similarity of appearance and behavioral similarity: Testing an assumption. *Behavior Genetics, 6,* 343–351.
Mulaik, S. A. (1987). Toward a conception of causality applicable to experimentation and causal modeling. *Child Development, 58,* 18–32.
Neale, M. C., & Cardon, L. R. (1992). *Methodology for genetic studies of twins and families.* Dordrecht, The Netherlands: Kluwer.
Newman, J., Freeman, F., & Holzinger, K. (1937). *Twins: A study of heredity and environment.* Chicago: University of Chicago Press.
Pedersen, N. L., Plomin, R., McClearn, G. E., & Friberg, L. (1988). Neuroticism, extraversion, and related traits in adult twins reared apart and together. *Journal of Personality and Social Psychology, 55,* 950–957.
Plomin, R. (1990). The role of inheritance in behavior. *Science, 248,* 183–188.
Plomin, R., & DeFries, J. C. (1985). *Origins of individual differences in infancy: The Colorado Adoption Project.* New York: Academic Press.
Plomin, R., DeFries, J. C., & McClearn, G. E. (1990). *Behavioral genetics: A primer* (2nd ed.). New York: W. H. Freeman.
Plomin, R., Willerman, L., & Loehlin, J. C. (1976). Resemblance in appearance and the equal environments assumption in twin studies of personality traits. *Behavior Genetics, 6,* 43–52.
Rende, R. D., Plomin, R., & Vandenberg, S. G. (1990). Who discovered the twin method? *Behavior Genetics, 20,* 277–285.
Rowe, D. C. (1983). Biometrical genetic models of self-reported delinquent behavior: A twin study. *Behavior Genetics, 13,* 473–489.
Rowe, D. C., Clapp, M., & Wallis, J. (1987). Physical attractiveness and the personality resemblance of identical twins. *Behavior Genetics, 17,* 191–201.
Rowe, D. C., & Gulley, B. (1992). Sibling effects on substance use and delinquency. *Criminology, 30,* 217–233.
Rowe, D. C., & Plomin, R. (1981). The importance of nonshared (E_1) environmental influences in behavioral development. *Developmental Psychology, 17,* 517–531.
Rowe, D. C., & Waldman, W. (1993). The question "how" reconsidered. In R. Plomin & G. McClearn (Eds.), *Nature, nurture, and psychology* (pp. 355–373). Washington, DC: American Psychological Association.
Scarr, S., & Carter-Saltzman, L. (1979). Twin method: Defense of a critical assumption. *Behavior Genetics, 9,* 527–542.
Scarr, S., Scarf, E., & Weinberg, R. A. (1980). Perceived and actual similarities in biological and adoptive families: Does perceived similarity bias genetic inferences? *Behavior Genetics, 10,* 445–458.
Scarr, S., & Weinberg, R. A. (1976). IQ test performance of black children adopted by white families. *American Psychologist, 31,* 726–739.
Scarr, S., & Weinberg, R. A. (1983). The Minnesota adoption studies: Genetic differences and malleability. *Child Development, 54,* 260–267.
Scott, J. P., & Fuller, J. L. (1965). *Genetics and the social behavior of the dog.* Chicago: University of Chicago Press.
Shields, J. (1962). *Monozygotic twins brought up apart and brought up together.* London: Oxford University Press.

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

freedom from social convention in the short-lived fad of "streaking." Today, college students are now pursuing other (more serious?) endeavors. Who became the streakers was certainly partly a function of individual personality—extraverts were probably more likely to do so than introverts. In this brief period, streaking was probably a good behavior to use to diagnose extraversion; conversely, we might have predicted that extraverts were more likely to streak. Although this behavior has now virtually ceased to exist on college campuses, extraverts still enliven parties, and introverts still avoid the social limelight. Indeed, there is little evidence that the distribution of the underlying personality dispositions has changed appreciably in a single generation, although the particular behaviors used to express a given disposition may change rapidly. The greater constancy and breadth of traits are good reasons to focus on their inheritance, rather than on the transmission of more molecular single "behaviors" or on the transmission of cultural artifacts. In Chapter 7, I discuss further the cultural transmission of both traits and specific cultural innovations.

Traits are usually inferred from clusters of behaviors that "hang together," correlating with one another. Figure 3.1 shows a general model of sociability. The large circle represents the trait itself, which is presumably a result of nervous system activity. The indicators in boxes on the right are the behaviors from which its existence has been inferred. The amount of the disposition to sociability depends on the presence or absence of these indicator behaviors. A sociable person should attend parties, talk to strangers in the supermarket, break into discussions at a business meeting, enjoy entertaining business associates, and so on. If we could pay a detective to trail people for several weeks, we would find that sociable people would display more of these behaviors during the time they were followed than shy people would. In this diagram, the arrows from the trait point to these behaviors because the trait is conceptualized as *one* cause of each behavior. I do not regard the use of a trait in a causal explanation as tautological: "When we call someone 'friendly' or 'aggressive' or 'generous,' we are saying something about how the person behaves (or would behave) in certain kinds of situations *and* about the functioning of his or her mind" (Funder, 1991, p. 32). For simplicity, the diagram omits other traits and immediate situational influences that are also causes of a behavior. Certainly, the behavior of breaking into a discussion at a business meeting would have other causes, including other traits (such as whether the boss was feared) and immediate situational influences (such as whether the individual cared about the issue under discussion).

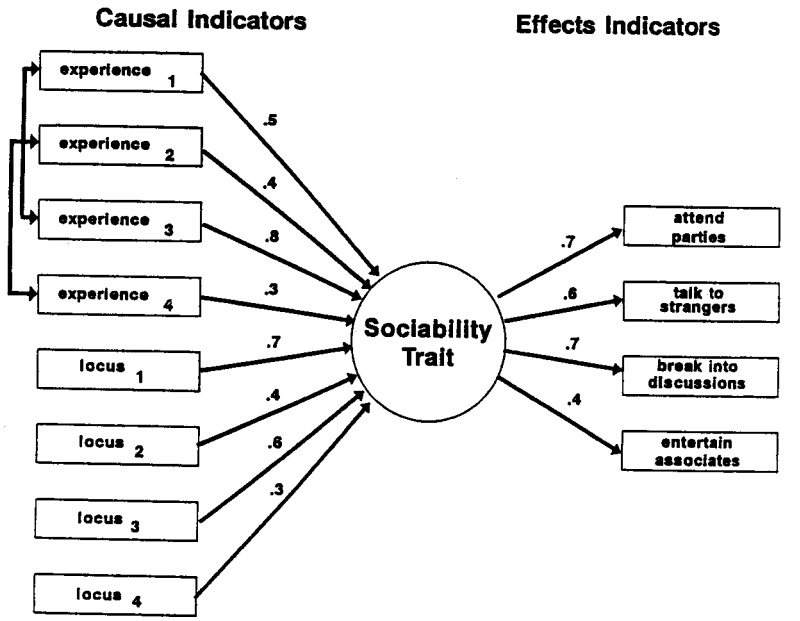

FIGURE 3.1. A model of sociability.

Many trait descriptions are the result of a factor-analytic investigation. Factor analysis is a statistical technique of grouping behaviors that occur together and inferring from these groupings the existence of underlying trait dispositions. The technique uses correlation matrices of many variables and reduces them to fewer underlying variables, which are correlated with each observed variable. In a correlation matrix of weight and height measurements—say, of arm length, waist size, shoulder width, head size, and neck size—factor analysis would quickly identify the underlying trait of *body* size. Some variables would correlate more strongly with the underlying body size trait than other variables; for instance, arm length is probably a better indicator than head circumference is.

The use of factor analysis has been attacked by some critics of personality research. In his book *The Mismeasure of Man*, the Harvard biologist Stephen Jay Gould (1981) noted that factor analysis can group indicators that have no underlying source of causality. Thus a single sta-

tistical factor could be found among the positive correlations since 1960 of stock market prices, the universe's expansion, the price of gold, and U.S. population size because all grow with time, not because a single cause links these disparate events. In particular, Gould objected to the inference that because behaviors tend to intercorrelate positively, they must imply a trait within the person as something physical and real in the brain. He asserted: "Factorists have often fallen prey to a temptation for *reification*—for awarding *physical meaning* to all strong principal components" (p. 250; emphasis in the original). Although Gould directed his criticism at IQ tests, his remarks apply equally to any trait inferred from statistical data.

Although the psychologist Gordon Allport (1966) also expressed reservations about factor analysis, he had little doubt about the existence of traits. Without a stable source of influence within the person, how can behavior remain stable over 20- and 30-year periods? If there is not some organizing source within the person, how can different observers come to see an individual in the same way (Kenrick & Funder, 1988)? Allport acknowledged that when people sweat outdoors, they do so because the temperature is hot. Nonetheless, the mechanism of behavioral adaptation lies within the individual: Evolution has given humans nerve sensors to detect temperature change and sweat glands to release cooling fluids. Dogs, with an absence of sweat glands in the skin, fail to adapt to temperature changes by sweating. The basic trait of sweating does reflect something real and physical in the design of human glands and the nervous system.

Allport failed to offer perhaps the strongest justification for identifying traits with brain structure and function: the behavioral resemblance of reared-apart MZ twins. When two separated MZ twins enter a laboratory and begin checking similar self-descriptions on a 400-item questionnaire, one has to wonder what source of resemblance could be responsible other than neurological similarity. The laws of physics do not permit extrasensory perception or brain-to-brain telecommunication; the source of reared-apart MZ twins' behavioral resemblance must lie within their nervous systems. (I have noted in Chapter 2 that the hypothesis of similarity in physical appearance is a red herring.) In one of the Minnesota twin study's well-publicized cases, one twin was reared in Czechoslovakia as a Nazi and the other in Trinidad as a Jew (Begley & Kasinborf, 1979)! Despite these nearly opposite life histories, their answers on the Minnesota Multiphasic Personality Inventory (MMPI) were extremely

similar. Although some of their attitudes were different—the Jewish-raised twin was more liberal than the other—different life histories may still lead to behavioral expressions of similar trait dispositions that have one neurological foundation. Thus, although "reification" is a somewhat unpleasant-sounding word, it describes a proper activity—looking for the neurological roots of behavioral dispositions. And given the heritability of personality traits, biological foundations will be found eventually for many of them.

