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# CHAPTER ONE

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## Introduction

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In 1925 psychiatrist Abraham Myerson, who was writing at the height of the eugenics movement's influence the belief in the overriding importance of genes, 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.'<sup>1</sup> 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 together, studies of twins reared apart, and molecular genetic research. In spite of the widespread view that the results of these investigations converge on the importance of genetics, there are serious problems associated with these methods. And as we shall see in Chapter 10, molecular geneticists have failed to find postulated genes for the major psychiatric disorders. Thus, Myerson's 1925 observation is more relevant to today's evidence than is commonly believed.

In 1996 twin researcher Irving Gottesman wrote, 'no educated person . . . can be oblivious to the fact we are in the midst of a genetic revolution.' He added that the younger generation of genetic researchers could hardly imagine 'the uphill battle that had been fought for the past 45 years.'<sup>2</sup> With what weapons was this 'battle' fought? Gottesman identified the 'old-fashioned strategies' of family, twin, and adoption studies. And he is correct that these methods helped pave the way for the ascendancy of the genetic position as articulated by the fields of behavior genetics and psychiatric genetics.

Newspapers often report claims that a specific gene has been linked to a psychiatric disorder, trait, or behavior, although most of these claims are subsequently found to have no merit. Several books have been published popularizing behavior genetic research, which include William Wright's *Born That Way*, Lawrence Wright's *Twins*, Hamer and Copeland's *Living With Our Genes*, and Judith Harris's *The Nurture Assumption*. Most have focused on the results of twin and adoption studies which, it is claimed, call forth a radical re-evaluation of the 'nature-nurture' debate on the side of nature.

This book provides an alternative view of this body of literature. Far from establishing the importance of genes, we will see that family, twin,

and adoption studies are plagued by researcher bias, unsound methodology, and a reliance on unsupported theoretical assumptions. It examines research methods, theories, and specific studies in the only way they should be examined — at their roots. Special attention is devoted to twin research because of its central position in the case for genetics.

### **Outline of the chapters**

Each chapter of this book can be read independently, and any particular chapter (other than Chapters 3 and 4) could be skipped without missing the basic argument. Chapters 7 and 8 consist mainly of in-depth analyses of published studies and may prove difficult reading for some people. They could be skimmed by readers who are already in general agreement with the conclusions I draw at the ends of these chapters.

Chapter 2 looks into the history of twin research, for a simple reason: the full story of this history has never been told. Unfortunately, twin researchers cannot be relied upon to provide details on the more unsavory aspects of this history. In fact, the origin of twin research as a tool of eugenics, ‘racial hygiene,’ and Nazism is rarely mentioned by twin researchers. Beginning with Galton, I discuss the various ways that twins have been used for research purposes, as well as some of the methodological problems as discussed by critics. We will see that interest in twin research waned in the late 1940s and 1950s, but began a revival in the 1960s that has continued up to the present day.

Chapter 3 is devoted to an analysis of the theoretical underpinnings of the most commonly used method in twin research, the ‘classical twin method.’ The twin method compares reared-together identical and reared-together same-sex fraternal twins, and its proponents claim that the greater correlation or concordance of identical twins points to the operation of genetic factors on the trait in question. However, this finding depends on the validity of a critical theoretical assumption which holds that identical and fraternal twins experience equal environments. Interestingly, most people — including many leading twin researchers — understand that identical twins’ physical and social environments are much more similar than fraternal. Nonetheless, twin researchers continue to uphold the validity of the twin method on the basis of a new definition of the equal environment assumption. I shall, however, demonstrate that this new definition is as untenable as its predecessor. Thus, contrary to the views expressed in countless textbooks and popular reviews, identical-fraternal comparisons cannot be relied upon to tell us anything about genetic influences on psychological trait differences.

Chapter 4 turns to studies of twins whom the investigators have called ‘reared-apart.’ Although these types of investigations might appear to eliminate the environmental factors confounding the twin method, we will see that they all suffer from important methodological problems, not the least of which is the fact that few pairs were actually reared apart

without knowledge of and contact with each other. I focus on the well-known Minnesota studies because they have been instrumental in strengthening the genetic position. I also look at cases of individual pairs who have been reported by journalists. Although their stories are interesting, there are many reasons why they don't tell us anything important about genetics.

