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CHAPTER 2

SEPARATING NATURE AND NURTURE

In a *New Yorker* cartoon, a pair of identical twins is shown reunited, sitting in the waiting room of a patent office, with identical Rube Goldberg inventions upon their laps. In actual cases, reunited twins are known for striking coincidences in their lives. Reunited twins Jim Lewis and Jim Springer were separated at 4 weeks of age and met for the first time when they were 39 years old (Chen, 1979). They had first wives named Linda and second wives named Betty; named their sons James Alan and James Allan, respectively; and named their dogs Toy. They worked as part-time deputy sheriffs in two different towns and pursued woodworking as a major hobby. As children, they had both liked math and disliked spelling; as adults, they had similar smoking and drinking habits. Coincidences? Perhaps so, as many boys are poor at spelling and many fathers want their names carried on. On the other hand, in the Minnesota study of twins reared apart, reunited fraternal twin pairs produced few such stories, whereas many examples of amazing similarities came from biographies of reunited identical pairs (Lykken, McGue, Tellegen, & Bouchard, 1992). This chapter reviews research designs for separating the effects of nature and nurture.

Variability

Social science seeks the causes of behavioral variability. Some children learn to read before first grade, others later. Some men and women are homosexual, others heterosexual. Shyness, impulsivity, honesty, and many other character traits vary enormously among individuals. In an early study of famous men, Francis Galton (1869), a pioneer of behavior genetics, sought to understand variability in social accomplishment—that

is, in the type of eminence that would today put a person on the cover of *Time* magazine. Of course, some nonvarying traits also exist in humans, such as walking on two legs and possessing a spoken language. Even here, though, a causal understanding will come only from making comparisons that will reveal variability; for instance, in our species' evolution, new genetic combinations led to walking on two legs and to language, because these human traits are missing in "cousin" primate species.

Ideally, to explain variability, a social scientist conducts an experiment manipulating potential causes. In animal studies, scientists have great freedom to manipulate both genetic backgrounds and rearing conditions. We could, for instance, study aggressiveness toward an unfamiliar person in two breeds of dogs: German shepherds and Labrador retrievers. These dog breeds should provide genetic variability, because gene substitutions have produced differences in many physical and behavioral traits. Dogs of both breeds could be reared under two sets of circumstances. In one, rearing would be relatively harsh and cold; for instance, mild physical discipline would be employed. In the other condition, the dogs could be reared by an affectionate trainer who would never use physical punishment. Thus, the experiment would have four groups of dogs: two dog breeds combined with two rearing conditions. In a test encounter with an unfamiliar person, each dog would be rated for number of snarls, barks, and other threats. The term "phenotype" refers to a measurable, expressed outcome of development. In this experiment, the dogs' aggressiveness phenotype could be predicted from the following equation:

Phenotype score (aggression)	=	Genetic score (breed)	+	Rearing score (condition)	+	Other environmental influences	+	Measurement error

This equation apportions dogs' aggressiveness to different causes. The dogs' breed should have an influence—German shepherds should be more aggressive, in general, than Labrador retrievers. So should their rearing conditions: Those dogs raised more harshly, we might predict, should be more aggressive. Other environmental influences should also contribute (e.g., unique incidents in the life of a particular dog, the dog's mood on the test day). Finally, all behavioral measurements would be imperfect; hence the need to include a measurement error term.

Scott and Fuller (1965) conducted actual studies of behavior genetics in dogs that were similar to the example above. For example,

cocker spaniels showed much less fear of people than basenji hounds. The authors then formed selective crossbreeds to test for the genetic determination of fear. Crossing cockers with basenjis resulted in offspring with half their genes from a cocker parent and half from a basenji parent. Because the crossbred dogs were raised either by a cocker mother or by a basenji mother, rearing effects were also tested. The maternal rearing environment had no influence on fear, but all the crossbred puppies were as fearful as their basenji parent. From this experiment and other genetic crosses, Scott and Fuller concluded that fearfulness of people may be determined by a dominant gene. The crossbred dogs, all inheriting one copy of this gene from their basenji parent, should always show fearfulness of people.

In human behavior genetics, these ideal experimental designs cannot be implemented. We cannot assign all redheads to one rearing condition and all brunettes to another, for both practical and ethical reasons. Nor can we make planned genetic crosses. So to determine the causes of behavior, a research design must capitalize on "experiments" of nature, in which either environment or heredity is "manipulated" by means of the social and genetic relatedness of pairs of relatives. Adoption affords a nuclear family structure like that of an ordinary family, but without the genetic relatedness of family members. Twins afford two levels of genetic relatedness—100% in monozygotic (MZ; one-egg) twins, and 50% in dizygotic (DZ; two-egg) twins. Uncle–nephew, aunt–niece, and grandparent–grandchild pairs afford a weaker (25%) level of genetic relatedness. If MZ twin brothers or sisters marry nontwin individuals, their children would be *socially* cousins, but *genetically* related at the same level as half-siblings (25%, instead of most cousins' 12.5%). These different relatedness levels occur because closer relatives share more genes affecting a trait than more distant relatives do.

As in the hypothetical dog study proposed above, human behavior genetic studies can apportion children's or adults' phenotypes to different causal influences. An equation expressing the phenotype in terms of underlying causes is as follows:

Phenotype score	=	Shared environment score	+	Nonshared environment score	+	Genetic score	+	Measurement error

It is essential to understand each component shown in this equation. Let us start with the two environmental terms—"shared" and

"nonshared" environment. The central message of this book is that we can learn much about the family environment by teasing shared and nonshared influences apart.

Environmental Components of Variation

As in the hypothetical dog animal study, shared environment is a "manipulation" of rearing conditions. The dog experiment would have had just two conditions: loving versus harsh discipline. All dogs in the "loving" condition would have been exposed to affectionate rearing; all dogs in the "harsh" condition would have had a cold, discipline-minded trainer. Family environments may also differ greatly. Some families have more resources, in terms of education and income, than others. Families may differ in their emotional climates and in their neighborhood characteristics as well.

Shared Environmental Variation

The shared environment score captures these broad family differences; by definition, composite shared environmental influences act in common on siblings (or on parent and child) to make them alike in their trait phenotypes. Imagine that we could score siblings for all environmental resources that affect their intellectual development equally (e.g., books in the home, parental vocabulary, nutrition). Now consider two families living in different parts of town. One family might have more resources to promote the growth of intelligence than the other. In the first one, children A and B might have a hypothetical shared environment score (when an average family scores 100) of 110. In the other family, on the poorer side of the town, the shared environment score of the two siblings might be just 90. That is, in the second family, this score would be exactly 90 for sibling A and exactly 90 for sibling B. A shared environment score is a *composite*, therefore, of all influences that the two siblings have in common.

