Developmental Review **20,** 539–567 (2000)
doi:10.1006/drev.2000.0511, available online at http://www.idealibrary.com on IDEAL®

Not in Their Genes: A Critical View of the Genetics of Attention-Deficit Hyperactivity Disorder

Jay Joseph

California School of Professional Psychology, Alameda

This article examines evidence cited in favor of the operation of genetic factors in attention-deficit hyperactivity disorder (ADHD). Like other psychiatric conditions, a belief in the genetic basis of ADHD is derived from the results of family, twin, and adoption studies. Because family studies are widely believed to be confounded by environmental factors, primary emphasis is placed on twin and adoption studies. ADHD twin studies depend on the validity of the equal environment assumption (EEA), which holds that the environments of identical (MZ) and fraternal (DZ) twins are the same. Here it is argued that however the EEA is defined, it cannot be accepted. Therefore, the greater similarity or concordance of MZ twins when compared to DZ twins is plausibly explained by environmental factors. Adoption studies constitute a third method for investigating the role of genetic factors in ADHD. It is argued that these studies are greatly flawed by factors including non blinded diagnoses and the failure to study the biological relatives of adoptees. After an examination of the total weight of evidence in favor of a genetic basis or predisposition for ADHD, it is concluded that a role for genetic factors is not supported and that future research should be directed toward psychosocial causes

Key Words: ADHD; adoption studies; children; equal environment assumption; genetics; hyperactivity; twin studies.

The current period is marked by the widespread acceptance of an important genetic influence on most psychological traits. This view is based on three pillars of support: (1) family studies, (2) twin studies, and (3) adoption studies. In psychiatry, schizophrenia has served as the model for the use of these methods. For the most part, the authors of over 2 dozen schizophrenia family studies, 14 schizophrenia twin studies, and 6 schizophrenia adoption studies concluded that their findings supported the existence of a genetic predisposition for the condition.

Here, we are interested in assessing the evidence in support of a genetic component for ''attention-deficit hyperactivity disorder'' (ADHD) which,

I thank David Cohen for his helpful comments on an earlier draft of this paper.

Address Correspondence and reprint requests to Jay Joseph, 2625 Alcatraz Ave., #328, Berkeley, California, 94705. E-mail: jayjoseph2@aol.com.

0273-2297/00 $35.00

like schizophrenia, is a psychiatric diagnosis made on the basis of a person's behavior and whose supposed genetic component is based on the evidence from family, twin, and adoption studies. For reasons of consistency, the term ''ADHD'' is used throughout this article in place of diagnoses which include ''hyperactive child syndrome,'' ''minimal brain dysfunction,'' ''hyperactivity,'' ''attention deficit disorder,'' and so on. As is shown, the way the condition has been defined is of secondary importance to an examination of the methods used to determine its possible genetic component.

According to Russell Barkley (1998a), ADHD is a ''developmental failure in the brain circuitry that underlies inhibition and self-control'' (p. 67). Barkley cites studies whose authors claimed to have found that a portion of ADHD children's brains are smaller than in normal children, which he links to genetic factors. Tannock (1998) also concluded that ADHD is caused by a brain dysfunction of probable genetic origin. Some have taken a more interactionist approach (e.g., Diller, 1998), while others have stressed environmental factors and have questioned the validity of the ADHD concept (e.g., Breggin, 1998; DeGrandpre, 1999). What follows is a review of the ADHD genetic study literature. In the concluding section, I will discuss possible future directions for research into the causes of the condition.

ADHD FAMILY STUDIES

Background

The family (or consanguinity) method of study constitutes the first systematic attempt to determine whether a condition clusters in families, thereby laying the basis for the possibility of finding a genetic component. Family studies locate persons affected with a particular trait or condition and attempt to determine whether their biological relatives are similarly affected more often than members of the general population or a control group. If a condition is found to cluster or ''run'' in families, it is said to be familial. Note that ''familial'' is not the same as ''genetic.'' Unfortunately, many people view these terms as being synonymous, when in fact they are not. As most genetic researchers now acknowledge, the aggregation of a particular condition in families is consistent with a genetic or an environmental etiology. Psychiatric geneticists Faraone and Tsuang (1995), for example, noted that a family study can provide ''the initial hint that a disorder might have a genetic component,'' while cautioning that ''Disorders can 'run in families' for nongenetic reasons such as shared environmental adversity, viral transmission, and social learning. . . . Although family studies are indispensable for establishing the familiality of disorders, they cannot, by themselves, establish what type of transmission'' (pp. 88–89). However, this has not always been the prevailing view.

The first schizophrenia family study (Rüdin, 1916) was published over 80 years ago, and the most influential study of this type was performed by Kall-

mann (1938). Most of the early family studies were authored by strong proponents of the genetic position, and most did not perform blind diagnoses. Kallmann believed that the familiality of schizophrenia *proved* that the condition was genetic in origin: ''The principal aim of our investigations was to offer *conclusive proof* [italics added] of the inheritance of schizophrenia and to help, in this way, to establish a dependable basis for the clinical and eugenic activities of psychiatry'' (Kallmann, 1938, p. xiv). Pam (1995) noted Kallmann's faulty logic and commented further on the family study method:

> The most serious breach in inductive logic committed by Kallmann was his use of kinship concordance rates to determine genetic transmission of psychopathology. We have already noted that no family inheritance study can control for environment in human research; such data, therefore, are nowhere near ''suggestive''—they are at best inconclusive and at worst misleading. . . . This inferential limitation holds with respect to any consanguinity finding, even if the design and technique employed in the investigation were scientifically impeccable. (p. 19)

Today, most behavior genetic and psychiatric genetic researchers (e.g., Faraone & Tsuang, 1995; Gottesman, 1991; Plomin, DeFries, & McClearn, 1990; Rosenthal, 1970; Wender, 1995) acknowledge that family studies by themselves cannot establish the existence of genetic factors and have cited twin and adoption studies as the primary evidence in favor of the genetic basis of schizophrenia and other conditions.

The authors of the ADHD family studies (Biederman et al., 1986, 1995; Biederman, Faraone, Keenan, Knee, & Tsuang, 1990; Cantwell, 1972; Faraone, Biederman, Keenan, & Tsuang, 1991a; Morrison & Stewart, 1971; Nichols & Chen, 1981; Welner, Welner, Stewart, Palkes, & Wish, 1977) have found consistent evidence for the familiality of the condition. Although several of these studies suffer from serious methodological problems (such as nonblind diagnoses), partisans of the genetic *or environmental* positions would be surprised if they *did not* find a familial clustering of ADHD.

In spite of the formal pronouncement that family studies by themselves cannot be used as evidence of genetic transmission, several important ADHD researchers have written that the evidence from these studies suggests a genetic basis for the condition. For example, in Barkley's authoritative handbook for the diagnosis and treatment of ADHD, the author writes, ''Family aggregation studies find that ADHD clusters among biological relatives of children or adults with the disorder, strongly implying a hereditary basis to this condition'' (Barkley, 1998b, p. 36). While Faraone and Tsuang (1995) viewed the results from family studies as providing only the ''initial hint'' of genetic factors, Barkley believes that these findings ''strongly imply'' such an etiology.

Several ADHD family researchers have implied that their results support the genetic position. For example, Nichols and Chen (1981) concluded that the ''greater risks to relatives of the severely affected children and to relatives of girls, the less frequently affected sex, provided some evidence that

the familial association was determined partly by polygenic inheritance'' (p. 276), and Biederman et al. (1995) have written, ''Additional lines of evidence from second-degree relative, twin . . . adoption, and segregation analysis studies suggest that the familial aggregation of ADHD has a substantial genetic component'' (p. 432). However, a method which by itself cannot be regarded as evidence in favor of the genetic hypothesis does not become evidence when combined with the supposed findings of another type of study. As noted by Diller, Tanner, and Weil (1995), ''Familial clustering, as noted in the [Biederman et al. 1995 family study] article, cannot distinguish between potential genetic and environmental etiologies. While the authors are careful to describe the new data as familial, they nevertheless discuss them only in the context of a genetic etiology'' (p. 451).

There is little reason to engage in a detailed discussion of the ADHD family studies, since their results are in accordance with the expectations of environmentalists and hereditarians alike. Therefore, this article focuses on the two methods most often cited in support of the genetic basis of ADHD: twin and adoption studies.

ADHD TWIN STUDIES

Overview

As we have seen, the finding that a trait or condition runs in families is consistent with both genetic and environmental etiologies. For this reason, the results from twin studies have been promoted as evidence in favor of the genetic position. According to Barkley (1998a), twin studies have provided ''the most conclusive evidence that genetics can contribute to ADHD'' (p. 68). Several ADHD twin studies have been published since 1965 (Eaves et al., 1993; Edelbrock, Rende, Plomin, & Thompson, 1995; Gilger, Pennington, & DeFries, 1992; Gillis, Gilger, Pennington, & DeFries, 1992; Gjone, Stevenson, & Sundet, 1996; Goodman & Stevenson, 1989a, 1989b; Levy, Hay, McStephen, Wood, & Waldman, 1997; Lopez, 1965; Nadder, Silberg, Eaves, Maes, & Meyer, 1998; Sherman, Iacono, & McGue, 1997; Silberg et al., 1996; Steffensson et al., 1999; Stevenson, 1992; Thapar, Hervas, & McGuffin, 1995; van den Oord, Verhulst, & Boomsma, 1996; Willerman, 1973). All of these studies utilized the so-called ''classical twin method'' (also known as the ''twin method''), which compares the concordance rates or correlations of reared-together identical twins (also known as monozygotic or MZ) to the same measures of reared-together fraternal twins (also known as dizygotic or DZ). A significantly greater similarity or concordance of MZ twins when compared with DZs is usually cited as evidence in favor of the genetic basis for the trait or condition under study. All ADHD twin studies have investigated pairs who were reared *together*; there have been no studies of reared-apart pairs. Separated twin studies typically look at similarities in personality and cognitive ability, but have been plagued

by methodological problems and questionable theoretical assumptions (see Farber, 1981; Joseph, in press-d; Kamin, 1974; Taylor, 1980).

