Chapter 1

Introduction: Methods, Meanings, and Morals

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The ambition to explain and predict criminal behavior scientifically has defined the field of criminology and inspired a vast number of studies in psychology, sociology, and economics. As central as this ambition has been to a wide variety of research programs, it has been called into question by those who doubt that criminal behavior is susceptible to scientific explanation or prediction or who worry that the desire to explain and predict masks a desire for pervasive social control. The recent pursuit of this ambition by human geneticists has inspired particularly strong hopes and fears. The success of genetics in understanding human disease suggests that it could be a powerful tool in the scientific investigation of human behavior, including criminal behavior. At the same time, the checkered history of human genetics suggests that it can easily be abused, misrepresented, or misunderstood, regardless of the validity of the studies or the motivation of the researchers.

At present, a variety of research programs investigates genetic influences on human behaviors, dispositions, and mental traits. Heritability studies seek to tease out genetic from environmental effects on human behavioral differences, largely by examining twins and adoptees; molecular researchers look for markers, and ultimately genes, associated with crime and violence; neurobiologists explore causal pathways by which genetic variations may affect aggressive and impulsive behavior (see generally Raine 1993; Reiss and Roth 1994; Stoff and Cairns 1996; Sherman et al. 1997). Although specific research programs differ greatly in aim and method, they share the assumption that it makes scientific sense to look for genetic contributions to important mental and behavioral differences among people.

These programs have had to wrestle with the legacy of human genetic research from the first half of the twentieth century, which studied heri-

table differences among people with the goal of improving the genetic stock of humanity (Kevles 1985; Duster 1990; Paul 1995). The confidence of many early researchers in their moral authority to control human reproduction was matched only by their ignorance of the complex patterns and mechanisms of human inheritance. Not only did their research programs yield little scientific insight, but they also lent scientific prestige to the restrictive immigration policies and sterilization laws of many Western nations at that time as well as to the campaigns of mass sterilization and "euthanasia" in Nazi Germany.

After a period of understandable quiescence following World War II, human genetic research on mental and behavioral traits has made a dramatic comeback. Researchers attribute this comeback in large part to the advent of sophisticated techniques for isolating and manipulating genetic material and statistically assessing patterns of inheritance. These techniques, they believe, will eventually make it possible to identify the specific genes and causal mechanisms that underlie the heritability of many psychological traits and behavioral dispositions, including some of those associated with criminal behavior. Although no genetic variations have yet been identified that can explain any significant proportion of criminal behavior, researchers see ongoing studies of the genetics of psychiatric and behavioral disorders as encouraging preliminaries (see, e.g., Carey and Gottesman 1996; Goldman 1996a).

Researchers also claim that the renewed interest in genetic contributions to personality and behavior represents a sensible retreat from the dogmatic environmentalism of the postwar era, which made unrealistic demands on families and institutions and promoted "one-size-fits-all" social interventions, doomed to failure by their neglect of individual differences. An understanding of the genetic and biological contributions to such differences, researchers insist, is critical for humane and effective social policies (e.g., Carey and Gottesman 1996; Fishbein 1996).

Critics contend that the comeback of genetic research on human behavior is largely due to the public's obsession with, and credulity toward, genetic explanations, and to a blanket repudiation of the optimistic environmentalist policies that enjoyed a brief ascendancy in the postwar years. They argue that the search for genetic factors involves the "medicalization" of social behavior and thus diverts attention and resources from the social and economic conditions largely responsible for crime. Although most mainstream researchers do not look for, or expect to find, racial differences in genetic predispositions to antisocial behavior, opponents argue that their work reinforces public perceptions of

criminal behavior as an essentially biological problem, affecting some races more than others (see Wasserman 1995; Miller 1996).

The public debate on current research has been characterized by the invocation of competing precedents. Researchers place behavioral genetics in the Enlightenment tradition of scientific progress, which has alleviated untold suffering by overcoming ignorance and superstition; critics place it in the more recent tradition of eugenics, which has justified terrible social inequalities by the invention or exaggeration of biological differences. Important as this debate is, its focus on historical precedents has tended to obscure critical issues about the scientific potential and moral relevance of claims of genetic influence on criminal behavior. While it would be naive to ignore history in appraising current human behavioral genetics, it would be equally mistaken to assume that an adequate appraisal can be made on that basis alone. Contemporary behavioral genetics appears to conform to prevailing norms of scientific inquiry and offers detailed and highly qualified findings, not the vague, sweeping assertions of causal influence that discredited eugenic research earlier in this century. We cannot judge whether contemporary research is likely to correct or repeat a history of scientific folly and social abuse without examining its assumptions, methods, results, and prospects. Similarly, we can hardly assess the social significance of current research, the motivation of those pursuing it, or the socially appropriate uses it may have without a clearer understanding of its potential for actually predicting, controlling, and explaining behavior, and its implications for the ascription of blame and punishment.

