Arthur R. Jensen

Within the past decade, empirical findings in behavior genetics have importantly changed how researchers in this field think heredity and environment affect individual differences in mental ability. These insights are hardly new. Some are even found in the writings of Sir Francis Galton (1822–1911), the father of behavior genetics. But the recognition, formalization, and empirical support given to them by behavior geneticists in recent years can be considered significant advances. The most surprising findings, only conjectured by Galton, concern the role of environment in the development of mental ability. The present picture is quite different from the beliefs generally held only a decade ago.

Genotype-environment covariance

One such idea is that the perceptible environment is like a cafeteria. People make different selections according to their genetic makeup, or *genotype*. The environment is not a "given" but is largely the person's own creation. This becomes increasingly true as persons develop from infancy to maturity.

Behavioral differences between persons that result from their selfselected and self-fashioned environments are the phenotypic expression of *genotype-environment covariance*.¹ It accounts for more of the total variance (i.e., individual differences) in abilities and achievements than was formerly thought. Genotype-environment (GE) covariance is neither a strictly genetic nor a strictly environmental component of phenotypic variance but reflects the genetically driven differential selection of experiences from the available environment. It also includes the effects of differential treatment by parents, teachers, and peers, because their responses are largely evoked by the person's distinctive genotypic characteristics.

Environmental forces peculiarly accommodate people's genotypic propensities. People seek out different environments, including friends and activities, that are congenial to their nature. The wider the variety of genotypes in a population, and the more varied the environment, the larger is the GE covariance component of the total phenotypic variance. The familiar phrase "nature and nurture" is now replaced by "nurture via nature." This is not just a subtle distinction; it proves theoretically crucial for understanding some essential data of behavior genetics. Researchers Robert Plomin and Sandra Scarr have most prominently furthered this idea (Plomin, 1986; Plomin & Bergeman, 1991; Plomin, DeFries, & Loehlin, 1977; Plomin & Neiderhiser, 1992; Scarr, 1985; Scarr & Carter-Saltzman, 1982; Scarr & McCartney, 1983; also see Bouchard & Segal, 1985; Rowe, 1987; Rowe & Plomin, 1981; Willerman, 1979).

Epistasis and emergenesis

Another new focus is on the genetic mechanism called *epistasis*. Epistasis is the interaction of two or more genes at different chromosomal loci to produce a distinct phenotypic effect, which cannot be explained by the additive effects of multiple genes. The idea of epistasis has been broadened to include interactions between polygenic systems that affect distinct phenotypic traits. Termed *emergenesis* in this context, it is essential for understanding the occurrence of conspicuous phenotypic differencess between close relatives in certain traits (Lykken, McGue, Tellegen, & Bouchard, 1992).

Occasionally, one sees a remarkable difference between one family member and all the others. The person's exceptional talent or trait seems *too* exceptional to be explained in terms of the usual *additive* effects of polygenes or differences in upbringing. For instance, Beethoven's brothers also had music lessons but showed only mediocre talent; the parents and siblings of the mathematical genius Ramanujan showed neither a mathematical bent nor any other intellectual distinction; and the parents, siblings, and children of the great conductor Toscanini had no outstanding musical talent. One could list countless other examples showing that most geniuses seem to just "come out of the blue."

According to emergenesis, the unusual development of certain abilities and talents depends on some rare combination of genes or polygenic systems that simultaneously influence several different abilities and traits. Only if this critical combination is present does the talent appear, given an appropriate environment. For example, Galton suggested that a higher than average level of general mental ability, energy, and persistence are involved in most outstanding achievements. Each parent's genotype may carry only some part of this combination. As parents pass on a random half of their genes to each of their children, there is some very small probability that any one child will get the particular combination of genes needed for the talent. It is like getting a royal flush in poker. All five of the critical cards must be in the shuffled deck when the game begins, but a habitual poker player's chances of getting them in his hand all at once are so slight that it rarely or never happens in his lifetime. Geniuses rarely pass on their extraordinary emergenic gift. Like anyone else, they transmit but a random half of their genes to each of their offspring. That this random half will include the genius's particular rare combination of genes is very unlikely. Hardly anyone questions the conclusion that the extraordinary achievements of genius exemplify both emergenesis and GE correlation. John B. Watson notwithstanding, there is no evidence that any special kind of environmental influences, if applied to a random sample of healthy infants, would be at all likely to produce the equivalent of a Shakespeare, a Beethoven, a Newton, a Michelangelo, a Gandhi, or a Babe Ruth.

In such examples, the importance of the environment is often overrated. Many parents who have hoped their seemingly talented child would become a great musician have done everything they could for their child to achieve this goal. Yet exceedingly few ever become famous musicians, even with unusual ambition and efforts of parents and child. In contrast, when Leonard Bernstein was a child, his parents even went so far as to get rid of the piano in their home, because their young son showed such intense devotion to practicing on it that they feared he might one day think of becoming a professional musician, a possibility his father extremely wished to preclude. Years later, in Carnegie Hall, after one of Leonard Bernstein's concerts with the New York Philharmonic, a family friend chided Bernstein's father for having tried early on to discourage his famous son's passionate interest in music. The elder Bernstein pleaded, "How was I to know he would become Leonard Bernstein?"

How do genetic researchers discover that a particular trait is emergenic? If, for the given trait, one finds a very high correlation (say, .75) between monozygotic (MZ) twins reared apart, and a very low correlation (say, .15) between dizygotic (DZ) twins reared together, one suspects emergenesis. Although DZ twins (and ordinary full siblings) have, on average, about half their genes in common, very rarely do they both have the same unique combination of genes associated with the emergenic trait. Also, if there is a high correlation between MZ twins reared apart, a very low correlation found between DZ twins reared together is not likely to be a result of environmental differences. Lykken et al. (1992) give many examples of emergenesis identified by the twin method (i.e., DZ correlation significantly less than one-half of the MZ correlation). Most of the psychological examples are in the realm of personality traits (e.g., extraversion), attitudes (e.g., religiosity), and interests (e.g., hunting and fishing, gambling). There are many examples of emergenesis in the realm of physical traits, where individual differences in some traits (e.g., a beautiful or handsome face) often depend on an ideal configuration of features that are genetically independent. So there is a very low probability that the relatives of an exceptionally

good-looking person will inherit the same combination of genes that make for an ideal configuration. A Greta Garbo or a Clark Gable typically has quite ordinary-looking parents and siblings.

It is noteworthy that general intelligence, as represented by IQ or by psychometric g (i.e., the general factor common to a diverse battery of cognitive tests), does not behave as an emergenic trait in genetic analyses. However, an above average level of g is often a critical condition for the development of an emergenic talent when the expression of the talent itself depends on the possession of certain complex cognitive skills. For example, acquiring the knowledge and technical skills needed to express an emergenic talent in mathematics or musical composition usually requires a superior level of general intelligence. It is exceedingly unlikely that there was ever a great mathematician or composer who was not above average in general intelligence. (Being above a threshold level in some trait for the manifestation of emergenesis, however, does not apply to every emergenic trait, particularly those in the personality domain.)

Sources of environmental variance

The most startling discovery in recent years concerns the locus of environmental effects on general intelligence. It was once believed that the most potent sources of environmental variance in IQ are conditions that differ between families. These are variables such as socioeconomic status (SES), cultural background, parents' education and occupation, style of child rearing, number of books in the home, and the like. In the last decade, we have seen the results of several large-scale studies of adopted children, such as the Texas, the Colorado, and the Minnesota adoption studies. They show, to everyone's astonishment, that these environmental differences between families account for little or none of the variation in the IOs of adolescents and adults, although these shared environmental factors account for about half of the total environmental variance in preadolescent children. Also, in childhood, the proportion of shared environmental variance among relatives is directly related to their degree of genotypic similarity, which decreases going from twins to siblings to parents-offspring to cousins (Chipuer, Rovine, & Plomin, 1990). But adoption studies based on adolescents and young adults show that the effects of shared (or between-families) environment have diminished to almost zero, with little change in the proportion of nonshared (or within-families) environmental variance and a marked increase in the proportion of genetic variance. Yet the adoptive families in these postadolescent studies range widely in SES and other variables on which many families typically differ from one another. Yet such differences scarcely contribute to the variance in IQ after childhood. This remarkable fact could have been discovered only by studying adopted children. In children reared by their biological parents, the effects of heredity and environment are completely confounded. The children's IQs and the quality of the environment are both correlated with the parents' genotypes. Countless studies of children reared by their biological parents report large correlations between IQ and environmental assessments. However, these correlations are unable to prove anything about the importance of environmental factors for individual differences in IQ. This is because the observed IQ–environment correlation reflects more than just the *direct* effect of environment on the person's mental development. It includes also the effect of the parents' genotypes on the environment, plus the parent–offspring correlation due to parents and their offspring having about half their genetic variance in common.

Past investigations of the effects of the home environment on IQ have too often overlooked the influence of the genetic correlation between parents and offspring. A careful study (Longstreth et al., 1981) that took this factor into account correlated children's IQs with ratings of the home environment (based on a 2-hour interview with the parents) on those aspects commonly believed to affect children's intellectual development. It demonstrated, as have many such studies, a significant and substantial correlation between the environmental measures and children's IQs. The correlation dropped to nonsignificance, however, when the mothers' IQs were partialed out.

Such outcomes are easily understood from the path model in Figure 2.1, which shows the causal effects of heredity and environment on the child's mental development (here indicated by IQ) for nonadoptive and adoptive children. As is evident in Figure 2.1, the crucial advantage of an adoption study is that it eliminates the effect of the parent-child genetic correlation from the connection between environment and IQ (or any other trait). Therefore, any significant correlation between adoptive children's IQs and the typical environmental differences *between* adoptive families must be due solely to environmental effects. (This is true, of course, only if the adoptees and their adoptive parents are not genetically related, and if adoptees are not selectively placed according to their supposed genotypes for intellectual development.)

Empirically, it turns out that this between-families source of environmental variance constitutes almost half of the total environmental variance in IQ in childhood but is practically nil in adolescents and adults (McGue, Bouchard, Iacono, & Lykken, 1993). By late adolescence, almost none of the environmental component of IQ variance results from differences *between* family environments – that is, those aspects of the environment that are *shared* by children reared together in the same family but that differ between one family and another. Most of the strictly environmental, or



Figure 2.1. Diagram showing causal connections (straight arrows) and correlations (curved lines) between the genetic and the systematic (i.e., nonrandom) environmental factors that influence a child's IQ when the child is reared by its biological parents. In the case of an adopted child whose biological parents are unrelated to its adoptive parents, the two dashed arrows are deleted.

nongenetic, variance exists *within* families. It comprises those environmental effects that are *unshared* or *specific* to each child in a family. To the exent that adult family members resemble each other in intelligence, they do so almost entirely because of their genetic similarity. Apparently, as individuals progress from childhood to adulthood and encounter an ever-increasing range of experiences, they discover and select from their widening environment those aspects that are most compatible with their own genotypic proclivities. Therefore, with increasing maturity, the individual's genotype is increasingly expressed in the individual's phenotypic characteristics, reflected by the diminishing proportion of environmental variance and the increasing proportion of genetic variance (broad heritability).

This amazing fact, which contradicts popular belief, is one of the major discoveries of behavior genetics in the past decade. And it poses an extremely important puzzle – the puzzle of nongenetic variance. To understand it, we need to review a few technical matters.

Variance components in behavior genetics

The total variance in a metric trait, such as IQ, can be partitioned into several components, as shown in Figure 2.2. Each main component, or



Figure 2.2. Branch diagram of the partitioning of the total phenotypic variance into the components (i.e., sources of variance) that can be estimated by the techniques of quantitative genetics. The main focus of the present article is on the environmental components (particularly *within-families*), shown highlighted.

source of variance, can be subdivided into more specific components. The genetic variance, for example, is analyzed into additive and nonadditive components, and each of these is analyzed into two components. Explanation of every component in Figure 2.2, and of how they are estimated by the methods of quantitative genetics, is beyond the scope of this chapter. It is covered in most textbooks of behavior genetics.

When the total phenotypic variance (V_P) is standardized (i.e., $V_P = 1$), the variance of each component then becomes a decimal fraction, or proportion, of V_P . Empirical studies usually report standardized values of the variance components.

This chapter focuses on the environmental variance (V_E) . It is analyzable into two components, called Between Families (BF) and Within Families (WF), with variances V_{BF} and V_{WF} .

It should be noted that some writers use other terms, as follows, for the BF and WF components of the environmental variance. They all have the same meaning.

Between Families (BF)	Within Families (WF)
Common Environment	Specific Environment
Shared Environment	Nonshared Environment
Systematic Environment	Random Environment
E_2	E_1

The BF environment, by definition, is any environmental influence on a trait that causes two or more persons who were reared together to be more alike, on average, than persons who were not reared together. The BF component is the variance of the means of each of many sets of persons who were reared together (e.g., sets of siblings or pairs of twins). A simple rule: The variance *between* families is the covariance *within* families.²

The WF environment, by definition, is the environmental influence on a trait that causes persons who were reared together to differ from each other. The WF variance component is the environmental variance specific to each person.