In Figure 3.1, the left-hand boxes show the causes of sociability. In statistical terms, they are "causal indicators" because their arrows point toward the latent trait, whereas the boxed behaviors are "effects indicators" because the latent trait's arrows point at them (Bollen & Lennox, 1991). The direction of causality is clearly the most important and defining difference between the two sets of indicators: We say that attending a party reveals the trait of sociability, not that it causes it. Another subtle difference has to do with the intercorrelations among the indicators. Positive correlations will be found among the effects indicators, and often these will be large. In this diagram, effects correlations can be calculated as the product of the numerical weights on their respective arrows. For instance, the correlation of "attend parties" and "break into discussions" is .49 (i.e., .7 × .7), and the average correlation among all pairs of effects indicators is .41. On the other hand, the weights on the causal indicators cannot be used to calculate the correlations among the causal indicators. Rather, their degree of intercorrelation is shown by the double-headed arrows, and causal indicators unconnected by double-headed arrows do not correlate. Two kinds of causal influences are shown: genes at various loci and specific experiences. The genes, being unlinked, do not all correlate with one another; this reflects the random assortment of genes, as described in Chapter 2. Some experiences may correlate, and others may be totally uncorrelated. Figure 3.1 thus visualizes the idea that uncorrelated and numerous influences may combine to produce a trait that is then inferred from its various manifestations (effects indicators). This book's thesis is that the experiential arrows in Figure 3.1 are neither child-rearing factors nor other environmental factors tied particularly to the family unit.

One difficulty is that we seek a general answer about family influence. The number of traits generated in the history of psychological research has been huge, and many traits going under different names may be similar. For instance, "self-esteem" may be defined as how much

a person respects and likes himself or herself, whereas "general anxiety" may be defined as a person's feeling tension and a knot in his or her stomach. Although on the surface the two concepts appear different, questionnaire measures of anxiety and self-esteem usually correlate negatively, making it unlikely that heritable influence on anxiety would be totally absent in a measure of self-esteem. Thus, part of the task is knowing the domain of traits. As each trait is investigated, we can determine whether it is heritable and whether it shows family environmental influence. As the number of traits showing the same pattern of genetic and environmental influence grows, the greater the strength of any generalization. If three, four, or five uncorrelated traits are investigated, and if they support a conclusion of genetic influence without composite shared environmental influence, the likelihood diminishes that any new trait that is named and discovered will be totally unrelated to the known traits. Thus, it becomes implausible that a new trait awaits discovery that will somehow reveal an entirely different pattern of genetic and environmental determination.

Behavior Genetic Studies of Personality Traits

The literature on the behavior genetics of personality is voluminous. But a new review of hundreds of twin and adoption studies is unnecessary to enable us to reach conclusions about personality variability. Instead, I rely on previous reviews and selective studies that have addressed issues commonly raised in this area relating to the strengths of the methods and their assumptions. One word of warning is in order: With the selection of a single study, just about any point can be made. Sampling variation is an inherent feature of the landscape of behavioral research—and it is often a serious problem, because investigators usually do not have the finances to study giant samples and employ representative sampling procedures that would minimize sampling variations. The practical science of opinion polling, however, has not foundered on the occasional miscalled election where the poll said that a losing candidate would win by a margin of a few percentage points. In the long run, opinion polling calls most elections correctly; in the long run, behavior genetic studies point in the direction of the underlying truths. When single studies are offered here, their results are representative of others in the field, and exceptions and qualifications are mentioned later.

In the personality field, a consensus has been reached that a "Big Five" set of trait dimensions spans the major naturalistic personality traits. These dimensions are found repeatedly in self-report questionnaires and in rating data (Digman, 1990). Many of the premier personality inventories—including Cattell's Sixteen Personality Factor Questionnaire (16PF), the Eysenck Personality Inventory, and the renowned MMPI—can be reduced to all, or a subset, of these five personality dimensions. The dimensions have been replicated in five language groups, from English to Japanese. They appear in studies from 1949 to the present. If these dimensions do not encompass the entire range of adult traits, they appear at least to capture a large portion of traits mentioned in everyday language as people describe one another. Each of the "Big Five" personality trait dimensions is named here according to one end of the continuum it represents:

1. Extraversion: traits such as "gregarious," "sociable," "dominant," and "adventurous."
2. Agreeableness: traits such as "kind," "affectionate," and "friendly."
3. Conscientiousness: traits such as "reliable," "organized," and "planful."
4. Emotional stability: traits such as "calm," "not worrying," and "stable."
5. Intellectual openness: traits such as "original," "insightful," "wide interests," and "inventive."

Reviewing the world's scientific literature on the "Big Five," we can reach general conclusions about the types of genetic and environmental influences operating to produce trait variation (Loehlin & Rowe, 1992; Loehlin, 1992). A colleague and I (Loehlin & Rowe, 1992) examined two kinds of family environmental influences. One was the environment shared by siblings (c^2), and the other was the environment shared by parent and a child, symbolized by the product pc. The first component of this product (p) represents the influence of parental phenotype on family environment; the second (c) is the effect of family environment on the child (the same environment that also contributes to siblings' resemblance).

Loehlin and I reviewed a heterogeneous set of studies that used neither exactly the same questionnaires, age groups, nor geographic

locations (indeed, some studies took place oceans apart). These included a diverse range of behavior genetic research designs: (1) the comparison of resemblances in MZ twins and DZ twins; (2) the comparison of adoptive and biological parent–child resemblances; (3) the comparison of adoptive and biological sibling resemblances; (4) the comparison of resemblances in the families of MZ twin pairs; and (5) the comparison of resemblances in MZ twins reared apart and together.

A sense of the richness of these data can be obtained by considering some observations for extraversion. In the extraversion data set, the sample-size-weighted correlation for MZ twins raised apart was .38; for MZ twins raised together, .55; for biological siblings, .20; and for all biologically unrelated siblings reared together in adoptive families, –.06. This pattern can be simply interpreted: *Individuals who share genes are alike in personality regardless of how they are reared, whereas rearing environment induces little or no personality resemblance.*

Data from twin-family studies elaborate these conclusions. In a twin-family study, adult MZ twins are recruited, and they are tested with their children. The unique genetic relatedness of MZ twins teases apart genetic and family environmental influences. For example, being a genetic duplicate, an MZ twin father should correlate as highly with his nephews and nieces as with his own children, because he correlates .50 genetically with both. The nephews and nieces, possessing the same genetic fathers, should correlate as highly as half-siblings, although they are raised as cousins (often in different towns or cities, with social contact mainly limited to holidays and special occasions). As shown in Table 3.1, the extraversion correlations we found (Loehlin & Rowe, 1992) followed lines of genetic relatedness. What family environmental mechanism could have generated correlations that were the same for parents and their own children as for uncles/aunts and the children of their brothers and sisters? What family environmental mechanism could have made cousins as alike as half-siblings? Only the adult MZ twins themselves showed a higher correlation (.43) than the first-degree biological relatives. Excluding the MZ twins, the overall heritability estimate (weighted by sample size) for Table 3.1 was .47, about the same as that estimated by the MZ twin correlation.

Table 3.2 summarizes our results for fitting the "Big Five" personality traits to various models (with the most information available on extraversion and emotionality). In the best-fitting models, the largest component of trait variation was unshared environment, followed closely by broad-sense heritability, while the component of siblings' shared

TABLE 3.1. Averaged Extraversion Correlations in Two Twin-Family Studies

	Mean r	No. of pairs	r_g[a]	Social relation
MZ twins	.43	116	1.00	Twins
Siblings in twin families	.23	177	.50	Full siblings
Twin parent to own child	.22	413	.50	Parent–child
Twin parent to brother's or sister's child	.21	192	.50	Uncle/aunt–nephew/niece
Cousins via MZ twins	.16	138	.25	Cousins

Note. Correlations reflect weighted average of two twin-family studies. Original sources: Price, Vandenberg, Iyer, & Williams (1982) and Loehlin (1986). Adapted from Loehlin & Rowe (1992). Copyright 1992 by Harvester Wheatsheaf. Adapted by permission.
[a] r_g is the relatives' genetic correlation.

environment was much smaller. The contribution of the rearing environment was statistically significant for all traits except extraversion, but accounted for only 2% to 9% of the total variation. The least important component was parent–child environmental influence: *The parameter* p *could be set to zero in all trait models.*

These results do not mean that the structure of personality trait variation is completely resolved. The statistical model just presented emphasizes nonadditive genetic influences as an additional source of MZ twins' behavioral resemblance. An alternative model is that MZ twins share a special environmental similarity that no other pairs of relatives experience. These two models, although differing in assumptions, gave nearly identical statistical fits to the observed correlations.

TABLE 3.2. Parameter Estimates for "Big Five" Personality Dimensions

Dimension	Unshared environment	Broad-sense h^2	Narrow-sense h^2	Siblings' shared environment
I. Extraversion	.49	.49	.32	.02
II. Agreeableness	.52	.39	.29	.09
III. Conscientiousness	.55	.40	.22	.05
IV. Emotional stability	.52	.41	.27	.07
V. Intellectual openness	.49	.45	.43	.06
Mean	.51	.43	.31	.06

Note. Adapted from Loehlin & Rowe (1992). Copyright 1992 by Harvester Wheatsheaf. Adapted by permission.

Although these statistical data were indecisive, I find the biological interpretation of genetic nonadditivity more intellectually compelling than one based on special MZ twin environments. A nonadditive trait's hallmark is an MZ twin correlation more than double that of first-degree relatives (e.g., siblings). Physical traits, such as brain midfrequency alpha level, show this property, correlating about .80 in MZ twins but only .13 in DZ twins (Lykken, McGue, Tellegen, & Bouchard, 1992). In addition, nonlinear combinations of ordinary traits may also correlate more than twice as strongly in MZ as in DZ twins. For instance, Lykken et al. (1992) found that the squared difference of height minus weight correlated .62 in MZ twins but only .15 in DZ twins. Thus, MZ twins were much more alike than DZ pairs in whether their weight was proportionate to their height. A further argument against the hypothesis of a special MZ twin environment is that MZ twins raised apart typically possess the same extraordinary resemblance for different traits as those raised together.

