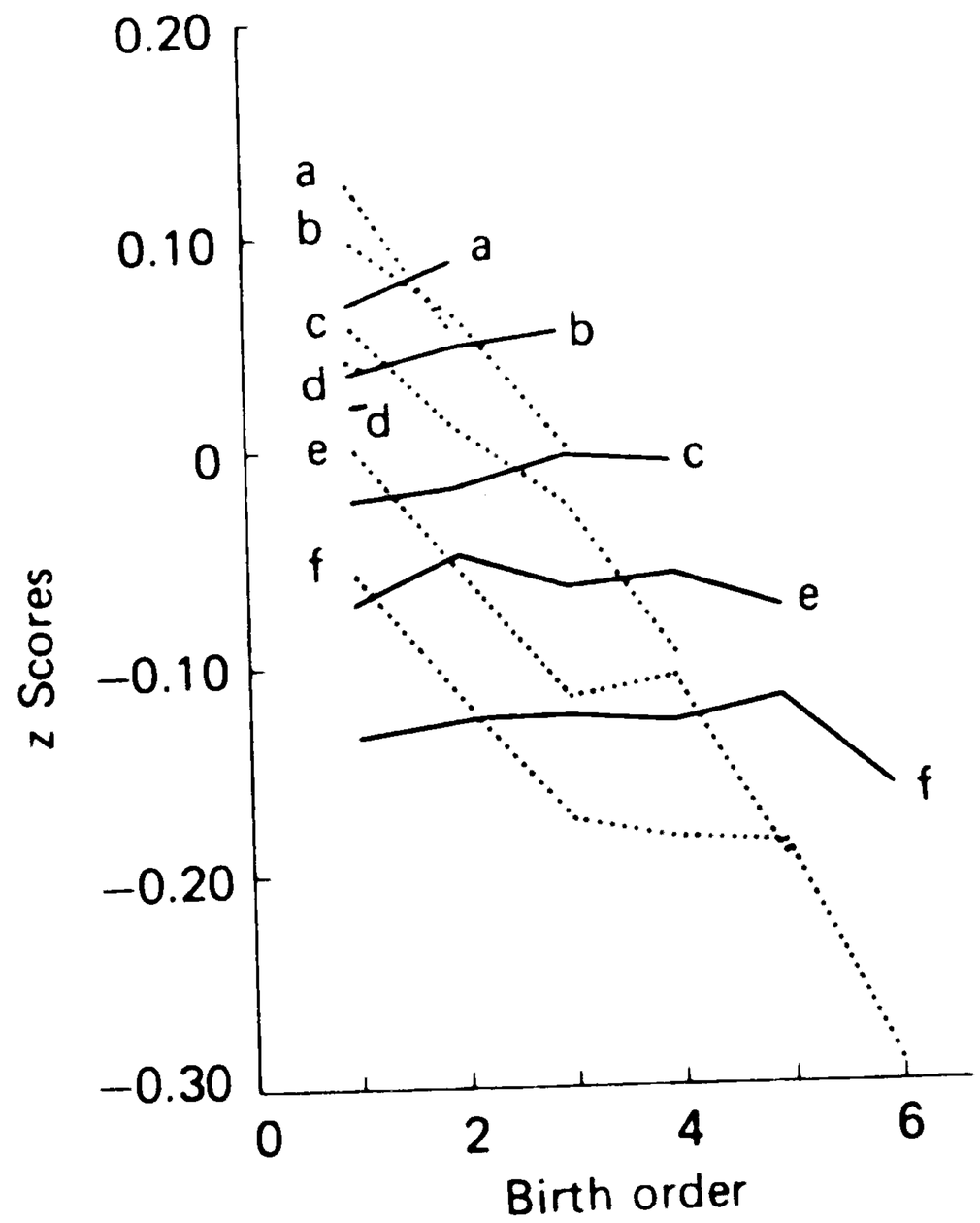


Figure 4.10. Height (solid line) and intelligence (broken line) as a function of birth order when (left panel) family size is confounded with birth order, and (right panel) birth order and family size are unconfounded. (From Belmont et al., 1975.)

of Belmont et al. (1975) is consistent with the failure to detect a significant within-families correlation between height (or weight) and intelligence in the previously described studies by Laycock and Caylor (1964) and Jensen (1980b). This is not to say that we believe the apparent birth-order effect on IQ shown by Belmont et al. is a direct causal effect of birth order per se. The observed effect is artifactual in the sense that it can be attributed to uncontrolled variables in the composition of the sample which are correlated with both IQ and birth order. An exceptionally thorough critical review of the literature on birth order and IQ, including the study by Belmont et al., points out these sampling artifacts and arrives at the following conclusion:

> In extremely *large and representative samples* IQ differences by birth order approach zero when [social] background variables are appropriately controlled. . . . The comparison of *sibs within the same sibship* excludes interfamilial differences and is of paramount importance for assessing whether birth order differences in IQ between unrelated sibs are within- or between-family differences. Among sibs within the same sibships birth order differences [are] near zero. Where they appear, they amount to 1 or 2 IQ points and are either sample-specific or due to disregarding the fact that when sampling younger sibs, those of large, narrowly spaced sibships are more likely to be included. (Ernst & Angst, 1983, p. 49)

Contradictory evidence. We have found only one study (Burks, 1940) that would appear to contradict our general conclusion that the correlation between body size and IQ exists between families but not within families. Yet the seeming contradiction is probably more apparent than real. In a group of 20 pairs of monozygotic twins, Burks obtained the correlations between the intrapair differences in IQ and the intrapair differences in several physical measurements. (The IQ and physical measurements are based on the average of measurements taken yearly over a period of several years [median of 7 years] between the ages of 3 to 11 years; this averaging has the effect of increasing the reliability of the composite mental and physical measurements used to obtain the intrapair difference correlations.) These correlations, with their 95% confidence intervals, are shown in the first column of Table 4.6. These are within-family (more precisely, within MZ twin-pair) correlations, and, at least for height, trunk length, and iliac width, are much higher than the usual between-family correlations of IQ with any physical traits. These quite large intrapair-difference correlations would seem to be inconsistent with the finding of many other studies that show negligible within-family correlations between IQ and measurements of body size. However, the fact that the correlations in Burks' study are based on MZ twins explains the apparent contradiction. Any differences between MZ twins, which have identical genotypes, necessarily arise exclusively from nongenetic, or environmental, causes. The fact that the nongenetic variance *within* pairs is very small relative to the variance *between* pairs (which is due to both genetic and nongenetic factors) is shown by the extremely high correlation between

twins on IQ and on the physical measurements (see second column of Table 4.6). The very small within-pair differences, perhaps arising from inequalities of the prenatal intrauterine environment, can affect both mental and physical development and hence can be quite substantially correlated *within* pairs, even though these particular environmental effects contribute probably as little as 1 percent to 3 percent of the total individual differences variance in either IQ or the body-size measures. In other words, the use of MZ twins eliminates the main source (i.e., the genetic component) of the within-family variance found with full siblings, who have only about half of their genes in common. The small amount of nongenetic variance, relative to the genetic variance, that could enter into within-family IQ × physical trait correlations based on full siblings would be almost completely swamped by the uncorrelated (within-family) genetic components of the mental and physical traits. Hence the results of Burks' study are what we should expect if the hypothesis is correct that the observed correlation between IQ and body size exists *between* families and not *within* families, as a consequence of a between-families simple genetic correlation between the traits. Note that the overall correlations in Burks' sample between *individual differences* in IQ and physical measures (last column in Table 4.6) are in the same range as those in most other studies. The real importance of Burks' study is that it shows that some part of the (IQ) difference between MZ twins is attributable to aspects of the environment which also affect physical growth and therefore are not entirely of a psychosocial nature.

Table 4.6. Correlation (and 95% Confidence Interval) of IQ and Anthropometric Measurements in Monozygotic Twins[a]

Trait	*Correlation between Intrapair IQ Difference and Intrapair Difference in Physical Trait[b]*	*Correlation between Twins[c]*	*Correlation between IQ and Physical Trait[d]*
IQ		.95 ± .06	
Height	.47 ± .35	.96 ± .06	.17
Weight	.12 ± .43	.98 ± .03	−.02
Leg Length	.11 ± .43	.92 ± .09	.29
Trunk Length	.40 ± .38	.98 ± .03	.08
Iliac[e]	.41 ± .38	.89 ± .12	−.11

[a]From Burks (1940, pp. 89–90).
[b]Based on average intrapair differences of 20 MZ pairs of both sexes.
[c]Based on 10 pairs of male MZ twins of ages 9 years 7 months to 10 years 6 months.
[d]Based on 21 males (members of 11 twin pairs), ages 9 years 7 months to 10 years 6 months. Confidence intervals not computed because of the high intrapair correlations on these traits.
[e]For a single measure of the iliac taken beyond age 12 years 6 months, the correlation with IQ drops to −.04 ± .46.

Causal factors. If the correlation between body size and intelligence is an exclusively between-families correlation and does not exist within families, as the preponderance of the evidence suggests, it necessarily follows that the correlation is not pleiotropic, but that it is instead the phenotypic expression of either a genetic correlation or an environmental correlation, or some amalgam of both. The fact that the correlation is found within every level of social class, in groups with little or no variation in nutrition or other health factors, in addition to the extremely high heritability of stature and the moderate heritability of intelligence, makes it more likely that a genetic correlation, rather than common environment or correlated environmental factors affecting both size and intelligence, is the principal cause of the phenotypic body size × intelligence correlation. This correlation would seem to be maintained at a "steady-state" level of close to + .20 in the young adult population, most probably as a result of assortative and cross-assortative mating for stature and intelligence in every generation. Men and women tend to choose mates of similar stature (with an assortative mating coefficient of about + .20) and even more similar intelligence (coefficient of about + .40), with the result that genes for both traits are assorted together in the offspring. Although it is easy enough to understand homogamy for these two traits, it seems somewhat more difficult to account for the existence of cross-assortative mating for stature and intelligence. Two hypotheses are suggested: (a) social class propinquity, that is, the greater probability of mating within, rather than outside of, one's own social class, plus the fact that social classes differ statistically in stature and intelligence, and (b) both high intelligence and tall stature tend to be valued as personal qualities in our culture and there is some degree of "tradeoff" between these qualities in mate selection.

We have found only one other type of explanation for the stature × IQ correlation. A positive relationship has been noted between stature and vanillyl mandelic acid (VMA) level in the urine (Henrotte, 1967). VMA is a breakdown product of catecholamine metabolism. As catecholamines are neurotransmitters involved in states of arousal, tone of awareness, and alertness, they may also be related to intelligence. It is on this basis that Henrotte suggests the hypothesis that the correlation between stature and the intelligence levels of different occupational groups is a result of differences in catecholamine metabolism. Unfortunately, the study does not distinguish between-families and within-family correlations of VMA level with stature. One would expect such a correlation to exist within, as well as between, families, possibly as a pleiotropic effect, that is, as the result of a gene affecting both catecholamine metabolism and stature. The apparent absence of a within-family correlation between height and intelligence, however, is inconsistent with the hypothesis that both stature and IQ are *causally* related to VMA. It is possible, of course, for there to exist a pleiotropic correlation between VMA and intelligence, and a (noncausal) genetic correlation between stature and intelligence. If such is the case, one would predict a within-family correlation between VMA level and intelligence and only

a between-family correlation between VMA level and stature. This prediction would seem more likely than the reverse, because of the known connection between VMA and neuronal activity. What is clearly needed is a sibling study of the correlation between VMA and intelligence.

Height-weight ratios and indices. Early students of the body size × intelligence relationship, finding only very small correlations of the primary measurements of height or weight with mental test scores, hoped to discover some nonadditive combination of bodily measurements that would yield a more impressive correlation. Combinations of various ratios were tried, such as weight/height^2, weight/height^3, sitting height/height, height–chest girth + weight, and so forth. The results of these efforts have been reviewed by Paterson (1930, pp. 166–169), who reports that none of the indices yielded significant correlations with intelligence. The correlations based on compound measures were no larger, and often smaller, than those found for simple measurements of height or weight. The correlation of the height/weight ratio with IQ in 206 high school seniors and college freshmen was an unimpressive + .10 ± .14, for example (Paterson, 1930, p. 167). Yet given the relatively small samples used in most of these studies, the 95% confidence intervals for most of the correlations are between $r \pm .17$ and $r \pm .20$. We have not found any correlational studies making use of such compound physical indices reported in the literature since Paterson's review.

Obesity and IQ. Although weight, being highly related to general body size, is positively correlated with IQ, obesity (defined as 20% or more overweight for sex, age, height, and build) has been found to be *negatively* correlated with IQ in adults (Kreze, Zelina, Juhas, & Garbara, 1974). The negative correlation is much higher in women than in men. The percentages of women in the lower and upper quartiles of IQ who were classified as obese are 41.4% and 10.7%, respectively. The corresponding percentages for men are 17.0% and 9.3%. The inverse relationship between IQ and obesity is most likely mediated by a third variable—social class, which is positively correlated with IQ and negatively correlated with obesity (Goldblatt, Moore, & Stunkard, 1965). The hypothesis that the *negative* correlation between obesity and social class in Western European and North American populations reflects different cultural norms associated with social class is further supported by the finding of a *positive* correlation between obesity and social class in India (Siddamma, 1978).

Children 6 to 7 years of age who are overweight (a weight/height ratio above the group median) performed significantly less well than underweight children on 15 Piagetian conservation tests involving the conservation of number, volume, matter, length, and weight; the overweight group scored lower than the underweight group in conservation performance even when IQ was controlled (Ewert, 1977). The author attributed the results to the greater susceptibility of the overweight subjects to cue salience, that is, they were more field-dependent as compared with the relatively field-independent underweight subjects. There is no evidence on this effect *within* families.

Head and Brain Measurements

The early literature on the relationship of head and brain measurements to intelligence, and even the treatment accorded to this topic in present-day psychology textbooks, affords little indication of the surprisingly complex technical problems that have made it difficult for scientists to agree as to the precise nature of this relationship. The most we can attempt here is to point out these technical problems and summarize the present state of the best available evidence, such as it is.

Interspecies comparisons of brain size. The apparent differences between various species of animals in what people commonly think of as the capacity for intelligent behavior, and the perceived relationship of such species differences to differences in brain size, are among the observations that have tempted both scientists and laymen to inquire whether individual differences in brain size among humans are correlated with individual differences in psychometric intelligence. The question has also been raised whether various racial groups among the species Homo sapiens differ in brain size, and whether such differences are reflected in the observed racial differences in psychometric intelligence.

In the five million years of human evolution, from Australopithecus to Homo sapiens, the brain has almost tripled in size, despite the anatomic and metabolic disadvantages of larger brain and head size. The chief advantage of a larger brain, in terms of natural selection, is the greater capacity it confers for complex adaptive behavior. Development of the cerebral cortex, the association areas, the frontal lobes, and, in general, those parts of the brain not directly involved in autonomic and sensory-motor functions, is related to the complexity of behavioral capacities such as perceiving, learning, reasoning, problem solving, and language. Hence, the question persists concerning the relationship between differences in brain size (or its correlate, head size) and intellect.

In considering this relationship among a wide range of mammalian species differing greatly in overall body size, brain size must be regarded in relation to body size, that is, allometrically. The size of every bodily organ is allometrically related to total body size, and a very high correlation exists between body weight and mean brain weight across species. For 93 species of mammals, varying in size from mouse to elephant, the correlation between the means of body and brain weight is .976; among only 15 species of primates the correlation is .973 (Armstrong, 1983). This high correlation, which represents a true functional relationship, and not just a statistical association, results from the fact that much of the brain serves vegetative and sensorimotor functions, and that the number of neurons subserving these functions is directly related to body size, or, more specifically, to the total surface area of the body. The high brain-body size correlation is also reflected in the extremely high correlation between body size and the cross-sectional area of the foramen magnum, an opening in the skull through which the spinal cord passes. Because all but a dozen or so cranial nerves pass through the foramen magnum, this measurement is useful as an index

of the total sensory-motor input-output of the animal's brain. The size of the medulla, a measurement that has been used as an alternative index of sensory-motor input-output (Passingham, 1975), is also strongly correlated with body size.

But it is the amount of brain tissue in excess of that part that is predictable from body size and subserves purely vegetative and sensory-motor functions that is most apt to be related to the animal's capacity for developing complex behavior.

This "excess" brain tissue in various species is measured from the regression of brain weight on body weight. In practice, the log of brain weight is plotted as a function of the log of body weight for a large number of mammalian species. The regression equation relating log brain weight to log body weight serves as a baseline for comparing various species on the amount of brain tissue above (or below) the amount predicted on the basis of body size. (Instead of the regression line, the principal axis of the plotted log brain weight by log body weight is recommended by some investigators.) The amount of deviation from the common regression line (or the principal axis) for the mean of any particular species has been termed an *encephalization index* (Jerison, 1973, 1982). This residual brain mass has been expressed mathematically with various modifications and refinements by different investigators (Passingham, 1975), but all such indices are essentially intended to express the degree of encephalization or development of the neocortex, especially that part of the brain that serves complex behavioral capacities, over and above the neural mass associated with general mammalian functions and closely related to overall body size.

The importance of the encephalization index for behavioral science derives from its close relationship, as contrasted with that of overall brain size, to the varying information-processing capacities manifested by different species. Degree of encephalization is found to be related to objective measures of animal intelligence such as "curiosity" (as measured by responsiveness to novel objects) and the speed of acquiring discrimination learning sets, which shows a rank-order correlation of +.96 with an index of encephalization (ratio of neocortex to medulla) among nine species of primates (Passingham, 1975). It is noteworthy that speed of acquisition of discrimination learning sets is also correlated with psychometric intelligence in children (Harter, 1965). On the basis of such evidence, Jerison (1982) claims this encephalization index as a measure of the "biological intelligence" of various species.

Jerison (1982), however, makes the important point that the high degree of relationship between encephalization and biological intelligence across different species does not hold up for individual differences or even for subspecies (or racial) differences within a major species. As Jerison expresses it, the intraspecies relationship of encephalization to behavioral capacity shows none of the orderliness of the interspecies picture. According to Jerison, "Individual variation, the source of microevolution, seems to be decoupled from interspecific

variation, which represents the effect of macroevolution'' (p. 743). Not all expert opinion agrees with Jerison on this point, however. In a review of Jerison's major work (*Evolution of the Brain and Intelligence,* 1973), Holloway (1974) dissents from Jerison's position as follows:

> If there is no regular relationship obtaining within the species between brain and body weights, and between brain weight and information-handling capacity, what are the driving forces or evolutionary dynamics that produce the lawful relationships between species or between higher taxa? Somewhere, there is a hiatus in explanations which claim a set of biological (functional) relationships at supra-species taxon levels but deny such a relationship within the biological unit (the species) undergoing evolutionary change. (p. 679)

Yet, only a slight (but significant) degree of statistical relationship is found between brain-size indices and measures of intelligence *within* any species, including modern Homo sapiens. It would appear, then, that the biological basis of intraspecies individual differences in intelligence probably resides much more in the fine structure of the neocortex (such as the amount of branching and the number of interconnections between neurons) and in the chemistry of neurotransmitters, than in the gross anatomy of the cerebrum. Nevertheless, we are still left with some small but significant correlation between brain size and psychometric intelligence in humans. Because of a number of technical difficulties, however, the precise value of this correlation remains arguable.

Measurements of brain size. The primary problem in the study of brain size and intelligence in humans is the measurement of brain size itself. As Van Valen (1974) notes, there is no study reported in the literature prior to 1974 that directly correlates brain size, as measured by weight, volume, or even cranial capacity, with intelligence test scores. With one exception (Willerman, Schultz, Rutledge, & Bigler, 1989), all existing studies are based on *estimates* of brain size or cranial capacity derived from external measurements of the head. The question naturally arises as to the validity of such estimates as measures of brain size. Studies based on direct measurements of excised brains or cranial vault capacity have had to depend on such rough estimates of intelligence as years of schooling or occupational level, as determined from death records. Another serious problem in this line of research is emphasized by Passingham (1979, p. 255), the author of a recent study of brain size and intelligence, who reports that there are no studies based on truly representative samples of the general population. Although Passingham does seem to have overlooked one excellent study by Susanne (1979) that is based on a highly representative sample of young men in Belgium, his point is well taken. In general, the use of nonrandom samples would tend to bias the data to some unknown degree toward a restriction of variance in the variables of interest, the variance of intelligence being most likely affected, with the inevitable result of some diminution of the obtained correlation.

Studies of the validity of external head measurements as estimates of direct brain measurements find correlations between the two variables in the range of .60 to .70 (Van Valen, 1974, p. 423). Head circumference is the most commonly used measure in correlational studies with IQ. Yet, in a study of Jørgensen, Parison, and Quaade (1961, cited by Van Valen, 1974, p. 423) of 89 persons, the correlation between brain volume and head circumference is only $+.50 \pm .21$. Two other studies of the same variables cited by Van Valen (1974, p. 423) give correlations of $+.55 \pm .10$ and $+.36 \pm .14$.

Linear measurements of the skull taken internally and externally are much more highly correlated with one another than are brain volume and head circumference. Hoadley and Pearson (1929) obtained internal $\times$ external correlations of $+.78$ for cranial length and $+.88$ for cranial breadth. The N-weighted mean of eight coefficients of correlation between direct measures of cranial capacity and linear measurements of the external size of the cranium was reported as $+.66 \pm .08$ (Macdonnell, 1904). Somewhat higher correlations have been found between external head circumference and internal skull diameter and volume measured from X-rays (Bray, Shields, Wolcott, & Madsen, 1969; MacKinnon, Kennedy, & Davies, 1956).