A coefficient of correlation (r) between persons is also a variance component. (This correlation should not be squared to represent a proportion of variance; it is itself the proportion of common or shared variance.) Correlation coefficients based on different classes of persons are used to estimate the variance components shown in Figure 2.2. For example, if persons take the same test twice (a few days apart), or take two equivalent forms of a test, the coefficient of correlation between the scores obtained on test and retest (or between equivalent forms) is the proportion of *true-score* variance. This correlation is also known as the test's *reliability coefficient* (r_{xx}). The proportion of error variance, therefore, is the complement of the reliability, or $1 - r_{xx}$.

The variance components of particular interest in this article are obtained from correlations based on the kinds of data shown in the accompanying chart. Regarding monozygotic twins reared apart (MZA), the genetic component V_g estimated by r_{MZA} includes some fraction of the GE covariance found in MZ twins reared together (MZT), so r_{MZA} actually estimates $V_g + kV_{ge}$, where k is some fraction of the V_{ge} of MZT. Also, the correlation between unrelated persons reared together (r_{UT}) excludes the genetic component only if the adopted children have not been selectively placed according to their supposed genotypes. All of the components listed in the table are attenuated by measurement error. The correction for attenuation, which eliminates the effect of measurement error, is to divide each correlation or variance component by the reliability (r_{xx}) of the test used to obtain it. The r_{xx} of IQ tests in an unrestricted sample is typically about .90. When possible, estimated variance components intended for theoretical interpretation should be corrected for attenuation.³

Correlation Between	Variance Components
Text-retest on same persons	True-score (V_{TS})
Monozygotic twins reared apart (r_{MZA})	Genetic (V_g)
Monozygotic twins reared together (r_{MZT})	Genetic (V_g) , GE interaction (V_i) GE covariance (V_{ge}) BF environment (V_{BF})
Unrelated persons reared together $(r_{\mu\tau})$	BF environment (V_{BF})

Other variance components that cannot be measured directly are estimated by subtracting one empirically obtained component from another. Certain components can be estimated by several different kinds of data.

The BF environmental component (V_{BF}) , for example, is estimated directly by r_{UT} and indirectly by the formula $r_{MZT} - r_{MZA}$. But this formula may underestimate V_{BF} , because the GE covariance (V_{ge}) is likely to be smaller in MZA than in MZT. MZ twins, of course, have identical genotypes, but those who are reared together usually have a more similar environment than those reared apart. The greater similarity in environment makes the GE interaction and GE covariance larger in MZT than in MZA. Therefore, the difference, $r_{MZT} - r_{MZA}$, comprises not only V_{BF} but some fraction of $(V_i + V_{ge})$, and thus overestimates V_{BF} . The preferred estimate of V_{BF} is the correlation between unrelated persons who were reared together (r_{UT}) . Another possible estimate of V_{BF} , though less compelling than r_{UT} , is the correlation $(r_{P_AC_A})$ between adoptive parents (P_A) and their adopted children (C_A) .

Estimating the WF environmental variance (V_{WF}) allows three options. The simplest is based on only one correlation and is therefore less liable to error than formulas based on two or more correlations. Many estimates of V_{WF} in the literature are based on $1 - r_{MZT}$, but this is spuriously inflated by variance due to measurement error. If one has a good estimate of the reliability of measurement (r_{xx}) , the better estimate, corrected for attenuation, is $1 - r_{MZT}/r_{xx}$.

Another estimate of V_{WF} is $V_{xx} - r_{MZA} - r_{UT}$. However, this method is proper only if the samples of MZA and UT are of about the same age. The relative sizes of the genetic component of IQ and the BF and WF environmental components all change between childhood and maturity (Plomin, 1986, chapter 14).

Still another estimate of V_{WF} is $r_{xx} - r_{MZA} - r_{P_AC_A}$. As in the previous formula, the MZA and C_A samples should be of similar age. Using correla-



Figure 2.3. Proportion of variance in Wechsler test performance associated with heritability estimates (top panel) and shared environmental components (bottom panel) derived from the ongoing University of Minnesota cross-sectional study of reared-together twins. (From McGue et al., 1993, p. 72, with permission of the authors and the American Psychological Association.)

tions based on different age groups in one and the same formula may result in seriously inconsistent estimates of the variance components.

Estimates of all these components are made more accurate when corrected for attenuation (by dividing each of the correlation coefficients on which they are based by r_{xx} , or the equivalent, by dividing the final variance component by r_{xx}), assuming, of course, that r_{xx} is itself quite accurate.

MZ twins reared together from birth, despite having identical genotypes, do differ significantly in many personal characteristics, including IQ. Although these differences are typically much smaller than the differences between dizygotic (DZ) twins or ordinary siblings reared together, they are nevertheless real. The true-score differences between MZT afford probably the least ambiguous estimate of the WF environmental variance $(1 - r_{MZT}/r_{xx})$. This WF component becomes the large part of the nongenetic truescore variance in IQ after early adolescence. From childhood to maturity, the BF environmental component dwindles almost to nonexistence. By adulthood, virtually the *only* nongenetic variance in IQ is the WF component. The marked increase in heritability and decrease in the betweenfamilies (or shared) environmental variance is well illustrated in Figure 2.3, which is based on data from the Wechsler intelligence scales obtained from two different age groups of reared-together MZ and DZ twins.

The term *nongenetic* here seems preferable to *environmental*. In a psychological context, most people think of *environment* as only the psychosocial-cultural milieu. But the main causes of WF variance are still uncertain. They could be more directly biological than psychosocial-cultural.⁴ The more neutral term *nongenetic*, therefore, is less apt to prejudice possible conceptions of the nature of WF variance.

Empirical estimates of BF and WF variance components

Reviews (Bouchard & McGue, 1981; Bouchard, Lykken, McGue, Segal, & Tellegen, 1991; Plomin, 1986, 1988; Plomin & Daniels, 1987) of studies of the kinship correlations used in the genetic analysis of human mental ability provide the evidence for the following conclusions. The most telling are studies of genetically unrelated persons who were adopted in infancy, reared together in the same family, and tested in late adolescence or adulthood (Scarr & Weinberg, 1978; Teasdale & Owen, 1984; Willerman, 1987). They show much smaller correlations (close to zero) than those obtained with adoptees tested in childhood. The *N*-weighted mean correlations (obtained via Fisher's *Z* transformation) based on all the available studies are probably the best estimates of the correlations used in the follow-

Relationship	Reared	Symbol	Number ^a	Correlation	Corrected ^b
MZ twins	Together	MZT	4,672	.86	.95
MZ twins	Apart	MZA	162	.75	.83
Childhood	•				
Unrelated	Together	UT	570	.29	.32
Postadolescent ^d	•				
Unrelated	Together	UT	385	.0025	.003
Parent-child ^e	Adopted	P _A C _A	1,397	.19	.21

Table 2.1. Weighted mean correlations used for WF variance estimates

"Number of pairs.

^bCorrection for attenuation based on reliability coefficient of .90.

^c Mean age of 9 years.

^dMean age of 17 years.

^eAdoptive parents and adopted child are genetically unrelated.

ing analysis are the N-weighted means of all the correlations reported in the published studies of each type of kinship data, it amounts to a meta-analysis of the estimated variance components.

Basic correlations. The *N*-weighted means of all the available correlations used for estimating WF variance are shown in Table 2.1.

Estimates of BF environmental variance. Four different estimates of the BF variance (with correction for attenuation in parentheses) are as follows.

- 1. $V_{BF} = r_{MZT} r_{MZA} = .86 .75 = .11$ (.12) 2. Childhood $V_{BF} = r_{UT} = .29$ (.32)
- 3. Postadolescent $V_{BF} = r_{UT} = .0025$ (.003)
- 4. $V_{BF} = r_{P,C} = .19$ (.21)

Estimates of WF nongenetic variance.

- a. $V_{WF} = r_{xx} r_{MZT} = .90 .86 = .04 (.044)$
- b. $V_{WF} = r_{xx} r_{MZA} r_{UT} = .90 .75 .29 = -.14 (-.15)$ c. $V_{WF} = r_{xx} r_{MZA} r_{UT} = .90 .75 .0025 = .15 (.16)$
- d. $V_{WF} = r_{xx} r_{MZA} r_{P,C_1} = .90 .75 .19 = .05$ (.06)

The observed differences between the various estimates of the nominally same component, whether BF or WF, call for some explanation. These differences are almost entirely a result of the fact that, for IQ, the relative sizes of the components of genetic variance, GE covariance, and BF and WF environmental variances systematically change with age. Genetic variance (V_s) and GE covariance (V_{se}) gradually increase from infancy to maturity. V_{BF} increases from early childhood to puberty, then decreases

markedly to late adolescence and maturity. V_{WF} decreases from early childhood to midchildhood and then remains nearly constant to maturity. From early childhood to maturity, the major trade-off is between the increasing $(V_{g} + V_{ge})$ and the decreasing V_{BF} . Therefore, the discrepancies in the estimates of V_{BF} and V_{WF} are mostly attributable to these age changes and the fact that the correlations for MZT, MZA, and UT are based on different age groups. It so happens that studies of MZT are mostly based on schoolage children, while MZA studies are nearly all postadolescents and adults. UT studies are based both on children and on postadolescents; the Nweighted average correlations obtained separately within each age group are used here. In the Texas adoption study, for example, pairs of unrelated adopted subjects reared together were tested for IQ as children (average age 10 years), and showed a correlation of .26. When they were tested again in late adolescence (average age 18 years), they showed a correlation of only .02 (Willerman, 1987). In view of these facts, several of the alternate estimates of the BF and WF components, as identified by the numbers or letters used earlier, call for comment.

1. Because MZT have more GE covariance in common than do MZA, the difference between r_{MZT} and r_{MZA} is a slightly inflated estimate of V_{BF} , which, by this estimate, really consists of $V_{BF} + kV_{ge}$, where k < 1. The value of k is not precisely known but is probably greater than 1/2. Therefore, a reasonable guesstimate of V_{BF} (corrected for attenuation) would be about .06.

4. This estimate of V_{BF} is based on the $r_{P_AC_A}$ for children, but because the adoptive parents are adults, they share less of the BF environment with their adopted children than is shared by two children of similar age reared together. Therefore, we should expect $r_{P_AC_A} < r_{UT}$, and this is what is found (i.e., .19 < .29).

a. Because MZT as children share more of the BF environment than MZT as adults, and studies of MZT are based mostly on school-age children, they have larger V_{BF} than adults. The formula $r_{xx} - r_{MZT}$, therefore, probably underestimates adult V_{BF} to some degree. The estimate obtained in (c), based entirely on postadolescent data, is predictably larger (.15 > .04).

b. This estimate is clearly anomalous, as it results in negative variance, which is impossible. The reason for the anomalous estimate is that the formula includes one correlation based on adults (i.e., $r_{MZA} = .75$) and one based on children (i.e., $r_{UT} = .29$). Note that $r_{xx} - r_{MZA}$ therefore estimates $V_{BF} + V_{WF}$ for adults, and if we subtract from it $r_{UT} (= V_{BF})$ based on children, a *negative* value is obtained, because V_{BF} is larger for children than for adults. Therefore the V_{WF} obtained in (c), which is based entirely on postadolescent data, is probably a good estimate.

d. Because $r_{P_xC_x}$ is based on children, it overestimates adult V_{BF} ; therefore, when it is subtracted from $r_{xx} - r_{MZA}$, it gives an underestimate of V_{WF} .

An exact estimate of the WF variance, however, is not crucial to the present argument. What we do know with reasonable certainty is that, beyond childhood, there is almost zero BF environmental variance in IQ. Whatever environmental variance exists is WF (.10 to .20 of the total true-score variance). The rest of the reliable IQ variance consists mainly of genetic variance (.60 to .70) and GE covariance (.10 to .20).

There is no significant evidence of a GE interaction component for IQ. One statistical test of GE interaction is the correlation (r_{md}) between the means of MZ pairs and the absolute differences between individuals in each pair (Jinks & Fulker, 1970). In the pooled 69 pairs of MZA for whom scores are available in the literature, $r_{md} = -.09$, p = .22. The weighted average of r_{md} in all studies of MZT (totaling 1,435 pairs) is -.04, p = .06. But this is at best a weak test, which assumes that the genes controlling sensitivity to the environment are the same as those that affect the average expression of the trait in MZ twins. Because other factors besides GE interaction, such as skewness of the score distribution, can cause r_{md} to differ from zero, this test may exclude the presence of GE interaction if the null hypothesis ($r_{md} = 0$) cannot be rejected but cannot prove the existence of GE interaction if the null hypothesis is rejected. The null hypothesis cannot be rejected on the basis of the existing studies of MZA. (The technical problems of detecting GE interactions are well discussed by Neale and Cardon [1992, pp. 22–3].)

Distinction between IQ and psychometric g for genetic analysis

Genetic models often make a distinction between the *phenotype* of interest and some particular *index* of the phenotype. *Intelligence*, as a psychological construct, and IQ, as a standardized score on a particular mental test, are examples of a phenotype and its index. There is not necessarily a perfect correlation between the true phenotype (if it were measurable) and an index of it. Therefore, analysis of the index variance into genetic and nongenetic components does not necessarily yield precisely the same proportional values of the components as would be obtained from a parallel analysis of the true phenotype.

For *intelligence*, however, this proposition cannot be examined, because there is no generally agreed upon meaning of *intelligence*. Undefined terms are unsuitable phenotypes for behavior-genetic analysis. So we are left with only an index, an IQ score based on a particular test, which is highly correlated but not perfectly correlated (even when corrected for attenuation), with other IQ scores based on different tests.