Lykken et al. (1992) have given this genetic nonadditivity a special name, "emergenesis"—that is, the "emergent properties of configurations of monomorphic genes." As mentioned in Chapter 2, unlike first-degree relatives, MZ twins share the entire *configuration* of their genes. For instance, a rare trait—say, charismatic leadership—conferred by a five-locus, recessive-gene system would appear only once in about 20 million random matings, but it would be shared by a pair of MZ twins. And whereas both MZ twins would possess world-class leadership skills, one would not be likely to find these in either their nontwin siblings or their parents. Thus, a trait can have a high degree of genetic determination without "breeding true" in families. As Lykken et al. (1992) remark, the random halves of genes from each parent may work additively, or may result in some unique new combination:

> Your tall mother held four queens, and she passed three of them along to you. Combining them (additively) with a queen from the paternal line, you can stand as tall as Mom. . . . The exciting thing about emergenesis is that you *might* receive the 10 and king of spades from Dad, and the jack, queen, and ace of spades from Mom, cards that never counted for much in either family tree but whose combination in you might produce a Ramanujan [a mathematical genius], a new Olympic record—or a True Crime miniseries for television. (p. 1575)

Or they might produce, as recounted by Lykken et al., a pair of reared-apart MZ twins who discovered, upon reunion, that they both used

Vademecum toothpaste, Canoe shaving lotion, Vitalis hair tonic, and Lucky Strike cigarettes.

Our model-fitting exercises (Loehlin & Rowe, 1992) were consistent in weakening the claims of the two major avenues of family environmental transmission of traits. First, there was a total absence of evidence that children resemble their parents in behavioral traits because some environmental process in the family transmits them from parent to child, whether that process be imitation, emotional identification, or anything else. The expectable parent–child resemblance would be merely one-half the *additive* (i.e., narrow-sense) heritability shown in Table 3.2. With mean additive heritability of .31, we would expect a rather modest parent–child correlation of about .16 for most personality traits. Note that the trait with the greatest additive heritability in Table 3.2, intellectual openness, is linked with the domain of intellectual ability (where, as we shall see later, additive genetic influences are more pronounced than they are for personality traits). Second, there was little evidence for environmental influences shared by siblings. This parameter estimate was weak, both in absolute terms and by comparison to unshared environmental influences. Children may grow up in one family, with many of the same objective experiences, yet they are nearly as unlike one another in personality as children reared in different families.

Family adoptions played a prominent role in our reaching these remarkable conclusions. The utter lack of familial resemblance in adoptive families—despite the early occurrence of adoption in infancy—directly implies that a family's emotional climate or parental example fails to set the direction of personality development. The socialization science view of strong family effects might be salvaged if parental treatments in adoptive homes were so different from those in biological families as to vitiate family influences. Hoffman (1985, 1991) suggests that merely knowing a child is adopted may place personality development on a different course:

> Simply knowing [that a child is] adopted may lessen the parents' efforts to mold the child to their own image either because the parents' identification with the child is less or because the parents feel more of an obligation to let the adoptive child develop independently. (1985, p. 132)

On the other hand, this response to adoption is speculative and *post hoc*. No one has demonstrated that adoptive families have parenting styles so original and different as to separate them from the normal

channels of socialization. In the Colorado Adoption Project, adoptive families were not exactly the same in rearing styles as the biological families with which they had been matched (Plomin, DeFries, & Fulker, 1988, pp. 73–74). For example, the adoptive families were more religious (the adoptees had been placed through a church-affiliated adoption agency) and exercised greater control over their children's behavior. Less family conflict also existed in the adoptive homes. The adoptive parents did not treat their children with greater warmth than the nonadoptive ones, however, and these *mean* group differences accounted for only a small part of the total variation in rearing styles. Even if adoptive parents could invent new rearing approaches, it is unclear that such innovations would necessarily lead to less parent–child resemblance. For instance, adoptive parents who express a lack of concern with whether their children strive academically (because they supposedly value the adoptive children's independent development) may nonetheless model a high regard for academic achievement in their own behavior (e.g., attending parent–child conferences, participating in school fund–raising activities) that belies their overt beliefs. One can more easily imagine the intentional socialization of behavior in some different direction than a complete absence of such mechanisms as modeling and imitation in adoptive families as opposed to biological families.

The Texas Adoption Study provides some unique data with which to evaluate these two different views (Loehlin, Willerman, & Horn, 1987). We have here the strong behavior genetic design of comparing biological and adoptive children raised in the same households; this should eliminate quarrels about whether two different sets of families are well matched. We also have rare personality data on the birth mothers of the adoptees. As noted in Chapter 2, the families recruited into this study all adopted children through a private agency in Texas; fortunately for research, the agency routinely administered personality and IQ tests to the unwed mothers before their children's birth. With these records available, the adoption design was completed by locating the adoptive families and administering tests to both the parents and children. About 40% of the adoptive families had biological children of their own, born either before or after the adoptive placement (contrary to popular mythology, adoption does not cure infertility, but some subfertile parents are able eventually to have a biological child). All the children were first tested at an average age of 7 years. In the initial round of the study, few personality resemblances were found in either biological or nonbiological comparisons; of course, however, these young children could not

complete the same personality inventories as were administered to adults. In a follow-up, children from 181 of the 300 families in the original study were recontacted; at this point, the adoptive children averaged about 17 years old. Their birth mothers had been on average just 2 years older (19 years old) at the time of the births. Thus, we can ask this question: Did the adoptees become like their birth mothers while living apart from them? These older adoptees were also able to complete the adult personality inventories.

I illustrate the Texas Adoption Study's general conclusions with the specific results for the MMPI. Developed at the University of Minnesota during the 1950s, the MMPI is widely known to clinicians, and it now enjoys wide use throughout the United States. Its nine scales—tagged with titles such as Hypochondriasis, Hysteria, and Schizophrenia—were originally intended for the diagnosis of psychopathology. Nonetheless, the inventory can also be used with normal populations, and response variability is great.

The Texas Adoption Study presents nearly an ideal design for evaluating familial effects on the MMPI. The adoptees were placed within a few days of birth and were permanently adopted; the adoptions were closed, so that adoptive parents did not have contact with the adoptees' biological relatives; nor could the adoptive parents be aware of the birth mothers' MMPI outcomes. Selective placement was minimal: The median correlation of the MMPI clinical scale scores from the birth mothers to the adoptive mothers and fathers were only .03 and .00, respectively.

Table 3.3 summarizes median correlations taken over eight MMPI scales. When relatives lacking biological relatedness were compared,

TABLE 3.3. Median MMPI Scale Correlations for Biologically Unrelated and Related Children

	Adoptive child	Biological child
Adoptive father	.02 (180)	.12* (81)
Adoptive mother	.00 (177)	.12* (81)
Adoptive midparent–midchild	.03 (135)	.24** (61)
Birth mother	.18** (133)	—

Note. *n*'s are in parentheses. Each correlation is a median of correlations taken over eight MMPI Scales. Data are taken from Loehlin, Willerman, & Horn (1987).
*$p < .10$.
**$p < .05$

MMPI scale scores did not correlate. The median midparent (i.e., average of mother's and father's score)–midchild correlation was just .03 (n = 135). Thus, rearing influences were negligible. In contrast, when relatives possessed biological relatedness—that is, a birth mother and her adopted-away child, or an adoptive mother and her own biological child—correlations were positive. Although the small sample size does not make the adoptive parent–biological child correlation of .12 statistically reliable, it is well within sampling variation of our expected value of a familial correlation for nonintellectual personality traits (.15). Heritability can be calculated as either the midparent–midchild correlation (h^2 = .24) or as twice the correlation between the birth mother and adoptee (h^2 = .36).

These data constitute a direct response to Hoffman's (1985, 1991) suggestion that adoptive parents treat adoptive and nonadoptive children differentially. It is true that an adoptive parent resembles only one kind of child in his or her own family, the biological child. And the Texas adoptees' MMPI traits could not be predicted from the adoptive parents'. Yet the adoptees' MMPI traits could be predicted from their *birth mothers'* MMPI traits (r = .18, n = 135). Thus we fully recover the fully expected degree of familial resemblance once we have information on a biological parent—even a biological parent whose contact with her child was limited to a few hours or days after birth. These observations lead strongly to the inference that what creates parent–child resemblance in natural families is biology, and that no process of imitation, modeling, or emotional identification is required to induce it. Table 3.4 drives this point home by showing birth mother–adoptee MMPI scale correlations. What we see here is the same amount of personality similarity as exists with shared rearing experiences (Hill & Hill, 1973).

The comparison of twins raised apart and together is another natural laboratory for weighing rearing influence. As noted earlier, the samples of twins raised apart tend to be more haphazard and idiosyncratic than those of twins raised ordinarily. The Minnesota study of twins reared apart also provides data on the resemblance of twins reared together, who completed the same physiological tests and personality inventories as the separated twins (Bouchard, Lykken, McGue, Segal, & Tellegen, 1990). The amount of variance on each measure that could be attributed to siblings' rearing was obtained by subtracting the raised-apart twins' correlation from the raised-together twins' correlation. As illustrated in Table 3.5, estimates of rearing influences were very low across a broad range of physical and personality measures. Indeed, in no case

TABLE 3.4. MMPI Correlations between Mothers and Children When Children Raised Apart

MMPI scale	r
Hypochondriasis	.06
Depression	.26
Hysteria	.13
Psychopathic Deviate	.27
Paranoia	.07
Psychasthenia	.18
Schizophrenia	.28
Hypomania	.17
Median	.18
Pairs	133

Note. From Loehlin, Willerman, & Horn (1987). Copyright 1987 by the American Psychological Association. Reprinted by permission.

did the difference between the separated and unseparated twins attain statistical significance—the correlations differed to within what sampling variation would allow.

No one can deny that occasional evidence for shared environmental influences crops up in twin and adoption studies. The evidence regarding parent-to-child influence, however, must be viewed in light of the many disconfirmations of such influences over a broad range of traits. The occasional statistically significant correlation between an adoptive parent and child may be merely a case of sampling chance. Indeed, the weight of evidence suggests that a higher standard of proof is needed for putative shared environmental influences than for putative genetic ones: The shared environmental result should be replicated across several studies before one begins to think about a family environmental mechanism to account for it.