In studies based on living subjects, IQ is usually correlated with head circumference (taken with a tape measure) or with linear dimensions of the head (measured by calipers). When linear dimensions are taken, an estimate of brain capacity may be derived from head length, width, and height measurements, in accordance with a formula devised by Lee and Pearson (1901). Their formula (No. 14, 1901, p. 252) estimates brain volume in cubic centimeters as follows:

For men: Brain $cm^3 = .000337(L - 11\text{ mm})(W - 11\text{ mm})(H - 11\text{ mm}) + 406.01$, where L, W, and H are length, width, and height in millimeters. For women: Brain $cm^3 = .0004(L - 11\text{ mm})(W - 11\text{ mm})(H - 11\text{ mm}) + 206.6$. The amount of 11 mm is subtracted as representing the average thickness of scalp and skull. Scalp thickness varies slightly with the amount of body fat, and this, of course, adds to the error of measurement. Brain weight in grams may then be estimated by multiplying the results obtained from Lee's formula by 0.87. (Brain weight is estimated from the *direct* measurement of cranial capacity by an equation given by Baker [1974, p. 429]: Brain weight [grams] $= 1.065\ cm^3 - 195$. Cranial capacity is measured directly by measuring, in a graduated cylinder, the amount of buckshot or mustard seed required to fill the skull.)

The main point of all this is that correlations between brain size and intelligence based on living persons for whom IQs are obtainable suffer attenuation from several sources between the external measurements at the surface of the scalp and the brain itself. Head size is far from perfect as a correlate of brain size and may even be correlated to some extent with total body size independently of brain size. If it is assumed that external measurements of head size (h) are correlated with intelligence (i) only through their relationship with brain size (b),

then it can be proved mathematically (see Van Valen, 1974, p. 423) that the correlation between head size and intelligence can be expressed as $\rho_{ih} = \rho_{ib}\rho_{bh}$, and the correction for attenuation of ρ_{ih} as an estimate of ρ_{ib} is, of course, simply ρ_{ih}/ρ_{bh}. For our purpose, a measure of overall body size should be partialed out of ρ_{ih} *and* ρ_{bh}, as we are interested in determining the degree of association between intelligence and that part of the variance in brain size which is independent of general body size.

Measurements of intelligence are also attenuated, with reliability in the best IQ tests generally ranging between .90 and .95. In addition, most IQ tests measure other factors of ability besides *g*, the general intelligence factor common to all complex cognitive tasks. As true scores from most ordinary IQ tests are correlated between .80 and .90 with *g*, moreover, it follows that the correlation between obtained scores and *g* falls somewhere between .75 and .88.

Age differences may also attenuate the correlation between head measurements and IQ, as brain size decreases relatively more after age 25 than does head size. Actual brain weight decreases some 100 grams or more between ages 25 and 80. There is an average negative correlation of about $-.20$ between brain weight and age between ages 25 and 80 (Ho et al., 1980a). When corrected for general body size (as measured by total body surface area) the correlation between brain weight and age (between 25 and 80 years) is $-.117 \pm .10$ for white men (Ho, Roessman, Straumfjord, & Monroe, 1980b). Hence, without proper correction for age differences, the head size $\times$ IQ correlation will be attenuated to some slight degree in samples of variable age; this effect is so slight between ages 18 and 60 (Ho, Roessman, Straumfjord, & Monroe, 1980a, 1980b), however, as to be of negligible impact when body size is statistically controlled. (Adult height is correlated negatively with age approximately $-.15$ to $-.20$.)

As may be seen in Figure 4.11, which shows the distribution of directly measured brain weights of 733 English men (Passingham, 1979), the distribution of brain size in adult humans roughly approximates the normal, or Gaussian, curve, although this distribution is not completely symmetrical around its mode. A similar-shaped distribution for 505 unselected European men, ages 21 and over, is shown in Figure 4.12. A normal curve has been fitted to both sets of data in Figures 4.11 and 4.12, and a chi-square test shows a close degree of approximation to normality, with neither set of data deviating significantly (at the 5% level) from the normal curve. A larger set of data is provided in a study by Miller (1926), in which cranial capacity (estimated by Lee's formula) was obtained on 4,012 school boys ranging in ages 7 to 17. We have age-standardized the cranial capacity measurements by converting them to *z* scores within each one-year age interval. The distribution of cranial capacities (age controlled) does not differ significantly ($p > .05$) from the normal distribution fitted to these data. In every study, the departures from normality, though not statistically significant in

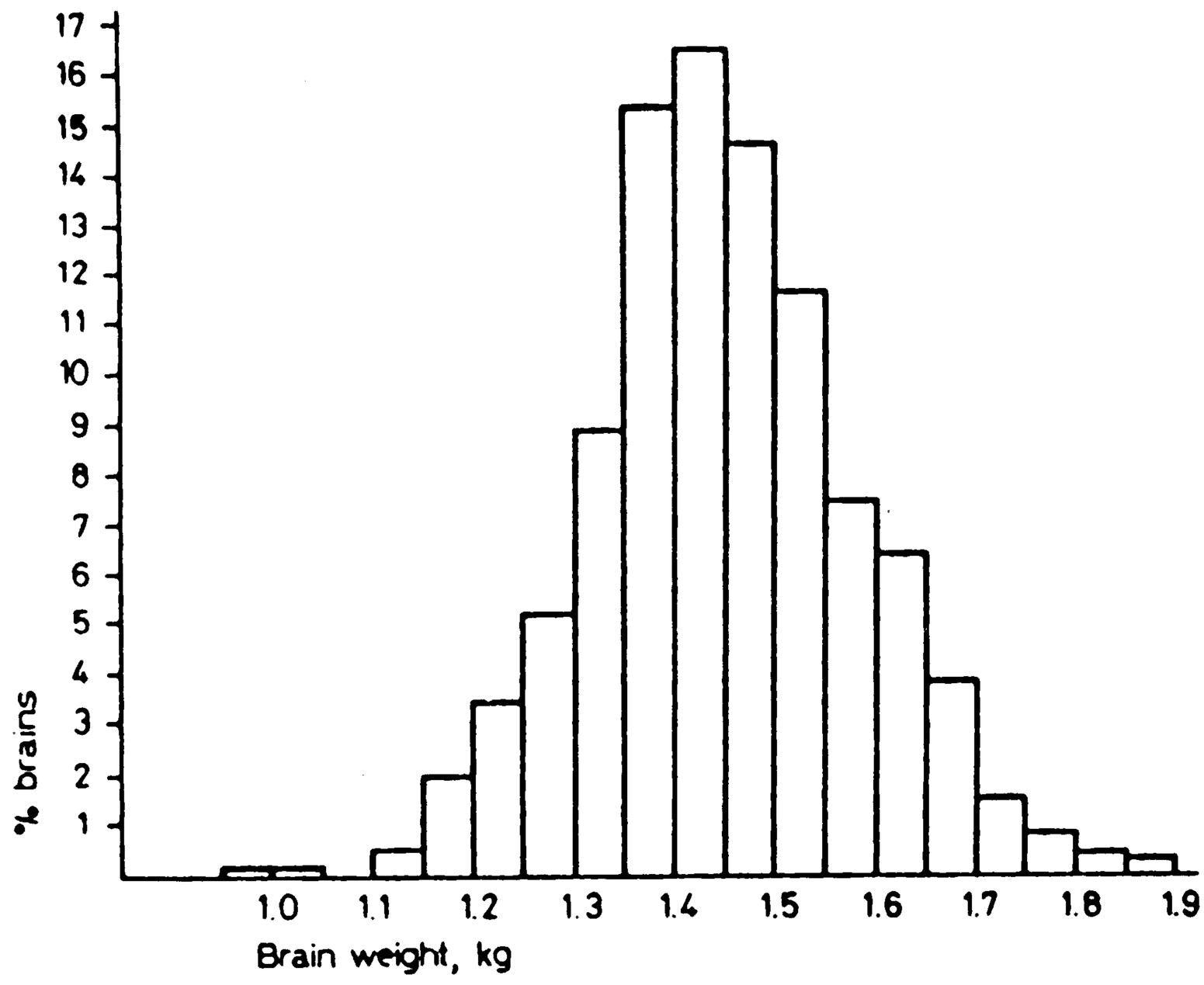


Figure 4.11. Histogram of brain weights (in kilograms) for 733 English men. (From Passingham, 1979, p. 261.) When a normal curve is fitted to these data, with $\overline{X}$ = 1.442 kg, *SD* = .134 kg, a chi-square test of the goodness of fit of the data to the normal curve gives χ^2 = 28.7, with 18 *df*, $.05 < p < .10$; i.e., the data depart significantly from normality at $p < .10$, but not at $p < .05$.

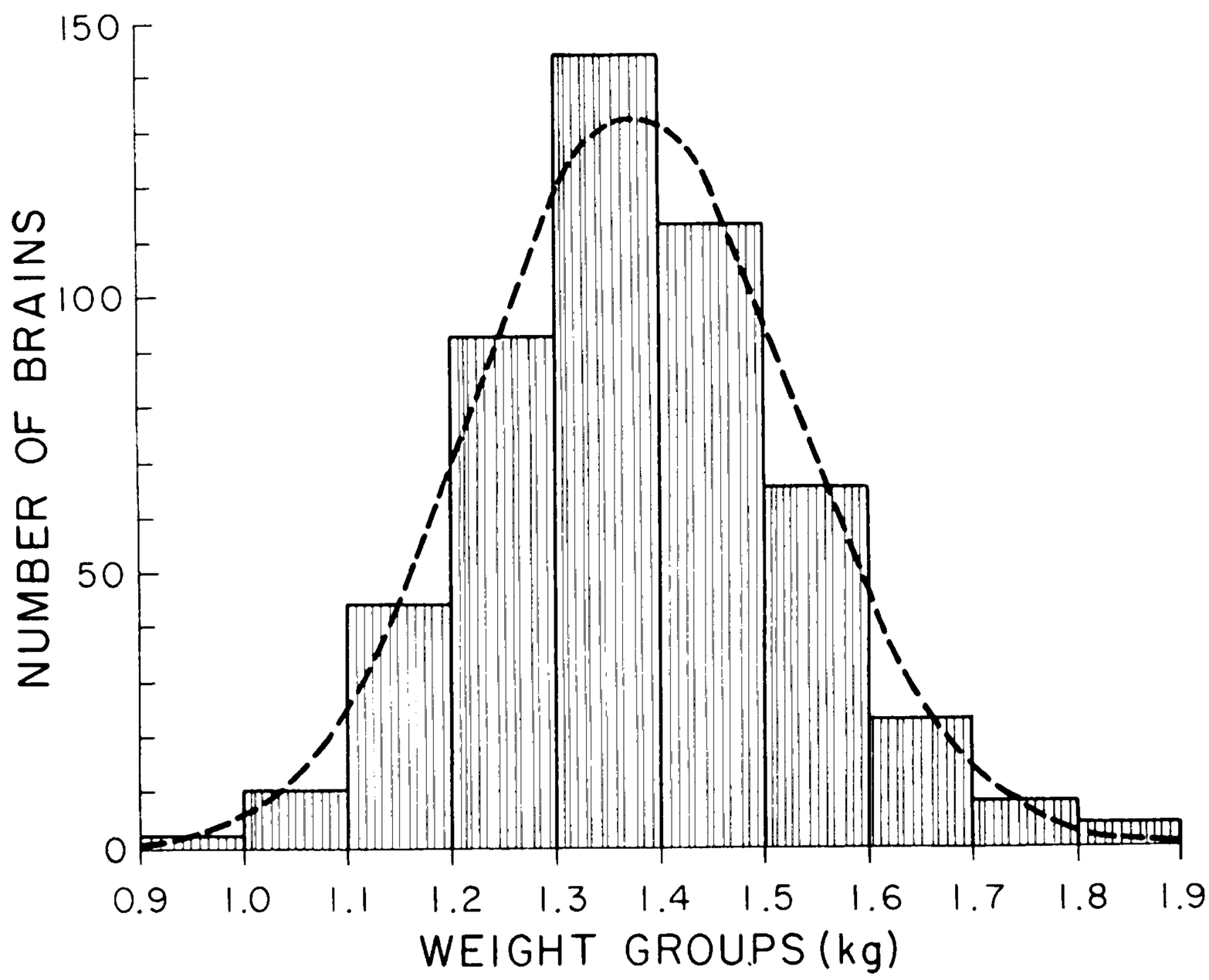


Figure 4.12. Histogram of brain weights of European men aged 21 and over. (From Baker, 1974, p. 430.) A normal curve has been fitted to these data, with $\overline{X}$ = 1.379 kg, *SD* = 0.151 kg. The data do not differ significantly from the normal distribution (χ^2 = 11.60, *df* = 9, $p < .30$).

separate studies, consist of greater relative frequencies in both the lower and the higher extreme tails of the distribution.

In view of the rather small correlation between brain size and IQ, it is arguable whether the approximately normal distribution of brain weights (and cranial capacities) has any more theoretically important connection with the similarly normal distribution of IQs than have the approximately normal distributions of many other somatic dimensions having no intrinsic relationship to mental ability, such as stature.

Brain size and body size. Because body size is correlated with both brain size (or head size) and IQ, it acts as a confounding factor in the correlation between these two variables. If it is assumed that a part of the variance in total brain size is correlated with body size independently of intelligence and a part is correlated with intelligence independently of body size, then the calculation of a partial correlation between brain size and IQ, with body size partialed out, makes theoretical sense. This is exactly what is done in the case of interspecies comparisons, for which brain size shows a close relationship to assessments of intelligence only when body size is controlled. The same reasoning can be applied within a species, despite certain theoretical complications which are illustrated here in terms of simple diagrams showing the possible ways in which correlation might arise between head size (*H*), brain size (*Br*), body size (*Bo*), and IQ. Two of these possibilities, which may be regarded as rival hypotheses, are shown in Figure 4.13.

Solid lines represent intrinsic or causal correlations (with direction of causation left unspecified); dashed lines represent adventitious or noncausal correlations. Figure 4.13a depicts the null hypothesis, that is, the hypothesis that no intrinsic correlation exists between brain size and IQ; the adventitious correlation shown in this figure is due to the fact that both variables are correlated with body size—brain size being causally correlated with body size and IQ being adventitiously correlated. The one part of this hypothesis for which we have already reviewed the evidence and found it fairly conclusive is the adventitious correlation between IQ and body size. Figure 4.13b represents the counterhypothesis, showing an intrinsic correlation between brain size and IQ. (Intrinsic correlation between brain, body, and head size are assumed to be quite reasonable, and so remain the same in Figures a and b.) The hypothesis represented in Figure 4.13b would be ruled out definitively if the brain × IQ correlation *within* families could not be shown to be statistically significant; such evidence, of course, would preclude the possibility of an intrinsic correlation between brain size and IQ, as seen in the case of the body-size × IQ correlation.

The head-size × IQ correlation is more problematic if head size is viewed as a proxy for brain size. The imperfect correlation between head size and brain size attenuates the correlation, and if the within-families zero-order correlation between head size and IQ is low to begin with, partialing body size out of the head size × IQ correlation could result in such a small partial *r* that only a very large

sample could make the null hypothesis convincing. We examine the evidence on this point later on. But first we should get some idea of the other correlations shown in Figure 4.13, that is, the correlations of body size with head size and brain size, because these are needed for partialing body size out of the IQ × brain-size (or head-size) correlation. The best justification for partialing out body size is the likelihood that some portion of the brain is more highly correlated with body size, and some portion is more highly correlated with intelligence. If this is true, the essential correlation between brain size and IQ should be revealed most clearly when body size is statistically controlled. Evidence shows that this is more than mere likelihood. When the brain is dissected into three portions—cerebral hemispheres, cerebellum, and pons plus medulla—all three portions are found to be positively correlated with body size; but the portion of the brain showing the lowest correlation with body size is the same portion in which the higher mental functions are known to be localized—the cerebral hemispheres (Marshall, 1892).

Table 4.7 summarizes studies prior to 1980 on the correlations of brain and head measurements with body height and weight in adult samples. The first three correlations are undoubtedly inflated as a result of their being computed on a combined sample of males and females; the inclusion of both genders in a sample of this kind has the effect of approximately doubling the correlation. The unit-weighted mean of same-sex correlations between *brain weight* and *body weight* is only +.21. This is lower than the mean correlation of +.31 between

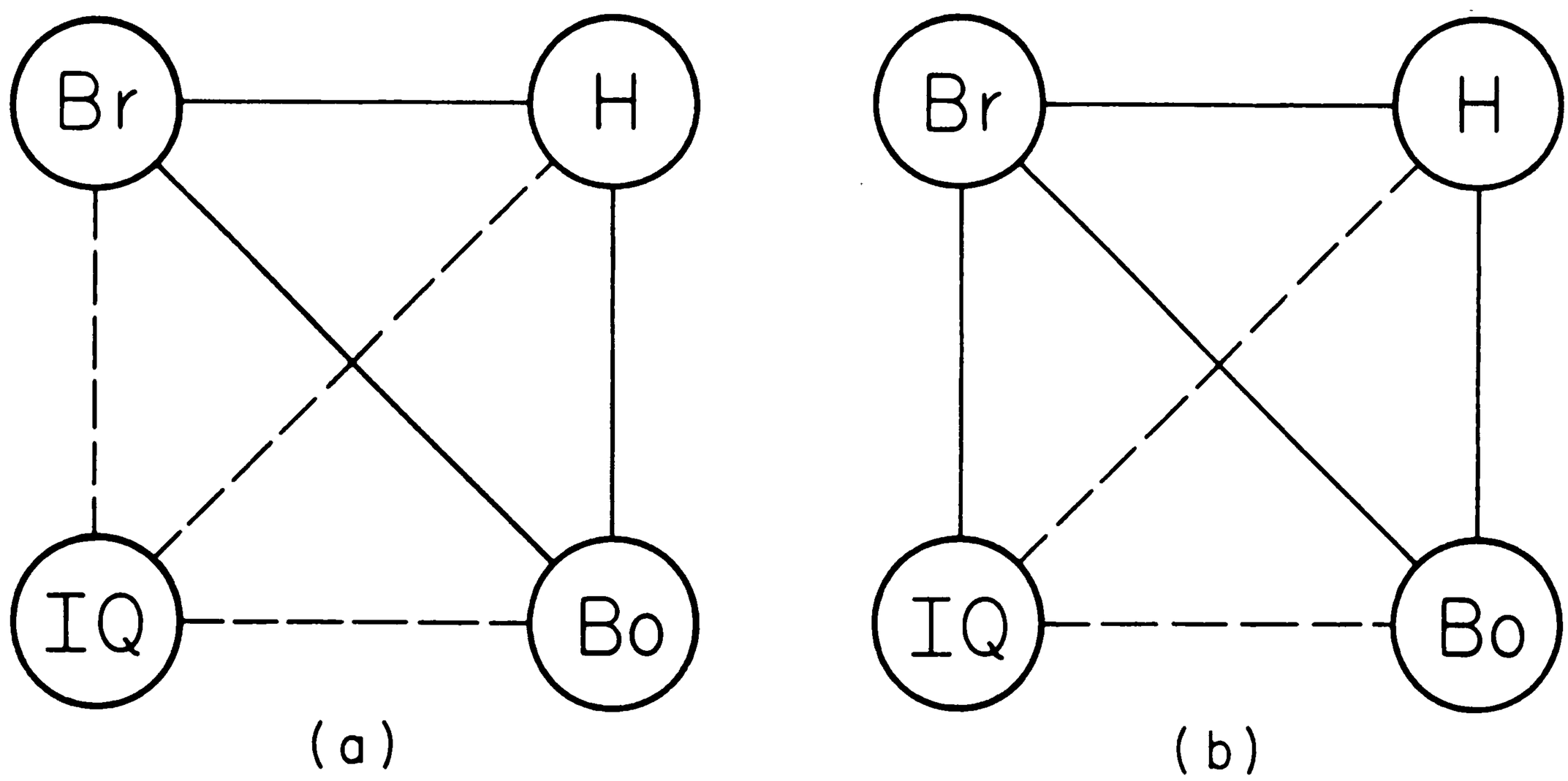


Figure 4.13. Possible correlations between brain size (Br), head size (H), body size (Bo), and intelligence (IQ). Solid lines indicate causal or functional connections, broken lines are correlations without functional relationship. Panel *a* represents a nonfunctional correlation between brain size and IQ; panel *b* represents an intrinsic or functional relation between brain size and IQ.