The authors of ADHD twin studies have found consistently that identical twins are more concordant for ADHD or correlate higher for ADHD-related behaviors than fraternals, and there is little doubt that in spite of these studies' methodological problems, MZ twins are significantly more similar than DZ twins. The question which concerns us here is whether the greater phenotypic similarity of MZ twins is caused by their greater genetic similarity, as the proponents of the twin method maintain. In order to answer this question, it is necessary to examine the theoretical underpinnings of the twin method itself. So before returning to the ADHD twin studies, we must assess the validity of the most important assumption of the twin method.

The ''Equal Environment Assumption'' in Twin Studies

Because MZs rate more similarly on ADHD-related measures than DZs, twin studies would be considered solid evidence in favor of a genetic predisposition for ADHD were it not for one important detail: Since its inception in the mid-1920s, the twin method has been based on the theoretical assumption that identical and fraternal twins share equal environments. The equal environment assumption (EEA) must be valid in order to claim that the MZ/DZ concordance rate difference, found in most types of human behavior, can be attributed to genetic factors. According to Kendler, the most prominent contemporary defender of the equal environment assumption, ''The EEA is crucial because, if the EEA is incorrect, excess resemblance of MZ twins compared with DZ twins ascribed to genetic factors could be partly or entirely due to environmental effects'' (1993, p. 906). Kendler is quite right: If the EEA is false, the twin method could be measuring nothing else than the more similar environment and greater emotional bond experienced by MZ twins.

Although the validity of the EEA is crucial to the viability of the twin method, it is not often discussed in detail by its defenders. The EEA has been the subject of at least two critical reviews (Joseph, 1998b; Pam, Kemker, Ross, & Golden, 1996), whose authors concluded that the assumption is untenable.

Until the late 1950s, the assumption of equal environments between MZ and DZ pairs was taken for granted by most twin researchers, although little theoretical or empirical justification for this clearly counterintuitive assumption was offered. In 1960, Don Jackson published a critique of the five schizophrenia twin studies which had been published up to that time. Jackson pointed out that female twins were consistently more concordant than male twins, that same-sex DZs were more concordant than opposite-sex DZs, and that fraternal twins were more concordant than ordinary siblings, though they each share the same genetic relationship to each other. Jackson noted that common environment, ''ego fusion,'' and association could explain these

differences, and he implied that the MZ/DZ concordance rate difference could also be explained on this basis. As a ''plausible hypothesis,'' Jackson (1960, p. 67) predicted that ''according to the degree of likeness in siblings, we will find an increased concordance for schizophrenia, without concern for genetic similarity.'' (Slightly modifying Jackson's position, we might say that according to the degree of *environmental similarity* among siblings, we would expect greater behavioral similarity, without concern for genetic relationship.) The reaction of the schizophrenia twin studying world followed, for the most part, two different paths. The first was an attempt to discredit Jackson's theory by claiming that its validity rested on Jackson's hypothesis that the identical twinship itself might create conditions more conducive to schizophrenia and that we would therefore expect to find a higher rate of schizophrenia among individual MZ twins than among the single-born population. Although Rosenthal (1960) and others claimed to have provided evidence that twins are no more susceptible than nontwins, the evidence is in fact equivocal (Joseph, 1998a; Kläning, Mortensen, & Kyvik, 1996). More importantly, Jackson's ''theory of identity confusion'' does not require twins to be more susceptible than singletons for the trait in question. The thrust of Jackson's theory dealt with the reasons why the *second* member of a twin pair fell to schizophrenia, not the first (Joseph, in press-c).

The second way that the proponents of the twin method responded to Jackson's ideas was to concede some of his most important points while continuing to uphold the twin method as a valid instrument for the detection of genetic influences. As demonstrated elsewhere (Joseph, 1998b), the most important twin researchers of the 1960s and 1970s were in agreement that environmental similarity and association were *partly* responsible for the MZ/DZ concordance rate difference. But I ask the reader: How did they know that environmental influences were not *entirely* responsible for the difference? In fact, they didn't know—they only *hoped* that their studies had measured genetic influences. In one of the early collaborations of Gottesman and Shields (1966), we find that the authors were willing to acknowledge that the greater psychological identification of MZ twins could affect concordance rates ''provided that the same proportion of potential schizophrenics are held back from overt illness by identifying with a normal twin as those who become ill by identifying with an abnormal one'' (p. 55). Gottesman and Shields provided no evidence in support of their attempt to balance the ledgers of the twin method. On what grounds, one might ask, did Gottesman and Shields insist on a one-to-one correspondence between those twins who became concordant for reasons of association and those who stayed ''well'' for the same reason? Could we not just as easily surmise that, for reasons of identification, there are *five* twins who become concordant for every *one* who remains well? The reasoning of Gottesman and Shields constituted little more than wishful thinking in the service of keeping a theory intact.

There are two main ways that the equal environment assumption of the twin method has been defined.

1. *The traditional EEA definition.* The traditional definition was used by most twin researchers prior to 1972 and continues to be used by some to the present day. The EEA is defined as the straightforward assumption that MZ and DZ twins experience similar environments and treatment and is exemplified by the following quotation from a leading twin researcher: ''The basic underlying assumption of the twin method is, of course, that environmental conditions of monozygotic twins do not differ from those of dizygotic twins'' (Kringlen, 1967, p. 20). At times, proponents of the traditional definition acknowledge that MZ twins experience more similar environments than DZs and that the twin method might therefore ''overestimate heritability'' in these cases.

2. *The ''equal trait-relevant environment assumption'' (Carey & DiLalla, 1994; referred to here as the ''trait-relevant EEA'').* One of the first examples of this new definition is found in Gottesman and Shields (1972), who defined the EEA, at least as it pertained to schizophrenia, as meaning the assumption of equal environments ''in respects which can be shown to be of etiological significance for schizophrenia'' (p. 25). More recently, Kendler and associates defined the EEA as follows:

> The traditional twin method, as well as more recent biometrical models for twin analysis, are predicated on the equal-environment assumption (EEA)—that monozygotic (MZ) and dizygotic (DZ) twins are equally correlated for their exposure to environmental influences that are of etiologic relevance to the trait under study. (Kendler, Neale, Kessler, Heath, & Eaves, 1993, p. 21)

Proponents of the trait-relevant EEA recognize that identical twins are treated more similarly and spend significantly more time together than fraternals, but claim that (a) the evidence shows that greater environmental similarity does not lead to higher concordance for psychiatric diagnoses or to greater psychological trait correlations; and (b) in order to invalidate the finding of genetic factors, critics must identify the trait-relevant environmental factors for which MZ and DZ twins experience dissimilar environments.

Today, Kendler and other twin researchers acknowledge that the environments of MZ twins are more similar than DZ twins (e.g., Bouchard, 1993; Gottesman & Shields, 1972; Kendler, Neale, Kessler, Heath, & Eaves, 1994; Morris-Yates, Andrews, Howie, & Henderson, 1990; Rose, 1991; Scarr, 1968; Scarr & Carter-Saltzman, 1979), and there is plenty of empirical evidence in support of this idea. Identical twins spend more time together than fraternals (Wilson, 1934) and more often have the same close friends, dress alike, study together, and attend social events together (Smith, 1965). In 1967, Kringlen published a survey of 117 twin-pairs (75 MZ, 42 DZ; where one or both were diagnosed with schizophrenia) which stands as one of the few attempts by a twin researcher to look at differences in association and

''ego fusion,'' which as we have seen was central to Jackson's theory of identity confusion. According to the results of Kringlen's survey (1967, p. 115), 91% of MZ twins experienced ''identity confusion in childhood,'' which was true for only 10% of the DZ twins. Because ADHD is seen most often in childhood, Kringlen's finding is of particular importance to the present analysis. Kringlen also found that MZ twins were more likely to have been ''considered as alike as two drops of water'' (76% vs 0%), ''brought up as a unit'' (72% vs 19%), and ''inseparable as children'' (73% vs 19%). The final question made a ''global evaluation of twin closeness.'' The results showed that 65% of identical twins were found to have an ''extremely strong'' level of closeness, which was true for only 19% of the fraternal pairs. Kringlen's findings illustrate the striking contrast between the environments (psychological or otherwise) of MZ and DZ twins.

Although the trait-relevant EEA is the most widely used definition today, it is in fact untenable. Proponents of the twin method are rarely able to pinpoint the environmental factors relevant to the condition they are studying, in spite of their belief that most psychiatric disorders require an environmental ''trigger'' in combination with a genetic predisposition. As mentioned, twin researchers attempt to place the burden of proof for the relevance of a particular environmental factor on twin method critics. According to Bouchard,

> The equal environment assumption [of the twin method] is required only for trait relevant features of the environment; features of the environment that have causal status. Causal status must be demonstrated, not assumed. . . . It is absolutely mandatory that Hoffman demonstrate that the differential treatments she cites have a causal influence on the traits whose similarity she is trying to explain. This is a very difficult task. (1993, p. 33)

For Bouchard, it is permissible to *assume* that the greater environmental similarity experienced by identical twins does not affect their greater similarity in behavior, but the assertion that the greater similarity of treatment leads to a greater similarity of behavior must be *demonstrated*. The ADHD diagnosis shows the utter implausibility of such thinking. As recently as 1991, a group of leading ADHD researchers could write that ADHD is a ''chronic condition of unknown etiology'' (Faraone, Biederman, Keenan, & Tsuang, 1991b, p. 109). However, if the etiology of ADHD is ''unknown,'' and it is demonstrated that identical twins are treated more alike, spend more time together, have more common friends, and experience greater levels of identity confusion; they are more likely to be similarly exposed to ''trait-relevant'' environmental factors—known or unknown. Contrary to the wishes of Bouchard and others, the burden of proof for showing that MZ and DZ twins are not differentially exposed to potential etiological environmental factors is placed on the shoulders of those who would claim that the twin method is able to separate genetic and environmental influences on a particu-

lar trait. However, if it could be shown that a condition required certain environmental factors, and *only* such factors, in the environmental component of its etiology, then one could conceivably test for equal exposure to this factor among groups as a prerequisite to making claims about genetic influences. This would be possible only upon the discovery of specific, definable, and measurable environmental factors. For now, the environmental etiological component of ADHD, which could well be 100%, remains unknown.