What about that small rearing influence that is occasionally significant for some traits? Does this mean that something is going on environmentally in the family? Perhaps not, because the evidence for c^2 is strongest when data from MZ twins are included in the models discussed earlier. Environments may be able to induce resemblance when genetically identical people are exposed to similar circumstances or when twins and siblings are able to influence one another directly. In one of my studies of teenagers, I found that nontwin siblings who spent time with

TABLE 3.5. Resemblance in MZ Twins Reared Together (MZT) and Apart (MZA): The Minnesota Study of Twins Reared Apart

Measure	MZA	MZT	Variance attributable to shared environment
Fingerprint ridge count	.97	.96	–.01
Height	.86	.93	.07
Weight	.73	.83	.10
Brain alpha activity (two measures)	.80	.81	.01
	.80	.82	.02
Systolic blood pressure	.64	.70	.06
Heart rate	.49	.54	.05
Mean of 11 Multidimensional Personality Questionnaire scales	.50	.49	–.01
Mean of 18 California Personality Inventory Scales	.48	.49	.01
Mean of 23 Strong–Campbell Vocational Interest Inventory scales	.39	.48	.09
Mean of 17 Minnesota Occupational Interest Inventory scales	.40	.49	.09

Note. Adapted from Bouchard, Lykken, McGue, Segal, & Tellegen (1990). Copyright 1990 by the American Association for the Advancement of Science. Adapted by permission.

the same friends were more similar in smoking and drinking behavior than were siblings who did not spend time with the same friends (Rowe & Gulley, 1992). It is not particularly surprising that an older sibling or the friend of such a sibling can give a cigarette or beer to a younger brother or sister. Adult twins who stay in contact with each other may frequent the same "watering holes" or teetotaling social groups, and so may influence each other's drinking. Twins and nontwin siblings may sometimes also be "partners in crime," committing delinquent acts together; sibling correlations for delinquency are greater than for other traits (Rowe & Gulley, 1992). In a sample of reared-apart MZ twins, time spent in direct social contact was also associated with personality similarity, independently of the twins' age or age at separation (Rose, Kaprio, Williams, Viken, & Obremski, 1990). These contemporaneous sibling influences, however, may not be mechanisms of long-term socialization, because their effects depend on siblings' immediate social contacts—influences that may dissipate once contact is lost. An observation of

contemporaneous environmental influences would not be proof that *rearing* environmental variation matters.

This review has slighted some areas of research that should be mentioned before I move on to studies of psychopathology. In an extensive series of behavior genetic studies, Robert Plomin and Arnold Buss (Buss & Plomin, 1984) have investigated childhood temperament, with particular emphasis on the traits of sociability, activity, and emotionality. In twin and adoption studies, these traits, as aspects of childhood personality, show a broad-sense heritability of .40 to .50. DZ twin correlations are often very low in childhood, sometimes even negative. Although additive genetic variation may be less important in childhood than in adulthood, a more plausible interpretation is that parents, who make these personality ratings, may contrast their children while rating them—scoring them as a bit more different than they are in actuality. The small sample size of many childhood twin studies also contribute to estimates' instability. On environmental effects, the twin and adoption studies of young children are in close agreement with the adult studies: There is little evidence of family environmental influences on temperamental traits assessed during childhood.

Another research focus is the issue of longitudinal stability versus change in personality. As Francis Galton (the English polymath who was the founder of modern behavior genetics) observed, twins who were alike at birth fail to develop great dissimilarities later in life, despite the accumulation of different experiences (Galton, 1876). I have concluded that directional change in personality traits is usually attributable to unshared environment, because MZ twins do not show a more similar profile of personality *change* than DZ twins (Rowe, 1987). That is, one MZ twin may become more sociable between the first time he or she takes a personality test and the second time, a year or a few years later. But the direction or amount of change in this MZ twin should *not* predict the direction or amount of personality change in his or her cotwin.

Conversely, "stability" refers to the tendency of individuals to maintain their same rank order on a trait dimension over time. It is apparently the result of genetics, as genetic factors continue to influence a trait throughout life. In my earlier article, I acknowledged that definitive proof could only come from lifelong studies of twins. Nonetheless, I asserted:

> The similarity of adult twins reared together, who have lived the greater part of their lives apart, is just a bit less surprising than the resemblance

of reared apart twins. Certainly, these results hint at a genetically driven stability to adult personality. (1987, p. 222)

Behavior Genetic Studies of Psychopathology

Problems of nomenclature and breadth of behavioral traits apply to psychopathology as well as to normal traits. One way to divide this domain is as follows: schizophrenia, mood disorders (bipolar disorder and unipolar depression), externalizing disorders (e.g., aggression and conduct problems), and internalizing disorders (e.g., high levels of anxiety). In this section, I briefly review relevant evidence on the influence of family environments in each of these types of disorders.

Schizophrenia

In both the popular mind and scientific circles, schizophrenia is the most devastating type of mental illness, with its great severity and sometimes bizarre symptomatology. Affecting about 1% of the population, schizophrenia includes such symptoms as an inability to form lasting emotional ties with friends and spouses; an absence of normal emotional responses to events; and delusions and hallucinations (usually auditory). I heard a women display symptoms of schizophrenia on one radio talk show when she complained that the government was listening to her while she shopped at the local supermarket. She thought that a large radar antenna at a local military base had been focused on her as she moved about the store. Although her beliefs were wildly delusional, her conversation with the talk show's host was otherwise quite reasonable. This example holds a bit of humor, but the life course of seriously ill schizophrenics can be tragic; their inability to hold jobs or to form lasting emotional bonds leaves them outside the friendships and pleasures of everyday life. Indeed, schizophrenics appear in disproportionate numbers in the population of the homeless and destitute in the United States.

During the dominance of Freudian influence from the 1920s to the 1950s (Torrey, 1992), the blame for schizophrenia was often placed on the "schizophrenogenic mother"—a women lacking in any emotional resonance toward her child. Postulating the existence of such a parent was reasonable, because about 10% of the offspring of one schizophrenic parent are affected with schizophrenia. And when observed in clinic or

interviewed, a mother with schizophrenia does not exhibit ideal parenting styles: She often lacks normal affect toward her children because she lacks emotional response in general, and the household is disorganized because she herself is disorganized in her behavior. Few would deny that a schizophrenic mother violates the tenets of good parenting; if there is a family environmental influence able to "mess up" children, it should be this one.

Yet the "schizophrenogenic mother" explanation of psychopathology is dead. The chinks in the explanation were always there: The majority of the children of such mothers failed to display psychopathology, and the ability of many children to develop normally in a hostile environment was impressive. Consider this story of a schizophrenic mother with three children (Segal & Yahraes, 1979). The mother said she was poisoning the food. The oldest child, a girl, tended to believe her, and so she refused meals. A second daughter would eat when the (normal) father was present in the home. But the youngest child, a 7-year-old boy, noted that "I'm not dead yet," and continued to accept the meals produced by a delusional mother. The so-called "shared" family environment was experienced very differently by the children in this family.

But what of the evidence for the familial effect—the 10% incidence in the children of one schizophrenic parent? I believe that this similarity of parent and child is genetically induced. Pooling data across adoptive studies yields a rate of schizophrenia in the children of a schizophrenic biological parent who are then relinquished and raised by normal adoptive parents of about 1 in 10 (DeFries & Plomin, 1978). Yet this rate is about the *same* as that of children who are raised with a schizophrenic parent (9.4%, n = 1,678; McGue & Gottesman, 1989). Thus, adding the environmental disadvantage to the genetic one does not increase risk over what is already seen with genetic disadvantage alone. In contrast, genetic disadvantage alone does elevate risk (10%, vs. 1% in the general population). More complex model-fitting analyses also confirm the absence of rearing influence attributable to schizophrenic disease in a parent (McGue, Gottesman, & Rao, 1985).

Although its generalizability is limited by a small sample size, a study of the families of Danish MZ and DZ twins who were discordant for schizophrenia (i.e., one twin in each pair had a diagnosis of schizophrenia, while the other had no mental illness) illustrates the subtlety of the environmental influences (Gottesman & Bertelsen, 1989). More of the normal twins (64%) had children than the abnormal twins (29%), consistent with schizophrenics' having fewer children than other people.

Rates of diagnosed schizophrenia in the 150 offspring followed lines of genetic relatedness. As many offspring of MZ twins who displayed schizophrenia (16.8%) were diagnosed as offspring of MZ cotwins who did *not* display the illness (17.4%). A lack of illness in a parent failed to reduce the risk to children who were at risk genetically, as described in this brief case history: One twin developed schizophrenia after the birth of her second child, at age 25 years. Her cotwin also had two children, but was described as completely normal throughout her life. The cotwin's daughter developed paranoid schizophrenia, the same diagnosis as her affected aunt. No other child was affected. In the DZ pairs, more schizophrenia was evident in the children of the twins displaying schizophrenia (17.4%) than in the children of the cotwins who were free of the illness (2.1%), because the latter presumably had fewer genes tending toward schizophrenia.

These data, like the main findings of combined twin and adoption studies, show that the putative environmental effect is not a shared one (i.e., child-rearing practices), in which one would see an increase in schizophrenia risk to the children of an affected MZ twin. Rather, the environmental influences were *unshared* ones uncorrelated with rearing environment (they were just as prevalent in the "bad" homes as in the "good" homes). Whatever these influences were, they caused a schizophrenia genotype to go unexpressed in some individuals—so-called "false negatives," who appeared outwardly normal, but still carried some genes that put their own offspring at risk for the disease. These unshared environmental influences may have had nothing whatsoever to do with stresses in individuals' social environments. The source of discordance in psychopathology could have been accidents of embryological development, exposure to viruses, or some other biological process differentiating the MZ cotwins during their development. In a study using brain imaging methods, affected MZ twins had larger ventricular areas (which contain spinal fluid but not nerve cells) and smaller anterior hippocampi (a brain area associated with the ability to form immediate memories) than their unaffected cotwins (Suddath, Christison, Torrey, Casanova, & Weinberger, 1990). Thus, a defect in the brain has been associated with schizophrenia in an affected twin of a discordant pair, suggesting that the psychopathology originates in some biological process or processes that have damaged one twin more than the other.

Given these data and other adoption data on schizophrenia, it is clear that exposure to a schizophrenic parent is not critical for the development of the illness. As Gottesman (1991) observed, "Both the necessity and the sufficiency of the specific kinds of schizophrenogenic environments provided by schizophrenic parents have been weakened by the adoption results. Recall that almost 90 percent of schizophrenics do not have schizophrenic parents" (p. 149). He cautions, however, that the adoption results permit gene × environment interactions, whereby children with schizophrenia-disposing genotypes would be more susceptible than nondisposed children to particular family influences (but not ones unique to schizophrenic parentage).