Table 4.7. Correlations between Head and Brain Measurements and Body Size (Height and Weight) in Various Adult Samples

Source	*Sample*	*Variables Correlated*	*Correlation*[a]
Passingham (1979, p. 258)	212 men, 213 women, English, ages 18–75 years	Cranial capacity estimated from Lee formula height	.62 ± .10
Passingham (1979, p. 258)	212 men, 213 women, English, ages 18–75 years	Cranial capacity estimated from Lee formula height (age partialed)	.623 ± .10
Passingham (1979, p. 258)	212 men, 213 women, English, ages 18–75 years	Cranial capacity estimated from Lee formula body weight	.57 ± .10
Passingham (1979, p. 262)	734 Englishmen	Brain weight height	.31 ± .07
Passingham (1979, p. 262)	305 English women	Brain weight height	.20 ± .11
Passingham (1979, p. 262)	1039 English adults, 18–75 years	Brain weight height	.45 ± .06
Passingham (1979, p. 262)	1039 English adults, 18–75 years	Brain weight height (age partialed)	.406 ± .06
Passingham (1979, p. 262)	290 English adults, 18–45 years	Brain weight height	.39 ± .11
Passingham (1979, p. 262)	290 English adults, 18–45 years	Brain weight height (age partialed)	.386 ± .11
Passingham (1979, p. 262)	198 English men, 18–45 years	Brain weight height	.20 ± .14
Passingham (1979, p. 262)	92 English women, 18–45 years	Brain weight height	.12 ± .21
Passingham (1979, p. 262)	1039 English adults, 18–75 years	Brain weight body weight	.26 ± .06
Passingham (1979, p. 262)	290 English adults, 18–45 years	Brain weight body weight	.36 ± .11
Schreider (1966)	European adults	Brain weight height	.25 ± .30
Whipple (1914, p. 82)	Oxford male students	Head length height	.31
Whipple (1914, p. 82)	Cambridge male students	Head length height	.28
Whipple (1914, p. 82)	3000 criminals	Head length height	.34 ± .03
Whipple (1914, p. 82)	Oxford male students	Head width height	.14
Whipple (1914, p. 82)	Cambridge male students	Head width height	.15
Whipple (1914, p. 82)	3000 criminals	Head width height	.18 ± .03
Susanne (1979)	2071 Belgian men, 17–25 years	Head circumference height	.355 ± .04[b]

[a]Correlation coefficient given with 95% confidence interval when the available information permits its exact computation.
[b]This corrrelation is not given in Susanne's article, but can be calculated from the given zero-order correlations between (a) head circumference and IQ, (b) height and IQ, and (c) the partial correlation between head circumference and IQ with height partialed out.

externally measured *head length* and *body height*. Head width is less correlated (mean $r = .16$) with height than is head length. Whenever possible, given the available information, the factor of age has been partialed out of these correlations. As can be seen, however, this partialing out of age has very little effect on the zero-order correlations.

The most recent, and in many ways the best, data on these relationships are provided in a study by Ho et al. (1980b). Conducted at the Institute of Pathology, Case Western Reserve University, this study offers a distinct advantage, in that all brain weights were obtained under uniform procedures for excising and weighing. Brains with any abnormalities known to affect brain weight were excluded. The subjects in this study varied in age from 25 to 80 years. The obtained correlations between brain weight and body measurements are shown in Table 4.8. It makes sense neurologically that, more than either body height or body weight, body surface area should be highly correlated with brain weight; this is because every square millimeter of the entire body surface is neurologically represented in the sensory cortex of the brain. (Body surface area [in square meters] = body weight [in kilograms]$^{0.425}$ $\times$ height [in centimeters]$^{0.725}$ $\times$ 0.007184.) Theoretically speaking, therefore, body surface area would seem to be the best variable to partial out of correlations between brain (or head) size and IQ. We note that the multiple correlation of height and weight with brain weight is a close approximation to the correlation between body surface area and brain weight.

In young children, head and body measurements are more highly correlated than in adults, even when age is controlled; this is probably because of individual differences in physical growth rates which are reflected in both head and body size. Some excellent data on the correlation of head circumference with body height and weight in white and black children from birth to 4 years are shown in Figure 4.14 (from Broman et al., 1975). The consistent decrease in the correlation between birth and 4 years probably continues slightly beyond 4 years, but the 4-year correlations appear to be closely approaching the asymptotes of the functions relating the magnitude of the correlations to age. We cannot explain

Table 4.8. Correlations[a] between Brain Weight and Body Measurements in White and Black Males and Females[b]

Sample	N	*Body Height*	*Body Weight*	*Body Surface Area*
White Males	414	.20 ± .10	.24 ± .10	.27 ± .10
Black Males	225	.20 ± .13	.15 ± .13	.20 ± .13
White Females	388	.24 ± .10	.23 ± .10	.29 ± .10
Black Females	218	.15 ± .13	.10 ± .13	.14 ± .13
Mean[c]		.204 ± .05	.196 ± .05	.241 ± .05

[a]Correlations given with 95% confidence interval.
[b]From Ho et al., 1980b.
[c]*N*-weighted mean.

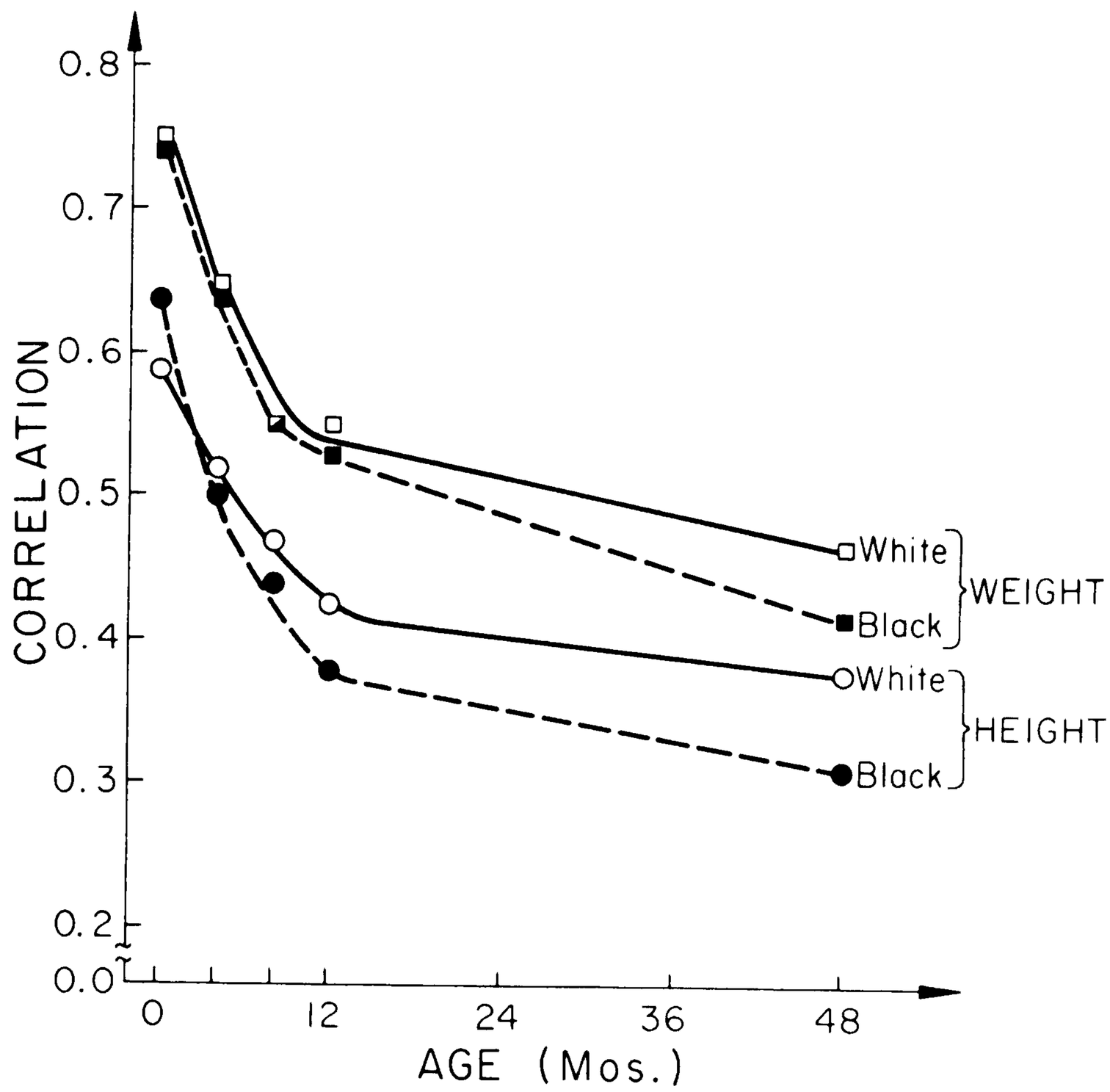


Figure 4.14. Correlation of head circumference with height and weight at various ages in white and black children. (Data from Broman et al., 1975, Table 9.2, p. 126.) The 95% confidence interval for all correlations is $r \pm .02$ except at age 8 months, which is $r \pm .03$.

why the correlations should be significantly, and substantially, lower for blacks than for whites; certainly, these lower values are not attributable to differences in variance in any of the correlated variables, as the variances of whites and blacks are nearly identical on every variable considered (Broman et al., 1975, Table 1, p. 174).

When *mean* cranial capacity (as estimated by Lee's formula, from measurements of the length, width, and height of the head of living persons) is obtained for a number of groups, and is correlated with *the mean* body weight of the groups, thereby eliminating individual variation, the resulting correlation is extremely high. Such data were obtained for 38 categories of military personnel in several different countries (NASA, 1978); the correlation between mean cranial capacity and mean body weight for these 38 groups was found to be +.937.

Head size, brain size, and IQ. Interest in the relationship between brain size and intelligence is at least as old as the history of psychology. Objective statistical evidence on the subject was not possible until after the turn of the century, however, when Binet, in 1905, invented the first demonstrably valid test of intelligence. Until then, the only evidence of an association between brain size and ability in humans had been based on the cranial measurements and brain

weights of deceased famous men of generally acknowledged intellectual distinction, such men as Gauss and Kant, among others. Some of these data are shown in Table 4.9. The mean brain weight of this group is 1,607 g(SD = 282), which is about 1.3 standard deviations above the mean brain weight (1,410 g, SD = 152) of a large random sample of European men. Although most of these specimens were clearly larger than the average volume or weight of male human brains, these data are regarded as hardly more than presumptive evidence of an association between brain size and ability. They leave much to be desired in terms of scientifically worthy evidence. Quite apart from the extremely small sample size, any one of a number of methodological problems—including marked differences in age and cause of death, as well as lack of uniformity in methods of removing, preserving, and weighing the brain specimens themselves—would serve to vitiate these antique curios as reliable scientific evidence. Some idea of the many problems of reliability posed by such data is provided by Baker (1974, pp. 429–432).

The earliest general review of the relation of skull size to intelligence that we have found is that of Whipple (1914, pp. 79–91). But aside from studies by Pearson (1906) and Pearl (1906), which were based on quite large samples, the studies prior to 1914 are hardly worth mentioning. Even the large-scale studies by Pearson and Pearl, based on subjective ratings of intelligence and college grades rather than objective test scores, leave much to be desired. Whipple's conclusions were much like those subsequently expressed by Paterson in a thorough and careful review published in 1930, by which time it was possible to include a few studies that were somewhat superior, methodologically, to those reviewed by Whipple (1914). Paterson concluded that most observed correlations between intelligence and head size fall within the range of .10 to .20, although the N-weighted mean of all the correlations available at that time was

Table 4.9. Brain Weight (in Grams) of Ten Famous Men[a]

Name	*Field*	*Age of Death[b]*	*Brain Weight*
Bismarck, Otto von	Statesman	83	1807
Broca, Paul	Anatomist	56	1424
Byron, George	Poet	36	1807
Cuvier, Georges	Naturalist	63	1820
France, Anatole	Novelist	80	1017
Gauss, Karl F.	Mathematician	78	1492
Kant, Immanuel	Philosopher	80	1631
Schumann, Robert	Composer	46	1413
Thackeray, Wm. M.	Author	52	1658
Turgenev, Ivan, S.	Novelist	65	2000
Whitman, Walt	Poet	73	1282

[a]From various sources (Baker, 1974; Cattell, 1971; Cobb, 1965).
[b]The correlation between age of death and brain weight in this sample is −.238

+ .20. Paterson noted that the reported correlations, though small, were always positive and clearly not due to chance. He also suggested that these reported correlations, because they are often based on samples that are restricted in range of intelligence (groups of university students, for example), may well underestimate the true correlation in the population. It now appears that Paterson may have been a bit carried away by his general mission of debunking popular myths concerning associations between physique and intellect, however. In his summary of conclusions, he writes as follows:

> Although inadequate statistical methods characterize most of the research studies and although no satisfactory standardized method of measuring head size is adopted in them, it can be said with considerable assurance that whatever positive correlation exists must be of a low order. . . . It appears that variation in head size is a function of race, sex, and family stock. It does not vary between individuals in correspondence with intellect. (Paterson, 1930, pp. 122–123)

It was this overwhelmingly negative conclusion which was to be perpetuated in psychology text books for more than half a century. An unwarranted conclusion in 1930, it is today flatly wrong in light of the best available evidence. As Cattell (1971) has remarked,

> Some of this was the sheer hubris of the specialist out to debunk any popular idea. Students were taught that there is no correlation of head size with intelligence; that some of the largest heads are those of hydrocephalic imbeciles (the head being enlarged by the disease process); and that men of genius have been known to have subaverage brain weight, e.g., the case of Anatole France. (He died at eighty with a somewhat subaverage brain weight [1,017 g, which is about 2.6σ below the average of European males], conceivably due to the usual shrinkage of weight which occurs with age.) (p. 178)

It is certainly true, and has long been acknowledged by all serious students of the subject, that, except at the pathological extremes of microcephaly and hydrocephaly, head size (or brain size) is practically useless as an indicator of intellect in individuals. As Cobb (1965) has noted, an extremely wide range of brain size, extending almost $\pm$ 3σ from the mean, is compatible with normal mental functioning. Brains as small as 800-900 grams (as compared with the average of 1,300-1,400) have shown normal intellect, and even nanocephalic dwarfs with brains of less than 700 g can converse fluently (Cobb, 1965, p. 558). Obviously, other features of the brain besides sheer size must account for all but a very small part of the variance in biological intelligence in humans.

We have found only one older study (Miller, 1926) which was not picked up in Paterson's (1930) comprehensive review. This study was based on the head measurements (and estimated cranial capacity) of 176 adult male prisoners, variously identified as mentally retarded, borderline, or normal, and more than

4,000 Tasmanian school boys. Significant differences in mean cranial capacity were found between the three mental levels and between age-matched pupils in academically select and nonselect schools. Unfortunately, no control for body size was provided, no tests of intelligence were used, and no correlation coefficients were reported. While these results do contribute additional evidence of some positive association between head size (or estimated cranial capacity) and a roughly assessed level of mental ability, therefore, they are not very informative for our purpose. Questions of intelligence aside, however, this study presents what is probably the most solid and precise data we have seen on the growth of estimated cranial capacity over the age-range from 7 to 17 years; these data are summarized in Figure 4.15.

Since Paterson's (1930) review, several valuable studies have appeared. Van Valen (1974) has reviewed virtually all the published studies prior to 1974. Van Valen reasons that the obtained partial correlation between head size and IQ, with body size partialed out, is attenuated by two factors—the imperfect measurement of intelligence and the far-from-perfect correlation between external head measurements and actual brain size as measured by volume or weight. In addition, Van Valen makes the reasonable assumption that cranial capacity (c) as estimated from external head measurements (controlled for body size) is corre-

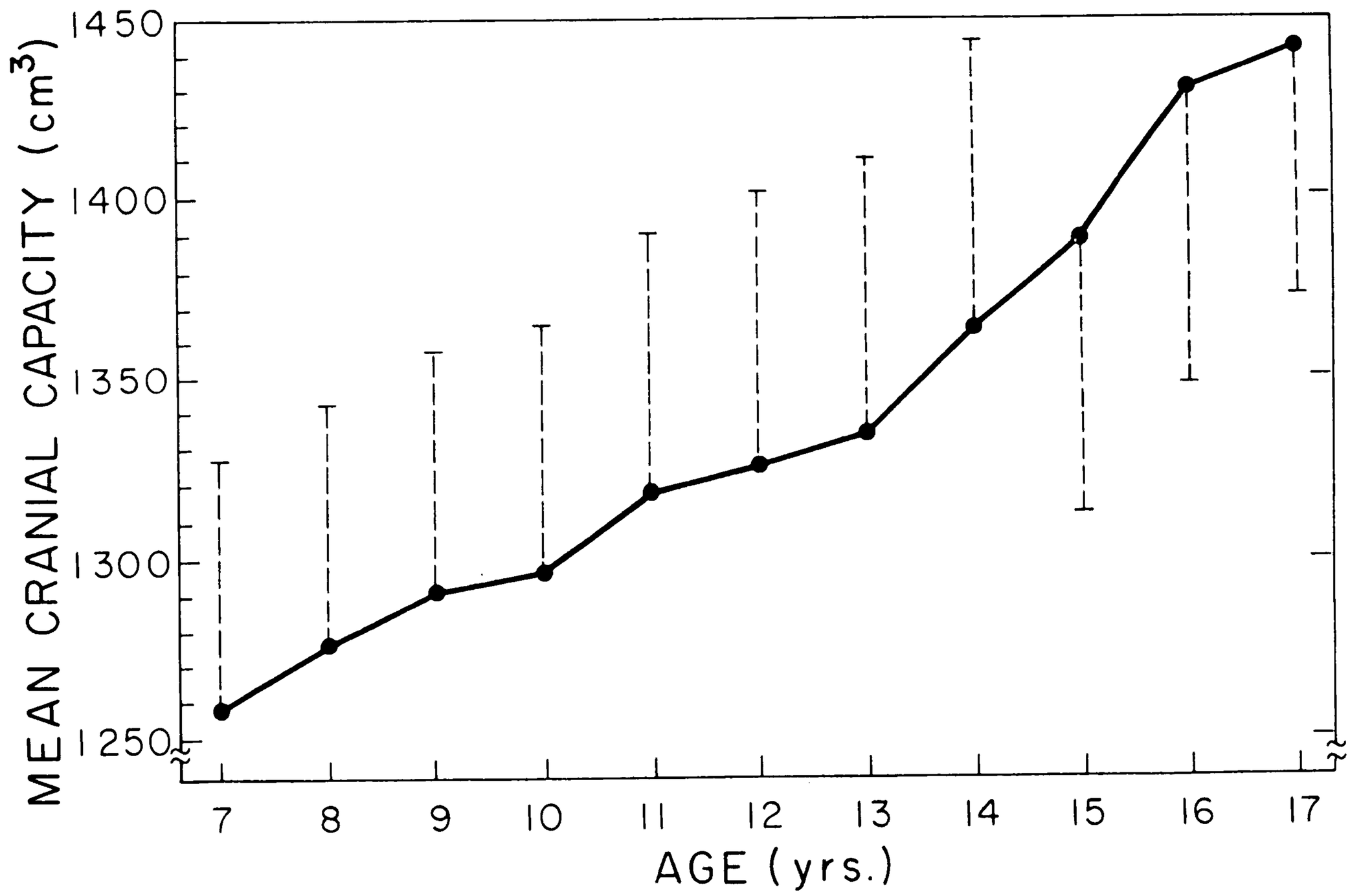


Figure 4.15. Mean estimated (by Lee's formula) cranial capacity in cm^3 of 4012 Tasmanian (Caucasian) school boys from ages 7 to 17 years. The vertical dashed lines indicate ± 1 standard deviation. These are not longitudinal data; each age is a different group, with average N = 565. (Drafted from data presented in Miller, 1926, Table 7, p. 30.)

lated with IQ (i) solely by way of brain size (b). Under this assumption, $\rho_{ic} = \rho_{ib}\rho_{bc}$, and so $\rho_{ib} = \rho_{ic}/\rho_{bc}$. Using the best available estimates of ρ_{ic} and ρ_{bc} and correcting for the attenuation of the intelligence measures, Van Valen concludes that the best estimate we now have of the correlation between brain size and intelligence in humans is about + .3. He sees this correlation as related to the increase in human brain size, noting that brain size "has increased dramatically and at an unusually high rate in human evolution" (p. 420). Van Valen also argues that there is a direct effect of brain size on intelligence and that "natural selection on intelligence at a current estimated intensity suffices to explain the rapid rate of increase of brain size in human evolution" (p. 417).

The most important studies cited in Val Valen's (1974) review, as well as those which have appeared since then, are summarized in Table 4.10. A problem with some of these correlations is that stature is not controlled. In Table 4.10, the N-weighted means are given for the head-size × intelligence correlation without stature being controlled and with stature controlled (in parentheses). Another problem with many of the correlations is that they are based on samples selected for superior intelligence, and hence, tend to underestimate the true correlation in the general population. For this reason, the large study by Susanne (1979) is probably most important. Susanne's subjects were a random sample of 2,071 drawn from a population of 43,452 young men eligible for military service in 1963 in Belgium. According to Susanne, the age and socioeconomic and geographic distribution of the sample was representative of the Belgian male population in general and was free of serious pathology. Subjects' ages ranged from 17 to 25 years, with a mean of 19.53 years. An overall intelligence measure, derived by averaging standardized scores obtained on five psychometric tests, afforded a highly reliable measure of general mental ability. Susanne's study employed 11 different measurements of the head and face. Only the correlation of the composite intelligence test score with cranial perimeter (head circumference) is shown in Table 4.10; before and after partialing out stature, the correlations are + .242 and + .194, respectively.

The appreciably lower correlations found in the recent studies by Swan and Miszkiewicz (no date) and by Passingham (1979) may be to some extent attributable to the fact that these studies sampled both males and females, a practice which tends to attenuate correlations of this kind, as we have seen. Such attenuation is probably overcome to some unknown degree by partialing out stature. As a general rule, however, correlations involving any body measurements should be calculated separately for males and females.