In the case of IQ, the total variation is 225 (the standard deviation of IQ scores, 15, is squared). If, say, behavior genetic studies have estimated that shared environmental variation accounts for 30% of this total, the amount of this variation would be estimated at 67.5 (one would multiply .30 × 225). This number represents the *maximum* variation in

IQ attributable to all family rearing conditions that make siblings alike in intelligence. Any measured (shared) rearing condition must explain *less* variation than the composite shared estimate (of which it is a part). For example, if "number of books in the home" correlated .27 with IQ, then it would explain 7% of the variation in IQ ("variance explained" is the correlation coefficient squared). Thus, its part of total IQ variation would be 15.7 (.07 × 225). Other shared rearing influences, such as parental vocabulary or neighborhood schools, must account for the remainder, 51.8 (i.e., 67.5 – 15.7).

Behavior genetic studies can give us a numerical estimate of composite shared rearing influences. The shared rearing estimate is defined as the ratio of shared environmental variation to phenotypic variation, and is given the symbol c^2. In the IQ example, c^2 is .30, a ratio of 67.5/225. This ratio has important policy implications, because it indicates how a phenotype might be changed by altering the rearing conditions of children with poor phenotypes to be like those of children with good ones. The greater the shared rearing estimate, the more change can be expected to follow from changing rearing conditions. Christopher Jencks (1980), writing for an audience of sociologists, has recognized the policy importance of shared rearing estimates (note that in this quotation, a different symbol is used for shared rearing):

> . . . many policy proposals consist, in essence, of providing all families with advantages currently enjoyed by the privileged. If e^2_c [the shared rearing estimate] is initially large for a given phenotype, successful efforts along this line can be expected to substantially reduce the total variance of the relevant phenotype and greatly improve the relative position of the disadvantaged. (p. 734)

Thinking of particular environmental influences, however, we must realize that they do not always make siblings alike. For instance, for siblings close in age, the quality of neighborhood schools is generally a shared rearing influence. But for particular siblings who are some years apart in age, it could be also partly unshared: For example, a school district might not pass a bond issue, so that educational quality would be worse for one sibling than for another. Hence a measured environmental influence might make some contribution to our theoretical estimate of shared rearing influence (i.e., making siblings alike); it might also make some contribution to the estimate of nonshared influence (i.e., making siblings different). To some extent then, shared and nonshared rearing influences may be like the two sides of a coin.

Nonshared/Unshared Environmental Variation

Unshared environmental influences touch each individual in a unique way. By definition, they are uncorrelated across siblings (or parent and child), and so operate to make family members dissimilar in a phenotype. First, all the accidents of embryological development are unshared; they can affect siblings differently, because each child has a different birth history. Even identical twins can have different *in utero* developmental courses (some MZ twins actually compete with each other for maternal nutrients, resulting in the twins' having very dissimilar weights and health statuses at birth). Parental favoritism can be an unshared influence: A sibling receiving more love may develop differently from his or her less favored sibling. Friendship networks can act as another unshared influence. Although some siblings may befriend the same individuals, most often, because of their different ages, they belong to different friendship groups. A special person (e.g., a particularly influential teacher or friend) or an emotionally intense experience (e.g., a very severe illness) can be yet another unshared influence. The list of potential unshared influences is extremely long (see Rowe & Plomin, 1981, for other examples).

Behavior genetic studies may yield a numerical estimate of composite nonshared (synonym: unshared) environmental variation. The nonshared rearing estimate is defined as the ratio of nonshared environmental variation to phenotypic variation, and is given the symbol e^2.

Genetic Variability

A discussion of genetic variability requires terminology that may be unfamiliar. These terms are defined as they appear. A "locus" is the physical location of a gene on a chromosome (e.g., the genetic material). Chromosomes come in pairs; in each pair, one is inherited from one's mother, and the other is inherited from one's father. Therefore, except for those genes located on the sex-determining X and Y chromosomes, a person has two distinct physical copies of each gene (one maternal in origin, the other paternal). In a population, all genes at a locus may be exactly the same in their internal composition. Such a gene is then said to be "monomorphic," meaning that it comes in just one form. If we use the letter D to symbolize this gene, then Joe's genotype would be DD, because he possesses this gene in two copies. Bob's genotype is also DD, as is everyone's in this population. Genes at other "loci" (the plural of locus) may have different internal compositions; that is, they can come in different forms. When this occurs, the gene is said to be "polymorphic." The technical term for different forms of a gene is "alleles." Thus, we may use a capital letter A to represent one allele, and a lowercase letter a to represent another. For this gene, an individual may have any one of three genotypes: AA, Aa, or aa. The term "genotype" refers to a person's exact genetic makeup at a genetic locus.

For instance, in the familiar ABO blood group, three alleles exist: A, O, and B. A child will inherit one allele from the mother and one from the father, yielding the child's blood group. At the ABO locus, one child may inherit the genotype AB, whereas a sibling may inherit AO. The first child has blood type AB; the second has blood type A, because a single O allele does not change blood type. With three alleles, there are six possible blood group genotypes in human populations: blood type A (AA or AO), blood type O (OO), blood type B (BO or BB), and blood type AB (AB).

Such varying alleles may influence trait phenotypes.[1] That is, when they influence a continuous (many-valued) trait (like IQ or height), a substitution of one gene for another will change its value. Suppose that a hypothetical A locus influences young children's activity levels when counted as the "number of fidgets per minute." Table 2.1 provides examples of different types of gene effects for this locus.

A gene locus's effect can be called "additive" when the substitution of one allele for another increases the fidgeting rate linearly. As shown in Table 2.1, if it is assumed that the effects of the A locus are additive,

TABLE 2.1. The Fidgeting Gene Locus: Examples of Different Gene Effects

	Fidgets per minute for fidgeting locus genotype:		
Type of effects	AA	Aa	aa
Additive effects	6	4	2
Dominance effects	6	6	2
Epistasis effects in presence of B locus genotype:			
BB	6	4	2
Bb	6	4	2
bb	2	2	2

children who possess genotype AA fidget an average of six times per minute; those who possess Aa fidget four times per minute; and those who possess aa fidget just twice per minute. In other words, the difference in fidgeting rates between genotypes is always *two* fidgets per minute (i.e., 6 – 4 is 2, and 4 – 2 is also 2), so that a substitution of one allele for another always increases the trait by an equal amount.

Additive gene effects may make biological relatives resemble one another in a trait. The average level of genetic similarity indicates how likely two individuals are to possess the same alleles. The greater the number of alleles matching in two people, the closer their numerical trait scores. In our example, two AA individuals both fidget six times per minute, but AA and aa individuals fidget at different rates. Siblings or parent–child pairs (first-degree relatives) share, on average, 50% of their alleles at different loci affecting a trait; half-siblings and uncle–nephew or aunt–niece pairs (second-degree relatives) share, on average, 25%; and MZ twins, of course, share 100%. Thus, allele substitutions influence a trait so that more closely related biological relatives are more alike in their trait scores.