A Finnish adoption study (Tienari et al., 1990, 1991) searched for interactions between schizophrenia-disposing genotypes and family environments. Their research design compared children of schizophrenic biological mothers adopted by nonrelatives with a case-by-case matched group of adoptive children of normal biological mothers. As in other adoption studies, rates of psychotic disorders were elevated only among adopted-away children of schizophrenic biological mothers (9.3% in index adoptees vs. 1.1% in controls; Tienari et al., 1990). This result again shows how the transmission of disposing genes may increase schizophrenia risk.

In all adoptive families, environmental quality was assessed through lengthy home interviews leading to clinical evaluations of family mental health. Healthy adoptive families were those in which conflicts were rare, anxiety and depression were mild, and role functioning was appropriate to a family's stage in the life cycle. The most disturbed adoptive families either had major unresolved conflicts or were openly chaotic. Poor mental health functioning in the adoptive families was associated with the degree of psychiatric disturbance in the adoptive offspring. An interaction between genetic background and family environment held, because this relationship was stronger for the genetically disposed index adoptees (who had schizophrenic biological mothers) than for the control adoptees. Of the index adoptees with mentally healthy adoptive families, 3.5% had psychotic spectrum mental disorders, as compared to 62.2% of those with severely disturbed adoptive parents.

In summary, these adoptive findings indicate a possible interaction between genotype and family environment for the development of psychosis. Two cautions must be mentioned, however. The majority of adoptive offspring in the Finnish study were adults when their adoptive families were interviewed. Hence, the study did not start prior in time to the offspring's development of psychiatric illness. This research design means that the direction of causality is a concern; it is possible that a severely disturbed child may harm an adoptive family's mental health

status, rather than vice versa. Second, the interviewing clinicians were aware of the offspring's mental health status, which may have confounded their reports of family mental health status. If the Finnish study leads to replications of the same type, then an exciting possibility of gene × environment interactions for psychosis would be assured. At least in this behavioral domain, familial influences may interact with children's genetic dispositions.

Although the genetics of schizophrenia fall outside this chapter's main focus, I present a brief summary of these results in Table 3.6. As would be expected for a familial genetic disorder, the risk to the relatives of affected individuals closely reflects their genetic relatedness. Still at issue is the exact mode of inheritance of schizophrenia (e.g., do genes with major effects exist?). Reviewing this evidence, McGue and Gottesman (1989) concluded that the evidence is most consistent with a polygenic model, because the nonlinear decline in concordance with degree of genetic relatedness is best satisfied by a multigene model. Nonetheless, the last word has not been heard on the issue, and a search is underway to find large-effect schizophrenia genes by means of linkage analysis with molecular genetic markers.

Mood Disorders

Two mood disorders constitute another major branch of mental illness. The two disorders are unipolar depression, which manifests itself as cyclic periods of severe depression; and bipolar disorder, in which depression cycles with mania (a state of high energy, euphoria, and sometimes delusional beliefs). Lifetime prevalences are greater for unipolar (about 6%) than for bipolar (about 0.5%) illness, with prevalences greater in women than in men (Tsuang & Faraone, 1990). Mood disorders have an unusual family correlate—greater creativity among the normal or mildly disturbed relatives of psychiatrically ill individuals. Greater creativity may be a biologically adaptive advantage conferred by the genes for mood disorders, when their number is below the threshold for severe illness (Andreasen, 1978; Richards, Kinney, Lunde, Benet, & Merzel, 1988).

Family, twin, and adoption data on mood disorders are less extensive than those on schizophrenia. Reviews in the area, however, suggest conclusions in accord with the schizophrenia findings (Moldin, Reich, & Rice, 1991; Tsuang & Faraone, 1990). Adoption studies demonstrate

TABLE 3.6. Schizophrenia Rates among the Relatives of Schizophrenics

Familial relationship	Sample size	% affected
Monozygotic twins	106	44.3
Offspring of two schizophrenic parents	134	36.6
Dizygotic twins	149	12.1
Siblings	7,523	7.3
Offspring of one schizophrenic parent	1,678	9.4
Half-siblings	442	2.9
Nieces or nephews	3,965	2.7
Grandchildren	739	2.8
First cousins	1,600	1.6
Spouses	399	1.0

Note. Sample size adjusted for age–risk curve (see Gottesman, Shields, & Hanson, 1982). Adapted from McGue & Gottesman (1989).

some familial genetic influence on both disorders. To date, the attempts to identify specific genes associated with the disorders through linkage analysis have been unsuccessful. Common family environmental influences are not suggested in the adoptive outcomes.

The twin data on mood disorders, however, are inconsistent with this book's thesis. The DZ twin correlations are more than one-half the MZ ones, suggesting considerable rearing influence. Tsuang and Faraone (1990) estimate that about 40% of variation in mood disorders is attributable to rearing.

At first glance, we would seem to have a puzzling exception to the general rule of a lack of influence from family differences. The growing skepticism about this influence, based on the general studies of personality that have been reviewed already, suggests looking for other possible explanations before too readily accepting some form of rearing experience here. One possibility immediately presents itself: nonrandom mating. Unhappy people preferentially marry each other. This does not mean that the unhappy necessarily prefer one another; the unhappy may have fewer choices in the marriage "marketplace" if happy people reject them. Nonrandom mating effects can mimic rearing influence in twin studies, because matched matings tend to increase the DZ twin correlations but cannot affect the MZ ones (because MZ twins cannot be made more alike genetically than they already are, whereas nonrandom mating tends to bring similar genes together in siblings). Given that the twin estimate of common sibling environment (c^2) is $2r_{DZ} - r_{MZ}$ (see chapter 2), a

greater DZ correlation tends to be "read" as shared environment even when it is induced by assortative mating. Thus Tsuang and Faraone (1990) conclude:

> The effects of common [shared] environment will be overestimated and those of heritability underestimated in the presence of assortative mating. Thus, the true magnitude of the genetic effect is likely to be larger than variance components suggest, because assortative mating is common among patients with mood disorders. (p. 91)

Externalizing Disorders

In childhood and adolescence, poor attention span, high activity levels, and conduct problems (i.e., disobedience, aggression) are more prevalent in boys than in girls. As with the major mental illnesses, a single diagnostic category may conceal considerable heterogeneity in pathways of genetic and environmental causation. Moreover, although these disorders often co-occur, they are imperfectly correlated. Some cases exist in which problems with attention span or activity level only are displayed, without co-occurring conduct problems; other cases exist in which conduct problems co-occur with normal attention span. The strong relationship of these "externalizing" behaviors—so called because these behavioral problems are easily seen by teachers and parents—to later crime and delinquency makes them of major concern in crime-ridden U.S. society.

Although behavior genetic studies of this disorder are not numerous, high-quality twin and adoption studies can serve to illustrate the absence of rearing influences. The twin study was completed in England by Goodman and Stevenson (1989), using 102 MZ and 111 DZ twin pairs. Parents, teachers, and observers provided ratings of the twins' hyperactivity. Table 3.7 presents the correlations for DZ twins and two kinds of MZ twins. One group consisted of MZ twins diagnosed as MZ on the basis of a standard questionnaire[1] (including questions such as "Are your twins as alike as two peas in a pod?"), but believed to be DZ by their parents. The other MZ twins were twins diagnosed as MZ and identified by their parents as MZ. (It may seem surprising that parents may not know what kinds of twins they have; one reason is that parents may be provided incorrect information at the time of their twins' birth, because delivery room doctors and nurses room must base their opinions about twin type on placental tissue, which can be unreliable for this

TABLE 3.7. Mean MZ and DZ Twin Correlations for Inattentiveness and Hyperactivity

Group	Twin *r*	No. of pairs
Recognized MZ	.62	64
Unrecognized MZ	.53	22
All MZ	.58	93
All DZ	.23	98

Note. Correlations reflect unweighted averages over different data sources in all categories of twins. Seven MZ pairs whose parents were uncertain or in conflict over twin classification were grouped with all MZ twins. Adapted from Goodman & Stevenson (1989). Copyright 1989 by Pergamon Press. Adapted by permission.

purpose.) The importance of this comparison is that it tested whether a *parental belief* that twins were MZ can make them more alike in behavior—a labeling bias influence, so to speak. As shown in Table 3.7, both kinds of MZ twins are about twice as similar as the DZ twins. The estimate of rearing influence was also effectively zero ($c^2 = -.12$).

Because twins experience high rates of premature birth, they constitute an ideal population for studying whether birth traumas have developmental consequences. Goodman and Stevenson (1989) examined this question as well. An MZ twin with a low birth weight (below 2,000 grams), however, was not more likely to be hyperactive than the genetically identical cotwin with a high birth weight. Regarding this discovery, Goodman and Stevenson commented: "In the absence of 'hard signs' of structural brain damage, childhood hyperactivity is unlikely to be the result of perinatal adversity, *even* if the child was at high perinatal risk, *e.g.* as a low birth weight twin" (1989, p. 707).

As in other samples, poor parenting practices were associated with hyperactivity in this study. Yet, given the lack of evidence for rearing influences from the twin analyses, the causality of this association must be viewed skeptically, as Goodman and Stevenson observed: ". . . hyperactivity is more likely to be a cause than a consequence of distorted family relationships" (1989, p. 706). Or both could be attributable to genes shared by parent and child—a possibility to which I will return in Chapter 5.

Parental treatments fared no better when evaluated in a study of adoptions. Jary and Stewart (1985) used a full adoption design (although information could not be obtained on *all* biological parents of the adoptees) and a comparison group of nonadopted children. All the chil-

dren were diagnosed as conduct-disordered. More antisocial traits appeared in the biological parents than in the adoptive parents, in whom these traits were nearly absent. For example, 0% of the adoptive fathers were diagnosed as antisocial, as opposed to 11% of the adoptees' biological fathers and 14% of the nonadoptees' biological fathers. The rates of diagnoses in the adoptees' biological parents were conservative because information was available for only 34% of their fathers, but the full sample size was used to compute the 11% rate. The unmistakable implication is that children can develop serious problem behaviors without being raised by problem parents:

> If it is true that these disorders in fathers are largely responsible for the factors known to be associated with aggressive conduct disorder, such as broken homes, wife and child abuse, and inconsistent discipline, then our findings suggest that these social factors are not necessary to the origin of the disorder. (Jary & Stewart, p. 10).

Again, the link between rearing experiences and child outcome is weakened.

