The Missing Gene

Psychiatry, Heredity, and the Fruitless Search for Genes

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Chapter 1. Introduction. The Twin Method: Science or Pseudoscience?

In 2003, American psychiatry heralded the beginning of the "genomics era" by proclaiming that gene discoveries "will change the way we treat psychiatric patients,"[1] will lead to the development of "targeted therapies," and will make possible the creation of "pharmacogenomics," which will use genetic information to predict the efficacy of psychiatric drugs. Finally, "one of the most important consequences of genomics will be to individualize treatment by allowing a clinician to tailor therapy on the basis of the unique genotype of each patient rather than the mean responses of groups of unrelated patients."[2]

A problem with these claims is that, despite having searched for over two decades with increasingly sophisticated technology, researchers have found no genes that cause the major psychiatric disorders. This might have compelled psychiatry to rethink the position that its disorders are caused by faulty genes, but this has not occurred. A major reason is that a belief in the genetic basis of mental disorders is a cornerstone of contemporary psychiatry. Yet, the evidence supporting this position is stunningly weak. In this book I show why this is so, and why there must be a massive rethinking of the role of genetics in causing psychiatric disorders. As we will see, it is very possible that genes for diagnoses such as schizophrenia, ADHD, autism, and bipolar disorder are not, as researchers often write, "elusive," but that they are nonexistent. Thus, the "genomics era" in psychiatry may develop into little more than a historical footnote.

In my previous book, *The Gene Illusion: Genetic Research in Psychiatry and Psychology under the Microscope*, I argued that the foundations of genetic theories —

1. Insel & Collins, 2003, p. 618.
2. Ibid., p. 618.

family, twin, and adoption studies — are based on poor methodology, bias, and a reliance on unsupported theoretical assumptions. My focus was split between research on psychiatric disorders such as schizophrenia and research into "normally distributed" psychological traits such as IQ and personality. In the present book I focus on genetic research in psychiatry, and cover several areas mentioned only briefly or not at all in *The Gene Illusion*. Moreover, some areas, such as molecular genetic research, have seen many changes and are in constant need of update. Two additional chapters focus on how genetic research in psychiatry has been *mis*reported by influential secondary sources. In fact, the misreporting of psychiatric genetic research is a running theme throughout the entire book.

The early part of the 21rst century is characterized by the *geneticization* of diseases and human differences, a term coined by Abby Lippman in 1992.[1] As Lippman subsequently described it, geneticization "capture[s] the ever growing tendency to distinguish people from one another on the basis of genetics; to define most disorders, behaviors, and physiological variations as wholly or in part genetic in origin."[2] This is particularly evident in psychiatry, where psychological, interpersonal, social, and cultural understandings of human suffering have taken a back seat to biological and genetic theories.

The field of *psychiatric genetics* was founded by Ernst Rüdin and his German colleagues in the early part of the 20th century.[3] German psychiatric geneticists used family and twin studies in an attempt to establish the genetic basis of psychiatric disorders. Their primary goal was to promote the eugenic program (called "racial hygiene" in Germany) of curbing the reproduction of people they viewed as carrying the "hereditary taint of mental illness," and in the process its leaders became willing accomplices of Hitler and the Nazis (see Chapter 6).[4] Rüdin and his colleagues played an important role in training people to conduct psychiatric genetic research, and people from other European countries came to Munich to study under them at the Genealogical-Demographic Department of the Kaiser-Wilhelm Institute of Psychiatry.

Contemporary psychiatric geneticists trace their discipline back to Rüdin's "Munich School," often utilizing the same methods and statistical formulations. Today, psychiatric geneticists investigate the causes of mental disorders in order to better treat and prevent them.[5] Unlike researchers in the previous era, they usually avoid discussions of eugenics in relation to their findings. The implications of their theories, however, are obvious, and they often promote the use of "genetic counseling," or recommending to people with certain psychiatric diagnoses not to conceive and bear children. The field of *behavior*

1. Lippman, 1992.
2. Lippman, 1998, p. 64.
3. See Proctor, 1988, Weindling, 1989 for a more detailed account of the birth of German psychiatric genetics.
4. Joseph, 2004b, Chapter 2; Müller-Hill, 1998a; Proctor, 1988.
5. Faraone, Tsuang, & Tsuang, 1999.

genetics uses methods similar to psychiatric genetics (e.g., family, twin, and adoption studies), but focuses on assessing genetic influences on "continuously distributed" psychological traits such as personality and IQ, and to a lesser extent on psychiatric disorders.

In this book I discuss behaviors and mental states classified by psychiatry as "mental disorders" or "mental illnesses." These include "schizophrenia," "bipolar disorder" "attention-deficit hyperactivity disorder," and so on. I use these terms only to show that, even if discrete mental disorders actually exist as valid and reliable (biological) entities, as mainstream psychiatry claims, there is little evidence that they have a genetic basis. A far better way of understanding human suffering and abnormal behavior is captured in psychologist Richard Bentall's 2003 "Post Kraepelinian Manifesto" (Emil Kraepelin was the Swiss pioneer of modern psychiatry). In Bentall's view, "There is no clear boundary between mental health and mental illness. Psychological complaints exist on a continuum with normal behaviors and experiences. Where we draw the line between sanity and madness is a matter of opinion." Bentall continued that there "are no discrete mental illnesses. Categorical diagnoses fail to capture adequately the nature of psychological complaints for either research or clinical purposes."[1]

A Brief Introduction to the Chapters

Chapter 2 examines evidence supporting the claim that attention-deficit hyperactivity disorder (ADHD) is influenced by genetic factors. Here, I highlight the invalidating flaws of family, twin, and adoption research in this area, and argue that there is little scientifically acceptable evidence supporting a genetic basis for the condition. I discuss ADHD molecular genetic research in Chapter 11.

Chapter 3 takes a close look a crucial aspect of schizophrenia adoption research: the "schizophrenia spectrum" concept. Expanding the definition of schizophrenia was an essential factor in the famous Danish-American schizophrenia adoption studies' conclusions in favor of genetics. However, we will see that the spectrum concept does not hold up to critical examination. My analysis is relevant today for two main reasons: (1) because these adoption studies remain the most frequently cited evidence in support of the genetic theory of schizophrenia, and (2) as an example of how a close examination of one aspect of genetic research can uncover serious and invalidating flaws.

Chapter 4 engages in a bit of historical speculation by predicting the results of twin and adoption studies of pellagra, an early 20th century disease ultimately discovered to be caused by a vitamin deficiency linked to malnutrition. My analysis suggests that the results from twin and adoption studies, often cited in

1. Bentall, 2003, p. 143.

support of genetic influences on psychiatric disorders, can be explained on the basis of environmental factors, in addition to methodological flaws and biases.

Chapter 5 looks closely at 43 psychiatry and psychology textbooks' discussions of schizophrenia adoption research. As I demonstrate, this body of research has been systematically misrepresented in these textbooks, and the original investigators' conclusions usually are taken at face value at the expense of a critical examination of their studies' glaring weaknesses. Chapter 5 will be of particular interest to professionals and academics, who often rely on textbooks for information on genetic research in psychiatry.

Chapter 6 takes a close look at perhaps the most relied upon secondary source in the "Genetics of Schizophrenia" area: Irving Gottesman's award-winning 1991 *Schizophrenia Genesis*. This book contains a diagram, reproduced or referred to in many contemporary psychiatry and abnormal psychology textbooks, listing various kinship risk factors for schizophrenia. I show that this book contains many errors and omissions, all in the direction of leading its readers to the mistaken conclusion that schizophrenia is strongly influenced by genetic factors. Although Gottesman is a well-known and respected psychologist, he provides a potentially misleading account of the topic to a new generation of students and professionals.