Following Van Valen, we can estimate the correlation between brain size and intelligence from the N-weighted means of the correlations between height (h), cranial size (c), brain size (b), and "IQ" (i). We assume virtually perfect reliability of the physical measurements, so there is no correction for attenuation. "IQ" is assumed to have a reliability of .90, which is typical, and correlations involving IQ are accordingly corrected for attenuation. Using the N-weighted

Table 4.10. Correlations between Head Size and Intelligence Reported in Various Studies

Source	*Subjects*	N	*Measure*	*Correlation*[a]	*Controlled Variables*
Pearson (1906)	Random	4486	Subjective ratings	.11 ± .03	Age, sex
Pearson (1906)	University students	1011	Grades	.11 ± .06	Age, sex
Pearl (1906)	Soldiers	935	Subjective ratings	.14 ± .06	Age, sex
Murdock & Sullivan (1923)	Random	595	IQ tests	.22 (.19)[b] ± .08	Age, sex
Reed & Mulligan (1923)	University students	449	Grades	.08 ± .09	Age, sex, body size
Sommerville (1924)	University students	105	IQ tests	.10 ± .19	Age, sex
Porteus (1937)	School children	200	IQ tests	.20 ± .14	
Schreider (1968)	Random?	80	IQ tests	.08 ± .22	Age, sex
Schreider (1968)	Random?	71	IQ tests	.12 ± .23	Age, sex
Robinow (1968)	Children, ages 3–13 years[c]	300	IQ tests	.18 (.09)[b] ± .11	
Swan, Haskins, & Douglas[d]	School children	547	IQ tests	.11 ± .08	Age
Swan & Miszkiewicz[e]	School children (grades K–12)	843	IQ tests (PMA total)	.075 to .084 (.05)[b] ± .07	Age
Passingham (1979)	English adults (18–75 years)	415	Wechsler Total IQ	.13 (.03)[b] ± .10	Age
Susanne (1979)	Random Belgian men, ages 17–25	2071	"IQ" (mean of 5 tests)	.242 (.194)[b] ± .04	Sex, height
N-weighted Mean				.142 ± .065 (.135 ± .06)[f]	

[a]Correlation with 95% confidence interval.
[b]Correlation in parentheses is with stature partialed out.
[c]These are longitudinal data from the Fels Research Institute. Stanford-Binet IQs at ages 3, 6, 9 years, Wechsler at 13 years. The unit-weighted mean *r* is given; its *SD* across ages is .078.
[d]Reported in Cattell (1982, Table 2.10, p. 38).
[e]Unpublished paper, not dated.
[f]In parentheses, the *N*-weighted mean of only those correlations for which body size (stature) is partialed out (total $N = 4{,}673$). The 95% confidence interval for the weighted means is calculated by the formula shown in footnote b of Table 4.2.

mean correlations (corrected for attenuation when involving IQ) as the best available empirical estimates of the true correlations, we can estimate the correlation between brain size (b) and IQ (i), as shown in Figure 4.16. The formula for theoretically estimating the correlation between brain size and IQ should be used only with the partial correlations, that is, only when height has been partialed out of all the correlations before they are entered into the estimation formula; thus

$$r_{bi \cdot h} = r_{ci \cdot h}/r_{cb \cdot h} = .135/.445 = .303,$$

which is our best estimate of the correlation between intelligence and brain size, as independent of stature, or body size. If we use only the disattenuated partial correlation ($r_{ic \cdot h} = .205$) from Susanne's (1979) study as the basis for estimation, we get $r_{ib \cdot h} = .205/.445 = .460$ as the estimated correlation between intelligence and brain size. The weak link in the estimation procedure is the empirical correlation between external cranial size and brain size. The mean r_{bc} used here is based on only three samples, but this is the best we can do with the available evidence. If we use the largest value of r_{bc} that we can find in the

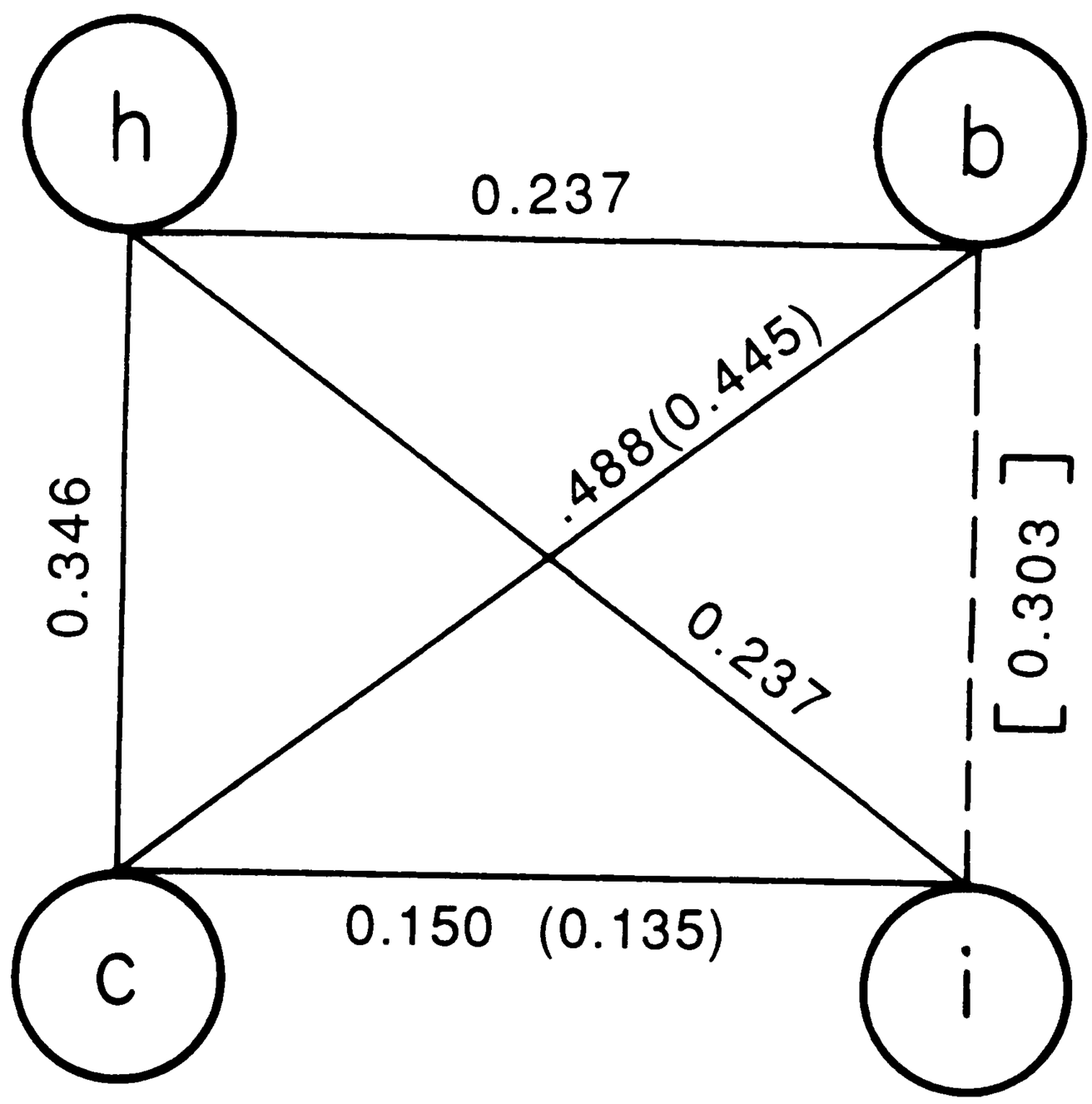


Figure 4.16 ***N*-weighted mean correlations (IQ correlations are corrected for attenuation) between height (*h*), external cranial size (*c*), brain size (*b*), and IQ (*i*). Solid lines indicate empirical correlations; the dashed line is a theoretically estimated correlation. Partial correlations, with *h* partialed out, are shown in parentheses. The correlation $r_{bi \cdot h} = .303$ is theoretically derived, following Van Valen (1974), from the formula**

$$r_{bi \cdot h} = r_{ci \cdot h}/r_{cb \cdot h} = .135/.445 = .303.$$

literature, it is .57 (Van Valen, 1974, p. 423), which, with height partialed out, becomes $r_{bc \cdot h}$ = .535. Using the value in the estimation, along with Susanne's disattenuated partial correlation $r_{ic \cdot h}$ = .205, we get $r_{ib \cdot h}$ = .205/.535 = .383 as the correlation between brain size and intelligence, independently of stature. In any case, if Susanne's study is considered to yield the best determination of $r_{cb \cdot h}$ that we now have, it seems safe to say that the correlation of brain size with intelligence is not less than .2 and may be considerably higher, depending on the size of $r_{bc \cdot h}$, which is not yet very firmly established by the present evidence.

The first (and, as yet, the only) *in vivo* study of the brain-size × intelligence relationship based on the direct measurement of brain size (rather than estimation from external head measurements) was made possible by the technique of magnetic resonance imaging (MRI), which permits, in effect, a 3-dimensional picture of a person's brain, from which precise measurements can be obtained by computer. This method completely overcomes the attenuation of the brain × IQ correlation that results from estimating brain size from external indices which are imperfectly correlated with actual brain size because of individual variation in thickness of the skull and tissues surrounding the brain. Using MRI, Willerman, Schultz, Rutledge, and Bigler (1989) compared two groups of non-Hispanic white college students whose total SAT scores were either high (≥ 1350) or low (≤ 940) and whose Wechsler IQs were ≥ 130 or ≤ 103. In the total group (N = 40), brain size, adjusted for body height and weight, was correlated r = .51 ($p <$ .01) with IQ. Corrected for the increased IQ variance because of selection of extreme groups, the correlation drops to r = .35, which may be regarded as the "best estimate" of the brain-size × IQ correlation in the general population of healthy people in this age group. This correlation is slightly, but not significantly, higher than Van Valen's (1974) "best estimate" of r = .30, based on external measurements of the head. Further studies using MRI should soon yield definitive answers regarding the relationship of IQ to brain size, but more importantly, MRI will permit study of the correlation of specific brain structures with psychometric abilities.

IQ and head circumference in young children. In a longitudinal study, Broman et al. (1975) reported correlations between Stanford-Binet IQ obtained at age 4 years and head circumference measured at birth, 4 months, 8 months, 1 year, and 4 years of age, in very large samples of whites ($N \simeq$ 10,000) and blacks ($N \simeq$ 12,000). The zero-order correlations are shown in Table 4.11, along with the partial correlations controlling for height and weight measured at each age. Unfortunately, the correlations (r_{CH}, r_{CW}, r_{HW}, given in Table 9.2 of Broman et al. 1975) required for computing the partials were not reported separately for males and females, and so in our Table 4.11 we have had to present only correlations based on the combined sexes. This pooling of male and female data very clearly attenuates the partial correlations, as can be seen from the within-sex correlations (r_{IC}) of IQ and head circumference (last 2 columns of Table 4.11) as compared with the substantially lower zero-order correlations

Table 4.11. Zero-Order Correlations[a] (Corrected for Attenuation[b]) and First-Order and Second-Order Partial Correlations [Height (H) and Weight (W)] between Stanford-Binet IQ (I) at Age 4 Years and Head Circumference (C) at Various Ages

	White (N ≃ 10,000)					*Black (N ≃ 12,000)*					*Mean Within-Sex* r^b_{IC}	
Age	r_{IC}	$r_{IC \cdot H}$	$r_{IC \cdot W}$	$r_{IC \cdot HW}$	*95% Conf. Int.*[d]	r_{IC}	$r_{IC \cdot H}$	$r_{IC \cdot W}$	$r_{IC \cdot HW}$	*95% Conf. Int.*[d]	*White*	*Black*
Birth	.088	.061	.046	.044	±.018	.110	.043	.031	.011	±.017	.120	.121
4 months	.121	.088	.065	.060	±.019	.110	.045	.006	.001	±.017	.181	.154
8 months	.121	.085	.080	.057	±.030	.132	.067	.064	.053	±.027	.186	.175
1 year	.132	.094	.086	.081	±.019	.110	.065	.041	.036	±.017	.197	.154
4 years	.165	.134	.123	.120	±.020	.132	.093	.070	.069	±.018	.213	.143
Mean[c]	.124	.092	.078	.073		.117	.061	.039	.030		.177	.160

[a]From Broman et al. (1975), Table 1, Appendix 4, and Table 9.2 (p. 126).
[b]Correlation for attenuation based on Stanford-Binet test-retest reliability of 0.83 (see Broman et al., p. 37); reliabilities of physical measures are assumed to be 1.00.
[c]*N*-weighted mean.
[d]All four corrrelations at each age have the same confidence interval.

(r_{IC}) for the combined sexes. Because the correlations used for partialing out height or weight may be spuriously inflated by any sex difference in these variables, the partial correlation between IQ and head circumference could be severely affected by amalgamation of the sexes. Hence, the partial correlations in Table 4.11 are best regarded as lower-bound estimates of the true correlation between IQ and head circumference.

The important conclusion that can be safely drawn from Table 4.11 is that all of the partial correlations are positive and (except for blacks at birth and 4 months) all are statistically significant (at 4 years, $p < .001$ in each racial group). It should also be noted that the correlations at ages 1 year and 4 years are in very close agreement with those of Robinow (1968, see our Table 4.10), who found a correlation (with age and sex controlled) of .18 (.09 with height partialed out) between IQ and head circumference in 300 children between ages 3 and 13. A graph of IQ plotted as a function of head circumference shows some nonlinearity of the relationship, which is therefore somewhat underestimated by the Pearson r. The plot for age 4 years, shown in Figure 4.17, is quite typical. The correlation between head size and IQ appears to increase with age, although there are not adequate data to establish this as a fact. In a representative sample (N = 2023) of North-Central United States children (94% Caucasian), Stanford-Binet IQ measured at age 7 years showed zero-order correlations with head circumference at birth, 1 year, 4 years, and 7 years of .08, .17, .22, and .23, respectively; all are significant beyond the .001 level of confidence (Fisch, Blick, Horrobin, & Chang, 1976).

Within-family correlation between head size and IQ. The fact that correlations between head size and IQ remain positive and significant when body size (height and weight) is controlled is proof that the head × IQ correlation is not entirely attributable to the correlation of both variables with a common factor—body size. But this evidence is not itself proof of an intrinsic, that is, functional or causal, connection between brain size and IQ. It is possible that the correlation between IQ and brain size (with body size controlled) might be only a nonintrinsic, or between-families, correlation, assuming there were common selection, or cross-assortative mating, for IQ and that part of the variance in brain size which is independent of body size. Although this theoretical possibility intuitively seems unlikely, it can be rigorously ruled out only by the finding of a *within*-family correlation between IQ and brain size (or head size as an attenuated proxy for brain size).

We have searched the literature for evidence of a within-family correlation between head size and IQ and have found only one study (Clark, Vandenburg, & Proctor, 1961). Unfortunately, the sample in this study is too small to provide a statistically adequate test of the obtained correlations. The evidence is based on 37 pairs of like-sex dizygotic twins; dizygotic twins are genetically equivalent to full siblings, but provide the added methodological advantage of being exactly the same age, thus obviating the need for age-standardization of measurements.

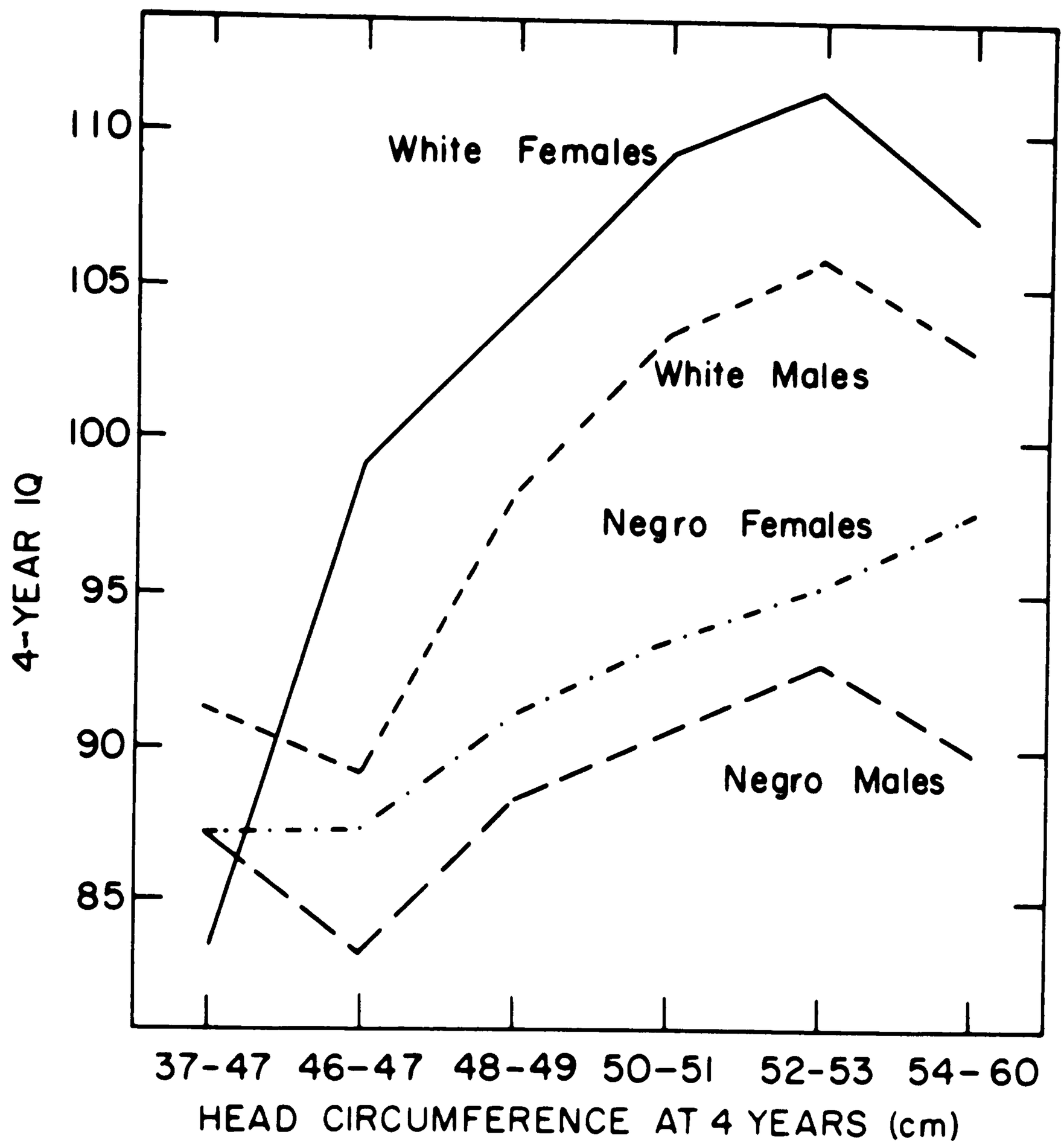


Figure 4.17. Four-year Stanford-Binet IQ plotted as a function of head circumference. Sample sizes: white males = 5046, white females = 4684; black males = 5964, black females = 6065. (From Broman et al., 1975, p. 186.)

The subjects were junior and senior high school students in Michigan. The within-family (i.e., within-pair) correlation (computed separately for males and females and averaged) between head circumference and IQ (based on Raven's Progressive Matrices, the Chicago Test of Reasoning Ability, and the Chicago Test of Verbal Ability) is + .03 ± .33 (95% confidence interval); with height and weight partialed out, the correlation becomes + .10. (The within-family correlation between IQ and the cross-sectional area of the head is − .09 ± .33, and remains the same with height and weight partialed out.) As these correlations are based on within-pair difference scores, they are more seriously attenuated than ordinary correlations. None of these within-family correlations is significant. Because of the small sample on which these correlations are based, however, we must conclude that it remains an open question whether a correlation between head size and intelligence (with body size controlled) exists within families.

In hopes of getting a better resolution to this question, Jensen (1987) obtained the between-family (BF) and within-family (WF) correlations between *g* factor scores (derived from a large battery of psychometric tests of mental abilities) and head size in MZ and DZ twins. All of these measurements are tabled in the appendix of a book by Osborne (1980). The variables of sex, age, and race were regressed out of both the test scores and head measurements. Multiple correlations (R) were calculated between the three head-size measurements (length, width, and circumference) as the independent variables and *g* as the dependent variable. The BF and WF multiple correlations for the 82 pairs of MZ twins and the 61 pairs of DZ twins are:

$$\text{MZ BF } R = +.39,\ p < .01$$
$$\text{MZ WF } R = +.17,\ p > .05$$
$$\text{DZ BF } R = +.15,\ p > .05$$
$$\text{DZ WF } R = +.28,\ p > .05$$

The within-individual (i.e., all twins treated as singletons, with total $N = 286$) multiple $R = +.30, p < .01$. The MZ data strongly suggest that the $g \times$ head-size correlation is largely genetic and that environmental factors contribute little or nothing to the correlation, probably because they have no effect on head size, at least in a population that has not suffered malnutrition. The DZ WF correlation of + . 28, which falls just short of significance at the .05 level, suggests an intrinsic correlation between head size and *g*. Since a larger correlation would hardly be expected, in view of the $R = +.30$ ($p < .01$) for the entire sample of MZ and DZ twins treated as singletons, a much larger sample of DZ twins (or full siblings) will obviously be needed for a rigorous test of the WF correlation between head size and *g* (or IQ). It is a crucial test, however, because without it we cannot really be certain that there is a truly intrinsic, or functional, relationship between head size (or brain size) and mental ability.