A statistical measure for similarity is the correlation coefficient. When marriage is approximately random in a population (i.e., spouses are not matched for a particular continuous trait to a greater extent than would happen by chance), then the genetic correlation for first-degree relatives is close to .50; for second-degree relatives, close to .25; and for third-degree relatives (e.g., cousins), close to .125 (see Falconer, 1981, for mathematical derivations; see also Plomin, DeFries, & McClearn, 1990). For MZ twins, it is 1.00. Thus, different groups of genetic relatives can be used to test for additive genetic influence on trait variation.

Other kinds of genetic influence are "nonadditive." These include genetic dominance and epistasis. "Genetic dominance" refers to intralocus interactions among alleles. As shown in Table 2.1, if it is assumed that the effects of the fidgeting locus are determined by genetic dominance, individuals with genotypes AA and Aa fidget an average of six times per minute, whereas aa individuals fidget at only two times per minute. Somehow, the A and a alleles interact, so that having one of each has the same total effect as having two A alleles.

Another kind of genetic nonadditivity is "epistasis," which refers to interlocus interactions among genes. As shown in Table 2.1, the effects of the fidgeting locus might depend on that of a hypothetical B locus, somewhere else in the genome. When a person possesses genotypes BB or Bb, the fidgeting locus acts just as we would expect: Fidgeting

increases from genotype aa to genotype AA. But in the presence of genotype bb, something unexpected happens: Regardless of the particular fidgeting locus genotype, the rate of fidgeting is only two times per minute. With epistatic effects, a trait's numerical value depends on the whole *configuration* of genes. Only identical twins share all nonadditive gene effects, because they are the only kind of biological relatives who possess exactly the same genotypes at all loci.

The "heritability coefficient" summarizes the strength of genetic influence on trait variation in a particular population. It is defined as the ratio of genetic variation to phenotypic variation, and is given the symbol h^2. The more a trait changes as one allele is substituted for another, the greater trait heritability is. As with shared environmental variation, heritability can be estimated from behavior genetic research designs. Heritability is called "narrow-sense" if it just estimates additive gene effects; it is called "broad-sense" if it estimates all (additive plus nonadditive) genetic effects.[2]

Heritability will vary from one population to another, depending on the kinds of genotypes and environmental exposures present. For instance, the heritability of skin color will be greater in a racially diverse population than in Sweden. In Sweden, environmental effects will be greater if we compare Swedes returning from a Mediterranean summer vacation with those staying home (untanned vs. tanned skin). Nonetheless, heritability coefficients may be generalizable over a range of environmental conditions, and the degree of generalizability can be evaluated empirically.

Research Designs for Separating Nature and Nurture

Separated-Twins Design

The study of separated twins is the most direct method for estimating a trait's heritability. Separated MZ twins are reared by different parents, and hence have different family environments, whereas they possess identical heredity. Thus the effects of heredity are distributed against a background of different family environments, and we can infer genetic influence on a trait from resemblance in separated twins. Data exist from five studies of separated twins—three quite small studies (Juel-Nielsen, 1980; Newman, Freeman, & Holtzinger, 1937; Shields, 1962) and two

larger, more recent studies (Bouchard, Lykken, McGue, Segal, & Tellegen, 1990; Pedersen, Plomin, McClearn, & Friberg, 1988). The Swedish twin study (Pedersen et al., 1988) is unique because the separated MZ and DZ twins were identified through national records, rather than being recruited through advertisement or word of mouth, and because of a larger sample size (about 200 pairs of twins of each type separated early in life). A sixth study of separated twins, by the British educational psychologist Cyril Burt (1955), has been the center of controversy over possible scientific fraud, and the proper response of most social scientists has been to exclude his data from consideration.

In a separated-twins study, the heritability estimate (h^2) is merely the trait correlation for all MZ twin pairs. Thus we can write:

$$r_{MZ} = \frac{\text{Genetic variation}}{\text{Phenotypic variation}} = h^2$$

As noted earlier, this heritability has a special interpretation: It is called "broad-sense" heritability because it reflects the action of all genes. MZ twins share the same pattern of genes at all loci, so that nonadditive gene effects also contribute to their behavioral resemblance in a way they cannot contribute to that of other biological relatives, who do not share the whole pattern of genes relevant to a trait. In the nature–nurture arena, we are particularly interested in the *maximum* possible influence of genes. Indeed, the best guess we could make about the psychological and physical traits of another person, without interviewing him or her directly, would be based upon characteristics of the person's MZ twin (if one could be found). Nothing we might discover about conditions of rearing, schooling, neighborhood, religion, or schoolyard friends would come close to the usefulness of an MZ twin in providing information about this person's height, weight, eye color, temperament, mental illness, habits, IQ, values, or nearly any other trait.

Any single type of study design is, of course, subject to particular weaknesses; therefore, my emphasis in this book is on the number of different studies using different approaches and converging on similar conclusions. Critics of separated-twins studies mention two weaknesses of this design: (1) lack of total separation of the twins and (2) selective placements.

Because MZ twins are typically separated for a great number of reasons, and because they are sometimes adopted by relatives, their separations are not always as complete as they would be in an ideal (but

impractical) experimental study. A trio of ardent critics of separated-twins studies, Lewontin, Rose, and Kamin (1984), mention the stories of separated twins who were not completely separated, such as the following: "Benjamin and Ronald had been 'brought up' in the same fruit-growing village, Ben by his parents, Ron by the grandmother. . . . They were at school together. . . . They have continued to live in the same village" (p. 108). Although it is true that MZ twins reared apart are sometimes not perfect separations, it is also true that first cousins often have similar degrees of contact: They too may live in the same village, in houses not that far apart, with parents who are siblings and socialize together, and with many opportunities to play and visit together. If social contacts can miraculously make people alike, then why do we not find great resemblance in cousins—to say nothing of nontwin siblings, who have even more social contact?

A more scientifically convincing response to this objection is that the timing of separation, the frequency of social contacts, and other contact measures have been included in separated-twins studies as variables themselves. Thomas Bouchard (1983), in his reanalysis of the three older studies of separated twins, found that separation age and degree of contact failed to condition the MZ twins' IQ resemblance. Similar results have been obtained for separation variables in the Swedish (Pedersen et al., 1988) and Minnesota (Bouchard et al., 1990) separated-twins studies: Separated MZ twins who have had later contact have generally not been found to be more alike in either personality or IQ.

