Chapter-by-Chapter Summary of

THE GENE ILLUSION:
GENETIC RESEARCH in PSYCHIATRY and PSYCHOLOGY UNDER the MICROSCOPE

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Algora Publishing, 2004
2003 United Kingdom Edition by PCCS Books

Available at Amazon.com

THIS SUMMARY REVISED AND UPDATED JANUARY, 2011

CHAPTER ONE | Introduction

In 1925 psychiatrist Abraham Myerson, who was writing at the height of the eugenics[1] movement's influence, observed, "We often hear of hereditary talents, hereditary vices, and hereditary virtues, but whoever will critically examine the evidence will find that we have no proof of their existence." [2]

The evidence of Myerson's era consisted of family pedigrees, preconceived notions, and prejudice. Today it consists mainly of family studies, adoption studies, studies of twins reared-

[1] *Eugenics* is a doctrine which holds that humans can be "improved" by selective breeding to eradicate "undesirable" traits in society. Eugenicists argue that many social problems and psychiatric disorders are caused by inherited genetic traits in people, which can be bred out of the population for the benefit of future generations. Many German eugenicists of the first half of the 20th century preferred the term *racial hygiene* to eugenics.

[2] Myerson, A. (1925, p. 23). *The Inheritance of Mental Diseases.* Baltimore: Williams & Wilkins.

together, studies of twins reared-apart, and molecular genetic research. Although it is widely believed that the results of these investigations converge on the importance of genetics, Joseph argues convincingly in *The Gene Illusion* that conclusions in favor of genetics based on family, twin, and adoption studies are faulty. Moreover, molecular genetic research has failed to identify genes for the major psychiatric disorders, or genes that influence variation in normal psychological traits such as IQ and personality.[3] Thus, Myerson's 1925 observation is more relevant to today's evidence than is commonly believed.

Although the media frequently reports that specific genes have been linked to psychiatric disorders and psychological traits, in almost every case subsequent research has failed to substantiate these findings.

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It has been observed that "the knowledge that certain diseases run in families is thousands of years old."[4] Today, most genetic researchers understand that a trait or condition "running in the family" can be explained by any number of environmental factors related to the physical, social, and psychological environments shared by family members. Thus, they recognize that family studies are unable to disentangle the potential influences of genes and environment. For example, a pair of prominent genetic researchers in psychiatry acknowledged that family studies can provide only "the initial hint that a disorder might have a genetic component," because "disorders can 'run in families' for nongenetic reasons such as shared environmental adversity, viral transmission, and social learning."[5] And behavior geneticist Robert Plomin and his colleagues wrote, "many behaviors 'run in families,' but family resemblance can be due to either nature or nurture."[6] They concluded, "Family studies by themselves cannot disentangle genetic and environmental influences."[7]

Some researchers, however, in addition to the popular media, continue to erroneously cite family data in support of genetics. As one of countless such examples, the author of a 2010 editorial in the *American Journal of Psychiatry* wrote, "We know that schizophrenia has genetic

[3] See Akil, H., Brenner, S., Kandel, E., Kendler, K. S., King, M., Scolnick, E., Watson, J. D., Zoghbi, H. Y. (2010). The future of psychiatric research: Genomes and neural circuits. *Science, 327,* 1580-1581; Jablensky, A. (2010). The diagnostic concept of schizophrenia: Its history, evolution, and future prospects. *Dialogues in Clinical Neuroscience, 12,* 271-287; Joseph, J. & Ratner, C. (2010). The fruitless search for genes in psychiatry and psychology: Time to re-examine a paradigm? *Council for Responsible Genetics:* "*Gene Myths Project.*"Available online at http://www.councilforresponsiblegenetics.org/pageDocuments/1NX6VC0254.pdf ;
Latham, J., & Wilson, A. (2010). The great DNA data deficit: Are genes for disease a mirage? *The Bioscience Research Project* (retrieved online 12/18/10 from http://www.biosciencesource.org/commentaries/article.php?id=46); Plomin, R., DeFries, J. C., McClearn, G. E., & McGuffin, P. (2008, p. 70). *Behavioral Genetics* (4th ed.). New York: Worth Publishers.

[4] Alper, J. (2002, p. 17). Genetic Complexity in Human Disease and Behavior. In J. Alper et al. (Eds.), *The Double-Edged Helix: Social Implications of Genetics in a Diverse Society* (pp. 17-38). Baltimore: The Johns Hopkins University Press.

[5] Faraone, S. V., & Tsuang, M. T. (1995). Methods in Psychiatric Genetics. In M. Tsuang, M. Tohen, & G. Zahner (Eds.), *Textbook in Psychiatric Epidemiology* (pp. 81-134). New York: Wiley-Liss.

[6] Plomin et al. (2008, p. 70).

[7] Plomin et al. (2008, p. 151).

causes, since the most significant risk factor is having a first-degree relative with schizophrenia."[8]

Because family studies are unable to disentangle the roles of heredity and environment, genetic researchers have turned to twin and adoption studies in an attempt to clearly separate (disentangle) these potential influences. As Joseph attempts to show, however, this theorized separation is far more difficult to accomplish than is currently believed.

Some ways that twins have been used for research purposes include:

- The twin method (twins reared-together)
- Studies of twins reared-apart
- The co-twin control method
- Genetic studies of the offspring of discordant monozygotic twin pairs
- Studies of discordant monozygotic twin pairs (investigating environmental differences)

The main tool of behavior genetics[9] and psychiatric genetics[10] is the "classical twin method," more commonly known as "the twin method." The twin method compares the resemblance of reared-together identical twin pairs (also known as monozygotic, or MZ), who share 100% genetic similarity, versus the resemblance of reared-together same-sex fraternal twin pairs (also known as dizygotic, or DZ), who average a 50% genetic similarity. (Twin resemblance is usually measured with concordance[11] rates or correlations.) Based on the assumption that the childhood and adult environments of both types of twins are comparable, known as the "equal environment assumption" or "EEA," twin researchers attribute to genetic factors the usual finding of a significantly greater resemblance among identical versus same-sex fraternal twins.

[8] Gilmore, J. H. (2010, p. 8). Understanding what causes schizophrenia: A developmental perspective. *American Journal of Psychiatry, 167,* 8-10.

[9] *Behavior genetics* is a discipline, rooted in the field of psychology, that uses family, twin, and adoption studies to assess possible genetic influences on "continuously distributed" psychological traits such as personality and I.Q., and also on psychiatric disorders. In other areas of behavior genetics, researchers work primarily with non-human animals.

[10] *Psychiatric genetics* is a discipline founded by Ernst Rüdin and his German colleagues in the early part of the 20th century. 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," by sterilization or other means. After the Nazi seizure of power in 1933, the leaders of Rüdin's "Munich School" of psychiatric genetics helped conceive and popularize Hitler's program of forcibly sterilizing "eugenically undesirable" people. Contemporary psychiatric geneticists investigate the causes of mental disorders in order to better treat and prevent them. Unlike 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.

[11] When both members of a twin pair are diagnosed with the same trait or disorder, they are said to be *concordant.* When one twin is diagnosed with the trait or disorder but the other is not, they are said to be *discordant.*

CHAPTER TWO | Twin Research: Misunderstanding Twins, From Galton to the 21st Century

The history of twin research is usually told by twin researchers. These typically brief accounts are written from the standpoint that twin studies are a valuable tool for assessing the influence of genetic factors on human trait variation. They tend to portray the history as a linear path from necessarily crude methods and uncertain biology, to the scientifically precise twin studies of the current period.

The story cannot be told without an integration of the social views and motivations of twin researchers, as well as the social and political environments in which they carried out their research. As Joseph shows, most pioneers of twin research believed strongly in the importance of heredity, which often led them to advocate the use of selective breeding programs for humans (eugenics). According to Lawrence Wright, a journalist generally supportive of twin research, "It is certainly true that the history of twin research is one of the most appalling chapters in science, having been born in Galton's aristocratic notions of the natural worthiness of the English upper class, taken to its evil extreme by eugenicists, and too readily used by American scientists to rationalize racial injustice."[12]

Unfortunately, three generations of twin researchers have failed to fully disclose the complete history of their discipline. As Joseph documents, they tend to ignore, cover up, or distort the "appalling" history of twin research.