Bouchard's position becomes even more implausible when we realize that, as we have seen, most genetic researchers acknowledge that *family studies* do not prove the existence of genetic factors. Bouchard himself has written that in family studies, ''genetic and environmental effects are confounded'' (1981, p. 23). The reason, as we know, is that the clustering of a condition among family members could be caused by purely environmental factors.[1] However, few people nowadays argue for a ''trait-relevant EEA'' *for family studies*; i.e., the claim that family studies prove the existence of the operation of genetic influences unless specific environmental factors shared by family members are demonstrated to have a causal relationship to the condition in question. Quite the contrary; it is acknowledged that the simple fact that family members share a common environment confounds the results of family studies. Similarly, the twin method is confounded by the fact that MZ twins experience more correlated environments than DZ twins, even if the specific trait-relevant environmental factors are unknown. As an example of the failure of genetic researchers to apply their observations about family studies to the twin method, we turn to the words of a prominent group of twin method defenders (Lyons, Kendler, Provet, & Tsuang, 1991). These investigators recognized that family studies are ''confounded'' because ''offspring are likely to share exposure to toxic or infectious agents that could lead to similar outcomes for the siblings'' (p. 124), but they failed to recognize that *the twin method is similarly confounded by the fact that identical twins share exposure to potential toxins to a greater degree than fraternal twins.* Bouchard, Kendler, Lyons, Tsuang, and others ask for, in effect, a sort of ''special exemption'' for the twin method in the face of the overwhelming evidence that, in addition to having a greater genetic similarity, MZ twins experience more similar environments than DZs.

Ironically, the logic of the trait-relevant EEA leads not to the validity of the twin method *but rather to its obsolescence*. The reasoning behind the trait-relevant EEA leads to the conclusion that family studies offer definitive proof that the familiality of a condition demonstrates its genetic basis, unless it can be shown that the affected families under study were exposed to an

[1] As was the case with pellagra, a disease which clusters in families and was once thought to carry a strong genetic component. In the early part of the 20th century, it was discovered to be caused by a niacin deficiency (see Chase, 1980; Joseph, 2000).

environmental agent *proven* to be relevant to the condition under study. Since family studies would thereby settle the question of the existence of genetic factors, there would be little reason to conduct twin studies at all. Because twin studies are difficult and often expensive to perform, it is likely that the now-definitive nature of the new ''trait-relevant'' family studies would drive the twin method out of existence as a research method.[2] *The trait-relevant equal environment assumption has transformed the twin method into little more than a special type of family study.*

Several proponents of the twin method (e.g., Kendler, 1983) maintain that MZ twins experience more similar environments because the genetically produced similarities in their personalities induce parents and others to treat them more similarly. In other words, for Kendler and others identical twins are treated more alike because they act more alike, as opposed to the conventional idea that they act more alike because they are treated more alike. The validity of the twin method, therefore, would seem to rest on the direction of causality. As Kendler has written, ''Although the similarity in environment might make MZ twins more similar, the similarity in behavior of MZ twins might *create* for themselves more similar environments'' (1987, p. 706). This idea has been referred to as the ''twins create their environment theory'' (Joseph, 1998b),[3] and it was argued that the concept is implausible because, among other reasons, it ascribes a trait to children that it does not ascribe to adults:

> Advocates of the ''twins create their environment theory'' would have us believe that the postulated genetically predisposed personalities of children are able to greatly impact the necessarily similarly predisposed personalities and response modes of their parents. MZ and DZ twins are portrayed as genetically programmed to act in rough proportion to the number of genes they share in common, but their parents are seen as readily able to change their behavior and treatment of the twins on the basis of environmentally caused factors, i.e., the twins' personalities. Children are characterized by their inborn propensity to display inherited personality; parents are characterized by their plasticity in reacting to these personalities. If parents can change their behavior on the basis of environmental influences, as this theory explicitly maintains, it should follow that children (including twins) would *also* be able to adjust their behavior and personalities in response to environmental stimuli. If anything, genetic influences or not, we would expect adults to have personalities far less malleable than that of 5-year-old children. Yet children are portrayed as having a greater ability to change the genetically predisposed personality of adults than adults have to create, through their treatment, similar behavior in MZ twins. (Joseph, 1998b, p. 340)

---

[2] As an example of how this logic does not always play out in real life, Kallmann, in spite of having discovered ''conclusive proof'' for the genetic basis of schizophrenia in his family study (1938, p. xiv), spent the next several years studying 691 twin pairs for his famous schizophrenia twin study (1946).

[3] This concept has been referred to as a ''Reactive Genotype–Environment Correlation'' (Plomin, DeFries, & Loehlin, 1977, p. 310).

Arguing further, even if the ''twins create their environment theory'' were correct, the twin method might still be measuring nothing more than environmental effects. Contrary to Kendler's viewpoint, it is not important to determine the *reason* that identical twins spend more time together because the fact that they do means that they will be more similarly exposed to environmental etiologic agents. Suppose that ADHD were caused by an excess of lead in the brain. MZ twins would likely be more concordant for ADHD for the simple reason that they spend more time together and are therefore more similarly exposed to lead than DZ twins, who spend less time together.

The EEA and ADHD Twin Studies

Because the equal environment assumption is so crucial to the validity of the twin method, we would expect that ADHD twin researchers would address the merits of the assumption and provide evidence in support of the traditional or trait-relevant definitions of the EEA. For the most part, however, this is not the case. To the extent that they discuss the EEA at all, most ADHD twin researchers adhere to the traditional EEA definition—in spite of the fact that, in the words of twin method proponents Scarr and Carter-Saltzman, ''the evidence of greater environmental similarity for MZ than DZ twins is overwhelming'' (1979, p. 528). Let us examine how the EEA has been discussed in ADHD twin studies:

1. Lopez (1965): The EEA was not discussed.
2. Willerman (1973): The author stated that ADHD family studies are biased because ''genetic and environmental influences are not distinguishable'' and noted, remarkably, that the twin method is only ''somewhat less subject to this bias'' (p. 288).
3. Goodman and Stevenson (1989a, 1989b): The authors discovered that the effect of parents' and teachers' (incorrect) *belief* about a twin-pairs' zygosity (MZ or DZ) had a ''substantial'' effect on their scoring of the twins (p. 696). Otherwise, they did not discuss the EEA.
4. Gilger et al. (1992): The EEA was not discussed.
5. Gillis et al. (1992): The researchers acknowledged that a violation of the EEA could explain higher MZ within-pair correlations, but concluded that the evidence suggested that ''MZ and DZ environmental influences are similar'' (p. 313).
6. Stevenson (1992): The EEA was not discussed.
7. Eaves et al. (1993): The EEA was not discussed.
8. Edelbrock et al. (1995): The EEA was not discussed.
9. Thapar et al. (1995): The authors wrote, ''assuming that MZ and DZ twins share environment to the same extent, MZ twins will be more alike than DZ twins for traits that are under genetic influence'' (p. 538). However, they cited no evidence in favor of the assumption that MZ and DZ twins share equal environments.

10. Gjone et al. (1996): The EEA was not discussed.
11. Silberg et al. (1996): The EEA was not discussed.
12. van den Oord et al. (1996): The EEA was not discussed.
13. Levy et al. (1997): The authors stated that ''the assumption of equal environments between MZ and DZ twins [is] often raised as a potential complication in twin studies. If MZ twins have more similar environments than DZ twins, this could be a reason for heritability being overestimated. The consistency here of resemblance to their nontwin siblings to both MZ and DZ twins justifies the conventional equal environment assumption'' (p. 742).
14. Sherman et al. (1997): The EEA was not discussed.
15. Nadder et al. (1998): The EEA was not discussed.
16. Steffensson et al. (1999): The EEA was not discussed.

As we can see, the EEA was discussed in only 2 of the last 11 published ADHD twin studies, and no investigator claimed that the environments must be equal only as they pertain to the trait-relevant environmental factors of ADHD. Surprisingly, no ADHD twin study cited previous articles supporting the validity of the EEA. Thus implicitly or explicitly, all ADHD twin studies have based their conclusions on the traditional assumption that the environments of MZ and DZ twins are equal, yet in only one (Gillis et al., 1992) do the authors claim that these environments are equal. It appears that for ADHD twin researchers, the validity of this critical theoretical assumption is taken for granted—or is indefensible.

Faraone (1996) has written, ''The twin study is well known for its ability to separate genetic and environmental sources of etiology. . . . [The] genetic features of twinning provide a straightforward means of quantifying the impact of environmental and genetic factors on psychopathology'' (p. 596). According to Faraone, a statistically significant MZ/DZ correlation difference ''must be due to genetic factors.'' He did add that ''this conclusion will be wrong if MZ twins have environments that are more similar than those of DZ twins'' (p. 596). It appears that the determination of genetic influences from twin data is not as ''straightforward'' as Faraone believes it is because he provided no evidence in support of the validity of the EEA. In an earlier paper, Faraone and Tsuang posited, ''if twin pairs are reared in the same household, then the degree of environmental similarity between MZ twins should be no different than between DZ twins. . . . The correctness of [the EEA] is key to the valid use of the twin method'' (1995, p. 89). Unfortunately for Faraone and associates, most people, including many leading psychiatric and behavior geneticists, recognize that ''the degree of environmental similarity'' for MZ pairs is far greater than for DZ pairs.