In Chapter 5, I explain the reasons why heritability figures are discussed only briefly in this book. The heritability concept was designed for use in agriculture to predict the results of a program of controlled breeding. Unfortunately, it has been falsely promoted as a 'nature-nurture ratio' for the relative influences of genes and environment on particular traits.<sup>3</sup> As I argue in this chapter, there is little reason to believe that heritability estimates can serve this function.

Chapter 6 begins a two-part series looking into the evidence supporting the widely held view that schizophrenia is a genetic disorder. The genetic basis of schizophrenia is nowadays so widely accepted that the question is almost never debated. Amazingly, however, the evidence supporting this position is extremely weak. The main evidence, we will discover, is derived from biased, methodologically unsound, and environmentally confounded research. In this chapter, I point to trends and data from the schizophrenia twin studies to further illustrate the problems with the twin method.

The schizophrenia adoption studies of the 1960s and 1970s are largely responsible for closing the 'genetics of schizophrenia' debate. Chapter 7 consists of an in-depth critical analysis of these studies, which were performed in the United States, Denmark, and Finland. We will discover that the schizophrenia adoption studies were plagued by methodological problems and bias. Most importantly, all schizophrenia adoption studies were performed in regions having eugenic sterilization laws in the era when adoptees were placed. This makes it unlikely that 'hereditarily tainted' children were placed into the same types of homes as other adoptees. Therefore, the basic assumption of adoption studies — that experimental and control adoptees had the same chance of being placed into available adoptive homes — was violated. On the basis of the serious problems with twin and adoption studies, I call for reopening the debate on the role of genetics in schizophrenia and other psychiatric disorders.

Chapter 8 examines twin and adoption studies of criminal and antisocial behavior as an example of how genetic research has been carried out in the area of human behavioral differences. Genetic theories of criminality have been regaining the foothold they had before they were discredited by their association with Nazism and German 'criminal biology.' In keeping with one of the main themes of this book, I demonstrate that the reported higher identical twin concordance for criminality can be plausibly explained on environmental grounds. Like schizophrenia, adoption studies have been put forward as important evidence in support of the role of genetics. Also like schizophrenia, there are several invalidating features of these studies, which I document.

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In Chapter 9, I examine the argument that intelligence (as allegedly measured by standardized IQ tests) has an important genetic component. Studies cited in support of this position are looked at only briefly, and emphasis is placed on the intelligence tests themselves. Specifically, we will see that assumptions about the genetic inferiority of lower classes and subordinated races are *built into* IQ tests. Thus, it is astonishing that anyone who knows how these tests are constructed could argue that the lower IQ scores of blacks versus whites, or working class versus upper class, are the result of genetic differences.

Chapter 10 examines the failure of molecular geneticists to find genes for psychiatric disorders and psychological trait variation. The belief that these genes exist derives from the results of family, twin, and adoption studies. As I argue throughout this book, these studies do not show what they are believed to show, and this is being borne out in the failure to find genes which in most cases are unlikely to exist. Unfortunately, the general public is sometimes led to believe that genes have been found. The sensational accounts of the original 'findings' are followed by brief reports of the inevitable failures to replicate. Behavior genetics and psychiatric genetics have arrived at a blind alley as they attempt to show us what they believed all along actually exist. Unlike family, twin, and adoption studies, in this case unsound methodology and glossed-over implausible assumptions cannot produce the desired results of finding actual genes.

In Chapter 11, I summarize and integrate the main arguments made in the preceding chapters, and propose that the entire body of evidence produced by behavior genetics and psychiatric geneticists be re-evaluated. I note further that biased and unsound research is not limited to these fields, but is a widespread problem. Finally, I provide an optimistic view of human progress; a view which stands in stark contrast to the bleak forecasts of most human genetic investigators.