Germany was the world center of twin research in the late 1920s and 1930s. It is commonly known that biology and genetics played a major role in the "Third Reich" and helped provide a "scientific" justification for the murder of mental patients and, ultimately, the Holocaust. What is not well known is that the intellectual seeds of the misuse of genetics were sown long before Hitler was named Chancellor in January, 1933. Joseph documents the largely unknown story of twin research's origins in eugenics and the German "racial hygiene" movement. He quotes from many documents published in the first half of the 20th century, many of which are translated from the original German for the first time. For example, German racial hygienist and inventor of the twin method Hermann W. Siemens, who holds the distinction of being one of the most unknown and unheralded inventors of a widely used research technique,[13] wrote in 1937 as follows:

> "Since the National Socialist seizure of power the political goals that we, the racial-hygienists, are in favor of, have now become a part—and not the least important one—of the German government program. 'Racial hygiene as a utopian dream' became 'Racial hygiene as political program'. . . . Our future will be governed by racial hygiene—or it will not exist at all."[14]

[12] Wright, L. (1997, p. 33). *Twins: And What They Tell Us About Who We Are.* New York: John Wiley & Sons.

[13] For an interesting article on Siemens and the origins of the twin method, see Teo, T., & Ball, L. C. (2009). Twin research, revisionism and metahistory. *History of the Human Sciences, 22,* 1-23.

[14] Forward to Siemens, H. W. (1937, quotation translated). *Grundzüge der Vererbungslehre, Rassenhygiene und Bevölkerungspolitik* [Foundations of Genetics, Racial Hygiene, and Population Policy] (8th ed.). Munich & Berlin: J. F. Lehmanns Verlag.

Later in the same work, Siemens discussed how the ideas of England's Francis Galton, who founded the eugenics movement in the 19th century and became the first person to propose using twins to study heredity, were being put into practice in Hitler's Germany:

> "Galton already saw the possibility of integrating racial-hygienic ideals—just like a new religion—into the national conscious. The national [völkische] state, however, is now called on to be really serious about it. According to its *Führer,* it is the obligation of the national state 'to declare children as a people's most precious commodity' so that 'it will one day be considered reprehensible to withhold healthy children from the nation [emphasis in original].'" [15]

The authors of two German twin studies of criminality published in 1936, Heinrich Kranz and Friedrich Stumpfl, called for the compulsory eugenic sterilization of criminals. According to Kranz,

> "One could ascertain so far on the basis of twin concordance rates that have been found that the imbecile criminals are undesirable in terms of racial hygiene [rassenhygienisch unerwünscht]; furthermore, some types of criminal psychopaths are borderline psychotics and severe alcoholics. All of these are already being recorded to a large extent through the [1933 Nazi] sterilization law."[16]

Kranz wrote that the genetic impairment of sex offenders "can hardly be questioned," while adding that "the castration law is simultaneously fulfilling the racial hygienic task [rassenhygienische Aufgabe]."[17]

Leading German racial hygienists such as Ernst Rüdin and Hans Luxenburger (the latter published the first schizophrenia twin study in 1928) pushed hard for eugenic sterilization laws well before Hitler took power in 1933. In the United States, many states had already passed eugenic sterilization laws, and most twin research was carried out by eugenicists eager to supply data in support of the cause. For the most part, however, contemporary twin researchers have obscured the fact that their discipline has its origins in eugenics and the German racial hygiene movement.

Beginning with Galton, Joseph discusses the various ways that twins have been used for research purposes, as well as some of the methodological problems discussed by critics. Due to its association with Nazism, eugenics, and the Holocaust, interest in twin research faded in the late 1940s and 1950s, but began a revival in the late 1960s that continues to the present time.

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[15] Siemens (1937, p. 180, quotation translated).

[16] Kranz, H. (1936, pp. 250-251, quotation translated). *Lebensschicksale Krimineller Zwillinge* [The Life Destiny of Criminal Twins]. Berlin: Julius Springer Verlag.

[17] Kranz (1936, p. 251, quotation translated).

An important development in the history of twin research was the publication of a 1960 critique of schizophrenia genetic research by psychiatrist Don Jackson, then Director of the Mental Research Institute in Palo Alto, California.[18] Jackson, who was a well known pioneer of family systems theory and family therapy, argued convincingly that the results of schizophrenia twin studies (which used the twin method) could be explained by non-genetic factors. Important observations Jackson made included:

- Disorders can run in families for environmental reasons

- There were no genetic studies of schizophrenia in which researchers made diagnoses blindly. The results of these studies were therefore susceptible to the researchers' bias

- There were other sources of bias in the twin study diagnostic process, such as the unreliability of schizophrenia diagnoses, and the finding that people had a better chance of being diagnosed with schizophrenia the longer they stayed in the hospital. A sampling bias was introduced by the methods used to obtain twin subjects, which could lead to inflated concordance rates

- Contrary to genetic expectations, fraternal twin pairs were more concordant for schizophrenia than non-twin sibling pairs, even though both sets have the same genetic relationship to each other

- Contrary to genetic expectations, female identical twin pairs were more concordant for schizophrenia than male identical twin pairs

- Contrary to genetic expectations, female fraternal twin pairs were more concordant for schizophrenia than male fraternal twin pairs

- Contrary to genetic expectations, same-sex fraternal twin pairs were more concordant for schizophrenia than opposite-sex fraternal twin pairs

- Individual case histories of reared-apart identical twins concordant for schizophrenia do not provide important evidence for genetic factors because they were few in number (two), and because the pairs grew up in similar environments and had an interactive relationship with each other

- Identical twin pairs grow up in a more similar environment and are treated more similarly than fraternal twin pairs. Therefore, greater resemblance of identical pairs for schizophrenia could be explained by the more similar environments they experience.

- The unique psychological bond or "ego fusion" of identical twin pairs contributes to their higher concordance rate for schizophrenia on the basis of mutual association and

[18] Jackson, D. 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.

identification. Furthermore, the nature of the identical twinship might itself create conditions leading to the identity problems often experienced by people diagnosed with schizophrenia

- There is a striking similarity between reports of folie à deux (shared psychotic disorder) and the case histories of identical twins concordant for schizophrenia

Jackson's chapter had an important impact on the debate over the causes of schizophrenia, and raised further doubt that the twin method measured anything more than the greater environmental similarity and "ego fusion" experienced by identical versus fraternal twin pairs. In the period following its publication, twin researchers carefully considered Jackson's observations and attempted to improve their methodology. But most failed to understand that Jackson showed convincingly that the twin method was a fundamentally flawed instrument.

Today, Jackson's critique is a forgotten document in the sense that the twin method is as popular as ever, even though *none of Jackson's major points has ever been refuted*.

Jackson suggested that the intimate emotional bond and "ego fusion" experienced by identical twin pairs might weaken or blur the psychological boundaries between them. His ego fusion theory describes a psychological process that could cause the co-twin of a person diagnosed with schizophrenia to receive the same diagnosis much more frequently than his or her non-twin sibling. As an alternative "plausible hypothesis" to genetic interpretations of the twin method's results, Jackson argued that environmental (non-genetic) explanations predict 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 could say that environmental theories predict that according to the degree of environmental similarity experienced by siblings and twins, we would expect greater behavioral similarity, without concern for their genetic relationship.

Jackson's most telling point was that—among pairs with the same genetic relationship to each other—those pairs experiencing a more similar environment and closer emotional bond were consistently more concordant for schizophrenia. For Jackson this suggested that the identical-fraternal concordance rate difference (already inflated by methodological bias) could be explained primarily on environmental grounds. Using today's behavioral genetic terminology, Jackson argued that the theoretical basis of the twin method—the identical-fraternal "equal environment assumption"—is false.

Today, twin studies constitute the main pillar of support for genetic theories in psychiatry and psychology, in part because the underlying assumptions of twin research are rarely questioned in the authoritative texts of these disciplines. However, past and present critics have made a strong argument that, due to researchers' reliance on unsupported theoretical assumptions and other factors, twin research provides no scientifically acceptable evidence in support of genetic influences psychological trait variation (such as IQ and personality) and psychiatric disorders.

CHAPTER THREE | The Twin Method: An Environmentally Confounded Research Method[19]

As most leading contemporary twin researchers now recognize, there is overwhelming evidence that identical twin pairs experience much more similar environments than fraternal pairs.[20] However, twin researchers continue to uphold the validity of the twin method and the equal environment assumption (EEA) on the basis of two main arguments.

The ***first*** argument is that, although identical and fraternal twin pair environments are different, *critics* must identify the "trait-relevant" environmental factors for which identical and fraternal twin pairs experience dissimilar environments.[21] (By "trait relevant," twin researchers mean aspects of the environment that have been shown to contribute to the trait in question. For example, exposure to trauma contributes to post-traumatic stress disorder.) However, because a basic tenet of science holds that the burden of proof falls on the claimant, not on critics,[22] twin researchers themselves bear the burden of proof for showing that identical and fraternal twin pairs are not differentially exposed to potentially relevant environmental factors.

Moreover, although faced with a similar problem, twin researchers never make the "trait relevant" argument when discussing potential environmental confounds in *family* studies. In this case we have seen that they readily concede, based on the simple fact that family members share a common environment, that family studies are unable to separate potential genetic and environmental influences.