In fact, the validity of the ''key'' assumption of the twin method as defined by Faraone and most ADHD twin researchers is not supported by the evidence and therefore no conclusions about genetic factors operating in ADHD

can be drawn from these studies. If MZ and DZ family environments are equal, how do we explain the remarkable finding by Kringlen (1967) that 91% of MZ twins—but only 10% of DZ twins—experienced identity confusion in childhood and that MZs were more likely to be ''inseparable as children'' (73% vs 19%)? According to the position of Faraone and Tsuang these figures should be equivalent. Since the evidence shows that identical twins are treated more alike, spend considerably more time together, and experience greater levels of identity confusion and closeness, we would expect that MZs—on purely environmental grounds—would demonstrate a greater correlation or concordance for most psychological traits and psychiatric conditions than DZs, including ADHD.

To summarize, ADHD twin studies are based on an unsupported theoretical assumption and therefore offer, like family studies, only a ''hint'' about the possible genetic basis of ADHD. It is quite possible, and even likely, that these studies have recorded nothing more than the greater psychological bond and environmental similarity experienced by identical twins.

ADHD ADOPTION STUDIES

Overview

The third method used to establish the genetic basis of a condition is the study of individuals who have been adopted. In theory, the adoption method is able to disentangle a person's genetic heritage from his or her rearing environment. Of course, if the twin method could satisfactorily accomplish this task, adoption studies would hardly be necessary, since they are more difficult to perform than twin studies. The well-known Danish/American schizophrenia adoption studies were performed by Kety, Rosenthal, Wender, and others. These researchers came together on the basis of a common belief that the twin method was unable to satisfactorily separate genetic and environmental influences. For example, Kety wrote,

> Twin studies are a more compelling form of genetic data [than family studies], but even twin studies depend on the assumption that the only thing that differentiates monozygotic from dizygotic twins is their genetic relatedness, and that environmental factors are somehow canceled out or randomized. But that is not the case. Monozygotic twins share much of their environment as well as their genetic endowment. They live together; they sleep together; they are dressed alike by parents; they are paraded in a double parambulator as infants; their friends cannot distinguish one from the other. In short, they develop a certain ego identification with each other that is very hard to dissociate from the purely genetic identity with which they were born. (Kety, 1978, p. 48)

And Rosenthal (1979) concluded, ''in both family and twin studies, the possible genetic and environmental factors are confounded, and one can draw conclusions about them only at considerable risk'' (p. 25). Wender, of course, is well known in the ADHD field in addition to being a schizophrenia researcher. He too has doubts about genetic inferences from twin studies:

‘‘The roles of ‘heredity’ (nature) and ‘environment’ (nurture) in the etiology of ADHD (as with other psychiatric disorders) cannot be determined by adding data from twin studies to the data from family studies’’ (Wender, 1995, p. 93). As an important advocate of adoption studies, Wender concluded that the roles of heredity and environment in ADHD ‘‘can, however, be more conclusively separated by adoption studies, in which the parents providing the genetic constitution (the biological parents) and those who provide the psychological environment (the adoptive parents) are different people’’ (p. 93).

While the method of studying adoptees as a way of definitively separating genetic and environmental influences may appear straightforward, the most important psychiatric adoption studies (e.g., Heston, 1966; Kety, Rosenthal, Wender, & Schulsinger, 1968; Kety, Rosenthal, Wender, Schulsinger, & Jacobsen, 1975; Kety et al., 1994; Rosenthal, Wender, Kety, Welner, & Schulsinger, 1971; Tienari et al., 1994) were likely confounded by the selective placement of adoptees on the basis of the socioeconomic and psychiatric status of index adoptees’ biological families (Joseph, 1999a, 1999b, in press-b; Lewontin, Rose, & Kamin, 1984). Like family and twin studies, adoption studies are susceptible to the confounding influence of environmental factors.

As of this writing, there have been five ADHD adoption studies (Alberts-Corush, Firestone, & Goodman, 1986; Cantwell, 1975; Morrison & Stewart, 1973; Safer, 1973; van den Oord, Boomsma, & Verhulst, 1994). In spite of the numerous flaws of the schizophrenia adoption studies, they possessed two important virtues not found in ADHD adoption studies: (1) most diagnoses were made blindly and (2) their authors studied or had information on the biological families of their adoptees.

Because of the difficulty in obtaining the carefully guarded records of adoptees’ biological parents, which the Danish/American researchers were able to use through their access to national registers, the authors of three ADHD adoption studies had to rely on the ‘‘Adoptive Parents’’ model, which was developed by Wender, Rosenthal, and Kety (1968). This method typically compares the psychiatric status of three (and sometimes four) types of families as follows:

1. *BH (Biological Hyperactive):* This group consists of children diagnosed with ADHD who were reared in the homes of their biological parent(s). Because the biological parents raised their own ADHD child, this group should not be confused with schizophrenia adoption studies that looked at the biological families of *adopted-away* individuals receiving diagnoses.
2. *AH (Adoptive Hyperactive):* This refers to children diagnosed with ADHD who were raised by adoptive parents with whom they share no genetic relationship.
3. *BN (Biological Normal):* This group typically consists of normal (non-

ADHD) children who are raised by their (normal) biological parents and is designated as a control group.

4. *AN (Adoptive Normal):* The AN group is comprised of adoptees with no record of ADHD or related diagnoses. Only one ADHD study utilized this group (Alberts-Corush et al., 1986).

ADHD adoption studies typically compare the diagnostic status of BH, AH, and BN families. It is important to remind the reader that no ADHD adoption study has investigated the biological families of adopted-away children. Therefore, no direct comparisons between the biological and adoptive families *of the same child* were made in these studies (Pauls, 1991). For the proponents of the genetic position, a greater rate of disturbance in the BH group when compared with AH families suggests the operation of genetic factors. According to Morrison and Stewart (1973), while consanguinity studies cannot determine whether familial clustering is due to genetic or environmental factors,

> Examining the legal parents of adopted hyperactive children could help decide the issue, for if a similar excess of ''personality disorder'' were found in the adopting parents, an environmental hypothesis for the transmission of behavior disorder could be sustained. However, if it were found that parents (and their extended families) who have adopted hyperactive children showed no such high prevalence of psychiatric illness, the argument for the genetic transmission of hyperactivity would be strengthened. (p. 888)

Morrison and Stewart, however, overlooked a crucial factor which is the Achilles heel of the Adoptive Family technique: that adoptive parents (or at least those who have gone through legal adoption procedures) constitute a population screened for mental health as part of the adoption process. They are, by definition, a group in which we would expect to find fewer psychiatric diagnoses than in the general population. Even Wender (1995) acknowledged, if only in passing, ''One problem with the adoptive parents method is that the prospective adoptive parents have usually been screened by adoption agencies and excluded if they had significant psychopathology'' (p. 95). And in an earlier discussion of the comparisons made in the Danish/American schizophrenia adoption series, Rosenthal (1971) noted, ''The screening with respect to adopting parents is well-known, since adoption agencies have long taken the view that mentally ill people do not make the kinds of parents that serve the best interests of the child'' (p. 194). Therefore a comparison of the diagnoses in two unrelated groups of families—one of which has been screened for mental health problems, and the other which has not been screened—tells us little about the possible operation of genetic factors in ADHD. Keeping this in mind, let us briefly examine the individual studies.

Safer (1973). Because Safer looked at the siblings and half-siblings of a group of 17 children who were in *foster care*, and because he compared rates among biological full-siblings and half-siblings, this report cannot be

considered a true adoption study. It would therefore be more proper to consider it a family study (as alluded to by Wender, 1995). The foster children had been born into abusive and neglectful families: ''In nearly every case, through neglect or cruelty, the natural parents mismanaged the care of these children and subsequently lost custody of them'' (Safer, 1973, p. 179). In addition, 5 of the 14 mothers for whom the researchers had information had been in jail, and three others were alcoholics; half of the fathers had been in prison, while three were alcoholics. The chaotic and abusive environments experienced by these children render the findings of this study of little value.

Safer found that the 17 index participants' full siblings were diagnosed with ADHD at a significantly higher rate than their half-siblings. However, it is not stated how much time the index children lived together with their siblings (although median ages at placement were provided). Safer concluded that it is likely that ''a genetic proclivity in association with a high rate factor increases the likelihood of [ADHD]'' (p. 184), but his study was far too limited and problematic to reach any such conclusion.

Morrison and Stewart (1973). Here, the researchers looked at the families of three groups of children: a BH group ($N = 59$), an AH group ($N = 35$), and a BN control group ($N = 41$). Relatives in each group were interviewed and diagnosed by nonblinded researchers. Morrison and Stewart claimed that their data supported the genetic position on the basis of two findings. The first was that BH group relatives were found to have a significantly greater rate of alcoholism, sociopathy, and hysteria, conditions ''for which there appears to be a genetic basis'' (p. 891). Since this study and previous family studies found an association between ADHD and these three diagnoses, Morrison and Stewart concluded that they had found evidence in favor of the genetic hypothesis. However, evidence for the genetic basis of alcoholism, sociopathy, and hysteria was (and still is) quite weak,[4] and even if it were strong it would be quite a stretch to conclude that ADHD is genetically related to them on the simple basis of association.