Chapter 7 examines the evidence supporting autism as a genetically-influenced disorder. Indeed, autism is often regarded as the psychiatric disorder most strongly influenced by genetics. Strikingly, however, we will see that the evidence, consisting mainly of four small twin studies, is plausibly explained on non-genetic grounds. In contrast to other psychiatric disorders, there is evidence suggesting that autism is caused by biological factors. However, "biological" is not the same as "genetic," and the current emphasis on alleged genetic factors detracts and drains away resources from research on potentially relevant biological causes, such as prenatal and postnatal exposure to mercury and other harmful substances. I discuss autism molecular genetic research in Chapter 11.

Chapter 8 focuses on an astonishing 1942 debate in the *American Journal of Psychiatry*. There, neurologist Foster Kennedy argued in favor of "euthanizing" (that is, killing) "defective" and "feebleminded" people. This had already occurred in Nazi Germany, where, even before the Holocaust, tens of thousands of "hereditarily defective" people were exterminated with the active participation of psychiatrists.[1] In response, child psychiatrist Leo Kanner argued against killing because, among other reasons, there would remain fewer people to perform society's dirty work. Kennedy and Kanner were followed by an anonymous editorial leaning towards Kennedy's position in favor of killing, whose authors called upon psychiatrists to focus their attention on the "morbid" attachment of parents opposed to the "disposal by euthanasia of their idiot offspring."

1. Lifton, 1986; Müller-Hill, 1998a.

Chapter 9 surveys a large body of literature cited in support of the equal environment assumption (EEA) of the twin method. Although I show later in Chapter 1 that the EEA is untenable regardless of how twin researchers have defined it, twin method results are accepted without question in mainstream contemporary psychiatry. A major reason is that leading twin researchers argue that the EEA is supported by a number of empirical studies. In Chapter 9, I critically review these studies and conclude that they do little to uphold the validity of the EEA. Moreover, I argue that the twin method is invalidated on its face by the fact that, as most twin researchers now recognize, identical twins experience much more similar environments than fraternals.

In *Chapter 10* I examine evidence put forward in support of genetic influences on bipolar disorder (manic depression). As with schizophrenia, ADHD, and autism, I show that the available evidence from kinship research lends little support to genetic theories of causation. I also show that many authoritative secondary sources have made incorrect or misleading claims about the results of bipolar disorder genetic research.

Chapter 11 consists of an up-to-the-moment assessment of psychiatric molecular genetic research. The most remarkable result of this research is that, despite over two decades of sustained work, genes for the major psychiatric disorder have not been discovered. Virtually all previous claims in favor of gene findings have failed replication attempts in subsequent studies. The standard explanation has been that many genes of small effect cause these disorders, and that we are on the threshold of gene discoveries. In this chapter I offer an alternative explanation, which relates to a major theme of this book: *It is unlikely that genes for the major psychiatric disorders exist*. Having shown in previous chapters that studies of families, twins, and adoptees are faulty, I analyze molecular genetic research in schizophrenia, ADHD, autism, and bipolar disorder. I argue that the fruitless search for genes may be the result of psychiatry's misplaced faith in the results of these previous kinship studies. Molecular genetic research in psychiatry is reaching the crisis stage as negative results continue to pile up, and researchers may be more open to questioning whether the genes they are looking for actually exist.

The Classical Twin Method

"The knowledge that certain diseases run in families," observed Joseph Alper, "is thousands of years old."[1] Today, it is widely understood that a condition "running in families" can be explained by any number of environmental factors related to the physical and psychological environments shared by family

1. Alper, 2002, p. 17.

members. For this reason, most psychiatric geneticists recognize that family studies are unable to disentangle the possible influences of genes and environment. Thus twin studies, which allegedly *are* able to separate these potential influences, are the most frequently cited evidence supporting genetic theories in psychiatry. However, we will see that the main premise of twin studies, upon which genetic theories are based, is faulty.

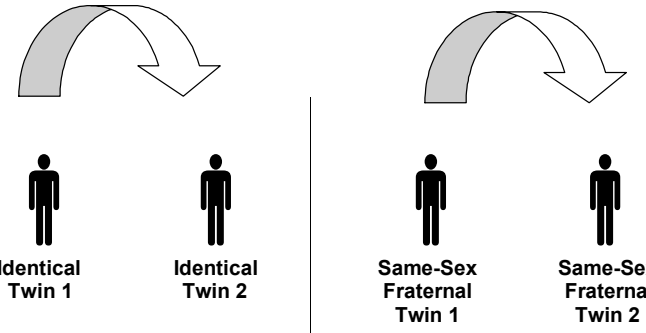
The main tool of psychiatric genetics is the "classical twin method," more commonly known as "the twin method." As seen in Figure 1.1, the twin method compares the concordance rates or correlations of reared-together identical twins (sharing 100% genetic similarity), versus the same measures of reared-together same-sex fraternal twins (averaging 50% genetic similarity). Based on the assumption that both types of twins experience the same kinds of environments, known as the "equal environment assumption" or "EEA," twin researchers attribute a statistically significant higher concordance rate among identical versus same-sex fraternals to genetic factors. Twins are said to be *concordant* when both members of a pair are diagnosed with the same disorder and *dis*cordant when only one is diagnosed.

Perhaps at this point I should briefly outline the concept of "statistical significance," which is central to genetic research. By convention, a result is considered statistically significant when its occurrence would be expected by chance less than 5 times in 100 (probability < 5%, or 0.05). If the probability value (often shortened to "p-value") that a finding occurred by chance exceeds 5% in a comparison between groups, the researcher should conclude that there is no difference between the groups. (The logic here is similar to that which applies when we are flipping a coin 10 times. Although we expect heads to come up 5 times with a fair coin, by chance alone it might come up 7 times, or perhaps 3 times. However, if it comes up heads 9-10 times, or 0-1 times, we might suspect that the coin has been altered in some way.)

Using twin research as an example, suppose a research team performs a schizophrenia twin study and finds that 7/17 (41%) identical pairs are concordant for schizophrenia, versus a same-sex fraternal rate of 2/15 (13%). Although the percentage difference appears large, the researchers use a test of statistical significance (in this case, a Fisher's Exact Test) and find a p-value of .086. They would therefore conclude, having failed to find a p-value below the conventional .05 threshold, that their study found no concordance rate difference between identical and fraternal twins. That is, they failed to uncover evidence that would lead them to reject the hypothesis that identical and fraternal twin concordance rates are the same (known as the "null hypothesis" in statistical language). The practice of using small representative samples of a population as a basis for making claims about the population as a whole uses the concept of *inferential statistics*. If results from the sample are unlikely to have been obtained by chance (statistical significance has been reached), researchers make inferences about this finding to the entire population.

Figure 1.1

THE CLASSICAL TWIN METHOD AND ITS ASSUMPTIONS



Identical Twins	***Same-Sex Fraternal Twins***
Share 100% of the same genes.	Share on average 50% of the same genes.
Reared together in the same family.	Reared together in the same family.

The greater resemblance of identical (MZ) versus same-sex fraternal (DZ) twins is attributable to genetic factors and is generalizable to the non-twin population, *assuming that all of the following are true:*

- There are only two types of twins, identical and fraternal.
- Investigators are able to reliably distinguish between these two types of twins.
- The risk of receiving the diagnosis is the same among twins and non-twins (generalizability).
- The risk of receiving the diagnosis is the same among individual identical twins as a population, versus individual fraternal twins as a population.
- Identical twin pairs and same-sex fraternal twin pairs experience the same emotional and psychological bond with each other, as well as experiencing roughly the same social, treatment, and physical environments (known as the "equal environment assumption" or "EEA").

Adapted with revisions from Joseph, 2004b, p. 22.