Thus, the occasional social contact of "separated" twins may not introduce strong biases. A more serious problem in adoption studies is that of selective placement. Although this was not the case in Jill Ireland's adoption of Jason (see Chapter 1), many adoption agencies do consciously attempt to match the social status of adopting parents with that of the biological parents. In a completely scientific study, adoption agencies would want to match adoptees randomly with adoptive parents—so that (for example) a factory worker would rear a child of a doctor, at least on occasion. The effect of selective placement is most serious for IQ and related traits, because these correlate most highly with years of education and income, which are used to assess social class. If we think that selective placement has occurred, its quantitative strength is the correlation between the trait as measured in the biological parent (usually the unwed mother of the adoptee) and as measured in the adoptive parent. The strength of selective placements varies among adoption studies. In Bouchard et al.'s (1990) study of twins raised apart, the social class

of the adoptive fathers of reared-apart twins correlated .27; in the Colorado Adoption Project (Plomin & DeFries, 1985), there was no association of biological and adoptive parents' social class.

Selective placement represents a potential bias for both genetic and environmental interpretations of adoptive studies. It upsets the environmental part of an adoptive design, because the adopted-away children may be raised in homes resembling what their homes would have been like had they stayed with their biological parents. It also upsets the genetic part of the adoptive design: Selective placement may make the genotypes of adoptive children and adoptive parents similar, whereas they would not be if the children were randomly assigned to adoptive families.

Fortunately, selective placement can be handled in behavior genetic studies. When analyzing their data statistically, behavior geneticists can allow for selective placement effects. For a genetic effect to be accepted, it must be greater (quantitatively) than all the adoptee–adoptive parent resemblance that could occur as a result of selective placement. Replication of results across studies with low or high levels of selective placement is another protection against false leads. Finally, because placement selectivity is most strongly oriented toward the IQ trait domain, it is seldom a concern when other traits are investigated.

Nontwin Adoption Designs

In the last 20 years, adoption in the United States has become more diverse. In the traditional adoption process, a child was adopted in early infancy, and records on the biological parent(s) were closed to the adoptive parents. Most children were given up for adoption by unwed mothers, who sometimes left home and lived in church-run homes where they could bear their children while avoiding social stigma and ostracism. Today, a pregnant girl is not a social outcast, and she may decide to keep her baby rather than relinquish the child for adoption. Moreover, abortion is an option for ending an unwanted pregnancy. If a child is relinquished today, the varieties of adoption are much greater: Placements are increasingly made by private attorneys rather than agencies, and it is not uncommon for the adoptive parents to know the biological parent(s). Despite these many changes, the major U.S. adoption studies since World War II have used traditional adoptions. The largest postwar American adoption studies, cited later in this book, are the Minnesota Adolescent Adoption Study (Scarr & Weinberg, 1983); the Minnesota Transracial Adoption Study (Scarr & Weinberg, 1976); the Colorado Adoption Project (Plomin & DeFries, 1985); and the Texas Adoption Project (Horn, Loehlin, & Willerman, 1979).

The adoption study is the most direct means of estimating the shared rearing component of variation. If selective placement is ignored, a trait correlation for unrelated sibling pairs—who may be either different children adopted into a family, or an adoptee and a biological child of the adopting parents—directly estimates shared rearing variation (c^2):

$$r_{\text{unrelated pairs}} = \frac{\text{Shared environmental variation}}{\text{Phenotypic variation}} = c^2$$

The same equation holds for shared rearing influence when the adoptive parent and adoptee are correlated.

Although these comparisons are completely informative for shared environmental effects, it is desirable for an adoption study also to estimate genetic effects. The two main strategies are (1) the "full" adoption design and (2) the "matched" adoption design. In the full adoption design, traits are assessed on the biological parent(s) (usually the mother) who relinquished a child for adoption. The Texas Adoption Project is an example of a study employing this design. The study was initiated when Joseph Horn and his colleagues found a private adoption agency in Texas that routinely gave the unwed mothers IQ and personality tests. With this information, the investigators completed the full adoption design by relocating the adoptees and their adoptive families. In this design, genetic effects are estimable from the trait correlation of the adoptees and their biological parent.

In an alternative design, adoption families can be compared to biological families matched for parental age and social class. In the latter, the correlations contain both rearing and heredity components. For example, the sibling correlation in biological families estimates the following:

$$r_{\text{related pairs}} = \frac{\text{Shared environment}}{\text{Phenotypic variation}} + \tfrac{1}{2} \times \left\{ \frac{\text{Shared heredity}}{\text{Phenotypic variation}} \right\}$$

Given this mathematical expectation, we can find the genetic effect by subtracting the correlation for *unrelated* pairs from that for *related* pairs in the matched families, assuming that the match of the two sets of families was good. The latter can be checked by comparing environmental assessments on the two sets of families for differences in mean levels and variances. Other opportunities may arise when an adoptive family

has both a biological child and an adoptee. In these cases, there is no need to match two sets of families, because the comparison can be made solely within the adoptive families in which the two children were reared. Even critics of behavior genetic methods acknowledge that this is a powerful research design. As Lewontin et al. (1984) observe, "There is plenty of room for any genetic effect to display itself in a higher correlation for the biological parent–child pairs" (p. 113).

The main criticism of adoption studies is the possibility that adoptive parents treat adoptees differently from the way that biological parents treat their own children. A hypothesis of differential treatment is worthy of some concern, but it is *post hoc* and nonspecific. After all, treatments may differ, but adoptive parents still use the same rewards, punishments, and examples to influence their children. If this is true, then rearing in adoptive families should show a direct relationship to child outcome (especially in young children, who cannot cognitively appreciate that they are "adopted"), even if the differences in treatment move personality development in a somewhat different direction in adoptive families than in biological ones.

As to a special knowledge of adoption, at least one test of its possible biasing effects yielded no significant findings. If a sense of similarity to a child *makes one similar to that child*, then parents' perceptions of similarity should drive actual similarity. This test was applied by Scarr, Scarf, and Weinberg (1980) in both adoptive and biological families. In the domains of intelligence and temperament, family members were completely inaccurate in guessing whether they were alike or unalike. Because the perceptions of resemblance were such a poor guide to actual resemblance in this case, it is hard to imagine how feelings about similarity might have guided personality development.

Classical Twin Designs

Most twin studies do not involve the rare pairs of twins who were separated and raised apart. Instead, two kinds of reared-together twins (MZ and DZ) are compared. It was only early in the 20th century that the existence of two biologically distinct types of twins was first acknowledged. It was then realized that the number of same-sex DZ twins must equal the number of opposite-sex twins: DZ twins are simply genetic siblings who happen to have shared a pregnancy, and half of all siblings are born boy–girl pairs and the other half are born either boy–boy

or girl–girl pairs. By counting the number of twins, and determining how many of these are opposite-sex twins, a researcher can do a simple calculation to find the approximate number of MZ twins. In 1924, an American psychologist, Merriman, and a German dermatologist, Siemans, first proposed using the method of comparing MZ with DZ twins to infer the degree of genetic influence. They discovered that MZ twins' correlation for the size of birthmarks was .40 and that for DZ twins was .20, exactly as one would expect for a trait with purely genetic variation and unshared environmental variation (cited in Rende, Plomin, & Vandenberg, 1990). For birthmarks, doubling the DZ twin correlation reproduces the MZ twin correlation, as expected on the basis of these equations:

$$r_{\text{MZ pairs}} = \frac{\text{Genetic variation}}{\text{Phenotypic variation}} = h^2$$

and

$$r_{\text{DZ pairs}} = \tfrac{1}{2} \times \left\{\frac{\text{Genetic variation}}{\text{Phenotypic variation}}\right\} = \tfrac{1}{2}\, h^2$$

The remaining variation—the difference between the MZ twin correlation and 1.00—represents the proportion of phenotypic variation that is attributable to unshared environmental sources and measurement error (reliability limitations are also inherent in physical measurements).