The fact that IQ tests are all quite highly correlated (about .80) with each

other, however, means that they measure some factor in common, whatever that factor may be. Many analyses have shown that this factor is the same one that is common to individual differences in performance on virtually all cognitive tests and other manifestations of mental ability, however diverse these may be in the specific information content and particular skills involved (Jensen, 1992). This *general factor* is called Spearman's g, or psychometric g, or just g. It can be estimated with varying degrees of accuracy by factor-analyzing large and diverse batteries of cognitive tests – the larger the battery and the more diverse the tests, the better the estimate of g.

Typical IQ tests, when factor-analyzed with a large and varied assortment of other cognitive tests, have large g loadings. Some 75–85% of the reliable variance in IQ consists of g variance. This distinction between IQ and g should be kept in mind in genetic analyses of IQ, because the results are slightly different for the same analyses applied to g factor scores. The difference is theoretically important. IQ variance has a smaller genetic component than g, even though IQ may also reflect the genetic component of other ability factors besides g, such as verbal and spatial ability (Bouchard, Lykken, McGue, Segal, & Tellegen, 1990, 1991).

Most but not all of the genetic variance in a battery of diverse tests is contained in the g factor, while the environmental variance resides mainly in the group factors and the variance specific to each test (Cardon, Fulker, & Plomin, 1992; Luo, Petrill, & Thompson, 1994). However, verbal and spatial group factors show some slight heritability independent of g. Various mental tests differ markedly in heritability (i.e., the proportion of genetic variance in test scores), and the tests' heritability coefficients are positively correlated to a high degree (.6 to .8) with the tests' g loadings (Jensen, 1987; Pedersen, Plomin, Nesselroade, & McClearn, 1992). In other words, the more a test reflects genetic variance, the larger its g loading. Factor analysis was developed by Pearson and Spearman at the turn of the century without any thought of genetics behind it. Yet the process of extracting the general factor, or g, from a number of diverse cognitive tests by means of factor analysis filters out, so to speak, much of the environmental variance (not including GE covariance).

In computer terms, the g factor reflects mostly individual differences in the genetically conditioned "hardware" of information processes, while individual differences in the "software" arise from environmental influences, learning, and experience. Individual differences in IQ based on any particular test typically reflect more of the "software" or experiential component of variance than does the g factor. Even so, the best estimate of the heritability of IQ as measured in adults by a single IQ test is about .75, which if corrected for attenuation would be about .80.

The nature of the nongenetic WF variance in IQ

The puzzle of nongenetic variance is this: By late adolescence, the betweenfamilies (BF) environmental variance in IQ has diminished to near zero, and the only remaining source of nongenetic variance is within families (WF). What, then, are the kinds of environmental effects that could be the source of the WF variance? This is a puzzling question, because psychologists have generally believed that the main environmental effects on IO exist between families as differences in the psychological-educational-socioeconomiccultural environment (PESC). Also, psychologists have generally believed that BF differences in these PESC effects are a much greater source of IQ variance than the more subtle differences in the psychological environment that cause differences between persons who were reared together. If these beliefs were true, why should the BF environmental variance in IQ diminish to almost zero from childhood to maturity, while the WF variance remains nearly constant throughout this period? Evidently, BF environmental differences, or PESC effects, do not have a strong or lasting influence on mental development, at least as it is indexed by IQ.

This important conclusion, however, should not be generalized to include individual differences in how effectively people have used their general mental ability in various attainments. Educational and occupational achievements reflect considerably more than mental ability as indexed by IQ (Jensen, 1993). Many other variables are involved, such as opportunities, interests, values, energy level, ambition, persistence, work habits, lifestyle, and other aspects of character and personality. Some of these personal variables involve genetic factors, although to a probably lesser degree than IQ, and they may be more influenced by the PESC aspects of the BF environment. But this is a separate issue and beyond the scope of this chapter.

To understand the nature of the predominant environmental effects on the distribution of IQ, at least within the typical range of the PESC environment in our population, we must focus on the WF environmental variance. One way to do this is to propose a working hypothesis and seek relevant evidence. As a working hypothesis, which is not yet tested and implies no theoretical commitment, it focuses examination of a class of nongenetic variables that has been peculiarly slighted in research on individual differences in IQ.

The physical microenvironment as a cause of WF variance. To account for nongenetic variance that shows up in genetic analyses even of very highly heritable physical characteristics, such as height, Sir Ronald Fisher (1918)

hypothesized what he termed the *random somatic effects* of the environment. The causes of these random somatic effects can be prenatal, perinatal, or postnatal. Each such effect can be so slight as not to be individually detectable. Because the single effects are many and random, however, their net effect may differ considerably between persons. For the same reasons, according to the law of errors, individual differences in the net effects would have a normal distribution in the population.

The innumerable causes of these effects can be called the *micro-environment*. We can hypothesize that the microenvironment affects both physical and mental development and that the nonshared or WF nongenetic variance in IQ mainly reflects microenvironmental effects.

This is illustrated by the following analogy. Suppose that the microenvironment is represented by a huge stack of cards. Each card bears a single integer number ranging, say, from -3 to +3, where the negative and positive numbers indicate the degree of unfavorable or favorable effect of a single microenvironmental factor. The numbers occur with equal frequencies in the stack. The cards are shuffled, and 10 cards are dealt at random to each of 10,000 persons. Each person's net score is the sum of the numbers on the 10 cards received. The total range of scores, therefore, would have an approximately normal, or bell-shaped, distribution extending from -30 (very bad luck) to 0 (average luck) to +30 (very good luck). This normal distribution of luck would have a mean of 0 and a standard deviation (SD) of approximately 10. (The particular numerical values in this analogy are of course wholly arbitrary.) If we aggregate scores into many small groups (analogous to families), the variance of the means of these random net effects (analogous to the between-families variance) will be much smaller than the variance of individual scores. (The total variance between individuals is the sum of the between-groups variance and the within-groups variance. The between-groups variance is 1/nth of the individual variance, where n is the average number of individuals in a group.) Therefore, the small between-groups variance may be swamped by other, larger sources of trait variance (e.g., genetic). Behavior-genetic analyses intended to estimate BF environmental variance, such as the correlation between unrelated children reared together, will scarcely reflect the microenvironmental effects responsible for the WF environmental variance.

This random model of the microenvironment is essentially the same as the genetic model for the inheritance of polygenic traits.⁵ The approximately normal distribution of a polygenic trait (e.g., height) in all the offspring of the same parents results from the net effect of a different random assignment to each offspring of one-half of each parents' genes. Each gene produces a small positive or negative effect on the phenotype, with a different net effect for each offspring (unless they are MZ twins). Differences between full siblings in their genetic endowments in any polygenic trait are like a random lottery. Some individuals have better luck than others.

Geneticists also recognize certain rare or mutant genes that singly can have a large phenotypic effect (usually deleterious), which overrides the normal polygenic determinants. (These are called *major gene* effects.) By analogy, a similar feature can be incorporated in our model of the microenvironment. Some single, rare environmental factors, such as accidental trauma or disease, can have a large, overriding phenotypic effect. These *macro*environmental effects are added to the normal distribution of microenvironmental effects in the population. One or both tails of the resulting composite distribution, therefore, would deviate from a normal curve. The amount of deviation would reflect the proportion of the total WF environmental variance contributed by macroenvironmental effects. These effects can be incorporated in the cards analogy by including in the stack of cards a small proportion bearing large numbers (e.g., ranging from ± 20 to ± 30).

The idea of randomness, or luck, as a source of important behavioral difference has been neglected in psychology. It is not a new idea. Galton (1908/1974) may have been the first to suggest its explanatory value. More recently, Paul Meehl (1978) has invoked a *random walk* hypothesis to explain the discordance of MZ twins for the development of psychiatric illnesses that have a strong genetic component, such as schizophrenia. The random walk hypothesis is attractive because research has failed to confirm hypotheses about MZ twin discordance in highly heritable traits that posit only a few categorical or systematic environmental variables, each with a big effect.

It has also been hypothesized that random epigenetic effects, or developmental noise, is an intrinsic phenomenon in all complex biological systems and may occur even under conditions of identical genotypes and uniform environment. This phenomenon is even regarded by Molenaar, Boomsma, and Dolan (1993) as a *third* source of variance, distinct from and in addition to genetic and environmental variance. These geneticists cite much relevant literature on what might be termed *autonomous chaos* in the developmental process, and they mention examples of it in physical traits studied in isogenic strains of animals raised under uniform conditions. It would as likely affect the structural and functional variance in neural networks as it does other anatomic features. They suggest, therefore, that some part of the variance classified as nonshared (or within-family) environmental variance cannot be traced to any exogenous effects. They write: "In our opinion, an important reason why the sources of these [nonshared environment] influences are still unknown is because a significant part of nonshared environmental influences may not be due to environmental differences at all, but result from intrinsic variability in the output of deterministic, selforganizing developmental processes" (p. 523). A similar view has been expressed by the Nobel laureate biochemist Gerald M. Edelman (1987):

As a result of the dynamic character of this model [of neural development], vast amounts of connectional variability will be found at all places in the nervous system, but particularly at the level of axonal and dendridic arbors in their finest ramifications. This insures individuality – while identical twins may have closer neuroanatomic structures than outbred individuals, it is predicted that they will nonetheless be found to have functionally significant variant wiring. (p. 323)

Our problem, then, is to try to get an empirical handle on the microenvironmental and epigenetic component of the WF nongenetic variance in IQ.

Intrapair IQ differences in MZT. Intrapair differences between monozygotic twins reared together (MZT) are probably the most direct measure of the WF environment. Any other kinship differences necessarily include genetic, GE covariance, and GE interaction effects, which together swamp the WF environmental effects. Because microenvironmental effects are hypothesized to be random, they cannot contribute to GE covariance, and their contribution to IQ variance therefore must be entirely nongenetic. Intrapair MZT differences consist exclusively of true WF nongenetic effects plus measurement error (e). The e must be taken into account, because it is a considerable part of the average intrapair difference, and we are really interested in the true-score differences.

The one possible disadvantage of using twins to measure environmental effects is that twins share the same uterine environment during gestation; this may present unique and unequal biological hazards, possibly increasing the intrapair differences in critical ways (Bulmer, 1970). Such effects, if unique to twins, would not add to the nongenetic differences between single-born children. The Oxford cytogeneticist C. D. Darlington (1954) argued that MZ twin differences overestimate environmental effects, because some of the difference is due to unequal division of the fertilized ovum, creating what Darlington terms cytoplasmic discordance and asymmetry.

These differences in the epigenetic landscape thus occur at the earliest stage of development. Their enduring differential effects on the twins are not genetic and are not really environmental but are probably best viewed as random biological noise – the "random somatic effects" mentioned by R. A. Fisher. Sometimes, there is even a marked inequality in placental blood supply, a condition peculiar to MZ twins. It also causes differences in development. The authors of a well-known twin study stated, "Such differences are neither genetic, in the ordinary sense, nor environmentally induced. In comparing the variability of identical [MZ] and fraternal [DZ] twins, therefore, it is not proper to consider all differences in identical twins reared together as environmentally determined" (Newman, Freeman, & Holzinger, 1937, p. 51).

The use of MZT for estimating the WF environmental variance, therefore, might overestimate the WF environmental variance in the population of singletons. However, these uniquely biasing prenatal factors in twin differences might be offset by the fact that MZ twins both share the same prenatal conditions and early environment on many variables, such as the mother's age, compatibility (or incompatibility) with the mother's blood group, health, parity, mother's medication before or during childbirth, and similarity of infant and early childhood experiences. Such variables probably contribute to the *unshared* environmental effects, or WFE variance, in the population of singletons.

Analysis of MZT intrapair differences in IQ. In the total literature on MZT, I have found ten studies in which all of the twins were tested on the Stanford-Binet, and the individual IQs were reported. They total 368 pairs, all school-age children (ranging in age from 5 to 16 years, with an average age of 10 years). Because of sampling differences, it would be statistically undesirable to pool all of these studies. Therefore, every sample was compared with every other sample to find out whether the means and variances of the IQ distributions differences and Bartlett's test for homogeneity of variances.

Six samples (from studies by Hirsch, 1930; Merriman, 1924; Stocks, 1930, 1933; Wingfield & Sandiford, 1928) did not differ significantly (p > .05) from each other and therefore can be treated statistically as samples from the same population. They comprise 180 pairs tested on the 1916 revision of the Stanford-Binet.⁶ The pooled sample (with total N = 360) has a mean IQ of 96.91 and *SD* of 15.78. (The mean IQ of twins is typically a few points below the population mean of approximately 100.)