Returning to our imaginary schizophrenia twin study, although a larger sample showing the same concordance rates would have produced statistically significant results ($p < .05$), we cannot assume that this would have occurred,

and it might be necessary for the researchers to obtain a larger twin sample for the next study. We must also be aware that even if a study finds a statistically significant difference between groups, it has not necessarily shown what *causes* the difference. In the case of twin concordance for psychiatric disorders, a significantly higher identical versus fraternal concordance rate could be caused by identical twins' greater genetic similarity, as twin researchers maintain, or to identicals' greater environmental similarity, as maintained by critics of the twin method. As we will see throughout this book, genetic theories in psychiatry are based in large part on the claim that identical versus same-sex fraternal twin concordance rate differences are caused by genetic factors.

Although the twin method depends on additional assumptions (listed in Figure 1.1), the equal environment assumption has been the main area of contention between twin researchers and their critics. Identical and fraternal twins are usually referred to as monozygotic (MZ) and dizygotic (DZ), respectively, in the scientific literature (as seen in many quotations in this book). I use "identical" and "fraternal," here. Unless otherwise mentioned, all twin studies discussed in this book are of twins reared together in the same family. For an analysis of problems in reared-apart twin research (other than the brief critique below), I refer readers to previous publications.[1]

Twin studies constitute the main evidence cited in support of genetic influences on psychiatric disorders and all types of psychological traits, as counterintuitive as the genetic basis of some traits may seem.[2] In *The Gene Illusion*, I discussed important problems in applying the twin method to psychiatric disorders, which I summarize here.

Methodological problems with studies utilizing the twin method have included (1) the acceptance of unsupported theoretical assumptions; (2) investigator bias in favor of genetic conclusions; (3) the lack of an adequate and consistent definition of the trait or condition under study; (4) the use of non-blinded diagnoses; (5) diagnoses that were made on the basis of sketchy information; (6) inadequate or biased methods of zygosity determination (whether a pair is identical or fraternal); (7) the fact that twins, and identical twins in particular, might have received similar hospital diagnoses because hospital psychiatrists viewed them as sharing a common genetic heritage; (8) the unnecessary use of age-correction formulas; (9) the use of non-representative sample populations; and (10) the lack of adequate descriptions of the methods.

The main problem, however, has been the unwarranted acceptance of the equal environment assumption. From the development of the twin method in the mid-1920s until the early 1960s, twin researchers defined the EEA, without qual-

1. See Farber, 1981; Joseph, 2001c, 2004b; Kamin, 1974; Kamin & Goldberger, 2002; Taylor, 1980.

2. In 2004, for instance, a group of researchers claimed that twin studies found important genetic influences on "perfectionism," and another group claimed genetic influences on "breakfast eating patterns" (Keski-Rahkonen et al., 2004; Tozzi et al., 2004).

ification, as the assumption that identical and fraternal twins share the same types of behavior-influencing, physical, and treatment environments. I have called this the "traditional EEA definition."[1] However, most research assessing these environments has shown, as critics have charged since the 1920s, that identical twins spend more time together, more often have the same friends, are treated more similarly by parents and others, and so forth.[2] Moreover, identicals share a closer emotional bond than fraternals, and more often view themselves as being two halves of the same whole (i.e., they experience what some psychologists call identity confusion[3]).

Faced in the early 1960s with mounting evidence against the EEA, twin researchers should have abandoned the twin method as being unable to disentangle possible genetic and environmental influences on psychiatric disorders. Today, most twin researchers concede the fact that identical twins experience more similar environments than fraternal twins. However, they continue to uphold the twin method as a valid instrument for the detection of genetic influences.

Twin researchers accomplished this by *redefining* the EEA. The new definition, which today predominates, has been called the "equal trait-relevant environment assumption" by behavior geneticists.[4] Here, I will shorten this term to read "trait-relevant EEA." An early example of this crucial change in definition was put forward in 1966 by twin researchers Irving Gottesman and James Shields, who wrote that, to invalidate schizophrenia twin studies, the "environments of MZ twins [must be] systematically more alike than those of DZ twins in features which can be *shown* to be of etiological significance in schizophrenia."[5] More recently, psychiatric genetic twin researcher Kenneth Kendler and his colleagues have defined the EEA in the trait relevant sense:

> 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* [emphasis added].[6]

Trait-relevant EEA proponents recognize that identical twins experience more similar environments than fraternals, but argue or imply that the burden of proof for demonstrating that identical and fraternal twins experience dissimilar trait-relevant environments falls not on twin researchers but on twin method *critics*.

1. Joseph, 2004b.
2. Ibid.
3. Jackson, 1960.
4. Carey & DiLalla, 1994.
5. Gottesman & Shields, 1966a, pp. 4-5. Emphasis in original.
6. Kendler, Neale, et al., 1993, p. 21.

Although now widely used, the trait-relevant EEA rests on shaky theoretical foundations. 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 get around this problem by charging critics with responsibility for showing that identical and fraternal twin environments differ on trait-relevant dimensions (see the discussion below).

What twin researchers fail to recognize, however, is that the trait-relevant EEA has transformed the twin method into little more than a special type of family study. The reason is that the comparison groups in both family studies and the twin method (in family studies, the general population or a control group in the twin method, fraternal twins) are acknowledged to experience environments different to those of the experimental groups (in family studies, the families of people diagnosed with the disorder in question; in the twin method, identical twins). Although contemporary twin researchers retain the trait-relevant requirement for the twin method, but *not* for family studies, virtually every argument they make in defense of drawing genetic inferences from identical-fraternal comparisons they could also make in defense of drawing genetic inferences from family studies. Yet, strangely, these investigators uphold the validity of the EEA and the twin method even as they admit that genetic conclusions based on family studies are confounded by environmental factors. Although most psychiatric geneticists recognize that the results of family studies prove nothing about genetics because "all mechanisms that could lead to a familial clustering of disease should be considered,"[1] they arbitrarily fail to consider all mechanisms that could lead to higher identical versus fraternal twin concordance rates for psychiatric conditions.

The results of "twins reared-apart" (TRA) studies are sometimes put forward in support of the EEA and the twin method, based on the claim that these studies have shown that the status of being reared together in the same home does not lead to much greater identical twin resemblance than if identical twins had been reared apart in different homes. However, these studies are plagued by invalidating problems which include (1) the dubious "separation" of twins, who in many cases grew up together and had quite a bit of contact over much of their lives; (2) the similarity bias of the samples; (3) researchers' failure to publish or share raw data and life history information for the twins under study (Thomas J. Bouchard, Jr.'s Minnesota TRA study, for example[2]), and (4) the impact that the researchers' bias in favor of genetic explanations had on the interpretation of their results. The main problem with TRA studies such as Bouchard's, however, is that the investigators mistakenly compared reared-apart

1. Faraone, Tsuang, & Tsuang, 1999, p. 21.
2. Bouchard et al., 1990.

identical twins ("monozygotic twins reared-apart," or "MZAs") to *reared-together* identicals — thereby failing to control for the fact that both sets share several important environmental similarities. These include common age (birth cohort), common sex, similar appearance, and similar political, socioeconomic, and cultural environments. (In addition to MZAs, the Minnesota study used reared-apart fraternals as a comparison group, but they also share several of the environmental influences experienced by MZAs. The investigators also attempted to correct MZA correlations for age and sex effects, but their adjustments were inadequate and unclear.) Thus, behavior geneticists and TRA researchers such as Bouchard and his colleagues used the wrong control group, leading to their erroneous conclusions in favor of genetics. A scientifically acceptable study would compare the resemblance of a group consisting of MZAs reared apart from birth and unknown to each other, versus a control group consisting not of reared-together identical twins, but of *biologically unrelated pairs of strangers* sharing all of the following characteristics: they should be the same age, they should be the same sex, they should be the same ethnicity, the correlation of their rearing environment socioeconomic status should be similar to that of the MZA group, they should be similar in appearance and attractiveness, and the degree of similarity of their cultural backgrounds should be equal to that of the MZA pairs. Moreover, they should have no contact with each other until after they are evaluated and tested. After concluding such a study, we might find that the biologically-unrelated pairs correlate similarly to MZAs, which would suggest that MZA correlations are the result of environmental influences. Because no study of this type has ever been attempted, and because of the major flaws and biases in the studies that have been undertaken, we can draw no valid conclusions in support of genetic influences on psychological trait variation, or in support of the twin method's validity, from reared-apart twin studies published to date.