Twin researchers' qualitative distinction between twin studies and family studies is puzzling and contradictory. If differing environments automatically invalidate genetic interpretations of family study data, then the differing environments of identical vs. fraternal twin pairs must automatically invalidate genetic interpretations of twin method data as well.

The ***second*** argument twin researchers make in defense of the twin method is that identical twins "create" more similar environments for themselves by virtue of their greater genetically-caused similarity of behavior.[23] Therefore, according to most leading twin researchers, the twin method's validity is based on determining why—not whether—identical twins experience more similar environments than fraternals. Twin researchers and popularizers of their work, however, fail to understand that the *reason* identical pairs experience more similar

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[19] A *confound* is an unforeseen or uncontrolled-for factor that threatens the validity of conclusions researchers draw from their studies. Although twin and adoption researchers usually interpret their findings as supporting genetic factors, uncontrolled-for environmental influences might lead others to interpret their findings solely in terms of environmental influences.

[20] See Joseph, J. (2010). Genetic research in psychiatry and psychology: A critical overview. In K. Hood, C. Tucker Halpern, G. Greenberg, & R. Lerner (Eds.), *Handbook of Developmental Science, Behavior, and Genetics* (pp. 557-625). Malden, MA: Wiley-Blackwell.

[21] Examples of genetic researchers attempting to reverse the burden of proof from themselves to critics include Bouchard, T. J., Jr. (1993b). 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 Academic Publishers; Faraone, S. V., & Biederman, J. (2000). Nature, nurture, and attention deficit hyperactivity disorder. *Developmental Review, 20,* 568-581.

[22] Lilienfeld, S. O., Lynn, S. J., & Lohr, J. M. (2003, p. 3). "Science and pseudoscience in clinical psychology: Initial thoughts, reflections, and considerations." In S. Lilienfeld, S. Lynn, & J. Lohr (Eds.), *Science and Pseudoscience in Clinical Psychology* (pp. 1-14). New York: Guilford.

[23] Kendler, K. S. (1983). "Overview: A current perspective on twin studies of schizophrenia." *American Journal of Psychiatry, 140,* 1413-1425.

environments than fraternal pairs, be it environmental or genetic, is irrelevant in assessing the validity of the EEA.

For example, suppose that schizophrenia is caused solely by exposure to the chemical mercury. Because identical pairs spend much more time together than fraternal pairs, it is much more likely that both members of an identical twin pair will be exposed to mercury, and subsequently be diagnosed with schizophrenia, than it is that both members of a fraternal pair will be exposed and diagnosed. Let us further imagine that identical pairs are more similarly genetically predisposed than fraternals to enjoy spending time at the beach. Although identical pairs may well show much higher concordance for skin cancer than fraternals, this does not mean that skin cancer is a genetically-based disease.

On a psychological level, the theorized genetically-programmed behavioral resemblance of identical pairs could elicit more similar abusive, abandoning, or traumatizing parental treatment that could lead to higher concordance for childhood or adult disorders such as, for example, anxiety, depression, or psychosis. In these cases it is not heredity, but rather abuse, abandonment, or trauma that plays a major role in causing psychiatric disorders.

Thus, even if identicals do indeed "create" more similar environments for themselves than do fraternals on the basis of their greater genetic similarity, it would be erroneous to conclude that higher identical versus fraternal concordance for psychiatric disorders is evidence that these disorders have a genetic component. In the example of mercury causing schizophrenia—regardless of *why* identical pairs are together more often—higher identical twin pair concordance is caused solely by identical pairs' propensity to be together more often than fraternal pairs, which leads them to be more similarly exposed to mercury.

Finally, proponents of the "twins create their own environment" position use circular reasoning. That is, *they assume the very thing they need to demonstrate*. Thus, their claim that twins' behavioral resemblance is caused by genetics is based implicitly on the results of previous twin studies. In other words, modern twin researchers circularly rely on the twin method to validate the twin method, and in the process they assume that twins' behavioral resemblance is caused by genetics, in order to circularly conclude the very same thing.

Thus, the only relevant question in assessing the validity of the twin method and the EEA is whether—not why—identical twin pairs experience more similar environments than fraternal pairs.[24]

There are two main conclusions that one can reach on the basis of twin method data:

1) **Twin Researchers' Conclusion**: The greater resemblance of identical vs. same-sex fraternal twin pairs provides solid evidence that a sizable portion of the population variance of psychological traits can be explained by genetic factors, **or**

2) **Twin Method Critics' Conclusion**: The twin method is a faulty instrument for assessing the role of genetics, given the likelihood that identical vs. same-sex fraternal comparisons measure environmental rather than genetic influences. Therefore, *all previous interpretations of the twin method's results in support of genetics are wrong*.

[24] See Joseph (2010).

Joseph argues that Conclusion #2 is the correct one, and that it is unlikely that the twin method measures anything other than the more similar treatment, socialization resemblance, environment, and emotional bond experienced by identical vs. fraternal twin pairs. Thus, twin researchers' position that "Family studies by themselves cannot disentangle genetic and environmental influences" is equally true for the twin method.

Most contemporary twin researchers hold that, although identical twin pairs do indeed experience more similar environments than fraternal pairs, a body of research has tested the validity of equal environment assumption (the "EEA Test" literature) and the results suggest that the twin method is not subject to major environmental confounds. This was psychiatric genetic twin researcher Kenneth Kendler's main defense of the EEA in 2006, which led him to conclude that the heritability estimates of psychiatric disorders he presented "are substantially correct."[25]

Interestingly, the authors of most EEA test studies found that identical pairs experience more similar environments than fraternal pairs. They then argued, however, that the greater environmental similarity of identical pairs does not contribute to their greater behavioral resemblance, or if it does, that identicals' greater behavioral resemblance is caused by their greater genetic similarity. However, we have seen that what EEA-test researchers must demonstrate–without qualification–is that identical and fraternal pairs experience roughly equal environments. Joseph critiques a few of the most frequently cited EEA test studies in this chapter, and examines this body of literature in much more detail in Chapter 9 of *The Missing Gene*.[26]

CHAPTER FOUR | Genetic Studies of Twins Reared Apart: A Critical Review

The past few decades have seen a great deal of attention paid to studies of reared-apart twins. The intuitive appeal of these studies is understandable, since studying twins separated at birth and reared apart in different families would appear to overcome the problems of environmental confounds in the twin method. Yet, Joseph shows that these studies are also subject to environmental confounds and other biases.

Twins reared-apart (known as "TRA") studies compare the psychological trait resemblance of reared-apart identical pairs (known as "MZAs") to the resemblance of reared-together identicals (known as "MZTs"), the latter serving as a control group. Some studies have included a group of reared-apart fraternal pairs ("DZAs"). TRA researchers usually conclude that, because MZA correlations are far greater than zero and are comparable to MZT correlations, their results support important genetic influences on psychological trait differences. Others have cited the results of TRA studies in support of the validity of the twin method,[27] and

[25] Kendler, K. S., & Prescott, C. A. (2006, p. 125). *Genes, Environment, and Psychopathology.* New York: Guilford.

[26] Joseph, J. (2006). *The Missing Gene: Psychiatry, Heredity, and the Fruitless Search for Genes.* New York: Algora.

[27] For example, see Alford, J. R., Funk, C. L., & Hibbing, J. R. (2005). Are political orientations genetically transmitted? *American Political Science Review, 99,* 153-167.

in support of the claim that family environment has a negligible influence on human psychological development.[28]

Joseph begins Chapter 4 with an examination of stories released by twin researchers, and reported by journalists, about reared-apart twins who, upon being reunited, are said to share an amazing set of common features, traits, preferences, etc. He points out, however, that most pairs come to the attention of researchers and journalists *because of* their similarities. It's similar to the old "dog bites person versus person bites dog" rule in journalism. A dog biting a person isn't newsworthy because it's a common occurrence, whereas a person biting a dog is news because it is an *un*usual occurrence. If we read several articles describing a person biting a dog, we would be wrong to conclude that people bite dogs more frequently than dogs bite people. The same is true for twins. Stories of similar reared-apart twins are news because they are interesting and compelling; stories about dissimilar twins are not.

More importantly, there are many *environmental* (non-genetic) factors shared by MZAs (and by MZTs as well) that would lead them to resemble each more than two randomly selected members of the world's population. These factors include:

- They are exactly the same age
- They are the same sex
- They are almost always the same ethnicity
- Their appearance is strikingly similar, which will elicit more similar treatment
- They usually are raised in the same socioeconomic class
- They usually are raised in the same culture
- They shared the same prenatal environment
- They typically spent a certain amount of time together in the same family environment, were aware of each other's existence when studied, and often had regular contact over a long period of time

All of these factors work towards increasing the psychological and behavioral trait resemblance of reared-apart twins for non-genetic reasons, yet are rarely discussed in popular accounts of individual MZA pairs. Together, these factors contribute to the *cohort effect*, which accounts for similarities in people's behavior and preferences that arise from the characteristics of the historical periods and cultural milieu in which they experience stages of life at the same time. In other words, we would expect two genetically-unrelated adults of the same gender, who are born at the same time, to resemble each other more for psychological traits, behaviors, tastes, etc. than would two randomly selected members of the population, spanning the entire adult age range.