### **Family studies**

Although twin and adoption studies are a major focus of this book, family studies are sometimes cited in support of the genetic position, and therefore deserve mention. The family (or consanguinity) method of study constitutes a systematic attempt to determine whether a condition clusters in families, thereby laying the basis for the possibility of finding a genetic component with other methods. Family studies locate persons affected with a particular trait or condition and attempt to determine whether their biological relatives are similarly affected more often than members of the general population or a control group. If a condition is found to cluster or 'run' in families, it is said to be familial. Note that 'familial' is not the same as 'genetic.' Unfortunately, many people view these terms as being synonymous, when in fact they are not. As most genetic researchers now acknowledge, the aggregation of a condition in families

is consistent with genetic or environmental causation. Psychiatric geneticists Steven Faraone and Ming Tsuang, for example, noted that a family study 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.’ They concluded that ‘Although family studies are indispensable for establishing the familiarity of disorders, they cannot, by themselves, establish what type of transmission.’<sup>4</sup> However, this has not always been the prevailing view.

The first schizophrenia family study was published in Germany during World War I by Ernst Rüdin, and the most influential study of this type was published by Franz J. Kallmann in 1938. (Kallmann, as we will see, also studied twins.) Most of the early family studies were authored by strong proponents of the genetic position, and most did not perform blind diagnoses. Kallmann believed that the familiarity of schizophrenia *proved* that the condition was genetic in origin: ‘The principal aim of our investigations was to offer conclusive proof of the inheritance of schizophrenia and to help, in this way, to establish a dependable basis for the clinical and eugenic activities of psychiatry.’<sup>5</sup> Psychologist Alvin Pam noted Kallmann’s faulty logic and commented further on the family study method,

The most serious breach in inductive logic committed by Kallmann was his use of kinship concordance rates to determine genetic transmission of psychopathology. We have already noted that no family inheritance study can control for environment in human research; such data, therefore, are nowhere near ‘suggestive’ — they are at best inconclusive and at worst misleading . . . This inferential limitation holds with respect to any consanguinity finding, even if the design and technique employed in the investigation were scientifically impeccable.<sup>6</sup>

Today, most behavior genetic and psychiatric genetic researchers acknowledge that family studies cannot establish the existence of genetic factors, and have cited twin and adoption studies as the primary evidence in favor of the genetic basis of schizophrenia and other conditions.

In spite of the formal pronouncement that family studies cannot be used as evidence of genetic transmission, some researchers attempt to use the results from these studies in support of genetic conclusions. For example, in Russell Barkley’s authoritative 1998 handbook for the diagnosis and treatment of attention-deficit hyperactivity disorder (ADHD), he wrote, ‘Family aggregation studies find that ADHD clusters among biological relatives of children or adults with the disorder, strongly implying a hereditary basis to this condition.’<sup>7</sup> And schizophrenia researchers Frangos and colleagues concluded their 1985 family study by writing, ‘We consider the results to provide evidence that even narrowly defined schizophrenia . . . has a genetic component . . .’<sup>8</sup> Whereas

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Faraone and Tsuang correctly viewed the results from family studies as providing only an 'initial hint' that genetic factors might be operating, investigators such as Barkley and Frangos et al. believed that these results provide evidence in favor of genetics.

Even before the first family studies were performed, patterns found in the family pedigrees of affected individuals were seen as definitive proof of genetic influences. The case of pellagra provides us with an example. Pellagra is a disease that ravaged poor people in the southern part of the United States during the first half of the twentieth century. Before then, it had been known in southern Europe for almost 200 years. The often fatal disease, still found among the world's poor, is characterized by severe skin rash, gastrointestinal problems, and mental disturbance. Between 1730 and 1930 pellagra claimed over half a million lives, including tens of thousands of poor blacks and whites in the southern United States. On the basis of the pioneering work of Joseph Goldberger and others in the early part of the twentieth century, it was firmly established that pellagra is caused by a dietary deficiency linked to malnutrition among the southern poor. In other words, pellagra was (and is) a disease of hunger and poverty. But it has not always been viewed this way.