The second and ''perhaps more striking'' (p. 891) evidence cited by Morrison and Stewart was the finding of significantly more cases of ADHD among BH vs AH relatives. These diagnoses, however, were made retrospectively on the basis of whether the relative had been ''hyperactive, aggressive, or reckless as a young child; had been involved in antisocial behavior such as lying, cheating, fighting or truancy at home or at the school; had suffered from distractibility, poor concentration, or had specific learning problems or failure in school'' (1973, p. 889). Morrison and Stewart did not disclose whether a person could have received an ADHD diagnosis on the sole basis

[4] The question of whether these diagnoses constitute definable, discrete entities is debatable and is beyond the scope of this article. For a critique of genetic studies of criminal and antisocial behavior, see Joseph (in press-a) and Walters and White (1989). For genetic studies of alcohol, see Peele (1986).

of having had, for example, a ''learning problem'' in their youth. And as McMahon (1980, p. 148) noted, the nonblinded interviewers could have ''unwittingly'' encouraged BH relatives to provide information leading to an ADHD diagnosis.

Morrison and Stewart's claim that BH relatives had a significantly higher rate of childhood ADHD is based on the information provided in a table (p. 890). There is no indication that BH parents, who were the first-degree relatives of the hyperactive child, had significantly more childhood ADHD diagnoses than AH parents; statistical significance was found only by combining parents with aunts and uncles. Conspicuously missing from this table are the rates among grandparents and among the siblings of the hyperactive children, even though grandparents were counted in another table, and BH children had an average ''sibship size'' of 3.6 while AH children averaged 2.2 (p. 890). Therefore, the diagnostic status of over 190 biological and adoptive siblings was not reported in this study.

Most certainly, the authors' conclusion that ''These data clearly favor a genetic hypothesis for transmission'' (p. 891) cannot be sustained.

Cantwell (1975). This study looked at the families of 139 boys (50 BH, 39 AH, and 50 BN). These groups were matched on the basis of age, sex, race, and social class. Like Morrison and Stewart, Cantwell was interested in examining the relationship between ADHD and alcoholism, hysteria, and sociopathy as well as comparing ADHD rates between groups. Diagnoses were made on the basis of nonblind interviews with parents. Diagnoses of other relatives were based on information gathered in these interviews. Cantwell found significantly more cases of ADHD among BH relatives when compared to AH and BN families and also found significantly more psychiatrically ill persons. He concluded that his findings ''are strongly suggestive of genetic factors operating'' in ADHD (p. 278) and that the data supported a genetic relationship between ADHD and alcoholism, sociopathy, and hysteria.

Like Morrison and Stewart, Cantwell based his conclusions on the results of nonblind retrospective diagnoses, and in the case of relatives other than parents, diagnoses were made on the basis of the parents' descriptions. McMahon (1980) questioned the method of relying on interviews to make diagnoses:

> It would have been important to attempt to validate these diagnostic procedures using independently rated behavioral observations; medical records; reports of friends, relatives and coworkers; and, perhaps, psychological test data. The need for independent, concurrent validation of diagnoses is especially critical when dealing with demonstrably unreliable parental attempts to assess retrospectively patterns of hyperactive behavior in themselves and in their relatives. (p. 148)

McMahon noted further that there were serious problems with the reliability and validity of the children's diagnoses due to the differing methods of diagnosis used in each group.

Also like the Morrison and Stewart adoption study, we see that sibling

diagnoses are absent from the study's tables even though they comprised an easily accessible group of (BN) first-degree or (AH) adoptive relatives.

In a later essay, Cantwell (1989) wrote,

> Almost 90% of adopted children are illegitimate. They are at greater potential risk, then, for poor prenatal care and certain types of birth hazard, such as low birth weight. Single mothers, particularly pregnant teenagers, may be exposed to greater social stress during the pregnancy and at the time of the decision to give up the child, so there are both biological and social factors related to the pregnancy that may make the adopted child at greater risk. (p. 82)

If as Cantwell suggested, social and prenatal factors are sometimes responsible for an adoptee's ADHD diagnosis, it is possible that Cantwell's (and Morrison & Stewart's) AH group recorded little more than the damage done to a child before placement, which is consistent with the low rate of psychiatric diagnoses among the AH adoptive parents.

There is evidence that, in the general population, adoptees are more likely than nonadoptees to receive an ADHD diagnosis (Deutsch, 1989; Deutsch et al., 1982). If true, this casts further doubt on the already shaky conclusions of the ADHD adoption studies. In addition to the factors discussed by Cantwell (1989), we might be seeing the results of trauma produced by the abandonment by one's primary caregivers, which would again partly explain the lack of diagnoses among AH adoptive parents. As noted by Cassou, Schiff, and Stewart (1980), a more proper designation for adoption studies would be *the study of abandoned children* (''Les Études D'Enfants Abandonnés'').

Alberts-Corush et al. (1986). Utilizing psychological tests, Alberts-Corush and associates assessed attention deficits and impulsivity among the parents in groups BH, AH, BN, and AN. The researchers found significantly more attention deficits among the BH parents, but no differences in impulsivity. Alberts-Corush and associates concluded that their data ''provide support for an association between childhood hyperactivity and attentional deficits in the biological parents of hyperactives'' (p. 423), but did not conclude that they had found evidence in favor of the genetic hypothesis. For Alberts-Corush et al., the Adoptive Parents method is apparently an unsatisfactory technique for the determination of genetic factors in ADHD: ''Cross-fostering studies involving the biological and adoptive parents of the same hyperactive child would assuredly provide a more definitive analysis of the gene–environment interaction'' (p. 422).

van den Oord et al. (1994). This study differs from the others because it compared the similarities of two groups of adopted sibling pairs. The participants were international adoptees (mean age = 12.4 years, SD = 1.2 years) who had been placed into Dutch adoptive homes. About two-thirds of the adoptees were born in Korea and other Asian countries, and another 18% were born in Colombia. Three groups of adoptees were studied: a *biological sib group* consisting of 111 pairs of biologically *related* sibling adoptees

raised in the same home, a *nonbiological sib group* consisting of 221 pairs of biologically *unrelated* pairs of adoptees raised in the same home, and a third group of 94 ''only child'' adoptees was studied in order to test for the effect that having a sibling might have on adoptee behavior. Adoptees were scored on the basis of their adoptive parents' responses on the Child Behavior Checklist (CBCL).

According to van den Oord et al., a greater behavioral similarity among the biological sibs when compared to the non biological sibs would suggest the operation of genetic factors. The authors found that the biological sib group correlated significantly higher on the CBCL Attention Problems and Externalizing scales and concluded that they had found an important genetic effect on these behaviors.

Although the van den Oord and associates 1994 study has been cited by others as supporting the genetic hypothesis (e.g., Barkley, 1998b; Tannock, 1998), it contains several serious problems. First, the investigators found no significant differences between the biological and nonbiological pairs on the CBCL Total score or on the Internalizing behavior category. Looking at the Attention Problems category for pairs of boys (who are diagnosed more often than girls), we find that the biological siblings correlated at a modest .169, while the nonbiological siblings correlated at .089 (p. 200). There is no indication that this difference is statistically significant.

There are also problems with the assumption that ''the common environments were similar for the two groups of siblings'' (p. 203). While all biological pairs had the same country of origin, this was true for only 75% of the nonbiological sibling pairs (p. 195). Although most of these children were raised in The Netherlands from an early age, the ethnic composition of the sibling pairs would likely affect their level of association. It is reasonable to assume that a pair of ethnically Korean siblings would be emotionally closer than a sibling pair consisting of a Korean and an Austrian, yet a pair of the latter type could only have been found in the nonbiological group. It is also likely that non-White ''foreigners'' living in The Netherlands would have experienced greater levels of discrimination and mistreatment than the European adoptees, who constituted 14.2% of the nonbiological group, but only 2.7% of the biological sibs (p. 195). It is therefore likely that siblings with the same biological heritage (and more similar appearance) would be treated more similarly by parents and the social environment. Goodman and Stevenson (1989b) found evidence that parents' ratings of twins were affected by their expectations of how MZ and DZ twins should act. Because biologically related siblings might be expected to be more similar than nonbiologically related pairs, it is possible that just such an ''expectancy effect'' was operating in the parents' CBCL rating of their adopted siblings in the van den Oord and associates adoption study.

Another problem with this study was that although the adoptees' mean age, age difference, and age at placement were provided, there was no indica-

tion of how much time the pairs lived together or whether there was a correlation between time living in the same home and behavioral similarity. It seems more likely that the biological sibs would be placed in their adoptive home at the same time. More importantly, the biological siblings were raised in the same preplacement environments in their native countries, meaning that they were more similarly exposed to potential behavior-influencing environmental factors than the nonbiological group, who lived in more dissimilar preplacement environments. The study assessed the adoptees' preplacement environments by quantifying factors such as ''abuse,'' ''caretaking,'' ''health,'' and ''neglect.'' Mean scores were derived from an ordinal rating system and were based only on the adoptive parents' knowledge. These figures do not provide an adequate picture of the preplacement rearing environments of the two groups.

The investigators were unable to control for age of placement, which represents another potentially confounding factor in this study. The biological sibs averaged a much later age of placement (43.5 months) than the nonbiological sibs (20.7 months). An earlier study (Verhulst, Althaus, & Versluisden Bieman, 1990b) based on a larger sample of international adoptees found that for 10- to 15-year-old adoptees, ''the older the child at placement the greater the probability that the child will develop behavioral/emotional problems and/or will perform less well in school'' (p. 104). Because the biological sibs in the 1994 van den Oord et al. study were, on average, 2 years older than the nonbiological sibs at the time of placement, an important difference in disturbance-creating environments existed between the two groups.