Twin researchers' recognition that identical twins experience more similar environments and treatments than fraternals invalidates genetic interpretations of identical-fraternal comparisons, for the exact same reason that genetic interpretations of family studies are invalid. Therefore, there is no reason to accept that the twin method measures anything other than the more similar environments of identical versus fraternal twins (plus error), and all conclusions in favor of genetic influences on psychiatric disorders derived from the twin method must be disregarded.

A Closer Look at Kendler's Defense of the EEA

Since the mid-1980s, Kenneth S. Kendler and his colleagues have published a large quantity of psychiatric genetic twin data derived from their work with the Virginia Twin Registry. Kendler has been one of the world's leading twin researchers and psychiatric geneticists for over two decades, and heads the Vir-

ginia Institute for Psychiatric and Behavioral Genetics. He is a highly honored psychiatric investigator, and is currently co-Editor of *Psychological Medicine*, as well as a member of the Editorial Boards of *Archives of General Psychiatry*, and *The American Journal of Psychiatry*. Since the 1990s he has also been involved in molecular genetic studies of psychiatric disorders.

In most of the twin studies with which Kendler has been associated, he and his colleagues concluded in favor of important genetic influences on the psychiatric disorders in question. But as Kendler well understands, these conclusions are based on the acceptance of the equal environment assumption. Kendler published a 1983 article in the *American Journal of Psychiatry*, entitled "Overview: A Current Perspective on Twin Studies of Schizophrenia," where he presented a detailed theoretical and empirical argument in defense of the EEA and the twin method. Although he has made some modifications since then, this article remains one of the few detailed defenses of the EEA ever published.[1]

Kendler began by outlining the traditional EEA definition which states that, because identical and same-sex fraternal twins "share environmental factors to approximately the same extent, differences in concordance between the two twin types must be due to the influence of genetic factors."[2] Although the critics' argument that identical twins "share more of their social environment" than do same-sex fraternal twins is true, wrote Kendler, it would be "premature" to invalidate the twin method.[3] The validity of the EEA, he argued, comes down to a pair of competing hypotheses explaining the causal relationship between identical twins' greater behavioral resemblance and their more similar social environments. The *first hypothesis*, put forward by the critics, "is that the similar phenotypes [e.g., behavior, psychiatric disorders] in monozygotic twins are caused by their similar social environment." The *second hypothesis*, put forward by Kendler and others, "is that the similar phenotypes of monozygotic twins are caused by their genetic similarity. The similar phenotypes of these twins are then responsible for creating their similar social environment."[4]

1. Kendler, 1983. Recent books promoting psychiatric and behavior genetic research, such as Jang's (2005) *The Behavior Genetics of Psychopathology*, and Carey's (2003) *Human Genetics for the Social Sciences*, provide little in the way of new arguments in support of the EEA.
2. Kendler, 1983, pp. 1413-1414.
3. Ibid., p. 1414.
4. Ibid., p. 1414.

Two "Competing Hypotheses" Discussed by Kendler

Hypothesis #1 (Critics)

The greater behavioral resemblance and greater concordance for psychopathology of identical vs. fraternal twins is caused by identicals' more similar treatment, and by the greater similarity of their social environments.

Critics' Conclusion: The twin method is unable to disentangle possible genetic and environmental influences on behavior and psychopathology.

Hypothesis #2 (Twin Researchers)

The greater behavioral resemblance and greater concordance for psychopathology of identical vs. fraternal twins is caused by identicals' greater genetic similarity. Therefore, identical twins create more similar environments for themselves than do fraternals.

Twin Researchers' Conclusion: The twin method is a valid instrument for the detection of genetic influences on behavior and psychopathology.

Although Kendler recognized that "if the first hypothesis is correct, then critics of twin studies are justified in their criticism," he argued that the second hypothesis is more plausible and that the twin method is, therefore, a valid instrument for the detection of genetic influences on psychiatric disorders. In support of the second hypothesis, Kendler cited a body of empirical "EEA-test" literature. (I review these and subsequent studies in Chapter 9, where I show that they do little to uphold the EEA.) Thus, for Kendler, the validity of the EEA and the twin method comes down to the position that the more similar physical, social, and treatment environments experienced by identical versus fraternal twins are caused by the former's greater genetic similarity.

But, wait! No further analysis is necessary to invalidate the twin method! Apparently, Kendler did not realize that the *reason* identical twins experience more similar environments than fraternal twins — be it environmental or genetic — *is completely irrelevant in assessing the validity of the EEA*. The only relevant question is *whether* — not why — identical twins experience more similar environments.[1]

Kendler attempted to bypass this obvious problem, however, writing that the evidence suggests that:

> the behavioral similarity of monozygotic versus dizygotic twins cannot be ascribed to differences in treatment of the twins by the social environment.... The behavioral similarity of monozygotic twins appears not to result from the similarity in social environment of the twins. Rather, the available evidence suggests that the similarity

1. Joseph, 2004b.

> of the social environment of monozygotic twins is the result of the behavioral similarity of the twins.[1]

Thus, Kendler acknowledged that identical twins experience more similar environments than fraternals, which he believed is caused by their greater genetic similarity. In other words, for Kendler, identical twins are treated more alike because they behave more alike (second hypothesis), as opposed to the critics' position that they behave more alike because they are treated more alike and experience more similar environments (first hypothesis). The validity of the twin method, for Kendler, rests on the direction of causality: "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."[2] Kendler approaches these two statements as if they were counterposed, when in fact they are potentially complementary because identical twins could still "create" environmental conditions or exposure leading to greater concordance for psychiatric disorders or physical disease. For example, if identical twins are more genetically predisposed to enjoy sunbathing than fraternal twins, identical twins may well show much higher concordance for skin cancer than fraternals. However, this does not mean that skin cancer is a genetically-based disease.

Another problem with Kendler's argument, which I have called the "twins create their environment theory,"[3] is that it ascribes to parents — but not to children — the ability to alter behavior on the basis of the actions of other family members.[4] Moreover, Kendler's argument is circular because the evidence for twins' behavioral similarity being caused by genetics is implicitly derived from the results of previous twin studies.

Finally, Kendler maintained that "the behavioral similarity of monozygotic versus dizygotic twins cannot be ascribed to differences in treatment of the twins by the social environment," even though his second hypothesis allows that more similar treatment might create more similar twins, but that the twin method remains valid because twins supposedly "are responsible for creating their own environment." In truth, there is little difference between the "first" and "second" hypotheses, because *both* imply that greater identical versus fraternal concordance is related to the more similar environments experienced by identical twins. Moreover, the second hypothesis renders the "trait-relevant" argument irrelevant in the sense that, even if critics were able to demonstrate that identical twins experience more similar trait-relevant environments than fraternals, Kendler and his co-thinkers would still argue for the twin method's validity on the basis of identical twins having "created" their more similar environments.

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1. Kendler, 1983, p. 1416.
2. Kendler, 1987, p. 706. Emphasis in original.
3. Joseph, 1998.
4. Joseph, 1998, 2004b.