The chapters in this volume were originally commissioned for an interdisciplinary conference that sought to examine the scientific prospects and social implications of genetic research into crime and violence. The proposed conference was funded in 1992 by the Ethical, Legal, and Social Implications (ELSI) Program of the National Center for Human Genome Research, at the National Institutes of Health (NIH). The same year that the grant was awarded, a leading federal research psychiatrist announced plans for a comprehensive "Violence Initiative" that would investigate the genetic and biological sources of "individual vulnerabilities" to criminal and antisocial behavior. That announcement provoked a furor over public funding for behavioral genetic research, and the proposed conference soon became part of the controversy it had been organized to explore. Although it had no connection to the Violence Initiative, and its agenda featured some of the leading critics of human behavioral genetics, the conference was assailed as lending sup-

port to racist assumptions about crime and repressive programs of crime control. In response, the NIH suspended, then terminated, the grant. Funding was restored, however, and the conference rescheduled, after an administrative review by the Public Health Service, the parent agency of the NIH, held the termination "arbitrary and capricious." Although it was conducted in a harsh limelight (the entire proceedings were filmed by C-SPAN and attended by dozens of print and television journalists) and briefly interrupted by a demonstration, the conference succeeded in getting entrenched and often bitter adversaries to talk to each other with civility and mutual respect.

That success was due in part, we believe, to the circulation of early drafts of the commissioned papers. The relevance and quality of those papers were widely recognized by the participants, and several were repeatedly cited during the conference, providing a common framework for adversaries whose very terms of discourse had often been at odds. The papers did not, for the most part, directly address the issues on which the conference focused: the justifications for government funding of genetic research on violence and the impact of such research on law enforcement, criminal justice, and vulnerable minorities. Rather, they offered a scholarly assessment of the potential of such research for predicting, explaining, and controlling crime, and for assigning responsibility for its commission. That assessment was recognized by many conference participants as critical to the resolution of the issues they were debating. If genetic research had little prospect of predicting, explaining, or reducing crim inal and antisocial behavior, and little relevance for the imposition of punishment or blame for such behavior, its pursuit and its funding, would be misguided, based on unreasonable expectations or ulterior motives. Furthermore, even if the research had substantial value, its explanatory potential and social implications may have been greatly exaggerated or distorted in the public debate.

The chapters collected in this volume are largely a subset of the commissioned papers, significantly revised and supplemented by additional contributions. They focus on two sets of issues central to the appraisal of current research into the genetics of criminal behavior:[1]

1. What are the assumptions about criminal behavior, causation, and scientific explanation underlying research programs that seek genetic factors in criminal behavior, and are they justifiable? Given the widely acknowledged importance of environmental factors, and the

fact that crime is a social category, what can a genetic investigation possibly tell us about criminality?

2. How would credible evidence of genetic influence on criminal behavior affect our practices of blaming and punishing? Would such evidence compel us to revise our conceptions of moral and legal responsibility or modify our assessment of particular agents?

Unfortunately, much of the discussion regarding genetics and violence has taken place without serious reflection on these issues. This volume presents such reflection by a diverse group of philosophers, many of whom had not previously written in this area. Because a great deal of bad philosophy pervades the public debate – in arguments for and against behavioral genetic research – it was important to elicit papers from contributors who could subject these arguments to close critical scrutiny.

For the remainder of this Introduction, we briefly describe the basic methods and assumptions of recent behavioral genetics, highlighting the claimed findings and the controversies most relevant to the prediction, explanation, and appraisal of criminal behavior. We do not intend this to be a freestanding review, but rather an introduction to the essays that appear in this volume, many of which offer their own careful exposition of particular aspects of the research. We then describe how the essays in this volume approach the issues we have just outlined.[2]

CLASSICAL AND CONTEMPORARY BEHAVIORAL GENETICS

Before summarizing the methods that behavioral geneticists employ, it is important to specify what it is that they investigate. Although the obvious answer is "behavior," we must distinguish the study of behavior from the study of behavioral *differences*. The latter is the primary focus of behavioral genetics. Unfortunately, this distinction and its significance have not been generally appreciated by nonscientists.

Conceptually, the contrast is clear enough; it is as clear as the difference between asking "Why is this person behaving criminally?" and "Why is this person behaving more criminally than this other person?" Presumably a complete answer to the first type of question can lead to answers to the second type; but the converse is not necessarily true: factors that account for behavioral differences may play a minor role in accounting for the behavior itself. By way of analogy, think of two people, one six feet

tall and the other six feet, one inch tall. The factors that explain the one-inch difference may play hardly any role in explaining the six feet in stature that each has attained. For this reason, it would be a mistake to take the answer to one question as the answer to the other. Behavioral geneticists may hope that an explanation of differences will provide, or at least lead to, an explanation of the behaviors they study. But they cannot assume this connection at the outset, and there may be reasons to suspect that behavioral commonalities and differences have distinct sources.