The pioneers of the American eugenics movement believed that pellagra had a hereditary basis. According to a 1916 article by Charles Davenport, 'Pellagra is not an inheritable disease in the sense in which brown eye color is inheritable. The course of the disease does depend, however, on certain constitutional, inheritable traits of the affected individual.'<sup>9</sup> Davenport based his conclusions on the results of his family pedigrees, then known as 'eugenical family studies.'<sup>10</sup> Yet even before the advent of eugenical family studies, Davenport believed that 'many physical, mental and moral traits have been proved to have an hereditary basis, and it seems probable that in practically all there is an hereditary factor of more or less importance.' He saw eugenical family studies as 'afford[ing] the means of studying this [proven] hereditary factor.'<sup>11</sup>

In support of his position, Davenport produced 15 pages of pedigrees of families affected by pellagra. These diagrams showed that pellagra clustered in families at a rate far greater than would be expected in the general population. Although Davenport mistakenly believed that the condition was communicable, his fundamental position was that 'the constitution of the organism must be held to be the principal cause of the diversity which persons show in their reaction to the same disease-inciting factors. This constitution of the organism is a racial, that is, hereditary factor.'<sup>12</sup> In Davenport's paper one searches in vain for any mention of the fact that most pellagrins lived in dire poverty. Conspicuously absent from his study are words such as 'poor,' 'poverty,' 'hunger,' or 'malnutrition,' in spite of the fact that several researchers had pointed to nutritional factors in pellagra and that there were no reported cases of health care providers, working in close contact with pellagrins, who had developed the disease. Above all, pellagra was known to be correlated

with poverty and the consumption of corn.

That Davenport and others were blinded by hereditarian views and failed to pay serious attention to the possible environmental confounds in pellagra family pedigrees is hardly an original observation; indeed, it is a classic example of the potential fallacy of reaching conclusions about genetic factors when studying people who share a common environment as well as common genes.<sup>13</sup> This is true for virtually all psychiatric conditions, psychological traits, behaviors, and medical conditions. Thus, it is now generally agreed that the results from family studies are explainable on either genetic or environmental grounds.

### Terms used in the book

I would like to define some terms that will be used throughout the book. I use **human genetics** as a shorthand for 'behavior genetics and psychiatric genetics,' although several aspects of human genetic research do not fit into these categories. I frequently discuss the underlying **assumptions** of genetic research. In this sense, an assumption is something that is taken to be true. The conclusions of behavior genetic research are based on many questionable assumptions, some of which the researchers identify and discuss, and many others that are not identified. **Concordance rates** are used in psychiatric twin studies. Twins are said to be concordant for a condition when both members of the pair are affected, and *discordant* when only one is affected. Unless otherwise noted, all discussions of twins in this book refer to pairs reared together in the same home. **Correlation coefficients** quantify the degree of relationship or association between two variables. A positive correlation is expressed as a coefficient ranging from 0.0 to 1.0. A 'relationship' means that two occurrences vary together, not necessarily that they are similar. In addition, correlation says nothing about the cause of the association. A classic example is that ice-cream sales increases are often correlated with an increase in the crime rate, which does not mean that eating ice cream causes people to commit criminal acts. The correlation is spurious because warm weather contributes to the causes of both higher ice-cream sales and a higher crime rate. Although the position that correlation does not imply cause is a basic statistical principle, it is frequently misunderstood or ignored by people attempting to use correlational data to imply cause and effect. When I say that a study is **methodologically** unsound, I mean that it violates a set or system of methods and principles used in psychological or psychiatric research.

The word **environmental** refers to all non-genetic factors influencing a trait or contributing to the causes of a condition. Examples of environmental factors include family or social milieu, viruses, prenatal complications, air and water pollution, and socioeconomic status. When discussing environmental and genetic influences, it should be emphasized that they frequently interact. For example, human height is influenced

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by heredity, but the social environments of adults in western societies who are three feet tall and those who are six feet tall will be vastly different.