Another example of how genetically oriented researchers use "the twins create their environment" argument in support of the EEA is found in the introductory chapter of the 2002 edition of *The Genetic Basis of Common Diseases*. According to King, Rotter, and Motulsky, the authors of the chapter and editors of the volume, "A higher concordance rate in MZ than in DZ twin pairs (especially like-sexed DZ pairs) indicates that a significant part of the familial aggregation [of a disease] is due to genetic factors...." However,

> A qualification is that it can sometimes be difficult to disentangle heredity from environment, because MZ twins, likely because of their genetic identity, tend to select similar environments. Coronary heart disease, celiac disease, inflammatory bowel disease, diabetes, and schizophrenia are examples of disorders in which the concordance rate is higher in MZ than in DZ twin pairs.[1]

There are at least three problems with this argument:

1. The argument is circular, because the assumption that identical twins select similar environments due to their more similar genetic makeup is based largely, though implicitly, on the results from previous twin studies of behavior and personality.

2. As mentioned above, the idea that identical twins are able to select similar environments ascribes to parents, but not to twins, the ability to change and adjust their behavior to the needs of others.

3. Even if identical twins' more similar environments are due to their greater genetic identity, higher identical versus fraternal concordance for the diseases, syndromes, conditions, and putative diseases listed by King et al. could still be completely caused by environmental factors (see the example of skin cancer I mentioned earlier).

Although King and colleagues devoted only a few sentences to the validity of the equal environment assumption, the next 1,000+ pages of their edited volume cited twin studies as the main evidence supporting a genetic basis for the medical and psychiatric disorders in question.

Let's return to Kendler's position that identical twins' greater behavioral resemblance "cannot be ascribed to differences in treatment of the twins by the social environment." Another problem that Kendler overlooked is that his theory must generalize to mean that no one's behavior — neither twins' nor non-twins — is influenced by their social environment. The reason, of course, is that each twin is an individual human being receiving treatment by the social environment. But, according to the logic of Kendler's EEA theory, the social environment experienced by each *individual* twin cannot alter his or her behavior.

The position that social and cultural influences *do* influence human behavior is so obvious that I will refrain from burdening myself and readers with a discussion of research supporting it. It's far simpler to cite some concrete examples that don't even address the importance of family (psychodynamic)

1. King et al., 2002a, p. 12.

rearing environment, since there are untold behavior-modifying influences in modern society. For example, what is a *law* if not a means of controlling human behavior through a system of rewards and punishment? Whether or not you break a particular law, such as speeding on an open highway or stealing an MP3 player, is at least partly influenced by whether you think it is worth the chance of receiving an expensive traffic ticket or being arrested for shoplifting. And what about income tax? Do people file honest returns solely because of moral virtue or patriotism, or do they also fear an audit and possible punishment if they don't? A small fraction of the behavior-influencing agents in society might include abortion laws, apartment rules, athletic scholarships, being a crime victim, billboards, billy clubs, birth control pills, bubble gum flavored toothpaste, bullies, cell phones, child labor laws, condoms, corporal punishment, cosmetic surgery, coupons, court marshals, culture, cruise missiles, diplomatic immunity, ethics codes, fashion, fences, freedom of speech, God, guns, "gunboat diplomacy," health insurance, heaven, hunger, income, jaywalking tickets, military dictators, muggers, mutual fund performance, newspaper headlines, oil prices, paychecks, peer pressure, penicillin, police, promotional opportunities, racism, radio, report cards, security guards, sexism, social security, spies, stock market crashes, surveillance cameras, strikes, strikebreakers, teachers, television, the devil, the Internet, the price of gasoline, the size of the lottery jackpot, the Ten Commandments, timecards, "time outs," traffic lights, traffic signs, trends, warnings by the Surgeon General, word of mouth, and zero percent financing.

However, for Kendler's theory to hold, we must deny that these factors influence human behavior. We must believe that humans are hardwired at birth to behave in a predetermined way, uninfluenced by the social environment and by society's system of rewards and punishments.

When Kendler says that critics must demonstrate that twins' environments are "trait-relevant," what he is really saying is that *everyone's* environment is trait-*ir*relevant.

Let's look to the advertising industry to illustrate another example of this untenable position. Advertising agencies and their clients understand that messages created in certain ways for particular audiences can indeed "influence behavior." If Coca-Cola pays $2 million for a thirty-second Super Bowl television commercial, they certainly expect that thousands or millions of people will change their cola buying *behavior*. Furthermore, modern advertising not only targets behavior but seeks to change the way people feel about themselves in society. It's not enough to tout the effectiveness of the latest dandruff shampoo; one also needs to make people feel embarrassed, if not humiliated, that they have dandruff. Self evidently, if the environment does not influence twins' and others' behavior and psychology, as Kendler implies, then corporations would not spend untold billions of dollars each year on advertising. Twin researchers and their EEA test studies (see Chapter 9) resemble the proverbial "blind men" who

cannot see the massive "elephant" of environmental influences on the behavior of both twins and non-twins alike.

Kendler went on to assert that:

> Differential treatment of twins by their social environment does not appear to be responsible for the greater similarity of monozygotic versus same-sex dizygotic twins for such characteristics as intelligence, personality, and language and perceptual skills. Therefore, it seems unlikely that such differential treatment could be responsible for the greater concordance for schizophrenia in monozygotic versus same-sex dizygotic twins.[1]

But what if we keep non-twins in mind and rephrase this statement to read: "Differential treatment of people by their social environment does not appear to be responsible for differences in characteristics such as intelligence, personality, and language and perceptual skills. Therefore, it seems unlikely that differences in treatment could be responsible for schizophrenia." The logic in both statements is the same, but the first statement's folly becomes obvious when we strip away the confusion created by the special situation of twins. Moreover, although many genetically-oriented commentators would allow for environmental effects playing some role in schizophrenia when combined with a genetic predisposition, the logic of Kendler's argument implies that the environment has no influence on schizophrenia. However, by 2005 Kendler would recognize that "a large body of descriptive literature shows convincingly that cultural processes affect psychiatric illness,"[2] and that the "impact of genetic risk for psychiatric disorders or drug use can be modified by the rearing environment...by stressful life experiences...and exposure to cultural forces."[3]

According to Kendler's EEA theory, identical twins experience similar environments because they share an identical genetic makeup. However, if identical twins have the same genetic makeup and experience necessarily equal within-pair environments, and psychopathology is the result of genetic plus environmental influences (or of their interaction), identical twin pairs should approach 100% concordance for psychiatric disorders. Moreover, they should be virtual "personality clones" of each other. Yet, as most people are aware, in many cases identical twins differ substantially in values, interests, tastes, psychiatric status, and so on. Indeed, we will see in Chapter 6 that the pooled identical twin schizophrenia concordance rate from the more methodologically sound studies is only about 20-22%.[4]

Kendler missed another essential point in his assessment of the relationship between twins' treatment and schizophrenia. It is not simply a matter of parents and others treating a pair of twins more similarly in a potentially psychologically

1. Kendler, 1983, p. 1416.
2. Kendler, 2005c, p. 436.
3. Ibid., p. 437.
4. See also Joseph, 2004b.

harmful manner, but that identical twins are socialized to experience a much stronger emotional bond with each other than are fraternal twins.[1] From the environmental perspective, the twin relationship is a more important aspect of concordance than identical twins merely being treated in a more similar etiologically-relevant manner than fraternals. Thus, twin studies of psychosis may have revealed little more than identical twins' greater propensity to experience folie à deux (shared psychotic disorder) than fraternals (see Chapter 6).[2]

In previous publications I have pointed to a 1967 survey by Norwegian schizophrenia twin researcher Einar Kringlen, who, in addition to computing concordance rates for schizophrenia, assessed his identical and same-sex fraternal twin pairs for emotional closeness and environmental similarity. The results of this survey, which to my knowledge have never been mentioned by anyone other than Kringlen and myself, are presented in Table. 1.1.