Composite shared rearing influences can also enter into this twin design. In a pure case of shared rearing influence, the MZ and DZ twin correlations would be equal because of the twins' common rearing experiences. Thus, the correlations can be expressed as follows:

$$r_{\text{MZ twins}} = r_{\text{DZ twins}} = \frac{\text{Shared environmental variation}}{\text{Phenotypic variation}} = c^2$$

When twin samples are large, a twin study can reveal both genetic variation and composite shared rearing variation. Algebraically, the shared rearing variation can be estimated as $2r_{DZ} - r_{MZ}$ and the heritability as $2 \times (r_{MZ} - r_{DZ})$.

In one twin study, Coon and Carey (1989) found that for high school musical interest and honors, MZ and DZ twin correlations were nearly equal when both twins had taken music lessons together (because parents had not wanted to arrange them for just one). For instance, MZ twin brothers correlated .71 for musical interest, whereas DZ twin brothers correlated .63. With this pattern, we can infer that about half the

variation (as noted above c^2 is twice the DZ correlation – the MZ correlation, or .55) in high school musical interest was attributable to the music lessons and other shared experiences. Coon and Carey's study shows that the twin method can successfully demonstrate composite shared rearing influence.

In another example, twin studies of delinquency have also demonstrated shared environmental influences. In a study of adolescents' deliquent acts (Rowe, 1983), the twin correlations for confessed delinquent acts yielded quite large estimates of shared rearing. The MZ male twin correlation (r_{MZ} = .62) was close to that of DZ male twins (r_{DZ} = .52). From these correlation coefficients, the shared family effect can be inferred to be substantial (c^2 = .42). Similarly, in female twins, the estimated shared environmental effects were also large (c^2 = .26). In the case of official delinquency records, twin similarity can be estimated from twin concordances (i.e., given a delinquent index twin, the probability that the cotwin is also delinquent). From a summary review of twin studies, DiLalla and Gottesman (1989) concluded that shared influences had large effects on adolescent crime, because MZ and DZ twin concordances were close in value: "The weighted concordances across the delinquency studies were 87% for MZ twins and 72% for DZ twins. The high concordance rates for DZ twins, greater than 50%, suggest a fairly large influence of shared family environment" (pp. 341–342).

Although detailed explanations of this shared effect lie outside the scope of this chapter, I interpret it as one of sibling mutual influence. In a study of nontwin siblings' delinquent behavior, the sibling correlation was *conditioned* on the degree of siblings' mutual contact (Rowe & Gulley, 1992). Siblings who liked each other (or who had the same friends) were substantially more alike in their rates of delinquency than those who were emotionally distant (or who belonged to different peer groups). Consider, for example, that the correlation coefficient of delinquency in "close" brothers (r = .63) was about triple that in "distant" brothers (r = .20). From this pattern of correlations, I infer that "close" brothers imitated each other's delinquency (or the delinquent behavior of their mutual friends). Similar findings obtain for twin siblings (Carey, 1992). On the basis of his mathematical models, Carey concluded that sibling mutual influence appears to explain a part of twins' resemblance for officially recorded delinquent acts. From these behavior genetic studies, what is surprising is that for delinquent behavior, a "shared family effect" estimated from twin models may involve ***shared sibling influences*** rather than, as would be commonly assumed, parental treatments. Nonetheless, in both the musical performance and delinquency examples, the twin method has been shown to identify the component of shared environmental variation successfully.

In contrast to these examples, twin studies typically find an absence of shared environmental influences on behavioral traits (as will be amply documented later in this book). This persistent failure to find shared rearing effects has led to accusations against the method rather than against its message—a natural tendency to blame the messenger for news many do not wish to hear. So Lewontin et al. (1984) have explained the greater behavioral resemblance of MZ than DZ twins in terms of uneven treatments of the two types of twins:

> . . . there are also some obvious environmental reasons to expect higher correlations among MZ than among DZ twins. . . . Because of their striking physical similarity, parents, teachers, and friends tend to treat them very much alike and often even confuse them for one another. MZ twins tend to spend a great deal of time with one another, doing similar things, much more so than is the case with same-sexed DZ twins . . . (p. 115)

Do parental treatments mold twins' traits alike? Or do twins' similar genetic traits provoke a search for similar, mutually reinforcing environmental opportunities? In the tendency of MZ twins to receive similar treatments, or to seek them out, the arrow of causation is certainly bidirectional (Lytton, 1980; Scarr & Carter-Saltzman, 1979).

Moreover, the question is not whether MZ twins receive more similar treatments (they do, and to claim otherwise would be foolish), but whether those treatments influence a particular trait. Some traits may be influenced by MZ twins' similar treatments, and others not. A useful example is the case of dressing twins alike. Three-year-old MZ twins, when dressed alike, seem adorable, and simple observation shows that MZ twins are dressed alike more often than DZ twins. Yet this particular treatment is not likely to mold a trait such as IQ. Intuitively, we know that clothes do not make a person's intelligence, and putting the same T-shirts on siblings does not homogenize variation in their IQs.

Dressing alike, though, may be just one example of a broader range of parental treatment similarities that could affect personality development. If so, those twin pairs whose parents attempt to treat them alike should be more alike in personality and intelligence than those twins given more *laissez-faire* treatment. Just this prediction was tested by the psychologists John Loehlin and Robert Nichols (1976) with 850 twin pairs. They constructed a scale of differential experience including these

items: "dressed alike," "played together" (ages 6–12) or "spent time together" (ages 12–18), "same teachers in school," "slept in same room," and "parents tried to treat alike." The scores on these items, and a composite score based on all five items, were correlated with the (absolute) difference in twins' intellectual abilities, personality traits, vocational interests, and interpersonal relationships. Loehlin and Nichols described their findings as follows:

> We will probably not be accused of extravagance if we say that these correlations are not very large. More of them *are* positive than negative (393 to 181, as a matter of fact), but the typical *r* is not greater than +.05 or +.06. . . . it is clear that the greater similarity of our identical twins' experience in terms of dress, playing together, and so forth cannot plausibly account for more than a very small fraction of their greater observed similarity on the personality and ability variables of our study. (1976, pp. 51–52)

Of course, the typical failure of differential treatments to matter does not mean that Lewontin et al. (1984) are always wrong. Social scientists must be alert to occasions when unequal environmental contexts may affect behavior—for instance, siblings' mutual closeness (see Carey, 1992; Rowe & Gulley, 1992). But for most broad personality and intellectual traits, concern over treatment differences has been misplaced.