The intraclass correlation (r_i) between the twins is .878, which differs little from the average $r_i = .88$ of the ten studies (totaling 661 pairs) of MZT based on Stanford-Binet IQs or the *N*-weighted average $r_i = .86$ of all existing studies of MZT (totaling 4,672 pairs [Bouchard & McGue, 1981]). The r_i is also the proportion of total shared variance, which, for MZT, consists of genetic variance, GE covariance and interaction, and shared, or BF, environmental influences. All the rest (i.e., $1 - r_i$) is WF environmental variance and error variance.

	f	cf	cIQ	cIQH	cIQL	cVIQ	cVIQH	cVIQL
0	18	180	96.91	99.76	94.06	249.12	242.78	239.22
1	24	162	97.34	100.51	94.17	260.06	249.26	250.80
2	14	138	96.70	100.33	93.07	263.12	253.07	246.81
3	20	124	97.09	101.02	93.16	274.68	260.44	258.07
4	17	104	96.95	101.35	92.56	254.97	237.86	233.46
5	19	87	97.16	102.02	92.30	271.84	249.63	246.76
6	15	68	97.83	103.35	92.31	286.70	253.99	258.42
7	9	53	98.90	105.13	92.66	290.09	241.47	260.94
8	8	44	99.07	105.86	92.27	330.06	271.03	296.74
9	4	36	97.39	104.81	89.97	343.13	283.88	292.36
10	3	32	97.63	105.41	89.84	345.23	278.37	291.01
11	2	29	97.10	105.17	89.03	360.27	291.38	298.93
12	3	27	96.48	104.74	88.22	379.03	310.19	311.43
13	5	24	97.08	105.63	88.54	361.95	285.23	292.75
14	0	19	96.18	105.26	87.11	370.52	287.98	288.20
15	4	19	96.18	105.26	87.11	370.52	287.98	288.20
16	1	15	98.17	107.67	88.67	337.07	238.76	254.89
17	3	14	97.96	107.57	88.36	355.96	255.67	271.66
18	1	11	96.09	106.00	86.18	319.81	216.73	226.51
19	5	10	95.50	105.50	85.50	339.85	235.65	244.05
20	2	5	92.90	103.40	82.40	283.69	170.24	176.64
21	1	3	94.17	105.00	83.33	369.47	244.67	259.56
22	2	2	83.00	94.00	72.00	125.00	4.00	4.00

Table 2.2. Distribution of MZT intrapair absolute differences inStanford-Binet IQ

|D|: absolute intrapair difference

- f: frequency
- cf: cumulative frequency
- cIQ: cumulative mean IQ (N = cf)

cIQH: cumulative mean IQ of higher-scoring twins

- cIQL: cumulative mean IQ of lower-scoring twins
- cVIQ: cumulative variance of IQ (N = cf)
- cVIQH: cumulative IQ variance of higher-scoring twins
- cVIQL: cumulative IQ variance of lower-scoring twins

Table 2.2 shows the frequency distribution of the absolute (i.e., unsigned) intrapair differences (|D|) in IQ, and the cumulative frequencies (cf) (going from the largest |D| of 22 IQ points to a |D| of zero), with the corresponding cumulative means and variances. Figure 2.4 shows the bivariate frequency distribution of the twins' IQs grouped in the class intervals 50–59, 60–69, and so on. What further information can we obtain from the statistics in Table 2.2 and Figure 2.3?

1. Given the total phenotypic variance $V_P = 249.12$ of IQs in the twin sample and the twin intraclass correlation $r_i = .878$, the *total* WF (i.e., within-

	IQ	60	70	80	90	100	110	120	130	140	150
	150										1
	140										
Ļ l	130								1		
Å. H	120							3	1	1	
<u>a</u>	110						10	6			
ų į	100					23	12	4			
ห่	90				25	24	3				
M	80			23	11	5					
ਮ	70		6	7	3			\mathbf{r}_{i}	= +	·. 87	'8]
	60	5	2	2					\ •		
	50	2									
	Σ	7	8	32	39	52	25	13	2	1	1

Higher-IQ Twin

Figure 2.4. Bivariate frequency distribution of lower-IQ and higher-IQ twins.

Note: The *intraclass* correlation, $r_i = +.878$, is the correlation between the twins regardless of their classification as higher or lower in IQ [or any other basis for classification]. The intraclass correlation is not the same as the Pearson correlation [or *interclass* correlation], r, between lower- and higher-IQ twins. The Pearson correlation between the lower-IQ and higher-IQ twins in the present sample is r = +.941. [Both interclass correlation and intraclass correlation are clearly explicated by R. A. Fisher (1970, pp. 213–49), the inventor of the intraclass correlation.]

pair) variance (including measurement error) is calculated as $V_{WFt} = V_P (1 - r_i) = 249.12 (1 - .878) = 30.4$. The *SD* of the WF IQs, then, is $(30.4)^{(0.5)} = 5.5$ IQ points. (This includes true-score environmental effects plus measurement error.)

2. The best estimate of the Stanford-Binet equivalent forms reliability in the age range and IQ level of the present sample is $r_{xx} = .93$ (McNemar, 1942, chapter 6). The twin correlation corrected for attenuation, then, is .878/.93 = .944. With the measurement error removed, we can estimate the WF environmental variance as $.93 \times 249.12(1 - .944) = 12.95$. So the *SD* of WF environmental effects is $(12.95)^{(0.5)} = 3.6$ IQ points. This may be compared with the total true-score *SD* of IQ, or $(.93 \times 249.12)^{(0.5)} = 15.2$ IQ points. All this can be most clearly presented in the typical form of an analysis of variance, as shown in Table 2.3.

The error variance is larger than the true-score WF variance in this sample. This is generally true in all of the studies of MZT reported in the

	U	ncorrected		Corrected for attenuation			
Source ^a	Variance	Percent	SD	Variance	Percent	SD	
BF	218.73	87.8	14.8	218.73	94.4	14.8	
WFE	12.95	5.2	3.6	12.95	5.6	3.6	
Error	17.44	7.0	4.2	0	0	0	
Total	249.12	100.0	15.8	231.68	100.0	15.2	

Table 2.3. Analysis of variance of MZT IQs

^{*a*}BF = between families (i.e., twin pairs).

WFE = within-families environment.

Note: Variances are additive; SDs are not additive.

literature. Given the weighted mean correlation of .86 based on all of the 34 published studies of MZT (totaling 4,672 pairs), and assuming that the average test reliability is .90, then the proportion of within-families environmental variance (V_{WFE}) is .04. If the population variance of IQ is $16^2 = 256$ (as on the Stanford-Binet), the V_{WFE} would be 10.24, and the *SD* of WF environmental effects would be $(10.24)^{(0.5)} = 3.2$ IQ points. By comparison, the *SD* of measurement errors are larger than WFE differences. The MZT intrapair differences in IQ attributable to WFE effects are almost swamped by measurement error. Unfortunately, for any given twin pair, we have no way of knowing how much of the intrapair IQ difference consists of measurement error and how much of true-score WFE effects. Yet we can make some theoretically informative inferences about the nature of the WFE from some further quantitative analyses of the total frequency distribution of intrapair differences (shown in the first two columns of Table 2.2).

Mean absolute difference $(|\overline{D}|)$. The model of random nongenetic effects with which the MZ twin data are to be compared posits that each twin's nongenetic deviation from the twin pair's common genotypic value is a normal random deviate, as would be expected for the distribution of many small and independent random effects. Therefore, the MZ twin differences, if they reflect random nongenetic effects and therefore conform to this model, should approximate the same distribution that would obtain for differences between pairs of values taken at random from a normal distribution. In the normal, or Gaussian, distribution there is an exact relationship between the σ and the mean absolute difference ($|\overline{\Delta}|$) between every pair of values in the distribution taken at random. The formula usually called *Gini's mean difference*, as given by the statistician Acardo Gini in 1914 (see Kendall & Stuart, 1977, pp. 48–9, 257), is $|\overline{\Delta}| = 2\sigma/\sqrt{\pi} = 1.1284\sigma$. Its standard deviation is $\sigma_{|\Delta|} \approx 0.8068\sigma$. The sample value of $|\overline{\Delta}|$ is signified by $|\overline{D}|$ and its *SD* by *SD*_{||D|}.

If, as hypothesized, all WF effects (i.e., both WFE and error) on IQ are random and therefore normally distributed, the theoretical values of $|\overline{\Delta}|$ and σ_{Δ} can be calculated from the *SD*s for WFE and Error shown in Table 2.3, using Gini's formulas. These theoretical values are as follows:

	Uncol	rrected	Corrected	
	$ \overline{\Delta} $	$\sigma_{i_{A_i}}$	IΔI	$\sigma_{\rm IAI}$
WFE	4.1	2.9	4.1	2.9
Error	4.7	3.4	0	0
WFE + Error	6.2	4.4	4.1	2.9

Because we cannot separate WFE effects from measurement error for each intrapair difference, we can only compare the uncorrected theoretical values of WFE + Error ($|\overline{\Delta}| = 6.2, \sigma_{|\Delta|} = 4.4, \text{CV}$ [coefficient of variation] = $\sigma/|\overline{\Delta}|$ = .7150) with the corresponding obtained values of all the twin differences in IQ, which are $|\overline{D}| = 5.70$, $SD_{|D|} = 5.37$, CV = .9421. These obtained values, calculated directly from all of the intrapair IQ differences, differ from the theoretical values based on Gini's formulas for $|\overline{\Delta}|$ and $\sigma_{|\Delta|}$ applied to the uncorrected SD of the combined WFE + Error = 5.51 [i.e., from Table 2.3: $(12.95 + 17.44)^{(0.5)} = 5.51$]. The standard deviations differ significantly ($F_{179,179}$ $= (5.37/4.44)^2 = 1.46, p < .05)$, and the CVs differ significantly (t = 2.80, df = 179, p < .01 [formula for standard error of the CV in Kendall & Stuart, 1977, p. 248]). This can only mean that the distribution of the obtained differences (|D|) is not consistent with the proposed working hypothesis that the differences result from random and normally distributed effects. And, as measurement errors are conventionally considered random and normally distributed, with $\mu = 0$, $\sigma_e = \sigma_x \sqrt{1 - r_{xx}}$, we must infer that a nonnormal distribution of WFE true-score effects is what causes the departure of the composite WFE plus error distribution from a normal distribution. The obtained distribution of |D| has a lower mean and a larger SD than theoretically expected because, compared to the theoretical distribution, there is an excess of very small |D| values having quite large frequencies (which lowers the overall mean of |D|) and an excess of large |D| values having relatively small frequencies (which increases the SD more than the mean). The nonnormal frequency distribution of |D| is most logically regarded as a composite of the normal distribution of measurement error and a nonnormal distribution of WFE effects. To infer the nature of the distribution of WFE effects on IQ, one has to try to read through the noise of measurement error. This requires a more detailed examination of the distribution of |D|.

The delta $|\Delta|$ distribution. The MZ twin intrapair difference |D| is the sum of each twin's deviation (d) from some value that both have in common. If that common value is the average of both twins, one twin's deviation is positive (+d), the other's is negative (-d), and their absolute values, |d|, are identical. Then, obviously, 2|d|, equals |D|, that is, the intrapair difference. In a group of twins, the mean of these intrapair deviations of course will be zero. (If the positive and negative deviations are normally distributed around the mean, their σ_d is given by a rearrangement of Gini's formula, viz., $\sigma_d = \frac{1}{2}|\overline{\Delta}|\sqrt{\pi}$.)

But the symmetry of +d and -d is merely a formalism, without any heuristic theoretical value or empirically testable implications. It would be theoretically more interesting to hypothesize that each twin's IQ is a deviation, not from the mean of both twins' IQs, but from their common genotypic value, whatever that may be. Unfortunately, we have no way of measuring any individual's (or any twin pair's) genotypic value. Over many twins, however, the means of each pair of twins are, on average, probably closer to their genotypic values than to any other values that could be directly calculated from the twin data. (Geneticists theoretically define genotypic value as the mean phenotypic value of all individuals with the same genotype.)

For any given pair of twins, the deviation of each twin's true-score deviation from the genotypic value, attributable to WFE effects, may be unequal. The higher-IQ twin, for example, could be less deviant from the genotypic value than is the lower-IQ twin (or vice versa). If this inequality were true more often than not in the twin population, the total distribution of twin deviations around their genotypic values would be nonsymmetrical and, ipso facto, nonnormal. Such asymmetry cannot be seen by direct examination of the distribution of |D| but must be inferred indirectly from certain statistics of the separate distributions of the higher-IQ and the lower-IQ twins from each pair. Certain other departures from normality, however, can be observed directly from a proper graph of the cumulative frequency distribution of |D|.

The absolute differences ($|\Delta|$) between all possible pairs of variate values in a unit normal curve ($\mu = 0$, $\sigma = 1$, z = a standardized deviation from μ) are distributed as the delta ($|\Delta|$) distribution. Its frequency distribution resembles the right-hand side of one-half of the normal curve, but it has different parameters.⁷ Some of the parameters of the $|\Delta|$ distribution are:

```
Range:0 to +\inftyMean:\mu_{i\Delta i} = i\overline{\Delta i} + 2/\sqrt{\pi} = +1.1284zStandard deviation:\sigma_{i\Delta i} \approx .8068zCoefficient of variation:CV = \sigma/\mu = .7150
```

These parameters are useful for comparison with the corresponding statistics of an empirical distribution of |D|, to determine its resemblance to a $|\Delta|$



Figure 2.5. A cumulative frequency distribution of the absolute intrapair IQ differences (|D|) in 180 pairs of MZ twins, plotted on normal probability paper. (The one open data point for |D| = 14 is an interpolation, as no twins in this sample have an intrapair difference of 14 IQ points. With this one exception, the values of |D| range continuously from 0 to 22 IQ points. Any single value of |D| is properly interpreted as falling within the class interval $|D| \pm 5$.)

distribution. If the distribution of all the intrapair twin differences in IQ very closely resembles a $|\Delta|$ distribution, it would be consistent with our working hypothesis that the WFE effects are normally distributed and therefore most likely the result of many small, random positive and negative environmental influences on IQ.