Table 1.1

IDENTICAL VS. SAME-SEX FRATERNAL TWIN RELATIONSHIP AND TREATMENT: KRINGLEN'S 1967 TWIN STUDY

	Identical – 75 Pairs				Same-Sex Fraternal – 42 Pairs				
	F	M	Total	%	F	M	Total	%	p*
Identity confusion in childhood									
Yes	37	31	68	90	3	1	4	10%	<.0000000001
No	0	7	7	9%	17	21	38	90%	
Mistaken for each other by									
Parents and/or sibs	10	6	16	21%	0	0	0	0%	.0004
Teachers	16	10	26	35%	0	0	0	0%	.000001
Strangers	11	15	26	35%	3	1	4	10%	.002
No one	0	7	7	9%	17	21	38	90%	<.000000001
Considered as									
Alike as two drops	31	26	57	76%	0	0	0	0%	<.000000001
More alike than	3	5	8	11%	2	1	3	7%	
Alike as sibs	2	5	7	9%	18	21	39	93%	<.000000001
Uncertain	1	2	3	4%	0	0	0	0%	
Dressed									
Alike	3	28	61	81%	10	18	28	67%	

1. Ainslie, 1985.
2. Jackson, 1960; Joseph, 2004b.

Not alike	2	2	4	5%	4	1	5	12%	
Uncertain	2	8	10	13%	6	3	9	21%	
Brought up "as a unit"									
Similar	2	25	54	72%	3	5	8	19%	.00000003
Dissimilar	2	4	6	8%	11	6	17	40%	
Uncertain	6	9	15	20	6	11	17	40%	
Inseparable as children									
To an extreme	31	24	55	73%	2	6	8	19%	.0000002
Partly inseparable	2	3	5	7%	6	7	13	31%	
Not inseparable	2	7	9	12%	12	8	20	48%	
Uncertain	2	4	6	8%	0	1	1	2%	
Inseparable as adults									
To an extreme	2	12	14	18%	0	0	0	0%	.001
Partly inseparable	2	11	35	47	4	6	10	24%	
Not inseparable	7	12	19	25	14	13	27	64%	
Uncertain	4	3	7	9%	2	2	4	10%	
Mutual friends as children									
Mutual friends	2	23	49	65	7	12	19	45%	.022
Partly	5	3	8	11%	8	5	13	31%	
Different friends	2	5	7	9%	5	4	9	22%	
Uncertain	3	7	10	13%	0	1	1	2%	
GLOBAL EVALUATION OF TWIN CLOSENESS									
Extremely strong	2	21	49	65	2	5	7	17%	.0000003
Moderately strong	5	5	10	13%	8	8	16	38%	
As siblings	1	6	7	9%	7	7	14	33%	.002
Uncertain	3	6	9	12%	3	2	5	12%	

*Source: Adapted from Kringlen, 1967, p. 115. * Probability value determined by Fisher's Exact Test, one-tailed—identical total vs. fraternal total. F = Female, M = Male. For all pairs, one or both were diagnosed with a psychotic disorder.*

As seen in Table 1.1, identical twins were much more likely than fraternals to have been "inseparable" as children and as adults, to have had mutual friends as children, and to have been mistaken for each other. More important are the assessments of twin closeness and "identity confusion." Regarding the latter, 90% of identical twins — but only 10% of fraternal twins — experienced "identity confusion" as children. Moreover, identicals were more often mistaken for each other, considered as "alike as two drops of water," and "brought up as a

unit." The final "evaluation of twin closeness" showed that 65% of identical twins had an "extremely strong" level of closeness, which was true for only 19% of fraternals.

Kendler and other defenders of the EEA simply ignore these results. They choose instead to focus on confirmatory data which they cite in support of the EEA and the twin method (see Chapter 9). Still, results such as Kringlen's support the validity of objections made by critics since the twin method's inception in the mid-1920s. Moreover, Kendler's EEA theory stands in direct contrast to the views of most schizophrenia twin researchers, who recognized that at least a portion of identical-fraternal concordance differences can be attributed to environmental factors.[1] Even a psychologist and behavior geneticist as prominent as Michael Rutter has acknowledged that "there will be violations of the Equal Environments Assumption (EEA), which is fundamental to the twin design...The violation arises because part of the difference between MZ and DZ pairs will stem from environmental effects..."[2]

It is widely understood both inside and outside psychiatry that identical twins experience much more similar environments than do fraternals. Moreover, qualifications regarding trait-relevance, or twins "creating their own environments," do nothing to alter the conclusion that the twin method is, like a family study, unable to disentangle possible genetic and environmental influences on psychiatric disorders. Although popularizers of genetic research such as William Wright, in his 1998 book *Born That Way*,[3] claimed that Kendler "refuted" the critics' objections in his 1983 "Overview" article, Kendler's theoretical defense of the EEA is, as we have seen, rather easy to refute.

The Twin Method as Pseudoscience

Because it rests on at least one clearly false theoretical assumption, one could argue that the twin method can be understood within the framework others have created to separate science from pseudoscience.

In the opening chapter of their 2003 edited volume, *Science and Pseudoscience in Clinical Psychology*, psychologists Scott Lilienfeld, Steven Lynn, and Jeffrey Lohr outlined ten "warning signs" of pseudoscience. "The more such warning signs a discipline exhibits," they argued, "the more it begins to cross the murky dividing line separating science from pseudoscience."[4] The ten warning signs outlined by Lilienfeld and his colleagues (who did not address the twin method, or list it as a

1. Joseph, 2004b, Chapter 6.
2. Rutter, 2003, pp. 935-936.
3. Wright, 1998.
4. Lilienfeld et al., 2003, p. 5. All subsequent quotations from these authors are taken from Lilienfeld et al., 2003, pp. 3-10.

pseudoscience) are "an overuse of *ad hoc* hypotheses designed to immunize claims from falsification," "absence of self-correction," "evasion of peer review," "emphasis on confirmation rather than refutation," "reversed burden of proof," "absence of connectivity," "over reliance on testimonial and anecdotal evidence," "use of obscurantist language," "absence of boundary conditions," and "the mantra of holism." Let's take a look at how the twin method and the arguments of its defenders fit into Lilienfeld and colleagues' pseudoscience framework.

An overuse of ad hoc *hypotheses designed to immunize claims from falsification.* An *ad hoc* hypothesis has been defined as "any hypothesis or hypothetical explanation developed to explain a particular set of data that does not fit into an existing theoretical framework. An *ad hoc* hypothesis is one developed after the data have been collected."[1] According to Lilienfeld et al., "The repeated invocation of *ad hoc* hypotheses to explain away negative findings is a common tactic among proponents of pseudoscientific claims. Moreover, in most pseudosciences, *ad hoc* hypotheses are simply 'pasted on' to plug holes in the theory in question."

The twin method is not as vulnerable to negative findings as it is to alternative explanations of its results. As we have seen, in the face of overwhelming evidence that identical twins experience much more similar environments than fraternals, Kendler and others, instead of abandoning the twin method, attempted to "plug holes" created by obvious environmental confounds with ad hoc hypotheses such as the "twins create their environment theory" and the trait-relevant stipulation. Clearly, Kendler and others "pasted on" these internally contradictory theories in order to salvage psychiatric geneticists' main research method.

Absence of self-correction. Pseudosciences tend to avoid eliminating errors from their methods and theories. The only significant changes in psychiatric twin research (apart from more complicated statistical formulas) occurred after psychiatrist Don Jackson, who was not a twin researcher, published a cogent critique of schizophrenia twin research in 1960.[2] Thus, twin researchers made corrections only after environmental confounds in twin research had been exposed by a well-respected critic from outside of their discipline.