People often claim that because identical twins look alike, they act alike. As in other exaggerated criticisms, there may be a few grains of truth in this one. Surely, more MZ than DZ female twins go on a similar number of dates, because their physical beauty is more closely matched. But to extrapolate an occasional effect of physical appearance to all traits is plainly wrong. If IQ or personality could be so easily read in a face, then the 19th-century phrenologists, who looked in the face for signs of "atavism" (large jaws, extreme size of the eye orbits, monkey-like noses, and other "primitive" features) would have earned great scientific dividends from their explanation of criminality.

To appreciate the weakness of this "similarity of appearance" explanation, let us consider physical attractiveness and a trait phenotype, such as self-esteem. Suppose, for example, that attractiveness is correlated .20 with self-esteem for each twin. Now, using the rules of path analysis, one can determine how much appearance may contribute to MZ twins' resemblance in self-esteem. As shown in Figure 2.1, two causal pathways connect MZ twins' self-esteem phenotypes. One of these pathways is through attractiveness. It depends on the correlation of the twins'

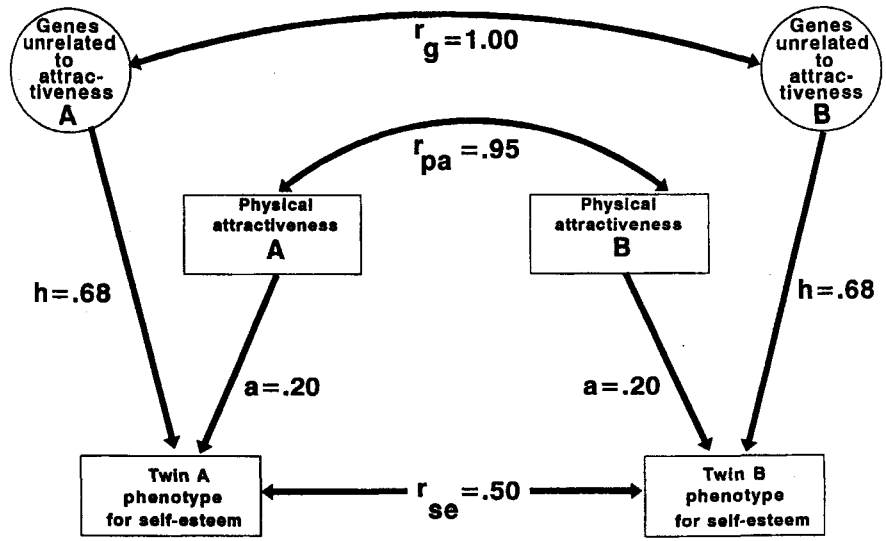

FIGURE 2.1. Physical attractiveness and MZ twins' phenotypic resemblance in self-esteem. *a*, correlation between attractiveness and self-esteem in each twin; *h*, correlation between genes unrelated to attractiveness and self-esteem in each twin; r_g, genetic correlation between the twins; r_{pa}, physical attractiveness correlation between the twins; r_{se}, self-esteem correlation between the twins.

physical attractiveness—which, because MZ twins are not *exactly* alike in appearance, is less than 1.00 (in Figure 2.1, this r =.95). It also depends on the correlation of attractiveness and self-esteem for each twin, already given here as .20. Now, to calculate the expected correlation between the twins' phenotypes that is attributable to this causal pathway, one multiplies the path coefficients connecting them (i.e., .20 squared [.20 for twin A × .20 for twin B] is multiplied by .95). This correlation works out to be .038. Genes that are unrelated to attractiveness may also contribute to behavioral resemblance. In Figure 2.1, they are assumed to correlate .68 with the phenotype of self-esteem. The twins' phenotypic correlation attributable to this causal pathway works out to be .462 (when .68 squared is multiplied by the twins' genetic correlation, 1.00). According to path analytic methods, the twins' self-esteem correlation should be the sum of these two pathways; it equals .50. But notice, too, that just a tiny fraction (7.6%) of this association is attributable to the twins' appearance; most of their behavioral similarity results from other genetic influences, the ones *independent* of physical appear-

ance. Because the numerical values chosen for this hypothetical example are conservative ones, its lesson is that appearance similarity should be only a weak determinant of MZ twins' behavioral resemblance.

Empirical data also tend to discount appearance similarity. For instance, MZ twins remain alike in personality, even after their degree of facial attractiveness is statistically controlled for (Rowe, Clapp, & Wallis, 1987). In two twin studies, those twins who were rated as more alike in appearance were not more alike in their personality traits (Matheny, Wilson, & Dolan, 1976; Plomin, Willerman, & Loehlin, 1976).

In summary, merely matching people in physical appearance should have little effect on similarity in their psychological traits, because, whatever greater treatment similarity lookalikes receive, it cannot make them alike in psychological traits if these treatments lack causal influence on the biological functions relevant to broad traits. How alike in personality or musical talent is even the best Elvis Presley lookalike to the King of Rock and Roll? If we gathered 10 Elvis lookalikes together, would they be alike in personality at all? The remarkable similarity of MZ cotwins is attributable to genes' creating matching neurons. Personality and temperament reside in the brain, not in a face.

Model-Fitting Designs

In behavior genetic studies, the state of the art consists of model-fitting research designs, which can combine features of the aforementioned designs (Neale & Cardon, 1992). Model fitting uses equations defining the *expected* correlations for different groups of biological and/or social relatives, so that relatives of many types can be combined into a single study that yields estimates of shared environmental variation and heritability. Figure 2.2 gives a simple example of the model-fitting approach. Each diagram describes a causal process on a phenotype in relatives of a particular social or biological type, and each can be expressed as an equivalent equation according to path-analytic rules. The three diagrams yield equations for MZ twins, related siblings, and unrelated siblings reared together, respectively. Arrows labeled with an "*h*" mark genetic influence; arrows labeled with a "*c*" mark shared environmental influence. The circle labeled "*u*" represents unshared environmental influence.

In this example, $h = .7$ and $c = .4$. The equations in Figure 2.2 thus result in the following expected correlations: MZ twins, $r = .65$; related siblings, $r = .41$; and unrelated siblings reared together, $r = .16$. With actual data, one works in the reverse direction, using the three equations with two unknowns (h and c) to estimate the values of those unknowns. Because there are more equations than unknown values, one also obtains a sense of how well the model fits the data. For instance, if the real correlations were .60, .45, and .11 for MZ twins, related siblings, and unrelated siblings, respectively, the equations would fit well with $h = .7$ and $c = .4$, but *not* perfectly. Discrepancies between expected correlations and obtained ones allow for a statistical test of a model's "goodness of fit."