Figure 2.5 shows the percentiles of the cumulative frequency distribution of |D| (based on the column labeled cf in Table 2.2), plotted on a normal probability grid. The reason for this kind of plot is that if the distribution of effects (e.g., WFE + Error), from which the values of |D| arose, were randomly and normally distributed, all of the plotted data points should fall along a straight line, as would the theoretical $|\Delta|$ distribution. Any systematic and significant departure from the straight line indicates that the |D| values could not have been generated solely from random differences between normally distributed values. This outcome is apparent in Figure 2.5. The values of |D| fit a straight line in the range from 0 to 9, which comprises about 80% of the twin pairs. However, values of |D| larger than 9, which comprise about 20% of the twin pairs, depart significantly and systematically from a straight line. They are considerably larger than the |D| values predicted from the hypothesis that all values of |D| arise from normally distributed nongenetic effects on IQ.

The possibility that some same-sex DZ twins have been misclassified as MZ cannot be dismissed. (For DZ twins, $|\overline{D}| = 11.4$ IQ points.) This type of misclassification, however, has been found to be not more than about 3–4% with the method of zygosity diagnosis used in these early studies, which would amount to 6 or 7 DZ pairs included in the present sample of 180 pairs that were misdiagnosed as MZ.

To what extent is this departure of values of |D| > 9 from their expected values attributable to environmental effects and to errors of measurement? Errors are presumed to be normally distributed, so they should not deviate significantly from the straight line. McNemar (1942, chapter 6) has shown that the distribution of children's IQ (and mental age) differences between equivalent forms L and M of the Stanford-Binet conform almost perfectly to the theoretical $|\Delta|$ distribution throughout the full range of IQ. Deviations of the obtained values from the theoretical values are extremely small. When the properties of the $|\Delta|$ distribution were used to predict the equivalent-forms test-reliability coefficients based on the actual test-retest correlation, the predicted reliability coefficients have a mean absolute deviation of only .005 from the obtained reliability coefficients.

From this fact, it seems reasonable to infer that the distribution of twin differences, plotted as |D| in Figure 2.5, deviates from the hypothetical $|\Delta|$ distribution for |D| > 9, not because of measurement errors but because of the nonnormality of WFE effects. The nonnormality involves only the most extreme 20% of the twin pairs' |D| values, and this 20% could be evenly divided between the left and right tails of the nonnormal distribution (each tail with 10%), or it could be divided asymmetrically. A high degree of symmetry would mean that the extreme WFE effects are as frequently positive as negative; that is, large environmental effects would raise IQ as often as they lower it. Is this in fact what happens?

Before examining this question, we should take another look at the distribution of measurement errors for individuals who were tested with equivalent forms of the Stanford-Binet when the test-retest interval is a considerable period. Changes in IQ then reflect not only measurement error in the strict sense (or the complement of the internal consistency reliability) but also true-score developmental variation. In the course of mental development, as in physical development, children show lags and spurts in growth, which are partly environmental but also, we know, partly genetic, because MZ twins show higher concordance than DZ twins in the pattern of lags and spurts in their cognitive development (Wilson, 1974, 1983).

Thorndike, Fleming, Hildreth, and Stranger (1940) provide ideal data on which we can check the fit of Stanford-Binet IQ changes over a minimum test-retest interval of 2.5 years in 1,167 elementary-school children. The distribution of test-retest differences has a mean absolute difference $(|\overline{D}|)$ of 10.5 IQ points, and the test-retest correlation is +.65. I have plotted this |D| distribution on a normal probability grid as in Figure 2.5. All of the |D| values in Thorndike et al.'s test-retest data fall on a straight line, without the least suggestion of the kind of deviation from linearity seen in Figure 2.5.

This suggests that whatever environmental and genetic effects are reflected in IQ changes over a period of more than 2.5 years are normally distributed and could have resulted from many small, randomly distributed effects. The twin differences greater than 9 IQ points, on the other hand, do not fit this model. Some proportion of them deviate more than would be expected from the normal distribution of many small random effects. Because developmental changes in IQ during the elementary-school years do not show nonnormal effects, as Thorndike et al.'s data seem to indicate, it is a likely hypothesis that some part of the large nonnormal twin differences originated prenatally or before school age.

Another test of nonnormality of the twins' IQ deviations is to look at the distribution of IQ deviations (d) of each twin from the mean of each pair. This distribution of d, which ranges from -11 to +11, has a mean = 0, SD = 3.91. The distribution is, of course, necessarily symmetric about the mean, so it cannot be informative about skewness. It can, however, be informative about another possible index of nonnormality – namely, *kurtosis*. Kurtosis refers to the degree of peakedness or flatness of the distribution and is indexed by the ratio of the 2nd and 4th moments (μ_2 and μ_4) of the distribution, the measure of kurtosis being Pearson's $\beta_2 = \mu_4/\mu_2^2$. For the normal distribution, $\beta_2 = 3$. A $\beta_2 < 3$ indicates a *platykurtic* distribution; $\beta_2 > 3$ indicates a *leptokurtic* distribution. In our twin sample's distribution of d, $\beta_2 = 3.93$, which is very significantly (p < .001) greater than 3. So this d distribution is decidedly nonnormal. It is leptokurtic, which means that, compared to the normal curve, it has an excess of small absolute deviations (|d| > 5).

The small deviations are scarcely larger than would be expected from measurement error alone, when the reliability of the IQ is .93. For the whole distribution of |D| (as shown in Figure 2.5), the mean difference, $|\overline{D}|$,

is 5.70 (SD = 5.37). But assuming a test-retest reliability of .93 (with error variance = 1 - .93 = .07), the mean difference between repeated measures on the same persons (with the same total IQ variance [249.12] as in the present twin sample) would be 4.71 (SD = 3.37). The intraclass correlation (r_i) between the 80% of twins with |D| < 10 is .85; for twins with $|D| \ge 10$, the $r_i = .63$. Because the *SDs* of the IQs on which these two correlations are based differ considerably (15.11 and 18.50, respectively), the correlations should be corrected for this difference. When the correlations are thus corrected to a common *SD* equal to that of the whole sample (15.78), the r_i for twins with |D| < 10 is .86, and the r_i for twins with $|D| \ge 10$ is .57. This difference (.86 - .57 = .29) in the twin correlations implies that most of the WFE effects that are visibly larger than measurement error occur in only about 20% of the twin pairs – that is, those with $|D| \ge 10$ IQ points. The WFE effects in the 80% of twin pairs with |D| < 10 must be quite small.

It should be noted that the picture shown in Figure 2.5 is not peculiar to this set of MZ twin data. When other sets of MZT IQ data (Osborne, 1980; Rosanoff, Handy, & Plesset, 1937) and three combined studies (totaling 69 pairs) of MZ twins reared apart (Juel-Nielsen, 1965; Newman et al., 1937; Shields, 1962) were each plotted in the same fashion as in Figure 2.5, the same distinctive features of the plot shown in Figure 2.5 are seen in each of the other sets of twin data.⁸

Asymmetry of WFE effects. Are the 20% of twin differences that are large (i.e., $|D| \ge 10$) attributable to WFE effects that enhance IQ or depress IQ by equal amounts, on average, for the higher- and lower-IQ twins in each pair? Or do the higher- and lower-scoring twins reflect unequal effects of the WFE? The answer to this question may be revealed by looking for systematic differences between certain features of the IQ distribution of the higher-IQ (HIQ) members and of the lower-IQ (LIQ) members of each twin pair.

Michael Bailey and Joseph Horn (1986) were probably the first researchers to apply this strategy to MZ twin data. They reported a larger IQ variance for the LIQ than for HIQ twins, which led them to conclude that in MZ twin pairs, the LIQ twin reflects disadvantageous nongenetic effects. That is, the LIQ twin's phenotype deviates, on average, further below the pair's common genotypic value than the HIQ twin's phenotype deviates above it. Their finding at least suggests that the IQ distributions of the HIQ and the LIQ twins differ in ways other than their defining mean difference in IQ. Their distributions are in some way asymmetrical.

The difference in variances reported by Bailey and Horn, however, is subtle at best, and several other MZ twin studies do not consistently show the variance of IQL > variance of IQH. The Bailey and Horn variance ratios (i.e., F = LIQ variance/HIQ variance) based on five well-known MZ twin studies, are all larger than 1 (averaging 1.14), but only two of the five are significant. In our present twin data, including all levels of |D|, the F = 0.99, which seems not to replicate the Bailey and Horn finding. However, looking at the cumulative variances (cVIQH and cVIQL) of the HIQ and LIQ twins in the last two columns of Table 2.2, we see that in 18 out of 23 comparisons the VIQL > VIQH. Going from twin differences of 22 to 6 IQ points, the cumulative variances are consistently larger for the LIQ twins, but this trend reverses markedly when we add in the twins who differ by only 5 to 0 points. For twin pairs with $|D| \ge 6$, F = 1.02; for twins with $|D| \le 5$, F = 0.75. Besides their defining difference in |D|, twins with small intrapair differences appear to differ also in other ways from twins with large intrapair differences falls at a |D| of about 9 or 10 IQ points.

Bailey and Horn (1986) also noted that if there was a significant difference in the degree to which the IQs of the LIQ and the HIQ twins predicted |D|, it would suggest that one of the groups accounts for a larger part of the twin differences than the other group. They hypothesized that the IQs of the LIQ twins would show a larger correlation with |D| than would the HIQ twins. If this hypothesis were borne out, it would mean that the WFE effects that cause MZ twins to differ in IQ are larger (in a negative direction) for the lower-scoring twins than for their higher-scoring co-twins (in a positive direction). Probably the best way to look at this is to compare the correlation between |D| and the cumulative means of the HIQ twins with the correlation between |D| and the cumulative means of the LIQ twins (columns cIQH and cIQL of Table 2.2). This correlation for the HIQ twins is -.075; for the LIQ twins, the correlation is +.293. There is no proper test for the significance of this difference, although the direction of the difference is consistent with the Bailey and Horn hypothesis.

But, as previously noted, we are really dealing with two distinct distributions demarcated by |D| < 10 and $|D| \ge 10$. So we should look at the Bailey and Horn hypothesis separately within each distribution. For |D| < 10, the correlation between |D| and the cumulative means of the HIQ twins is +.678; the corresponding correlation for the LIQ twins is +.035. For $|D| \ge 10$, the correlation for HIQ twins is -.45; for LIQ twins, the correlation is -.76. (If the largest twin difference, |D| = 22, is regarded as an outlier and is omitted from the calculations, the correlations for $|D| \ge 10$ are -.05 for the HIQ twins and -.87 for the LIQ twins.)

The absolute size of these correlations is unimportant here. It is the *difference* between the correlations for the HIQ and LIQ twins that is most

informative. It shows that the intrapair IQ differences are not symmetrical, for if they were symmetrical, these correlations should be nearly the same. But we see that for twin pairs with |D| < 10, the HIQ twins' IQs predict |D| better than the LIQ twins' IQs do. And for twin pairs with $|D| \ge 10$, the LIQ twins' IQs twins' IQs predict |D| much better than the HIQ twins' IQs do.

Finally, to look at this phenomenon with greater statistical power than is afforded by the 180 twin pairs in the above analyses, I have analyzed MZT data from several studies totaling 1,435 twin pairs (studies by Hirsch, 1930; Merriman, 1924; Newman, Freeman, & Holzinger, 1937; Osborne, 1980; Rosanoff, Handy, & Plesset, 1937; Stocks, 1930, 1933; Wingfield & Sandiford, 1928.)

Because these studies used different IQ tests and the various samples are heterogeneous in means and SDs, it was necessary to standardize the twins' IQs and the intrapair differences within each study, scaling both variates as $(X - \overline{X})/SD = z$ scores, where X is an individual IQ (or an intrapair |D|) and \overline{X} is the sample mean IQ (or the sample $|\overline{D}|$), and SD is the sample standard deviation of each variate. With the twin data in each sample separately transformed from IQ to z_{10} and from |D| to z_{10} , the total of 1,435 MZT pairs in these samples then could be pooled. The z scores for IQ (z_{10}) were regressed on the z scores for intrapair difference (z_{1D1}) , separately for the HIQ and LIQ twins. (The regression coefficient for z scores is identical to r, the Pearson correlation coefficient.) If, at each level of $z_{(D)}$, the HIQ and LIQ twins' standardized IQs (z_{10}) in each pair differed, on average, equally (but in opposite directions) from the grand mean $\bar{z}_{IO} = 0$ of all the twins, then the regression coefficients (or correlations) of the HIQ and LIQ twins' z_{IO} on z_{DI} should not differ significantly in absolute size. (Of course, they necessarily have opposite signs.)

As it turns out, however, the correlations are +.14 for the HIQ twins and -.26 for the LIQ twins, as depicted in Figure 2.6. The difference between the two absolute values of r is highly significant (t > 4, df = 1,434, p < .001). The crucial point is that r_L is a significantly larger correlation than than r_H . This result can be interpreted as showing that whatever specific, or unshared, or within-family nongenetic effects (WFE) cause MZ twins to differ in IQ, these effects are more strongly negative than positive. That is, the lower-IQ twins are more disadvantaged than the higher-IQ twins are advantaged by whatever nongenetic factors make MZ twins differ in IQ. Apparently, the nongenetic influences on mental development are more frequently deleterious than they are advantageous. However, in the general population (excluding MZ twins), this possibility remains only an untested hypothesis. It would be exceedingly difficult to test this hypothesis in samples composed entirely of persons who differ in genotypes, including



Figure 2.6. The linear regression (or correlation, r) of the IQs of the higher-(H) and lower-(L) scoring twins in each pair on the absolute twin differences, |D|. Both IQ and |D| were transformed to z scores, with the H and L intercepts both set at 0.

dizygotic (DZ) twins, because of positive genotype \times environment covariance in IQ, which is a component of intrapair differences in DZ twins (and single-born siblings). Even if both genetic and environmental effects were perfectly symmetrical, on average, for DZ twins or siblings, the effects of GE covariance would not necessarily be symmetrical. Persons with different genotypes elicit and seek different environmental conditions, which may interact nonadditively with genetic effects.