Evasion of peer review. There is no reason to believe that twin researchers of the past few decades have evaded the peer review process. This has not been necessary, given the largely uncritical acceptance of twin research in mainstream psychiatry and psychology. Thus, twin researchers submitting their findings to psychiatry and psychology journals do not fear that their work will be rejected because their findings are explainable on environmental grounds. Twin studies are cited frequently in support of the claim that mental disorders are biologically based, a position vitally important to psychiatry and to its partners in the psychopharmaceutical industry. It therefore is unfortunate that so many twin

1. Reber, 1985, p. 12.
2. Jackson, 1960. For an analysis of Jackson's argument, see Joseph, 2001b, 2004b.

studies could pass the psychiatric journal peer review process without being rejected on the grounds that their authors' conclusions are based on an unsupported, yet critical, theoretical assumption.

Emphasis on confirmation rather than refutation. This is a hallmark of Kendler's defense of the twin method and the EEA. In Chapter 9, we will see how Kendler has focused mainly on research claiming to uphold the EEA, while ignoring "real world" examples running counter to his argument. "Pseudoscientists," wrote Lilienfeld and colleagues, "tend to seek only confirming evidence for their claims.... a determined advocate can find at least some supportive evidence for virtually any claim..." Moreover, "most pseudosciences manage to reinterpret negative or anomalous findings as corroborations of their claims." An example is found in a 1998 adoption study by prominent behavior geneticists Robert Plomin and colleagues, who, despite having found no personality-test score correlation between birth parents and their 245 adopted-away biological offspring, concluded that a "nonadditive genetic influence, which can be detected by twin studies but not by adoption studies, is a likely culprit."[1]

Reversed burden of proof. We have seen that Kendler's defense of the EEA and the twin method implies that the burden of proof for showing that identical and fraternal environments differ on trait-relevant dimensions falls not on twin researchers themselves but on critics. As Lilienfeld et al. point out, however, "a basic tenet of science is that the burden of proof always falls squarely on the claimant, not the critic...Consequently, it is up to the proponents of these techniques to demonstrate that they work, not up to the critics of these techniques to demonstrate the converse." But since the twin method would indeed be relegated to the museum of pseudosciences if *twin researchers* bore the burden of proof for showing that identical and fraternal twins experience equal (trait-relevant) environments, they place this burden on critics.

In his 1983 "Overview" article, Kendler wrote,

> For a familial-environmental bias in twin studies of schizophrenia to be a tenable hypothesis, nongenetic familial factors must be shown to be of major etiologic importance in the disorder.[2]

When he wrote that familial-environmental factors "must be shown," Kendler clearly meant that these factors must be shown *by critics*. Kendler does this even though (1) shifting the burden of proof to critics runs counter to a "basic tenet of science," (2) family environment is a major factor in environmental explanations of schizophrenia and other psychiatric disorders, and (3) he takes the opposite position when he discusses potential environmental confounds in psychiatric *family* studies. In this case, Kendler does not require critics

1. Plomin et al., 1998, p. 211.
2. Kendler, 1983, p. 1416.

to show that "familial factors must be shown to be of major etiologic importance in the disorder" to invalidate conclusions in favor of genetics.

According to twin researcher Michael Lyons and his colleagues, "it would seem that the burden of proof rests with critics of the twin method to demonstrate that 'trait-relevant' environmental factors are more similar for identical than same-sex fraternal twins."[1] Although they wrote, without any justification, that the burden of proof "seems" to fall on critics, a stronger case can be made that twin researchers bear this burden. After all, aren't *they* responsible for demonstrating the validity of their research method?

Similarly, according to twin researcher Thomas J. Bouchard, Jr.,

> It is certainly true that MZ twins experience more similar environments than do DZ twins, but it is also true, if perhaps surprising, that no one has been able to show that such imposed similarities in treatment are trait-relevant.[2]

Elsewhere Bouchard argued, in response to criticisms of the EEA by psychologist Louise Hoffman,

> The equal environment assumption 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 [emphasis added].[3]

And in a response to my criticism of the EEA, Faraone and Biederman wrote in 2000,

> The second claim made by Joseph is that twin studies of ADHD are flawed by the equal environment assumption, which holds that the trait-relevant environments of identical and fraternal twins are the same. He finds this assumption untenable for two reasons. First, several studies have shown that, compared with fraternal twins, identical twins are treated more alike, spend more time together, have more common friends, and experience greater levels of identity confusion. Second, he *infers* from these data that identical twins are more likely to be similarly exposed to "trait-relevant" environmental factors. Notably, Joseph presents no data to support his inference. Thus, readers should view it as a hypothesis to be tested rather than a conclusion to be accepted [emphasis in original].[4]

Thus, in addition to reversing the burden of proof, twin researchers commit the "*ad ignorantium* fallacy," which Lilienfeld et al. described as "the mistake of assuming that a claim is likely to be correct merely because there is no compelling evidence against it."

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1. Lyons et al., 1991, p. 126.
2. Bouchard, 1997, p. 134.
3. Bouchard, 1993, p. 33.
4. Faraone & Biederman, 2000, p. 570.

Absence of connectivity. By this it is meant that pseudoscience proponents tend to disconnect their field from other disciplines. Kendler's citations in defense of the EEA come mainly from the fields of psychology and psychiatry, and even then only from genetically-oriented researchers in these fields. Findings from neuroscience, criminology, sociology, anthropology, and other areas of psychiatry and psychology demonstrating the importance of the environment on behavioral differences, hardly exist for Kendler (at least until recently) and other leading proponents of the twin method. This is, according to Lilienfeld, consistent with pseudoscientific practices, which "often purport to create entirely new paradigms out of whole cloth rather than to build on extant paradigms. In doing so, they often neglect well-established scientific principles or hard-won scientific knowledge."

Over reliance on testimonial and anecdotal evidence. Although the twin method does not rely on anecdotal evidence, it was used to a great extent in Bouchard and colleagues' reports of allegedly separated twin pairs in their famous Minnesota reared-apart twin studies.[1] In turn, the claimed similarity of these "reared-apart" pairs strengthened the twin method, since twin researchers can and do argue that being reared together does not cause twins to resemble each other much more than if they had been reared apart. In 1987, critic Val Dusek described eight years of published Minnesota anecdotes in the absence of peer reviewed studies as "bewitching science," and compared them "to the sort of evidence often offered as proof for astrology or parapsychology such as extrasensory perception (E.S.P.)."[2]

Use of obscurantist language. This has been a common feature in twin research published since the early 1990s. In many cases, concordance rates or correlations are downplayed in favor of complex path analysis and model fitting diagrams, once described by psychologist Richard Lerner as "dazzling statistical pyrotechnics," accompanied by language difficult to understand by people not directly involved in twin research.[3] For example, in a 1992 twin study of phobias in women, Kendler and his colleagues wrote, in reference to their statistical formulations,

> In univariate analysis, information regarding the causes of variation is obtained by comparing the resemblance of MZ and DZ twin pairs for a single variable. In the multivariate case, the correlation between two or more variables is the primary unit of analysis. By comparing the cross-twin, cross-variable correlation in MZ and DZ twins, and contrasting that to the cross-twin within-variable and within-twin cross-variable correlations, the covariation of two or more variables can be partitioned into its genetic and environmental components.[4]

1. See Joseph, 2004b, Chapter 4.
2. Dusek, 1987, p. 21.
3. Lerner, 1995, p. 148.
4. Kendler et al., 1992b, p. 275.

Even if one could decipher this not atypical passage, all statistical formulations, path analyses, model fitting, multiple regression coefficients, and so on depend on the validity of the EEA as a prerequisite for concluding in favor of genetics. The expression "Garbage in, garbage out" could be modified for twin research to read, "False assumptions in, false conclusions out." Twin research publications turn readers' attention away from untenable assumptions and in the direction of impressive looking scientific language and diagrams which, in the words of Lilienfeld and colleagues, "may be convincing to individuals unfamiliar with the scientific underpinnings of the claims in question, and may therefore lend these claims an unwarranted imprimatur of scientific legitimacy."