Thus, for reasons having nothing to do with genetics, we should expect to find a much higher "video game playing behavior" correlation in the United States among pairs of randomly selected biologically-unrelated 15-year-old boys than we would expect to find among randomly selected pairs drawn from the entire 15-100-year-old male and female population of the United States.

The above example illustrates one of the central fallacies of TRA studies, including the frequently cited Minnesota studies (see below).

[28] For example, see Harris, J. R. (1998). *The Nurture Assumption: Why Children Turn Out The Way They Do.* New York: The Free Press.

An example illustrating the failure to recognize cultural and cohort influences on twin resemblance, and to erroneously attribute this resemblance to genetics, is found in journalist Kay Cassill's 1982 description of reared-apart identical twins Keith Heitzman and Jack Hellback, who grew up in Louisiana:

> "Although the mighty Mississippi divided these two physically, it could not separate their parallel lives. The welder from one side and the pump mechanic from the other found that they are both allergic to ragweed and dust. Both had done poorly in school. Both disliked sports and had cut their gym classes whenever they could. They are both addicted to candy. Their similarity of dress includes a penchant for wearing cowboy hats, which matches their parallel interest in guns and hunting."[29]

As one commentator noted, "Even if 'the mighty Mississippi divided' the twins, the fact that they both wear cowboy hats and like hunting is not that unusual for two [white] working-class men in the same region of Louisiana."[30] The same point can be made about the celebrated reared-apart "Jim Twins," two working-class white men who grew up in the same region of Ohio at the same time.

Joseph concludes that the stories of individual pairs of reared-apart identical twins, while interesting, tell us little to nothing about genetic influences on human psychological trait differences. As behavior geneticist Richard Rose commented, these stories make "good show biz but uncertain science."[31] Judith Harris, author of *The Nurture Assumption*, has written that "there are too many of these stories for them all to be coincidences."[32] And she is correct that they are not all coincidences. Rather, these stories are selectively reported "show biz" combined with a stunning failure to recognize the environmental factors influencing these twins' similar behaviors.

Joseph then turns his attention to the handful of systematic "twins reared apart" (TRA) studies published since the late 1930s, the most well known being the Minnesota Study of Twins Reared Apart (MISTRA) by Thomas J. Bouchard, Jr. and his colleagues.[33] Many people who remained unconvinced about genetics by the twin method have been convinced by these TRA studies. (TRA studies record twin pair test score correlations for psychological traits such as IQ and personality. There have been no systematic TRA studies of psychiatric disorders.)

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[29] Cassill, K. (1982, p. 183). *Twins: Nature's Amazing Mystery.* New York: Atheneum.

[30] Dusek, V. (1987, p. 21). Bewitching science. *Science for the People, 19,* 19-22.

[31] Rose, R. J. (1982, p. 960). Separated twins: Data and their limits. *Science, 215,* 959-960.

[32] Harris (1998, p. 293).

[33] Bouchard, T. J., Jr., Lykken, D. T., McGue, M., Segal, N. L., & Tellegen, A. (1990). Sources of human psychological differences: The Minnesota Study of Twins Reared Apart. *Science, 250,* 223-228.

However, there are important problems with TRA studies. Among these problems we find:

- It is doubtful that most studied MZAs deserve the status of having been "reared-apart," since most pairs had significant contact with each other for many years
- In several studies, there were biases favoring the recruitment of MZA pairs who resembled each other more for behavioral traits than MZA pairs as a population
- The Minnesota researchers failed to publish life history information for the twins under study, and then denied independent reviewers access to raw data and other unpublished information
- There is controversy about whether "intelligence" and "personality" are valid and quantifiable constructs
- The impact that the researchers' bias in favor of genetic explanations appears to have had on their results and conclusions.

The main problem with TRA studies such as Bouchard's, however, is that the investigators based their conclusions on comparing MZA vs. MZT correlations—thereby failing to control for the fact that both sets share several important environmental similarities. As we have seen, these include common age (birth cohort), common sex, similar appearance, and similar political, socioeconomic, and cultural environments. (Bouchard's group attempted to correct MZA correlations for age and sex effects, but these adjustments were inadequate and unclear.[34]) Thus, Joseph argues that all TRA researchers used the wrong control group, leading to their erroneous conclusions in favor of genetics.

A scientifically acceptable TRA 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 MZTs, 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, Joseph argues that we can draw no valid conclusions in support of genetic influences on psychological trait variation from the reared-apart twin studies published to date.

[34] McGue, M., & Bouchard, T. J., Jr. (1984). Adjustment of twin data for the effects of age and sex. *Behavior Genetics, 14,* 325-343.

CHAPTER FIVE | The Heritability Concept: A Measure of Inheritance or Inherently Misleading?

In Chapter 5 Joseph argues against the use of the *heritability* statistic in psychology and psychiatry. Heritability statistics (coefficients) are widely disseminated 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 frequently pointed out, a heritability estimate is *not* a "nature/nurture ratio" of the relative contributions of genes and environment.[35]

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."[36]

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*. This is because 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 on the basis of an environmental occurrence, 100% of the "armedness" *variation* in the 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 reminded us that although "heritable" and "inherited" are very different concepts, many people wrongly believe them to be synonymous because they sound alike.[37] 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.[38] 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

[35] Hirsch, J. (1997). Some history of heredity-vs-environment, genetic inferiority at Harvard (?), and The (incredible) Bell Curve. *Genetica, 99,* 207-224.

[36] Lewontin, R. C. (1974, p. 400). The analysis of variance and the analysis of causes. *American Journal of Human Genetics, 26,* 400-411.

[37] Hirsch (1997).

[38] Lush, J. L. (1949). Heritability of quantitative characteristics in farm animals. *Hereditas* (Suppl.). G. Bonnier & R. Larsson (Eds.), 356-375.

traits and disorders. Moreover, heritability estimates are based on rarely-met assumptions about humans.[39]

Heritability estimates are dubious for the additional reason that they are derived from family, twin, and adoptions studies, which are subject to the invalidating environmental confounds and biases documented in *The Gene Illusion*.

Thus, while it is theoretically possible that genetic factors underlie psychiatric disorders and psychological trait variation, it is inappropriate and misleading to use the heritability statistic to estimate the magnitude of these potential factors. 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."[40] Clearly, Joseph's views are similar to other "harsh critics" of the heritability concept. According to the critical behavior geneticist Douglas Wahlsten, "The only practical application of a heritability coefficient is to predict the results of a program of selective breeding."[41]

CHAPTER SIX | The Genetics of Schizophrenia I: Overview.

Chapter 6 begins a two-part critical examination of the evidence behind the claim that genetic factors play an important role in causing schizophrenia, the classical psychiatric disorder. Although the genetic basis of schizophrenia is currently seen as a virtual proven fact in psychiatry and psychology, Joseph shows that the evidence supporting this position is stunningly weak.

On the basis of the analysis he made in previous chapters, Joseph argues that schizophrenia twin research, which includes studies using the twin method, individual case reports of supposedly reared-apart twins, and studies of the offspring of discordant identical twins, provides no scientifically acceptable support to genetic theories of schizophrenia. These "predisposition-stress" theories hold that schizophrenia is caused by a genetic predisposition in combination with exposure to environmental triggers. Joseph writes, paradoxically, that the genetic predisposition concept speaks more to what we *don't* know (or are unable or unwilling to change) about the environment than to what we *do* know about genetics.

Several early schizophrenia twin researchers provided detailed case histories of identical twin pairs judged concordant for schizophrenia. A closer look at these case histories suggests that the close psychological association and social isolation of identical pairs played a major role in their greater concordance for schizophrenia when compared to the histories of fraternal twin pairs. Throughout the case histories of identical twin pairs we encounter researchers' observations such as "they rather shut themselves up together," "never troubled to make separate

[39] Hirsch, J. (2004). Uniqueness, diversity, similarity, repeatability, and heritability. In C. Coll, E. Bearer, & R. Lerner (Eds.), *Nature and Nurture: The Complex Interplay of Genetic and Environmental Influences on Human Behavior and Development* (pp. 127-138). Mahwah, NJ: Erlbaum.