Internalizing Disorders

People with a variety of psychological problems report the negative emotions of depression, fear, and anxiety (commonly referred to as "internalizing" disorders, because they are not usually outwardly evident). Given the lack of rearing influences on personality traits of normal intensity, it is unsurprising that when these same emotions are experienced more intensely, they show the same mix of determinants. This point can be made by considering adult twins (n = 3,798 pairs) who completed a short questionnaire for symptoms of anxiety and depression (Kendler, Heath, Martin, & Eaves, 1986). About 30% of the twins admitted symptoms of anxiety, such as feelings of panic and worry. About 4% reported that they had suicidal thoughts. A finding relevant to one assumption of the twin method was that the frequency of the twins' current social contact was unassociated with the similarity of the reported symptoms. Various statistical models were fitted to the individual items; the main finding was genetic influence on all symptoms, and a singular lack of rearing influence on them. In the cautious phrasing of scholarly writing, Kendler et al. ventured:

> . . . an etiologic role for familial factors could not be unambiguously demonstrated for any of the items studied. . . . Though these results do not eliminate a possible role for common [shared] environmental variables, they do suggest that factors such as rearing environment and culture play a smaller role than has previously been thought in the etiology of common symptoms of anxiety and depression. (1986, pp. 220–221)

Summary

In summary, the picture for psychopathology is not different from that for normal traits: There is little influence of common rearing experience on child or adult psychopathology. Exceptions may be found to this general rule, but they are not many. Delinquency in the teenage years is one trait that does not seem to fit the pattern; as noted by Cloninger and Gottesman (1987), twin resemblance for delinquency is not much greater in MZ than in DZ pairs. But this exception may be merely the result of the tendency of both types of twins to be "partners in crime" and to run around with the same adolescent crowd, as noted earlier (Rowe & Gulley, 1992). The general pattern for personality and psychopathology is now so reliable that it must be explained.

Behavior Genetic Studies of Social Attitudes

People hold beliefs about a wide variety of political, social, and religious topics. Socialization science assumes that many of these beliefs are acquired via social learning in the family. One can easily imagine exposure to an "Archie Bunker" father, who spouts racial slurs and advocates conservative social policies to anyone who will listen. All children old enough to understand Archie's political views will be exposed to them, and may therefore acquire them. At the other extreme, politically liberal parents may encourage humanitarian impulses toward the less fortunate, and may use the abundance of the American dinner table as an object lesson in the need for social generosity. Our intuition is that rearing effects are strong, because we know that social attitudes must be learned somewhere. Unlike physical activity or emotional outbursts, attitudes do not seem to spring immediately from physiology. Yet the fact that these attitudes must be learned does not mean that family experiences are crucial exposures for acquiring them. It is easy to forget that

even a young child's life is wider, more varied, and more rich in experiential opportunities than most parents will readily acknowledge.

In their review of earlier studies and report of original findings, Eaves, Eysenck, and Martin (1989) applied the full armamentarium of behavior genetic model-fitting research to explore underlying influences on social attitudes. They examined both individual attitudinal items from major attitude self-report inventories and composite, multifactor scales. Their data consisted of twin samples in England and Australia, with sample sizes in the hundreds of pairs; hence, they reported empirical replications across continents.

It was quickly apparent that social attitudes do not present the same kinds of data patterns as personality traits do. DZ twin correlations were higher for social attitudes than for personality traits. Thus, from basic model fitting, it looks as though *both* heredity and rearing experience influence social attitudes. For example, male twins' authoritarianism correlated .74 in MZ pairs and .44 in DZ pairs. According to our algebraic rules, the estimates of heritability (h^2) and rearing influence (c^2) would be 60% and 14%, respectively. For males twins' religion (a scale of belief in particular religious precepts, not membership in a particular religious faith), the respective correlations were .66 and .51, with $h^2 = .30$ and $c^2 = .36$. For female twins' prejudice, they were .61 and .48, with $h^2 = .26$ and $c^2 = .35$. Other examples could be given, but the pattern is clear: The twin data include a component of genetic influence *and* a component of rearing influence. Table 3.8 presents short one- and two-word items from the Australian questionnaire, separated according to whether the items had large or small rearing effects. For each item, respondents indicated whether they approved, disapproved, or were indifferent. No difference in content or emphasis is immediately apparent in the items where variation was statistically more versus less "family environmental." Possibly the difference between the items was merely one of sampling variation.[2]

The conclusion that family environments influence social attitudes has one important caveat, however, that could entirely undermine our inference of nongenetic family influence. That is, nonrandom marriage effects are also greater for social attitudes than for personality traits, or even for intellectual ability. For instance, in one British study, the spouse correlation for religion was .52; for authoritarianism, .56; for socialism, .54; and for prejudice, .35 (Eaves et al., 1989, p. 378). As we have seen for mood disorders, if spouses match on a behavioral trait, greater genetic similarity may be induced in offspring, which in turn can inflate the value

TABLE 3.8. Social Attitudes with High and Low Family Environmental Influence (Marriage Assortment Not Considered)

Low family environmental influence, high genetic influence

1. Death penalty
2. Self-denial
3. Working mothers
4. Military drill
5. White superiority
6. Cousin marriage
7. Chaperones
8. Empire building
9. Computer music
10. Fluoridation
11. Women judges
12. Conventional clothes
13. Teenage drivers
14. Apartheid
15. Censorship
16. White lies
17. Strict rules
18. Jazz
19. Learning Latin
20. Divorce
21. Inborn conscience

High family environmental influence, low genetic influence

1. School uniforms
2. Birth control
3. Divine law
4. Nudist camps
5. Bible truth
6. Co-education

Note. Adapted from Eaves, Eysenck, & Martin (1989). Copyright 1989 by the Academic Press Ltd. Adapted by permission.

of a DZ twin correlation in a way that mimics the effects of family environment. These high spousal correlation coefficients did not appear to be the result of social influence in the marriage; people who had been married a long time were not more alike in their attitudes than were newlyweds. Initial assortment, rather than influence, thus seems to be the cause of spousal behavioral resemblance.

Readers may notice an ironic parallel here with this book's theme. I have argued that rearing experiences do not influence the traits of children—whose youth, potential for developmental growth, and inexperience make them seem like potential candidates for an influence pro-

cess. Yet we accept quite readily the idea that our spouses are hard to influence; it is easier to avoid an area of divergent opinion than to try to get our wives or husbands to agree. How intuitive it is that spouses are hard to change! Yet the great change that occurs in children does not mean that their *direction* of change is any more malleable to our wishes than that of our spouses, to whom we also apply pressure by social example and by levers of reward and punishment, but to little advantage.

In statistical models including nonrandom mating, Eaves et al. (1989) were able to show that a statistical parameter representing parental influence on children's social attitudes could be omitted without degrading statistical model fits:

> The degree of assortative [nonrandom] mating for attitudes is so high that its genetic consequences could account for all the additional resemblance between twins that our earlier analyses had ascribed to the "family environment." . . . This result does not agree with our initial intuition that cultural factors derived from parents are major determinants of family resemblance in attitudes. (p. 387)

Given these countervailing models, Eaves et al. (1989) concluded that one must turn to other behavior genetic designs to rule out rearing influences decisively. The nonrandom mating model suggests that an adoptive parent and child will lack resemblance for social attitudes, whereas a rearing environment model expects familial resemblance among biologically unrelated individuals raised in one household.

Not many adoptive data exist for social attitudes, but the few existing examples are consistent with a lack of rearing influence. In the Minnesota study of twins raised apart, separated twins correlated as highly as unseparated twins for religious traditionalism (Bouchard et al., 1990). Another important test comes from Sandra Scarr's (1981) adoption study of authoritarianism (as assessed via the F or Fascism scale). This area of research has been described in Chapter 1; although Adorno, Frenkel-Brunswik, Levinson, and Sanford (1950) viewed the F scale as tapping into an underlying dimension of personality, the items themselves refer to generalized social attitudes. Table 3.9 presents a few items from the F scale. Reading through items 3, 4, and 8, where agreement represents authoritarianism, one can sense an extremity of emphasis. Phrases such as "complete faith," "somehow get rid of," and "publicly whipped or worse" suggest a conservative extreme on some dimension of opinion—Archie Bunker rather than Thomas Jefferson. And such broadly stated

TABLE 3.9. Illustrative Authoritarianism Items

1. One of the most important things children should learn is when to disobey authorities. (R)
2. People ought to pay more attention to new ideas, even if they seen to go against the American way of life. (R)
3. Most of our social problems could be solved if we could somehow get rid of the immoral, crooked, and feebleminded people.
4. Every person should have complete faith in a supernatural power whose decisions he obeys without question.
5. The artist and professor are probably more important to society than the business man and the manufacturer. (R)
6. The findings of science may some day show that many of our most cherished beliefs are wrong. (R)
7. In spite of what you read about the wild sex life of people in important places, the real story is about the same in any group of people. (R)
8. Sex crimes, such as rape and attacks on children, deserve more than mere imprisonment; such criminals ought to be publicly whipped or worse.

Note. R = agreement means lack of authoritarianism. From Adorno, Frenkel-Brunswik, Levinson, & Sanford (1950). Copyright 1950 by the American Jewish Committee. Reprinted by permission of HarperCollins Publishers Inc.

opinions invite disagreement from people who hold a more differentiated view of the social world.

Scarr (1981) compared parent–child and sibling attitudinal resemblance in 112 adoptive families and 120 matched biological families in Minnesota. All children were placed with adoptive families before their first birthdays. The adoptive parents held more authoritarian attitudes than the biological ones; Scarr speculated that this difference was partly attributable to the location of relatively more adoptive families in small towns and rural areas. The mean differences, however, were not great compared to the variability of attitudes held within the two groups of families: Some parents were highly authoritarian, others just the opposite, and children in both sets of families were exposed to a wide range of beliefs. As we have seen for social attitudes generally, nonrandom mating effects existed for authoritarian attitudes (the spouse correlation for biological families was .43, and that for adoptive families was .34).

Scarr's data can be used to test the expectation that variation in rearing should influence authoritarianism, if adoptive family members' authoritarian beliefs correlate. But Scarr did not find this. Adoptive relatives' attitudinal resemblance was weak (and, statistically, could have been attributable to chance). In contrast, biological family members resembled one another strongly. The mother–child authoritarianism correlations showed the greatest contrast: In the adoptive families this

correlation was .00, whereas in the biological families it was .41. The sibling correlations were .14 and .36, respectively, and the father–child correlations were .06 and .44, respectively. Thus similar beliefs occurred whenever families shared genes, but not when they shared rearing experiences alone. It takes a small act of imagination to think of the possible mismatches represented by a correlation of zero: a mother who believes that criminals should get the gallows, while her adolescent adoptive son would vote against the death penalty; or a mother who belongs to the Sierra Club and votes Democratic, while her adolescent adoptive daughter would think that social problems can be solved, in Adorno et al.'s (1950) phrase, by ridding the world of the "immoral, crooked, and feebleminded."