Another study by Verhulst and associates (Verhulst, Althaus, & Versluisden Bieman, 1990a) found that, among 12- to 15-year-old boys, adoptees were three times more likely than nonadoptees to score in the deviant range on the CBCL Hyperactivity scale and twice as likely to score in the Externalizing deviant range. Regardless of the cause, it is clear that the international adoptees were at greater risk for hyperactive behavior than non adoptees. It is therefore unlikely that the results of an adoption study of this type can be generalized to the population of nonadoptees. Of course, van den Oord and colleagues would argue that the effects of the adoption process were controlled for by the use of a nonbiological adoptee group, but we have seen that the investigators were unable to control for the differing environments of the two groups of adoptee pairs.

Van den Oord and associates attempted a new approach to the study of the causes of ADHD; nevertheless, a flawed model coupled with contradictory findings does not allow for the acceptance of the researchers' conclusion that a genetic basis for ADHD was found.

Summary and Discussion of the Findings of ADHD Adoption Studies

Because it is unable to make a direct comparison between the biological and adoptive relatives *of the same adoptee,* it is unlikely that the Adoptive

Family method (used in three of the five studies) is able to provide evidence in favor of the genetic hypothesis. Even in the case of a scrupulously performed study of this type (which the ADHD Adoptive Family studies certainly were not), it is doubtful that the Adoptive Family method could offer any more than a suggestion of the operation of genetic factors (McMahon, 1980).

The fact that ADHD adoption studies typically fail to perform blind evaluations of their participants is reason enough to question their conclusions. As a leading schizophrenia adoption researcher has noted, ''With respect to all such research, in which the dependent variable is the diagnosis of relatives, it is essential that the diagnostician not know whether the individual examined is related to an index or control proband . . . because it is easy to be swayed by knowledge regarding index or control status'' (Rosenthal, 1975, p. 20). For Rosenthal, who had intimate knowledge of how these studies were performed, blind diagnoses are ''essential'' because it is ''easy'' to be influenced by knowledge of the group status of the participant under study. The authors of the ADHD adoption studies noted the difficulty of remaining blind to the status of their participants because details of the adoption process are usually disclosed in the interview process. Nevertheless, our understanding of the difficulties faced by these researchers does not mean that we must accept their conclusions.

Summarizing the evidence in favor of the genetic basis of ADHD, Wender (1995) wrote, ''What have these adoption studies added to the data on ADHD from the family and twin studies? First, they have provided more solid data showing that 'hyperactivity' (broadly defined) has genetic contributions'' (p. 99). Because, as we have seen, Wender considered family and twin studies to be confounded by environmental factors, one might ask what ''solid data'' he was referring to. Like other genetically oriented commentators, Wender implied that the alleged findings from one research method can legitimize—or ''unconfound''—the results from another. However, if family and twin studies are contaminated by environmental factors, the results from an adoption study cannot alter this finding. According to Wender, another important finding of the ADHD adoption studies was that ''they have shown that some psychiatric disorders associated with conduct disorder—'alcoholism,' Antisocial Personality Disorder ('psychopathy,' 'sociopathy'), somatization disorder ('Briquet's Syndrome,' 'hysteria')—are associated with hyperactivity and are also genetically transmitted'' (1995, p. 99). The authors of the original ADHD adoption studies (Cantwell, 1975; Morrison & Stewart, 1973) believed that there was a genetic link between ADHD and alcoholism, sociopathy, and hysteria on the basis of the (extremely weak) evidence in support of the genetic foundation of these diagnoses. That Wender continues to see a genetic linkage is based on two unlikely assumptions: (1) that the evidence in favor of the genetic basis of alcoholism, sociopathy, and hysteria is solid; and (2) that the mere association of psychiatric conditions is evidence for their *genetic* association. The most outstanding example of Wender's em-

brace of assumption 2 was his support of the questionable Danish/American ''schizophrenia spectrum'' concept (see Joseph, 1998a).

Psychiatric genetics has a long history of the mistaken belief that the mere association of conditions implies their genetic relationship. Kallmann's (1938) consanguinity study looked at the families of 1087 people diagnosed with schizophrenia who had been admitted to Herzberge Hospital in Berlin. In addition to finding that the relatives of his ''probands'' were diagnosed with schizophrenia at rates significantly higher than population expectations, he also found that patients and their relatives had died of tuberculosis at rates several times greater than in the general population. This finding led Kallmann to conclude, with certainty, that tuberculosis and schizophrenia were genetically related diseases:

> Because in our estimate of the causes of death we naturally counted only the absolutely assured deaths from tuberculosis, the assumption will have to be made for the probands that at least one third of them, and possibly even more, died of tuberculosis. Thus no doubt can remain that *within our own proband material the death rate from tuberculosis was also much higher than in the general population, and that, on the whole, a very particular significance must be assigned to tuberculosis in the entire heredity-circle of schizophrenia* [emphasis in original]. (Kallmann, 1938, p. 86)

Today it is apparent that Kallmann's ''finding'' was actually a textbook example of what is known as a spurious correlation, which has been defined as a ''correlation that results not from any direct relationship between the variables under assessment, but because of their relationships to a third variable (or fourth, or more) that has no connecting relationship between them'' (Reber, 1985, p. 161). Kallmann failed to recognize that the high rate of tuberculosis among schizophrenia patients and their relatives was the result of environmental conditions common to both schizophrenics and tuberculosis sufferers: namely that the socioeconomic and hygienic conditions of mental patients and their family members were inferior to the conditions of a typical German family. Similarly, the conclusion that alcoholism, sociopathy, and hysteria are genetically related to ADHD could be the result of a correlation as spurious as Kallmann's.

To summarize, the ADHD adoption literature reveals a handful of greatly flawed studies which, even when combined, provide (at best) inconclusive evidence in favor of either a genetic basis for ADHD or its genetic relationship to any other condition.

SUMMARY AND CONCLUSIONS

We have seen that the genetic basis of ADHD has been supported with the same types of studies cited in favor of the genetic basis of schizophrenia and other psychiatric diagnoses. There are three main ways that psychiatric geneticists and behavior geneticists have made the case for the genetic basis of ADHD: family, twin, and adoption studies. We have seen that although family studies might be able to demonstrate the familiality of ADHD, the

fact that families share a common environment as well as common genes does not permit any conclusion about a genetic component for the diagnosis.

It was argued that the classical twin method is no less confounded by environmental factors than family studies because identical twins clearly share a more similar environment than fraternals. Twin researchers have attempted to defend the assumption of equal environments but have failed to provide convincing evidence that the EEA, whether using the traditional or trait-relevant definition, is valid. It is therefore likely that the greater similarity of MZ vs DZ twins on measures related to ADHD symptoms records nothing more than the greater environmental similarity and identification of MZ twins. Typically, ADHD twin study articles discuss the EEA briefly or not at all, and in no study do the authors come out in favor the trait-relevant EEA. The conclusions of these studies, therefore, are based on the simple assumption that MZ and DZ environments are equal when it is clear that these environments are not equal.

ADHD adoption studies are greatly inferior to the already flawed schizophrenia adoption studies which preceded them and therefore offer no important evidence in favor of the genetic position. Apart from the other methodological problems with these studies, the fact that most made nonblind diagnoses and did not assess adoptees' biological relatives invalidates any inferences of the operation of genetic factors. After an examination of the total weight of evidence in favor of a genetic basis or predisposition for ADHD, it is concluded that a role for genetic factors is not supported and that future research should be directed toward psychosocial causes.

A reevaluation of the genetic evidence is important in the context of how ADHD is viewed and what directions will be taken in future research. Proponents of the brain dysfunction model of ADHD (and other psychiatric conditions) often point to the evidence from genetic studies in support of their position, since defective genes are seen as creating associated biological defects. The belief in the biological/genetic basis of ADHD has hindered investigation into possible environmental factors (McCubbin & Cohen, 1999), but it is just this area that demands greater attention. While there is little solid evidence in support of specific environmental factors, there are theories requiring further investigation. DeGrandpre (1999) sees the condition as the result of some children's problems with impulse control in our increasingly ''rapid-fire culture,'' leading to children's ''rapid-fire consciousness'':

> At the heart of the developmental problem lies the emergence of a phenomenological experience of unsettledness, characterized by feelings of restlessness, anxiety, and impulsivity. Hyperactivity and the inability to attend to mundane activities exemplify the type of escape behavior that the ''sensory addicted'' child or adult uses in order to maintain his or her needed stream of stimulation. (p. 32)

It is reasonable to propose that future research be directed toward psychosocial theories such as DeGrandpre's. If future studies are also able to detect

genetic factors, this information could be used to identify children in need of special intervention. Unfortunately, history has shown that the results of genetic studies have often been used to stigmatize individuals and groups, to discourage the search for other relevant and necessary factors, and to support the use of psychotropic drugs to treat problems caused by social and psychological factors. This article, therefore, is a necessary counterweight to the prevailing biopsychiatric/pharmacological view of ADHD.

REFERENCES

Alberts-Corush, J., Firestone, P., & Goodman, J. (1986). Attention and impulsivity characteristics of the biological and adoptive parents of hyperactive and normal control children. *American Journal of Orthopsychiatry,* **56,** 413–423.

Barkley, R. (1998a, September). Attention-deficit hyperactivity disorder. *Scientific American,* 66–71.

Barkley, R. (1998b). *Attention-deficit hyperactivity disorder: A handbook for diagnosis and treatment* (2nd ed.). New York: The Guilford Press.

Biederman, J., Faraone, S., Keenan, K., Knee, D., & Tsuang, M. (1990). Family-genetic and psychosocial risk factors in DSM-III attention deficit disorder. *Journal of the American Academy of Child and Adolescent Psychiatry,* **29,** 526–533.

Biederman, J., Faraone, S., Mick, E., Spencer, T., Wilens, T., Kiely, K., Guite, J., Ablon, S., Reed, E., & Warburton, R. (1995). High risk for attention deficit hyperactivity disorder among children of parents with childhood onset of the disorder: A pilot study. *American Journal of Psychiatry,* **152,** 431–435.