[40] Rende, R. (2004, p. 112). Beyond heritability: Biological process in social context. In C. Coll, E. Bearer, & R. Lerner (Eds.), *Nature and Nurture: The Complex Interplay of Genetic and Environmental Influences on Human Behavior and Development* (pp. 107-126). Mahwah, NJ: Erlbaum.

[41] Wahlsten, D. (1990, p. 119). Insensitivity of the analysis of variance to heredity-environment interaction. *Behavioral and Brain Sciences, 13*, 109-120.

friends," "no contact with other people," "they seemed to share one illness between them," "were never separated from one another," "longing intensely for her sister's company," "did not like to mix too much with others," "always clung together," "inseparable," "couldn't make a move without the other," and so on. Moreover, there are several trends in schizophrenia twin research, such as a 2- to 3-fold higher rate among same-sex versus opposite-sex fraternal pairs, that are difficult to explain on genetic grounds. Thus, as Jackson suggested, twin studies of psychosis may have revealed little more than identical twin pairs' greater environmental similarity, and their greater propensity to experience folie à deux (shared psychotic disorder) than fraternal pairs.

No researcher has been able to perform a systematic study of schizophrenia using reared-apart identical twins. However, several individual case histories of ostensibly separated pairs have been reported. In Susan Farber's exhaustive 1981 review of these cases she concluded that, according to her "lenient criteria," nine identical pairs warranted consideration as legitimately separated twins.[42] However, in all of these cases (6 pairs were considered concordant by Farber) the twins were aware of each other's existence and had periodic contact.

Regardless of how many individual reared-apart pairs are reported concordant for schizophrenia, however, they do not constitute scientifically acceptable evidence in favor of genetics. As we have seen, twin pairs often come to the attention of researchers because of their similarities. In this case, researchers or hospital administrators might become aware of a pair of identical twins hospitalized for schizophrenia, whereas a discordant pair, where only one twin is hospitalized, would not come to their attention as often. Moreover, most cases were reported by genetically-oriented investigators, whose bias influenced which pairs they chose to report, how they reported them, and how they diagnosed the twins. In any case, a basic principle of science is that a collection of anecdotes does not equal data.

Another method of studying twins compares schizophrenia rates among the offspring of *dis*cordant identical twin pairs (that is, one member of the pair is diagnosed with schizophrenia, and the other is not). The most frequently cited study in this area was published by Gottesman and Bertelsen in 1989.[43] After performing a detailed review of this body of research, Joseph concludes that it provides no evidence in support of genetic influences on schizophrenia.

Joseph ends Chapter 6 with the following conclusion:

> "Genetic influences on schizophrenia cannot be established by the results of family studies, twin studies, published studies of the offspring of discordant identical pairs, or individual cases of 'reared apart' identical twins. Taken together, this body of research points merely to the *possibility* that genes influence schizophrenia, and nothing more."

[42] Farber, S. L. (1981, p. 165). *Identical Twins Reared Apart: A Reanalysis.* New York: Basic Books.

[43] Gottesman, I. I., & Bertelsen, A. (1989). Confirming unexpressed genotypes for schizophrenia. *Archives of General Psychiatry, 46,* 867-872.

CHAPTER SEVEN | The Genetics of Schizophrenia II: Adoption Studies.

The schizophrenia adoption studies of the 1960s and 1970s were largely responsible for closing the "genetics of schizophrenia" debate in favor of genetics. In Chapter 7, Joseph undertakes an in-depth critical analysis of these studies, which were carried out in the United States, Denmark, and Finland. He argues that this body of research is flawed on several critical dimensions rarely discussed in mainstream accounts. The task of highlighting these invalidating flaws has been left to critics, who are usually ignored or dismissed by mainstream sources intent on demonstrating the definitive nature of this research.

In theory, an adoption study is able to disentangle potential genetic and environmental influences on psychiatric disorders because adoptees receive their genes from one family, but are raised in the environment of another family. However, the theorized separation of genetic and environmental factors is not as clear cut as it might appear on paper.

To date, there have been six major schizophrenia adoption studies.[44] The first, published by Leonard Heston in 1966, compared the rate of schizophrenia among 47 adopted-away biological offspring of women diagnosed with schizophrenia who were confined to Oregon state mental hospitals, versus a control group of 50 adoptees of non-diagnosed mothers. In 1968, American psychiatric investigators Seymour Kety, David Rosenthal, Paul Wender and their Danish colleagues published the results of studies based on the records of Danish adoptees and their families. Rosenthal and colleagues studied the adopted-away biological offspring of parents diagnosed with schizophrenia, "schizophrenia spectrum disorders," or manic depression. Using a different design, in 1968 Kety and colleagues began with the records of adoptees from the greater Copenhagen area, identified those diagnosed with a schizophrenia spectrum disorder, and recorded diagnoses among their adoptive and biological relatives. In a 1975 follow-up, the investigators interviewed and re-diagnosed many of the 1968 relatives. The study was then extended to the rest of Denmark, and the final results were published in 1994. Wender and his Danish-American colleagues published a study using the "crossfostering" design in 1974.

The final study was performed by Pekka Tienari and colleagues in Finland, who first published results in the 1980s and continued to publish into the 21rst century. In contrast to the earlier investigations, Tienari studied adoptees' family environments as well their genetic background. He and his colleagues concluded that both genes and adoptive family rearing environment are "predictors" of schizophrenia.[45]

* * *

[44] Lichtenstein and colleagues published a study in 2009 which used hospital and population records of Swedish parents and children (including some adoptees) to assess whether schizophrenia and bipolar disorder are genetically distinct disorders. They did not personally investigate family members or adoptees. They found an elevated risk for schizophrenia among the adopted-away biological offspring of parents diagnosed with schizophrenia. However, they did not state how many adoptees were studied. Lichtenstein, P., Yip, B. H., Björk, C., Pawitan, Y., Cannon, T. D., Sullivan, P. F., & Hultman, C. M. (2009). Common genetic determinants of schizophrenia and bipolar disorder in Swedish families: A population-based study. *Lancet, 373,* 234–39.

[45] Tienari, P., Wynne, L. C., Sorri, A., Lahti, I., Läksy, K., Moring, J., Naarala, M., Nieminen, P., & Wahlberg, K. E. (2004). Genotype-environment interaction in schizophrenia-spectrum disorders. *British Journal of Psychiatry, 184,* 216-222.

In all of these studies, the investigators concluded in favor of important genetic influences on schizophrenia, while Tienari and colleagues added a finding that disturbed family environments also contribute to the condition. However, Joseph and previous reviewers have detailed several important biases and methodological flaws in schizophrenia adoption research. Looking specifically at the famous Danish-American studies, problem areas include:

- The investigators decided to expand the definition of schizophrenia to include non-psychotic "schizophrenia spectrum disorders," and they would not have found statistically significant results without such an expansion. In fact, the Kety et al. 1968 study found *zero* cases of chronic schizophrenia among the 65 identified first-degree biological relatives of adoptees diagnosed with a schizophrenia spectrum disorder, and Rosenthal and colleagues found that only 1 of the 76 adopted-away biological offspring of a parent diagnosed with a spectrum disorder had received a hospital diagnosis of schizophrenia

- In Kety and colleagues' famous 1968 study, there is evidence suggesting that the researchers decided to change the design of their study after the initial relative group comparisons failed to obtain statistically significant results in the genetic direction[46]

- The researchers failed to adequately define schizophrenia and "schizophrenia spectrum disorders"

- In Rosenthal and colleagues' 1971 study, the researchers counted manic depression (bipolar disorder) as a "schizophrenia spectrum disorder" despite their insistence elsewhere that this diagnosis is genetically *un*related to schizophrenia. For example, Kety, Rosenthal and colleagues wrote, "manic-depressive illness was never thought to be in the schizophrenia spectrum by us."[47] Without these manic-depressive subjects, Rosenthal would not have been able to claim statistically significant results in the genetic direction

- In the Kety et al. studies using interviews to make diagnoses, there were inconsistencies in the way that the researchers decided to count and diagnose dead or unavailable relatives

[46] Joseph documents the contrast between Rosenthal's 1967 description of the relative groups the researchers planned to compare, versus the description of relative group comparisons in the 1968 Kety et al. publication. See Rosenthal, D. (1967). An historical and methodological review of genetic studies of schizophrenia. In J. Romano (Ed.), *The Origins of Schizophrenia: Proceedings of the First Rochester International Conference on Schizophrenia, March 29-31, 1967* (pp. 15-26). New York: Excerpta Medica Foundation, versus Kety, S. S., Rosenthal, D., Wender, P. H., & 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.

[47] Kety, S. S., Rosenthal, D., Wender, P. H., & Schulsinger, F. (1976, p. 417). Studies based on a total sample of adopted individuals and their relatives: Why they were necessary, what they demonstrated and failed to demonstrate. *Schizophrenia Bulletin, 2,* 413-427.