What happened? Why didn't the adolescent adoptive children share their parents' beliefs? Scarr's data contain several clues suggesting a possible explanation, the first being the strong association of verbal IQ and authoritarian attitudes: The Adorno et al. items were endorsed in the authoritarian direction by *less* verbally bright individuals. Of course, this does not mean that high-IQ individuals never hold authoritarian beliefs; however, such individuals may better sense the social opprobrium attached to endorsing authoritarian items, and thus may not choose not to make a public expression of these beliefs. Authoritarian beliefs also may be truly rarer among highly intelligent individuals, who may be loath to respond to social complexities with the blunt instrument of authoritarian solutions. Whatever the exact process, the general lesson is that individuals reason according to their own beliefs, independently of parental example. The genes for IQ congregate in biological families, and therefore so do the reasoning abilities that lead to similar attitudes.

In Scarr's data, authoritarian attitudes were also positively associated with personality traits reflecting fear in social situations. More fearful individuals may grope for direct solutions to social conflicts. Genes for personality traits, like those favoring intellectual traits, congregate in biological families, and therefore so does the tendency to develop similar social attitudes when personality favors one belief over another. In Scarr's (1981) words, "authoritarian attitudes are not learned in rote fashion from one's associates (parents, teachers, colleagues) but rather represent conclusions one has reached by applying one's cognitive skills to social and political experiences" (p. 423). Thus we reach the final conclusion that, most likely, both genes (for IQ and personality traits) and nonrandom mating give the appearance of rearing influences in twin studies of social attitudes. The people one is raised with have little lasting importance for what one finally believes.

Behavior Genetic Research on Religious Affiliation

Variation in rearing does matter, however, for religious affiliation. It is true that religious denomination is neither a personality trait nor a social attitude, and as such does not qualify as a trait. Nonetheless, exceptions are interesting because behavior genetic methods seem so regularly to fail to show rearing influence that, if nothing else, we need to see an example where they can come to the opposite conclusion. In Eaves, Martin, and Heath's (1990) twin-family study in Australia, religious denominations (mainly Anglican, other Protestant, and Catholic) revealed what was missing from other traits: Children were like the parents who reared them in religion, and the twin siblings were also like each other. Resemblances were not perfect, because some individuals changed religious denominations; furthermore, when children grew up and left home, they became less like their parents. I do not intend to describe Eaves et al.'s statistical analysis in detail. Their model-fitting procedures led to the main conclusion that these patterns of imitation were environmental—that they were rearing effects.

At first glance, the environmental inheritance of religious denomination may seem to conflict with the genetic transmission of social attitudes; however, once one realizes the wide range of belief and opinion held within a large denomination, the conflict vanishes. Some Catholics use birth control and have had abortions, whereas others are ardently pro-life. Some Anglicans are politically conservative, whereas others are liberal. What Eaves et al. (1990) tested was merely the transmission of a religious label—not whether the professed faith was accepted, not whether religious ceremonies were observed, and not whether faith was shallow or deep. Indeed, the implication of a religious life is unverified in naming a denomination. Genetic influence appears strongly in the transmission of religious *beliefs* (Waller, Kojetin, Bouchard, Lykken, & Tellegen, 1990).

Nonetheless, certainly pollsters and church leaders care to count their numbers, and we must ask why religious denomination shows rearing influence. One answer may be that it takes time and effort before

the doctrine and ceremonies of a faith are learned. Parents provide the opportunity for this learning, so a choice based on a child's own genetic disposition is impossible. Once the child becomes an adult, the discomforts of switching faiths are many: Neither the liturgy, the music, the doctrines, nor the traditions of a new faith are known, nor are they conveniently acquired. It is easy to find friendship and emotional support from members of one's own faith, and easy to step into its comfortable routines. If another faith would be inherently more attractive (e.g., Unitarianism in place of Catholicism for the religiously dubious), there is still the cost of time and effort in making the transition from one faith to another. So it may be simply that the costs of exploring new alternatives outweigh the immediate benefits of staying in the original faith. People may remain ensconced in their own faith, unless pulled away from it by an interfaith marriage or by movement into a different social class, where opportunities to practice the faith learned in childhood become greatly reduced. Ironically, the Eaves et al. (1990) data gave a hint of genetic influence on female twins' choice of religious denomination, once the twin sisters had left home. In an environment of greater choice and opportunity, genetic dispositions may begin to influence choice, when they may not have before.

Niche Picking

In his book *The Extended Phenotype*, Richard Dawkins (1982) cleverly imagines how genotypes may extend beyond the confines of nucleic acid to encompass a constructed environment that nurtures, protects, and supports the organism. The extended environment is as much a creation of the genes as is the body that houses them. To pick two of many illustrations, the paper wasp creates a paper-like nest to house its young and protect them from rain and wind. The beaver plays construction worker, deftly cutting down trees with its razor-sharp teeth and building dams that rechannel streams, flooding a basin for its mud and wood home. How can these acts be regarded as acts of DNA, when genes code only for proteins and do not contain within them a blueprint of a paper nest or dam? The answer is that the genes may construct a nervous system—and that hormones and neurotransmitters may then motivate behaviors resulting in the dramatic redesign of an environment. The way a beaver will restructure its environment is as genetically shaped as its flat tail and keen hearing.

The extended phenotypes of paper wasps and beavers emerge from a hard-wired, instinctive sense of what things ought to be. Little tutoring is needed for the expression of either behavior, although I suspect that the dam building of a beaver must improve with experience, as even bees show a capacity for learning.

Dawkins's examples are undeniably examples of instincts—of stereotyped inherent patterns of behavior (Wessells & Hopson, 1988). Such instincts are shared by most individuals in a species, but the present topic is individual differences in traits. Yet the conceptual distance between the human expression of individual differences and the extended phenotype of a species may not be so great. Genes can produce dispositions, tendencies, and inclinations, because people with subtly different nervous systems are differently motivated. Admittedly, the process of causality is a probabilistic one—but it is reliable, on the average, when one looks at enough people, or examines a long enough period in a single person's life. The genes themselves do not pick the environment; only the whole person, not DNA sheltered within the nuclei of trillions of cells, can act. Yet, given enough environmental opportunities, the ones chosen are those most reinforcing for a particular nervous system created by a particular genotype.

The role of "genotypes" in modifying and selecting environments has been labeled in behavior genetics with the terms "reactive" and "active" gene–environment correlations (Plomin, DeFries, & Loehlin, 1977; Scarr & McCartney, 1983). The reactive correlation refers to other people's responses to one's genetic disposition. For instance, a highly active child triggers more parental surveillance than an inactive one. A beautiful woman attracts the attention of more men than an unattractive one. The other type is the active correlation, whereby individuals with different genetic dispositions eventually discover and frequent the environmental contexts that reinforce them. The chess-playing prodigy, who spends hours practicing, reading about chess, and competing in tournaments, fertilizes an innate talent with the kinds of challenges that will nurture it.

In a behavior genetic study, the gene–environment correlation, like the extended phenotypes of wasps and beavers, counts as part of *genetic variation* because the direction of the growth curve of development, and the limit ultimately attained, is set in the genes and in their effects on the environment. Perhaps this is unfair, because the environment plays a direct causal role in developing and maintaining a genetic disposition; certainly, without the environmental opportunities, neither the talent of a chess prodigy nor the creativity of a recording artist could flourish.

But the character of these environmental influences is qualitatively different from that of rearing ones. Rearing environments are imposed on children, whereas the reactive and active environments are created in response to the particular genotype. To some extent they are chosen, as Scarr and McCartney (1983) observe: ". . . most differences among people arise from genetically determined differences in the experiences to which they are attracted and which they evoke from their environments" (p. 433). Socialization science looks to rearing to change developmental growth curves, but it is exactly the *imposed* rearing environment that loses its sway over development until, as we have seen, adoptive children no longer resemble their (biologically unrelated) siblings or parents, and until the similarity seen in biological families is merely the happenstance of overlapping heredity. In biological families too, the genetic differences among siblings often generate very different life courses as, through the reactive and active processes, children discover the environmental opportunities for the genes' extended phenotype. In a newspaper column, Curtis Austin (1991) warned against attributing too much influence to rearing:

> Yet I can't help but feel another side of the story receives far less attention. It's not always the parents' fault. Sometimes, truly loving and caring parents have troubled kids. . . . In the middle-class neighborhood where I was raised, the Wheeler boys were a study in contrast. Gary, the older by about two years, was well-mannered, hard-working and a straight-A student. Carlton, was loud, abrasive and a constant trouble-maker. Gary went on to college, got married and raised a family. His brother became a professional criminal, spending his life in and out of prison. (p. D1)

In the diversity of American society, the environmental opportunities exist to manifest almost any behavioral disposition. Children may discover friends among peers whose values are as foreign to their own parents as another culture's. In Austin's example, Carlton Wheeler, despite being reared under the same roof with the same caring parents as Gary, displayed a very different developmental trajectory. The implication is that parents are often given too much credit for children who turn out well, and too much blame for children who turn out poorly. The source of causal influence is not in rearing variation, but in the genes and in unshared environmental variation.

This chapter has covered the nonintellectual personality traits. Historically, intellectual abilities have held a more central position in the

nature–nurture debate because their social importance merges a scholarly concern with the concerns of social policy makers and parents. So the next chapter returns to this old battlefield, and examines the hypothesis of rearing influences on IQ.

Notes

[1]The questionnaire could not assign 8% of the cases, which were omitted from the analysis. Questionnaire identification of twin type is usually quite accurate (about a 95% hit rate) when compared to biological means of determining twin type, such as human blood groups.

[2]Differences in the heritability of beliefs may be important. Tesser (1993) found that more heritable beliefs are more resistant to change in response to social pressure.

References

Andreasen, N. C. (1978). Creativity and psychiatric illness. *Psychiatric Annals, 8*, 113–119.

Adorno, T. W., Frenkel-Brunswik, E., Levinson, D. J., & Sanford, R. N. (1950). *The authoritarian personality*. New York: Harper.