The reason for the scientific emphasis on behavioral differences may be largely historical: earlier generations of human geneticists were preoccupied with the question of why people *vary* with respect to significant traits and behaviors. Perhaps as a result, the research methods adopted by behavioral genetics are generally better suited for investigating behavioral differences than the behavior itself. This is particularly true of heritability research.

Heritability Research

Until techniques were developed in the 1970s for isolating and manipulating genetic and other molecular material, human behavioral genetics was largely confined to heritability studies. Heritability research begins with the scarcely debatable observation that many behaviors and psychological traits travel in families. Parents usually confound the assessment of genetic influence, however, by providing their children with rearing environments as well as genes. Thus, it may be difficult to say whether their genes or their parenting is responsible for the transmission of behavior from one generation to the next. Behavioral genetics exploits two processes that can highlight the differential impact of genetic and environmental contributions: twinning and adoption. Twinning produces offspring that share either half their genes (dizygotic/DZ), the same proportion as in normal siblings, or all their genes (monozygotic/MZ). If the rearing environments of DZ twins can be assumed to be as much alike as those of MZ twins, and if other, more technical, assumptions are satisfied, then any greater similarity in the behavior or traits of the MZ twins can be attributed to their greater genetic commonality.

The second process that can highlight the contrast between genetic and environmental contributions is adoption. A true experiment would randomly assign children immediately at birth (or, better yet, at conception) to other parents, and compare their traits and behaviors with

those of their biological and adoptive parents. Social practice very roughly approximates such an experiment by assigning children somewhat fortuitously, if nonrandomly, to adoptive parents, at some point after birth. The greater the similarity of the children to their biological parents in the trait or behavior studied, in comparison to their adoptive parents, the greater the estimated genetic contribution to that trait or behavior. A hybrid approach combines twinning with adoption, by studying the similarities of MZ twins who are reared apart. Although the logic of these comparisons has been understood and debated for over a century, the implementation of careful studies only became possible with the bureaucratic record-keeping and refined statistical techniques of the early and mid-twentieth century.

Studies conducted over the past thirty years have reported significant heritabilities for a variety of psychiatric and behavioral conditions, including schizophrenia, intelligence, neurosis, antisocial behavior, and property crime. Interestingly, they have failed to find significant heritabilities for other behavior, such as violent crime, which the public assumes to have a substantial genetic component (Raine 1993; Plomin, Owen, and McGuffin 1994; Carey 1994, 1996).

There has been much discussion about the validity of the assumptions on which these findings rest: to what extent does the departure from randomness in these studies introduce bias in the results? To what extent must, or can, we control for ways in which genetic similarities can affect rearing environments? For example, are the rearing environments of DZ twins as much alike as those of MZ twins, or are identical twins environmentally as well as genetically more alike, down to their identical wardrobes? Does the time adoptees spend with their biological parents, or do the adoption agencies' nonrandom placement practices, introduce systematic biases in the studies' results? The researchers themselves are keenly aware of these challenges to the validity of previous studies, although they tend to be more optimistic than their critics about the prospects for dispensing with controversial assumptions or controlling for their violation (Plomin et al. 1997, chs. 5, 11).

Beyond these specific methodological concerns, researchers must confront the general limitation of heritability studies we noted earlier: these studies are designed to investigate only behavioral differences, not behavior. Indeed, even with regard to behavior differences, they can reveal only variation, not causation. They proceed by calculating the variation in a population of some measure of a trait or behavior (e.g., IQ score, number of arrests) and comparing it with the genetic variation in

that population. Thus, a typical finding of an MZ/DZ twin study might be that 60 percent of the variance in IQ score in a particular population is due to the variation in genetic constitution – IQ has a heritability of 60 percent. This finding emphatically does not mean that 60 percent of the IQ of an individual is caused by his genes. In this respect, the term "heritability" can be very misleading. Nevertheless, researchers would argue that heritability studies make a valuable contribution by identifying traits and behaviors that are good candidates for genetic influence in light of the strong evidence that *something* is being genetically transmitted.

Molecular Genetics Research

Researchers and critics agree that heritability research will play a diminishing role in the behavioral genetics of the twenty-first century. But they disagree about the scientific potential of the techniques that are superseding it (Wasserman 1996). These techniques, which attempt to link behavioral and other mental traits to specific genes, advance beyond heritability research in two respects: they seek to identify the genes being transmitted, and they attempt to trace causal pathways from genes to traits or behavior, thereby moving part way from the population to the individual. On the other hand, these techniques maintain the focus of heritability research on the genetic basis for *differences* among individuals.