- The researchers failed to provide case history information on adoptees or relatives, and failed to study important environmental variables

- As an earlier critic noted, in Kety's Adoptees' Family study, the "procedure of counting up all the possible relatives of each index case and pooling them as if they were independent samples . . . would allow some families to disproportionately affect the results."[48] Thus, the investigators' decision to emphasize the diagnostic rate among individual *relatives*, as opposed to individual *families*, violated the assumption of independent observations underlying the statistical comparisons they used

- In the Kety studies, the researchers decided to count first- and second-degree relatives with equal weighting

- The researchers decided to include many late-separated and late-placed adoptees in their samples. This meant that, during critical developmental periods, these adoptees (a) were reared for a certain period of time by their biological parent(s), (b) suffered a disruption of attachment bonds with their biological parent(s), and/or (c) were placed in unstable environments between separation and adoption. Research performed over the past two decades suggests that disturbed parent-child attachment patterns can influence brain development during critical developmental periods.[49] This body of research raises the possibility that there are environmentally-caused (although possibly reversible) biological differences between the brains of some adoptees and the brains of securely-attached non-adoptees, which leads to even more questions about the generalizability of adoption research to the non-adoptee population

- The investigators used substandard interviews to make diagnoses. In the Kety et al. studies, many of these "interviews" never took place, but instead were *fabricated* by the investigators on the basis of hospital records.[50] In the unpublished raw data Kety and colleagues called them "pseudointerviews," but there was no indication in any of the Danish-American investigators' publications that, in some cases, "interviewed" adoptees and relatives were not actually interviewed. Of the interviews that *were* conducted, the researchers believed that a five-minute doorstep conversation at a person's home was sufficient to diagnose that person with schizophrenia[51]

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[48] Benjamin, L. S. (1976, p. 1130). A reconsideration of the Kety and associates study of genetic factors in the transmission of schizophrenia. *American Journal of Psychiatry, 133,* 1129-1133.

[49] Shore, A. N. (2001). Effects of a secure attachment relationship on right brain development, affect regulation, and infant mental health. *Infant Mental Health Journal, 22,* 7-66; Siegel, D. J. (1999). *The Developing Mind: Toward a Neurobiology of Interpersonal Experience.* New York: Guilford.

[50] Kendler, K. S., & Gruenberg, A. M. (1984). An independent analysis of the Danish adoption study of schizophrenia. *Archives of General Psychiatry, 41,* 555-564; Lewontin, R. C., Rose, S., & Kamin, L. J. (1984). *Not In Our Genes.* New York: Pantheon.

[51] Paikin, H., Jacobsen, B., Schulsinger, F., Godtfredsen, K., Rosenthal, D., Wender, P. H., & Kety, S. S. (1974). Characteristics of people who refused to participate in a social and psychopathological study. In S. Mednick, F. Schulsinger, J. Higgins, & B. Bell (Eds.), *Genetics, Environment and Psychopathology* (pp. 293-322). New York: American Elsevier Publishing Company.

- The genetic bias of the investigators appeared to influence how they decided to count relatives, how they decided to define schizophrenia, the types of comparisons they decided to make, and the conclusions they reached

- Problems with Wender's 1974 crossfostering study include (a) the researchers' decision to use global mental health ratings in place of diagnosing schizophrenia, (b) the use of selected post-hoc comparisons which were used to support the genetic position, (c) the failure to find statistically significant differences between important comparison groups, (d) the researchers' failure to consider alternative explanations of their results, and (e) that the mean age of the crossfostered adoptees at the time their adoptive parents were diagnosed with a spectrum disorder was 12-years-old. By the 1980s, Wender himself would admit that, in his 1974 study, "the question of what would happen if children born of normal parents were placed in the homes of typical schizophrenics cannot be answered"[52]

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Like the twin method, adoption studies contain their own set of critical theoretical assumptions. The most critical is the assumed absence of selective placement (called the "no selective placement assumption"). Researchers must assume that factors relating to the adoption process (including the policies of adoption agencies) did not lead to the placement of experimental (index) group adoptees into environments contributing to a higher rate of the condition or trait in question. They must assume that children were not placed into homes correlated with the biological or socioeconomic status of their biological family.

In the various studies, however, the evidence suggests that experimental group adoptees did experience more harmful rearing environments than those experienced by the control adoptees. This suggests that children whose biological family had a history of mental disorders were seen as inferior potential adoptees, and were more likely to be placed into more chaotic and potentially pathogenic adoptive families.

The adoptees who became the subjects of schizophrenia adoption research were placed in the early-to-middle part of the 20th century in three regions: Denmark, the United States (Oregon), and Finland. However, all three countries had laws permitting the compulsory eugenic sterilization of people diagnosed with schizophrenia and other "mental disorders." These laws were passed on the basis of a widespread belief in these countries that people diagnosed with schizophrenia and other disorders were the dangerous carriers of "hereditary taint."

Denmark. In 1929, Denmark became the first European nation to pass a eugenically-inspired sterilization law. A more comprehensive statute was passed in 1935.[53] These laws were in force until well after the last studied Danish adoptees were placed (placements were made between 1924 and 1947). Moreover, the Danish adoption agencies clearly stated that a potential adoptee's genetic family background was checked to determine his or her suitability (or

[52] Wender, P. H., & Klein, D. F. (1981, p. 175). *Mind, Mood, and Medicine.* New York: Farrar, Straus, & Giroux.

[53] Hansen, B. S. (1996). Something rotten in the state of Denmark: Eugenics and the ascent of the welfare state. In G. Broberg & N. Roll-Hansen (Eds.), *Eugenics and the Welfare State: Sterilization Policy in Denmark, Sweden, Norway, and Finland* (pp. 9-76). East Lansing, MI: Michigan State University Press.

desirability) for adoption.[54] One can therefore conclude that, under the conditions then existing in Denmark, the most qualified potential adoptive parents, who were usually informed of "deviance" in the adoptee's family background, would not have selected children with a biological family history of mental disorders.

Oregon. Similar conditions existed in Oregon, where the adoptees under study were placed between 1915 and 1945. Although Heston and virtually all subsequent reviewers and textbook authors failed to mention it, in 1917 Oregon passed a law creating a "State Board of Eugenics," whose duty was to authorize, in the words of the law, the compulsory sterilization of "all feeble-minded, insane, epileptic, habitual criminals, moral degenerates and sexual perverts," because they might produce "inferior" offspring. The law gave the Board of Eugenics power to examine the "family traits and histories" of such persons, and to perform "a type of sterilization as may be deemed best by said board."[55] An additional law passed in 1919 stipulated that the mere fact that a person had been admitted to a mental hospital constituted "prima facie evidence that procreation by any such person would produce children with an inherited tendency to feeble-mindedness, insanity, epilepsy, criminality or degeneracy." Given that all of Heston's experimental group adoptees were born to women hospitalized with schizophrenia, it is extremely unlikely that their children were placed into the same types of adoptive homes as the "untainted" control adoptees (and many were placed in an orphanage for several months or years).

Finland. Finland also had a long history of eugenics-inspired legislation aimed at curbing the reproduction of "hereditarily tainted" people.[56] A government commission was created in 1926 to look into the desirability of promoting the sterilization of people seen as "mentally retarded," "mentally ill," or epileptic. In 1935, the Finnish parliament passed the Sterilization Act, which allowed the compulsory eugenic sterilization of "idiots," "imbeciles," and the "insane," which included people diagnosed with schizophrenia and manic-depression. The law permitted the compulsory sterilization of people if there was reason to believe that their conditions could be genetically transmitted to their children. In 1950, Finland passed the Castration Act, which permitted the compulsory eugenic castration of criminals, the mentally retarded, and the "permanently mentally ill." Compulsory eugenic sterilization was not legally abolished in Finland until 1970.

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Because the Kety studies began with diagnosed adoptees (as opposed to diagnosed biological parents), they might appear less vulnerable to selective placement bias. However, in 8 of 33 experimental group *adoptive* (rearing) families, a parent had been admitted to a Danish

[54] Mednick, S. A., & Hutchings, B. (1977). Some considerations in the interpretation of the Danish adoption studies in relation to asocial behavior. In S. Mednick & K. Christiansen (Eds.), *Biosocial Bases of Criminal Behavior* (pp. 159-164). New York: Gardner Press.

[55] Olson, C. P. (Ed.). (1920). *Oregon Laws: Showing All the Laws of a General Nature in Force in the State of Oregon* (Vol. 2). San Francisco: The Bancroft-Whitney Company.