Allport, G. W. (1966). Traits revisited. *American Psychologist, 21*, 1–10.

Austin, C. (1991, November 16). Kids' wrong turns don't always lead back to parents. *Dallas Times Herald*, Section D, 1.

Begley, S., & Kasindorf, M. (1979, December 3). Twins: Nazi and Jew. *Newsweek*, 139.

Bollen, K., & Lennox, R. (1991). Conventional wisdom on measurement: A structural equation model perspective. *Psychological Bulletin, 110*, 305–314.

Bouchard, T. J., Lykken, D. T., McGue, M., Segal, N. L., & Tellegen, A. (1990). Sources of human psychological differences: The Minnesota study of twins reared apart. *Science, 250*, 223–228.

Buss, A. H., & Plomin, R. (1984). *Temperament: Early developing personality traits*. Hillsdale, NJ: Erlbaum.

Cloninger, C. R., & Gottesman, I. I. (1987). Genetic and environmental factors in antisocial behavior disorders. In S. Mednick, T. Moffit, & S. Stack (Eds.), *The causes of crime* (pp. 92–109). New York: Cambridge University Press.

Dawkins, R. (1982). *The extended phenotype: The gene as the unit of selection*. Oxford: Oxford University Press.

DeFries, J. C., & Plomin, R. (1978). Behavioral genetics. *Annual Review of Psychology, 29*, 473–515.

Digman, J. M. (1990). Personality structure: Emergence of the five-factor model. *Annual Review of Psychology, 41*, 417–440.

Eaves, L. J., Eysenck, H. J., & Martin, N. G. (1989). *Genes, culture and personality: An empirical approach*. London: Academic Press.

Eaves, L. J., Martin, N. G., & Heath, A. C. (1990). Religious affiliation in twins and their parents: Testing a model of cultural inheritance. *Behavior Genetics, 20*, 1–22.

Funder, D. C. (1991). Global traits: A neo-Allportian approach to personality. *Psychological Science, 2*, 31–39.

Galton, F. (1876). The history of twins as a criterion of the relative powers of nature and nurture. *Royal Anthropological Institute of Great Britain and Ireland Journal, 6*, 391–406.

Goodman, R., & Stevenson, J. (1989). A twin study of hyperactivity: II. The aetiological role of genes, family relationships and perinatal adversity. *Journal of Child Psychology and Psychiatry, 30*, 691–709.

Gould, S. J. (1981). *The mismeasure of man*. New York: Norton.

Gottesman, I. I. (1991). *Schizophrenia genesis: The origins of madness*. New York: W. H. Freeman.

Gottesman, I. I., & Bertelsen, A. (1989). Confirming unexpressed genotypes for schizophrenia: Risks in the offspring of Fischer's Danish identical and fraternal discordant twins. *Archives of General Psychiatry, 46*, 867–872.

Gottesman, I. I., Shields, J., & Hanson, D. R. (1982). *Schizophrenia: The epigenetic puzzle*. Cambridge, England: Cambridge University Press.

Hill, M. S., & Hill, R. N. (1973). Hereditary influence on the normal personality using the MMPI: I. Age-corrected parent–offspring resemblances. *Behavior Genetics, 3*, 133–144.

Hoffman, L. W. (1985). The changing genetics/socialization balance. *Journal of Social Issues, 41*, 127–148.

Hoffman, L. W. (1991). The influence of family environment on personality: Accounting for sibling differences. *Psychological Bulletin, 110*, 187–203.

Jary, M. L., & Stewart, M. A. (1985). Psychiatric disorder in the parents of adopted children with aggressive conduct disorder. *Neuropsychobiology, 13*, 7–11.

Kendler, K. S., Heath, A., Martin, N. G., & Eaves, L. J. (1986). Symptoms of anxiety and depression in a volunteer twin population. *Archives of General Psychiatry, 43*, 213–221.

Kenrick, D. T., & Funder, D. C. (1988). Profiting from controversy: Lessons from the person–situation debate. *American Psychologist, 43*, 23–34.

Loehlin, J. C. (1986). Heredity, environment, and the Thurstone Temperament Schedule. *Behavior Genetics, 16*, 61–73.

Loehlin, J. C. (1992). *Genes and environment in personality development*. Newbury Park, CA: Sage.

Loehlin, J. C., & Rowe, D. C. (1992). Genes, environment, and personality. In G. Caprara & G. L. Van Heck (Eds.), *Modern personality psychology: Critical reviews and new directions* (pp. 352–370). New York: Harvester Wheatsheaf.

Loehlin, J. C., Willerman, L., & Horn, J. M. (1987). Personality resemblance in adoptive families: A 10-year follow-up. *Journal of Personality and Social Psychology, 53*, 961–969.

Lykken, D. T., McGue, M., Tellegen, A., & Bouchard, T. J., Jr. (1992). Emergenesis: Genetic traits that may not run in families. *American Psychologist, 47*, 1565–1577.

McCrae, R. R., & Costa, P. T., Jr. (1990). *Personality in adulthood*. New York: Guilford Press.

McGue, M., & Gottesman, I. I. (1989). Genetic linkage in schizophrenia: Perspectives from genetic epidemiology. *Schizophrenia Bulletin, 15*, 453–464.

McGue, M., Gottesman, I. I., & Rao, D. C. (1985). Resolving genetic models for the transmission of schizophrenia. *Genetic Epidemiology, 2*, 99–110.

Moldin, S. O., Reich, T., & Rice, J. P. (1991). Current perspectives on the genetics of unipolar depression. *Behavior Genetics, 21*, 211–242.

Plomin, R., DeFries, J. C., & Fulker, D. W. (1988). *Nature and nurture during infancy and early childhood*. Cambridge, England: Cambridge University Press.

Plomin, R., DeFries, J. C., & Loehlin, J. (1977). Genotype–environment interaction and correlation in the analysis of human behavior. *Psychological Bulletin, 84*, 309–322.

Price, R. A., Vandenberg, S. G., Iyer, H., & Williams, J. S. (1982). Components of variation in normal personality. *Journal of Personality and Social Psychology, 43*, 328–340.

Richards, R., Kinney, D. K., Lunde, I., Benet, M., & Merzel, A. P. C. (1988). Creativity in manic–depressives, cyclothymes, their normal relatives, and control subjects. *Journal of Abnormal Psychology, 97*, 281–288.

Rose, R. J., Kaprio, J., Williams, C. J., Viken, R., & Obremski, K. (1990). Social contact and sibling similarity: Facts, issues, and red herrings. *Behavior Genetics, 20*, 763–778.

Rowe, D. C. (1987). Resolving the person–situation debate: Invitation to an interdisciplinary dialogue. *American Psychologist, 42*, 218–227.

Rowe, D. C., & Gulley, B. (1992). Sibling effects on substance use and delinquency. *Criminology, 30*, 217–233..

Scarr, S. (1981). *Race, social class, and individual differences in I.Q.* Hillsdale, NJ: Erlbaum.

Scarr, S., & McCartney, K. (1983). How people make their own environments: A theory of genotype → environment effects. *Child Development, 54*, 424–435.

Segal, J., & Yahraes, H. (1979). *A child's journey: Forces that shape the lives of our young*. New York: McGraw-Hill.

Suddath, R. L., Christison, G. W., Torrey, E. F., Casanova, M. F., & Weinberger, D. R. (1990). Anatomical abnormalities in the brains of monozygotic twins discordant for schizophrenia. *New England Journal of Medicine, 322*, 789–794.

Tesser, A. (1993). The importance of heritability in psychological research: The case of attitudes. *Psychological Review, 100*, 129–142.

Tienari, P., Kaleva, M., Lahti, I., Laksy, K., Moring, J., Naarala, M., Sorri, A., Wahlberg, K. E., & Wynne, L. (1991). Adoption studies of schizophrenia. In C. Eggers (Ed.), *Schizophrenia and youth: Etiology and therapeutic consequences* (pp. 40–51). Berlin: Springer-Verlag.

Tienari, P., Lahti, I., Sorri, A., Naarala, M., Moring, J., Kaleva, M., Wahlberg, K., & Wynne, L. C. (1990). Adopted-away offspring of schizophrenics and controls: The Finnish adoptive family study of schizophrenia. In L. N. Robins & M.Rutter (Eds.), *Straight and devious pathways from childhood to adulthood* (pp. 365–379). Cambridge, England: Cambridge University Press.

Torrey, E. F. (1992). *Freudian fraud: The malignant effect of Freud's theory on American thought and culture*. New York: HarperCollins.

Tsuang, M. T., & Faraone, S. V. (1990). *The genetics of mood disorders*. Baltimore: John Hopkins University Press.

Waller, N. G., Kojetin, B. A., Bouchard, T. J., Lykken, D. T., & Tellegen, A. (1990). Genetic and environmental influences on religious interests, attitudes, and values: A study of twins reared apart and together. *Psychological Science, 1*, 138–142.

Wessells, N. K., & Hopson, J. L. (1988). *Biology*. New York: Random House.

CHAPTER 4

LIMITED REARING EFFECTS ON INTELLIGENCE (IQ)

In *Nature's Gambit* (Feldman, 1986), we can read case histories of extraordinary intellectual achievement. Adam Konantovich could speak in grammatical sentences at 3 months of age and read simple books at 1 year. At the age of 5 years, when attending a puppet show for preschoolers at the Boston Museum of Science, Adam answered a rhetorical question about what whales eat as follows: "Krill, they're small shrimp, but they're not microscopic." Billy Delvin was reading about particle physics at age 7 and scored better on the mathematics Scholastic Aptitude Test (SAT) than many junior high school students. Yet another story of precocity was told to me by a friend who is a professor at Harvard. His young daughter, then only about 18 months old, was greeted in the supermarket by a women who smiled and said, "Coochie, coochie, coo." His daughter then turned to her mother and asked, "Is she trying to talk to me?" These stories tell us that some children are born with unusually great aptitude for intellectual achievement. We recognize intuitively that no amount of "intellectual stimulation" (even the 3,000 books in the home of Adam Konantovich) could produce such talent in a child lacking special "gifts," but these unusual cases cannot tell us how important rearing environment is for intellectual development more generally—the issue broached in this chapter.

General Intelligence: Definitions and Controversies

Most social scientists recognize that "academic intelligence" refers to the ability to acquire the kinds of information taught in schools. Indeed, the