Biederman, J., Munir, K., Knee, D., Habelow, M., Autor, S., Hoge, S., & Waternaux, C. (1986). A family study of patients with attention deficit disorder and normal controls. *Journal of Psychiatric Research,* **20,** 263–274.

Bouchard, T. (1981). The study of mental ability using twin and adoption studies. *Twin research: Vol. 3. Intelligence, personality, and development* (pp. 21–23). New York: A. R. Liss.

Bouchard, T. (1993). Genetic and environmental influences on adult personality: Evaluating the evidence. In J. Hettema & I. Deary (Eds.), *Basic issues in personality* (pp. 15–44). Dordrecht, The Netherlands: Kluwer.

Breggin, P. (1998). *Talking back to Ritalin.* Monroe, ME: Common Courage Press.

Cantwell, D. (1972). Psychiatric illness in the families of hyperactive children. *Archives of General Psychiatry,* **27,** 414– 417.

Cantwell, D. (1975). Genetic studies of hyperactive children: Psychiatric illness in biologic and adopting parents. In R. Fieve, D. Rosenthal, & H. Brill (Eds.), *Genetic research in psychiatry* (pp. 273–280). Baltimore: The Johns Hopkins Press.

Cantwell, D. (1989). Comments by mentor. In L. Bloomingdale & J. Swanson (Eds.), *Attention deficit disorder, Volume IV: Current concepts and emerging trends in attentional and behavioral disorders of childhood* (pp. 79–84). New York: Pergamon Press.

Carey, G., & DiLalla, D. (1994). Personality and psychopathology: Genetic perspectives. *Journal of Abnormal Psychology,* **103,** 32–43.

Cassou, B., Schiff, M., & Stewart, J. (1980). Génétique et schizophrénie: Réévaluation d'un consensus [Genetics and schizophrenia: Re-evaluation of a consensus]. *Psychiatrie de l'enfant,* **23,** 87–201.

Chase, A. (1980). *The legacy of Malthus: The social costs of the new scientific racism.* Urbana, IL/Chicago: Univ. of Illinois Press.

DeGrandpre, R. (1999). *Ritalin nation.* New York: Norton.

Deutsch, C. (1989). Adoption and attention deficit disorder. In L. Bloomingdale & J. Swanson (Eds.), *Attention deficit disorder: Volume IV. Current concepts and emerging trends in attentional and behavioral disorders of childhood* (pp. 67–79). New York: Pergamon.

Deutsch, C., Swanson, J., Bruell, J., Cantwell, D., Weinberg, F., & Baren, M. (1982). Overrepresentation of adoptees in children with the attention deficit disorder. *Behavior Genetics,* **12,** 231–238.

Diller, L., Tanner, J., & Weil, J. (1995). Etiology of ADHD: Nature or Nurture? [Letter to the editor]. *American Journal of Psychiatry,* **153,** 451–452.

Diller, L. (1998). *Running on Ritalin.* New York: Bantam Books.

Eaves, L., Silberg., J., Hewitt, J., Meyer, J., Rutter, M., Simonoff, E., Neale, M., & Pickles, A. (1993). Genes, personality, and psychopathogy: A latent class analysis of liability to symptoms of attention-deficit hyperactivity disorder in twins. In R. Plomin & G. McClearn (Eds.), *Nature, nurture, & psychology* (pp. 285–303). Washington, DC: American Psychological Association Press.

Edelbrock, C., Rende, R., Plomin, R., & Thompson, L. (1995). A twin study of competence and problem behavior in childhood and early adolescence. *Journal of Child Psychology and Psychiatry,* **36,** 775–785.

Faraone, S. (1996). Discussion of ''Genetic influence on parent-reported attention-related problems in a Norwegian general population twin sample.'' *Journal of the American Academy of Child and Adolescent Psychiatry,* **35,** 596–598.

Faraone, S., Biederman, J., Keenan, K., & Tsuang, M. (1991a). A family-genetic study of girls with DSM-III attention deficit disorder. *American Journal of Psychiatry,* **148,** 112–117.

Faraone, S., Biederman, J., Keenan, K., & Tsuang, M. (1991b). Separation of DSM-III attention deficit disorder and conduct disorder: Evidence from a family-genetic study of American child psychiatric patients. *Psychological Medicine,* **21,** 109–121.

Faraone, S., & Tsuang, M. (1995). Methods in psychiatric genetics. In M. Tsuang, M. Tohen, & G. Zahner (Eds.), *Textbook in psychiatric epidemiology* (pp. 81–134). New York: Wiley–Liss.

Farber, S. (1981). *Identical twins reared apart: A reanalysis.* New York: Basic Books.

Gilger, J., Pennington, B., & DeFries, J. (1992). A twin study of the etiology of comorbidity: Attention-deficit hyperactivity disorder and dyslexia. *Journal of the American Academy of Child and Adolescent Psychiatry,* **31,** 343–348.

Gillis, J., Gilger, J., Pennington, B., & DeFries, J. (1992). Attention deficit disorder in reading-disabled twins: Evidence for a genetic etiology. *Journal of Abnormal Child Psychology,* **20,** 303–315.

Gjone, H., Stevenson, J., & Sundet, J. (1996). Genetic influence on parent-reported attention-related problems in a Norwegian general population twin study. *Journal of the American Academy of Child and Adolescent Psychiatry,* **35,** 588–596.

Goodman, R., & Stevenson, J. (1989a). A twin study of hyperactivity: I. An examination of hyperactivity scores and categories derived from the Rutter Teacher and Parent Questionnaires. *Journal of Child Psychology and Psychiatry,* **30,** 671–689.

Goodman, R., & Stevenson, J. (1989b). 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.

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

Gottesman, I., & Shields, J. (1966). Contributions of twin studies to perspectives on schizo-

phrenia. In B. Maher (Ed.), *Progress in experimental personality research* (Vol. 3, pp. 1– 84). New York: Academic Press.

Gottesman, I., & Shields, J. (1972). *Schizophrenia and genetics: A twin study vantage point.* New York: Academic Press.

Heston, L. (1966). Psychiatric disorders in foster home reared children of schizophrenic mothers. *British Journal of Psychiatry,* **112,** 819–825.

Jackson, D. (1960). A critique of the literature on the genetics of schizophrenia. In D. Jackson (Ed.), *The etiology of schizophrenia* (pp. 37–87). New York: Basic Books.

Joseph, J. (1998a). *A critical analysis of the genetic theory of schizophrenia.* Unpublished doctoral dissertation, California School of Professional Psychology, Alameda.

Joseph, J. (1998b). The equal environment assumption of the classical twin method: A critical analysis. *Journal of Mind and Behavior,* **19,** 325–358.

Joseph, J. (1999a). A critique of the Finnish Adoptive Family Study of Schizophrenia. *Journal of Mind and Behavior,* **20,** 133–154.

Joseph, J. (1999b). The genetic theory of schizophrenia: A critical overview. *Ethical Human Sciences and Services,* **1,** 119– 145.

Joseph, J. (2000). Potential confounds in psychiatric genetic research: The case of pellagra. *New Ideas in Psychology,* **18,** 83–91.

Joseph, J. (in press-a). A critical review of twin and adoptive studies of criminality and antisocial behavior. *Journal of Mind and Behavior.*

Joseph, J. (in press-b). The Danish-American adoptees' family studies of Kety and associates: Do they provide evidence in support of the genetic basis of schizophrenia? *Genetic, Social, and General Psychology Monographs.*

Joseph, J. (in press-c). Don Jackson's ''A critique of the literature on the genetics of schizophrenia''—A reappraisal after 40 years. *Genetic, Social, and General Psychology Monographs.*

Joseph, J. (in press-d). Separated twins and the genetics of personality differences: A critique. *American Journal of Psychology.*

Kallmann, F. (1938). *The genetics of schizophrenia: A study of heredity and reproduction in the families of 1,087 schizophrenics.* New York: J. J. Augustin.

Kallmann, F. (1946). The genetic theory of schizophrenia: An analysis of 691 schizophrenic twin index families. *American Journal of Psychiatry,* **103,** 309–322.

Kamin, L. (1974). *The science and politics of I.Q.* Potomac, MD: Erlbaum.

Kendler, K. (1983). Overview: A current perspective on twin studies of schizophrenia. *American Journal of Psychiatry,* **140,** 1413–1425.

Kendler, K. (1987). The genetics of schizophrenia: A current perspective. In H. Meltzer (Ed.), *Psychopharmacology: The third generation of progress* (pp. 705–713). New York: Raven Press.

Kendler, K. (1993). Twin studies of psychiatric illness: Current status and future directions. *Archives of General Psychiatry,* **50,** 905–915.

Kendler, K., Neale, M., Kessler, R., Heath, A., & Eaves, L. (1993). A test of the equal-environment assumption in twin studies of psychiatric illness. *Behavior Genetics,* **23,** 21–27.

Kendler, K., Neale, M., Kessler, R., Heath, A., & Eaves, L. (1994). Parental treatment and the equal environment assumption in twin studies of psychiatric illness. *Psychological Medicine,* **24,** 579–590.

Kety, S. (1978). Heredity and environment. In J. Shershow (Ed.), *Schizophrenia: Science and practice* (pp. 47–68). Cambridge, MA: Harvard Univ. Press.

Kety, S., Rosenthal, D., Wender, P., & Schulsinger, F. (1968). The types and prevalence of mental illness in the biological and adoptive families of adopted schizophrenics. In D. Rosenthal & S. Kety (Eds.), *The transmission of schizophrenia* (pp. 345–362). New York: Pergamon Press.