Figure 2.2's model illustrates several points. First, with model fitting, data from many different behavior genetic studies can be combined in a single analysis to recover estimates of genetic and environmental influence. Second, the more groups that are included, the more vigorously assumptions can be tested. For instance, the simple model shown does not allow for special MZ twin environments, and discrepant values of this correlation could reject the model of Figure 2.2. Third, model fitting can ground our conclusions by means of statistical tests of significance and goodness of fit. The powerful model-fitting approaches, based on statistical procedures, should only follow a direct examination of the observed statistics.

Molecular Genetics and Behavior Genetic Designs

Behavior genetic studies may seek to identify specific genetic and specific environmental influences in the composite estimates of variance components (Plomin, 1990; Loehlin, Horn, & Willerman, 1989; Rowe & Waldman, 1993). Until recently, the possibility of identifying specific genes in composite heritable influences seemed remote; with the advent of molecular genetics, however, cautious optimism has increased in regard to identifying some genes that are relatively more powerful causes of variation in behavioral traits. This section briefly reviews advances in molecular genetics that may lead to biologically grounded behavior genetic research designs.

Specific details of genetic inheritance are known more precisely than ever before. The basic unit of inheritance, a gene, is a stretch of DNA molecule containing a sequence of chemical information. Like a written sentence, it has a meaning for the biological cell as a set of instructions for creating proteins, which are both the structural materi-

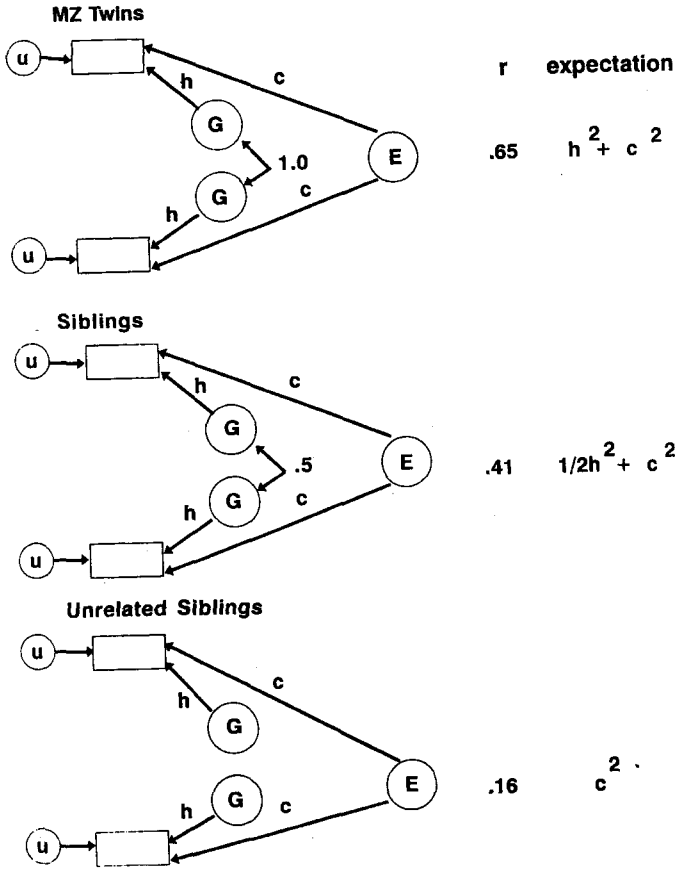

FIGURE 2.2. The phenotypic resemblance of MZ twins, related siblings, and unrelated siblings. G, genotype; E, shared environment. See text for details.

als of all cell components and also the enzymes that regulate the rates of biochemical reactions. The message in a gene is coded much like words in a sentence, except that instead of 26 letters, the alphabet is four letters: the chemical bases adenine (A), thymine (T), guanine (G), and cytosine (C) that physically lie within the double helix of DNA. And each "word" in the gene sentence is just three letters (chemical bases) long.

The genetic code is among the greatest discoveries of 20th-century science. Each three-chemical base word in the gene sentence corresponds to a particular amino acid, the chemical components of proteins, except for three "words" that are like a period at the end of a sentence, marking where a gene ends. For example, the sequence CAT is the amino acid valine; AAT is leucine; and ATT is one "stop" signal that indicates the end of a gene. A typical gene codes for 400 amino acids (or, equivalently, is about 1,200 bases long), when read sequentially from its start to the stop signal.

Estimates of the genetic loci coding for different proteins (or for RNA transcripts) in the human genome vary; a range from 50,000 to 100,000 is currently favored. Even if the genes at two-thirds of these loci were found to be monomorphic, there would be still as many as 33,000 loci that have two or more alleles and that are thus able to contribute to behavioral variability. This is a gigantic domain in which to find genes influencing a particular trait. Robert Plomin (1990) has likened this problem to finding the proverbial "needle in a haystack."

Two general approaches exist for finding the genes affecting a particular trait (viz., the needles) in the background of thousands of irrelevant genes (viz., the haystack): association studies and linkage studies (Plomin, 1990). In an association study, candidate loci with likely physiological relevance to a trait are first identified. Ideally, the alleles at this loci differ in mutational changes that alter the amino acid sequence of the gene product—not in "silent" mutations that may change a gene's "base" sequence without altering the amino acid sequence (e.g., ATC and ACG both code for valine) or in mutations in nonfunctional regions of a gene (i.e., in regions called "introns"). Genotypes at the loci are then scored numerically, and these values are subsequently correlated with variation in the trait. Association is shown if particular genotypes or alleles are more frequent in individuals who express a particular trait.

For example, alcohol dehydrogenase is an enzyme involved in the breakdown of alcohol into nontoxic compounds that are removed from the body (Crabb, Edenberg, Bosron, & Li, 1989). This locus is polymorphic, with a normal allele and a mutant one that is less effective in breaking down compounds derived from alcohol. This mutant gene is also genetically dominant over its normal counterpart. One effect of having the mutant gene is that the faces of affected people tend to flush red after these people drink alcohol. This mutant gene is more common in Asian racial groups than in other populations. In these Asian populations, alcoholics are several times more likely to carry normal alleles than abnormal ones; for instance, only 2.3% of Japanese alcoholics, as opposed to 41% of the general population in Japan, were found to have a geno-

type with the mutant allele (Crabb et al., 1989). Thus, this single gene exerts a considerable influence on the risk of alcoholism by making drinking less attractive for many individuals.