Social class differences in brain size. As there is a correlation between socioeconomic status (SES) and IQ (Eysenck, 1979, Chap. 7), we might expect a corresponding correlation between head or brain size and SES. SES is usually indexed by occupational status, and many years ago sociologists and anthropologists reported average differences in head size and other body measurements between men in various occupations, with those in the higher-status occupations, in general, averaging larger values on the physical measures (e.g., Hooton, 1939; Sorokin, 1927). We now have somewhat better data, based on much larger samples and more satisfactory measurements.

An important distinction in this context is between a person's SES of origin, which is the SES attained by the person's parents, and attained SES, which is the level of SES attained by the person in adulthood. Correlations of IQ, head size, and other variables with SES of origin are always considerably smaller than with attained SES. For example, the average correlation between IQ and SES of

origin is between .30 and .40, whereas the correlation between IQ and attained SES, or occupational level, is between .50 and .70 in various studies (Jensen, 1980a, Chap. 8).

The largest set of data (approximately 10,000 white and 12,000 black children) showing a correlation between head circumference and SES of origin, as indexed by a composite of family income and the amount of education and occupational status (10 categories) of the head of household, is reported by Broman et al. (1975). These data are summarized in Table 4.12. Small but significant correlations are seen in both racial groups between SES and head circumference, even when height is held constant. Control for height also controls for the possible influence of nutritional factors, both prenatal and postnatal, associated with SES. However, head growth is less vulnerable to nutritional deprivation than total body growth (Robinow, 1968).

A relationship between directly measured brain weight and occupational level is reported by Passingham (1979). Persons (734 men and 305 women) were classified according to occupation, or, in the case of women, according to husband's occupation, into one of three categories, which we have labeled *high* (professional), *middle* (skilled workers and tradesmen), and *low* (semiskilled and unskilled workers). The occupational groups for the total sample were matched on age, and there were no significant differences between the three groups in cause of death or in ratings of edema of the brain. The three groups were compared (separately within each sex) by analysis of variance on brain weight and on a "brain index," which is the antilog of the deviation of log brain weight from the linear regression line of the regression of log brain weight (g) on log height (cm). This index of brain size is independent of body height, showing a correlation of only $-.01$ with height and .03 with body weight in both men and women, who do not differ significantly on the brain index ($t = 0.26$, $df = 1{,}034$, $p = .795$). Thus this brain index fulfills essentially the same purpose as the encephalization index of Jerison (1973).

An analysis of variance shows that, for men, the three occupational categories differ significantly ($F = 6.97$, $df = 663$, $p < .01$) in mean brain weight and in mean brain index ($F = 3.904$, $df = 663$, $p < .05$). For women, none of the

Table 4.12. Zero-Order and Partial Correlations (Height Controlled) between Head Circumference and Socioeconomic Status of Origin[a]

Age[b]	*White (N ≃ 10,000)*	*Black (N ≃ 12,000)*
8 months	+0.11 (.08)[c] ± .03[c]	+0.11 (.04)[c] ± .03[d]
1 year	+0.14 (.11) ± .02	+0.08 (.05) ± .02
4 years	+0.16 (.13) ± .02	+0.11 (.05) ± .02

[a]Data from Broman et al. (1975).
[b]These are longitudinal data, the same children being measured at each age.
[c]Height-partialed correlations in parentheses.
[d]95% confidence interval for both the zero-order and partial correlations.

mean differences between categories was significant for either brain weight or brain index, although with one exception they were in the expected direction. It should be noted that the women were classified by husband's occupation. The low correlation between husband's occupational level and wife's intelligence, which is probably not higher than about + .3, would greatly attenuate the mean differences between the categories in brain size for women. In other words, husband's occupational level is a rather poor proxy for the wife's own level of intelligence, and therefore the nonsignificance of the category differences in mean brain size for women should not be surprising.

As we have been expressing relationships in terms of correlation coefficients throughout this review, we have used the means and standard deviations given by Passingham (1979, Table VII, p. 264) to compute the point-biserial correlation (r_{pb}) between dichotomized occupational levels and brain size (brain index, which holds height constant, and brain weight). We have also calculated the t test of significance of each r_{pb} and have determined the exact one-tailed p values corresponding to each t. As can be seen in Table 4.13, for men, the High vs. Middle and High vs. Low occupational categories are very significantly differentiated with respect to both brain weight and brain index. Assuming that the correlation between occupational category and brain size is mediated solely by the correlation of each variable with intelligence, and assuming a correlation of .50 between intelligence and men's occupational category, the significant correlations for the brain index in Table 13 are consistent with an inferred correlation of about .24 to .28 between intelligence and brain size (independent of body size).

Race differences in brain size. Throughout the history of investigation of this subject, the prevailing notions at any given time and place seem to have shifted about more as a result of social and political attitudes than as a result of the actual scientific evidence. It is interesting, for example, that all editions of the *Encyclopædia Britannica* subsequent to the 18th edition (1964) have omitted any reference to one racially distinguishing characteristic of black people of African origin claimed in earlier editions, namely, "a small brain in relation to their size." Yet a preponderance of the evidence has been consistent with the position taken in earlier editions, and no contradictory evidence had been brought to bear in the meantime. The prevailing sociopolitical zeitgeist of the 1960s, however, abjured all evidence that could possibly be construed as suggesting a biological, rather than exclusively cultural, basis for the well established black deficit in psychometric intelligence. One frequently cited article by Tobias (1970), a South African anatomist and anthropologist, offered some reassurance for this position by arguing that the quality of the evidence, as of 1970, did not permit clear-cut rejection of the null hypothesis with respect to black-white differences in brain size, provided differences in body size and other possible artifacts were taken into consideration. Based entirely on studies of postmortem brains per se, Tobias's critique ignores all of the much more

Table 4.13. Point-Biserial Correlation (r_{pb}) between Dichotomized Occupational Categories (High, Middle, Low[a]) and Brain Size, with t Tests and Exact (One-Tailed) Probability (p)[b]

Contrast	Men						Women					
	Brain Weight			Brain Index			Brain Weight			Brain Index		
	r_{pb}	t	p	r_{pb}	t	p	r_{pb}	t	p	r_{pb}	t	p
High-Middle	.144	2.81	.002	.118	2.49	.006	.089	1.10	.135	.112	1.10	.135
High-Low	.188	2.93	.002	.140	2.88	.002	.025	0.27	.393	.015	0.17	.432
Middle-Low	.054	1.27	.101	.023	0.53	.298	−.064	−0.87	.807	−.098	−1.29	.901

[a]Sample sizes: For men: High = 98, Middle = 291, Low = 278; for women: High = 49, Middle = 97, Low = 72.
[b]Correlations and *t* tests calculated from data in Passingham (1979, Table VII, p. 264).

plentiful evidence of cranial measurements and focuses exclusively on the shortcomings and ambiguities of a number of studies of the autopsied brains of blacks and whites. Without question, there are numerous potentially biasing factors (Tobias lists 14 in all) when comparisons are made between brain weights (or volumes) obtained from different studies, for example, the level at which the brain is severed from the spinal cord, the presence or absence of covering membranes, the type of chemical preservative used, the length of time between removal and weighing, the temperature during preservation, as well as age and cause of death. Clearly, such a large number of uncontrolled factors would likely increase random errors of measurement, or unreliability, and hence obscure or attenuate the statistical significance of any true differences between racial groups. What seems most unlikely, however, is that such error factors should consistently produce a *racial* bias in brain weights. Tobias's argument is severely weakened by the fact that the brain weight (or volume) differences between the races are in close agreement with well established measures of cranial volume directly obtained from skulls, which are not subject to the same measurement errors listed by Tobias in the case of autopsied brains. Internally measured cranial capacity sets the upper limit of brain size, which is very highly correlated with cranial capacity, as brain growth creates tension on the cranial suture lines, causing bone deposition and growth of the skull.

In any case, virtually all of the methodological deficiencies cited by Tobias (1970) have been avoided in a recent large-scale comparative study (Ho, Roessmann, Straumfjord, & Monroe, 1980a) of brain size in blacks and whites. In this study, weights of fresh brains, excised under uniform conditions, were obtained over a five-year period at the Institute of Pathology at Case Western Reserve University in Cleveland, Ohio. Brains showing any pathology (lesions, tumors, hemorrhage, infarct, or edema) were excluded from study. The study sample consisted of 1,261 autopsy cases in a general hospital (222 black females, 228 black males, 395 white females, 416 white males). Ages ranged from 2 to 80 years, with a mean age of 60 years for both blacks and whites. The overall results are shown in Table 4.14. The average brain weights for blacks of both sexes is between 7 and 8 percent lower than the average for whites; expressed in standard deviation units, this is about 0.8σ. The differences are significant; for men, $t = 9.51$, $df = 642$, $p < .001$; for women, $t = 6.23$, $df = 615$, $p < .001$. The sex difference within each race is somewhat larger than the race difference between blacks and whites; note, however, that sex difference is more strongly a function of differences in body size between the sexes, whereas the racial groups show comparatively little difference in body size, as measured by height, weight, and total body surface area. The black women in this study are slightly larger, in fact, than the white women. As can be seen in Figure 4.18, the race difference in brain size is not significantly present at birth and becomes increasingly evident in early childhood, being well established by six years of age, after which the difference remains about the same throughout life.

Table 4.14. Mean and Standard Deviation of Brain Weight (g) of White and Black Men and Women[a]

	Men		*Women*					
Group	*Mean*	SD	*Mean*	SD	*Diff.*	*% Diff.*[b]	*Diff./SD*[c]	r^e_{pbs}
White	1,392	130	1,252	125	140	10.1	1.10	0.49
Black	1,286	138	1,158	119	128	10.0	0.99	0.44
Difference	106		94					
% Diff.[b]	7.6		7.5					
Diff./*SD*[c]	0.80		0.77					
r_{pb}[d]	0.36		0.35					

[a]Data from Ho et al. (1980a), Table 1. (*N*s = 416 white males, 228 black males; 395 white females, 222 black females.)
[b]% Diff. = 100 (larger mean − smaller mean)/larger mean.
[c]The difference expressed in units of the average standard deviation ($\overline{SD}$) of the two groups.
$\overline{SD} = [(N_1 s_1^2 + N_2 s_2^2)/(N_1 + N_2)]^{1/2}$, where N is sample size, s is standard deviation, and subscripts 1 and 2 refer to each group.
[d]Point-biserial correlation between race and brain weight.
[e]Point-biserial correlation between sex and brain weight.

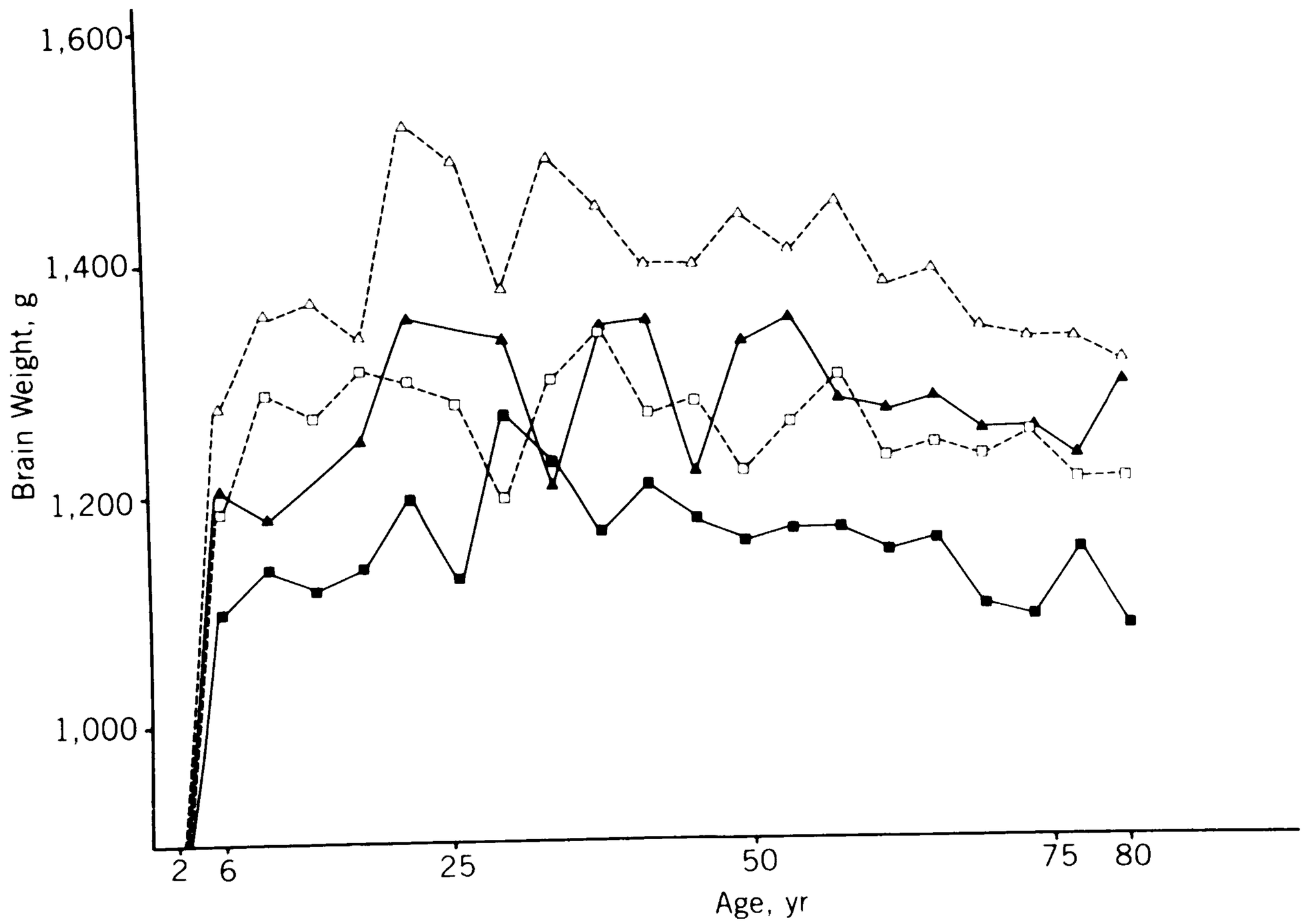


Figure 4.18. Mean brain weight of white males (open triangle), black males (solid triangle), white females (open square), black females (solid square). Brain weight is plotted at midpoint of each 4-year age interval, for example, point at 6 years represents average brain weight of all cases between 4 and 8 years. (From Ho et al., 1980a.)

In another publication based on the same data, Ho et al. (1980b) explicitly examined the racial difference in brain weight in relation to body size (height, weight, body surface area). When body size is held constant, the overall racial difference in mean brain weight is reduced to 0.48σ, as compared with the unadjusted difference of 0.79σ. The adjusted difference remains highly significant ($p < .001$), however. Differences in brain weight between the sexes, when adjusted for body size, are much smaller and less clearcut. White women's brains are heavier than white men's, and the reverse is true for blacks. In summarizing their findings on racial differences, Ho et al. (1980b) offer the following general conclusions:

> Our first report (Ho et al., 1980a) indicated that the brain weight is higher for white men than for black men, and higher for white women than black women. The result is the same when the brain weight is adjusted to the body height, weight, and surface area. The absolute brain weight of black men is heavier than that of white women, but is heavier for white women than for black men when adjusted for height, weight, or body surface area. All the differences in brain weight between the white and black populations are statistically significant when adjusted for any of the body dimensions, except for that between white and black men, when adjusted for body weight. (p. 645)

A more recent article by Ho, Roessman, Hause, and Monroe (1981) analyzes the brain weights of white and black (total $N = 782$) newborn infants, aged 1 to 29 days, in relation to gestational age, body weight, and sex. No significant sex or race differences were found for full-term infants, although among premature infants, whites showed significantly heavier brain weight. Because a significant race difference in brain weight was manifested only in premature infants, Ho et al. suggest that the difference between the races found in their previous study of adult brain weights is best explained by environmental factors implicated in prematurity and low birthweight, both of which have a higher incidence in the black than in the white population. We take exception to this interpretation, for several reasons. First, many types of individual differences in physical (and mental) characteristics that emerge during the course of development are not present at birth. Many important genetic influences have the quality of time capsules, with effects that are not manifested until later stages of the individual's development. For example, the absence of a sex difference in brain weights in infancy and the later appearance of a sex difference is explainable as a difference in developmental growth rates approaching different asymptotes at maturity by the two sexes, and it requires no extraneous causal factors for explanation. Certainly, there is no compelling reason to believe that those differences which are fully manifested at maturity must necessarily be incipiently manifested at birth.

Second, the difference between whites and blacks in prematurity rates is far from being large enough to account for the brain-size difference at maturity, even if it is assumed that prematurity and low birth weight retard brain development much more severely in blacks than in whites. The National Center for Health Statistics reports a prematurity rate of 6.4% for white babies and 13.22% for black babies (Reed & Stanley, 1977). Let us assume, for the sake of argument, that the effect of prematurity on whites is negligible, but that the effect on blacks is to reduce their brain weight throughout their entire development into adulthood. Let us assume that in the absence of prematurity the distribution of brain weight is just the same for blacks as it is for whites. Given these assumptions, the mean brain weight of adult blacks should be a weighted composite of the brain weight of those 13.22 percent who were born premature and those 86.78 percent who were born mature and therefore would have the same average brain weight as white adults. Ho et al. (1980a, Table 1) report the mean brain weight for whites as 1,323 g and for blacks as 1,223 g. Thus, according to our assumptions, $.1322x + .8678(1{,}323\text{ g}) = 1{,}223\text{ g}$, where x is the mean brain weight of blacks who were premature. Solving for x, we get 567 g as the average adult brain weight of those blacks who were born premature. This low brain weight is obviously completely out of bounds; it is much less than the brain weight of true microcephalic imbeciles (Penrose & Haldane, 1969, p. 174), and the very lowest brain weights among the 450 blacks analyzed by Ho et al. (1980a) were only slightly less than 900 g. Therefore, the assumptions we made as the basis for arriving at the virtually impossible figure of 567 g—assumptions in accord with

the suggestion of Ho et al. that the greater rate of prematurity in blacks may account for the racial difference in adult brain weights—constitute a reductio ad absurdum disproof of their argument.

Finally, if the higher prematurity rate in blacks appreciably affected adult brain weight, and if, in the absence of prematurity, the distribution of brain weights in blacks were assumed to be the same as in whites, then we should expect negative skewness of the distribution for blacks as well as a larger standard deviation. In fact, as can be seen in Figure 4.19, the distributions of brain weights for black males and females are quite symmetrical (as are the white distributions), and the standard deviations of brain weights for whites and blacks (146 g and 144 g, respectively) do not differ significantly ($F = 1.03$, $df = 811/450$, $p > .05$).

Perhaps the most impressive study of population differences in cranial capacity (and by inference, brain size), based on virtually all of the extant measurements of endocranial volume found in the world literature (numerous studies comprising over 20,000 specimens) is the work of Beals, Smith, and Dodd (1984). They have quite conclusively shown that endocranial volume and cranial morphology vary according to the climatic conditions under which different populations have evolved in different parts of the globe, the main variable being distance from the equator. Linear regressions of cranial capacity on degrees north or south latitude on different continents show slopes of about 2.5 to 3.1 cm^3 increase in cranial capacity per degree of distance from the equator. Racial differences in cranial capacity appear merely as incidental correlates of their different long-term geographic-climatic distributions on the earth. The sex-

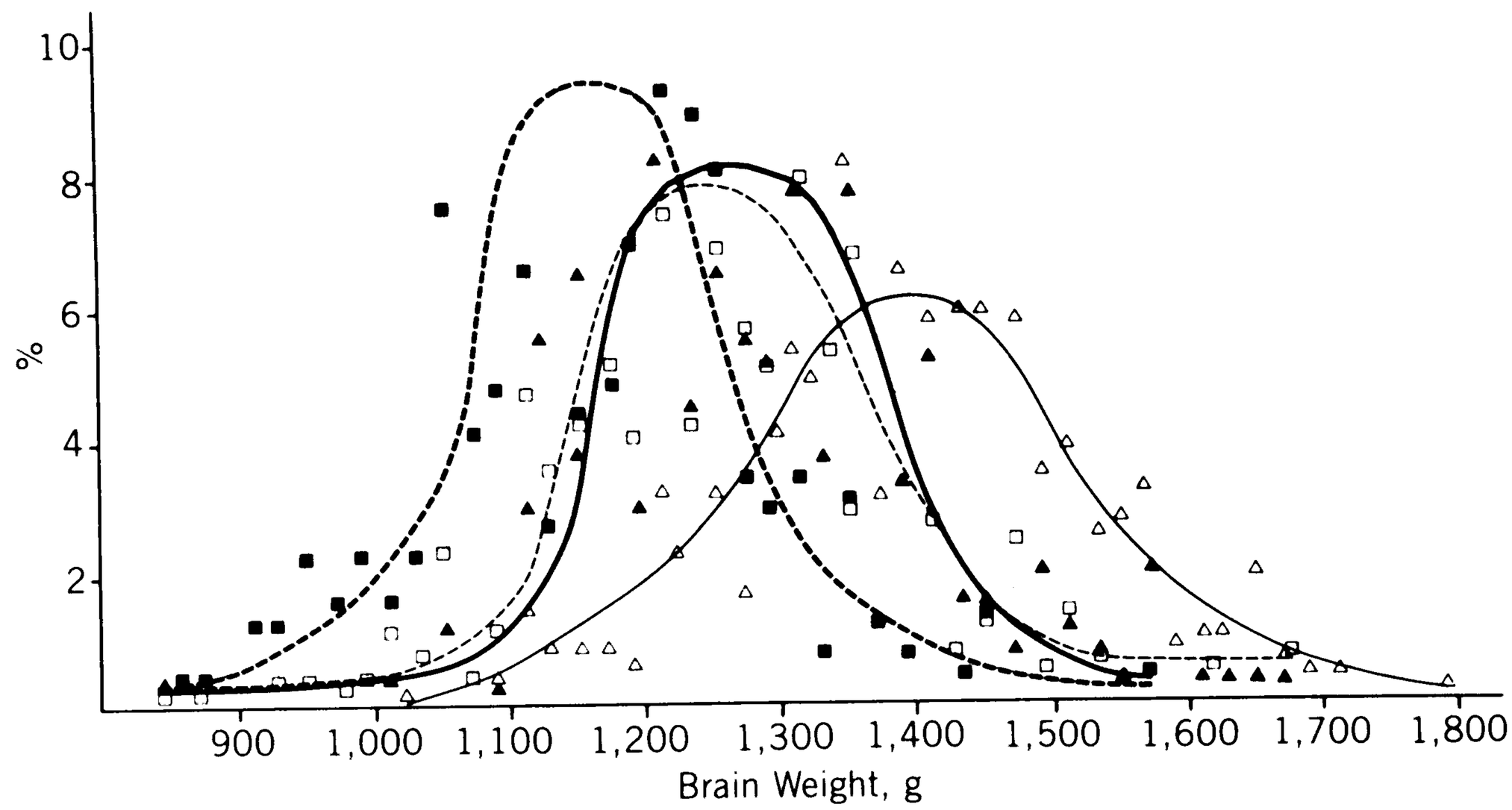


Figure 4.19. Distribution of brain weight in white men (open triangle, thin line), black men (solid triangle, thick line), white women (open square, thick dashes), and black women (solid square, thick dashes). (From Ho et al., 1980a.)

combined mean cranial capacities (in cm^3) of the specimens from the continents of Asia, Europe, and Africa, for example, are 1,380 (SD = 83), 1,362 (SD = 35), and 1,276 (SD = 84), respectively (Beals et al., 1984, Table 2, p. 306). The precise degree to which these differences in mean cranial capacity are reflected in psychometric measures of mental ability remains highly problematic, and a proper consideration of the relevant evidence is beyond the scope of this chapter.