Thus, differences due to GE covariance and differences due to WFE are confounded in DZ twin and sibling data. And for differences between genetically unrelated children reared together, there is no way to distinguish WFE variance from genetic variance or GE covariance.

On the other hand, there is little reason to believe that WFE effects are peculiar to MZ twins. Singletons are probably even more subject to such WFE effects than are MZ twins, because single-born siblings are exposed prenatally to differences in mother's age, parity, blood antigens, health, and other conditions that have an impact on development. Such conditions are the same for MZ twins but may differ markedly for singletons.

In summary, the results of these analyses of MZ twin differences in IQ are consistent with the hypothesis that for the vast majority (about 80%) of

twins (and probably singletons), the WFE variance results from many small and randomly distributed microenvironmental factors whose net effects in individuals are normally distributed. A minority of MZ twins (about 20%), however, show larger nongenetic deviations in IQ than can be assumed under this model of small, random microenvironmental effects. What could account for these more deviant individuals, of whom the downwardly deviant are the more affected? There are two likely possibilities, and both are probably true. The first is that in a minority of twins, one member of the pair encounters some exceptionally strong, or macroenvironmental, factor that has an impact on mental development. The second possibility is that an unusually unlucky combination of random microenvironmental factors may exceed a critical threshold in their phenotypic consequences such that a nonrandom stochastic process – a snowball effect – alters the trajectory of mental growth for better or (more often) worse.

Effects of the physical microenvironment on IQ

There is enough evidence of physical microenvironmental effects on mental development that this source of IQ variance cannot be ignored. The main reason that these effects have remained far in the background of research on intelligence is that, typically, each effect alone is so small as to be statistically insignificant and unrecognized, except in huge samples, which are rare. Also, in most studies, these effects are confounded with the much greater proportion of genetic variance and GE covariance.

Yet the physical microenvironment may well account for most of the specific or WF variance. Compared with the WF environmental variance to be explained, which is but a small percent (probably not more than about 5%) of the total IQ variance in MZT twins, the part attributable to all physical microenvironmental effects is large. It should be realized that any individual is not affected by more than some very small proportion of the total population of microenvironmental effects. In large samples, the physical factors with the strongest effects on IQ are so infrequent in the population that, in a random sample, each factor contributes a barely detectable increment to a multiple correlation with IQ. The effect has little chance statistically of being replicated in studies based on smaller-sized samples. Thus, it is highly likely that a significant correlation between IQ and a single physical variable found in one small-sample study will not replicate in another small-sample study. The significant correlation in the first study then is discarded as sampling error, and the variable in question escapes further investigation.

One example is seen in the famous study of 19 pairs of MZ twins reared apart (MZA), by Newman, Freeman, and Holzinger (1937). They found a

correlation of +.51 (p < .05) between intrapair absolute differences in Stanford-Binet IQ and intrapair absolute differences in fingerprint ridge count. The differences have to be nongenetic, of course, but whatever caused the intrapair differences in fingerprints must have occurred at some time during the first trimester of gestation, because fingerprints are fully developed by the fourth prenatal month. The set of twins (Gladys and Helen) with the largest intrapair IQ difference (24 points) showed by far the largest difference in fingerprints. Fingerprints usually differ between MZ twins no more than the fingerprints of a person's right and left hands. The larger MZ differences are due to epigenetic biological noise in embryonic development. Intrapair differences in MZ twins' palm-print ridge counts, attributable to developmental noise, are also significantly correlated with intrapair differences in certain personality traits measured by the MMPI (Rose, Reed, & Bogle, 1987).

On the other hand, the Minnesota Twin Study, with 48 MZA pairs, found a near-zero correlation between intrapair differences in fingerprints and IQs (Thomas Bouchard, personal communication). Small but significant effects that fail to replicate in small-sample studies are obviously hard to distinguish from Type I error. Feasible solutions are meta-analysis of the statistics from many studies, and analysis of mental measurements of MZ twins specially selected for much larger than the average intrapair differences either in IQ or in various physical characteristics.

In seeking clues to the nature and developmental timing of microenvironmental effects, MZ twin studies should correlate intrapair differences in a variety of physical variables with intrapair differences in IQ. Unfortunately, there have been few studies of this type. The results of one such study (Burks, 1940), based on 20 MZT twins, are shown in Table 2.4. The correlations are presented with their 95% confidence intervals; thus, three of the correlations in the first column are significant at p < .05. Note that twin intrapair differences in IQ, although the physical traits have little or no correlation with IQ (see the last column in Table 2.4). The intrapair differences in physical traits serve merely as signs of developmental noise.

Many physical conditions are correlated with IQ. Some of these were identified in the Collaborative Perinatal Project of the National Institute of Neurological Diseases and Stroke (Broman, Nichols, & Kennedy, 1975). This study, with nearly 27,000 subjects, reported the correlations of 169 prenatal, perinatal, and postnatal variables with the Stanford-Binet IQ at 4 years of age. Of the 169 variables, 32 are related to race, socioeconomic status, and family history. These contribute mainly to the between-families variance and usually involve genetic factors. Of the remaining 137 variables,

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	$.40 \pm .38$	$.98 \pm .03$.08
Iliace	.41 ± .38	.89 ± .12	11

Table 2.4. Correlation (and 95% confidence interval) of IQ and anthropometric measurements in monozygotic twins^a

^e Prepared from information in Burks (1940, pp. 89-90).

^bBased 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.

^dBased 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 $-.04 \pm .46$.

most are physical conditions that are environmental as far as the child is concerned; that is, they are not causally related to the child's genotype.

Some of these 137 conditions can be considered aspects of the WF environmental variance. I have classified them into five categories. Within each category, I have tabulated the total number of variables, the number significantly (p < .001) correlated with IQ at age 4, and the number of these that can be strictly regarded as a within-family environmental effect (WFE), in that it is unlikely to involve genetic factors and could differ between single-born siblings reared together. This tabulation is shown in Table 2.5. Also given is the variables' mean absolute (unsigned) zero-order correlations with IQ for correlations that are significant at p < .001.

The average correlations are quite small, but it should be remembered that each of these physical environmental variables alone affects only some fraction of the population; the increments or decrements in IQ could be considerable for the affected individuals. Also, different individuals are "hit" by different microenvironmental elements and some individuals are "hit" more or less often than others. Thus, even common but singly minute effects can accumulate randomly, with a substantial net effect on IQ for some individuals. When all these variables are combined in a multipleregression equation to predict IQ, they account for about 4 percent of the

		Type of variable								
	Maternal	Prenatal	Labor and delivery	Neonatal	Infancy and childhood					
Total	19	26	30	25	37					
p < .001	13	17	14	13	28					
WFE	8	3	2	0	2					
Mean r	.070	.085	.064	.066	.030					

Table 2.5. Tabulation of physical conditions correlated with IQ in the National Collaborative Prenatal and Perinatal Project and the mean absolute correlation (|r|, p < .001) in each category

Source: Based on Appendix 4, table 1, in a report by Broman, Nichols, & Kennedy, 1975.

total IQ variance. But this is scarcely less than the total WFE percent of the variance, about 5-6% as estimated from the disattenuated MZT correlation.

Some specific within-family microenvironmental effects

Maternal and prenatal factors. As the geneticist Geoffrey Ashton (1986) has stated, "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). When the gene at one (or more) of the chromosomal loci that controls a particular physical characteristic has identical alleles,9 such genes are called homozygous. Ashton's research, based on genetic markers (blood antigens) for 18 chromosomal loci, found that homozygosity is associated with higher scores on verbal and spatial tests. The genotype of a more homozygous fetus, having less intraindividual genetic variation, is less likely to be incompatible with the mother's genotype. Thus, the mother's greater immunological tolerance protects the more homozygous fetus from the developmental damage that could otherwise result from its antigenic incompatibility with the mother if the fetus were more heterozygous.

Effects of the Rhesus (Rh) blood antigen are well known. When the mother is Rh-negative and the fetus is Rh-positive (as happens when the

father is Rh⁺), the mother builds up antibodies that attack the Rh⁺ fetus's red blood cells. The effects of the maternal antibodies are so subtle in the first pregnancy as to go undetected, but the antibodies continue to build up in subsequent pregnancies, with serious consequences for the developing fetus, sometimes including stillbirth. Some 7-8% of pregnancies of Rhnegative mothers are at risk for Rh incompatibility. The average (negative) effect on IQ in this group is about -6 IQ points; children born in later ordinal positions show the greater effects (Costiloe, 1969). Children of unknown blood type whose mothers are Rh-negative average about onehalf an IQ point lower than children whose mothers are Rh-positive, a statistically significant effect (Mascie-Taylor, 1984). Fortunately, since the 1970s, it has been possible to make Rh-negative mothers immune to the Rh factor by vaccination with a blood extract called Rh immune globulin. Its widespread use and the gradual decrease in family size are probably among the many causes of the secular rise in IQ in industrialized countries in recent decades (Flynn, 1987).

But Rh is not the only blood antigen incompatibility that accounts for some fraction of the variance in IQ and other behavioral traits. The ABO blood groups of the mother are also correlated with IQ, although to a lesser degree than the Rh factor (Broman et al., 1975; Mascie-Taylor, 1984). These are only a few of the many blood antigens and other polymorphisms that could affect the developing brain because of mother-fetus incompatibility. Immunoreactive factors have also been invoked to explain the greater incidence of developmental disorders in the male fetal brain, as a male fetus is antigenically apt to be less compatible with the mother than a female fetus (Gaultieri & Hicks, 1985).

Other *prenatal* factors that are correlated (negatively) with IQ are the mother's age, parity (i.e., number of prior pregnancies), X-ray exposure during pregnancy, the mother's smoking or excessive use of alcohol or drugs during pregnancy, fever during pregnancy, a shorter or longer than normal period of gestation, maternal diabetes, and placental abnormalities (Broman et al., 1975; Mascie-Taylor, 1984). When a placental abnormality is present, the difference in IQ between MZ twins is related to whether they are monochorionic or dichorionic (Melnick, Myrianthopoulos, & Christian, 1978). (The chorion is the outer embryonic membrane.) Lower birth weight is related to lower IQ. That the effect is not genetic is shown by the fact that the MZ twin with the lower birth weight usually has a lower IQ at school age, a result explained by the twins' unequal sharing of nutrients during gestation (Churchill, Neff, & Caldwell, 1966; Scarr, 1969; Willerman & Churchill, 1967).

Several *perinatal* factors are correlated (negatively) with IQ. Anoxia at birth, usually a result of premature separation of the placenta from the

uterus, can have a drastic effect on early psychomotor development, but the deficit in IQ generally diminishes throughout childhood, averaging about 3 IQ points by 7 years of age (Corah, Anthony, Painter, Stern, & Thurston, 1956). Mother's pelvic size and the fetus's head position during delivery (Willerman, 1970a,b) and breech delivery (Broman et al., 1975) are also related to the child's later IQ.

The purported negative relation between *birth order* and IQ has often been regarded as a clear-cut example of a within-family environmental effect. But even the existence of any birth order effect on IQ, when all the methodological artifacts in this research are controlled, has been critically questioned in the most thorough review of the evidence available (Ernst & Angst, 1983). The birth-order effect claimed by some researchers accounts for at most about 2–3% of the total variance in IQ. The only purely psychological theory of the birth-order effect on mental development is the socalled *confluence theory* of Robert Zajonc (1976). This model, however, has been found decisively faulty (Retherford & Sewell, 1991), and other explanations not involving causal factors of a psychological nature better explain the data (Page & Grandon, 1979).

What little effect birth order may have on IQ is perhaps best explained by the increasing probability in successive pregnancies of maternal immune attack on the fetal brain. For example, the relative frequency of type AB blood increases with birth order, suggesting an increasing mother-fetus incompatibility and spontaneous abortion of fetuses with the other blood types in the ABO system. At least 25% of all conceptuses are spontaneously aborted, usually in the early stage of pregnancy. Spontaneous abortion is a critical threshold on a continuum of causal factors, which at subthreshold levels may result in subtle forms of disadvantage that contribute a part of the WFE variance. A considerable body of evidence has been adduced in support of this immunoreactive theory of the effect of birth order on IQ (Foster & Archer, 1979). A decrease in immunoreactive effects because of the gradually decreasing family size in all industrialized countries over the last three generations might account for some part of the secular rise in IQ during this period (Flynn, 1987).

One of the most striking *postnatal* environmental variables found to affect IQ is whether the infant is given breast milk or a formula. In a large (N = 300) and methodologically exemplary study in Cambridge, England, children born preterm (under 1,850g at birth) were fed by tube with either breast milk or a preterm formula. The neonates in both groups were well matched for birth weight, gestation, and other medical variables. The mothers' social class, education, family structure, and other potentially confounding factors were statistically controlled. The experiment continued in the hospital under professional supervision until the babies were discharged

or had reached 2,000g body weight. At 7.5 to 8 years of age, the children who had received breast milk scored, on average, 8.3 IQ points higher on the WISC than those who had received a formula, a difference significant beyond the .0001 level of confidence (Lucas, Morley, Cole, Lister, & Leeson-Payne, 1992). The authors explain this result in terms of nutritional factors that affect brain development and are present only in mothers' milk.