The other primary approach to identifying single genes is linkage analysis. In linkage studies, both genes and specific traits are followed through a family pedigree. The coincidence of particular genes with a specific phenotype throughout the family pedigree is evidence of genetic linkage. For example, if it were found that schizophrenia usually co-occurred with allele A at a locus affecting schizophrenia, whereas nonschizophenic family members inherited allele a, this would constitute evidence of genetic linkage. Sibling pairs also can be used in linkage studies. At a genetic locus, siblings can share no alleles, one allele, or two alleles—making them like unrelated individuals, like ordinary siblings, or like MZ twins in terms of their genetic relatedness at that locus (and in nearby genome regions). If siblings who share more alleles are also more alike in behavior, this correlation also demonstrates genetic linkage. Unlike candidate genes, linked genes may not be the ones directly influencing a trait phenotype, but may merely represent genetic loci close in physical proximity to the truly influential loci.

Both association and linkage methods are being pursued to locate genes for behavior. Although promising, both methods face considerable obstacles. The association research design suffers from the disadvantage that population histories may be confounded with direct gene effects; for instance, the association of skin color genes with IQ does not mean that the former directly cause intelligence. Linkage studies have a weakness of low statistical power, unless huge samples are used. Because the main conclusions of this book depend on the examination of environmental rather than genetic variation, they can stand independently of the direct identification of genetic loci affecting different behavioral traits. Nonetheless, the identification of these genes will provide an intellectually satisfying closure on the question of genetic influence on behavior, and will open new areas of research.

Environments and Behavior

In this review of genetics, many complexities of both inheritance and environment have been omitted. For instance, I have failed to discuss gene × environment interactions. At some level, the world is undoubtedly a more untidy place than that implied in my very simple description of how genetic and environmental variation combine. Yet, in a surprising number of applications, simple ideas capture enough of trait variation so that more complicated ones are hardly needed, or are a luxury for explaining the final few percentage points of variance.

Behavior genetic methods can be used to estimate the strength of shared family environmental influences, even when the specific mechanisms are unknown. A shared environmental estimate indicates how much changing family environments would change trait phenotypes; if this statistical term accounts for appreciable outcome variation, then changing the family environments of disadvantaged children to be like those of the most advantaged should change developmental outcomes for children. The thesis of this book is that the effects of shared family environments on children's developmental outcomes are limited. The data assembled in the next two chapters show, in particular, that for many personality and intellectual traits, variations in shared family environment have little influence on trait development. A lack of shared environmental influences would weaken, if not falsify, many explanations of behavioral variation tied to the family unit, including standard rearing variables such as the general emotional climate of the home (e.g., warmth vs. coldness), parental discipline patterns, home intellectual stimulation, family structure, and many other family variables. In the next two chapters, the phrase "rearing variation" is used to refer to those aspects of parental treatments that correspond to the theoretical shared environment component of variation, as defined above. This shorthand is used even though this variance component includes, in addition to parental treatments, other environmental effects tied to the family unit (e.g., neighborhood schools).

As discussed above, behavior genetic studies also estimate nonshared (also called unshared) environmental influences that vary within a family—they constitute the world of experience unique to each child. The behavior genetic studies reviewed in the following chapters consistently affirm the effects of different nonshared environmental influences on trait variation; after all, even genetically identical MZ twins are not perfectly concordant for behavioral characteristics. One part of unshared environmental influence may be the effects of parents' differential treatments of their children. Thus, an exclusion of shared environmental influences does not necessarily negate the influence of all family rearing influences. The possible role of differential parental treatments of

siblings is considered later in Chapter 5. The next chapter explores evidence regarding shared environmental influences on nonintellectual personality traits and psychopathology.

Notes

[1]The purpose of predicting developmental outcomes from genotypes is sometimes criticized as "genetic determinism." But there is really nothing wrong with a deterministic outlook in the social sciences. After all, reliable prediction of developmental outcomes is an implicit goal in environmentally oriented as well as genetically oriented research.

In general, social scientists have adopted a "probabilistic" view of causation, in which "the values of an independent or causal variable do not determine the specific *outcomes* of a dependent variable but rather the specific (conditional) *probability distributions* with which the values of the outcome variable occur" (Mulaik, 1987, p. 24; emphasis in the original). To apply this to the genotype → phenotype relationship, a particular genotype implies a probability distribution of the relevant phenotypes. In some single-gene diseases, the conditional distribution has low variance, so that a high degree of "determinism" obtains. For instance, all bearers of the dominant gene for Huntington's disease will develop this neurological disorder if they live long enough. Most conditional probability distributions have greater variance. In the case of Down's syndrome (a chromosomal abnormality that causes mental retardation), though the IQ distribution of affected children is definitely far below average, an affected child occasionally possesses normal intelligence.

With these ideas in mind, we can see that whether a developmental outcome is highly "genetically determined" depends both on its heritability and on how it is defined. Thus, the probability of one child of a schizophrenic mother's becoming a schizophrenic adult is about 1 in 10, a weakly determined outcome for a single child. Of the offspring of 1,000 schizophrenic mothers, about 100 will become schizophrenic adults. We may define another outcome as the probability that *at least 40* offspring will become schizophrenic adults. This latter outcome would be more strongly "genetically determined," with a probability close to 1.00, although the fate of any single child would be hard to know in advance.

Better prediction would be obtained if we had genetic tests for the specific genes that lead to schizophrenia. Even so, prediction would be imperfect because these genes do not always express themselves as schizophrenic disease, as shown by the imperfect concordance of MZ twins. If one MZ twin is affected, the probability is about 50% that the other twin is also affected.

[2]A variance component is sometimes expressed in a percentage form to refer to what part of phenotypic variance it explains. For example, if h^2 equals .25, then 25% ($100 \times h^2$) of phenotypic variation is attributable to genetic variation.

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CHAPTER 3

As the Twig Is Bent?: Families and Personality

Personality Traits and Their Identification

Traits are the enduring themes of our lives. In Robert McCrae and Paul Costa's summary of longitudinal studies of adults, one of the impressive findings was the consistency of personality over the adult years (McCrae & Costa, 1990). The top scorers on a given trait stayed high; the lowest scorers stayed low. For instance, the least shy members of any group studied remained more sociable than others over the years, and the most painfully shy remained relatively more shy than others. Although at high school reunions we easily slip into our old relationships—and perhaps thus overestimate the endurance of traits—we cannot help being struck by how people who have particular traits manage to maintain them and find social niches compatible with their personality dispositions and interests. In California, the words "personal growth" hold the promise of infinite change and variety, of discarding an old self like an old set of clothes; however, scientific evidence suggests that such recasting of the self is at best an extremely rare event. For those individuals prone to anxiety, panic, or depression, the inability to replace one personality trait with another is an impediment. On the other hand, stability makes us consistent social objects to others. It also allows us gradually to "know ourselves," and thus to find ways to satisfy the many complex requirements of our characters.

This book focuses primarily on traits rather than on specific behaviors, for several reasons. In human culture, technological and social innovation is a constant process, and new devices and behavior patterns are constantly being adopted and abandoned. Not long ago in historical time, buck-naked college students sprinted across campus lawns, asserting their