[56] Hietala, M. (1996). From race hygiene to sterilization: The eugenics movement in Finland. In G. Broberg & N. Roll-Hansen (Eds.), *Eugenics and the Welfare State: Sterilization Policy in Denmark, Sweden, Norway, and Finland* (pp. 195-258). East Lansing, MI: Michigan State University Press.

psychiatric facility, which was true for none of the 34 control adoptive families.[57] This statistically significant finding, which did not appear in any of the Danish-American researchers' publications, suggests that experimental adoptees were placed into more unstable and psychologically harmful adoptive homes than were the control adoptees. Thus, Kety and colleagues' finding of a higher rate of schizophrenia spectrum disorders among experimental versus control biological relatives might reflect little more than the agencies' placement of children with "tainted" biological relatives into more psychologically harmful adoptive homes. Psychosocial theories of schizophrenia emphasize, and Tienari's findings confirm, that these types of homes are more likely to produce psychologically unstable (and possibly "schizophrenic") adults.[58]

Thus, if we look at schizophrenia adoption research in the context of the social and political environments in which it was performed, it is clear that *the great majority of adoptees were given up for adoption at a time when the compulsory sterilization of "schizophrenics" for eugenic purposes was permitted by law in the country or state in which their adoptions took place (Denmark, Finland, Oregon).* Leaving aside all other problems, the evidence suggesting that selective placement occurred in these studies is reason enough to reject any conclusions in favor of genetics. Unfortunately, adoption researchers rarely discuss selective placement other than to briefly dismiss its impact on their results. When we consider these studies' other glaring methodological problems, there is little reason to accept their authors' conclusions in favor of a role for genetics in causing schizophrenia.

It is clear that investigators such as Kety, Rosenthal, and Wender intended to find—and desired to find—that genetic factors underlie schizophrenia. As seen clearly in their published works, they changed definitions, comparisons, and ways of counting to ensure that they would find what they were looking for, while at the same time turning a blind eye to the confounding influence of selective placement factors. It is not a matter of fraud, but rather of how ostensible scientific experiments are transformed into a statement of the beliefs and agendas of the investigators and their backers. Genetic research has a long history of these types of conclusions, going all the way back to Galton.

Thus, the results of family, twin, *and* adoption studies provide no scientifically acceptable evidence that genes influence the appearance of a set of behaviors given the name "schizophrenia." And at the beginning 2011, three decades of heavily funded molecular genetic research has failed to uncover any "schizophrenia genes."[59] It is likely that "schizophrenic" behavior is the way that some people respond to having experienced "seriously disturbed families," and seriously disturbing social and political environments. "All symptoms of schizophrenia," wrote psychologist Bertram Karon, "may be understood as manifestations of chronic terror and defenses against terror."[60]

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[57] Lewontin et al. (1984).

[58] See also Read, J., Mosher, L., & Bentall, R. (Eds.). (2004). *Models of Madness: Psychological, Social and Biological Approaches to Schizophrenia.* Andover, UK: Taylor & Francis.

[59] Akil et al. (2010); Latham & Wilson (2010); Jablensky (2010); Need, A. C., Ge, D. et al. 2009. A genome-wide investigation of SNPs and CNVs in schizophrenia. *PLoS Genetics, 5 (2)*; Risch, N., Herrell, R., Lehner, T., Liang, K., Eaves, L., Hoh, J., Griem, A., Kovacs, M., Ott, J. & Merikangas, K. R. (2009). Interaction between the serotonin transporter gene (5-*HTTLPR*), stressful life events, and risk of depression. *Journal of the American Medical Association, 301,* 2462-2471.

[60] Karon, B. P. (1999, p. 3). The tragedy of schizophrenia. *General Psychologist, 34,* 1-12.

CHAPTER EIGHT | Is Crime in the Genes? A Critical Review of Twin and Adoption Studies of Criminal and Antisocial Behavior.

In Chapter 8, Joseph critically examines twin and adoption studies of criminal and antisocial behavior. At the same time, he outlines the historical and political context in which these studies were performed. Genetic theories of criminality are currently regaining the foothold they had before they were discredited by their association with eugenics, Nazism, and German "criminal biology." Joseph argues that, like other types of behavior, the reported greater resemblance of identical versus fraternal twin pairs for criminality found in some of the studies can be plausibly explained on environmental grounds. Joseph then moves on to adoption studies of criminal and antisocial behavior, where he highlights several invalidating flaws and biases. He concludes the chapter as follows:

> "Family, twin and adoption studies provide no scientifically acceptable evidence for the existence of a genetic predisposition for any type of 'criminal,' 'psychopathic,' or antisocial' behavior, however it has been defined at any given time or in any given society. Finally, given (1) the potential social impact of criminal genetic research, which includes the further unwarranted stigmatization of ethnic minorities; (2) the well-known social factors leading to crime; and (3) the political aspects of deciding who is and is not labeled a criminal, it is questionable whether this type of research should even be performed."

CHAPTER NINE: Genetics and IQ.

The "genetics of intelligence" question has been a central issue in the "nature-nurture debate" for over 100 years. Historically, eugenicists and behavior geneticists used family, twin, and adoption studies of IQ test scores, the latter allegedly measuring innate or "native" intelligence, to argue that intelligence has an important genetic component (or is "significantly heritable"). In previous chapters Joseph highlighted problems with family, twin, and adoption research in general, as well as problems with the heritability concept. In Chapter 9, he extends his analysis to genetic studies of IQ.

It is important to determine what IQ tests actually measure. Genetic studies of IQ depend on the assumption that the tests accurately measure some agreed upon definition of intelligence. The claim that standardized IQ tests measure intelligence (or general intelligence, represented as *g*) has been the subject of intense debate. Over the years, critics have highlighted many problems in IQ testing, which include (1) that general intelligence is merely the product of a mathematical formula, and therefore has no physical reality; (2) that there is no consensus definition of "intelligence"; (3) that IQ tests measure school learning more than innate intelligence; and (4) that IQ tests measure only narrow abilities and ignore "real world" intelligence.

Joseph notes these and other problem areas, and goes on to argue that IQ test creators' assumptions about the lower intelligence of the working class and oppressed ethnic groups are *built into* their IQ tests. Psychologist Ken Richardson captures this position in the following quotation:

> "In effect, then, Galton's aim, and that of his followers, became simply an attempt to reproduce an existing set of ranks (social class) in another, the test scores, and pretend that the latter is a measure of something else. This is, and remains, the fundamental strategy of the intelligence-testing movement."[61]

It is therefore puzzling how anyone who understands how these tests are constructed could argue that the lower IQ scores of African-Americans versus whites, or working class versus upper class, are the result of genetic differences. Indeed, the tests are *designed* to obtain these results.

To illustrate this point, imagine that the creators a new IQ test in development find that laborers have a mean IQ score of 115, while brain surgeons have a mean IQ of 91. What would the test creators do after obtaining these results? Most likely, they would reload their IQ test with tasks and questions which they already knew brain surgeons performed much better than laborers, and they wouldn't stop until they produced a test in which brain surgeons scored 115, and laborers scored 91. Correspondingly, suppose that the same IQ test developers find that their test produces a mean IQ of 95 for whites, and a mean IQ of 108 for blacks and Latinos. In this case they would probably adjust the questions to at least allow whites to score as high as blacks and Latinos, based on an *a priori* assumption that whites are not less intelligent than ethnic minority groups. (IQ test creators have traditionally assumed that males and females are equally intelligent, and their IQ tests have reflected this assumption.) However, IQ test creators historically have made no such assumption for ethnic minority groups versus whites.

Thus, in reality, IQ tests are designed to match their creators' assumptions about which members of society are and are not intelligent, as opposed to their claim that they are merely recording the distribution of intelligence "in nature."

A belief in the existence of genetically-based racial and class differences in native intelligence has existed since the beginning of IQ testing. In the first half of the 20th century, this idea was axiomatic among large sections of academia and the upper classes. Eugenically oriented psychologists' use (and creation) of IQ tests in support of a "scientific" basis for racism has been well documented by many authors.[62] Compulsory eugenic sterilization laws in the United States, Germany, Scandinavia and elsewhere targeted "mental defectives" and the "feebleminded." A low IQ score for a German child in the late 1930s was sometimes a death sentence.[63]

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[61] Richardson, K. (2000). *The Making of Intelligence.* New York: Columbia University Press.

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[62] For example, see Chase, A. (1980). *The Legacy of Malthus: The Social Costs of the New Scientific Racism.* Urbana, IL/Chicago: University of Illinois Press (Originally published in 1977); Gould, S. J. (1981). *The Mismeasure of Man.* New York: W. W. Norton & Company.

[63] Aly, G. (1994). Medicine against the useless. In G. Aly, P. Chroust, & C. Pross (Eds.), *Cleansing the Fatherland* (pp. 22-98). Baltimore: Johns Hopkins Press.