Another word I employ frequently is **confound**. A basic study tests the effect of a factor or factors (the independent variable) on another factor (the dependent variable). The investigators attempt to eliminate (control for) factors other than the independent variable which could influence (confound) the results. For example, a serology laboratory takes precautions to keep foreign substances out of its samples, since contamination could lead to false results. In human genetic research, conclusions favoring genetic factors are dependent upon being able to control for environmental confounds. Human genetic investigators usually state that the results of family studies are confounded by environmental factors, but that twin and adoption studies are not.

In discussions of psychiatric adoption studies, the terms **index group**, **experimental group**, and **control group** are used frequently. I use 'index group' and 'experimental group' interchangeably in reference to subjects identified because they are, or because they are related to, people diagnosed with the condition under study. Control groups consist of people matched with members of the index (experimental) group, but who are not diagnosed with the condition under study. The original investigators' conclusions are drawn from comparisons between the index group and the control group. The **genetic theory of schizophrenia** refers to the view that although environmental factors might be important, genetic factors are equally if not more important.

It is also important to distinguish between genetic influences on a **trait** versus genetic influences on **trait differences**. A trait is a distinguishing characteristic or quality of a person. Clearly, there are many inherited physical and mental traits that distinguish humans from other species. Behavior genetic research takes this basic fact as a starting point and seeks to establish that genetic factors contribute greatly to differences among people for particular human traits such as personality and intelligence. As behavior geneticist Robert Plomin has written, 'It is critically important to recognize that behavioral genetic research is limited to the investigation of the genetic and environmental origins of individual differences within a species, not species-typical development.'<sup>14</sup> Still, the critical differentiation between genetic influences on behavior versus genetic influences on behavioral *differences* is often conflated. In a 1994 article in *Science*, Plomin added to this problem by writing, 'Just 15 years ago, the idea of genetic influence on complex human behavior was anathema to many behavior scientists.'<sup>15</sup> While hardly anyone in 1979 doubted that genes influence human behavior, many disputed the contention that genes were involved in human behavioral *differences*.

I also discuss various aspects of the way that the **eugenics** movement impacted genetic research. This movement was founded by Francis Galton in the nineteenth century. Eugenics refers to the belief that human beings can be improved by means of selective breeding. 'Positive eugenics' refers to measures used to increase the reproduction of those seen as genetically

'fit,' whereas 'negative eugenics' refers to attempts to curb or eliminate the reproduction of the 'unfit.' Examples of negative eugenic measures include marriage restrictions, laws against miscegenation (racially mixed marriages), sterilization, and in the case of the Nazi Germany, genocide. The eugenics movement was very active and influential in the United States in the first four decades of the twentieth century, until the crimes of the Nazis in the name of eugenics and 'racial hygiene' demonstrated what the logic of eugenics could ultimately lead to. The mainstays of eugenic research were family and twin studies. Although we have seen that family studies are no longer seen as conclusive evidence for genetic factors, twin studies remain 'the workhorse of behavioral genetics.'<sup>16</sup> The use of twins in an attempt to prove that heredity is important has a long and yet largely unknown history. In the next chapter, I attempt to give the reader a better sense of this history.

## **Notes**

<sup>1</sup> Myerson, 1925, p. 23.

<sup>2</sup> Gottesman, 1996, p. 52.

<sup>3</sup> Hirsch, 1997.

<sup>4</sup> Faraone & Tsuang, 1995, pp. 88–9.

<sup>5</sup> Kallmann, 1938a, p. xiv.

<sup>6</sup> Pam, 1995, p. 19.

<sup>7</sup> Barkley, 1998, p. 36.

<sup>8</sup> Frangos et al., 1985, p. 385.

<sup>9</sup> Davenport, 1916, p. 13.

<sup>10</sup> Davenport & Laughlin, 1915.

<sup>11</sup> *Ibid.*, p. 3.

<sup>12</sup> Davenport, 1916, p. 4. Davenport mistakenly believed that blacks were constitutionally less susceptible to pellagra than whites.

<sup>13</sup> See Chase, 1975, 1980.

<sup>14</sup> Plomin, 1996, p. 30.

<sup>15</sup> Plomin, 1994, p. 187.

<sup>16</sup> Plomin & DeFries, 1998, p. 64.