Absence of boundary conditions. "Most well-supported scientific theories," wrote Lilienfeld et al., "possess boundary conditions, that is, well-articulated limits under which predicted phenomena do and do not apply." This relates to twin research in cases where the investigators insist that their findings can be generalized to the non-twin population. It is even more problematic in adoption research, where little attention is given to whether results can be generalized to the non-adoptee population (see Chapter 6). This is not to say that the results of twin and adoption studies (assuming that their assumptions are valid) are never generalizable, but that researchers, in their haste to conclude in favor of universal genetic principles, pay little attention to this crucial question.

"The mantra of holism."[1] According to Lilienfeld and colleagues, pseudoscience proponents "typically maintain that scientific claims can be evaluated only within the context of broader claims and therefore cannot be judged in isolation." An example is found in psychiatric genetic investigators Faraone, Tsuang, and Tsuang's 1999 *Genetics of Mental Disorders*. Following a discussion of family, twin, and adoption studies, and the limitations of each, they wrote,

> Although some methodological problems limit the effectiveness of twin studies, there is no conclusive evidence that these limitations substantially bias twin study results. Instead, there is a consensus among psychiatric geneticists that the twin method provides an informative source of converging evidence in determining the importance of genetic factors in psychiatric disorders.[2]

Moreover,

> Because each method has its limitations, we cannot rely on either a single study or class of studies to draw conclusions about the effects of genes and environment on mental illness. Instead, from an examination of many studies we seek a pattern of converging evidence that consistently confirms genetic and/or environmental hypotheses about the familial transmission of the disorder.[3]

While Faraone et al. recognized that, due to its "limitations," we "cannot rely" on twin studies to provide evidence in support of genetics, they put these

1. This phrase was coined by John Ruscio (2001).
2. Faraone, Tsuang, & Tsuang, 1999, p. 39.
3. Ibid., p. 45.

studies forward in support of genetics in the context of the supposed "converging evidence" from *other* types of studies. In a similar vein, twin researcher Irving Gottesman (whose work is the topic of Chapter 6) wrote that although schizophrenia family, twin, and adoption studies each "contribute to the genetic argument.... No one method alone yields conclusive proof or disproof."[1] Gottesman's argument is noteworthy in the sense that, if the EEA were truly sound, the twin method would indeed provide "conclusive proof" in favor of genetics.

In 2000, Faraone and Biederman used the "holism" argument in response to my criticism of ADHD twin and adoption studies, claiming that genetic theories of ADHD make better "predictions" and are more "parsimonious."[2] As we have seen, in defense of the EEA they placed the burden of proof on me by invoking the trait-relevant condition for ADHD twin studies.

In their 1998 adoption study of personality, Plomin and colleagues utilized the "holism" argument to explain away results consistent with environmental explanations of personality differences. Rather than highlight their study's failure to find a significant correlation between personality and genetic relationship, the investigators discussed their results in the context of previous twin studies of personality: "The most obvious implication of these results is that other family and adoption studies are needed to triangulate with twin studies on the estimation of genetic influence for personality as assessed by self-report personality questionnaires."[3]

* * *

The framework provided by Lilienfeld and others suggests that the twin method may belong in the pseudoscience category. Indeed, twin researchers' defense of the EEA has done little if anything to dispel the idea that the greater environmental similarity experienced by identical twins explains their greater concordance for psychopathology when compared to same-sex fraternal twins.

The "Heritability" Fallacy

The *heritability* concept is widely used in reference to genetic influences on psychiatric disorders and psychological trait variation. However, heritability estimates falsely claim to approximate "how much" genetic influence there is. As dissident behavior geneticist Jerry Hirsch has pointed out for many years, a numerical heritability estimate (coefficient) is not a "nature/nurture ratio" of the relative contributions of genes and environment.[4]

1. Gottesman, 1991, p. 93.
2. For my original article, Faraone and Biederman's response, and my response to them, see Faraone & Biederman, 2000; Joseph, 2000c, 2000e.
3. Plomin et al., 1998, p. 215.
4. Hirsch, 1997, 2004; McGuire & Hirsch, 1977; Joseph, 2004b.

Contrary to popular belief, whether heritability is 10% or 90% says nothing about the potential efficacy of a particular environmental intervention, nor does a heritability estimate greater than 50% imply that genes are more important than the environment. An example is phenylketonuria (PKU), a genetic disorder of metabolism which, without a specific environmental intervention, causes mental retardation. Although the population variance for PKU susceptibility is completely explained by genetic factors (heritability = 1.0, or 100%), the administration of a low phenylalanine diet to the at-risk infant during a critical period prevents the disorder from appearing. PKU is an excellent example of biologist Richard Lewontin's observation that a "trait can have a heritability of 1.0 in a population at some time, yet could be completely altered in the future by a simple environmental change."[1]

Approaching this question from a different angle, although the human trait of having two arms is inherited, the heritability of humans having two arms is *zero*. The reason is that the heritability statistic describes *variation* in a population attributable to genes. Because virtually everyone is born with two arms, and because people with one arm become that way because of an environmental occurrence, 100% of the "armedness" variation in a population is caused by the environment, and 0% of the variation is caused by genes. At the same time, of course, having two arms is a genetically programmed human trait. Thus, a trait could be 100% *inherited*, yet have a *heritability* of 0%. Hirsch has reminded us that although "heritable" and "inherited" are very different concepts, many people wrongly believe them to be synonymous because they sound alike.[2] Unfortunately, the genetic literature does little to help people avoid such confusion.

A heritability estimate, which is applicable only in a specific population, in a specific environment, and at a specific point in time, was developed in agriculture as a means of predicting the results of a selective breeding program for economically desirable traits.[3] Unfortunately, the invalid extension of the heritability statistic from a breeding predictor to a quantification of the genetic contribution to psychiatric disorders and psychological trait variation has led to a great deal of misunderstanding about the role of genetic influences on these traits and disorders. Moreover, heritability estimates are based on rarely-met assumptions about humans. According to Hirsch,

> Heritability estimation assumes both random mating in an equilibrium population (including therein the equally likely occurrence of every culturally tabooed form of incest) and the absence of either correlation or interaction between heredity and environment. In fact, when one or more of those assumptions are violated, that

1. Lewontin, 1974, p. 400.
2. Hirsch, 1997.
3. Lush, 1949. According to Kendler, "contrary to common usage, 'heritability' does not designate a characteristic of a disorder or a trait but only of a disorder or trait in a specific population at a specific time" (Kendler, 2005b, p. 5).

> is, random mating in an equilibrium population, correlation or interaction, heritability is undefined.[1]

Heritability estimates are dubious for the additional reason that they are derived from twin and adoptions studies, which are subject to the invalidating environmental confounds and biases I discuss in this book.[2]

Thus, while it is theoretically possible that genetic factors underlie psychiatric disorders, it is inappropriate and misleading to use the heritability statistic to estimate the magnitude of this possible component. Behavior geneticist Richard Rende has written that the heritability statistic serves as "a useful statistical indicator to some, a rather meaningless index to others, and a potentially harmful, biased, and even blatantly incorrect calculation to the harshest critics."[3] Clearly, my views are similar to other "harsh critics" of the heritability concept.

While other terms are preferable, it may be acceptable to use the word "heritable" to indicate that a disorder is influenced by genetics. However, as the example of PKU shows, a heritability percentage or estimate says nothing about the ability to treat or prevent a disorder, or about the magnitude of genetic influences, and its use should therefore be discontinued in psychiatry and psychology.

* * *

Psychiatric geneticists and others view attention-deficit hyperactivity disorder, the most frequently diagnosed psychiatric condition among school-age children, as being strongly influenced by genetic factors. In the following chapter I examine the evidence they put forward in support of this claim.

1. Hirsch, 2004, p. 137.
2. Joseph, 2004b.
3. Rende, 2004, p. 112.