Kety, S., Rosenthal, D., Wender, P., Schulsinger, F., & Jacobsen, B. (1975). Mental illness in the biological and adoptive families of adopted individuals who have become schizophrenic: A preliminary report based on psychiatric interviews. In R. Fieve, D. Rosenthal, & H. Brill (Eds.), *Genetic research in psychiatry* (pp. 147–165). Baltimore: The Johns Hopkins Press.

Kety, S., Wender, P., Jacobsen, B., Ingraham, L., Jansson, L., Faber, B., & Kinney, D. (1994). Mental illness in the biological and adoptive relatives of schizophrenic adoptees: Replication of the Copenhagen study to the rest of Denmark. *Archives of General Psychiatry,* **51,** 442–455.

Kläning, U., Mortensen, P., & Kyvik, K. (1996). Increased occurrence of schizophrenia and other psychiatric illnesses among twins. *British Journal of Psychiatry,* **168,** 688–692.

Kringlen, E. (1967). *Heredity and environment in the functional psychoses: An epidemiological-clinical study.* Oslo: Universitetsforlaget.

Lewontin, R., Rose, S., & Kamin, L. (1984). *Not in our genes.* New York: Pantheon.

Levy, F., Hay, D., McStephen, M., Wood, C., & Waldman, I. (1997). Attention-deficit hyperactivity disorder: A category or a continuum? Genetic analysis of a large-scale twin study. *Journal of the American Academy of Child and Adolescent Psychiatry,* **36,** 737–744.

Lopez, R. (1965). Hyperactivity in twins. *Canadian Psychiatric Association Journal,* **10,** 421–426.

Lyons, M., Kendler, K., Provet, A., & Tsuang, M. (1991). The genetics of schizophrenia. In M. Tsuang, K. Kendler, & M. Lyons (Eds.), *Genetic issues in psychosocial epidemiology* (pp. 19–52). New Brunswick, NJ: Rutgers Univ. Press.

McCubbin, M., & Cohen, D. (1999). Empirical, ethical, and political perspectives on the use of methylphenidate. *Ethical Human Sciences and Services,* **1,** 81–101.

McMahon, R. (1980). Genetic etiology in the hyperactive child syndrome: A critical review. *American Journal of Orthopsychiatry,* **50,** 145–150.

Morris-Yates, A., Andrews, G., Howie, P., & Henderson, S. (1990). Twins: A test of the equal environments assumption. *Acta Psychiatrica Scandinavica,* **81,** 322–326.

Morrison, J., & Stewart, M. (1971). A family study of the hyperactive child syndrome. *Biological Psychiatry,* **3,** 189–195.

Morrison, J., & Stewart, M. (1973). The psychiatric status of the legal families of adopted hyperactive children. *Archives of General Psychiatry,* **28,** 888–891.

Nadder, T., Silberg, J., Eaves, L., Maes, H., & Meyer, J. (1998). Genetic effects on ADHD symptomatology in 7- 13-year-old twins: Results from a telephone survey. *Behavior Genetics,* **28,** 83–99.

Nichols, P., & Chen, T. (1981). *Minimal brain dysfunction.* Hillsdale, NJ: Erlbaum.

Pam, A. (1995). Biological psychiatry: Science or pseudoscience? In C. Ross & A. Pam (Eds.), *Pseudoscience in biological psychiatry: Blaming the body* (pp. 7–84). New York: Wiley.

Pam, A., Kemker, S., Ross, C., & Golden, R. (1996). The ''equal environment assumption'' in MZ–DZ comparisons: An untenable premise of psychiatric genetics? *Acta Geneticae Medicae et Gemellologiae,* **45,** 349–360.

Pauls, D. (1991). Genetic factors in the expression of attention-deficit hyperactivity disorder. *Journal of Child and Adolescent Psychopharmacology,* **1,** 353–360.

Peele, S. (1986). The implications and limitations of genetic models of alcoholism and other addictions. *Journal of Studies on Alcohol,* **47,** 63–73.

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

Plomin, R., DeFries, J., & McClearn, G. (1990). *Behavioral genetics: A primer* (2nd ed.). New York: W. H. Freeman.

Reber, A. (1985). *The Penguin dictionary of psychology.* London: Penguin Books.

Rose, R. (1991).Twin studies and psychosocial epidemiology. In M. Tsuang, K. Kendler, & M. Lyons (Eds.), *Genetic issues in psychosocial epidemiology* (pp. 12–32). New Brunswick, NJ: Rutgers Univ. Press.

Rosenthal, D. (1960). Confusion of identity and the frequency of schizophrenia in twins. *Archives of General Psychiatry*, **3,** 101–108.

Rosenthal, D. (1970). *Genetic theory and abnormal behavior.* New York: McGraw-Hill.

Rosenthal, D. (1971). A program of research on heredity in schizophrenia. *Behavioral Science*, **16,** 191–201.

Rosenthal, D. (1975). The spectrum concept in schizophrenic and manic-depressive disorders. In D. Freedman (Ed.), *Biology of the major psychoses* (pp. 19–25). New York: Raven.

Rosenthal, D. (1979). Genetic factors in behavioural disorders. In M. Roth & V. Cowie (Eds.), *Psychiatry, genetics and pathography: A tribute to Eliot Slater* (pp. 22–33). London: Oxford Univ. Press.

Rosenthal, D., Wender, P., Kety, S., Welner, J., & Schulsinger, F. (1971). The adopted-away offspring of schizophrenics. *American Journal of Psychiatry*, **128,** 307–311.

Rüdin, E. (1916). *Zur Vererbung und Neuentstehung der Dementia praecox.* Berlin: Springer-Verlag OHG.

Safer, D. (1973). A familial factor in minimal brain dysfunction. *Behavior Genetics*, **3,** 175–186.

Scarr, S. (1968). Environmental bias in twin studies. *Eugenics Quarterly*, **15,** 34–40.

Scarr, S., & Carter-Saltzman, L. (1979). Twin method: Defense of a critical assumption. *Behavior Genetics*, **9,** 527–542.

Sherman, D., Iacono, W., & McGue, M. (1997). Attention-deficit hyperactivity disorder dimensions: A twin study of inattention and impulsivity-hyperactivity. *Journal of the American Academy of Child and Adolescent Psychiatry*, **36,** 745–753.

Silberg, J., Rutter, M., Meyer, J., Maes, H., Hewitt, J., Simonoff, E., Pickles, A., & Loeber, R. (1996). Genetic and environmental influences on the covariation between hyperactivity and conduct disturbance in juvenile twins. *Journal of Child Psychology and Psychiatry*, **37,** 803–816.

Smith, R. (1965). A comparison of socioenvironmental factors in monozygotic and dizygotic twins, testing an assumption. In S. Vandenberg (Ed.), *Methods and goals in human behavior genetics* (pp. 45–61). New York: Academic Press.

Steffensson, B., Larsson, J., Fried, I., El-Sayed, E., Rydelius, P., & Lichtenstein, P. (1999). Genetic disposition for global maturity: An explanation for genetic effects on parental report of ADHD. *International Journal of Behavioral Development*, **23,** 357–374.

Stevenson, J. (1992). Evidence for a genetic etiology in hyperactivity in children. *Behavior Genetics*, **22,** 337–344.

Tannock, R. (1998). Attention deficit hyperactivity disorder: Advances in cognitive, neurobiological, and genetic research. *Journal of Child Psychology and Psychiatry*, **39,** 65–99.

Taylor, H. (1980). *The IQ game: A methodological inquiry into the heredity–environment controversy.* New Brunswick, NJ: Rutgers Univ. Press.

Thapar, A., Hervas, A., & McGuffin, P. (1995). Childhood hyperactivity scores are highly

heritable and show sibling competition effects: Twin study evidence. *Behavior Genetics,* **25,** 537–544.

Tienari, P., Wynne, L., Moring, J., Lahti, I., Naarala, M., Sorri, A., Wahlberg, K., Saarento, O., Seitamaa, M., Kaleva, M., & Läsky, K. (1994). The Finnish adoptive family study of schizophrenia. *British Journal of Psychiatry,* **164**(Suppl. 23), 20–26.

van den Oord, E., Boomsma, D., & Verhulst, F. (1994). A study of problem behaviors in 10- to 15-year-old biologically related and unrelated international adoptees. *Behavior Genetics,* **24,** 193–205.

van den Oord, E., Verhulst, F., & Boomsma, D. (1996). A genetic study of maternal and paternal ratings of problem behaviors in 3-year-old twins. *Journal of Abnormal Psychology,* **105,** 349–357.

Verhulst, F., Althaus, M., & Versluis-den Bieman, H. (1990a). Problem behavior in international adoptees: I. An epidemiological study. *Journal of the American Academy of Child and Adolescent Psychiatry,* **29,** 94–103.

Verhulst, F., Althaus, M., & Versluis-den Bieman, H. (1990b). Problem behavior in international adoptees: II. Age at placement. *Journal of the American Academy of Child and Adolescent Psychiatry,* **29,** 104–111.

Walters, G., & White, T. (1989). Heredity and crime: Bad genes or bad research? *Criminology,* **27,** 455–485.

Welner, Z., Welner, A., Stewart, M., Palkes, H., & Wish, E. (1977). A controlled study of siblings of hyperactive children. *Journal of Nervous and Mental Disease,* **165,** 110–117.

Wender, P. (1995). *Attention-deficit hyperactivity disorder in adults.* New York: Oxford Univ. Press.

Wender, P., Rosenthal, D., & Kety, S. (1968) A psychiatric assessment of the adoptive parents of schizophrenics. In D. Rosenthal & S. Kety (Eds.), *The transmission of schizophrenia* (pp. 235–250). New York: Pergamon Press.

Willerman, L. (1973). Activity level and hyperactivity in twins. *Child Development,* **44,** 288–293.

Wilson, P. (1934). A study of twins with special reference to heredity as a factor determining differences in environment. *Human Biology,* **6,** 324–354.

Received: September 15, 1999