Anatomical Age and Mental Age

Individual differences in rates of both physical and mental growth are well established. The question of interest in the present discussion, however, is whether mental growth rate is correlated with growth rate as indexed by physical characteristics.

Paterson's (1930) review of the relevant evidence showed that rate of physical maturation, as indexed by the degree of ossification of cartilage in the bones of the wrist and by the eruption of permanent teeth, is correlated about +.10 with Binet Mental Age, with chronological age controlled. Indeed, even various aspects of physical maturation are not highly correlated. Hence, there is little functional unity in rates of physical and mental growth.

More recent studies are consonant with these conclusions, and provide additional information which explains the low correlations (around +.10) which are usually found between indices of physical and mental maturity. The main factor is socioeconomic status; when it is held constant, the correlations are close to zero. Physical and mental growth rates are both positively correlated with SES. However, no direct causal connection between growth rates and SES per se should be inferred.

Age of menarche. The age of first menstruation, or menarche, is one index of rate of physical maturity in girls which shows a low significant correlation with IQ, particularly in older studies (pre-1940). For example, data from the Harvard Growth Study (Dearborn et al., 1938), based on children born in the late 1920s and early 1930s, show a correlation of about −.20 between age of menarche and IQ. The basis of the correlation appears to be that age at menarche is correlated with social class; higher SES girls begin menstruating earlier (Tanner, 1963). Later studies have also shown a decrease in the correlation between SES and age of menarche; but there remains a negative correlation between menarche and family size (Dann & Roberts, 1969; Douglas, 1966; Nisbet & Illesley, 1962). As family size is negatively correlated with IQ, not causally, but because both variables are correlated with certain social background factors, it mediates most of the small remaining correlation between IQ and age of menarche. But even with SES and family size controlled, there is still found to be some slight IQ superiority of early over late maturing children (Nisbet & Illesley, 1963). The slight relationship between rates of physical and

mental maturity is most probably a *between*-families correlation. We have found no evidence for a *within*-family correlation, although no studies have obtained the necessary data for determining a within-family correlation.

Puberty praecox. Precocious puberty, a condition due to the abnormally premature development of the gonads and the sex hormones, provides a natural experiment on the effects of puberty on mental growth independent of chronological age. Occurring at any age in the period from early childhood to just before the age of normal puberty, puberty praecox has marked accelerating effects on overall physical development, reflected in height and weight, muscular development, strength, ossification of metacarpal and carpal bones, closure of epiphyseal junctures, body hair (especially pubic hair), and genitalia, which become equal in development to those of normal adults, and secondary sexual characters. Is this marked acceleration of physical maturation reflected also in an acceleration of mental maturation? The answer is afforded by a review of 62 cases of puberty praecox on which there was evidence concerning mental development as well as physical development (Stone & Doe-Kulmann, 1928). The authors concluded that "there is no evidence for genuine mental precocity regularly associated with any glandular disturbance underlying puberty praecox. Most probably, the rate of mental development is either unaffected by the glandular disorder or is retarded" (p. 393).

Two highly detailed longitudinal developmental studies of girls with puberty praecox were made by Gesell (1928). In these two cases, menstruation began at 3 years, 7 months and at 8 years, 3 months. The girls were observed over a period of several years after the onset of puberty and were periodically subjected to physical and mental measurements. The results are quite clear: Although these girls physically manifested all of the features of puberty, with mature primary and secondary female sexual characteristics, their mental development was normally consistent with their chronological age and reflected not at all their precocious physical maturation. Gesell's (1928) conclusion:

> The nervous system, among all the organs of the body, manifests a high degree of autonomy, in spite of its great impressionability. It is remarkably resistant to adversity, even to malnutrition. This relative invulnerability gives it a certain stability in the somatic competition between the organ systems. *It tends to grow in obedience to the inborn determiners, whether saddled with handicap or favored with opportunity.* For some such biological reason, the general course of mental maturation is only slightly perturbed by the precocious onset of pubescence. (p. 409)

OCULAR VARIABLES

Myopia

Myopia, or nearsightedness, is a visual deviation in which light rays entering the eye reach their focal point at some distance before the retina. This condition is

part of a continuum which includes *emmetropia* (normal vision, with focal point on the retina), and *hyperopia* (farsightedness, with focal point falling beyond the retina). Positive and negative deviations from perfect emmetropia are continuously and symmetrically distributed in the general population of adults, forming a rather markedly leptokurtic bell-shaped curve, as compared with the normal, or Gaussian, distribution (François, 1961; Sorsby, Benjamin, Davey, Sheridan, & Tanner, 1957). Individual differences on this dimension are mainly the result of differences in the refractive power of the lens, but are also affected by differences in the axial length of the eyeball and corneal refraction. Hence, to the extent that hereditary factors may be involved, the overall variation in refraction is most probably polygenic.

The general consensus among contemporary researchers on myopia has been that genetic factors are strongly involved (François, 1961; Karlsson, 1973, 1974, 1975a, 1975b, 1976; Sorsby, Sheridan, & Leary, 1962; Sorsby & Fraser, 1964, 1966; Waardenburg, Franceschetti, & Klein, 1963). Although the evidence for the inheritance of myopia is now substantial, the exact mechanism of inheritance is not yet firmly established. However, few geneticists, if any, still favor the hypothesis proposed by early investigators that myopia is a single-gene, or Mendelian, character. Two facts demand a polygenic model of the total variance in refractive error: (a) the refractive error underlying myopia is a continuous variable with a slightly skewed leptokurtic frequency distribution; and (b) at least three separate but correlated optical factors involving the cornea, lens, and axial length of the eye from the cornea to the retina contribute to the refractive error that makes for myopia. A segregation analysis specifically aimed at testing the single-gene model found no support for it (Ashton, 1985b). However, a number of studies of family pedigrees and studies based on monozygotic (MZ) and dizygotic (DZ) twins, siblings, and other kinships are impressively consistent with a model of polygenic inheritance involving both additive and nonadditive (recessive) effects (Furusho, 1957; Karlsson, 1974, 1975b, 1976; Sorsby et al., 1957, 1962; Sorsby, Leary, & Fraser, 1966; Sorsby & Fraser, 1964; Wold, 1949; Young, 1958). Heritability analysis of the MZ and DZ twin data suggests that additive genetic effects account for almost 50% of the variance in ocular refraction, and additive plus nonadditive genetic factors account for most of the variance. There is no direct evidence that myopia is causally dependent on nearwork or reading or other scholastic activity. Moreover, the nearwork hypothesis seems to be contradicted by the fact that myopia occurs with comparable frequency in both literate and nonliterate populations (Post, 1982) and is as frequent in persons with Down syndrome as in other persons from the same population (Lowe, 1949).

Correlation with intelligence. The massive evidence now leaves no doubt that myopia is positively correlated with IQ and with its behavioral correlates, particularly scholastic achievement. A positive relationship between myopia, or nearsightedness, and mental ability was first documented in a European population more than a century ago (Cohn, 1883, 1886). Since then, the positive

correlation between myopia and various measures of intelligence and scholastic aptitude and achievement has been substantiated by numerous studies (Ashton, 1985a; Baldwin, 1981; Benbow, 1986a, b; Benbow & Benbow, 1984; Douglas, Ross, & Simpson, 1967; Dunphy, 1970; Grosvenor, 1970; Heron & Zytoskee, 1981; Hirsch, 1959; Karlsson, 1973, 1975a; McManus & Mascie-Taylor, 1983; Young, 1963). The degree of relationship between myopia and mental ability in the general school population, as estimated in three large studies (Ashton, 1985a; Hirsch, 1959; Karlsson, 1975a), when expressed as a coefficient of correlation, is between about .20 and .25, which is equivalent to an IQ difference of about 6 to 8 points between myopes and nonmyopes. A study by Karlsson (1975a), based on 2,527 California high school seniors, found a difference of 8 IQ points on the Lorge-Thorndike IQ test between pupils wearing correctional lenses for myopia (N = 377) and nonmyopes (N = 2,150). The largest study, by Rosner and Belkin (1987), based on a national sample of 157,748 Israeli Jewish males, aged 17–19 years, recruited for military service, found a strong positive association of myopia with measures of intelligence on both verbal and nonverbal tests and also with years of schooling independent of intelligence.

A recent study of 5,943 myopic and 9,891 nonmyopic 18-year-old men examined for military service in Denmark provides further information on the form of the relationship between mental ability and myopia (Teasdale, Fuchs, & Goldschmidt, 1988). The measurements of myopia in terms of the power (in diopters) of the correcting lens required ranged from mild to severe. The overall intelligence test score differences between myopes and nonmyopes corresponds to approximately 7 IQ points and is, of course, highly significant. Figure 4.20 shows the prevalence of myopia as a function of total scores on a 45-minute group intelligence test consisting of subtests measuring verbal, numerical, spatial, and logical reasoning abilities. Figure 4.21 shows the relation of myopia to educational level (in years of schooling). The authors note that if reading were the cause of the association of myopia with intellectual ability, one should expect a stronger correlation of myopia with *education* than with *intelligence*. (The correlation between these variables was +.572.) Yet both variables are equally related to myopia, and each variable is related to myopia independently of the other, as was found also in the large study by Rosner and Belkin (1987).

The largest intelligence and educational differences occur between myopes and nonmyopes, while differences in degree of myopia beyond 2 to 3 diopters account for relatively less of the variance in abilities, as shown in Figure 4.22. The nonmyopic group differs significantly from all myopic groups from mild to severe; and groups with degrees of myopia beyond 2 diopters show no significant differences in intelligence test scores or level of education.

Within-family correlation between intelligence and myopia. The correlations in all of the studies reviewed so far are only evidence of a *between-families* (BF) relationship between myopia and IQ. There are only two studies reported in the literature that specifically looked at the *within-family* (WF) correlation,

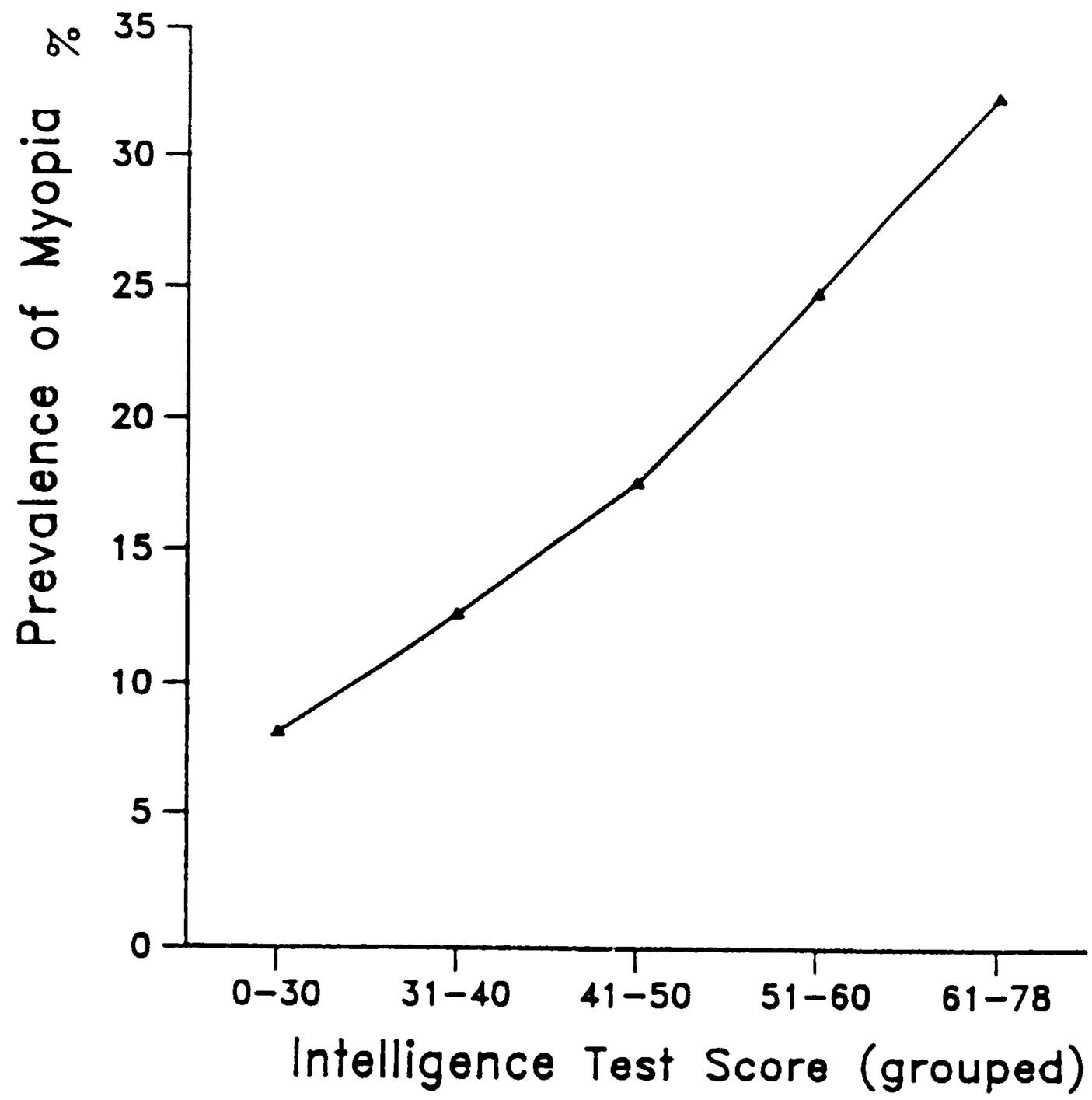


Figure 4.20. Prevalence of myopia in relation to intelligence test score. (From Teasdale et al., 1988.)

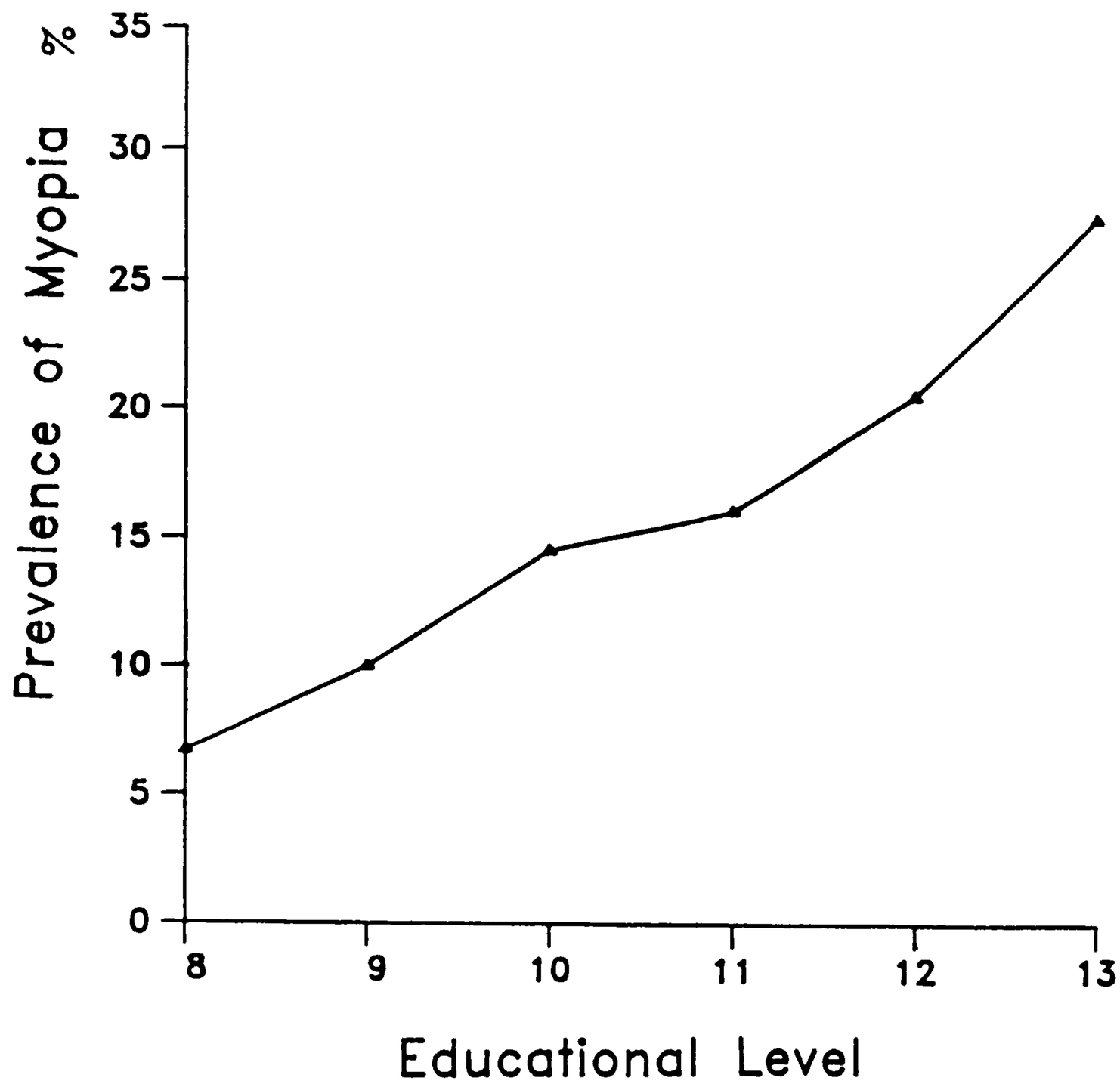


Figure 4.21. Prevalence of myopia in relation to educational level. (From Teasdale et al., 1988.)