Many other physical health-related factors are probably correlated with IQ, but these have not yet been studied in detail. Lubinski and Humphreys (1992) found that medical and physical well-being are considerably above the norm in the mathematically gifted; they are more highly associated with giftedness even than extreme levels of socioeconomic privilege. It is also likely that common childhood diseases, such as whooping cough, measles, mumps, and chicken pox, could each take a toll on IQ, perhaps of one IQ point. Inoculation against these diseases would prevent this negative effect. As inoculation is a mild induction of the disease that stimulates the body's immune system, it might also favorably affect physical growth, including brain development. Some part of the gradual secular rise in IQ over the past three generations could be attributable to such factors, which, along with improved nutrition, have become widespread in industrialized countries.

Summary

From an evolutionary standpoint, the genetic inheritance, or innateness, of fitness characteristics is essential. The evolutionary process has ensured normal development to the vast majority of every species by biologically programming the ontogeny of their crucial characteristics, at the same time maintaining enough genetic diversity in certain traits for adaptation to changing environmental conditions. In humans, intelligence and the ability to learn are such characteristics. An overly plastic nervous system, with its functions shaped too easily by the environment, would put the organism's adaptive capacity at risk of being wafted this way or that by haphazard experiences. A half-century of research in physical anthropology and behavioral genetics supports the idea that general mental ability, or g, is a fitness trait with increasing cybernetic stability during its course of development. As argued by Moffitt, Caspi, Harkness, & Silva (1993), it is elastic rather than plastic in its temporary deviations from its biologically programmed trajectory. Genetic variance, genotype-environment covariance, and $G \times E$ interaction are the major components of g variance. The variance attributed to shared, or between-families, environmental factors, which is considerable throughout childhood, gradually shrinks to near-zero between early adolescence and maturity. During this period, most of the environmental variance is converted into genotype-environment covariance, as

persons elicit, seek, select, and modify those elements of the available cognitive-social-cultural milieu that are most compatible with their genotypically conditioned proclivities.

After such sources of IQ variance have been accounted for, psychologists generally try to explain the one remaining source of variance – the specific, or within-family environment (WFE) – in wholly psychological terms of social learning and possible differences in opportunities and motivation that may exist among full siblings who are reared together. Environmental variables of a biological nature are most often slighted. Yet the physical-biological microenvironment might well contribute most of the WFE variance in g.

According to the microenvironmental theory of WFE, the neural basis of mental development is affected in each individual by a limited number of physical events beginning shortly after conception, each with a biologic effect usually too small to be detected individually. Their reliably detectable effects result from their aggregation in some individuals. These small biologic events are a random selection from among all such microenvironmental events that may affect development. Because they "hit" individuals more or less at random, they vary in both number and kind for different individuals. The net effects of these small, independent physicalenvironmental influences for individuals are deviations (positive and negative) of individuals' phenotypic IQs from their genotypic values. The statistical properties of these deviations can be inferred from the intrapair IQ differences between MZ twins. Because the net deviations have resulted from many small, independent events, they are normally distributed in the population.

Superimposed on this normal distribution of random environmental effects on IQ is a distribution resulting from a small number of comparatively large environmental effects, more often negative than positive, that "hit" only a fraction of the population. They are attributable to (1) a nonrandom, stochastic snowball effect on a few unlucky individuals who by chance have received a critical preponderance of unidirectional small effects, which increases the likelihood of incurring still more effects in the same direction; and (2) the occurrence of rare events with large effects that "hit" only a small fraction of the population. The composite of these two distributions of net environmental effects forms a population distribution that is leptokurtic, with excess frequencies in the two tails, especially in the tail on the negative side. This, then, is the form of the distribution of phenotypic IQ deviations that behavior-genetic models attribute to the specific, or within-family, environment.

A host of nongenetic but biologic factors – prenatal, perinatal, and early postnatal – are known to affect mental development, each factor alone

having only a small effect. But the number of these presently known factors is probably only a fraction of all the biologic factors that affect mental growth. The additive and interactive effects of their random combinations probably accounts for most of the g variance ascribed to WFE. If so, it makes more understandable the notably unsuccessful efforts of researchers to produce any bona fide evidence that g can be significantly and lastingly raised by any purely psychological or educational means (Detterman & Sternberg, 1982; Jensen, 1989; Spitz, 1986). Because g reflects individual differences mainly in the neural mechanisms of information processing, it is more susceptible to biological than to psychological influences.

The secular increase of IQ in industrialized countries over the past three generations can be attributed in part to the widely increased availability of improved health care, nutrition, obstetrical advances, and other factors with biologic effects on mental growth. In First World countries, such benefits have almost universally minimized a significant portion of the microenvironmental factors that negatively affect mental development.

In the picture we see emerging from behavior-genetic analyses of mental abilities, the psychometric construct called g appears to be a biological phenomenon with many behavioral correlates, including performance on IQ tests. Some of these correlates are trivial, except as knowledge of them may help to advance understanding of the nature of g. However, g has correlates of great significance in their own right. The phenomenon represented by g is an undoubtedly crucial factor in understanding individual differences in many educationally, economically, and socially important variables.

Notes

1. It is important to distinguish between genotype-environment (GE) *covariance* (or correlation, which is simply the standardized covariance) and genotype-environment *interaction*. They are entirely different concepts, but each may account for some part of the phenotypic variance in a trait.

GE covariance is the result of the nonrandom occurrence of different genotypes in different environments. In other words, genotypes and environments may be correlated. Persons whose genotype is favorable for the development of a certain trait (e.g., musical talent) are more likely than chance to grow up in an environment that is favorable to the development of the trait (e.g., parents with musical interests, opportunity for music lessons, etc.). The correlation of genotypes and environments for a given trait in the population increases the phenotypic variance over what it would be if the correlation were zero. Assuming for simplicity that there is no GE interaction, the total phenotypic (P) variance (V) is the sum of the genetic (G) variance and the environmental (E) variance *plus* twice the covariance (Cov) of G and E, or, as it is expressed in biometrical genetics, $V_P = V_G + V_E + 2CovGE$. (Regarding CovGE = $r_{GE}\sqrt{V_G}\sqrt{V_E}$, note that CovGE depends on there being substantial values of V_G and V_E ; if either one is zero, there can be no GE covariance, or

correlation either, because GE correlation depends on variance in both genetic and environmental effects.)

GE interaction is a component of the phenotypic variance that is due to different genotypes reacting differently to the same environmental condition. That is, an environmental condition that favors the phenotypic development of individuals who have genotype A may have no effect, or may even have an unfavorable effect, on individuals who have genotype B. A classic example is a condition known as galactosemia. Most infants thrive on milk, but a small number have a genotype that prevents their normally metabolizing milk, and the abnormal metabolites damage the infant's brain, resulting in severe mental retardation. Another example: A pair of orphaned monozygotic twins (hence, identical genotypes) separated in infancy, one reared by a very unmusical family, the other by an intensely musical family; neither twin even shows any sensitivity to music or develops any interest in it. Another pair of MZ twins in the identical circumstances shows a very different outcome: The twin reared in the unmusical family shows little sensitivity or interest in music, while the twin reared in the musical family turns out to be a highly accomplished musician. One set of twins (i.e., one genotype) is insensitive to the musical environment, and the other set (i.e., another genotype) is highly sensitive to a musical environment if exposed to music. The phenotypic variance in musicality among these two sets of twins would have a large component of GE interaction.

- 2. The statement "the variance *between* families is the covariance *within* families" is most easily explained in terms of the analysis of variance and its relation to the intraclass correlation. If we perform a simple one-way analysis of variance on a population of persons grouped in families, we arrive at three variances: the Between-Families variance (V_{BF}) , the Within-Families variance (V_{WF}) , and the Total variance $(V_T = V_{BF} + V_{WF})$. The correlation between persons *within* families is the *intraclass correlation*, which is $r_i = V_{BF}/V_T$. But a correlation coefficient is just a standardized covariance – that is, a covariance divided by the total variance. So if we multiply the intraclass *correlation* (between the persons within families) by the total variance, we have the *covariance* between family members, which is equal to V_{BF} . Therefore, the variance between families is the covariance within families.
- 3. One rarely sees corrections for attenuation in behavior-genetic literature, although it is often called for when the aim is to estimate components of variance that certainly comprise some variance due to measurement error, which is unique to the particular measuring instrument and is of no theoretical interest. Surely if error-free measurements of the variables of interest were available, investigators would prefer to use them. Although there are problems with the correction for attenuation, such as the reliability of the reliability coefficient itself, it is still possible to more closely approximate the true-score variance components by correction for attenuation than by not correcting at all; noncorrection, in effect, assumes perfect reliability, which we know is impossible. We cannot achieve perfection with the correction for attenuation, but we can come somewhat nearer to the error-free values of the correlations and variance components if we do make the correction, provided we have a reliable reliability coefficient. Therefore, I consider it preferable in the present analysis to correct for attenuation, using a "best estimate" of the measurement's reliability coefficient. The best estimate for intelligence tests, based on the standardization data of a variety of individual and group tests, is .90 (see Jensen, 1980, chapter 7). (Specifically for the Stanford-Binet, beyond age 6, the best estimate is .93.) The most relevant reliability is test-retest or equivalent forms reliability rather than internal consistency (Kuder-Richardson) reliability, although these two conceptually distinct types of reliability are usually of comparable magnitude.
- 4. By psychosocial-cultural, I mean environmental influences that arise from the individual's subjective waking experiences that involve personal interactions, identification with role

models, learning opportunities, language, customs, parental and peer demands, and values and interests acquired in the individual's environment. By *biological* or *physical* environmental influences, I refer to factors, both endogenous and exogenous, that directly impinge on and affect the individual's anatomy and physiology, or that directly affect the physical growth process of any organ system, particularly the nervous system. By *directly affect*, I mean that conditioning, learning, and awareness are not the agencies of the influence. Examples of physical influences on an individual are the mother's health and nutrition during the individual's prenatal development, perinatal anoxia, childhood diseases, malnutrition, brain injury, hormonal imbalance, sensory defects, and the like. Other physical environmental effects are mentioned later on in this chapter.

- 5. A polygenic trait is one whose genetic variance is contributed by genes at two or more chromosomal loci and for which all the genes have small and more or less equal effects, whether their alleles have additive effects, or are interactive within the same locus (i.e., dominance) or between different loci (i.e., epistasis). Intelligence is a polygenic trait; the number of genes involved in IQ variance has been variously estimated in the genetics literature at between 20 and 100, although these numbers are not taken very seriously, they represent reasonable limits within which the true value probably falls.
- 6. The question arises of whether a sample size of 180 MZ twin pairs can afford the statistical power needed for the analysis of WF variance, which is a small proportion (about .10) of the total phenotypic variance. Neale and Cardon (1992, chapter 9) point out that enormously larger samples of twins than that used here are needed to estimate certain small variance components (e.g., the shared environmental variance) in genetic analyses with a satisfactory degree of statistical confidence, such as p < .05. The main concern of the present analysis, however, is not with the estimation of the relative size of the BF and WF environmental components of variance (which in any case would not be possible using only MZ twins reared together) but with the form of the distribution of IO differences between MZ twins and whether this distribution conforms to the distribution of effects that are predicted from a model of environmental effects that are purely random. The twin data show that certain statistics of the obtained distribution of twin differences depart from the corresponding parameters (σ and CV = σ/μ) of the theoretical distribution predicted by the random effects model at p < .05 and p < .01, respectively. Also, the highly significant (p < .01) .001) asymmetry of the lower- and higher-scoring twins (Figure 2.6) contradicts the randomeffects model.
- 7. The right-hand (+z) half of the normal curve has the following parameters, where z is a standardized deviate (x/σ) :

Range:0 to + ∞ Mean: $\mu_{+z} = \sqrt{2}/\sqrt{\pi} = +.79788z$ Standard deviation: $\sigma_{+z} = \sqrt{1/2} = .7071z$ Coefficient of variation: $CV = \sigma/\mu = .8862$

- 8. It is noteworthy that the same distinctive features seen in Figure 2.5 are also found when such a plot is performed on Burt's (1966) reputed data on 53 pairs of MZ twins reared apart (MZA), with their IQ correlation of .771. Since 1976, Burt's MZA results have been excluded from all meta-analyses in behavior genetics because of their questioned authenticity. Yet there is no evidence in Burt's publications that the idea of plotting twin differences in this fashion had ever occurred to him. If he had faked his MZA data, as alleged by his detractors, he would have to be credited with clairvoyant intuition.
- 9. Every normal person's 46 chromosomes come in 23 identifiable homologous pairs, one chromosome from each parent. The genes, each at different loci on a chromosome (like beads on a string) control the production of enzymes, which in turn affect the development of all of the body's physical structures and functions. Many genes (called *polymorphic* or *segregating genes*) have two or more forms, called *alleles* (or *allelomorphs*), which all have somewhat different developmental effects on the same system. When a gene at a particular

chromosomal locus has identical alleles (e.g., AA or aa, instead of Aa) in the two chromosomes, it is said to be *homozygous*. When the alleles of a gene at a particular chromosomal locus are different (e.g., Aa instead of AA or aa) in the two chromosomes, the gene is said to be *heterozygous*.