Researchers expect that molecular and neurogenetic research will resolve the ambiguities about causation by tracing the complex pathways through which specific genes affect traits or behavior (Carey and Gottesman 1996; Goldman 1996a, 1996b). By replacing global estimates of heritability with narrow and testable hypotheses, this work, it is claimed, will establish behavioral genetics on a solid biological foundation. Critics, on the other hand, fear that neurogenetic research will fall prey to the same kind of oversimplification as heritability research (Balaban 1996; Balaban, Alper, and Kasamon 1996).

Thus far, researchers have only identified one genetic variation closely associated with criminal behavior, in one family. It is instructive to compare the scientific issues raised by this apparent link between a *genetic* abnormality and criminal behavior with those raised almost thirty years earlier by the claimed association of a *chromosomal* abnormality with criminal behavior. The comparison suggests that old issues of interpretation will continue to confront the new molecular genetics.

From Extra Chromosomes to Mutant Genes

The first microbiological marker thought to be linked with human misbehavior was the XYY karyotype (possessed by males born with an extra Y chromosome). In 1965, researchers found an apparently high prevalence of that karyotype among prison inmates in Britain. That is to say, the percentage of prison inmates with XYY was higher than the percentage of XYY males in the general population (Jacobs et al. 1965). Unfortunately, many people quickly took this finding to be evidence of a direct link between an extra Y chromosome and a tendency to hyperaggressivity and violence. That assumption was eventually rejected by genetic researchers, but it held sway in the popular imagination long enough to stigmatize a generation of XYY males and (reportedly) lead to the abortion of a significant number of fetuses with that karyotype. It is now widely believed that if an extra Y chromosome leads to incarceration, it is by a indirect route. XYY individuals are no more aggressive than average, but they may be taller and less intelligent, hyperactive, and generally more impulsive (see Rutter, Griller, and Hagell 1998). Their increased risk of arrest or conviction may stem from an increased likelihood of getting caught, or of committing crimes more likely to be detected, rather than from heightened aggressiveness or greater disregard for social norms.

Both researchers and critics regard the XYY story as a cautionary tale, but in different ways. Where critics see an illustration of the risks inherent in any inquiry into biological markers for social behavior, researchers see a modest triumph of scientific self-correction. Critics observe that the early XYY investigators, in their rush to find a direct link between genes and behavior, assumed that an extra male chromosome would make a specific contribution to violence or aggression, instead of having the generally impairing effects typically associated with an extra chromosome. Researchers, on the other hand, note that it was behavioral geneticists who ruled out any association between the XYY karyotype and violence or aggression (while confirming the high incidence of XYY in prisons and other institutions; Witkin et al. 1976). Those findings, researchers note, came from the very studies of XYY individuals that critics vehemently opposed, on the grounds that they stigmatized their subjects and created a significant risk of a self-fulfilling prophecy.

In the twenty-five years since the XYY controversy, the techniques

for identifying biological markers may have changed more than the issues concerning their interpretation. With the development of recombinant DNA technology in the late 1970s, researchers were able to identify and manipulate individual genes and genetic material. That technology became relevant to behavioral genetics with the discovery of genetic markers for a variety of diseases and traits. These markers – highly variable (polymorphic) but functionally inert DNA segments – were found to be associated with various phenotypic traits, presumably because they were located in close proximity to genes that actually contributed to those traits. (In most cases, the markers were assumed to vary by family, such that a particular DNA variation [allele] at a given location on the genome would mark a given trait in one family, while a different allele at that location would mark that trait in another family. In other cases, it was hypothesized that the same allele would be linked with a trait in all individuals bearing it, greatly simplifying the task of identifying that allele.)

In the late 1980s and early 1990s, markers, and in some cases genes, were identified for a number of diseases known by inheritance patterns to have a significant genetic component. Behavioral geneticists were quick to adopt the same methods, hoping to replicate the dramatic success of medical genetics. They were soon reporting markers for a number of psychiatric and behavioral conditions, including bipolar disorder and alcoholism, though the first finding was retracted and the second has remained mired in controversy (Plomin et al. 1994; Holden 1994).

In 1993 researchers finally found a marker, then a gene, associated with violence and aggression, in the male members of a Dutch family (Brunner et al. 1993). Although the family was atypical in several relevant respects, the study had enormous impact. In part, this was because the affected gene was known to be responsible for a protein, MAO, involved in regulating the metabolism of serotonin, one of the neurotransmitters thought to play a critical role in mediating between genes and behavior and implicated in psychiatric and behavioral conditions ranging from depression to impulsive violence.

A comparison of the MAO and XYY studies suggests both significant advances in scientific technique and similar issues of interpretation. The connection between genotype and phenotype was closer in several respects for MAO than for XYY. First, a statistically significant association was found between MAO and aggressive and criminal behavior in one family; in contrast, no correlation was established between XYY and any form of criminal behavior until a decade after the karyotype was iden-