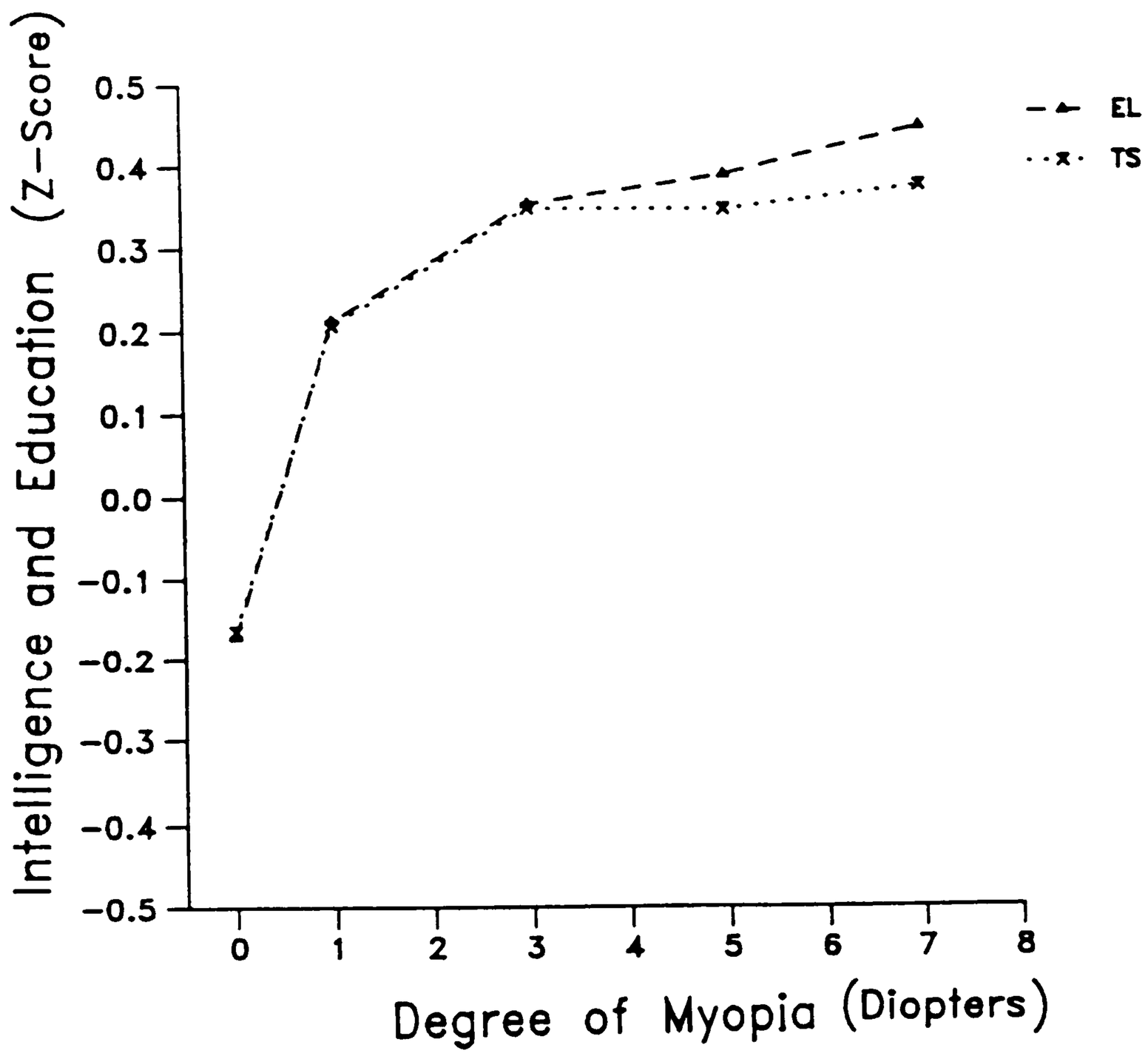


Figure 4.22. Standardized (*z*) intelligence test score (TS) and educational level (EL) in relation to degree of myopia measured in diopters. (From Teasdale et al., 1988.)

which, if significant, would be consistent with the hypothesis of a pleiotropic relationship.

In a study by Benbow (1986b; see also Benbow & Benbow, 1984) of extremely precocious students (the 417 most academically gifted youths selected from over 100,000 gifted students identified in a talent search covering the entire United States), it was discovered that over 50% of the mathematically precocious (and even more of the verbally precocious) were myopic, while fewer than 5% were hyperopic, as compared with about 15–20% of myopes in the general high school population. Myopia in the precocious group was determined by questionnaires mailed to their parents, who were also asked to report on the presence of myopia in any siblings of the precocious probands; 36% of the siblings were reported to be myopic, which is a significantly smaller percentage than the 53% of precocious probands who were reported to be myopic. Among the parents of the extremely precocious, 55% reported being myopic. The greater incidence of myopia in the parents than in their gifted children (55% vs. 53%) is most likely attributable to the increase in myopia with age. Although the Benbows did not measure the IQs of the gifted probands' siblings or parents, it seems safe to infer from simple regression considerations that the IQs of the siblings and parents were lower, on average, than the IQs of the highly selected sample of gifted youngsters. Hence we would conclude that these data indicate a WF correlation between mental ability and myopia.

The WF correlation between myopia and general mental ability was investigated more precisely by Cohn, Cohn, and Jensen (1988), who measured myopia directly as a continuous variable in a group of 60 gifted students (average age about 15 years) and their full siblings. The gifted (G) and their nearest-in-age siblings (S) differed 0.92σ (equivalent to 14 IQ points) on the Raven Matrices (Standard and Advanced forms combined). The G and S groups also differed significantly ($p < .05$) in degree of myopia (measured as refraction error) in both eyes, the G being more myopic, with an average effect size of 0.39σ.

This study, along with the Benbow (1984), leaves little doubt that there is a WF correlation between myopia and mental ability. Cohn et al. consider alternative hypotheses to account for the WF correlation, and conclude that pleiotropy is the most plausible explanation. (A finding of no difference in degree of myopia between the gifted and their nongifted siblings would clearly contradict pleiotropy.) The two alternative hypotheses, which find little supportive and considerable contradictive evidence in the literature, are that (a) genetically determined myopia leads to a preference for close work and studiousness, which in turn leads to higher performance on IQ tests; and (b) genetically and environmentally conditioned higher IQ leads to a preference for reading and studiousness, which in turn strains the eyes, causing myopia. The second link of the first hypothesis lacks plausibility because it posits a weak cause (studiousness) for a large effect in the case of intellectually gifted and their siblings, who differ almost one standard deviation on a test of intelligence that does not involve reading comprehension or bookish knowledge. No one has yet demonstrated any environmental intervention that will raise the IQ by anything near one standard deviation (Spitz, 1986). Hence the second hypothesis seems more plausible than the first, although it must be recognized that the evidence is quite inconclusive that reading, near-work, or studiousness are among the causes of myopia, and most modern investigators have discounted this "near-work" hypothesis of the relation between IQ and myopia.

Eye Pigmentation

We have found only three studies of the relation between eye color (i.e., pigmentation of the iris) and intelligence. These studies leave much to be desired methodoligically and none can be considered compelling.

In connection with the Harvard Growth Study, Estabrooks (1929) compared the IQs of four subgroups of large samples of students from grades 6 through 8 in three school systems in New York and Massachusetts: blue-eye and brown-eye, and light-hair and dark-hair, with each pigmentation group divided by sex. All subjects were Caucasian of North European extraction. In the New York sample, the blue-eyed children's IQs were 3 to 4 points higher than those of the brown-eyed children. Because this effect was confounded with age in this sample (the

younger children had both higher IQs and a greater frequency of blue eyes), Estabrooks argued that pigmentation increased with age in childhood and he attributed the eye color × IQ correlation to age differences. In the Massachusetts study, all of the subjects were of about the same age (8 years), and there was no relationship between pigmentation and IQ as measured by five different group IQ tests.

Riley (1978) compared the SAT scores of 85 Caucasian college undergraduates, ages 18 to 19, who were classified by self-report as having blue eyes or brown eyes. Despite considerable heterogeneity of the SAT scores, their relation to eye color was virtually nil, and far from even borderline significance ($t < 1$). Nor was the variance of IQ related to eye color.

A study of 122 college undergraduates found correlations between eye color (rated on a 12-point scale from very light − 1 to very dark − 12) and scores on the Verbal and Spatial subtests of the Differential Aptitude Tests of −.21 and −.19, respectively; both are significant ($p < .05$). Because both abilities are related to eye color, it suggests that the correlation is actually with the *g* factor common to both abilities, but this hypothesis was not tested nor does the article present the necessary correlations for the required test (Gentry, Polzine, & Wakefield, 1985). In the same study, two other genetic markers (ability to curl the tongue into a tube shape and a yes-no taste test for phenylthiocarbamide [PTC]) also showed significant ($p < .05$) correlations (about .18) with both Verbal and Spatial test scores. Because no information at all is given about the degree of ethnic heterogeneity of the subject samples, these correlations can support no really interesting interpretation. In all three of the studies reviewed here, it seems to be totally unrecognized by their authors that the only type of correlation between eye pigmentation (or any other genetic markers) and mental ability that would be of more than trivial interest from a genetic standpoint is a within-family correlation, which would indicate either genetic linkage or, more probably, pleiotropy. At best, between-family correlations can only provide a clue of which genetic variables to investigate for the presence of a within-family correlation.

BLOOD CORRELATES

Two main classes of blood correlates of mental ability have been studied: (a) antigen polymorphisms, or blood types, and (b) blood serum chemistry, particularly serum urate level. Aside from the fact that they both require the drawing of blood samples, these two classes of variables call for quite different research methods, and they differ markedly in their theoretical implications for study of the biological basis of individual differences in ability.

Blood Polymorphisms

Classification of individuals by blood group (also known as blood type) is based on various antigens, or specific enzymes, in the blood. Each antigen is attributa-

ble to a polymorphic gene, that is, a gene whose particular chromosomal locus can be occupied by two or more alternate forms of the gene, known as alleles. Hence, identification of the various forms of a particular antigen provides a phenotypic marker for a single gene. Over 70 such antigens have been discovered in human blood, some 30 of which are rather commonly used in genetic research. The ABO and Rhesus (Rh) systems of antigens are best known and most widely used, mainly because of their importance in blood transfusion.

The fact that blood antigens correspond to single genes makes them especially valuable in the genetic analysis of other genetic traits. Linkage analysis may show that variance in a given trait (or some fraction of its variance in the case of a polygenic trait) is conditioned by a gene (or genes) located on the same chromosome as the gene for a particular blood antigen. Establishing a genetic linkage between a given trait and a single-gene marker is a large step beyond merely demonstrating the heritability of the trait. Linkage analysis can ultimately lead to the precise chromosomal locus of the gene that affects the phenotypic trait. One of the major tasks awaiting future research in behavior genetics is the linkage analysis of various phenotypic within-family correlates, both behavioral and physiological, of mental abilities. The success of such an endeavor will probably depend as much on the componential analysis of abilities and the measurement of elementary cognitive processes with the techniques of mental chronometry (Jensen, 1985) as on the methodology of quantitative genetics.

Pitfalls. There are two major methodological pitfalls that were unrecognized by some earlier researchers in this field: (a) the capitalization on chance, or random variation, in the use of multiple regression techniques, and (b) reliance on between-family data, which capitalizes on what is referred to in population genetics as "heterogeneity of population structure" or "population stratification" (Thomson & Bodmer, 1975). These terms refer to the fact that most natural populations are a composite of a number of genetically differentiated subpopulations, arising from immigration or invasions, and are maintained by social stratification and assortative mating. Such differentiation is often longstanding and not recognizable from superficial appearance. Different traits which are not at all correlated *within* subpopulations but which differ *between* subpopulations will be correlated in the *combined* subpopulations. But such correlations due to population heterogeneity are utterly trivial from the standpoint of understanding the causal nature of the correlated traits. Certain subpopulations, even within the same major racial classification, are known to differ in the relative frequencies of blood groups. To the extent that the subpopulations also differ in mental abilities, for whatever cause, it should be possible to find correlations between blood groups and abilities in any large sample randomly drawn from a composite of different subpopulations. The obtained correlations, however, will most likely not be replicable in some other population sample.

Both of these pitfalls are clearly demonstrated in an early study (Osborne & Suddick, 1971), which showed multiple correlations (R) between a number of blood types and three mental test factors ranging from .45 to .77, in white (N =

54) and black ($N = 42$) samples. The observed R for 18 predictors (blood types) in the white sample was .69, which is not significant at the .05 level. By selecting from the 18 blood types five that were the best predictors, the R was still large (.58) *and* significant ($p < .05$) for this small number of predictors. This *a posteriori* procedure of selecting a subset of predictors from a much larger set of predictors that in toto is not significant, of course, capitalizes on chance and is a well-known statistical fallacy. This was pointed out by Owen (1972), who attempted to replicate these results using the same set of predictors and regression weights on a similar population sample. The observed sample R of .28 was totally nonsignificant for an N of 84, and the estimated population correlation (i.e., the shrunken R) was a mere .08. The failure to replicate the large but fallacious correlations of the original study is not at all surprising. But even if such a study had been analyzed correctly and showed a significant and substantial correlation which could be replicated in other samples from the same population, the finding would still be trivial, because correlation due merely to population heterogeneity simply cannot be ruled out in this type of study. The reason that such a correlation is regarded as trivial is that no scientifically interesting inferences can follow from it.

Another instructive example is the significant ($p < .001$) correlations of .06 and .07 between IQ and the ABO blood types and Rh factor found in the total sample ($N = 26{,}760$) of white and black children studied by Broman, Nichols, and Kennedy (1975, p. 279). But there were no significant correlations *within* either the white or black samples, even though correlations as small as .02 would be significant beyond the .001 level for such large samples. This can only mean that the observed correlations of IQ with the ABO and Rh systems are attributable to the genetic heterogeneity of the combined black and white samples, which have different gene frequencies for these blood groups.

Between-families studies of the ABO groups. The ratio of A to (O + B + AB) blood type frequencies was found to differ by social class in two districts in England (Beardmore & Karimi-Booshehri, 1983). The social class differences in A/(O + B + AB), with the upper classes having the highest frequencies of A type blood, were consistent in the two separate locales and in males and females. The well-established correlation between social class and IQ makes it reasonable to hypothesize that the correlation between social class and blood type comes about through their mutual association with IQ. The authors of this study favor linkage or pleiotropy to explain their finding, arguing that the two regions studied have different invasion and immigration histories and that generations of social mobility would have homogenized originally different gene pools. If their conclusion is correct, other studies in different populations should reveal a correlation between the ABO system and IQ. As noted previously, no correlation between ABO and IQ was found in very large white and black samples (Broman et al., 1975).

A study of the residents of seven English villages found significant correlations between Wechsler IQ and the ABO system ($A_2 > 0 > A_1$), but the

correlation is confounded by IQ differences between locally born residents and immigrant residents—again a case of population heterogeneity (Gibson, Harrison, Clark, & Hiorns, 1973). A later study in much the same locales, with larger samples, found no significant correlation of ABO with IQ when "localness" was controlled, but turned up two other significant blood group correlations with Wechsler Performance IQs—the Kell ($p > .05$) and Haptoglobin (Hp) ($p < .001$) markers (Mascie-Taylor, Gibson, Hiorns, & Harrison, 1985). The Hp correlation was entirely attributable to the Block Design subtest, and Kell was associated only with Performance IQ. The test specificity of these correlations leads the study's authors to suggest that the relationship is probably not a result of population heterogeneity. The correlations remained significant when sex, localness, social class, village, birth order, family size, and handedness were statistically controlled. Six other blood groups (ABO, MNS, Rh, PGM, ADA, and Tf) showed no significant correlations with mental test scores. But the significant correlations (Kell and Hp) will remain of little interest until they can be replicated in different populations, and the possibility of their representing pleiotropy or linkage could be investigated only by a within-families study. The study by Mascie-Taylor et al. (1985) did find a within-family difference in Haptoglobin types between male siblings who were downwardly mobile in social class and those who remained in their father's social class. This one within-family correlation would seem to make Haptoglobin worthy of further study in relation to the spatial visualization factor manifested in the Wechsler Block Design subtest, with which it showed the highest correlation.

Blood-type homozygosity. The Haptoglobin (Hp) groups, however, showed no significant correlations with any of the 15 tests, including 5 tests of visual-spatial abilities, used in the large Hawaii Family Study of Cognition (Ashton, 1986). A total of 18 different blood polymorphisms and 15 diverse tests of mental abilities turned up about the same number of significant ($p < .05$) correlations as would be expected by chance alone, with a total $N = 4{,}469$. However, another kind of relationship between blood polymorphisms and tests of verbal and spatial abilities was discovered at a high level of significance ($p < .001$). (Speed and memory factors did not show this relationship.) This was the individual's overall amount of homozygosity on all 18 blood groups, that is, whether the alleles for any particular blood group were the same (homozygous) or different (heterozygous). Because each allele at a given locus is contributed by a different parent, the homozygous condition suggests less hybridization, but more important, it suggests a lower probability of maternal-fetal incompatibility of blood antigens. It was found that increasing homozygosity, based on the number of homozygous polymorphisms among the 18 that were tested, was significantly correlated with higher test scores for verbal and spatial abilities. This relationship was found in both Caucasian and Asian samples, and in the parent and offspring generations, so it appears to be a genuine phenomenon. Although the mental test variance accounted for by zygosity in this study is exceedingly small, it could amount to a considerable effect over a much greater

number of genetic loci. Ashton (1986) estimates that the effects of zygosity at 50 loci could account for 7.5% of the variation in verbal and spatial abilities. (The common ability factor here is most likely *g*.) The mechanism by which zygosity can affect cognitive development is explained by Ashton as follows:

> The advantage of being homozygous is most evident in immunological tolerance expressed during grafting or transplanting. A developing fetus is a special kind of graft in which the fetus is potentially incompatible with the maternal genotype at all polymorphic loci. . . . [A] reasonable biological hypothesis is that antigenic incompatibility exists at many loci and is expressed through subtle effects during brain development *in utero*. The more homozygous an individual is, the less developmental deficit is incurred. (p. 528)

Rh factor and fetal development. The one blood polymorphism that has long been known to have an effect, under certain conditions, on brain development and intelligence is the Rhesus, or Rh, factor, which can result in antigenic incompatibility between the mother and fetus. Rh incompatibility, known in its clinical forms as kernicterus of hemolytic disease of the newborn, can occur when the mother is Rh-negative (*dd*) and the father is Rh-positive (DD or Dd). The Rh-negative offspring are safe, as they are compatible with the mother. The Rh-positive offspring are at risk, because they are antigenically incompatible with the mother, who builds up antibodies to attack the "foreign" fetus. This maternal immunization against an Rh-positive fetus generally has little if any detectable effect on the fetus in the first pregnancy, because the mother's immunization is not sufficiently developed. The build-up of antibodies continues to increase after delivery, and with each subsequent pregnancy the Rh-positive fetus is at increased risk, often requiring a complete blood exchange transfusion at the time of birth in order to maintain the neonate's viability or to lessen brain damage.

The behavioral effect of this condition on mental development varies from very slight retardation to idiocy, although the technique of exchange transfusion greatly improves the prognosis (Penrose & Haldane, 1969, pp. 237-239). A Finnish study of 43 youths, 17 to 19 years of age, who were Rh incompatible with their mothers and had required postnatal replacement transfusions, were compared with 43 matched controls and were found to perform less well than the controls in general intelligence, visual-motor coordination, and scholastic achievement (grade-point average, attention, and concentration). Degree of deficit was positively related to the length of delay after birth in the neonate's being given an exchange transfusion (Wuorio, 1974).

Penrose and Haldane (1969, p. 238) cite other studies that lead them to suspect that maternal-fetal incompatibility in other blood groups, particularly the ABO system, might have similar, though less pronounced, effects on fetal viability and mental development. More recently, another geneticist, Karlsson (1978), wrote:

> Blood group incompatibilities, involving both the Rh factor and the ABO system, are known to cause progressively greater damage to the fetus as the number of pregnancies increases. Presumably there are additional factors of this type, which still have not been discovered. (p. 182)

The slight negative correlations between birth order and IQ which show up significantly only in studies of very large groups (Ernst & Angst, 1983) could be a reflection of the effects of such antigenic incompatibilities. The importance of this phenomenon in terms of its contribution to the nonheritable component of IQ variance has not yet been adequately investigated.

Within-family studies. As previously emphasized, the study of within-family relationships between genetic polymorphisms and mental ability is the only methodology that can potentially establish genetic linkage, pleiotropy, or an intrinsic, or directly causal or functional, connection between different traits. The method has been applied in only a few studies of the blood-ability relationship.

Bock, Vandenberg, Bramble, and Pearson (1970) compared the within-pair variances of Wechsler Verbal IQ for 526 DZ twins classified as concordant or discordant on each of four blood groups systems: ABO, MNSS, Rh, and Duffy, or Fy. Discordance only for Rh and Duffy showed increased within-pair variance in Verbal IQ, with Rh showing the stronger effect. The authors interpret this result as evidence for linkage between the blood-group loci and a major gene that enhances verbal intelligence. Other plausible explanations have not been ruled out, such as maternal incompatibility with Rh-positive, or linkage of Duffy with Rh (both loci are on the same chromosome), or pleiotropy. In any case, the fact that DZ twins who are discordant for certain blood groups show greater differences in IQ, as compared with DZs who are concordant for the same blood groups, seems worthy of much further investigation. If the phenomenon replicates, a definitive explanation of it would be of great importance.

In an attempt to replicate the findings of Bock et al. (1970), Rose, Procidano, Conneally, and Yu (1979) examined full-sib pairs who were concordant (N = 56) and discordant (N = 74) on six different blood groups, including Rh and Duffy. Only Rh discordance showed a greater within-pair variance on Wechsler Vocabulary, but the effect is only suggestive and not significant (p = .12). Because of the polygenic nature of complex mental abilities, and the linkage of only one or very few genes with any single blood group marker, the effect of sibling discordance on ability for any one marker gene would necessarily be quite small if there were linkage, and hence a very large sample of sibling pairs would be needed to detect linkage with a high level of confidence.

X-linkage. So-called sex-linkage, which is usually linkage occurring on the X chromosome, of which females normally inherit two X chromosomes (i.e., XX) and males only one (i.e., XY), is easier to detect by studies of male full siblings than is autosomal linkage, simply because males possess but a single X chromosome. The required data consist of a large number of sets of three male

full siblings in which only two members of the set are concordant for a given genetic marker, such as a blood group antigen. Degree of similarity on another trait (in this case mental ability) is compared within male sib-pairs that are either concordant or discordant for the X-linked marker gene. If there is X-linkage between the marker gene and the trait of interest, the brothers who are concordant for the marker gene will be more similar on the trait than are the brothers who are discordant for the marker.