References

- Ashton, G. C. (1986). Blood polymorphisms and cognitive abilities. *Behavior Genetics*, 16, 517–29.
- Bailey, J. M., & Horn, J. M. (1986). A source of variance in IQ unique to the lower-scoring monozygotic (MZ) twin. Behavior Genetics, 16, 509-16.
- Bouchard, T. J., Jr., Lykken, D. T., McGue, M., Segal, N., & Tellegen, A. (1990). Sources of human psychological difference: The Minnesota study of twins reared apart. *Science*, 250, 223–8.
- Bouchard, T. J., Jr., Lykken, D. T., McGue, M., Segal, N. L., & Tellegen, A. (1991). IQ and heredity. *Science*, 252, 191–2.
- Bouchard, T. J., Jr., & McGue, M. (1981). Familial studies of intelligence: A review. *Science*, 212, 1055–9.
- Bouchard, T. J., Jr., & Segal, N. L. (1985). Environment and IQ. In B. B. Wolman (Ed.), Handbook of intelligence (pp. 391-464). New York: Wiley.
- Broman, S. H., Nichols, P. L., & Kennedy, W. A. (1975). Preschool IQ: Prenatal and early developmental correlates. Hillsdale, NJ: Erlbaum.
- Bulmer, M. G. (1970). The biology of twinning in man. Oxford: Clarendon.
- Burks, B. S. (1940). Mental and physical developmental patterns of identical twins in relation of organismic growth theory. In G. W. Whipple (Ed.), *Intelligence: Its nature and nurture* (Part II, 399th Yearbook of the NSSE). Bloomington, IL: Public School Publishing Co.
- Burt, C. (1966). The genetic determination of differences in intelligence: A study of monozygotic twins reared together and apart. British Journal of Psychology, 57, 137-53.
- Cardon, L. R., Fulker, D. W., & Plomin, R. (1992). Multivariate genetic analysis of secific cognitive abilities in Colorado Adoption Project at age 7. *Inelligence*, 16, 383-400.
- Chipuer, H. M., Rovine, M. J., & Plomin, R. (1990). LISREL modeling: Genetic and environmental influences on IQ revisited. *Intelligence*, 14, 11–29.
- Churchill, J. A., Neff, J. W., & Caldwell, D. F. (1966). Birth weight and intelligence. *Obstetrics* and Gynecology, 28, 425-9.
- Corah, N. L., Anthony, E. J., Painter, P., Stern, J. A., & Thurston, D. (1956). Effects of perinatal anoxia after seven years. *Psychological Monographs*, 79, No. 3, 1–34.
- Costiloe, T. M. (1969). Ordinal position in sibship and mother's Rh status among psychological clinic patients. *American Journal of Mental Deficiency*, 74, 10-16.
- Darlington, C. D. (1954). Heredity and environment. Proceedings of the 9th International Congress of Genetics, 9, 370-81.
- Detterman, D. K., & Sternberg, R. J. (Eds.) (1982). How and how much can intelligence be increased. Norwood, NJ: Ablex.
- Edelman, G. M. (1987). Neural Darwinism: The theory of neuronal group selection. New York: Basic Books.
- Ernst, C., & Angst, J. (1983). Birth order: Its influence on personality. Berlin: Springer-Verlag.
- Fisher, R. A. (1918). Correlation between relatives on the supposition of Mendelian inheritance. *Transactions of the Royal Society of Edinburgh*, 52, 399–433.
- Fisher, R. A. (1970). Statistical methods for research workers (14th ed.). New York: Hafner.
- Flynn, J. R. (1987). Massive IQ gains in 14 nations: What IQ tests really measure. Psychological Bulletin, 101, 171–91.
- Foster, J. W., & Archer, S. J. (1979). Birth order and intelligence. *Perceptual and Motor Skills*, 48, 79–93.

- Galton, F. (1908/1974). Memories of my life. New York: AMS Press.
- Gaultieri, T., & Hicks, R. E. (1985). An immunoreactive theory of selective male affliction. Behavioral and Brain Sciences, 8, 427-41.
- Hirsch, N. D. M. (1930). Twins: Heredity and environment. Cambridge: Harvard University Press.
- Jensen, A. R. (1987). The g beyond factor analysis. In J. C. Connoly, J. A. Glover, & R. R. Ronning (Eds.), *The influence of cognitive psychology on testing and measurement*. Hillsdale, NJ: Erlbaum.
- Jensen, A. R. (1989). Raising IQ without increasing g. Developmental Review, 9, 234-58.
- Jensen, A. R. (1992). Commentary: Vehicles of g. Psychological Science, 3, 275-8.
- Jensen, A. R. (1993). Psychometric g and achievement. In B. R. Gifford (Ed.), Policy perspectives on educational testing (pp. 117–227). Boston: Kluwer.
- Jinks, J. L., & Fulker, D. W. (1970). Comparison of the biometrical, genetical, MAVA, and classical approaches to the analysis of human behavior. *Psychological Bulletin*, 73, 311–49.
- Juel-Nielsen, N. (1965). Individual and environment: A psychiatric-psychological investigation of MZ twins reared apart. Acta Psychiatrica Scandinavia, Supplement 183. Cophenhagen: Munksgaard.
- Kendall, M., & Stuart, A. (1977). *The advanced theory of statistics* (4th ed.) (Vol. 1). New York: Macmillan.
- Longstreth, L. E., Davis, B., Carter, L., Flint, D., Owen, J., Richert, M., & Taylor, E. (1981). Separation of home intellectual environment and maternal IQ as determinants of child IQ. Developmental Psychology, 17, 532–41.
- Lubinski, D., & Humphreys, L. G. (1992). Some bodily and medical correlates of mathematical giftedness and commensurate levels of socioeconomic status. *Intelligence*, 16, 99–115.
- Lucas, A., Morley, R., Cole, T. J., Lister, G., & Leeson-Payne, C. (1992). Breast milk and subsequent intelligence quotient in children born preterm. *Lancet*, 339, 261-4.
- Luo, D., Petrill, S. A., & Thompson, L. A. (1994). An exploration of genetic g: Hierarchical factor analysis of cognitive data from the Western Reserve Twin Project. *Intelligence*, 18, 335–47.
- Lykken, D. T., McGue, M., Tellegen, A., & Bouchard, T. J., Jr. (1992). Emergenesis: Traits that may not run in families. *American Psychologist*, 47, 1565-77.
- Mascie-Taylor, G. C. N. (1984). Biosocial correlates of IQ. In C. J. Turner & H. B. Miles (Eds.), *The biology of human intelligence*. Proceedings of the 20th Annual Symposium of the Eugenics Society, London.
- McGue, M., Bkouchard, T. J., Iacono, W. G., & Lykken, D. T. (1993). Behavioral genetics of cognitive ability: A lifespan perspective. In R. Polomin & G. E. McClearn (Eds.), *Nature, nurture & psychology*. Washington, DC: American Psychological Association.
- McNemar, Q. (1942). The revision of the Stanford-Binet Scale. Boston: Houghton Mifflin.
- Meehl, P. E. (1978). Theoretical risks and tabular asterisks: Sir Karl, Sir Ronald, and the slow progress of soft psychology. *Journal of Consulting and Clinical Psychology*, 46, 806– 34.
- Melnick, M., Myrianthopoulos, N. C., & Christian, J. C. (1978). The effects of chorion type on variation in IQ in the NCPP twin population. *American Journal of Human Genetics*, 30, 425-433.
- Merriman, C. (1924). The intellectual resemblance of twins. *Psychological Monographs*, 33, 1–49.
- Moffitt, T. E., Caspi, A., Harkness, A. R., & Silva, P. A. (1993). The natural history of change in intellectual performance: Who changes? How much? Is it meaningful? *Journal of Child Psychology and Psychiatry*, 14, 455–506.
- Molenaar, P. C. M., Boomsma, D. I., & Dolan, C. V. (1993). A third source of developmental differences. *Behavior Genetics*, 23, 519–24.
- Neale, M. C., & Cardon, L. R. (1992). Methodology for genetic studies of twins and families. Boston: Kluwer.

- Newman, H. H., Freeman, F. N., & Holzinger, K. J. (1937). Twins: A study of heredity and environment. Chicago: University of Chicago Press.
- Osborne, R. T. (1980). Twins: black and white. Athens, GA: Foundation for Human Understanding.
- Page, E. B., & Grandon, G. M. (1979). Family configuration and mental ability: Two theories contrasted with U.S. data. American Educational Research Journal, 16, 257–72.
- Pedersen, N. L., Plomin, R., Nesselroade, J. R., & McClearn, G. E. (1992). A quantitative genetic analysis of cognitive abilities during the second half of the life span. *Psychological Science*, 3, 346–53.
- Plomin, R. (1986). Development, genetics, and psychology. Hillsdale, NJ: Erlbaum.
- Plomin, R. (1988). The nature and nurture of cognitive abilities. In R. J. Sternberg (Ed.), Advances in the psychology of human intelligence (Vol. 4). Hillsdale, NJ: Erlbaum.
- Plomin, R., & Bergeman, C. S. (1991). The nature of nurture: Genetic influences on "environmental" measures. *Behavioral and Brain Sciences*, 14, 373-427.
- Plomin, R., & Daniels, D. (1987). Why are children in the same family so different from one another? *Behavioral and Brain Sciences*, 10, 1–60.
- Plomin, R., DeFreies, J. C., & Loehlin, J. C. (1977). Genotype-environment interaction and correlation in the analysis of human behavior. *Psychological Bulletin*, 84, 309-22.
- Plomin, R., & Neiderhiser, J. M. (1992). Genetics and experience. Current Directions in Psychological Science, 1, 160-3.
- Retherford, R. D., & Sewell, W. H. (1991). Birth order and intelligence: Further tests of the confluence model. *American Sociological Review*, 56, 141-58.
- Rosanoff, A. J., Handy, L. M., & Plesset, I. R. (1937). The etiology of mental deficiency with special reference to its occurrence in twins. *Psychological Monographs*, 48(4).
- Rose, R. J., Reed, T., & Bogle, A. (1987). Asymmetry of *a-b* ridge count and behavioral discordance on monozygotic twins. *Behavior Genetics*, 17, 125–40.
- Rowe, D. C. (1987). Resolving the person-situation debate. *American Psychologist*, 42, 218–27.
- Rowe, D. C., & Plomin, R. (1981). The importance of nonshared (E1) environmental influences in behavioral development. *Developmental Psychology*, 17, 517–31.
- Scarr, S. (1969). Effects of birth weight on later intelligence. Social Biology, 16, 249-56.
- Scarr, S. (1985). Constructing psychology: Facts and fables for our times. American Psychologist, 40, 499–512.
- Scarr, S., & Carter-Saltzman, L. (1982). Genetics and intelligence. In R. J. Sternberg (Ed.), Handbook of human intelligence. Cambridge: Cambridge University Press.
- Scarr, S., & McCartney, K. (1983). How people make their own environments: A theory of genotype-environment effects. *Child Development*, 54, 424-35.
- Scarr, S., & Weinberg, R. A. (1978). The influence of "family background" on intellectual attainment. American Sociological Review, 43, 674–92.
- Shields, J. (1962). Monozygotic twins brought up apart and brought up together. London: Oxford University Press.
- Sitz, H. H. (1986). The raising of intelligence: A selected history of attempts to raise retarded intelligence. Hillsdale, NJ: Erlbaum.
- Stocks, P. (1930, 1933). A biometric investigation of twins and their brothers and sisters. Annals of Eugenics, 4, 49–108, 5, 23–5.
- Teasdale, T. W., & Owen, D. R. (1984). Heredity and familial environment in intelligence and educational level A sibling study. *Nature*, 309, 620–2.
- Thorndike, R. L., Fleming, C. W., Hildreth, G., & Stranger, M. (1940). Retest changes in IQ in certain superior schools (pp. 351–61). In National Society for the Study of Education, *39th yearbook*.
- Willerman, L. (1970a). Fetal head position during delivery, and intelligence. In C. R. Angle & E. A. Bering (Eds.), *Physical trauma as an etiological agent in mental retardation* (pp. 105–8). Washington, DC: U. S. Dept. of Health, Education, and Welfare.

- Willerman, L. (1970b). Maternal pelvic size and neuropsychological outcome. In C. R. Angle & E. A. Bering (Eds.), *Physical trauma as an etiologic agent in mental retardation* (pp. 109–12). Washington, DC: U. S. Dept. of Health, Education, and Welfare.
- Willerman, L. (1979). Effects of families on intellectual development. American Psychologist, 34, 923-9.
- Willerman, L. (1987, April). Where are the shared environmental influences on intelligence and personality? Paper presented at the Society for Research on Child Development, Baltimore, MD.
- Willerman, L., & Churchill, J. A. (1967). Intelligence and birth weight in identical twins. Child Development, 38, 623-9.
- Wilson, R. S. (1974). Twins: Mental development in the preschool years. Developmental Psychology, 10, 580-8.
- Wilson, R. S. (1983). The Louisville Twin Study: Developmental synchronies in behavior. Child Development, 54, 298-316.
- Wingfield, A. H., & Sandiford, P. (1928). Twins and orphans. Journal of Educational Psychology, 19, 410–23.
- Zajonc, R. (1976). Family configuration and intelligence. Science, 192, 227-36.