Given (1) the controversy over what IQ tests actually measure; (2) the racial and class bias built into the most widely used IQ tests; (3) environmental confounds in family, twin, and adoption studies; (4) problems with applying the heritability concept to IQ and other psychological traits; and (5) that IQ genetic research is based on a number of questionable assumptions, the argument put forward by the authors of 1994's *The Bell Curve*[64] and others in favor of the innate cognitive inferiority of ethnic minorities and the working class is preposterous, to say the least. "Nothing," wrote Richard Lewontin, Steven Rose, and Leon Kamin in 1984, "demonstrates more clearly how scientific methodology and conclusions are shaped to fit ideological ends than the sorry story of the heritability of IQ."[65]

CHAPTER TEN | Molecular Genetic Research in Psychiatry and Psychology: An Exercise in Futility?

In Chapter 10 Joseph points out that genes for the major psychiatric disorders remain undiscovered, and that the reason may be that such genes do not exist.[66] Similarly, molecular genetic studies searching for IQ and personality genes may also be doomed to failure. The belief that such genes exist is based on the results of family, twin, and adoption studies, which molecular genetic researchers interpret as providing unequivocal evidence in favor of genetics. As Joseph argues throughout his book, however, this body of research provides no scientifically acceptable evidence in favor of genetics. This may explain the decades-long failure to find genes in psychiatry and psychology, which continues into the year 2011. Unfortunately, over three decades of media reports of subsequently unreplicated "gene findings" have misled the general public to believe that genes have been found for the major psychiatric disorders, and for IQ and personality.

Buried in the avalanche of sensational claims by researchers and reporters, we find more realistic evaluations by some prominent molecular genetic researchers. For example, by 2005 Kendler would write,

> "The strong, clear, and direct causal relationship implied by the concept of 'a gene for …' does not exist for psychiatric disorders. Although we may wish it to be true, we do not have and are not likely to ever discover 'genes for' psychiatric illness."[67]

Three years later, psychiatric genetic researcher Stephen Faraone his colleagues observed, "It is no secret that our field has published thousands of candidate gene association studies but few replicated findings"[68]

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[64] Herrnstein, R. J., & Murray, C. (1994). *The Bell Curve.* New York: The Free Press.

[65] Lewontin et al. (1984, p. 100).

[66] Joseph, J. & Ratner, C. (2010). The fruitless search for genes in psychiatry and psychology: Time to re-examine a paradigm? *Council for Responsible Genetics:* "*Gene Myths Project.*"Available online at http://www.councilforresponsiblegenetics.org/pageDocuments/1NX6VC0254.pdf

[67] Kendler, K. S. (2005, p. 1250). "A gene for …": The nature of gene actions in psychiatric disorders. *American Journal of Psychiatry, 162,* 1243-1252.

In a 2009 article published in the *Journal of the American Medical Association*, molecular genetic researcher Neil Risch and his colleagues recognized the failure of gene finding efforts in psychiatry and psychology, and that "few if any" findings have been substantiated:

> "Despite progress in risk gene identification for several complex diseases, few disorders have proven as resistant to robust gene finding as psychiatric illnesses. The slow rate of progress in psychiatry and behavioral sciences partly reflects a still-evolving classification system, absence of valid pathognomonic diagnostic markers, and lack of well-defined etiologic pathways. Although these disorders have long been assumed to result from some combination of genetic vulnerability and environmental exposure, direct evidence from a specific example has not been forthcoming. Few if any of the genes identified in candidate gene association studies of psychiatric disorders have withstood the test of replication."[69]

In a 2010 "Policy Forum" article published in the prestigious journal *Science*, three Nobel Prize winning researchers and their colleagues recognized the "frustrating lack of progress" in understanding the genetics of mental disorders.[70]

Although most researchers continue to believe that genes both exist and await discovery, commentators Jonathan Latham and Allison Wilson concluded in late 2010 that the "dearth of disease-causing genes is without question a scientific discovery of tremendous significance," and that, on the basis of this discovery, "most disease, most of the time, is essentially environmental in origin." It must follow, according to these authors, that "heritability studies of twins are inherently mistaken or misinterpreted."[71] Indeed, they are.

Instead of recognizing the possibility that decades of failed gene finding efforts may indicate that genes do not exist, which would necessitate a thorough re-examination of family, twin, and adoption studies, most researchers choose instead to emphasize *optimism* and de-emphasize *failure*. As Latham and Wilson observed, "The history of scientific refutation…is that adherents of established theories construct ever more elaborate or unlikely explanations to fend off their critics."

A recent example of the use of optimistic rhetoric in place of drawing the balance sheet on the unexpected, decades-long failure to discover genes, is psychiatric geneticists James Hudziak and Stephen Faraone's 2010 assessment of the status of molecular genetic research in child psychiatry:

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[68] Faraone, S. V., Smoller, J. W., Pato, C. N., Sullivan, P., & Tsuang, M. T. (2008, p. 1). The new neuropsychiatric genetics. *American Journal of Medical Genetics Part B (Neuropsychiatric Genetics) 147B*, 1–2.

[69] Risch et al. (2009, p. 2463).

[70] Akil et al. (2010, p. 1580).

[71] Latham, J., & Wilson, A. (2010). The great DNA data deficit: Are genes for disease a mirage? *The Bioscience Research Project* (retrieved online 12/18/10 from http://www.biosciencesource.org/commentaries/article.php?id=46).

> "Only 10 years ago, the first draft of the human genome was reported. In the intervening period, genetic research on developmental psychopathology has grown exponentially, as reflected not only in the number of published papers but also in the power of molecular genetic and statistical technologies. Although we are only in the infancy of our field, the pathway to discovery is clear. One can only imagine the incredible progress that will be made in the next decades."[72]

Rather than emphasize the failures to uncover the genes they believe underlie childhood psychiatric disorders such as ADHD and autism, and to recognize the possibility that they were wrong in believing that these genes exist, Hudziak and Faraone speak of the "exponential" growth of genetic research and the "power" of their technologies. They imply that the "number of published papers" constitutes scientific progress, instead of emphasizing that the findings of these published papers—literally thousands of them as Faraone had written in 2008—were not replicated. Hudziak and Faraone also implied that failures are to be expected because the field is only in its "infancy." Ten years earlier, Faraone, in a reply to Joseph's 2000 article on genetics and ADHD, also characterized ADHD molecular genetic research as being in its "infancy," while claiming that "molecular genetic studies have already implicated several genes as mediating the susceptibility to ADHD."[73]

Finally Hudziak and Faraone, again choosing to de-emphasize decades of failure, claimed that the "pathway to discovery is clear," and that "incredible progress…will be made in the next decades." However, psychiatric geneticists have been saying this for 30 years. Rather than being in its infancy, 15 or so years of molecular genetic studies of childhood psychiatric disorders have produced an important finding: The genetic basis of these disorders has been refuted.[74] Thus, the main finding of the molecular genetic research they speak of is that *genes for childhood psychiatric disorders do not appear to exist*.

[72] Hudziak, J. J., & Faraone, S. V. (2010, p. 734). New genetics in child psychiatry. *Journal of the American Academy of Child and Adolescent Psychiatry, 49,* 729-735.

[73] Faraone & Biederman (2000, p. 572); see also Joseph, J. (2000). Not in their genes: A critical view of the genetics of attention-deficit hyperactivity disorder. *Developmental Review, 20,* 539-567, and Joseph, J. (2000). Problems in psychiatric genetic research: A reply to Faraone and Biederman. *Developmental Review, 20,* 582-593.

[74] Latham & Wilson (2010) made a similar point about the failure of molecular genetic studies to uncover genes causing common diseases. If we take the results "at face value," they wrote, "genetic predispositions as significant factors in the prevalence of common diseases are refuted. "

CHAPTER ELEVEN: Where Do We Go From Here?

In the final chapter, Joseph sums up his critique of family, twin, and adoption studies, and calls on the fields of psychiatry and psychology to perform a critical reassessment of this body of research. In addition, he suggests ways that all research could be improved, and calls for a reassessment of behavior geneticists' use of concepts such as "heritability," "IQ," and "personality."

Joseph also warns that the current ascendancy of genetic theories and genetic determinism, albeit on the basis of faulty and misinterpreted research, could lead to a rebirth of the eugenics movement. He concludes his book as follows:

> "In contrast to the bleak hereditarian view of humans and their future, there exists a radically different perspective. Human psychological distress, to the extent that it goes beyond people's normal reactions to life events, is primarily the result of well-known and well-documented psychologically traumatic environments and events, and conditions such as racism, sexism, homophobia, unemployment, economic inequality, war, and social alienation. Future societies free of these conditions will see a dramatic reduction in human suffering, as well as a flourishing of ability and innovation, and any possible role of genetic influences in shaping human psychological differences will be of interest mainly to historians."

THIS SUMMARY REVISED AND UPDATED JANUARY, 2011

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