Because of the well-established sex difference in the cognitive style known as field-dependence and in spatial-visualization ability, it was hypothesized that the genetic component of these variables is X-linked. The hypothesis was tested, with the male siblings method described above, in an exemplary study by Goodenough et al. (1977), using a known X-linked blood group, $X_g(a)$. The hypothesized linkage effect was found at a significant level for the two measures (Rod-and-Frame, Embedded Figures) of field-dependence, but in none of the six tests of spatial-visualization ability could the null hypothesis be rejected. Brothers who were identical in $X_g(a)$ blood group were more similar to each other in degree of field dependence than brothers who differed in $X_g(a)$. The authors conclude:

> If the result can be cross-validated . . . the conclusion that a gene on the X chromosome is involved in the development of field dependence would appear to be established. Whether an individual carries the $X_g(a^+)$ gene or not has no known consequence of any importance to him. Certainly neither he nor his associates can detect that fact. If field dependence is transmitted in association with $X_g(a)$, then it would appear difficult to account for such a finding except by appeal to an X-linked model. (p. 383)

The number of identified genetic markers with known loci on specific chromosomes is constantly increasing, a fact which should make it more productive in the future to conduct linkage studies of various mental ability factors. The success of such an effort would counter the common complaint that, despite the great many studies showing high heritability for mental abilities, not a single specific gene has been identified as related to normal variation in mental ability.

Serum Uric Acid

Belief in an association between gout and eminence dates back to antiquity. This belief first gained more than mere anecdotal status when Havelock Ellis (1904), in his study of a large, representative sample of intellectually eminent Britishers, actually discovered a much higher incidence of gout than is found in the general population. The question naturally arises why gout—a painful inflammation usually of the joint in the big toe—should be associated with notable intellectual achievement.

Gout is the direct result of inflammation due to the formation of uric acid crystals in a joint. Uric acid is the end product of the metabolism of purines, which are constituents of animal proteins and fats. Uric acid is excreted in the urine and is carried in low concentrations in the blood as serum urate, or serum uric acid (SUA). At higher concentrations, SUA forms crystals which are deposited in the joints, most commonly of the big toe, which cause the painful inflammation known as gout.

Interestingly, in all other mammals except man and the higher apes, uric acid is not the end product of purine metabolism. In other mammals, the enzyme uricase oxidizes uric acid to allantoin, which is excreted as the end product of this metabolic chain. In the course of evolution, the higher apes and man lost the capacity for synthesizing uricase, with the result that uric acid was the end product of purine metabolism.

More interestingly, the molecular structure of uric acid is highly similar to that of caffeine, which is a central nervous system stimulant. (High serum uric acid level is also related to faster reaction times, as is caffeine.)

These observations led Orowan (1955) to hypothesize that the uric acid in the blood, SUA, as an endogenous stimulant to the brain, contributed to the evolution of human intelligence by the constant cortical arousal caused by this stimulant. Orowan's hypothesis prompted behavioral scientists to look for mental correlates of SUA. Correlational studies are entirely feasible, as SUA can be quite reliably measured, is quite stable in adults, and shows a wide range of individual differences. The distribution of SUA has a higher mean, larger standard deviation, and is more platykurtic in men than in women (Mikkelsen, Dodge, & Valkenberg, 1965). Proportionally fewer women than men have high levels of SUA. The sex difference is not evident until puberty. SUA then increases more for men, reaching a peak between ages 20 and 24 years, after which there is a gradual decline and plateau. After menopause, women's SUA increases gradually to about the SUA level of men in their fifties.

Genetic factors are implicated in individual differences in SUA. Studies of MZ and DZ twins, of MZ twins reared apart, and of parent-offspring regression indicate heritability coefficients around .30 to .40 (Mueller et al., 1970). The fact that the distribution of SUA is a slightly skewed unimodal curve is consistent with polygenic inheritance.

Orowan's hypothesis gave rise to a large and complex literature on the behavioral correlates of SUA. Fortunately, most of this literature has been excellently and thoroughly reviewed by Mueller, Kasl, Brooks, and Cobb (1970). The 122 references in their article will not be cited here. We will cite only the more recent studies, not included in the Mueller et al. review, in connection with our summarization of the findings in the earlier review that are most germane to the present chapter. A more extensive catalog of the literature related to behavioral aspects of SUA is provided by Stevens (1973).

It should be realized that the causal nature of the observed behavioral corre-

lates of SUA is still obscure. Although there is evidence that the experimental manipulation of uric acid level in rats is significantly related to their maze learning and retention (Essman, 1970), a direct causal connection between SUA and behavior has not yet been established in humans. Many different hypotheses have been suggested to explain the correlation of SUA with behavioral variables: direct neural stimulation affecting certain cognitive processes, increased drive or motivation, effects of other chemical products in the metabolic process ending in uric acid, environmental factors that affect both SUA and behavior, and brain activity that yields uric acid as a product.

Ability correlates of SUA. There is a significant positive correlation between SUA levels and mentally retarded/nonretarded treated as a dichotomous variable, but the SUA correlation with IQ (or psychometric *g*), though significant, is quite small. Also, groups with higher than average IQ (e.g., Ph.D.s, medical students, business executives) show higher levels of SUA than the average for the general population. But such groups, of course, also differ from the average in many other ways besides their above-average intelligence.

When we look at the correlations of SUA level with IQ itself, however, the result is less impressive, suggesting that other factors besides IQ per se are probably involved in the elevated SUA levels of professional groups. We note that in the three largest studies, with a total $N = 1104$, the N-weighted mean Pearson r is only $+.085$ (unit-weighted mean $= +.093$), which is significant beyond the .01 level. In 149 high school students, the r was $+.10$ (Kasl, Brooks, & Rodgers, 1970), which is nonsignificant for this sample size, but highly consistent with other reported correlations. (We have not come across any negative correlations, but of course there is a greater risk of failure to report negative or inconsistent results.)

As most of the correlations reported in the literature are Pearson r (i.e., a linear correlation), it was suggested that the SUA $\times$ ability relationship may actually be nonlinear—an inverted U—and hence is grossly underestimated by the Pearson r (Stevens & Cropley, 1972). This very reasonable hypothesis merits examination. Unfortunately, the one study that attempted to demonstrate the hypothesized nonlinear relationship between SUA level and level of mental ability, with the latter decreasing beyond some optimal level of SUA, yielded equivocal results, showing a significant nonlinear trend on only two out of the four different *g*-loaded tests administered to 115 male university students (Stevens, Cropley, & Blattler, 1975). The hypothesized inverted-U relationship of ability to SUA remains unsubstantiated, but the evidence is also far too inadequate to dismiss it.

Cognitive styles and SUA. On the hypothesis that elevated SUA level is related to high ambition, acceptance of conventional goals, and high drive for achievement, it was predicted that SUA level would *be negatively* related to a cognitive style known as "divergent thinking" (Cropley, Cassell, & Maslany, 1970). Divergent thinkers are claimed to be relatively unconcerned about strict observance of rules, and are more impulsive, unconventional, and willing to take

risks than are persons who are characterized as "convergent" thinkers, who excel in typical IQ tests, do well in school, and succeed in conventional occupations. The prediction was significantly borne out, but only for the male subjects, in the study by Cropley et al. (1970). Divergent thinking was measured by the Wallach and Kogan battery; convergent thinking by Thurstone's Primary Mental Abilities Test. Male (but not female) high "divergers" showed significantly *lower* SUA levels. There was no significant relationship of SUA to the convergent thinking measures. In another study, of men only, with superior methodology, such as control of diet and exercise (which can affect SUA), Kennett and Cropley (1975) again found a significant inverse relationship between SUA level and divergent thinking, and virtually no relationship with convergent thinking. These findings seem to suggest that SUA levels may be more related to certain personality variables than to intellectual ability per se. The personality variables may simply be influential in intellectually demanding pursuits.

Achievement and SUA. If an elevated level of SUA acts as a cortical stimulant, like caffeine, we should expect its main effect to be manifested not in higher intelligence per se, but in a higher level of activation of cognitive functions which would lead to greater *achievement* for any given level of intelligence. This, in fact, is what is most commonly reported in the literature on the behavioral correlates of SUA. The evidence most strongly substantiates the positive association of SUA with achievement in mentally demanding pursuits. In a study of 149 male high school students, SUA level was most notably correlated with *over*achievement, defined as level of achievement (indexed by school grade-point average) adjusted for IQ (Kasl, Brooks, & Rodgers, 1970). SUA level was also positively related to students' number of extracurricular activities. When university faculty ($N = 144$) were rated on overall level of achievement, the ratings were correlated $+.50$ with SUA level (Mueller & French, 1974). Among nontenured faculty, the chief criterion for promotion to tenure—number of publications—was correlated $+.37$ with SUA level.

The relation between SUA and achievement seems well established for males, but too few females have been subjects in SUA studies to warrant any conclusion about SUA and achievement in women.

Socioeconomic status and SUA. The cortical stimulant nature of SUA and its relation to achievement has suggested the hypothesis that elevated SUA level would be related to upward social mobility, as indexed by level of education and occupation. Numerous studies have found evidence for this hypothesis, although at least one study (not included in the extensive review by Mueller et al., 1970) showed no relation of socioeconomic status to SUA level (Acheson, 1969). SUA is correlated with the dichotomous variable "school drop-out/persistence in school." It is related to ratings of ambition and "need for achievement," and shows a substantial correlation with "achievement orientation behavior" in professional groups.

One large study (total $N = 1500$) contrasted professionals and executives, on

the one hand, with farmers and unskilled workers, on the other, and found, on average, a higher SUA level in the former than in the latter group (Dodge & Mikkelsen, 1968). Perhaps the most interesting feature of this study is that SUA was measured also in the wives of the men in these contrasting occupational groups, and there was no difference in their average SUA levels. This suggests that the differing levels of SUA by occupation in the males is probably not attributable to possible social class differences in dietary habits or general lifestyle, which would presumably be shared by the wives.

MISCELLANEOUS VARIABLES

Our literature search has turned up a number of other physical variables that studies have found to be related to mental ability. But the studies are single or very few for each variable, and hence afford an inadequate basis for conclusions or theoretical interpretation. Some, however, are probably promising for further research and should be cited in this review. Replication, of course, is essential for these variables to accrue scientific interest. Some of the correlations are merely opportunistic—calculated from sets of physical and mental measurements that were originally obtained for other purposes. Hence, the reported correlations, though significant, are considerably more liable to Type I Error than are correlations found in studies specifically designed to test a particular hypothesis. With this caveat, we mention these studies only briefly.

Vital capacity. The maximum amount of air that can be expired from the lungs, which is related to stature, age, and physical fitness, showed correlations of +.23 and +.29 with mental test scores (Schreider, 1968; Whipple, 1914).

Handgrip strength. Related to intellectual level and efficiency in various aged samples (total $N = 2000$) of healthy males and females (Clement, 1974).

Facial features. Judges' ratings of intelligence from photographs of faces of regular students are correlated (about .20) with teachers' assessments of the subjects' intelligence (Burt, 1919), and with intelligence test scores (Kiener & Keiper, 1977). The latter study details the specific facial characteristics that discriminate significantly between the 14 most and the 14 least intelligent among 84 women students.

Hair color. In 1100 Swedish school children, IQ was positively related to darkness of hair, and the mean IQ of red-haired pupils was slightly below average and significantly fewer went on to high school (Lundman, 1972).

Basal metabolic rate. High (+.6 to +.7) correlations with IQ were found in two studies (Ns = 90 and 200) of children, ages 5 to 16 years (Hinton, 1936, 1939), but no significant correlations between BMR and IQ were found in a study of 87 adolescents (Shock & Jones, 1939), or between BMR and scholarship in six studies of college students (Yarbrough & McCurdy, 1958). The studies of children suggest a sharp drop in the BMR × IQ correlation after about age 10; the relationship may be an early developmental phenomenon and, in

view of the large correlation reported by Hinton, warrants further investigation.

Masa intermedia. This mass of neural tissue connects the two halves of the thalamus. It is absent in about one-third of men and one-fourth of women. In a group of 74 adult neurological patients there was a significant difference in the means of the Wechsler-Bellevue Performance scales of men who possessed the *masa intermedia* and those who did not, the latter scoring *higher* (Landsdell, Davie, & Clayton, 1972). Strangely, among those in whom the *masa intermedia* was present, there was a *positive* correlation ($+.43, p < .01$) between the size of the *masa intermedia* and Performance scores. The use of magnetic resonance imaging, with neurologically normal persons, should be able to verify these puzzling relationships if they are generalizable beyond the particular study sample.

Asthma and other allergies. Intellectually precocious children, especially those with exceptional mathematical reasoning ability, show a much higher incidence of allergies of various kinds than is found in the general population. Some 55% of verbally and mathematically precocious students suffer from allergies, in contrast to about 10 percent in the general population (Benbow, 1986a, 1986b; Benbow & Benbow, 1986). The authors relate this highly significant finding to a hypothesis of Geschwind and Behan (1982) to the effect that immune disorders (and left-handedness, also more prevalent in the precocious) are related to fetal exposure to high levels of testosterone or high sensitivity to testosterone. Testosterone slows development of the left hemisphere, which may lead to enlargement of the right hemisphere, which is presumably involved in the kind of spatial-visualization ability that is often correlated with exceptional mathematical reasoning ability. Whatever the merits of this hypothesis, the essential empirical finding seems an important one in view of the fact that the relation between allergies and precocity is a *within*-family correlation. The incidence of allergies is 15 to 20% higher among the precocious probands than among their parents and siblings.

Phenylthiocarbamide (PTC) tasting ability. PTC, a synthetic chemical, is of interest to geneticists because the ability of individuals to taste this chemical (which is unpleasantly bitter if it can be tasted) is due to a single Mendelian dominant gene. About 70 percent of European and Asian populations are "tasters." Hence, PTC tasting ability is frequently used in determining the zygosity of twins and as a genetic marker. Two independent studies, with Ns = 122 and 141, have both found that nontasters score significantly higher than tasters in g and spatial ability, with correlations about .20 (Gentry, Polzine, & Wakefield, 1985; Mascie-Taylor, McManus, McLarnon, & Lonigan, 1983). PTC showed no correlation (-.04) with a test of clerical speed and accuracy, which has a minimal loading on g.

Tongue-curling ability. Whether or not a person can curl the tongue into a tube shape behaves like a single-gene Mendelian trait and hence can be used as a genetic marker in studies of linkage and pleiotropism. There seems little likelihood of assortative mating or cross-assortative mating with intelligence for this

character, although there remains the possibility of population heterogeneity for both traits and a consequent between-families correlation. In a study of 122 unrelated college undergraduates (hence between-families), tongue-curling ability was found to be positively and significantly ($p < .05$) correlated with verbal reasoning ($r = +.18$) and spatial reasoning ($r = +.20$), but not with clerical speed and accuracy ($r = .03$) (Gentry, Polzine, & Wakefield, 1985). As the authors correctly point out,

> The smaller size (about .20) of the significant correlations between genetic markers . . . and intelligence . . . does not detract from their importance. Indeed if the markers are genetically simple and the psychological variables are genetically complex, small correlations would be expected to occur and can be expected to point the way to specific genetic contributors to personality and intelligence. (p. 113)

However, we emphasize again that such correlations can have no causal status unless they are established in a within-families study. If the variables are significantly correlated within families, then the appropriate studies can be done to determine whether the correlation is attributable to linkage disequilibrium or to pleiotropy.

SUMMARY AND CONCLUSIONS

The massive evidence of significant correlations between scores on psychometric tests of mental ability and a wide variety of physical traits shows beyond a reasonable doubt that the population variance on standard mental tests reflects latent traits that are profoundly enmeshed with organismic variables in complex ways.

The causes of the relationship between individual differences in mental ability, on the one hand, and individual differences in anatomical, physiological, serological, and biochemical characteristics, on the other, are both numerous and different for various physical traits. Hence, the causal nature of the correlation between mental and physical traits is often unique and must be studied separately in each physical trait for which such correlation is found. This kind of analysis is further complicated by the fact that some of the physical traits that are correlated with mental ability are themselves correlated with one another. The causal pathways therefore do not yield easily to analysis.

The nature of the association between mental and physical traits can be classified in several ways that are useful theoretically. These systems of classification are not all completely independent or mutually exclusive, and any particular correlation can be classified in various ways.

The association between a mental and a physical trait can be (a) *functional*, or intrinsic, and closely related to the biological mechanisms directly involved in

the mental activity, such as brain anatomy and biochemistry; or it can be (b) a *nonfunctional*, or indirect, by-product of a long chain of other causes, often reflecting cultural values, such as the nonfunctional relationship between intelligence and physical stature.

Another system of classification is the distinction between (a) significant correlations that occur *within* families (i.e., correlation between different traits among full siblings), and (b)) correlations that are essentially zero *within* families but significantly greater than zero *between* families. The first classification (i.e., (a)) is more likely to include functional or intrinsic relationships (i.e., (a) above), while the second classification (i.e., (b) is more likely to include the nonfunctional types of relationships (i.e., (b) above).

Within each of these categories, there are still other types of correlation between mental and physical traits.

Within-family correlations are most often explained in terms of

1. *genetic linkage* (i.e., the genes for different traits having loci in close proximity on the same chromosome), but this correlation is detectable only in family pedigree studies or in genetically heterogeneous populations that are still in a state of linkage disequilibrium for the traits in question
2. *pleiotropy* (i.e., one gene that affects the development of two phenotypically distinct traits)
3. a *structure-function* relationship whereby performance is directly dependent on a particular physical structure or biochemical mediator; and
4. *exogenous* (not heritable) prenatal or postnatal factors that affect siblings differentially with respect to the parallel development of both mental and physical traits.

Between-family correlations (when accompanied by zero within-family correlations) are most often explained in terms of

1. *genetic heterogeneity*, or genetic stratification, of the population with respect to certain mental and physical traits, making them correlated in a random sample from such a composite of genetic subpopulations
2. *selective mating* for each of two genetically independent traits, usually because the particular physical and the mental traits both happen to be valued in a given culture, for whatever reason
3. *assortative mating* for each of two distinct traits
4. *cross-assortative mating* for both traits, that is, a tradeoff between a particular trait in one mate for a different trait in the other mate (e.g., intelligence and stature), usually because in the particular culture both traits are perceived as desirable
5. *common environment*, that is, factors (e.g., nutrition, ordinary childhood diseases, and the like) that generally affect all of the siblings within a family

but may differ between families, and which simultaneously affect both mental and physical development.

The origins of exclusively *between*-family correlations must be sought in the immediate environment (e.g., nutritional differences, health-care standards) and in the cultural factors that determine mating preferences.

The origins of *within*-family correlations, particularly pleiotropy and linkage, are more obscure and probably must be sought in the evolutionary history of a given population. There may be little or no evident rhyme or reason to a particular correlation due to pleiotropy (e.g., the positive correlation between myopia and intelligence) or to linkage. But pleiotropy originates through genetic mutation, which is a random phenomenon, and so the effects of mutation are essentially random and without rhyme or reason. However, the pleiotropic mutations that endure in a population do so through selection, by virtue of the increment in overall Darwinian fitness conferred by the *net* adaptive effect of the two or more phenotypic expressions of the pleiotropic gene. Hence there need not be any obvious or inherently "logical" or psychologically explainable connection between the distinct phenotypic characteristics that are correlated pleiotropically. Their continued existence in the population, however, indicates that, adaptively, their net effect (i.e., the algebraic sum of their separate positive or negative effects) has, so to speak, passed the test of natural selection in terms of fitness. A good case can be made that psychomteric *g*, the main latent trait measured by conventional intelligence tests, has evolved as a fitness character during the course of human evolution.

The significant associations between mental ability and physical traits which are the most securely established by research are all quite low—their true or error-corrected values are virtually all represented by correlation coefficients between about .10 and .40.

Stature (general body size, height, and weight) shows correlations with IQ averaging close to +.20. This correlation is now so strongly established by such massive evidence that further demonstrations of it would seem pointless. It appears to be a between-families type of correlation, most likely attributable to assortative and cross-assortative mating for stature and intelligence, and probably also to shared nutritional and health factors within families.

Brain-size correlations with IQ, controlling for overall body size, differ depending on the precision with which brain size (or the encephalization quotient, which controls for body size) is measured. IQ is correlated with external head measurements between +.10 and +.20, with intracranial volume about +.25 to +.30, and with direct *in vivo* measurement of the brain by means of MRI, about +.35. This is probably a structure-function type of correlation, but this point has not yet been adequately researched to draw any compelling conclusion.

Age of menarche, a developmental variable, is correlated with IQ about −.20.

Myopia shows a within-family correlation with IQ, which is consistent with the hypothesis that the well-established population correlation of about +.20 to +.25 between IQ and myopia is pleiotropic.

Blood groups lend themselves to genetic linkage studies when significant within-family correlations are found between particular blood groups and mental abilities. Research in this vein so far has turned up little, but there is some evidence, surely in need of replication, that certain blood groups may be linked to particular ability factors other than *g*, such as spatial visualization. There is stronger evidence that *maternal-fetal blood antigen incompatibility*, particularly in the Rh and ABO systems, is probably related to the afflicted offspring's mental development, although only some very small proportion of the total population variance in IQ is likely to be accounted for by this factor. *Serum urate* (uric acid in the blood) level is correlated only about +.10 with IQ, but shows considerably higher correlations with various achievement indices, suggesting that it acts as a cortical stimulant affecting intellectual drive more than level of intelligence per se.

Also reviewed briefly were a number of other claimed physical correlates of IQ based on single or very few studies and for which any scientifically worthy conclusions must await replications. Reports in the literature of particular physical correlates of mental ability, when not the main aim of a systematic program of research, are probably more prone to Type I error than might be expected for other findings in the general behavioral science literature.

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